

MAM KALLIATH

HIV Physician Training Course 2002,  
Christian Medical College, Vellore

## DISTANCE LEARNING COURSE

# HIV AND FEVER

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**MODULE 5**

## INSTRUCTION SHEET - HIV AND FEVER (MODULE 5)

1. In addition to this module you will find X-ray 5A in separate cover for activity 5.8.
2. After you complete the module tear: (a) Tutor marked assignment (page 25); (b) the module evaluation form (at the end of the module) and enclose it the stamped envelop. Send it by registered post by: January 11, 2003.
3. Please write your name and roll number on the tutor marked assignment before dispatching it.

## OVERVIEW

Fever is a common symptom during the course of HIV infection and left untreated may cause considerable morbidity and death. Fever is usually due to an underlying infection that is eminently treatable. These infections may be due to virulent pathogens (that commonly cause infections in an immunocompetent host) or opportunistic infections (that affect immunocompromised hosts).

Short duration fevers are usually due to virulent pathogens. The etiology of prolonged fever is dependent on the stage of the disease. In the early stages of HIV disease, prolonged fever may be due to an acute sero-converting illness or virulent infections such as tuberculosis, pneumococcal pneumonia, and typhoid. In later stages of disease, prolonged fever may be due to opportunistic infections (such as cryptococcosis or toxoplasmosis) as well as virulent pathogens. Non-infectious causes of fever are less common and include malignancies and drug fever.

In most cases, fever is associated with symptoms and signs that suggest the source of fever. However immunosuppression may blunt the body's immune response and alter the typical clinical presentation of an infection. In patients with advanced HIV infection, fever may be the only evidence of a pathologic process.

Fever of unknown origin (FUO) is defined as a temperature elevation of 101<sup>0</sup> F (38.3<sup>0</sup> C) or higher for 3 weeks or longer the cause of which is not diagnosed after 1 week of intensive in-hospital investigation. FUO is classified according to the clinical setting in which it occurs: classical FUO; FUO in neutropenic patients; FUO in HIV patients; and nosocomial FUO. Not all patients with HIV infection may fit the above definition and temperature recording at home may be required to demonstrate fever when it is irregular and remittent. An alternative definition of FUO in HIV infection is a fever: (a) which has no specific localizing symptom or sign and (b) which has been present long enough for a self-limited illness to be ruled out.

Proper management of prolonged fever in HIV infection requires diagnosis of the underlying cause based on clinical symptoms and the stage of HIV disease. In contrast to classical FUO, clinical algorithms driven by the presenting symptoms guide the evaluation of fever. This module will help you to increase your knowledge about: (a) the various causes of fever; (b) clinical syndromes associated with fever; (c) and the diagnostic approach and treatment of these syndromes.

### **OBJECTIVES**

After completion of this module should be able to:

1. List the common causes of fever in Human Immunodeficiency Virus (HIV) infection.
2. Recognise the different clinical syndromes and the main causative agents/diseases responsible for them.
3. Use clinical algorithms to diagnose and manage these clinical syndromes.

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The first activity aims at the evaluation of a patient with short duration fever. Before undertaking this activity, read "WHO staging of HIV infection" (pg.29), "Fever-short duration and prolonged fever" (pg. 27), "Clinical rules in the management of fever in HIV infection" (pg. 30) and "Correlation of CD4 count and absolute lymphocyte count" (pg. 31).



### ACTIVITY 5.1

#### SHORT DURATION FEVER (TIME: 10 min)

A 25 year old man from Chittoor presents with fever of one week duration associated with chills and rigors. He was diagnosed to have HIV infection after routine screening for blood donation 6 months ago. He has been asymptomatic and has not suffered from any serious opportunistic infections. His WBC count done 2 months ago was TC 7000 cells/mm<sup>3</sup> and differential count: neutrophils 55%, lymphocytes 40%, eosinophils 2%, basophils 2%.

On examination: Weight 70 Kg, well built. No skin or mucous membrane findings, no lymph nodes enlarged. Spleen 2 cm. No cardiovascular, respiratory or neurological findings.

1. What is his stage of HIV infection based on the WHO staging?

Stage 1

2. List the likely causes of fever in this patient, -in order of probability.

Malaria, flu, ? Ur. Infection

3. What tests will you order to evaluate the fever?

Blood smear MD, CBP, Urine routine, Widal

**FEEDBACK 5.1**

1. What is his stage of HIV infection?

WHO clinical group 1.

Clinically he is asymptomatic and has no clinical signs of immunodeficiency. His absolute lymphocyte count is 2800 cells/mm<sup>3</sup> which approximately correlates to a CD4 count of >200 cells/mm<sup>3</sup>.

2. List the likely causes of fever in this patient, in order of probability.

Malaria

Typhoid

Hepatitis

Viral fever

Since he does not have clinical evidence of immunodeficiency and is presenting with a short duration fever, the clinical differential diagnosis is the same as in an immunocompetent person. The presence of splenomegaly suggests a diagnosis of malaria or typhoid. Since Chittoor is endemic for malaria, that is the most probable diagnosis.

3. What tests will you order to evaluate the fever?

Total and differential white cell count

Malarial smear - thick and thin

Widal test

Blood culture (if available)

Urine microscopy



## ACTIVITY 5.2

## SHORT DURATION FEVER- II (TIME: 10 min)

His MP smear is found to be positive for *P. vivax*.

1. What treatment would you start him on?

Chloroquine + Primaquine (By 20) 45 days  
4+4+2

2. Is there any role for prophylaxis for malaria for him?

Yes. - Though presently not immunocompromised  
immunos challenges from intercurrent infection  
could speed up immunodeficiency



**FEEDBACK 5.2**

His MP smear is found to be positive for *P. vivax*.

1. What treatment would you start him on?

Chloroquine 600 mg (4 tablets) - Day 1

Chloroquine 600 mg (4 tablets) - Day 2

Chloroquine 300 mg (2 tablets) - Day 3

Primaquine 15 mg OD for 5 days

(NMEP regimen)

2. Is there any role for prophylaxis for malaria for him?

There is no increased risk for the development of malaria in HIV infection. Since he is residing in an endemic area, there is no role for malaria prophylaxis. ?

This activity is designed to give you practice in clinical evaluation of a HIV infected person who presents with prolonged fever. Before doing this activity, read "History in a patient presenting with fever" and "Clinical examination findings in fever" (pg. 28) in the reader.



### ACTIVITY 5.3

## CLINICAL EVALUATION OF PROLONGED FEVER

(TIME: 20 min)

Mr. Vengaih, 35 year old manual labourer from Namakkal, was diagnosed to have HIV infection two years ago. He presented with symptoms of fever and chills of two months duration.

1. What are the specific questions you would like to ask in the history?

- relating to immunosuppression
- relating to TB & respiratory
- Neurologic syndrome
- OI prophylaxis / history
- clinical staging

2. What are the clinical findings that you would look for?-

- Lymphadenopathy & immunosuppression w/ TB
- Respiratory signs
- CNS signs
- Skin manifestations
- ? Anorectal

**FEEDBACK 5.3**

1. What are the specific questions you would like to ask in the history?

1. Duration of HIV infection
2. Associated symptoms – respiratory, CNS, urinary, abdominal
3. History of tuberculosis and TB treatment
4. Other opportunistic infections
5. Opportunistic Infections prophylaxis
6. Highly active antiretroviral therapy
7. Co-existent morbidity: Alcoholism, IVDU

2. What are the clinical findings that you would look for?

Oral candida, oral hairy leukoplakia, pigmentation  
Significant generalized lymphadenopathy  
Skin lesions - nodules, papules  
Hepatosplenomegaly, intra-abdominal glands  
Respiratory distress, lower respiratory signs  
Neck stiffness  
Limb weakness  
Fundal lesions - haemorrhages  
Elevated JVP, heart murmurs  
Genital examination - ulcers, glands  
Per Rectal examination - prostatic tenderness and fluctuation

The next exercise focuses on the differential diagnosis of prolonged fever. Before you undertake the exercise, study: "Pyrexia of unknown origin-classification" (pg. 27); "Etiology of fever -correlation to CD4 counts" (pg. 31) and "Frequency of etiologies of prolonged fever at Vellore (pg. 32).



#### ACTIVITY 5.4

### DIFFERENTIAL DIAGNOSIS OF FEVER

(TIME: 15 min)

Mr. Vengaiah was diagnosed to have HIV infection when he consulted his local GP for recurrent genital ulceration. He had significant loss of weight and appetite. He had noticed darkening of skin and loose stools on and off. He also had occasional headache.

On examination: An emaciated individual, temperature-38°C, respiratory rate-24/min, Pulse rate-100/min. Darkening of palms and soles and generalized pruritic papular rash. Two 0.5 x 0.5 cm lymph nodes in the deep cervical region, a few small axillary nodes. Abdominal examination-mild hepatosplenomegaly. CNS examination-no signs of meningeal irritation, no papilloedema or focal deficits.

1. What is his WHO clinical stage? *Stage III*

Based on his clinical stage, what differential diagnosis would you consider in order of probability?

Stage - *Stage III*

Interpretation of clinical signs -

Differential diagnosis - *TB - Pulm or extra pulm (disseminated)*  
*Lymphomas / G.I. Infections.*

**FEEDBACK 5.4**

1. What is his clinical stage of HIV infection?

Based on his clinical stage what differential diagnosis would you consider in order of probability?

Stage

WHO clinical group 3

Interpretation of clinical signs

Pruritic rash and pigmentation indicate significant immunodeficiency. Generalised lymphadenopathy and hepatosplenomegaly may indicate the presence of disseminated TB. The increased respiratory rate may indicate respiratory involvement probably due to pulmonary TB.

Differential diagnosis:

Disseminated TB

Cryptococcosis

Lymphoma

The next exercise aims to make you familiar with the P<sub>UO</sub> algorithms. Before you do the exercise, study:

**Fever, respiratory and central nervous system algorithms**  
(pg. 36-38).

**ACTIVITY 5.5****PUO ALGORITHMS (TIME: 10 min)**

1. Which P<sub>UO</sub> algorithm will you use in Mr. Vengaiiah's case (the patient referred to in Activity 5.3 and 5.4)?

Which tests will you order in correct sequence?

Algorithm

1

**Tests**

Step 1 - FNB

Step 2 - Ultrasound - Head + FNB

Step 3 - Culture for AFB of bone marrow

**FEEDBACK 5.5**

Which algorithm will you use? Which tests will you order in correct sequence?

Algorithm - Fever algorithm (Figure 1).

Tests:

Step 1- Chest x-ray, sputum AFB

Step 2 - Lymph node FNAC or smear/ ultrasound of the abdomen

Step 3 - Bone marrow

Step 4 - Liver biopsy

The list and order of tests in the algorithm provide a guide to investigation. These may require modification in the individual case based on clinical judgement.

The next exercise aims to make you familiar with the empiric therapy in PUO. Before you do the exercise, study: "Prolonged fever etiological agents" (pg. 35) and "TB in relation to prolonged fever in HIV infection" (pg. 33-34).



### ACTIVITY 5.6

#### EMPERIC THERAPY (TIME: 10 min)

Mr. Vengaiah's laboratory reports are as follows:

WBC Total count: 4200 cells/mm<sup>3</sup> Neutrophils 75, Lymphocytes 20, Eosinophils 3, basophils 2.

Chest x-ray - Bilateral hilar adenopathy. Sputum AFB - negative. Lymph node FNAC was non-diagnostic. Ultrasound of abdomen- multiple hypoechoic areas in the liver and spleen, no lymph nodal masses. Bone marrow smear and biopsy- no specific lesions. In view of financial constraints a liver biopsy was not attempted.

1. What is your interpretation of the findings?

*Resp* Focalizing lesion, with disseminated feature, leucopenia and hilar masses and hypoechoic lesions in liver & spleen suggest infiltration - Disseminated TB, Cryptococcal meningitis

2. What treatment will you start at this point, mentioning drug regimens?

TB - Regime I - 2 RHZE<sub>3</sub>  
4 RH<sub>3</sub>





### FEEDBACK 5.6

1. What is your interpretation of the findings?

There is absolute lymphopenia indicating advanced immunodeficiency. The hilar adenopathy and hypoechoic lesions suggest a disseminated infiltrative process which is probably tuberculosis. Cryptococcal infection is still a possibility but there are no meningeal signs.

2. What treatment will you start at this point mentioning drug regimens?

The most probable diagnosis in this patient is disseminated tuberculosis for the following reasons: (a) clinical features point to the diagnosis; and (b) TB is the most common cause of prolonged fever in HIV infection. The negative result on sputum AFB testing does not exclude the diagnosis.

The high probability of diagnosis and the negative investigation workup warrant the initiation of empiric anti-tubercular therapy. He should be followed up carefully to assess response to therapy. In the case of failure of response further diagnostic evaluation would be indicated.

This patient should be started on short course chemotherapy. According to the RNTCP program this patient would fit into Category 3 (seriously ill extra-pulmonary disease-disseminated TB).

His drug regimen will be as follows:

2(HRZE)<sub>3</sub>, 4(HR)<sub>3</sub>. Rifampicin 450 mg, Isoniazid 600 mg, Pyrazinamide 1500 mg and Ethambutol 1200 mg thrice weekly for 2 months. Followed by Rifampicin 450 mg, Isoniazid 600 mg thrice weekly for 4 months.

The principles of drug therapy, drug choice, dose and duration are the same as in a immunocompetent patient with tuberculosis. Some physicians would extend the total duration of treatment up to 1 year.

He was started on empirical anti-tuberculous therapy with which he noted remarkable improvement.

This activity takes you through the steps for evaluating a patient with fever and respiratory symptoms.



**ACTIVITY 5.7**

**FEVER AND RESPIRATORY SYMPTOMS -  
DIFFERENTIAL DIAGNOSIS (TIME: 10 min)**

Mr. Albert, 45 years old a known case of HIV infection presents with complaints of fever, cough and slowly progressive breathlessness of 2 weeks duration.

He has lost 10 kg of weight. He has a history of diarrhoea on and off. There is no history of dimness of vision or headache.

He received a complete course of tuberculosis treatment 20 years ago for pulmonary TB.

On examination: 50 Kg. He is tachypneic at rest. Respiratory rate- 40/mt., temperature 37.6° C and blood pressure 110/70 mm Hg. On examination of the respiratory system there are crackles in right infrascapular and infraaxillary regions and an occasional wheeze. Central nervous system, including fundus, was normal.

*not significant pathology*

1. Write down your differential diagnosis in order of probability and also state the reasons for your first diagnosis?

*Dulm. T.B - Past history of cough & slowly progressive tachypnea along with crackles in lower chest denotes a parenchymal problem.*  
*bact. pneumonia - However not rapid in onset*  
*Cryptos - No supportive signs*

2. What tests will you order to confirm your diagnosis?

*Routine blood, sputum chest, PNAc if relevant, Sputum Bacteriology (after enhancement) Gram stain - Ultrasound abdomen and related follow up*

**FEEDBACK 5.7**

Write down your differential diagnosis in order of probability and also state the reasons for your first diagnosis?

Differential diagnosis:

1. Pneumocystis carinii
2. Pulmonary TB-relapse
3. Bacterial pneumonia

Reasons for considering PCP as first diagnosis:

Previous complete TB treatment makes TB less likely.

Symptoms of dry cough, progressive breathlessness, minimal respiratory signs and respiratory distress make PCP the likely diagnosis.

He has not taken TMP/SMX prophylaxis.

What tests will you order to confirm your diagnosis?

Chest X-ray

Sputum AFB smear and gram stain

Induced sputum with 3% saline for PCP smear (silver staining)

Serum LDH

Pulse oximetry



## ACTIVITY 5.8

### FEVER AND RESPIRATORY SYMPTOMS- MANAGEMENT (TIME: 10 MIN)

? Pulse oximetry - oxygen saturation 80%

Serum LDH - 800 U/L, Sputum AFB smear - negative

? Induced sputum for PCP smear could not be performed.

Chest x-ray- 5-A (in labeled cover)

1. Write down the abnormalities on the chest x-ray. State the differential diagnosis?

Findings: - Prominent bronchial markings  
- ? Intersegmental infiltrates (R) (L)  
- no evidence of consolidation/effusion

Differential diagnosis:

1. PCP
2. Broncho pneumonia
3. Cryptococcal
4. ? disseminated Kochs

2. What treatment will you start this patient on (drug, dose and duration)?

- TMP+SM 2 bd x 2 wks + 20D for life  
- Oxygen  
- Predni colone - 10mg tid - taper of 3 weeks

3. If he failed to respond to the above treatment what further tests will you require?

- ? Bronchoscopy  
- Biopsy

**FEEDBACK 5.8**

1. Write down the abnormalities on the chest x-ray. State the differential diagnosis?

Findings: Bilateral diffuse interstitial infiltrates.

Differential diagnosis:

1. PCP
2. TB
3. CMV
4. Cryptococcal pneumonia
5. Lymphocytic interstitial pneumonitis
6. Non-Hodgkin's lymphoma

2. What treatment will you start this patient on?

Co-trimoxazole (TMP 160 mg/SMX 800 mg) 2 tablets three times per day × 21 days.

In the critically ill patient the IV co-trimoxazole may be used (dose TMP 20mg/kg/day IV q6-8h).

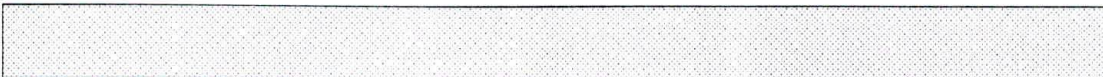
This patient has significant hypoxemia based on the pulse oximeter reading and therefore warrants the initiation of corticosteroid therapy at the following dose:

Prednisolone 40 mg twice daily × 5 days; followed by Prednisolone 40 mg once daily × 5 days; and then followed by Prednisolone 20mg once daily for 11 days.

3. If he failed to respond to the above treatment what further tests will you require?

If he fails to respond to the above treatment bronchoscopy and bronchoalveolar lavage will be required.

Mr. Albert responded to co-trimoxazole and prednisolone combination which he received for 21 days. Following this he was started on life long TMP/SMX prophylaxis with TMP/SMX DS one tablet daily.



The next exercise focuses on the evaluation of fever and headache.



### ACTIVITY 5.9

#### FEVER AND HEADACHE – I (TIME: 10 min)

Mr. Sundaram, 37 years old was diagnosed to have HIV infection 5 years ago, received complete anti-TB treatment for TB lymphadenitis and is currently on Bactrim prophylaxis. He presents with fever of 3 months and headache with vomiting for 20 days. On examination his general physical examination is normal. He has no neck stiffness, fundus is normal and there are no focal neurological deficits. - He had no sinus tenderness.

What diagnoses will you consider and what tests will you order?

- Meningitis - Cryptococcal / Tubercular  
 - Space occupying lesion  
 - Encephalitis

CSF - biochemical, microscopy, special stain, ~~proteins~~  
 - MRI  
 cultures

**FEEDBACK 5.9**

What diagnoses will you consider and what tests will you order?

Diagnosis: Chronic meningitis probably due to cryptococcal infection.

Tests:

CSF - opening pressure, total count, sugar, protein, gram stain, India ink, AFB smear.

Routine, AFB and fungal cultures if available.

Chest X-ray and sputum AFB smear and malarial parasite and Para-nasal sinus x-ray.



## ACTIVITY 5.10

## FEVER AND HEADACHE – II (10 min)

Mr. Sundaram's laboratory reports are as follows:

CSF- Pressure 180 mm. Total cell count -100 cells/mm<sup>3</sup>

Differential cell count - Lymphocytes 96 Neutrophils 4.

Protein - 44 mg/dl, sugar - 53mg/dl, India Ink test - negative.

Fungal culture - *Cryptococcus Neoformans*.

Chest x-ray and sputum AFB negative.

What treatment will you administer to this patient (drug, dose and duration)?

Amphotericin B - 7mg/kg/day x ?

~~200mg~~

Fluconazole 800mg stat + 400mg x 8wks + 200mg/d lifelong



**FEEDBACK 5.10**

What treatment will you give?

T. Fluconazole 800 mg stat, followed by 400 mg OD for 8-12 weeks.

Then lifelong prophylaxis with Fluconazole 200 mg once daily.

Fluconazole may be given to this patient as he has good prognostic features: consciousness preserved, increased CSF cell count, normal sugar, CSF pressure normal.

## NOTES

- Hematology - Liver, liver, kidney, Serology
- Biochemistry - Rontin ambiguous,
- Micro. - Smears - (No cultures / biopsy)
- X-ray / Ultrasound

- TB - Pulm / disseminated
- Malaria
- Typhoid
- chr. focal infections - Ur. tract / brain / ocular

- Resp. - TB
- Cryptococcal
  - PCP
  - Lymphocytic infiltrates

Cerebral - - Meningitis - TB / Crypto / syph.

- Focal - CMV. / Herpes disseminated
- Conception - Toxoplasma

- Lymphomas
- Drug fever



## READINGS

**PYREXIA OF UNKNOWN ORIGIN - CLASSIFICATION (Durack and Street)**

	CLASSIC	NEUTROPENIC	HIV	NOSOCOMIAL
<b>Patient type</b>		< 500 neutrophils/mm <sup>3</sup>	Seropositive	Hospitalised
<b>Duration</b>	3 days hospitalization or 3 OP visits	3 days hospitalization	3 days hospitalization or 4 weeks	3 days hospitalization
<b>Etiologies</b>	Infections Malignancy Inflammation	Peri-anal infection Aspergillosis Candidemia	TB MAC PCP Toxoplasmosis	Urinary infection Respiratory infection Drugs Phlebitis

MAC- Mycobacterium avium intracellulare

TB - Tuberculosis

PCP - Pneumocystis carinii

**SHORT DURATION FEVER AND PROLONGED FEVER**

<b>Short duration fever (&lt;2 weeks)</b>	<b>Prolonged fever (&gt; 3 weeks)</b>
Viral fever	Tuberculosis
Malaria	Pneumocystis carinii pneumonia
Typhoid	Cryptococcosis
Bronchitis/sinusitis	Toxoplasmosis
Pneumococcal pneumonia or bacteremia	Disseminated Cytomegalovirus infection
Urinary tract infection	Disseminated Mycobacterium avium-intracellulare infection
Pyogenic skin infections	Disseminated herpes infection
<i>S. typhimurium</i> septicaemia	Infective endocarditis
	Lymphoma
	Drug fever

**SPECIFIC HISTORY IN A PATIENT PRESENTING WITH FEVER**

1. Duration of HIV infection
2. Associated symptoms - respiratory (cough, breathlessness and sputum), CNS (headache, seizures, limb weakness, visual symptoms), urinary (dyuria), abdominal (abdominal pain, diarrhea, vomiting and jaundice).
3. History of tuberculosis and TB treatment
4. Other opportunistic infections
5. Opportunistic Infections prophylaxis
6. Highly active antiretroviral therapy
7. Co-existent morbidity - Alcoholism, IVDU
8. History to enable clinical staging - Degree of weight loss, activity level, mucocutaneous lesions

**CLINICAL EXAMINATION FINDINGS IN FEVER**

<b><u>Findings</u></b>	<b><u>Inference</u></b>
Oral candida, oral hairy leukoplakia, pigmentation	Signs of immunodeficiency
Sinus tenderness and nasal discharge	Sinusitis, upper respiratory tract infection
Significant generalized lymphadenopathy	TB, cryptococcosis, lymphoma
Neck stiffness	Chronic meningitis
Fundal lesions- hemorrhages	CMV retinitis
Skin lesions- nodules, papules	Cryptococcosis, nocardiosis, Penicillium infection, TB
Respiratory distress, lower respiratory signs	Pneumonia, PCP, TB, Plueral effusion
Elevated JVP, heart murmurs	Infective endocarditis, pericardial effusion
Hepatosplenomegaly, intra-abdominal glands	TB, Lymphoma
Genital examination-ulcers, glands	STD's
Pelvic examination- forniceal tenderness	Pelvic inflammatory disease
Per rectal examination- Prostatic tenderness and fluctuation, peri-anal abscess	Prostatic abscess
Limb weakness	Focal cerebral lesion-toxoplasmosis

Proposed WHO staging system for patients infected with HIV

Stage 1:

Asymptomatic

Persistent generalised lymphadenopathy

Stage 2:

Weight loss between 5% and 10% of body weight

Minor mucocutaneous manifestations (seborrhoeic dermatitis, prurigo, fungal nail infections, recurrent oral ulcerations, angular stomatitis)

Herpes zoster within the past five years

Recurrent upper respiratory tract infections (for example, bacterial sinusitis)

*And/or*

Performance scale 2: symptomatic, normal activity

Stage 3:

Weight loss >10% body weight

Unexplained chronic diarrhoea for longer than one month

Unexplained prolonged fever (intermittent or constant) for longer than one month

Oral candidiasis

Oral hairy leukoplakia

Pulmonary tuberculosis within last year

Severe bacterial infections (for example, pneumonia, pyomyositis)

*And/or*

Performance scale 3: bedridden for less than 50% of the day during the last month

Clinical stage 4 (AIDS):

HIV wasting syndrome

Pneumocystis carinii pneumonia

Toxoplasmosis of the brain

Cryptosporidiosis with diarrhoea for more than one month

Cryptococcus, extrapulmonary

Cytomegalovirus infection of an organ other than liver, spleen, or lymph nodes

Herpes simplex virus infection mucocutaneous for more than 1 month or visceral of any duration

Progressive multifocal leukoencephalopathy

Any disseminated endemic mycosis

Candidiasis of the oesophagus, trachea, bronchi, or lungs

Atypical mycobacteriosis, disseminated

Non-typhoidal salmonella septicaemia

Extrapulmonary tuberculosis

Lymphoma

Kaposi's sarcoma

HIV encephalopathy

*And/or*

Performance scale 4: bedridden for more than 50% of the day during last month

**CLINICAL RULES IN THE MANAGEMENT OF FEVER IN HIV INFECTION**

- Major opportunistic infections are uncommon with a CD<sub>4</sub> counts above 200 cells/mm<sup>3</sup>.
- Patients with HIV infection and a CD<sub>4</sub> count exceeding 500 cells/mm<sup>3</sup>. should be evaluated for fever as an immunocompetent host.
- Patients with CD<sub>4</sub> cell counts of 200-500/cu.mm. are at increased risk of infections caused by relatively virulent pathogens that occur in the immunocompetent hosts as well.
- Three-fourths of prolonged fevers are due to tuberculosis. Therefore in the case of a negative diagnostic evaluation, empiric anti-TB treatment is warranted.
- In the following situations suspect unusual causes of fever:

**Prolonged fever**

- In a patient who has completed TB treatment
  - Occurring at low CD<sub>4</sub> count (<50-100 cells/mm<sup>3</sup>)
  - With skin lesions, fundal lesions
  - Not responding to empiric anti-TB treatment
  - In an IV drug user
- Approach to short duration fever is similar to an immunocompetent host. However investigation and empiric therapy may start earlier.

**CORRELATION OF CD4 COUNT AND ABSOLUTE LYMPHOCYTE COUNT**

- Absolute lymphocyte count =  $\frac{\text{WBC total count} \times \text{Lymphocyte count}}{100}$
- Absolute lymphocyte count (ALC) of 1000 cells/mm<sup>3</sup> roughly correlates to a CD4 count 200 cells/mm<sup>3</sup>.
- The positive predictive value of ALC < 1000 cells for a diagnosis of AIDS is 88%.

*Journal of Association of Physicians of India 1997; 45: (6):455-6.*

**Etiology of fever in HIV infection - correlation to CD<sub>4</sub> counts**

CD <sub>4</sub> counts cells/mm <sup>3</sup>	Etiologies of fever
> 500	Acute retroviral syndrome Causes of fever in non-immunocompromised patient
200-500	Bacterial pneumonias Tuberculosis Herpes zoster Lymphomas
<200	Pneumocystis Carinii Pneumonia (PCP) Disseminated/Chronic herpes simplex Toxoplasmosis Cryptococcosis Disseminated histoplasmosis Tuberculosis (TB) (miliary/extrapulmonary)
<50	Disseminated Cytomegalovirus (CMV) Disseminated MAC

? Not including list above

16889  
DIS-325p02

**CAUSES OF PROLONGED FEVER IN HIV INFECTION****(STUDY OF 100 PATIENTS AT CMCH, VELLORE FROM REFERENCE 3)**

Disseminated TB (definite)	21%	66%
Disseminated TB (presumed)	19%	
Pulmonary TB	16%	
Extra-pulmonary TB	10%	
Pneumocystis Carinii	7%	
Cryptococcosis	10%	
Cerebral Toxoplasmosis	1%	
Bacterial pneumonia	2%	
Amoebic liver abscess	2%	
Disseminated Histoplasmosis	1%	
Sinusitis	1%	
Spontaneous Peritonitis	1%	
Pyogenic meningitis	1%	
Malaria	1%	

**UNUSUAL CAUSES OF FUO****Drug Fever**

- Usually accompanied by a skin rash.
- Associated with TMP/SMX, dapsone, penicillin, cephalosporins, phenytoin, carbamazepine and clindamycin.
- Resolves within 24-48 hours after withdrawal of drug.

**Immune reconstitution Syndrome**

- Patients initiated on HAART may develop fever, leucocytosis, lymphadenopathy and worsening chest x-ray in the first few weeks to months.
- This is due to development of a vigorous immune response to sub-clinical opportunistic infections.
- It has been frequently reported with MAI and tuberculosis.



### TB IN RELATION TO PROLONGED FEVER IN HIV INFECTION

1. The commonest cause of HIV related FUO in India is tuberculosis.
2. Disseminated and extra-pulmonary TB (lymph node, pleural effusion, abdominal TB) occur frequently with advanced HIV infection.
3. Clinical pointers to a diagnosis of tuberculosis include: hepatosplenomegaly, weight loss and lymph node enlargement.
4. Abnormal laboratory findings in disseminated TB include: anemia, elevated alkaline phosphatase and lymphopenia (<1500/mm<sup>3</sup>).
5. Chest X-ray findings of tuberculosis in HIV infection may be atypical. These findings include interstitial or lobar infiltrates and lower lobe sub-segmental infiltrates, hilar and mediastinal lymphadenopathy and pleural effusion.
6. The principles of chemotherapy of tuberculosis in an HIV infected patient are the same as an immunocompetent host.
7. There is no evidence that multi-drug resistant (MDR) tuberculosis occurs with increased frequency in the HIV infected patients in India.
8. In a patient presenting with prolonged fever and a negative physical examination and laboratory investigation, empiric anti-tuberculosis treatment may be justified.
9. Disseminated mycobacterium avium intracellulare (MAI) infection is uncommon in India. Prior tuberculosis could confer immunity against atypical mycobacteria. It is also possible that patients do not survive till to the stage of advanced immunodeficiency at which MAI infections occur.

**TUBERCULOSIS TREATMENT RECOMMENDATIONS**  
**REVISED NATIONAL TUBERCULOSIS CONTROL PROGRAM**

Category of Treatment	Type of patient	Regimen*
Category I	New sputum smear positive Seriously ill sputum smear negative Seriously ill extra-pulmonary tuberculosis**	2(HRZE) <sub>3</sub> 4(HR) <sub>3</sub>
Category II	Sputum smear positive relapse*** Sputum smear positive failure*** Sputum smear positive treatment after default	2(HRZES) <sub>3</sub> 1(HRZE) <sub>3</sub> 5(HRE) <sub>3</sub>
Category III	Sputum smear negative Extra-pulmonary not seriously ill	2(HRZ) <sub>3</sub> 4(HR) <sub>3</sub>

\* The number before the letters refers to the months of treatment. The subscript after the letters refers to the number of doses per week. H: Isoniazid (600 mg), R: Rifampicin (450 mg), Z: Pyrazinamide (1500 mg), E: Ethambutol (1200 mg), S: Streptomycin (750 mg). Patients weighing more than 60 Kg should receive an additional dose of Rifampicin of 150 mg. Patients of age > 50 year or weight < 30 Kg should receive streptomycin 500 mg. Patients in category I and II who are sputum smear positive at the end of the intensive phase of treatment should receive an additional month of intensive phase of treatment.

\*\* Examples of seriously ill extra-pulmonary TB cases are meningitis, disseminated TB, tuberculous pericarditis, bilateral and extensive pleurisy, spinal TB with neurological complications and intestinal and genitourinary TB.

\*\*\* In rare and exceptional cases, patients who are smear negative or have extra-pulmonary disease can have relapse or failure. The diagnosis in all such cases should be supported by culture or histological evidence of active tuberculosis. In these cases the patients should be categorized as "other" and given category II treatment.

**Prolonged Fever - Etiological Agents**

Agent	Course	Typical Findings	Diagnosis	Treatment
<b>Tuberculosis (MTB)</b>	Chronic, sub-acute or asymptomatic usually has productive cough ± hemoptysis	Variable: focal infiltrates, reticulonodular, cavitary disease, hilar adenopathy, lower and middle lobe involvement common, pleural effusion; early-stage HIV infection-upper lobe cavitary; late-stage HIV-pneumonitis mid or lower lobes or miliary pattern with minimal granuloma formation. Extrapulmonary TB is common - esp. meningitis, adenopathy	Sputum AFB stain and culture, if no sputum production - induced sputum ; requires 3-8 weeks for growth on conventional media; sensitivity of sputum AFB smear - 50%	See page 34.
<b>M. arium Complex</b>	Chronic or asymptomatic	History- Fever, weight loss, night sweats, diarrhea. Examination- lymphadenopathy, hepatosplenomegaly	Sputum, FOB or induced sputum AFB stain and culture; must distinguish from MTB; MA may colonise airways without causing pulmonary disease.	Clarithromycin + ethambutol ± rifabutin*
<b>S.Pneumoniae</b>	Acute; purulent sputum ± pleurisy	Lobar or broncho-pneumonia ± pleural effusion	Blood cultures often positive; sputum gram stain, culture (sensitivity of culture is 50%).	Oral: Amoxicillin, IV: Crystalline penicillin
<b>Cryptococcus</b>	Chronic, sub-acute or symptomatic	Nodule, cavity, diffuse or nodular infiltrates	Sputum, induced sputum, or FOB stain and culture; serum cryptococcal antigen usually positive; CSF analysis indicated if antigen or organism found at any site.	Fluconazole or Amphotericin B + Flucytosine*
<b>Pneumocystis Carinii</b>	Acute or sub-acute; non-productive cough; dyspnea	Interstitial infiltrates with characteristic ground glass appearance; negative x-ray in early stages about 15-20%;	Induced sputum (mean yield of 60%) and FOB with BAL (mean yield of 95%); technical expertise is highly variable.	TMP-SMX or Pentamidine pO <sub>2</sub> < 70 mm Hg; Prednisone*

\* See Sanford Guide to HIV/AIDS therapy for details of drug doses

**MANAGEMENT ALGORITHMS FOR HIV-INFECTED PATIENTS WITH PROLONGED FEVER**

Fig. 1

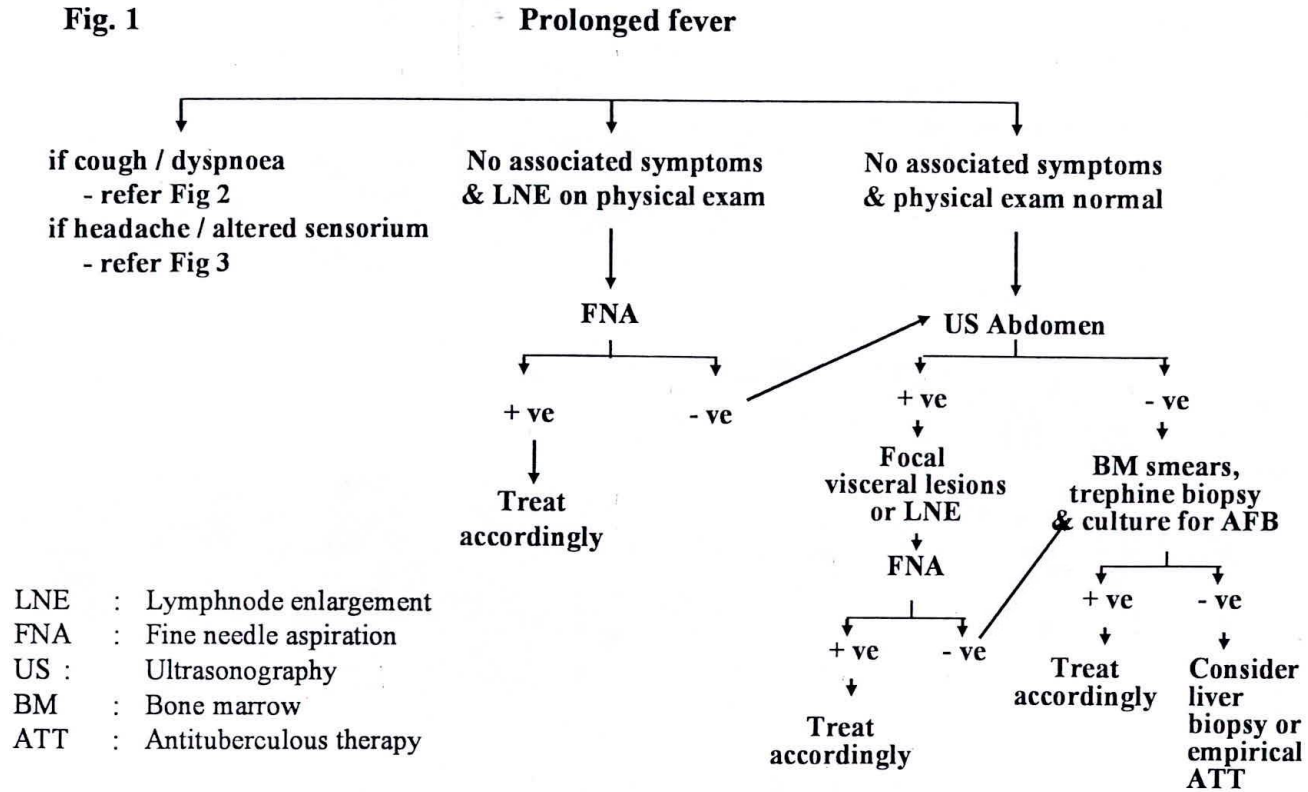


Fig. 2 Prolonged fever and pulmonary symptoms

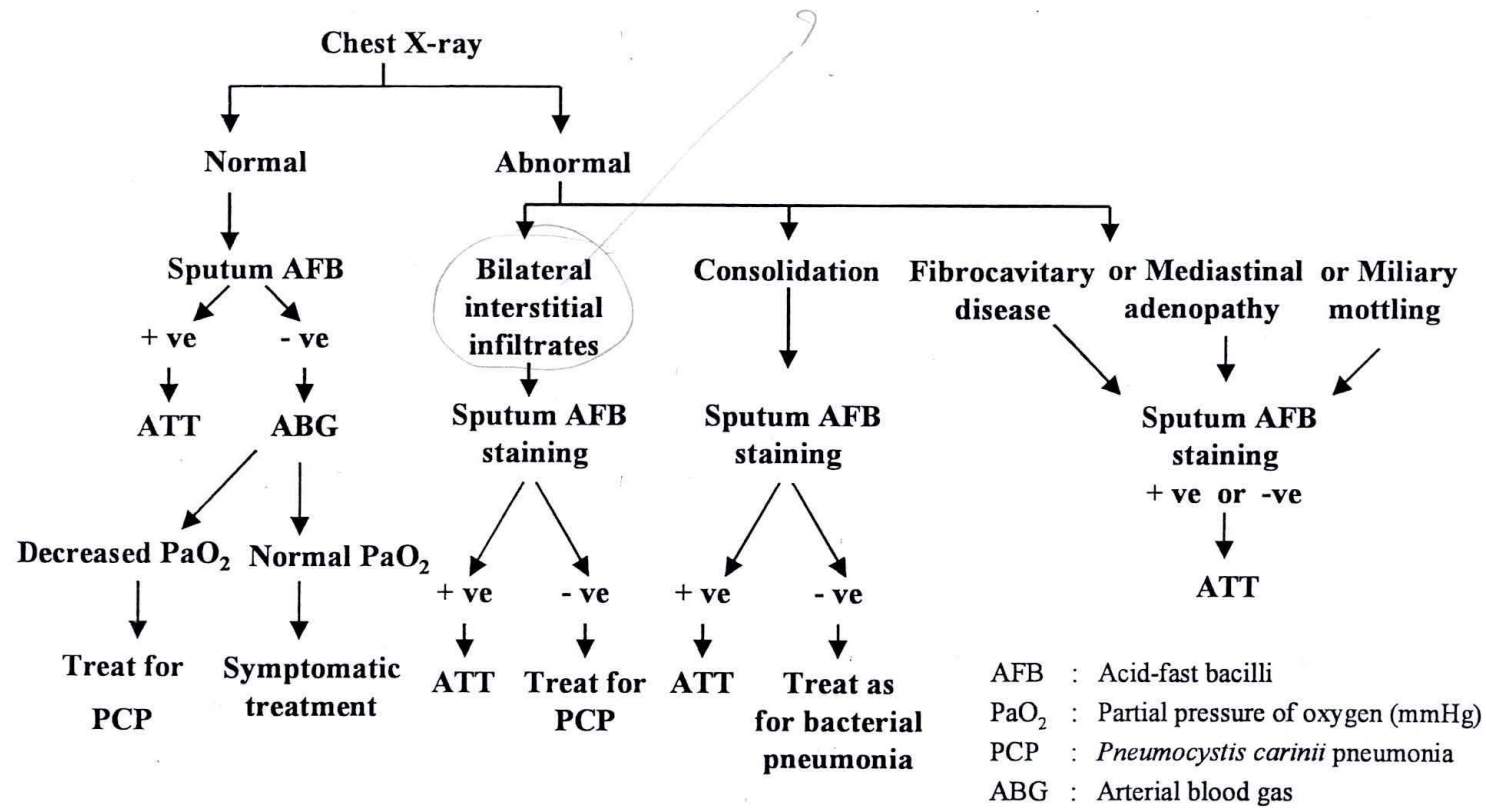
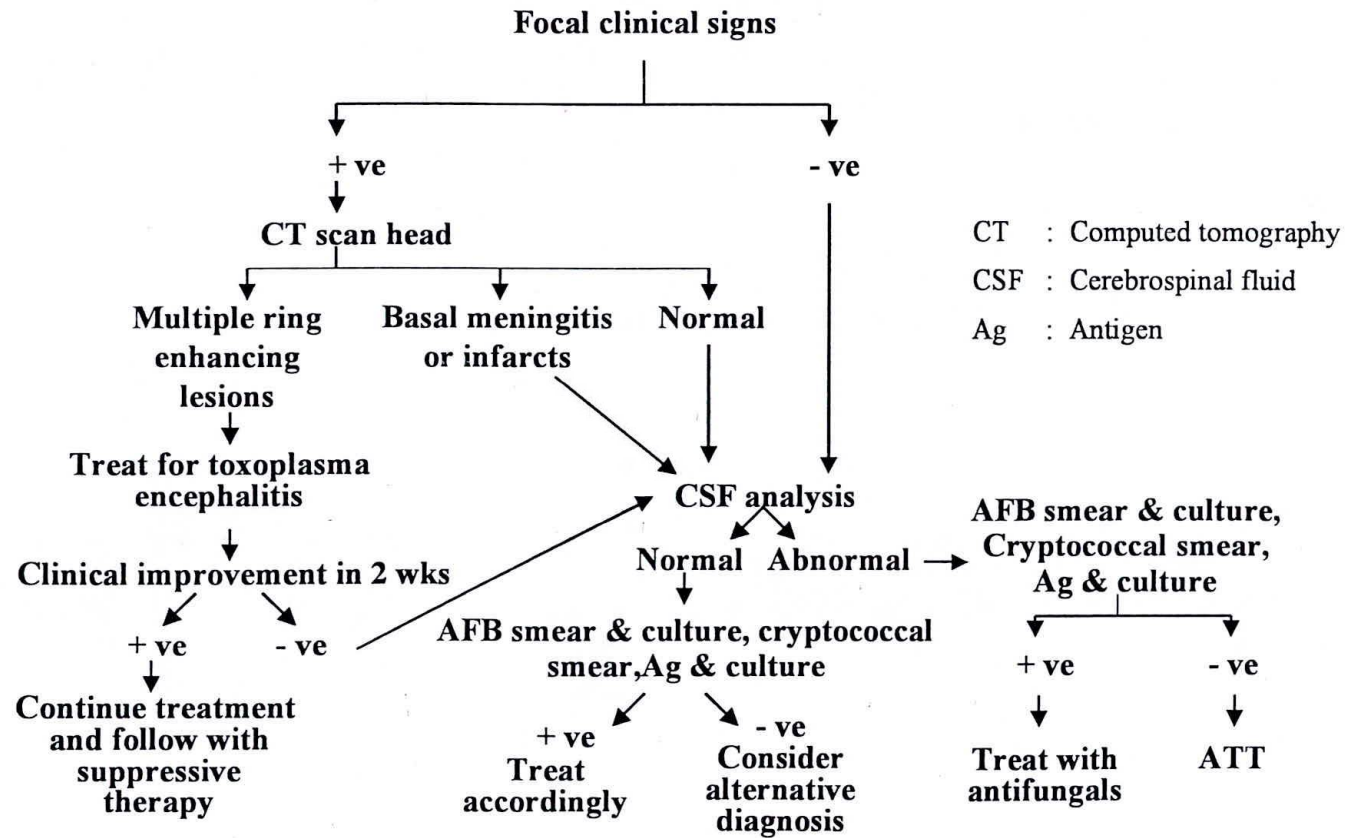


Fig. 3 Prolonged fever and neurological symptoms



**REFERENCES**

1. USPHS/IDSA (2002) Guidelines for the prevention of opportunistic infections in persons infected with Human Immunodeficiency Virus- 2002.  
<http://www.cdc.gov/mmwr/PDF/RR/RR5108.pdf>
2. Rupali P. (1998) Profile of prolonged fever in HIV infection. *In* M.D dissertation to the M.G.R Medical University.
3. Sullivan M, Feinberg J. Bartlett JG (1996) Fever in patients with HIV infection. *Infect Dis Clin North Am* ; 10 : 149-165.
4. World Health Organization (1990) Acquired immune deficiency syndrome (AIDS): interim proposal for a WHO staging system for HIV-1 infection and disease. *Wkly Epidemiol Rec* 65: 221-228.

DISTANCE LEARNING COURSE

**INFECTION CONTROL  
&  
EXPOSURE PREVENTION**

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**MODULE 6**



**INSTRUCTION SHEET -INFECTION CONTROL AND EXPOSURE**  
**PREVENTION (MODULE 6)**

1. After you complete the module tear: (a) Tutor marked assignment (page 31-32); (b) the module evaluation form (at the end of the module) and enclose it the stamped envelop. Send it by registered post by: January 18, 2003.
2. Please write your name and roll number on the tutor marked assignment before dispatching it.

## OVERVIEW

Today HIV physicians are faced with the challenge of providing quality HIV care to patients while also ensuring a safe working environment for the hospital staff. The HIV epidemic has provided us with a better understanding of the risks from blood borne pathogen to health care workers. This has helped define approaches to reduce these risks.

The first case of transmission of HIV infection to a health worker was documented in 1984. Till 1997 there were 95 proven cases and 191 probable cases of occupational transmission of HIV worldwide. The magnitude of the problem and risks posed by occupational transmission of Hepatitis B and Hepatitis C is far greater.

The chief risk factors for occupational transmission are: (i) contaminated needle stick injuries (risk of transmission from needle stick injury 0.3% for HIV, 1.8% for Hepatitis C and 6-30% for Hepatitis B) and (ii) mucous membrane or non-intact skin exposures (risk of transmission 0.09% for HIV). Out of the 267 cases of occupationally acquired HIV in United States, 35% were in nurses, 16% in laboratory technicians, 10% in physicians and 6% in surgeons.

The Center for Disease Control in 1987 has identified "Universal Precautions" as the central approach to prevention of transmission of blood borne pathogens. In the application of universal precautions, all patients and their blood and specified body fluids are regarded as potentially infectious. The two cardinal principles of universal precautions are: (i) reduction of exposure to infectious body fluids by use of physical barriers (gloves, masks, protective eye wear and gown) and (ii) safe disposal of sharps to prevent needle injuries.

The other important components of a health worker safety program are:

Mandatory hepatitis B vaccination of all health workers at risk

Waste segregation and disposal

Regular worker education

Reporting services for occupational exposures

Provision of post-exposure prophylaxis

Introduction of new safety devices

This module focuses on how to implement these policies at the level of your facilities and hospitals. The practical aspects of these programs and detailed discussion of segregation and disposal of wastes and health worker education will be covered during the Contact Course II.

## OBJECTIVES

After completing this module you should be able to:

1. Define the extent of the problem of infections due to blood borne pathogens among health workers in India and the risks posed by specific pathogens and types of exposures.
2. Outline the components of a safety program for health care workers to reduce risks of transmission of blood borne pathogens with specific reference to:
  - (a) Implementation of universal precautions
  - (b) Hepatitis B vaccination
  - (c) Exposure reporting service
  - (d) Provision of post-exposure prophylaxis.
3. Identify the weaknesses of infection control practice in your set-up and outline possible strategies to address these weaknesses.

<b>CONTENTS</b>
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	<u>Title</u>	<u>Time (minutes)</u>	<u>Page</u>
<b>Activity 6.1</b>	<b>Blood borne pathogen infections in health workers in India</b>	<b>10</b>	<b>4</b>
Reading	<i>Occupational exposures in US</i>	20	33-34
<b>Activity 6.2</b>	<b>Universal precautions- the use of barriers</b>	<b>20</b>	<b>6</b>
Reading	<i>Universal precautions for the prevention of transmission</i>	10	35-37
<b>Activity 6.3</b>	<b>Infectious Body fluids</b>	<b>5</b>	<b>8</b>
<b>Activity 6.4</b>	<b>Universal precautions- sharp disposal</b>	<b>15</b>	<b>10</b>
Reading	<i>Prevention of injuries with sharps</i>		
	<i>Sharps disposal container</i>	10	38
<b>Activity 6.5</b>	<b>Housekeeping in HIV infection</b>	<b>5</b>	<b>12</b>
<b>Activity 6.6</b>	<b>Hepatitis B vaccination</b>	<b>20</b>	<b>14</b>
Reading 6.6	<i>Hepatitis B immunisation in health workers</i>	10	40
	<i>Hepatitis B and the health worker</i>	10	56-57
<b>Activity 6.7</b>	<b>Post-exposure care</b>	<b>5</b>	<b>16</b>
Reading 6.7	<i>Handling exposure</i>	10	41
<b>Activity 6.8</b>	<b>Post-exposure risk assessment</b>	<b>10</b>	<b>18</b>
Reading 6.8	<i>Factors affecting transmission</i>	5	42
	<i>Risk of occupational transmission</i>		
	<i>Exposure evaluation – initial tests</i>	5	43
<b>Activity 6.9</b>	<b>Filling up an exposure report form</b>	<b>10</b>	<b>19</b>
<b>Activity 6.10</b>	<b>Post-exposure prophylaxis –HIV (A)</b>	<b>20</b>	<b>22</b>
Reading 6.10	<i>Recommended HIV post-exposure prophylaxis</i>	20	44-48
<b>Activity 6.11</b>	<b>Post-exposure prophylaxis –HIV (B)</b>	<b>5</b>	<b>24</b>
<b>Activity 6.12</b>	<b>Post-exposure prophylaxis –HBV</b>	<b>10</b>	<b>26</b>
Reading 6.12	<i>Hepatitis B and the health worker</i>	10	56-57
<b>Activity 6.13</b>	<b>HIV testing for invasive procedures</b>	<b>10</b>	<b>28</b>
Reading 6.13	<i>Blood borne pathogen screening for invasive procedures</i>	10	49
<b>TMA</b>		<b>60</b>	<b>31</b>
<b>Total estimated study time</b>		<b>325</b>	

This activity is aimed at helping you learn about the risk of different types of blood borne exposures and the extent of the problem of blood borne pathogen infection among health workers. Please read, "Occupational Exposures in the United States" (pg. 33-34) in the reader. After completing the reading, attempt the following activity.



### ACTIVITY 6.1

## BLOOD BORNE PATHOGEN INFECTIONS IN HEALTH WORKERS IN INDIA (TIME: 10 MIN.)

1. Based on the below formula given at the end, calculate the estimated number of seroconversions to HIV, Hepatitis C and Hepatitis B per year among health workers in India. The data provided is hypothetical.

Estimated number of needle stick injuries (NSI) in India - 900,000 per year. (This figure is based on the following assumptions: 600,000 hospital beds in India, 30 injuries/100 beds, 60% of injuries being unreported, 50 % of exposures being outside the hospital)

Estimated prevalence of blood borne pathogens in hospitalized patients: HIV 2%, HCV 2%, HBV 4%.

Risk of Infection following contaminated needle stick injury: HIV 0.3 %; HCV 1.8%; HBV 6%.

Formula:

Estimated number of sero-conversions / year =  

$$\frac{\text{No. of NSI} \times \text{seroprevalence in hospital} \times \text{risk of infection}}{10000}$$

Eg. No. of seroconversions due to HIV =  $\frac{900,000 \times 2 \times 0.3}{10,000}$

Calculate the following using the above formula:

Estimated number of seroconversion / year in India-

HIV - 54

Hepatitis C - 324

Hepatitis B - 2160

$$\frac{150,000}{600,000} \times 3 \times \frac{100}{50} \times 2 = 900,000$$

$$90 \times 2 \times 0.3 = 54$$

$$90 \times 2 \times 1.8 = 324$$

**FEEDBACK 6.1**

Estimated number of seroconversions / year to health workers in India-  
HIV – 54

Hepatitis C – 324

Hepatitis B – 2160

The above figures are estimates of seroconversions but may not reflect actual number of health workers who are infected by blood borne pathogens. They are meant to emphasise the magnitude of risk of blood borne pathogen infection among health workers.

*The following activity will help you learn about the use of barriers in universal precautions. Before undertaking it read: "Universal precautions for the prevention of transmission of HIV and other blood borne infections" (pg.35-37).*



## ACTIVITY 6.2

## UNIVERSAL PRECAUTIONS -USE OF BARRIERS

(TIME: 20 MIN.)

Thamarai Selvi, a 24 years old primigravida has been married for two years. She is unbooked and comes in labour to your clinic.

Write down in the table below: (a) what exposures (hand contact, spray, splash) are likely to take place with infectious body fluids related to the procedures listed; (b) what barriers are appropriate (gloves, mask, eye wear and gown).

Procedure	Probable means of exposure to infectious body fluids	Appropriate barriers
Vaginal examination	hand contact	gloves
Normal vaginal delivery	hand contact spray	Gloves Gown Mask & eye wear
Caesarian section	hand contact spray splash	"
Abdominal ultrasound	Nil (unless open wound)	—
Care of the new born	Hand contact	Gloves


**FEEDBACK 6.2**

<b>Procedure</b>	<b>Probable means of exposure to infectious body fluids</b>	<b>Appropriate barriers</b>
<b>Vaginal examination</b>	Hand contact	Gloves
<b>Normal vaginal delivery</b>	Hand contact Spray Splash	Gloves Mask Eye wear Full arm apron Plastic overshoes
<b>Caesarian section</b>	Hand contact Spray Splash	Gloves Mask Eye wear Full arm apron Plastic overshoes
<b>Abdominal ultrasound</b>	Nil	Nil
<b>Care of the new born</b>	Hand contact Spray Splash	Gloves Mask Eye wear Plastic apron Overshoes



The following activity will help you learn which body fluids to consider potentially infectious.

**ACTIVITY 6.3****INFECTIOUS BODY FLUIDS (TIME: 5 MIN)**

The ward attender accidentally spilled urinal contents onto his clothes and hands while emptying it. The source patient was undergoing treatment for HIV infection and cryptococcal meningitis. The attender was not wearing any protective clothing at the time. The urine was not blood stained, but contained small amount of faeces. The attendant is sent for risk assessment and for counseling regarding post-exposure prophylaxis.

1. Is the attender at risk of acquiring HIV infection?

No -

2. What would you advise to the attendant?

- Reassure  
- On protective wear

**FEEDBACK 6.3**

1. Is the attendant at risk?

No, the attender is not at risk. Urine and faeces do not require application of universal precautions and do not pose risks of transmission to the health worker.

2. What would you advise to the attendant?

The attender needs to be reassured. It would also be appropriate to review with him the principles of universal precautions, the need for using barriers and appropriate disposal of sharps. The attender would be better off wearing gloves and apron while handling body fluids, equipment or linen soiled with body fluids so as to avoid skin contact. He needs to check his hands for cuts regularly and to use a bandage as needed. You should review his Hepatitis B immunisation status.

*The next activity will help you learn about preventing needle injuries and how to dispose sharps. Attempt the activity after reading: "Prevention of injuries with sharps" and "Sharp Disposal Container" (pg.38).*



## ACTIVITY 6.4

## UNIVERSAL PRECAUTIONS- SHARP DISPOSAL

(TIME : 15 MIN)

1. List measures to reduce the risk of needle injury during the following procedures: (a) blood drawing; (b) surgery.

	Measures to reduce risk of needle injury
Blood drawing	<ul style="list-style-type: none"> <li>- One hand procedure</li> <li>- Covered hand</li> <li>- No capping / one hand capping</li> <li>- Used needle container closely</li> </ul>
Surgery	<ul style="list-style-type: none"> <li>- Instrument barrier zone</li> <li>- No hand retraction / or holding sharps</li> <li>- Full personal barriers</li> <li>- Remove wound out of the field</li> <li>- Sharps - wires and pins</li> </ul>

2. Write down a list of things that you should not do to a hollow bore needle once it is used.

1. capping	3. transporting uncovered
2. breaking	4. flushing

3. Answer the following questions related to a sharps disposal container

What are three characteristics that a sharps disposal container must have?	Puncture proof, Leak proof, covered
Where should a sharps disposal container be placed?	- Easy reach, and visibility at eye level (at the site of use)
What should the colour of a sharps disposal container be and how should it be labelled?	Red - Biohazard
When should a sharps disposal container be emptied?	When 3/4 full



### FEEDBACK 6.4

1.

	Measures to reduce risk of needle injury
Blood drawing	Gloves Tourniquet Avoid recapping Sharps disposal container on the blood drawing tray Needles not to be left lying around after the procedure
Surgery	Use instruments and not hands to grasp needles/sharps Do not retrieve needles / sharps with hands Move unused needles/sharps out of the surgical field Shield scalpel to prevent injury of the assistant Care with wires and pins Never pass sharp instruments from hand to hand - use "neutral zone"

2. Write down a list of things that you should not do to a hollow bore needle once it is used.

1. Do not recap needles.
2. Do not bend or break needles.
3. Do not leave needles lying around.
4. Do not dispose needles in the regular waste bin.

3.

What are three characteristics that a sharps disposal container must have?

*Puncture resistance, durability and being leak proof.*

Where should a sharps disposal container be placed?

*At the site of use – in the nursing station, treatment room, operating area, on the injection tray.  
It should be placed at table level and be easily accessible.*

What should the colour of a sharps disposal container be and how should it be labelled?

*Blue with biohazard symbol*

When should a sharps disposal container be emptied?

*As soon as it is three-fourths full.*

This activity will help you learn about housekeeping aspects of nursing a patient with HIV infection.



### ACTIVITY 6.5

#### HOUSEKEEPING IN HIV INFECTION (TIME: 5 MIN.)

P. Chandy is an HIV affected individual. He underwent an uneventful appendicectomy. After returning to the ward he vomited a large amount on the floor. Tick your choice.

1. Where should he be nursed?

Isolation ward  ICU  General ward  Single room

2. How will you clean the area?

Wipe dry  Cover with Dakins  Clean as usual for any other case

3. What will you do with his unsoiled linen and utensils?

Send for autoclaving  Soak in Dakin's solution  Proceed as usual

4. How will you prepare his bed for the next patient?

Wash with a disinfectant  Fumigate the room  Proceed as usual

5. Where will you dispose his wound dressing?

Yellow bag  Special bag for HIV patients  Metal container



### FEEDBACK 6.5

There are no special housekeeping needs for HIV positive individuals without secondary/opportunistic infections. Universal precautions should apply as with all patients. Special precautions will depend on the nature of the secondary infection; e.g. open tuberculosis will need isolation.

1. Where should he be nursed?

Isolation ward      ICU       General ward ✓      Single room

2. How will you clean the area?

Wipe dry       Cover with Dakins       Clean as usual for any other case ✓

3. What will you do with his unsoiled linen and utensils?

Send for autoclaving       Soak in Dakin's solution       Proceed as usual ✓

4. How will you prepare his bed for the next patient?

Wash with a disinfectant       Fumigate the room       Proceed as usual ✓

5. Where will you dispose his wound dressing?

Yellow bag ✓      Special bag for HIV patients       Metal container   
As for all  
infectious  
waste

This activity is aimed at helping you learn about Hepatitis B vaccination in health workers. Perform the activity after reading "Hepatitis B immunisation in health workers" (pg.40) and the article, "Hepatitis B and the health worker" (pg. 56-57).



### ACTIVITY 6.6

#### HEPATITIS B VACCINATION (TIME: 20 MIN.)

Your hospital has requested you to devise an immunisation strategy for Hepatitis B vaccination for the staff.

1. Which categories of staff would you provide hepatitis B vaccination to?

All HCW potential exposure to blood/body fluids  
(Not included - Administrative staff)

2. Is anti-HBc antibody testing required before vaccination to assess for prior infection?

No. There is no harm in additional dose.  
However for prophylaxis it is useful.

3. What are factors that may lead to lack of development of protective antibodies after immunisation?

Chl. kidney/liver disease, HIV, steroids, immunosuppression

4. What is the cost of vaccination for all the staff at risk in your hospital?

No. of staff requiring vaccination - 10

Total cost of vaccination - 20% wastage =

Cost of Shanvac B vaccine (10 ml) - Rs. 950.40

Cost per person for 3 dose Shanvac vaccination - Rs. 285.

5. What are some strategies to ensure complete vaccination for all the staff at risk in your hospital?

a. Rec. Policy & Education on the same -  
b. Records -  
c. Punitive steps



### FEEDBACK 6.6

1. Which categories of staff would you provide hepatitis B vaccination to?

All staff at risk - doctors, nurses, attenders, sweepers, lab technicians, physiotherapists, students involved in patient care.

Staff who may not require Hepatitis B vaccination are office staff.

2. Is anti-HBc antibody testing (evidence of prior hepatitis B infection) required before vaccination to assess for prior infection?

No. Routine testing for prior evidence of Hepatitis B infection is not required before vaccination.

3. What are factors that may lead to lack of development of protective antibodies after immunisation?

HIV infection

Chronic liver disease

Chronic renal failure

Smokers

Persons on immunosuppressive drugs

Persons above 50 years have less adequate antibody production after vaccination.

4. What is the cost of vaccination for all the staff at risk in your hospital?

No. of staff requiring vaccination - n

Total cost of vaccination (Rs.) -  $n \times 285$

5. What are strategies to ensure complete vaccination for all the staff at risk in your hospital?

a. Requirement for pre-employment vaccination.

b. Annual drive with education program.

c. Incentives for vaccination (provide vaccine free upto a certain date and thereafter charge for it).



This activity aims to enable you to learn the immediate steps of post-exposure care. Read "**Handling Exposure**" (pg.41). After completing the reading attempt the below activity.

**ACTIVITY 6.7****POST-EXPOSURE CARE (TIME: 5 MIN.)**

Dr. N. a 30 year old lady doctor drew blood from a patient Mr. K. Hospital number 3351, with HIV infection and cryptococcal meningitis. As she is withdrawing the needle after blood drawing, the patient jerked his hand and the needle pierced the doctor's gloved hand injuring her index finger. The needle (a 22 G disposable needle) went deep into her finger and there was visible blood on the surface of the needle.

1. What immediate steps of action should the doctor take?

1. Remove the gloves
2. Wash both running water, especially for 10 mins
3. Report to concerned authorities
4. Apply bandage
- 5.

**FEEDBACK 6.7**

1. What immediate steps of action should the doctor take?

1. Remove gloves
2. Go to nearest tap
3. Wash hands with running water expressing blood for 10 minutes.
4. Apply soap or antiseptic as is available.
5. Apply bandage
6. Report the exposure to the reporting authority.

This activity aims to enable you to learn the risk of transmission, factors influencing transmission from a HIV contaminated needle stick injury and tests to be performed at the initial evaluation of a needle stick injury. Undertake the activity after reading the following : "Factors affecting transmission", "Risk of occupational transmission of HIV" (pg.42) and "Exposure evaluation - initial tests to be performed" (pg.43).



### ACTIVITY 6.8

### POST-EXPOSURE RISK ASSESSMENT

(TIME: 10 MIN.)

Dr. N had removed her gloves immediately after the exposure and placed her hands under running water expressing blood and disinfecting it with spirit. She applied a bandage and then came to report her exposure to your clinic.

1. What is your assessment of the risk of transmission of HIV to Dr. N from this exposure?
2. What are the factors that may increase the risk of transmission in this case?

1. Risk of transmission of HIV

Yes, 0.3% (0.2-0.5)

2. Factors that may increase the risk of transmission:

- a. Symptomatic source patient
- b. hollow bore needles
- c. deep penetration
- d. visibly blood on the surface

3. What testing would you advise for the source patient Mr.K. and for Dr. N?

Source patient - HCV and HbSAg

Exposed doctor - " + HIV ELISA



### FEEDBACK 6.8

1. Risk of transmission of HIV - 0.3% (0.2-0.5%)
2. Factors that may increase the risk of transmission:
  - (a) Advanced HIV infection
  - (b) Hollow bore needle
  - (c) Needle had been introduced directly into a vein.
  - (d) Visible blood on the surface of the needle
  - (e) Deep injury

The only factor that may reduce the risk of transmission is that she was wearing gloves at the time of the injury.

- (f) What testing would you advise for the source patient and for doctor who has been exposed?

Source patient – HbsAg, Hepatitis C serology

Exposed doctor – the appropriate serology for which the source patient is positive in this case HIV ELISA (the doctor's blood is drawn and the tests ordered after the patient's report is known).

*This activity will help you fill up an exposure report form.*



### ACTIVITY 6.9

#### FILLING A EXPOSURE REPORT FORM

(TIME: 10 MIN.)

Mr. K (Hospital Number 3351) HbsAg - negative, HCV serology-negative

Dr. N - HIV serology negative Dr. N had received 3 doses of Hepatitis B vaccination in 2000. No followup anti-HBs test had been performed.

Fill up the exposure report form for Dr. N.

<b>Index case (source patient)</b>	
Name of source patient: <i>HV 12</i>	Hospital No. _____
Diagnosis: <i>HIV &amp; Cryptosporidiosis</i>	
Blood borne pathogen status known before the accident:	
HIV	<input checked="" type="checkbox"/> Yes/ No <input checked="" type="checkbox"/> Positive / negative
Hepatitis B	<input checked="" type="checkbox"/> Yes/ No <input checked="" type="checkbox"/> Positive / negative
Hepatitis C	<input checked="" type="checkbox"/> Yes/No <input checked="" type="checkbox"/> Positive / negative
Blood screen performed after the accident:	
HIV	<input checked="" type="checkbox"/> Yes/ No <input checked="" type="checkbox"/> Positive / negative
Hepatitis B	<input checked="" type="checkbox"/> Yes/ No <input checked="" type="checkbox"/> Positive / negative
Hepatitis C	<input checked="" type="checkbox"/> Yes/No <input checked="" type="checkbox"/> Positive / negative
<b>Infection status</b> in case of HIV infection (circle) :	
Class 1- Asymptomatic, low viral load	
Class 2 - <u>symptomatic</u> AIDS, high viral load, acute seroconverting illness	
<b>Hospital staff</b>	
Name: <i>DN</i>	Hospital No.: _____
Hepatitis B vaccination taken (circle) <input checked="" type="checkbox"/> 1 <sup>st</sup> dose/ <input checked="" type="checkbox"/> 2 <sup>nd</sup> dose/ <input checked="" type="checkbox"/> 3 <sup>rd</sup> dose <i>2000</i>	
Anti-HBs level - (Date _____ ) Level: <i>not known</i>	
Blood screen performed after the accident:	
HIV	<input checked="" type="checkbox"/> Yes/ No <input checked="" type="checkbox"/> Positive / negative
Hepatitis B	<input checked="" type="checkbox"/> Yes/ No <input checked="" type="checkbox"/> Positive / negative
Hepatitis C	<input checked="" type="checkbox"/> Yes/No <input checked="" type="checkbox"/> Positive / negative
Anti-HBs titre	<input checked="" type="checkbox"/> Yes / No <input checked="" type="checkbox"/> Positive / negative
<b>Injury</b>	
<i>Sharp injury</i> (describe the device and the nature of the exposure)-	
Type of device - <i>hollow bore</i>	
Visible blood on the surface of device- Yes/ No	
Device introduced into artery or vein - Yes / No	
Deep injury - Yes / No	
Was the health worker gloved: Yes / No	
<b>Exposure type</b> - Less severe (eg. solid bore needle, superficial injury)	
<input checked="" type="checkbox"/> More severe (eg. Large bore hollow needle, deep puncture, visible blood on needle surface, needle inserted into vein/artery)	
<b>Mucous membrane or non-intact skin exposure</b> (describe the exposure)	
Nature of fluid- _____ <i>NA</i>	
Site of exposure: _____	
Skin - _____	
Conjunctiva- _____	
Oral mucosa- _____	
Was the health worker using mask and protective eye-wear- Yes / No _____	
<b>Exposure type</b>	
Small volume (ie. A few drops)	
Large volume (ie. Major splash) _____	
Was appropriate post-exposure care initiated Yes / No _____	



### FEEDBACK 6.9

#### Index case (source patient)

Name of source patient: **Mr. K**

Hospital No. **3351**

Diagnosis: **Cryptococcal Meningitis**

Blood borne pathogen status known before the accident:

HIV **Yes**/ No **Positive** / negative

Hepatitis B Yes/ **No** Positive / negative

Hepatitis C Yes/**No** Positive / negative

Blood screen performed after the accident:

HIV Yes/ **No** Positive / negative

Hepatitis B **Yes**/ No Positive / **negative**

Hepatitis C **Yes**/No Positive / **negative**

#### Hospital staff

Name: **Dr. N**

Hospital No.:

Hepatitis B vaccination taken (circle) **1<sup>st</sup> dose/ 2<sup>nd</sup> dose/ 3<sup>rd</sup> dose**

Anti-HBs level – (Date ) Level: **Not done**

Blood screen performed after the accident:

HIV **Yes**/ No Positive / **negative**

Hepatitis B Yes/ **No** Positive / negative

Hepatitis C Yes/**No** Positive / negative

Anti-HBs titre Yes / No Positive / negative

**Infection status** in case of HIV infection (circle) :

Class 1- Asymptomatic, low viral load

**Class 2** – symptomatic, AIDS, high viral load, acute seroconverting illness

#### Injury

Sharp injury (describe the device and the nature of the exposure)-

Type of device – **Hollow bore needle 22 G needle**

Visible blood on the surface of device- **Yes** / No

Device introduced into artery or vein – **Yes**/ No

Deep injury – **Yes** / No

Was the health worker gloved: **Yes** / No

Exposure type – Less severe ( eg. solid bore needle, superficial injury)

**More severe** (eg. Large bore hollow needle, deep puncture, visible

blood on needle surface, needle inserted into vein/artery)

Mucous membrane or non-intact skin exposure (describe the exposure)

Nature of fluid-

Site of exposure:

Skin –

Conjunctiva

Oral mucosa-

Was the health worker using mask and protective eye-wear- Yes / No

#### Exposure type

Small volume (ie. A few drops)

Large volume (ie. Major splash)

Was appropriate post-exposure care initiated **Yes**/ No

The next activity aims to help you learn how to choose and initiate post-exposure prophylaxis for a health worker who has had an exposure from an HIV positive source patient. Undertake the activity after reading: "Recommended HIV postexposure prophylaxis for percutaneous injuries", "Recommended HIV postexposure prophylaxis for mucous membrane exposures and nonintact skin exposures", "Basic and Expanded HIV Postexposure Prophylaxis Regimens", "Timing and duration of PEP, Primary side effects associated with antiretroviral agents, Monitoring and managing toxicities", "Counselling of health workers exposed to HIV positive source", "Follow-up testing" (pg.44-48) in the reader.



### ACTIVITY 6.10

## POST-EXPOSURE PROPHYLAXIS – HIV (A)

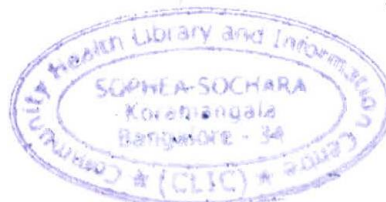
(TIME: 20 MIN.)

Write down in the exposure form what post-exposure prophylaxis would you advise for Dr. N?

#### EXPOSURE REPORT FORM (CONTINUED)

Exposure to HIV positive source patient:

Needle injury: ✓		
Exposure type- Less severe / More severe ✓		
Mucous membrane or non-intact skin exposure: N/A		
Exposure type- Small volume / large volume		
HIV infection status of source: Class 1 / Class 2 ✓		
PEP recommendation:		
Basic 2 drug PEP- Consider / Recommend		
Expanded 3 drug PEP- Recommend ✓		
Drugs advised		
Drug	Dose	Duration
1. Zidovudine	? 800mg bid	1/12
2. Lamivudine	400mg bid	1/12
3. Nevirapine	200mg bid	
Side-effects to be monitored for:		
1. Anaemia / Neutropenia		
2. Hepatitis / Pancreatitis		
3. - skin rash		
Followup visits:		Lab tests
1. 72 hrs		
2. 2 wks		- blood count - LFT / kidney
3. 1/12		- HIV ELISA
Counselling:		
1. Thoroughly wash with soap & water		
2. Maintain drug regimen		
3. Side effects of drugs		
4. Avoid exposure to others		
- Condom / No donating blood		



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### FEEDBACK 6.10

#### EXPOSURE REPORT FORM (CONTINUED)

Exposure to HIV positive source patient:

Needle injury: Exposure type- Less severe / <b>More severe</b> Mucous membrane or non-intact skin exposure: Exposure type- Small volume / large volume HIV infection status of source: Class 1 / <b>Class 2</b> PEP recommendation: Basic 2 drug PEP Consider / Recommend <b>Expanded 3 drug PEP</b>		
Drugs advised:		
Drug	Dose	Duration
1. Indinavir	800 mg q8h (on empty stomach)	4 weeks
2. Zidovudine	300 mg bd	4 weeks
3. Lamivudine	150 mg bd	4 weeks
(Alternate basic regimens- Lamivudine + Stavudine or Didanosine + Stavudine) (Alternate additional drug for expanded regimen- Nelfinavir or Efavirenz)		
Precautions to be taken and side-effects to be monitored for: Indinavir – On empty stomach with low fat meal, to take large amount of fluids Nausea, vomiting, increase in bilirubin. Kidney stones. Avoid astemizole, terfenadine, cisapride, Rifampicin, statins and benzodiazepines Zidovudine – Headache, nausea, vomiting, malaise, anemia		
Followup visits:		
	Lab tests	
1. 2 weeks	Assess for toxicities (blood counts, creatinine, LFT)	
1. 6 weeks	HIV serology (other blood tests as needed)	
2. 3 months	HIV serology (other blood tests as needed)	
3. 6 months	HIV serology	
Counselling:		
1. Reassure Dr. N- explain that while there are risks, they are really low. 2. Sexual abstinence or use condom with partner for the 6 months of followup. 3. Not to donate blood. 4. Explain regimen, side-effects, drugs to be avoided and need for adherence. 5. To review in case of any symptoms suggestive of an acute seroconverting illness (fever, lymphadenopathy, rash, neurological syndromes). 6. Emphasise to Dr. N that you will be available in case she would like to discuss anything.		





## ACTIVITY 6.11

## POST-EXPOSURE PROPHYLAXIS – HIV (B)

(TIME: 10 MIN.)

Mr. N, a lab technician drew blood from Mr. R. and spilled a few drops of blood on his hands after the procedure. He noticed a superficial cut on the hand on which he spilled blood. He immediately washed his hands with soap and water and reported the exposure to you. The post-exposure testing result showed that R's HIV ELISA is reactive and Mr. N's HIV test is negative. Mr. R is asymptomatic and had come for a general checkup.

Fill up your recommendations for post-exposure prophylaxis for Mr. N. on the exposure report form.

EXPOSURE REPORT FORM (CONTINUED)

Exposure to HIV positive source patient:

Needle injury:		
Exposure type- Less severe / More severe		
Mucous membrane or non-intact skin exposure: ✓		
Exposure type- Small volume / large volume		
HIV infection status of source: Class 1 / Class 2		
PEP recommendation:		
Basic 2 drug PEP Consider / Recommend		
Expanded 3 drug PEP		
Drugs advised		
	Drug	Dose
1.	Zido	300 bid
2.	Lami	40 mg
3.		
Duration		
		4/12
		1'
Side-effects to be monitored for:		
1.		
2.		
3.		
Followup visits: ✓		
Lab tests		
1.		
2.		
3.		
Counselling: ✓		
1.		
2.		
3.		
4.		



## FEEDBACK 6.11

### EXPOSURE REPORT FORM (CONTINUED)

Exposure to HIV positive source patient:

Needle injury: Exposure type- Less severe / More severe <b>Mucous membrane or non-intact skin exposure:</b> Exposure type- <b>Small volume</b> / large volume HIV infection status of source: <b>Class 1</b> / Class 2 PEP recommendation: <b>Basic 2 drug PEP Consider</b> / Recommend Expanded 3 drug PEP		
Drugs advised		
Drug	Dose	Duration
1. <b>Nil</b>		
2.		
3.		
Side-effects to be monitored for:		
1.		
2.		
3.		
Followup visits:	Lab tests	
1. 6 weeks	HIV ELISA	
2. 3 months	HIV ELISA	
3. 6 months	HIV ELISA	
Counselling:		
<p>Mr. K had a low risk exposure and this needs to be explained to him. While basic regimen of PEP with 2 drugs can be offered, it need not be recommended. You might actually dissuade Mr. K from taking PEP as the risk of side effects from PEP may outweigh any benefit to him. You would advise him to come for follow up.</p> <p>He needs to wear gloves while drawing blood and apply banadage to cuts. You should advise sexual abstinence or use of condom with partner, not to donate blood and to review in case of any symptoms suggestive of an acute seroconverting illness (fever, lymphadenopathy, rash, neurological syndromes).</p>		

The next activity aims to help you learn how to choose and initiate post-exposure prophylaxis for a health worker who has had an exposure from a Hepatitis B source patient. Undertake the activity after re-reading: "Hepatitis B and the health worker" (pg.56-57).



### ACTIVITY 6.12

## POST-EXPOSURE PROPHYLAXIS – HBV

(TIME: 10 MIN.)

Dr. S sustains a needle stick injury with an 18 G needle which he has used for a road traffic accident patient Mrs. D, for fluid resuscitation. There was visible blood on the needle and it had punctured his gloved finger. He washes his hands and reports the exposure. Mrs. D HbsAg test is positive. Dr. S had three dose hepatitis B vaccination 5 years ago and his anti-HBs titre following the exposure was found to be 8 mIU/ml.

Write down your recommendation for post-exposure prophylaxis for Dr. S.

#### EXPOSURE REPORT FORM (CONTINUED)

Exposure to HBSAg positive source patient:

Needle injury	✓
Mucous membrane or non-intact skin exposure:	
Vaccination status:	
Unvaccinated	
Partially vaccinated	
Completely vaccinated	✓
Anti-HBs antibody titre after vaccination:	< 10 Miu/ml / > 10 Miu/ml / not known ✓
Anti-HBs antibody titre after exposure:	< 10 Miu/ml / > 10 IU/ml / not known ✓
<u>PEP Recommended</u>	
HBIG 0.06 ml/Kg IM	✓
Hepatitis B vaccine booster	
Hepatitis B vaccine full course 0,1, 6 months	✓
Followup visits:	1/12
Lab tests	Hb s Ag.
Counselling:	<p>Non responder - Hence full course.          Precaution till observation period          Risk of infection.</p>



## FEEDBACK 6.12

### EXPOSURE REPORT FORM (CONTINUED)

Exposure to HBSAg positive source patient:

**Needle injury:**

Mucous membrane or non-intact skin exposure:

Vaccination status:

Unvaccinated

Partially vaccinated

**Completely vaccinated**

Anti-HBs antibody titre after vaccination: < 10 Miu/ml / > 10 Miu/ml / **not known**

Anti-HBs antibody titre after exposure: **≤ 10 mIU/ml** / > 10 IU/ml / not known

**PEP Recommended**

**HBIG 0.06 ml/Kg IM**

**Hepatitis B vaccine booster**

Hepatitis B vaccine full course 0,1, 6 months

Followup visits:

Lab tests

1 month after last dose of vaccine

Anti-HBs level

**Counselling:**

Dr. S should be reassured that the risk may be low as he has had prior vaccination. It is because of his lower anti-body level that he is being given HBIG and booster vaccination. No specific behavioural modification or restriction in patient contact is required.

The following exercise aims to help you learn about the debate on screening of blood borne pathogens before invasive procedures. Read "Blood borne pathogen screening before invasive procedures" (pg.49) in the reader. Then proceed to the next activity.



### ACTIVITY 6.13

## HIV TESTING FOR INVASIVE PROCEDURES

(TIME: 10 MIN.)

A 35-year-old lorry cleaner from Namakkal presenting with a left sided neck swelling and fever is found to have matted lower deep cervical lymph nodes and is referred for a fine needle aspiration of the lymph node. The surgeon who the case is referred to requests for an HIV test.

1. Is the risk of transmission of blood borne pathogens to the health worker while drawing blood for HIV screening more or less than the risk of performing a needle aspiration of the lymph node?

Same

2. In which situations may HIV screening be performed before doing an invasive procedure?

- Much bleeding expected

3. Would precautions would you take if the patient was known to be HIV positive?

- Post at the end of the test  
- otherwise universal precautions

**FEEDBACK 6.13**

1. Is the risk of transmission of blood borne pathogens to the health worker while drawing blood for HIV screening more or less than the risk of performing a needle aspiration of the lymph node?

The risk of transmission through a hollow needle used to draw blood is more than the risk of transmission from a needle used to aspirate tissue. The infectiousness of blood is greater than aspirated tissue fluid and the needle used for drawing blood can deliver more inoculum than a fine needle used for the aspiration test.

2. In which situations may HIV screening be performed before doing an invasive procedure?

Risk prone procedures such as deep pelvic or vascular surgery .

3. Would precautions would you take if the patient was known to be HIV positive?

Operative precautions do not change based on HIV status and all universal precautions should be followed irrespective of the results of a positive test. It makes more sense to invest resources in universal precautions rather than in universal testing.



**NOTES**



## READINGS

### OCCUPATIONAL EXPOSURES IN THE UNITED STATES

From the : International Health Care Safety Centre

The following figures were calculated based on 1996 EPINet data (a surveillance system for needle exposures to health workers in the US). We do not know to what degree new HIV treatments have affected health care worker risk of HIV infection. They have probably reduced the risk somewhat since there are now fewer AIDS patients in hospitals.

We estimated percutaneous injuries and blood and body fluid exposures in one year, based on:

- 30 injuries per 100 occupied hospital beds reported (from our national EPINet data for 1996)
- 600,000 occupied hospital beds in the U.S.
- 180,000 injuries in one year reported in hospitals ( $0.3 \times 600,000$ )
- 39% of incidents not reported (according to surveys conducted in 6 EPINet hospitals in 1996-1997) = 295,082 injuries occurred in hospitals
- double this figure because 50% of health care workers work outside of hospital settings (total = 590,164 percutaneous injuries)
- according to EPINet data for 1996, an additional 1/3 of reported exposures (total = 196,721 mucocutaneous exposures) involve skin/non-intact skin or mucous membrane contact with blood or at-risk biological substances which can also transmit HIV, HBV, HCV
- Total annual percutaneous and mucocutaneous exposures to blood or at-risk biological substances in the U.S. in 1996 = 786,885

$$180^3 \times \frac{100}{60}$$

$$\frac{590,164}{1,000}$$

Risk of Infection Following a Single HIV, HBV, or HCV-Contaminated Needlestick or Sharp Instrument Injury

- HIV 0.25% - 0.4%
- HBV 6% - 30%
- HCV 0.4% - 1.8%



The CDC estimates that 400 new occupational HBV infections occurred in 1995 among U.S. health care workers, down from 17,000 in 1983. (Arch Intern Med 1997;157:2601-2603)

Assuming that between 1% and 2% of patients are HIV-positive (and therefore that 1% to 2% of needlesticks are HIV-contaminated) between 18 to 35 new occupational HIV infections would occur from percutaneous injuries each year. Infections resulting from blood exposures to non-intact skin or mucous membranes would add between 2 to 4 cases (based on a transmission rate of .09% for a mucous membrane exposure).

Assuming that between 2% and 10% of patients are HCV-positive (Dr. Richard Garvin, Hepatitis Branch, CDC), between 59 to 1,180 new occupational HCV infections would occur each year. Infections resulting from blood exposures to non-intact skin or mucous membranes would add between 16 to 393 cases (assuming that the transmission rate was between 0.4% and 1.8% per exposure, with lower limit from Dr. Giuseppe Ippolito, Italy, 1999).

The consequences of occupational exposure to bloodborne pathogens are not only infections. Each year, thousands of health care workers are affected by psychological trauma during months of waiting for notification of serological results. Other personal consequences can include postponement of childbearing, altering sexual practices, side effects of prophylactic drugs, infection, chronic disabilities, loss of employment, denial of worker compensation claims, liver transplant, and premature death.

From: <http://hsc.virginia.edu/medcntr/centers/epinet/cdcestim.html>

### UNIVERSAL PRECAUTIONS FOR PREVENTION OF TRANSMISSION OF HIV AND OTHER BLOODBORNE INFECTIONS

"Universal precautions," as defined by CDC, are a set of precautions designed to prevent transmission of human immunodeficiency virus (HIV), hepatitis B virus (HBV), and other bloodborne pathogens when providing first aid or health care.

Under universal precautions, blood and certain body fluids of all patients are considered potentially infectious for HIV, HBV and other bloodborne pathogens.

Universal precautions took the place of and eliminated the need for the isolation category "Blood and Body Fluid Precautions" in the 1983 CDC Guidelines for Isolation Precautions in Hospitals. However, implementing universal precautions does not eliminate the need for other isolation precautions, such as droplet precautions for influenza, airborne isolation for pulmonary tuberculosis, or contact isolation for methicillin-resistant *Staphylococcus aureus*.

Universal precautions apply to: blood, other body fluids containing visible blood, semen, and vaginal secretions. Universal precautions also apply to tissues and to the following fluids: cerebrospinal, synovial, pleural, peritoneal, pericardial, and amniotic fluids.

Universal precautions do not apply to: feces, nasal secretions, sputum, sweat, tears, urine, and vomitus unless they contain visible blood. Universal precautions do not apply to saliva except when visibly contaminated with blood or in the dental setting where blood contamination of saliva is predictable.

Universal precautions involve:

1. The use of protective barriers such as gloves, gowns, aprons, masks, or protective eyewear, which can reduce the risk of exposure of the health care worker's skin or mucous membranes to potentially infective materials.
2. In addition, under universal precautions, it is recommended that all health care workers take precautions to prevent injuries caused by needles, scalpels, and other sharp instruments or devices.

## 1. GLOVING, GOWNING, MASKING, AND OTHER PROTECTIVE BARRIERS AS PART OF UNIVERSAL PRECAUTIONS

All health care workers should routinely use appropriate barrier precautions to prevent skin and mucous membrane exposure during contact with any patient's blood or body fluids that require universal precautions.

### a. Gloves should be worn:

i. *for touching blood and body fluids requiring universal precautions, mucous membranes, or nonintact skin of all patients*

ii. *for handling items or surfaces soiled with blood or body fluids* to which universal precautions apply.

iii. Gloves should be changed after contact with each patient.

iv. Hands and other skin surfaces should be washed immediately if contaminated with blood or body fluids requiring universal precautions.

v. Hands should be washed immediately after gloves are removed.

vi. Gloves should reduce the incidence of blood contamination of hands during phlebotomy, but they cannot prevent penetrating injuries caused by needles or other sharp instruments. Institutions that judge routine gloving for all phlebotomies is not necessary should periodically reevaluate their policy. Gloves should always be available to health care workers who wish to use them for phlebotomy. In addition, the following general guidelines apply:

Use gloves for performing phlebotomy when the health care worker has cuts, scratches, or other breaks in his/her skin.

Use gloves in situations where the health care worker judges that hand contamination with blood may occur, e.g., when performing phlebotomy on an uncooperative patient.

Use gloves for performing finger and/or heel sticks on infants and children.

Use gloves when persons are receiving training in phlebotomy.

b. Masks and protective eyewear or face shields should be worn by health care workers to prevent exposure of mucous membranes of the mouth, nose, and eyes

during *procedures that are likely to generate droplets of blood or body fluids* requiring universal precautions.

c. Gowns or aprons should be worn during *procedures that are likely to generate splashes of blood or body fluids* requiring universal precautions.

## **2. PREVENTION OF NEEDLE AND SHARP INJURIES**

All health care workers should take precautions to prevent injuries caused by needles, scalpels, and other sharp instruments or devices during procedures; when cleaning used instruments; during disposal of used needles; and when handling sharp instruments after procedures. To prevent needlestick injuries:

- a. Needles should not be recapped by hand, purposely bent or broken by hand, removed from disposable syringes, or otherwise manipulated by hand.
- b. After they are used, disposable syringes and needles, scalpel blades, and other sharp items should be placed in puncture-resistant containers for disposal.
- c. The puncture-resistant containers should be located as close as practical to the use area.
- d. All reusable needles should be placed in a puncture-resistant container for transport to the reprocessing area.

General infection control practices should further minimize the already minute risk for salivary transmission of HIV. These infection control practices include the use of gloves for digital examination of mucous membranes and endotracheal suctioning, handwashing after exposure to saliva, and minimizing the need for emergency mouth-to-mouth resuscitation by making mouthpieces and other ventilation devices available for use in areas where the need for resuscitation is predictable.

Although universal precautions do not apply to human breast milk, gloves may be worn by health care workers in situations where exposures to breast milk might be frequent, e.g., in breast milk banking.

Adapted from : <http://www.cdc.gov/ncidod/hip/BLOOD/UNIVERSA.HTM>

### PREVENTION OF SHARP INJURIES

The following set of instructions lists measures to prevent needle injuries from taking place.

#### 1. Procedure related injuries:

##### a. *Blood drawing:*

- i. Use tourniquet and gloves
- ii. Take a sharps disposal container on your blood drawing tray
- iii. Avoid keeping your palpating finger on the artery or vein during blood drawing (as in arterial blood gas)
- iv. Remove the needle and dispose in sharps disposal container before transferring blood into test tube. An artery clamp or forceps may be used for removing needle from the syringe and disposing into sharps disposal container.
- v. Do not recap the needle after use.

##### b. *Surgery*

- i. Do not pass sharps from hand to hand. Use a neutral area or transit tray.
- ii. Do not retract with hand.
- iii. Do not use hand suturing.
- iv. Avoid needle-hand contact. Never retrieve needles and sharps with fingers.
- v. Move unused sharps out of the surgical field.
- vi. Shield the scalpel when using it to avoid injury to assistants.
- vii. Exercise care with wires and long pins.
- viii. While suturing the needle should be away from the knot. Clamp the needle and then only cut the suture.
- ix. Wear double gloves while operating.

#### 2. Disposal injuries

- a. Never leave unused sharps lying around.
- b. Dispose sharps only in the sharps disposal container.

- c. Remove and empty sharps disposal container when three-fourths full.
- d. Sharps disposal containers need to be emptied with great care.

### **SHARP DISPOSAL CONTAINERS**

1. Functional Criteria: Puncture resistant, durability and leak proof

2. General location and placement:

Sharp containers should be placed at the site of use. They should be readily visible and within easy horizontal reach of the user. There should not be obstacles between the sharp container and the site of use.

3. Colour and label: Blue or white translucent colour with biohazard label on the surface of the container.

4. Emptying: The sharp disposal container should be sent for emptying when it is three-fourths full.

### HEPATITIS B IMMUNISATION IN HEALTH WORKERS

Hepatitis B is the most important occupational blood borne pathogen infection among health workers. In the United States it was estimated that 17,000 new Hepatitis B infections occurred in health workers in 1983. With effective immunisation the number of new Hepatitis B infections in health workers was reduced to 400 in 1995.

All health workers at risk of exposure to Hepatitis B should be provided immunisation. The efficacy of 3 dose schedule of vaccination is 90-95%. The vaccination schedule has less efficacy in persons who have HIV infection, chronic liver disease, chronic renal failure, Diabetes Mellitus, smokers and persons on immunosuppressive drugs.

Routine prevaccination antibody testing is not required. Antibody testing is recommended 1-2 months after the third dose of vaccine. Persons who do not develop adequate antibody titre after the third dose are required to receive a repeat course of vaccination with three doses. Follow up antibody testing after 2 months of completion of course and booster vaccination is not required.

Following significant exposure of a vaccinated health worker to a Hepatitis B positive source patient, anti-HBs testing is required. If antibody levels are inadequate Hepatitis B immunoglobulin and booster dose of vaccination are to be administered. This regimen has an efficacy of about 75% in protecting the health worker against the exposure.

In situations where anti-HBs testing is not available, a reasonable option is:

- a. provide complete Hepatitis B vaccination course to all at risk health workers.
- b. In case of a significant exposure to Hepatitis B positive source patient-

If the health worker has had a full course of vaccination then the person may be followed up. If the person does not have documented vaccination record or has not received a complete course of vaccination, then Hepatitis B immunoglobulin injection followed by repeat course of vaccination should be advised. In case Hepatitis B immunoglobulin is not available for a significant exposure in an unvaccinated or partially vaccinated individual, then institute Hepatitis vaccination as soon as possible.

### HANDLING EXPOSURES

In case of an exposure:

*Don't panic.*

Stop what you are doing immediately.

Hand over to colleague if needed.

Call for help.

In case of needle stick injury:

1. Remove gloves
2. Go to nearest tap
3. Wash hands with running water expressing blood for 10 minutes.
4. Apply soap or antiseptic as is available.
5. Apply bandage

In case of mucous membrane exposure:

1. Irrigate copiously with water or normal saline for 10 minutes. In case of exposure to eyes keep your eyelids held open.
2. Do not apply soap or antiseptic.
3. Report exposure to reporting authority.

In case of non-intact skin exposure:

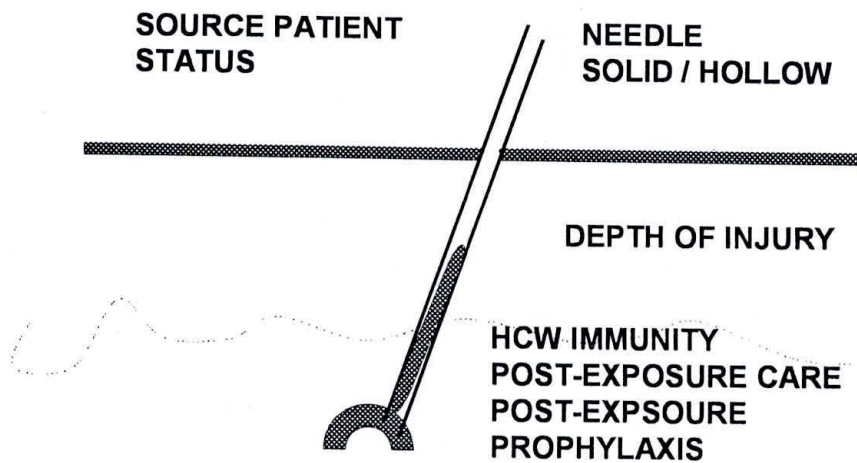
1. Wash with soap and water or antiseptic.
2. Report exposure to reporting authority.

**In case of needle stick injury, mucous membrane exposure or non-intact skin exposures:**

**Report the exposure as soon as possible so to initiate appropriate post-exposure prophylaxis.**



## FACTORS AFFECTING TRANSMISSION



### FACTORS AFFECTING TRANSMISSION FROM NEEDLE INJURY

1. Increased inoculum of blood-
  - a. Device visibly contaminated with blood
  - b. Needle had been placed directly in artery or vein
  - c. Deep injury
  - d. Hollow bore needle
2. Source patient with increased viral load: Symptomatic disease, AIDS, acute seroconverting illness

#### Factors which reduce risk of transmission from a needle stick injury

Post-exposure prophylaxis

### RISK OF OCCUPATIONAL TRANSMISSION OF HIV INFECTION

Contaminated needle stick injury - 0.3 % (0.2-0.5%)

Mucous membrane exposure - 0.09% (0.006-0.5%)

**EXPOSURE EVALUATION - INITIAL TESTS TO BE PERFORMED**

Source patient having no known infection.

**1. Tests for source patient**

HbsAg, HIV ELISA or rapid test and HCV serology.

**2. Tests for exposed health worker**

a. *If source patient's tests are all negative -*

No tests required for exposed health worker.

b. *If source patient HIV test is positive -*

HIV ELISA to be performed for exposed health worker.

c. *If source patient HbsAg test is positive and health worker has been partially or fully immunized -*

Anti-HBs titre to be performed for the health worker.

d. *If source patient HCV serology test positive -*

HCV serology to be performed for the health worker.

**RECOMMENDED HIV POSTEXPOSURE PROPHYLAXIS FOR  
PERCUTANEOUS INJURIES**

(Vol. 50 / No. RR-11 MMWR pg. 24)

Refer Guidelines and Policies in HIV Care, CMCH pg. 23-27 and note differences in management.

Exposure type	INFECTION STATUS OF SOURCE PATIENT				
	HIV positive Class 1*	HIV positive Class 2*	Source of unknown HIV status †	Unknown source§	HIV-Negative
Less severe ¶	Recommend basic 2- drug PEP	Recommend expanded 3- drug PEP	Generally, no PEP warranted Consider 2 drug PEP in source with known risk factors††	Generally, no PEP warranted Consider 2 drug PEP** in settings where exposure to HIV patients likely	No PEP warranted
More severe§§	Recommend expanded 3- drug PEP	Recommend expanded 3- drug PEP	Generally, no PEP warranted Consider 2 drug PEP in source with known risk factors††	Generally, no PEP warranted Consider 2 drug PEP** in settings where exposure to HIV patients likely	No PEP warranted

\* HIV- Positive, Class 1 – asymptomatic HIV infection or known low viral load (e. g., <1,500 RNA copies/ mL). HIV- Positive, Class 2 – Symptomatic HIV infection, AIDS, acute seroconversion, or known high viral load. If drug resistance is a concern, obtain expert consultation. Initiation of postexposure prophylaxis (PEP) should not be delayed pending expert consultation, and, because expert consultation alone cannot substitute for face- to- face counseling, resources should be available to provide immediate evaluation and follow- up care for all exposures.

†Source of unknown HIV status (e. g., deceased source person with no samples available for HIV testing).

§Unknown source (e. g., a needle from a sharps disposal container).

¶Less severe (e. g., solid needle and superficial injury).

\*\* The designation “consider PEP” indicates that PEP is optional and should be based on an individualized decision between the exposed person and the treating clinician.

††If PEP is offered and taken and the source is later determined to be HIV- negative, PEP should be discontinued.

§§More severe (e. g., large- bore hollow needle, deep puncture, visible blood on device, or needle used in patient’s artery or vein).

**RECOMMENDED HIV POSTEXPOSURE PROPHYLAXIS FOR MUCOUS MEMBRANE  
EXPOSURES AND NONINTACT SKIN EXPOSURES**

(Vol. 50 / No. RR-11 MMWR Page 25)

*Refer Guidelines and Policies in HIV Care, CMCH pg. 23-27 and note differences in management.*

Exposure type	INFECTION STATUS OF SOURCE PATIENT				
	HIV positive Class 1†	HIV positive Class 2†	Source of unknown HIV status§	Unknown source¶	HIV-Negative
Small volume**	Consider basic 2 drug PEP ††	Recommend basic 2 drug PEP	Generally, no PEP warranted Consider 2 drug PEP †† in source with known risk factors††	Generally, no PEP warranted Consider 2 drug PEP †† in settings where exposure to HIV patients likely	No PEP warranted
Large volume¶¶	Recommend basic 2- drug PEP	Recommend expanded 3- drug PEP	Generally, no PEP warranted Consider 2 drug PEP †† in source with known risk factors	Generally, no PEP warranted Consider 2 drug PEP †† in settings where exposure to HIV patients likely	No PEP warranted

\* For skin exposures, follow-up is indicated only if there is evidence of compromised skin integrity (e. g., dermatitis, abrasion, or open wound).

†HIV- Positive, Class 1 – asymptomatic HIV infection or known low viral load (e. g., <1,500 RNA copies/ mL). HIV- Positive, Class 2 –

symptomatic HIV infection, AIDS, acute seroconversion, or known high viral load. If drug resistance is a concern, obtain expert consultation. Initiation of postexposure prophylaxis (PEP) should not be delayed pending expert consultation, and, because expert consultation alone cannot substitute for face-to-face counseling, resources should be available to provide immediate evaluation and follow-up care for all exposures.

§Source of unknown HIV status (e. g., deceased source person with no samples available for HIV testing).

¶Unknown source (e. g., splash from inappropriately disposed blood).

\*\* Small volume (i. e., a few drops).

††The designation, "consider PEP," indicates that PEP is optional and should be based on an individualized decision between the exposed person and the treating clinician.

§§If PEP is offered and taken and the source is later determined to be HIV- negative, PEP should be discontinued.

¶¶Large volume (i. e., major blood splash).

**BASIC AND EXPANDED HIV POSTEXPOSURE  
PROPHYLAXIS REGIMENS**

(Vol. 50 / No. RR-11 MMWR pg. 47)

**BASIC REGIMEN**

- Zidovudine + Lamivudine
  - ZDV: 600 mg per day, in two or three divided doses, and
  - 3TC: 150 mg twice daily.

**ALTERNATE BASIC REGIMENS**

- Lamivudine (3TC) + Stavudine (d4T)
  - 3TC: 150 mg twice daily, and
  - d4T: 40 mg (if body weight is <60 kg, 30 mg twice daily) twice daily.
- Didanosine (ddI) + Stavudine (d4T)
  - ddI: 400 mg (if body weight is <60 kg, 125 mg twice daily) daily, on an empty stomach.
  - d4T: 40 mg (if body weight is <60 kg, 30 mg twice daily) twice daily.

**EXPANDED REGIMEN**

Basic regimen plus one of the following:

- Indinavir (IDV)
  - 800 mg every 8 hours, on an empty stomach.
- Nelfinavir (NFV)
  - 750 mg three times daily, with meals or snack, or
  - 1250 mg twice daily, with meals or snack.
- Efavirenz (EFV)
  - 600 mg daily, at bedtime.
- Abacavir
  - 300 mg twice daily.

**ANTIRETROVIRAL AGENTS FOR USE AS PEP ONLY WITH EXPERT  
CONSULTATION**

- Ritonavir
- Saquinavir (SQV)
- Amprenavir (AMP)
- Lopinavir/Ritonavir (KALETRA™)

**ANTIRETROVIRAL AGENTS GENERALLY NOT RECOMMENDED FOR USE AS PEP**

- Nevirapine (NVP)

### TIMING AND DURATION OF PEP

Post-exposure prophylaxis for HIV infection should be initiated as soon as possible after the exposure preferably within the first 24 hours. However PEP may be started even up to one week after the exposure. Post-exposure prophylaxis should be administered for 4 weeks if tolerated.

### PRIMARY SIDE EFFECTS ASSOCIATED WITH ANTIRETROVIRAL AGENTS

(Vol. 50 / No. RR-11 MMWR pg. 13)

#### Nucleoside reverse transcriptase inhibitors (NRTIs)

1. Zidovudine (AZT) anemia, neutropenia, nausea, headache, insomnia, muscle pain, and weakness
2. Lamivudine (3TC) abdominal pain, nausea, diarrhea, rash, and pancreatitis
3. Stavudine (d4T) peripheral neuropathy, headache, diarrhea, nausea, insomnia, anorexia, pancreatitis, increased liver function tests (LFTs), anemia, and neutropenia
4. Didanosine (ddI) pancreatitis, lactic acidosis, neuropathy, diarrhea, abdominal pain, and nausea
5. Abacavir nausea, diarrhea, anorexia, abdominal pain, fatigue, headache, insomnia, and hypersensitivity reactions

#### Nonnucleoside reverse transcriptase inhibitors (NNRTIs)

1. Nevirapine (NVP) rash (including cases of Stevens-Johnson syndrome), fever, nausea, headache, hepatitis, and increased LFTs
2. Delavirdine (DLV) rash (including cases of Stevens-Johnson syndrome), nausea, diarrhea, headache, fatigue, and increased LFTs
3. Efavirenz (EFV) rash (including cases of Stevens-Johnson syndrome), insomnia, somnolence, dizziness, trouble concentrating, and abnormal dreaming

#### Protease inhibitors (PIs)

1. Indinavir (IDV) nausea, abdominal pain, nephrolithiasis, and indirect hyperbilirubinemia  
Nelfinavir (NFV) diarrhea, nausea, abdominal pain, weakness, and rash
2. Ritonavir (RTV) weakness, diarrhea, nausea, circumoral paresthesia, taste alteration, and increased cholesterol and triglycerides
3. Saquinavir (SQV) diarrhea, abdominal pain, nausea, hyperglycemia, and increased LFTs
4. Amprenavir (AMP) nausea, diarrhea, rash, circumoral paresthesia, taste alteration, and depression
5. Lopinavir/Ritonavir (Kaletra™) diarrhea, fatigue, headache, nausea, and increased cholesterol and triglycerides

### MONITORING AND MANAGING TOXICITIES

After initiation of PEP the health worker should be monitored at baseline and after 2 weeks. Lab testing should include complete blood counts, liver and renal function tests. They should be advised the importance of completing their regimen. They should also know the drug side-effects and interactions. Minor side-effects may be treated symptomatically or with dose adjustment. Major side-effects may require stoppage or change of drugs.

### COUNSELLING OF HEALTH WORKERS EXPOSED TO HIV SOURCE

1. Exposed health workers are under tremendous stress and should be reassured and gently counseled.
2. While the risks of exposure need to be acknowledged the miniscule nature of most exposures needs to be emphasized as well the possibility for reducing the risks through PEP.
3. The pros and cons of taking PEP need to be discussed if it is indicated.
4. If PEP is initiated, then the dosing, side-effects and interactions and the follow-up schedule need to be clearly explained.
5. The health worker should refrain from sexual activity or use condoms appropriately during the 6 months after the exposure.
6. They should avoid blood donation during the period of follow-up.
7. The health worker should be advised to seek medical evaluation for any acute illness occurring during follow-up.

### FOLLOW-UP TESTING

(Vol. 50 / No. RR-11 MMWR Page 46)

#### **HBV exposures**

- Perform follow-up anti-HBs testing in persons who receive hepatitis B vaccine.
  - Test for anti-HBs 1–2 months after last dose of vaccine.
  - Anti-HBs response to vaccine cannot be ascertained if HBIG was received in the previous 3–4 months.

#### **HCV exposures**

- Perform baseline and follow-up testing for anti-HCV and alanine aminotransferase (ALT) 4–6 months after exposures.
- Perform HCV RNA at 4–6 weeks if earlier diagnosis of HCV infection desired.
- Confirm repeatedly reactive anti-HCV enzyme immunoassays (EIAs) with supplemental tests.

#### **HIV exposures**

- Perform HIV-antibody testing for at least 6 months postexposure (e.g., at baseline, 6 weeks, 3 months, and 6 months).
- Perform HIV antibody testing if illness compatible with an acute retroviral syndrome occurs.
- Advise exposed persons to use precautions to prevent secondary transmission during the follow-up period.
- Evaluate exposed persons taking PEP within 72 hours after exposure and monitor for drug toxicity for at least 2 weeks.

### BLOOD BORNE PATHOGEN SCREENING BEFORE INVASIVE PROCEDURES

There are three possible reasons doctors may order HIV test before invasive procedures.

#### 1. HIV status alters diagnostic and therapeutic considerations

In certain situations knowledge of the HIV status may alter the diagnostic and therapeutic considerations.

Eg. In case of renal transplant HIV screening is done on the recipient. If the recipient had HIV infection, the patient would not tolerate immunosuppression and may die of HIV and not chronic renal failure.

#### 2. Performance of risk prone procedures which pose serious risks to the operating surgeon

In performance of high-risk procedures, knowledge of the HIV test may allow the surgeon to alter surgical procedure so as to reduce risk to the operating team.

Eg. Risk prone procedures such as deep pelvic surgery or vascular surgery where the surgeons operating hands and sharps are placed together but are not visible to the surgeon. In this situation the surgeon is prone to the risk of sharp injury. Appropriate precautions need to be taken to prevent injury to the surgeon. In such a situation HIV testing may be considered as it may reduce the risk of transmission of HIV infection to the operating surgeon.

#### 3. Performance of procedures which are not in high risk category

HIV testing is not recommended in most invasive procedures, as they do not pose significant risks to the surgeon. The only method of reducing risks in these procedures is by following universal precautions for all patients.

There is a perception that knowledge of HIV test may lead to closer adherence to universal precautions in patients who pose risks to the surgeon. However a study from San Francisco suggested infection control precautions were no more carefully observed when HIV test was known to be positive.

Screening for blood borne pathogens requires testing for HIV, Hepatitis B and Hepatitis C. Screening tests may miss some patients who have infection in the window period or because of lack of sensitivity of the tests (eg. Hepatitis C). Also screening may miss unknown blood borne pathogens that we do not know about today.

A negative blood borne pathogen screen may lead a surgeon to be less careful and therefore more prone to injury. On the other hand a positive HIV test may lead to the surgeon to avoid performance of the procedure on the patient. This would reduce patients' access to medical care and would be ethically unacceptable.



### SAFE SURGICAL PRACTICE

While all categories of Health Care Workers (HCW) are at risk of exposure to blood borne pathogens, surgeons and obstetricians and gynecologists, by the very nature of their profession, are particularly at risk. General surgeons are higher risk than any other category of HCW <sup>1</sup>.

While this article has focused on HIV transmission, it must be remembered that the risk of contracting hepatitis B or C is higher than the risk of contracting HIV from blood contact or sharps injuries with solid or hollow needles <sup>3</sup>. Observing the following precautions will help minimize transmission of these and other unknown BBP

The risk to a surgeon is dependent on the type and number of operations performed by the surgeon and the prevalence of HIV in the population. This will vary with the seroprevalence of the patient population and the surgical injury rate. The frequency of percutaneous injury has been found in various studies to be about 1 in 40 operations. Based on a probabilistic model, the cumulative lifetime risk to a surgeon performing 350 operations per year for 30 years is about 1/100 <sup>3</sup>. For HIV prevalence of 1 in 100, the cumulative risk is about 1 in 100. When the seroprevalence is 1/10, the risk may be high as 1 in 5 i.e., the risk increases with increasing prevalence in the community.

There are five variables that can be altered to reduce the risk of HIV transmission to a surgeon: I) seroprevalence ii) efficiency of transmission iii) number of operations performed per year iv) number of years in practice and v) incidence of inoculation. Of these, the only practical strategy for risk reduction is to modify surgical practice so as to reduce the incidence of inoculation.

Inoculation during an operation can be substantially decreased by 1) Barrier protection 2) Alteration of surgical technique to avoid needlestick / sharps injury.

### Personal Protective Equipment (PPE): Barrier precautions and PPE are used to decrease a surgeon's exposure to blood.

1. **Gloves:** Glove perforations are common during operations, particularly if equipment such as drills are being used. Furthermore, surgeons are often unaware

that gloves have perforated and there has been skin contact with blood and body fluids.

Double gloving is recommended as this has been shown to reduce exposure rates from 51% to 7%. Double gloving has been criticized as being impractical, decreasing tactile sensation and dexterity, particularly by older surgeons. It is recommended that the inner pair be a half size bigger than the size normally worn by the surgeon, and the outer pair be the usual size. This prevents loss of tactile sensation, air pockets and pain<sup>3</sup>. Polyethylene and vinyl gloves, though resistant to tearing, have not been shown to decrease exposure to blood.

It should be noted that while gloves protect against blood exposure, they do not prevent or protect against needle stick or sharps injuries. However, wearing gloves reduces the amount of blood contact.

If the forearm is likely to be exposed to blood (eg. During manual removal of placenta), it should also be covered with gloves.

2. **Masks:** The role of masks has shifted from protecting the patient to protecting the surgeon. Hence, masks should be secured properly, covering the mouth and nose. They should be changed, ideally between cases and if soiled with blood. High filtration, disposable masks provide more protection than cloth masks traditionally worn. Where large volume splashes are anticipated, a full-face shield is recommended in addition.
3. **Protective eyewear:** Protective eyewear is necessary to protect the HCW from conjunctival contact with blood. Various models are available, the cheapest being standard glasses with side wings. In India, cheap, easily available varieties are motorcycle goggles. Motorcycle face shields can be used where large spills are anticipated, or when the surgeon wears corrective eyewear already. These should be made available in all operation theatres in adequate numbers.
4. **Gowns:** Ideally, gowns should be made of impervious material. While these are commercially available, they are expensive, and single use. A cheaper alternative is to wear a disposable plastic apron under the conventional cloth gown. While this protects the chest and abdomen up to mid thigh, the arms are unprotected. Protection of the arms will require elbow length gloves or disposable barrier sleeves or specially designed plastic gowns with sleeves. Provision of protection to the arms

is unsatisfactory at present in our practice. An easily adaptable technique using ordinary gloves is demonstrated in the photographs provided.

5. **Footwear:** The dorsum of the feet should be covered by impervious footwear that is thick enough to prevent accidental injuries like blood spills and falling scalpels etc. A pair of such footwear should be kept exclusively for OR use. These may be covered with a pair of overshoes to decrease bloodstaining and for ease of cleaning. (This *should not replace* impervious foot wear) The overshoes should ideally be of disposable, impervious material, but cloth overshoes over leather / plastic footwear is an acceptable alternative in our situation. Knee length rubber boots are advised where pooling of blood and fluid on the floor is likely, e.g. Caesarean section and urology.

*Thus a "well dressed" surgeon in the 21<sup>st</sup> century would have protective eye wear, a disposable mask, a plastic apron under the gown (or impervious gowns), double gloves, and well fitting shoes with an overshoe.*

6. One area that is frequently overlooked is that of aerosols created during surgery. These include CO<sub>2</sub> laser therapy, laparoscopic surgery, procedures using high-speed drills etc. It is prudent to assume that such aerosols may be infectious, and effective smoke evacuation systems should be in place. Laparoscopic surgery is not contraindicated in HIV positive individuals if aerosols are effectively dealt with. Masks should be well fitting and of the high filtration variety.

#### **Avoiding sharps injury**

Cuts and pricks by scalpels and solid needles are the commonest sharps injuries encountered by the surgeon and his team. Solid needles are less likely to result in seroconversion than hollow needles and most literature on this issue is with regard to hollow needles.

Minor changes in surgical practice and a progressive mindset are necessary to reduce the chance of intraoperative needle stick injuries.

The following modifications in technique are recommended.

1. Use of forceps or another needle holder to retrieve suture needles from tissues. While older surgeons, who were trained in an era where needles were reused and needed to be treated "with respect" often use their fingers to retrieve needles, younger surgeons and surgeon in training need to be taught to change practice in

order to avoid injury. The most common site of injury is the tip of the index finger of the non dominant hand.

2. Use of forceps or instruments to hold tissues while suturing / cutting and not the hand. This is especially important while closing the abdomen after a laparotomy.
3. Avoid attempts to palpate needle tips obscured by tissue or blood. Suck all blood and retract tissues with retractors, sponge sticks or mops. Assistants should not enthusiastically mop while the surgeon is suturing.
4. Do not pull needles out towards the non dominant hand or the assistant's hand.
5. Avoid tying sutures with the needle attached to the suture. It is safer, though more expensive to cut the needle off before tying.
6. Sharp instruments such as mounted needles, cautery tips, staples etc on the table or patient should be stored on a corner of the Mayo stand or the scrub nurses' trolley when not in use. Foam and sticky pads are available to store used needles. Magnetic pads are also available.
7. "Economy of movement". Certain practices need to be relearnt by all members of the surgical team. It is good practice to make sure that no two people touch the same sharp instrument at the same time. If they do, it is prudent to ensure that only one person's hand is moving at any one time. Rapid, jerky movements should be avoided. If a sharp is placed on the operating field or patient, it should be accompanied by a verbal warning such as "sharp", "knife" or "cautery". Scrub nurses have been traditionally taught to pass instruments to surgeons directly. Scrub nurses should avoid handling suture needles with their fingers and should use instruments to load them. All sharps should be passed between surgeons and nurses through a "neutral zone", usually a kidney tray.
8. Where major blood splashes are anticipated, eg. vascular surgery, vascular anastomoses can be inspected through a Petri dish cover. Irrigation and suction can also be performed behind such transparent shields.
9. Decreasing the number of personnel in the OR will decrease the opportunities for exposure.
10. When the patient is known to be seropositive, perhaps the one single factor that decreases such injuries is an experienced surgeon <sup>4</sup>. Assistants should be relaxed, not angry or anxious and assist without coercion.

11. Improvement in technology. Blunt tipped needles are available though the "industry" has been reluctant to introduce these in developing economies. Scalpel blades with blunt tips serve the same purpose as conventional blades.
12. Use of alternative instruments. The use of scalpels and scissors can be reduced significantly if the surgeon is willing to dissect with the electrocautery hand held handle, as most American surgeons do. Scissors in place of scalpels for dissection will reduce the chance of injury. Electrocautery can be used to cut skin, if properly used. Many institutions have eliminated the use of scalpel blades during surgery <sup>4</sup>. Staplers for bowel anastomoses and skin closure are recommended, but this has to be weighed against increased costs.
13. Performance of a lesser procedure often suffices in patients known to be seropositive, eg. Percutaneous drainage of intra abdominal abscess, FNAC instead of open biopsy, etc.

Thus, in this day and age, it is important that all HCW involved in and around operating theatres practice their craft with a high degree of discipline and integrity.

### REFERENCES

1. CDC (2001) Updated U.S Public Health Service Guidelines for the management of occupational exposures to HBV, HCV and HIV and recommendations for post-exposure prophylaxis. *MMWR* 50/No. RR-11: 1-54.
2. Hospital Infection Control Committee, Christian Medical College and Hospital (2001) Guidelines and policies in HIV care.
3. Universal precautions for prevention of transmission of HIV and other blood borne infections.  
<http://www.cdc.gov/ncidod/hip/BLOOD/UNIVERSA.HTM>
4. International Health Care Safety Centre (1998). Annual Number of Occupational Percutaneous Injuries and Mucocutaneous Exposures to Blood or Potentially Infective Biological Substances.  
<http://hsc.virginia.edu/medcntr/centers/epinet/cdcestim.html>
5. Immunisation Action Coalition. Hepatitis B and the health care worker  
<http://www.immunize.org>

### FURTHER READING

1. CDC(1987) Recommendations for prevention of HIV transmission in health-care settings. *MMWR* 36(suppl no. 2S).
2. CDC(1988) Update: Universal precautions for prevention of transmission of human immunodeficiency virus, hepatitis B virus, and other bloodborne pathogens in health-care settings. *MMWR* 37:377-388.
6. CDC (1989) Guidelines for prevention of transmission of human immunodeficiency virus and hepatitis B virus to health-care and public-safety workers. *MMWR* 38(S-6):1-36.
7. Lin EY, Burnicardi FC (1994) HIV Infections and Surgeons. *World J Surg*; 18(5): 753-8.
5. Smoot EC (1998) Practical Precautions for Avoiding Sharp Injuries and Blood Exposure. *Plastic Recons Surg*. 101(2): 528-34.
6. Schiff SJ (1990) A surgeon's risk of AIDS. *J Neurosurg* 73(5):651-60.
7. Crombleholme WR (1990) HIV Infection. Managing exposure risks for the Obstetrician / Gynecologist. *Obstet Gynecol Clin. North Am.* 17 (3): 627-36.
8. Stotter AT, Vipond MN, Guillou PJ (1993) The response of general surgeons to HIV in England and Wales. *Ann R Coll Surg Engl.* 75(5): 330-2.
9. Gerberding JL, Littell C, Tarkington A, Brown A, Schechter WP (1990) Risk of exposure of surgical personnel to patients' blood during surgery at San Francisco General Hospital. *N Engl J Med* 322:1788-93.
10. Zuger A, Miles SH. Physicians, AIDS, and occupational risk: Historic traditions and ethical obligation. *JAMA* 1987;258:1924-8.
11. [http://medocs.ucdavis.edu/osu/421/orthopedic ethics/case 6/case 6.htm](http://medocs.ucdavis.edu/osu/421/orthopedic%20ethics/case%206/case%206.htm)
12. Selecting, evaluating and using sharp disposal containers.  
<http://www.cdc.gov/niosh/sharps>

# Hepatitis B and the health care worker

## CDC answers frequently asked questions about how to protect health care workers

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### Which workers in the health care setting need hepatitis B vaccine?

Persons who have a reasonable expectation of being exposed to blood on the job should be offered hepatitis B vaccine. This does not include receptionists, executive directors, billing staff, general office workers, etc., as these groups would not be expected to have occupational risk.

### What is the appropriate site for administration of hepatitis B vaccine and what needle length and gauge should be used?

The deltoid is recommended for routine intramuscular vaccination in adults, particularly for hepatitis B vaccine. The suggested needle size is 1 to 1½ inches and 20 to 25 gauge.

### A health care worker's (HCW) first dose of hepatitis B vaccine was four months ago. Should the series be restarted?

No. The vaccine series does not need to be restarted. The person should receive the second dose at this time and third dose 2–6 months later.

### Is it safe for HCWs to be vaccinated during pregnancy?

Yes. Pregnant women in occupations with a high risk of HBV infection should be vaccinated. Hepatitis B vaccine contains no components that have been shown to pose a risk to the fetus at any time during gestation. However, HBV infection during pregnancy poses a significant risk to the fetus or newborn of perinatal or *in utero* infection.

### Which HCWs need serologic testing after receiving 3 doses of hepatitis B vaccine?

Persons at occupational risk of infection and with continued percutaneous or percutaneous exposures to blood or body fluids (e.g., HCWs with direct patient contact, HCWs who have the risk of needlestick or sharps injury, lab workers who draw and test blood) should be tested after vaccination. Testing should be done 1–2 months after the last dose of vaccine.

### What should be done if a HCW's serologic test comes back negative for anti-HBs?

Repeat the 3-dose series and then test for anti-HBs 1–2 months after the last dose of vaccine. If the

HCW is still negative after a second vaccine series, the HCW is considered a non-responder to hepatitis B vaccination. The HCW should be counseled that non-response to the vaccination series most likely means that the HCW is susceptible to HBV infection. It is possible, however, that the HCW is chronically infected with HBV and HBsAg testing should be recommended. Counseling of the HCW should then be done to discuss what non-response to the vaccination series means for that specific HCW and what steps should be taken in the future to protect his/her health.

### How often should anti-HBs titers be drawn on HCWs who perform invasive procedures?

No healthy person needs to be repeatedly tested for anti-HBs. Persons who perform invasive procedures should be treated no differently from other health care workers with respect to anti-HBs testing. If a health care worker has an exposure (e.g., needlestick) he or she should be evaluated for postexposure prophylaxis according to current recommendations (see table below).

**You need more than hepatitis B shots!**

To obtain the ACIP statement

"Immunization of Health-Care Workers" call (800) 232-2522

*Should a HCW who performs invasive procedures and who once had a positive anti-HBs result be revaccinated if the anti-HBs titer is rechecked and is less than 10mIU/mL?*

No. Postvaccination testing should be done only 1–2 months after the original vaccine series is completed. Testing showed that the HCW was protected as a result of the original vaccination series. Data show that adequate response to the 3-dose series of hepatitis B vaccine provides long-term immunologic memory that gives long-term protection. Only immunocompromised persons (e.g., hemodialysis patients, HIV-positive persons) need to have anti-HBs testing and booster doses of vaccine to maintain their anti-HBs concentrations of at least 10mIU/mL in order to be protected against HBV infection.

### If HCWs were vaccinated for hepatitis B in the past and not tested for immunity, should they be tested now?

No. A HCW does not need to be tested unless he or she has an exposure. If an exposure occurs, refer to the table below for management guidelines. In addition to following these guidelines, if prophylaxis (HBIG and a booster dose of vaccine) is indicated, the person should receive postvaccination testing 3–6 months afterwards. It is necessary to do postvaccination testing at 3–6 months as earlier testing may just measure antibody from HBIG. This postvaccination anti-HBs test result should be recorded in the person's health record.

Recommended postexposure prophylaxis for percutaneous or percutaneous exposure to hepatitis B virus, United States*			
Vaccination and antibody response status of exposed person	Treatment when source is		
	HBsAg <sup>1</sup> positive	HBsAg negative	Source not tested or status unknown
Unvaccinated	HBIG <sup>2</sup> x 1; initiate HB vaccine series <sup>3</sup>	Initiate HB vaccine series	Initiate HB vaccine series
Previously vaccinated:			
Known responder <sup>4</sup>	No treatment	No treatment	No treatment
Known non-responder	HBIG x 2 or HBIG x 1 and initiate revaccination	No treatment	If known high-risk source, treat as if source were HBsAg positive
Antibody response unknown	Test exposed person for anti-HBs <sup>5</sup> 1. If adequate <sup>4</sup> , no treatment 2. If inadequate <sup>4</sup> , HBIG x 1 and vaccine booster	No treatment	Test exposed person for anti-HBs 1. If adequate <sup>4</sup> , no treatment 2. If inadequate <sup>4</sup> , initiate revaccination

<sup>1</sup> Hepatitis B surface antigen

<sup>2</sup> Hepatitis B immune globulin; dose 0.06 mL/kg intramuscularly

<sup>3</sup> Hepatitis B vaccine

<sup>4</sup> Responder is defined as a person with adequate levels of serum antibody to hepatitis B surface antigen (i.e., anti-HBs ≥10 mIU/mL); inadequate response to vaccination defined as serum anti-HBs <10 mIU/mL

<sup>5</sup> Antibody to hepatitis B surface antigen

\* from "Immunization of Health-Care Workers," MMWR, 1997; 46 (RR-18).

Item #P2109 (3/01)

**For a pre-employment physical, a health care worker states she received all three hepatitis B vaccine doses as an adolescent. Would you do a titer?**

This is a situation that will become more common in the future and for which there are no specific guidelines. A reasonable approach, however, can be developed from current recommendations. Currently, CDC recommends postvaccination testing for antibody to hepatitis B surface antigen (anti-HBs) 1-2 months after the last dose of hepatitis B vaccine for persons vaccinated as health care workers or in training. This employee was vaccinated as an adolescent, and postvaccination testing was not done since it was not indicated at the time of vaccination.

If the health care worker has written documentation of three doses of vaccine given as an adolescent, that should be sufficient to meet the needs of the employer and the requirements of OSHA guidelines. Another option would be to test the person for the presence of anti-HBs, since a person vaccinated as an adolescent is still likely to have detectable antibody. If the person, however, is anti-HBs negative on testing, that does not mean s/he was not immunized, since s/he could have lost detectable antibody over time and still be protected. If the person is found to be anti-HBs negative, that status should be recorded on her/his employee health record along with the vaccination history. If the health care worker subsequently has a blood exposure, s/he should follow the current guidelines for postexposure immunoprophylaxis. If the health care worker has no written documentation of vaccination as an adolescent, the person should receive the 3-dose vaccine series and anti-HBs testing 1-2 months after the full series.

**I oversee the employees of a clinic in which all the health care workers decided to check their anti-HBs titers (15 employees got tested). Eight of them had titers less than 10 mIU/mL, although two of them had previously had adequate titers. The other seven had not been previously tested. What should I do?**

CDC does not recommend periodic testing for anti-HBs or booster doses of hepatitis B vaccine

for immune competent persons. When testing is done as described above, it places the employee health service in a difficult position. The two employees who previously had documented adequate titers should have nothing done as they are protected. It also appears that 7 of the 15 employees had adequate levels of anti-HBs when tested. That leaves 6 employees in which it is not known if they had previously responded to hepatitis B vaccination and now have undetectable anti-HBs. The most helpful approach to define the issue, would be to give one dose of vaccine to each of the employees and then test anti-HBs in one month. For employees with adequate anti-HBs ( $\geq 10$  mIU/mL), nothing more need be done, as they are protected. For employees with inadequate anti-HBs after one additional dose of vaccine, we would complete the revaccination series by giving two more doses of vaccine according to the recommended schedule and test 1-2 months after the third dose of vaccine. If anti-HBs is adequate, they are protected; if inadequate, they are "non-responders" to the vaccine.

**There are several physicians in our group who have no documentation of having received hepatitis B vaccine but are relatively sure they received the doses many years ago. What do we do now?**

Unfortunately, inadequate documentation of vaccination is common. Even if these physicians think they may have been fully vaccinated, but it is not documented, the three-dose vaccination series should be administered and post-vaccination testing should be performed 1-2 months after the three-dose series. There is no harm in receiving extra doses of vaccine.

Some might suggest giving only one dose of vaccine followed by post-vaccination testing. Although 30% of previously unvaccinated healthy adults will have a protective antibody response after only one dose of vaccine, these individuals will not have the long-term protection afforded by the three-dose series.

Each organization (hospital, clinic, etc.) should develop policies or guidelines as to the documen-

tation required to ensure valid hepatitis B vaccination. If policies are in place and documentation is not present, revaccination should be instituted. Care should always be taken to document vaccine lot, date, manufacturer, route, and vaccine doses. Postvaccination testing results should also be documented, including the date testing was performed.

**I'm a nurse who received the hepatitis B vaccine series over 10 years ago and had a positive follow-up titer. At present, my titer is negative. What should I do now?**

Nothing. Current data show that vaccine-induced anti-HBs levels may decline over time; however, immune memory (anamnestic anti-HBs response) remains intact indefinitely following immunization. Persons with declining antibody levels are still protected against clinical illness and chronic disease. For health care workers with normal immune status who have demonstrated an anti-HBs response following vaccination, booster doses of vaccine are not recommended nor is periodic anti-HBs testing. ♦

## Keep your own vaccination history!

Record the dates you received hepatitis B vaccine, as well as the results of your postvaccination serology (anti-HBs).

Remember to save records of any vaccinations you receive so you don't have to repeat them.



MANI KALLIDJA

HIV Physician Training Course 2002,  
Christian Medical College, Vellore

DISTANCE LEARNING COURSE

**HIV  
&  
GASTROINTESTINAL SYSTEM**

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**MODULE 7**

**INSTRUCTION SHEET - HIV AND GASTROINTESTINAL SYSTEM**  
**(MODULE 7)**

1. In addition to this module you will find X-ray 7-1 in separate cover for activity 7.5.
2. After you complete the module tear: (a) Tutor marked assignment (page 30); (b) the module evaluation form (at the end of the module) and enclose it the stamped envelop. Send it by registered post by: January 25, 2003.
3. Please write your name and roll number on the tutor marked assignment before dispatching it.

## OVERVIEW

This module will help you to improve your knowledge and help you develop your clinical services in relation to the gastrointestinal manifestations associated with HIV infection.

The gastrointestinal system is the largest lymphoid system in the human body. It plays a crucial role in the pathogenesis of AIDS. It may provide a portal of entry for the virus (eg. HIV infection transmitted through breast milk). HIV infection of the gastrointestinal tract results in local immunosuppression as a direct consequence of which the gut is vulnerable to a variety of opportunistic pathogens.

Ninety percent 90% of HIV patients from developing countries present with gastrointestinal symptoms. Particular enteric pathogens or neoplasms can help establish the diagnosis of AIDS.

The most common gastrointestinal presentations are: diarrhoea, dysphagia, abdominal pain and jaundice. This module will help you learn the clinical approach to these symptoms by working through a set of clinical problems.

**OBJECTIVES**

After completion of this module the student should be able to:

1. Describe:

- i. The clinical approach to the evaluation of syndromes of the gastrointestinal system: (a) odynophagia/ dysphagia; (b) abdominal pain; (c) diarrhoea; (d) jaundice.
- ii. The causative agents of the syndromes.
  - i. The diagnostic evaluation of these syndromes.
  - iii. The specific treatment of the syndromes and their etiological agents.

2. Apply the clinical algorithms to the evaluation and management of clinical case problems of gastrointestinal disease.

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<i>Reading 7.1 Dysphagia and odynophagia</i>	<i>30</i>	<i>33-36</i>
<b>Activity 7.2 Non-resolving dysphagia</b>	<b>10</b>	<b>7</b>
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<i>Reading 7.3 Abdominal pain</i>	<i>30</i>	<i>37-39</i>
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<b>Activity 7.5 Abdominal pain – Part III</b>	<b>10</b>	<b>13</b>
<b>Activity 7.6 Diarrhoea – Part I</b>	<b>10</b>	<b>15</b>
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<b>Activity 7.7 Diarrhoea – Part II</b>	<b>10</b>	<b>17</b>
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<b>Activity 7.11 Jaundice – Part II</b>	<b>10</b>	<b>25</b>
<b>Activity 7.12 Jaundice – Part III</b>	<b>10</b>	<b>27</b>
<b>TMA</b>	<b>60</b>	<b>30</b>
<b>Total estimated study time</b>	<b>320</b>	

The following exercise will help you learn the approach to diagnosis and management of a patient presenting with swallowing difficulty. Read the section, "Dysphagia and Odynophagia" (pg. 33-37) in the reader. After you have completed the reading you may proceed to the activity.



### ACTIVITY 7.1

#### APPROACH TO DYSPHAGIA (TIME: 15 MINUTES)

Mr. Manu Nair is a forty-five year old engineer working in a small manufacturing firm in Mumbai. He had an abdominal surgery ten years ago, during which time he was transfused one unit of blood. Two years ago he was found to be HIV seropositive during an annual office health checkup. He now presents with a history of difficulty in swallowing and occasional severe pain in the retrosternal region after swallowing.

1. What is the differential diagnosis that you will consider in Mr. Nair in order of probability?

1. - Oesophageal candidiasis
2. - CMV oesophagitis
3. - Herpes Simplex oesophagitis
4. - Tubercular oesophagitis
5. - Primary HIV oesophagitis

2. What additional history would you like to obtain from Mr. Nair?

1. of retinitis
2. of TB
3. of foreign body/mechanical obstruction

3. What specific findings will you look for when you examine Mr. Nair?

1. Oral candidiasis
2. Oral herpetic ulcers
3. Signs of AIDS disease
4. Retinitis

4. Examine the photograph (Fig. 7-A on page 51). What is your diagnosis?

Oral candidiasis

5. What treatment will you institute for this patient?

1. Fluconazole 100mg OD x 2 wks + local Fluconazole if no improvement → Refw.



### FEEDBACK 7.1

1. What is the differential diagnosis that you will consider in Mr. Nair in order of probability?

Mr. Nair has dysphagia and retrosternal pain suggesting the presence of esophagitis. The differential diagnosis of this complaint is:

1. Candida esophagitis
2. Herpes esophagitis
3. CMV esophagitis
4. HIV induced esophagitis
5. Drug induced esophagitis

2. What additional history would you like to obtain from Mr. Nair?

1. Prior history of candidal infection and use of fluconazole.
2. History of visual blurring or bloody diarrhea which may occur with CMV infection.
3. History of use of Doxycycline, AZT or DDC which can cause pill induced esophagitis.
4. Food and fluid intake in the past few days.

3. You are then asked to examine Mr. Nair. What are the specific things that you will look for in his condition?

1. Curdy white lesions on the surface of the tongue and the buccal mucosa diagnostic of candida esophagitis.
2. Grouped vesicular lesions on the surface of the mouth suggestive of herpes stomatitis and lesions of genital herpes.
3. Fundus examination for large yellow and white lesions with granular borders and perivascular exudates and haemorrhages suggestive of CMV retinitis (cottage cheese and ketchup appearance).
4. Sign of malnutrition and dehydration.

4. Examine the photograph (Fig. 7-A on page 51). What is your diagnosis?

The photo shows curdy white precipitate on the tongue suggestive of candidal infection. The presence of oral candidiasis in a patient with symptoms of esophagitis usually indicate the presence of candidal esophagitis.

5. What treatment will you institute for this patient?

T. Fluconazole 100-200 mg OD for 2 weeks.

If he has been on regular fluconazole prophylaxis, the dose of fluconazole may be increased to 400-800 mg daily or changed to Itraconazole solution. If he is unable to swallow, then Syrup Itraconazole or IV Fluconazole may be administered till he is able to swallow. If he is unable to swallow at all then in-patient admission and IV fluids are required.

The next activity will help you learn about non-resolving dysphagia.



## ACTIVITY 7.2

### NON-RESOLVING DYSPHAGIA (TIME: 10 MINUTES)

Mr. Nair initially improved after treatment with Fluconazole. However he returns to your clinic because of recurrence of symptoms.

1. What is your differential diagnosis and which further tests will you order?

- Resistance to Fluconazole - Culture & sensitivity
- Oesophageal endoscopy & biopsy

2. Examine the test reports shown in Fig. 7-B and 7-C (page 51) and write down your findings?

→ Inclusion bodies, acantho cells

3. What is your diagnosis based on these laboratory findings?

C. Oesophagitis

4. What treatment will you administer to this patient (drug choice, dose and duration)?

Baclofen 5mg od x 3-6 wks





## FEEDBACK 7.2

1. What is your differential diagnosis and which further tests will you order?

1. Recurrence of candida esophagitis
2. Herpes
3. CMV
4. HIV induced

The patient needs to be referred for an upper GI endoscopy.

2. Examine the upper GI endoscopy picture in Fig. 7-B and histopathology of lesion in 7-C (page 51) and write down your findings?

7-B - Large shallow superficial ulcers at the distal end of the esophagus.

7-C - The histopathology - numerous owl eye type inclusion bodies

3. What is your diagnosis based on these laboratory findings?

The presence of owl eye type inclusion bodies is diagnostic of CMV esophagitis.

4. What treatment will you administer to this patient (drug choice, dose and duration)?

Gancyclovir 5 mg/Kg IV OD (or 6 mg/Kg IV 5 days a week) for 4-6 weeks

The dose needs to be adjusted according to renal functions. The duration of the induction therapy is longer than for CMV retinitis for which induction therapy is 14-21 days. In patients who are resistant to Gancyclovir, IV Foscarnet may be used.

The following exercise will help you learn the approach to diagnosis and management of a patient presenting with abdominal pain. Read the section, "Abdominal pain" (pg. 37-39) in the reader. After you have completed the reading you may proceed to the activity.



### ACTIVITY 7.3

#### ABDOMINAL PAIN - PART I (TIME: 10 MINUTES)

37 year old Mr. Kumar a lorry driver, was diagnosed to have HIV infection 3 years ago when he presented with oral candidiasis. He presented with complaints of moderate to severe upper abdominal pain, with progressive weight loss and low grade fever for the last 2 months.

1. What history will you elicit from the patient to localise the anatomical site of the pathology?

- Periodicity and severity
- Generalized vs location
- Associated vomiting, vomiting, diarrhoea, swelling
- H/o TB, Oesophagitis,

2. What physical findings will you look for in this patient?

- Localizing pain vs guarding ~~and~~
- Organomegaly or free fluid
- Observable features in pancreatitis, arthritis/dysenteritis
- B.S.
- Hydration and nutrition

**FEEDBACK 7.3**

1. What history will you elicit from the patient to localise the anatomical site of the pathology?

Stomach and duodenum- burning upper abdominal pain with vomiting soon after food  
Pancreas- constant pain in the epigastrium and radiating to the back and relieved on leaning forward.

Cholecystitis and cholangitis- right hypochondrial pain radiating to the scapula and right shoulder, jaundice, fever

Enteritis- diarrhea and vomiting

Surgical causes (obstruction, perforation and peritonitis)

Progressive abdominal distension, vomiting

2. What physical findings will you look for in this patient?

Jaundice, tenderness, guarding, rigidity and rebound, Murphy's sign, liver and spleen, other masses, shifting dullness and fluid thrill, bowel sounds



## ACTIVITY 7.4

## ABDOMINAL PAIN - PART II (TIME: 10 MINUTES)

Mr. Kumar complained of upper abdominal pain which was fairly constant and radiated to the back. It was associated with vomiting which occurred soon after food. The bowels habits were fairly regular. He had lost about 10 Kg of weight over the last several months. He had no history of jaundice. He had been receiving Bactrim prophylaxis but had not received any anti-viral treatment. He does not smoke or consume alcohol.

On examination he was looking ill. Pulse rate 90/minute, Respiratory rate 16/minute, temperature 101° F. Pallor present, multiple cervical lymph nodes 1.5-2 cm in size, Liver 5 cm, Spleen 2 cm. Ill-defined tender and firm mass in the epigastrium 4-6 cm in size. Shifting dullness present.

1. What inference will you draw from the patient's description of abdominal pain?

Gastritis, duodenal/mesenteric involvement

2. What inferences can be drawn based on the examination findings and what are the possible differential diagnosis?

Generalized infiltrative process  
 - Disseminated TB ± infiltration of mesenteric nodes  
 - " KAPC  
 - Lymphoma.

3. What tests will you order for Mr. Kumar?

• Ascitic fluid - Gram, AFB, fungal stain, protein, cells  
 • ~~TIBC~~ Blood picture; S. Amylase  
 • PNBAC, U/S scan, LFT

? - Creatinine, fasting sugar



### FEEDBACK 7.4

1. What inference will you draw from the patient's description of abdominal pain?

The pain is of pancreatic origin as it has the characteristic description of upper abdominal pain radiating to the back. He has not been exposed to the common drugs and toxins which results in pancreatitis (alcohol, <sup>AZT</sup>ddI, 3TC and Pentamidine) in a HIV patient. The pain may be the result of the mass in the epigastrium which may arise from the pancreas or structures surrounding the pancreas.

2. What inferences can be drawn based on the examination findings and what are the possible differential diagnosis?

<u>Finding</u>	<u>Differential diagnosis</u>
Cervical adenopathy	TB, cryptococcosis, lymphoma, PGL
Hepatosplenomegaly	TB, cryptococcosis
Intraabdominal mass	TB lymphadenitis, lymphoma
Shifting dullness	TB
Sluggish bowel sounds:	TB

This patient has generalised lymphadenopathy, hepatosplenomegaly and probably intra-abdominal lymphadenopathy. The most likely diagnosis are disseminated tuberculosis and lymphoma. While MAI can present in a similar way it is not common in India and may not be strongly considered in the differential diagnosis.

3. What tests will you order for Mr. Kumar?

WBC total and differential count  
 Creatinine, Fasting sugar, Liver function tests ✓  
 Chest x-ray  
 Fine needle aspiration of lymph node and AFB smear  
 Ultra-sound of the abdomen  
 Serum amylase



## ACTIVITY 7.5

## ABDOMINAL PAIN - PART III (TIME: 10 MINUTES)

WBC total count - 2700 cells/mm<sup>3</sup> Differential count - Neutrophils 60, lymphocytes 26, bandforms 6, monocytes 12. Liver function tests- Total bilirubin 1.0 mg%, Direct bilirubin 0.8 mg%, SGOT 120 U/L, SGPT 150 U/L, Alkaline phosphatase 180 IU/L. Ultrasound of abdomen- multiple cystic peri-pancreatic nodes, hepatosplenomegaly, free fluid present in the abdomen (see X-ray 7-1 in x-ray cover). Serum amylase 70 U/L. Chest x-ray- mediastinal adenopathy. Fine needle aspiration of cervical nodes- Numerous AFB seen.

1. What is your interpretation of the results and what further tests will you order for this patient?

Disseminated TB, infiltrations in liver, Peri pancreatic nodes, bone marrow infiltrations  
 ? Fairly large infiltrations in liver - 3 times raised enzymes  
 ? Meningeal involvement / chronic obstruction  
 CSF, Barium swallow.

2. What treatment will you initiate?

Regime 1  $\frac{1}{4}$  R E.D. 2+4  
 $\frac{1}{4}$   $\frac{1}{2}$



### FEEDBACK 7.5

1. What is your interpretation of the results and what further tests will you order for this patient?

This patient has advanced immunodeficiency as indicated by the absolute lymphocyte count of  $<1000$  cells/mm<sup>3</sup>. The normal serum amylase rules out acute pancreatitis. The liver function tests show mild elevation of liver enzymes and alkaline phosphatase which could be consistent with a diffuse infiltrative process in the liver but does not rule out anicteric hepatitis. The chest x-ray confirms the presence of generalised lymphadenopathy. The ultra-sound confirms the presence of hepatosplenomegaly and intra-abdominal adenopathy with peri-pancreatic involvement and peritoneal free fluid. The demonstration of acid fast bacilli on the FNAC confirms the process to be due to disseminated tuberculosis. It does not rule out MAI infection or another co-existent intra-abdominal process.

The further tests that may be required are AFB culture on the lymph node aspirate. If a second diagnosis is strongly being considered then ultrasound guided aspiration of the peri-pancreatic node or peritoneal aspiration should be performed.

2. What treatment will you initiate?

Anti-TB treatment should be initiated with 4 drug regimen, Rifampicin, INAH, Ethambutol and Pyrazinamide at appropriate doses. Bactrim prophylaxis needs to be initiated. The abnormal liver function tests are not a contra-indication for initiation of hepatotoxic anti-TB drugs. However he needs to be carefully monitored for the development of hepatotoxicity.

Mr. Kumar was treated with IV fluids and anti-TB treatment with Rifampicin, Isoniazid, Pyrazinamide and Ehtambutol with which he showed gradual improvement of symptoms. At review after 3 months his symptoms had resolved and he was gaining weight. His AFB culture was reported as M. tuberculosis.

The following exercise will help you learn the approach to diagnosis and management of a patient presenting with diarrhoea. Read the section, "Diarrhoea" (pg. 40-44) in the reader. After you have completed the reading you may proceed to the activity.



### ACTIVITY 7.6

#### DIARRHOEA - PART I (TIME: 10 MINUTES)

Mrs. Selvi an asymptomatic HIV positive patient presented with small volume stools mixed with blood, cramping abdominal pain of 2 days associated with fever.

On examination pulse rate 120 /minute BP 90/70 mm Hg Temperature 101.5° F Dry tongue Abdomen- mild peri-umbilical tenderness

1. What are your inferences based on the history and examination?

Colonic, likely bacterial - ? Periumbilical

2. What are the likely causes of diarrhoea in this patient?

Protozoa - Amoeba - ? Micro/Cryptosporidia/Cyclospora  
Bacteria - Shigella, Salmonella, Campylo.

3. What tests would you like to order for her?

Stool cultures, ova cysts, saline, modified acid fast, iodine

4. What treatment will you administer?

Sipran - Albendazole / Ciproflox.



**FEEDBACK 7.6**

1. What are your inferences based on the history and examination?

This patient has an acute diarrheal illness with features suggestive of an acute bacillary dysentery and mild dehydration. She is febrile but there are no features which suggest the presence of sepsis syndrome.

2. What are the likely causes of diarrhea in this patient?

The causes of acute diarrhea illness in HIV infection include:

Shigella sp.

Salmonella typhimurium and enteritidis

Vibrio sp.

Campylobacter jejuni

E. coli

Viruses

Of these Shigella and Campylobacter can produce a dysenteric illness and are therefore the likely pathogens.

3. What tests would you like to order for him?

This patient could be treated without the aid of any laboratory tests. Stool examination for faecal leucocytes may be done to confirm the diagnosis of bacillary dysentery. Stool culture may be performed if facilities are available.

4. What treatment will you administer?

Oral rehydration

T. Ciprofloxacin 500 mg BD for 3-7 days.



## ACTIVITY 7.7

## DIARRHOEA - PART II (TIME: 10 MINUTES)

Mr. Selvam is a thirty five year old miner from Kolar was diagnosed to have HIV infection when he presented to his GP 3 years ago with gonorrhoea. He now complains of diarrhoea occurring 3-5 times day for the last 4 months and progressive weight loss. He has been treated by his local doctors with several course of antibiotics with out much relief.

1. Write down the further historical points that you would like to elicit from Mr. Selvam?

- details of diarrhoea - volume, blood, tenesmus, etc.  
 - H/o or malabsorption & malnutrition  
 - what drugs - esp anti-protozoal / oral

2. What specific examination findings would you look for in Mr. Selvam?

Localizing - upper vs lower segment  
 - proctoscopy & rectal exam.  
 - malabsorption features



### FEEDBACK 7.7

1. Write down the further historical points that you would like to elicit from Mr. Selvam?

CONDITION	QUESTIONS
Small bowel diarrhoea	Large volume Less frequent Borborygmi Periumbilical pain
Large bowel diarrhoea	Small volume Frequent Tenesmus Hematochezia
Anorectal disease	Rectal pain Fresh bleeding
Protein energy malnutrition	Weight loss Leg swelling Facial puffiness Night blindness
Vitamin deficiencies	Fissures over angles of mouth Loss of taste Tongue burning Paraesthesias
Mineral deficiencies	Proximal weakness
TB	Fever, respiratory symptoms
Cryptococcal meningitis	Central nervous system symptoms

2. What specific examination findings would you look for in Mr. Selvam?

General examination- Pallor, Bitot spots, angular stomatitis, glossitis, pedal oedema, echymoses,  
Respiratory examination- Lower respiratory signs  
Neurological signs- Peripheral neuropathy, proximal weakness, fundal signs of CMV retinitis  
Gastrointestinal system- Abdominal tenderness, peri-anal ulcers



## ACTIVITY 7.8

## DIARRHOEA - PART III (TIME: 10 MINUTES)

Mr. Selvam said that the diarrhoea was extremely troublesome, as it occurred up to six times a day and about four times in the night as well. The stools were large volume and watery. He had not noticed any blood or complained of tenesmus. He often experienced minimal pain around his umbilicus. He has lost significant weight in the last month. There is no history of prolonged fever or respiratory symptoms.

On examination:

Pulse rate 102/minute BP 90/70 mm Hg Weight 40 Kg Severe emaciation. Pallor, glossitis, angular stomatitis, koilonychia, pedal oedema present. No jaundice

Abdomen- no hepatosplenomegaly or free fluid. No perianal lesions

CNS- ankles jerks are absent, fundus examination

1. What are your inferences based on the history and examination?

Small bowel diarrhoea - Malnutrition - protein, mineral, vitamins. Also

2. What are the likely causes of diarrhea in this patient?

Protozoa (except *E. histolytica* & *Giardia*)

3. What tests would you like to order for him?

Stool acid fast, fungal, ? chrome



### FEEDBACK 7.8

1. What are your inferences based on the history and examination?

This patient has a chronic small bowel diarrhea non-responsive to antibiotic therapy. The clinical examination shows evidence of protein energy malnutrition (emaciation, weight 40 Kg), iron deficiency (anemia and koilonychias), hypoproteinemia (pedal oedema), riboflavin deficiency (angular stomatitis, glossitis), peripheral neuropathy (absent ankle jerks) which may be due to HIV infection or folic acid or B12 deficiency).

2. What are the likely causes of diarrhea in this patient?

The common pathogens causing chronic diarrhea in HIV infection in India are protozoal agents: *Isospora belli*, *Cyclospora*, *Cryptosporidia* and *Giardia lamblia*. However a variety of other pathogens may cause chronic diarrhea including: *Microsporidia*, *Shigella sp.*, *Salmonella sp.*, *Vibrio cholera*, *E. histolytica* and *Cytomegalovirus*. In a proportion of patients no pathogen may be found and the diarrhea may be due to HIV enteropathy.

3. What tests would you like to order for him?

? Stool examination-ova and cysts (Giemsa, Trichrome and acid fast stain)  
 ? Stool culture  
 Haemoglobin, WBC total count and differential count, blood picture  
 ? Liver function tests, Electrolyte profile (if possible)



## ACTIVITY 7.9

## DIARRHOEA - PART IV (TIME: 10 MINUTES)

## Laboratory reports:

Haemoglobin 7.2g%, WBC count 5200 cells/mm<sup>3</sup> Neutrophils 75  
Lymphocytes 15 Basophils 3 Monocytes 7 Blood smear-  
dimorphic blood picture (microcytosis, occasional  
macrocytes and hypersegmented neutrophils seen). Liver  
function tests- Total bilirubin 1.2 Direct bilirubin 0.9  
total protein 7.0 g/dl globulin 4.5 g/dl SGOT 30 SGPT 40  
Alkaline phosphatase 120

Stool examination - see Fig. 7-D (page 52)

Reported to be *Isospora belli* cysts seen

1. What treatment will you initiate for this patient?

**FEEDBACK 7.9**

1. What treatment will you initiate for this patient?

TMP/SMX DS I qid for 10 days

TMP/SMX DS I bd for 3 weeks

Followed by Bactrim DS I OD life long suppression

Rehydration

Nutritional supplementation-

Calorie, protein, fat ( as tolerated)

Ferrous sulphate 200 mg OD to tid as tolerated

Trace element preparation 1 capsule OD

Vitamin B complex I OD

Loperamide I prn

The following exercise will help you learn the approach to diagnosis and management of a patient presenting with jaundice. Read the section, "Hepatic Disorder" (pg. 45-49) in the reader. After you have completed the reading you may proceed to the activity.



### ACTIVITY 7.10

#### JAUNDICE- PART I (TIME: 10 MINUTES)

40-year-old Mr. Das presented to his GP with complaints of jaundice of 3 months and was referred from Calcutta after he was found to be HIV positive.

Mr. Das was transfused at the time of an acute appendicitis 5 years ago. He had been well since then. He has no history of abusing IV drugs or sexual risk factors for HIV infection. He has not received any other drugs. There is no history of low grade fever, weight loss or respiratory symptoms.

On examination:

Icterus present, loss of axillary hair, pedal oedema present. Abdomen examination- Liver 4 cm Spleen 2 cm

Free fluid - present

1. What are the inferences of your clinical examination?

- infiltrative condition -

- raised hepatic pressure - choledochal pancreatitis.

2. What lab tests would you order for Mr. Das?

LFT, chest xray, HbAg, HCAg, U/S abd.

Peripheral blood routine.



**FEEDBACK 7.10**

1. What are the inferences of your history and clinical examination?

Mr. Das has evidence of a chronic decompensated liver disease (loss of axillary hair, pedal oedema and ascites) probably with chronic active hepatitis (jaundice and hepatosplenomegaly). This may be Hepatitis B or C induced following the transfusion that he received 5 years ago.

The absence of significant drug history (anti-TB, anti-viral drugs, anti-epileptics) make drug induced hepatitis unlikely. Opportunistic infection which cause infiltration of the liver such as disseminated TB, disseminated MAI and cryptococcosis may present similarly. But the absence of systemic symptoms of fever and progressive weight loss make these unlikely.

2. What lab tests would you order for Mr. Das?

Liver function tests

HbsAG

Anti-HCV serology

HIV ELISA

Ultra-sound of the abdomen



## ACTIVITY 7.11

## JAUNDICE- PART II (TIME: 10 MINUTES)

Laboratory reports:

Liver function tests-

Total bilirubin 7 mg/dl Direct bilirubin 5 mg/dl Total protein 6.9 g/dl, Albumin 2.5 g/dl, SGOT 450 U/L, SGPT 350 U/L, Alkaline phosphatase 260 U/L

HbsAg - positive

Ant-HCV serology - positive

HIV ELISA- positive

Ultra-sound abdomen-

Hepatosplenomegaly with free fluid in the abdomen

CD4 count (Capsellia) - 150 cells/ mm<sup>3</sup>

Viral load (Amplicor assay)- 60,000 copies/ml

1. What is your interpretation of the laboratory reports?

*- AIDS / stage IV  
- Reactivation of HB & HC?  
- 'Hepatocellular damage continues' - Hepatitis*

2. What treatment options are available for Mr. Das?

Hepatitis B- *Lamivudine*

Hepatitis C- *''*

HIV- *? NRTI - > NNRTI*

**FEEDBACK 7.11**

What is your interpretation of the laboratory reports?

? Conjugated hyperbilirubinemia, increased SGOT, SGPT and alkaline phosphatase demonstrate the evidence of hepatitis. The low serum albumin and ascites on the ultrasound show evidence of hepatic decompensation. They confirm the presence of chronic active hepatitis which is due to Hepatitis B and C co-infection.

What treatment options are available for Mr. Das?

Hepatitis B-

Interferon IFN alpha 2b

Lamivudine

Hepatitis C-

Interferon IFN alpha with Ribavirin

HIV-

HAART

To avoid PI's, Nevirapine

To use Lamivudine

Possible regimen- Efavirenz, AZT and Lamivudine



## ACTIVITY 7.12

## JAUNDICE- PART III (TIME: 10 MINUTES)

Mrs. Braganza is a hairdresser from Mumbai. She had gone to Bangkok seven years ago and had been fascinated by the tattooing artistes. She got herself a tattoo and unfortunately acquired HIV via the infected needle. She has been a regular patient and is currently on HAART for the last two years. She had cryptococcal meningitis seven months ago and was treated appropriately. (4.1) Her last CD<sub>4</sub> count was 50/ml. She was noticed to have minimal jaundice in his last visit.

Her LFT is as follows:

Total bilirubin 2.6 mg%, Direct bilirubin 1.3mg%, Total protein 6.8gm%, Albumin 3.2gm%, SGOT 55 U/L, SGPT 60 U/L, Alkaline Phosphatase 650U/L

1. What are the main abnormalities on the liver tests?

1. Cholestatic picture - Alk Phos ↑, Indirect Bil ↑
- 2.
- 3.
- 4.

2. What is the interpretation of the liver abnormalities?  
What are the differential diagnosis of this abnormality?

1. ? Cryptococcal cholangitis
2. Non specific cholangitis
- 3.

3. Examine the ultra-sound Figure 7-E (page 52) and write down your findings. What is your inference?

- 1.
- 2.

4. What would be your next line of management?

- Cryptococcal - Fluconazole 400mg bid x 6wks
- Surgical

**FEEDBACK 7.12**

1. What are the main abnormalities on the liver tests?

1. Increased bilirubin
2. Markedly increased alkaline phosphatase
3. Marginally elevated transaminases

2. What is the interpretation of the liver abnormalities?  
What are the differential diagnosis of the LFT report?

The findings favour a biliary disease or an infiltrative disease of the liver. The different biliary disease presentations include acalculous cholecystitis and AIDS cholangiopathy.

3. Examine the ultra-sound X-ray 7-E (Page 52) and write down your findings. What is your inference?

The ultra-sound shows intra-hepatic biliary dilatation favouring a diagnosis of AIDS cholangiopathy.

4. What would be your next line of management?

This patient needs referral for an ERCP or CT of the abdomen.

**NOTES**



## READINGS

### DYSPHAGIA AND ODYNOPHAGIA

The oesophagus is a frequent site of infection by opportunistic infections. A large number of pathogens including fungal, bacterial, viral and even protozoal organisms can cause infectious oesophagitis. Three pathogens, *Candida*, *cytomegalovirus* and *herpes simplex virus* cause the majority of oesophageal infections. In addition, although obscure in pathogenesis, HIV associated oesophageal ulceration is an important disease condition.

The following table is a list of the important as well as less common disease conditions causing infectious oesophagitis in HIV patients.

TABLE 1: SPECTRUM OF OESOPHAGEAL INFECTIONS

	<u>COMMON</u>	<u>LESS COMMON</u>
Fungi	Candida	Aspergillus
Viral	Cytomegalovirus Herpes simplex virus	Epstein Barr virus
Bacterial		Bacterial Tuberculosis
Others	Idiopathic HIV associated ulcer	

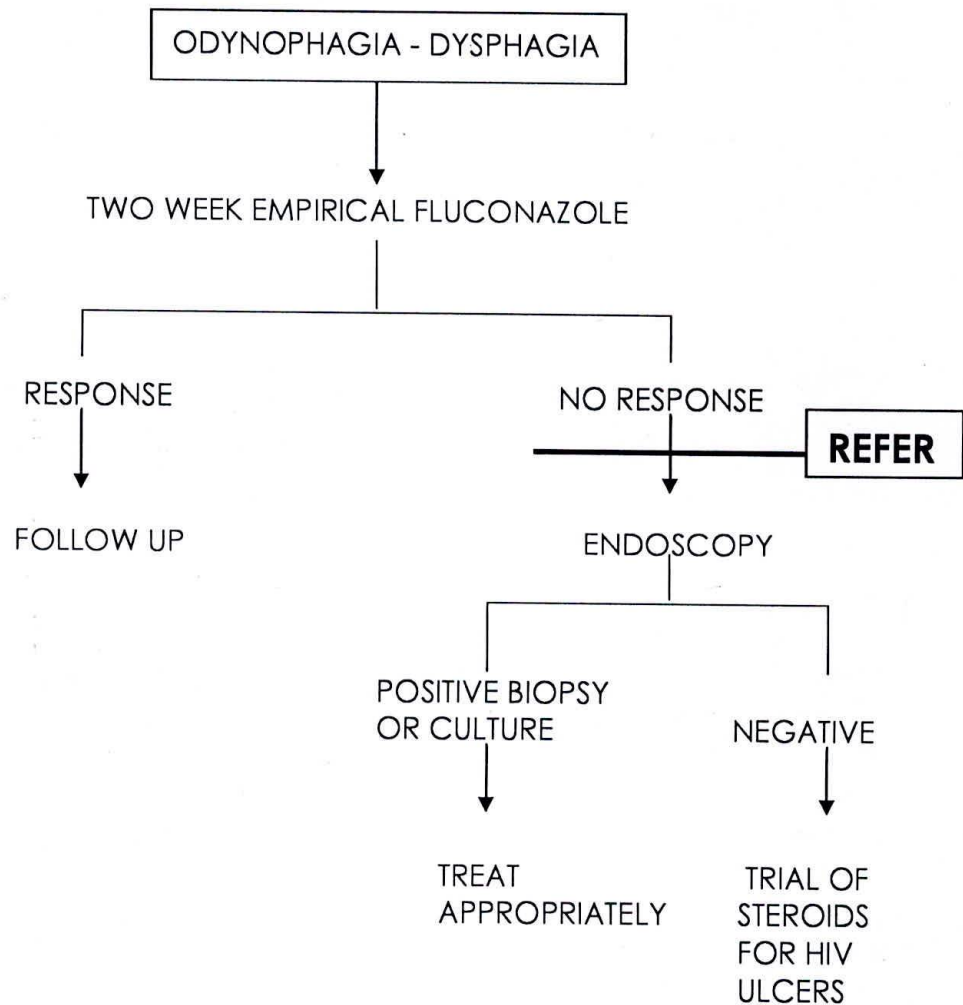
**COMMON CAUSES OF ESOPHAGITIS**

	<b>Candida Esophagitis</b>	<b>CMV Esophagitis</b>	<b>Herpes Esophagitis</b>	<b>HIV associated Esophagitis</b>
Etiology	Candida albicans	Cytomegalovirus	Herpes simplex	Idiopathic
Incidence	Commonest cause of esophagitis (64%) Develops in 20% of AIDS patients	Less frequent	Infrequent	Occurs with advanced HIV infection
Clinical features	Substernal dysphagia Odynophagia Oral thrush predictive of candida esophagitis Absence of oral thrush does not exclude diagnosis	Substernal pain Odynophagia Dysphagia less common Associated CMV retinitis, colitis may be present.	Odynophagia Dysphagia Retrosternal pain Oral herpetic ulcers may be present	Diagnosis considered after ruling out all other causes of esophagitis
Diagnosis	The diagnosis is based on clinical suspicion Gold standard- Upper GI endoscopy with histopathological examination	Upper GI endoscopy - Extensive ulcerations that are large and deep Biopsy- Cytopathic changes Characteristic inclusion bodies	Histopathological examination Initial phase- discrete ulcers that coalesce to form larger ulcers Viral cultures	Endoscopy- Moderate to large ulcers in mid and distal esophagus



**ALGORITHM FOR ODYNOPHAGIA AND DYSPHAGIA**

The first step in the management of odynophagia and dysphagia is an empirical trial with oral fluconazole. Further evaluation and treatment following non-response is as listed in the algorithm below:-



(Wilcox CM et al Gastroenterology 110: 1803-1809, 1996)

TREATMENT OF OESOPHAGITIS

DISEASE	TREATMENT
Candidal oesophagitis	T. Fluconazole 100 mg OD for 2 weeks In case of non-response increase to T. Fluconazole 400 mg OD for 2 weeks Refractory to Fluconazole consider: Syrup Itraconazole 100 mg bd for 10 days IV Amphotericin 0.3-0.5 mg/kg/day
CMV oesophagitis	Gancyclovir 5 mg/Kg OD for 3-6 weeks
Herpes oesophagitis	T. Acyclovir 400 mg tid for 5 days Or Acyclovir 5 mg/kg IV q8h for 5-10 days
Idiopathic HIV oesophagitis	T. Prednisolone 40 mg OD Taper by 10 mg/week Total course 4 weeks Treatment of HIV infection

ABDOMINAL PAIN

The importance of history taking in abdominal pain cannot be re-emphasized. It provides important clues in localizing the source of the problem. The following are patterns of abdominal pain commonly encountered in clinical practice.

EVALUATION OF ABDOMINAL PAIN IN AIDS PATIENTS

SYMPTOM COMPLEX	SUSPECT	DIAGNOSTIC TESTS
Dull pain, diarrhea, nausea and vomiting	Infectious enteritis	Stool parasites, cultures
Acute severe pain	Peritonitis	Abdominal films Surgical consultation
Right upper quadrant pain, jaundice	Cholecystitis Cholangitis	Liver function tests Ultra-sound abdomen
Epigastric pain with radiation to the back	Pancreatitis	Ultra-sound abdomen Serum amylase and lipase
Subacute pain and vomiting	Intestinal obstruction	Abdominal films Barium studies

**ABDOMINAL PAIN - DIFFERENTIAL DIAGNOSIS**

ORGAN	MANIFESTATION	DISEASE
Stomach	Gastritis, ulcer	H. pylori, CMV, lymphoma
Small Intestine	Enteritis	Enteric pathogens producing diarrhea (see pg.)
	Obstruction	TB, lymphoma
Colon	Colitis	Enteric pathogens producing colitis (see page)
Liver	Infiltration	TB, Cryptococcosis, MAC infection
Biliary tract	Cholecystitis, cholangitis	CMV, Cryptosporidium, Microsporidium
Pancreas	Pancreatitis	TB, CMV, Drugs (Didanosine, Lamivudine, Alcohol, Pentamidine)
Mesenteric nodes Peritoneum	Peritonitis, ascites Intra-abdominal lymphadenopathy	TB, Lymphoma, MAC infection

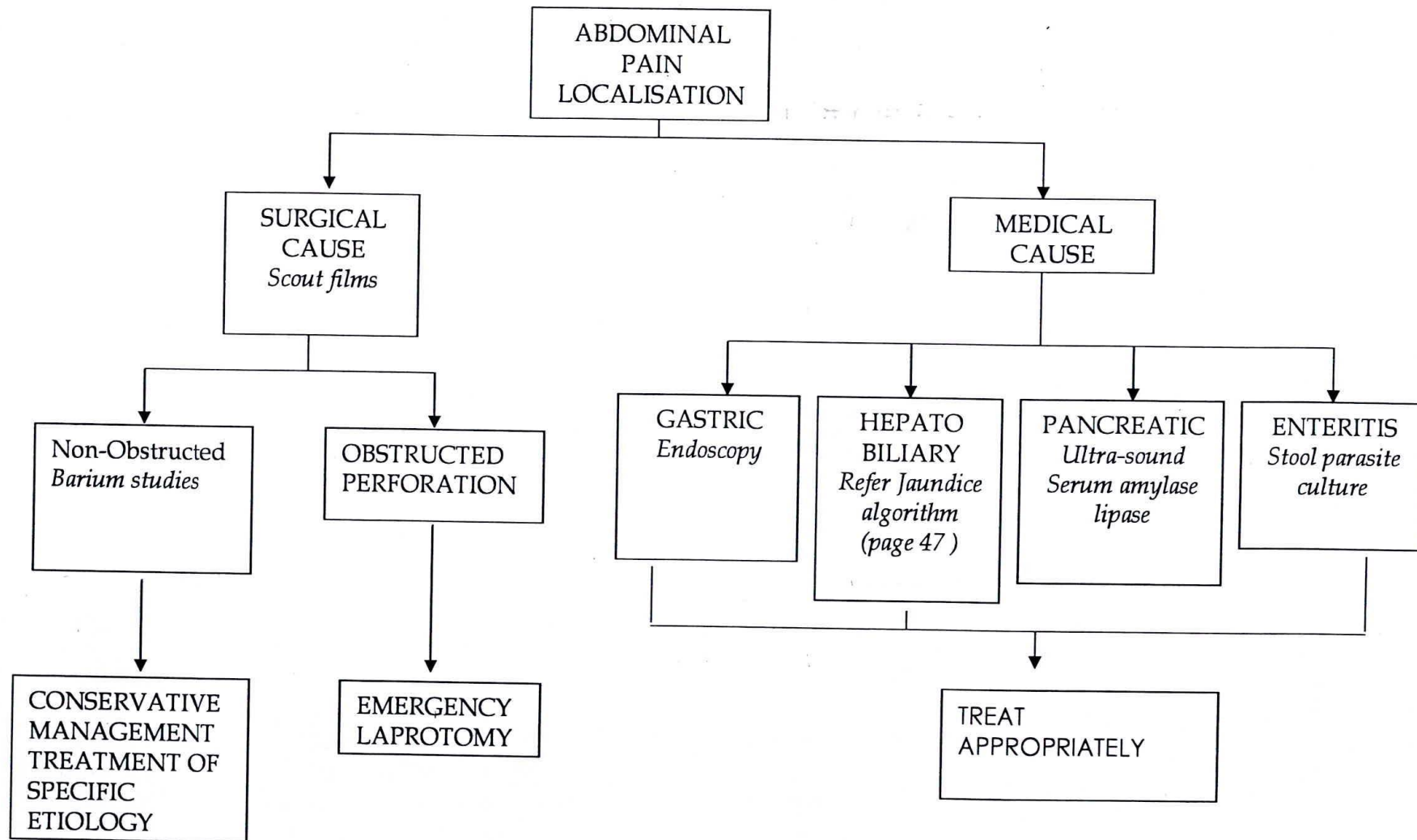
TB - Tuberculosis

CMV - Cytomegalovirus infection

MAC - Mycobacterium avium intracellulare infection

**MANAGEMENT OF ABDOMINAL PAIN**

The first decision in the evaluation of abdominal pain is to differentiate between a medical and surgical cause of abdominal pain. The indications for surgery are the same in HIV patients as that in the normal population. There is increased perioperative morbidity in AIDS patients due to their debilitation and malnutrition. All tissue specimens should be sent for viral, fungal and mycobacterial culture. The management of medical causes of abdominal pain is defined by the etiology of the abdominal pain.

ALGORITHM OF MANAGEMENT OF ABDOMINAL PAIN

### DIARRHOEA

Diarrhoea is the most frequent of gastrointestinal manifestations of AIDS, seen in up to 50-90% of patients with HIV infection. The factors that contribute to this predilection for enteric infections are: (a) immune dysfunction seen in the intestinal mucosa; (b) nutritional deficiencies; and (c) achlorhydria and hypochlorhydria.

Diarrhea in HIV infection may be due to multiple pathogens. Organisms which produce acute diarrhea in the normal population may cause chronic diarrhea in patients with HIV infection. Response to treatment may be partial and associated with high recurrence rates.

The principles of therapy are: (a) supportive therapy with fluids, electrolytes and anti-motility drugs; (b) to use specific antimicrobial therapy against the pathogen identified.

#### ACUTE DIARRHOEA

The pathogens causing acute diarrhea in HIV infection are similar to diarrheal pathogens in the normal population and include Shigella, Salmonella, E. coli, viruses, Campylobacter, G. lamblia and E. histolytica infections. Bacterial infections are the predominant cause of acute diarrhea. Bacterial gastroenteritis may be prolonged and severe in HIV patient. Therefore earlier initiation of antibiotic therapy in acute diarrhea is required in patients with HIV infection.

#### CAUSES OF CHRONIC DIARRHOEA IN HIV PATIENTS AT VELLORE

<b>Protozoa (70%)</b>	<b>Bacteria (23%)</b>	<b>Helminths (6%)</b>
Isospora belli Giardia lamblia Cryptosporidia Microspora Cyclospora Blastomycosis Balantidium coli E. histolytica D. fragilis	Shigella sp. Salmonella sp. Aeromonas	S. stercoralis

**CHRONIC DIARRHOEA IN HIV PATIENTS**

	<b>PROTOZOA (70%)</b>			<b>VIRUSES</b>		<b>BACTERIA (23%)</b>	
<b>Etiology</b>	Cryptosporidia	Isospora belli	Miscrosporidia	Cytomegalovirus	Herpes simplex	Shigella Salmonella Campylobacter	MYCOBACTERIUM AVIUM INTRACELLULARE
<b>Clinical features</b>	Profuse, non-bloody, watery diarrhea, abdominal pain, weight loss, nausea			Hemaorrhagic diarrhea, fever, weight loss, abdominal pain, haematochezia	Proctitis with anorectal pain, bleeding and mucopurulent discharge	Recurrent or chronic diarrhea, fever, abdominal pain and cramps Protracted illness	Diarrhoea Abdominal pain Malabsorption Fever, weight loss Hepatosplenomegaly
<b>Diagnosis</b>	Stool ova and cysts- Modified acid fast stain Light and electron microscopy of intestinal biopsy	Stool ova and cysts- Modified acid fast stain Large spherical oocytes	Stool ova and cysts Trichrome stain Specific fluorescent stain	Colonoscopy- Discrete haermorrhagic erosions or ulcerations Histopathology- Owl eye inclusion bodies	Stool and blood culture	Stool and blood culture	Stool examination- AFB in stools Endoscopic biopsy- AFB in macrophages No granuloma formation

### EVALUATION OF DIARRHOEA IN HIV PATIENTS

Prior to evaluation as detailed above, a careful history should be obtained. Unfortunately the history may not help in establishing a specific diagnosis as more than one organism may be the cause of diarrhoea. Abdominal cramps, bloating and nausea suggest gastric and/or small intestinal involvement seen with *Cryptosporidium*, *Isospora* or *Giardia*. Severe watery diarrhoea is characterized by *Cryptosporidium*. Hematochezia implies, colitis, seen in CMV, *Shigella* or *Campylobacter* infection. Tenesmus is seen in bacterial colitis. Anal intercourse followed by tenesmus and dyschezia suggest herpes, gonococci or Chlamydial infection leading to proctitis.

#### STAGE 1

1. Stool culture for Bacteria. E.g. *Shigella*, *Salmonella*, *Campylobacter* and *Clostridium difficile*.
2. Stool specimens examined for parasites using saline, iodine, Trichrome, and acid fast preparations for mycobacteria.

If stage one tests are negative empiric therapy may be considered. If the empiric therapy fails the patient needs to be referred for stage 2 and 3.

#### STAGE 2

1. Gastroduodenoscopic or colonoscopic inspection of tissue and biopsy.
2. Biopsy stained with Hematoxylin and Eosin for protozoa, methenamine silver or Giemsa for fungi, and AFB stains for mycobacteria.
3. Duodenal or colonoscopic biopsy culture for mycobacteria.
4. Duodenal fluid examination for parasites.

#### STAGE 3

1. Biopsy specimens examined by electron microscopy

The goal of evaluating diarrhoea is to identify a treatable cause with the minimal work up. There are inter-institutional variations of the expertise available for diagnosis and this can alter the sensitivity or pick up rate of various tests. Geographical patterns of infections also may play an important role in determining yield of pathogens.

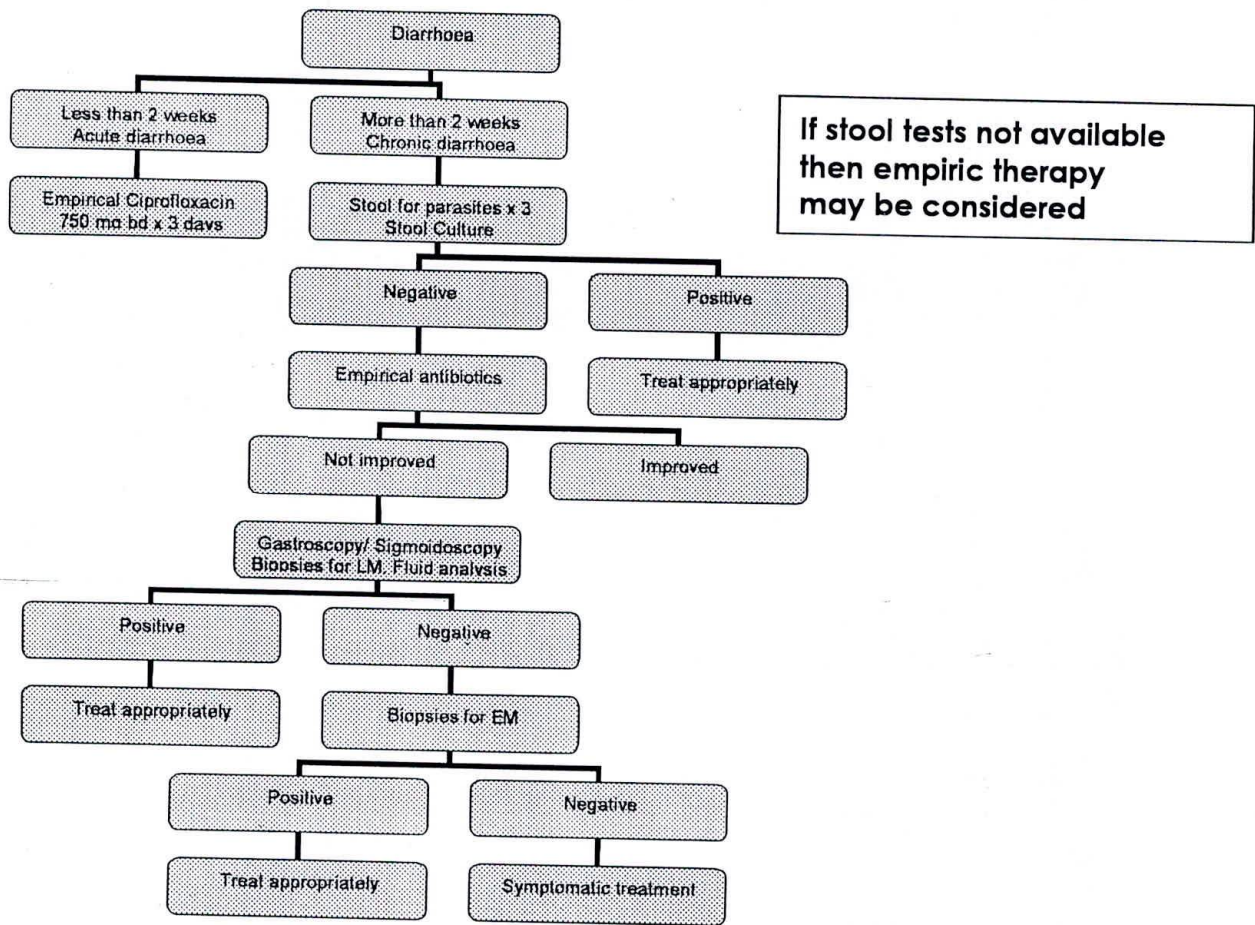


**TREATMENT OF HIV ASSOCIATED DIARRHOEA****DRUG TREATMENT**

<b>Pathogen</b>	<b>Treatment</b>
Isospora belli	TMP/SMX II BD for 2-4 weeks or TMP/SMX DS I QID for 10 days followed by TMP/SMX DS I BD for 3 weeks or Ciprofloxacin 500 mg BD for 7 days
Cryptosporidium parvum	No therapy proven efficacious Nitazonoxide 2 G/day or Octreotide
Cyclospora cyatenensis	TMP/SMX DS I QID for 10 days or TMP/SMX I BD for 3 weeks
Entamoeba histolytica	Metronidazole 750 mg tid for 10 days Tinidazole 1G q12h for 3 days
Giardia lamblia	Metronidazole 250 mg tid for 5 days
Microsporidium	Albendazole 400 mg BD for 4 weeks
Campylobacter jejuni	Ciprofloxacin 500 mg BD for 3 days Azithromycin 500 mg OD for 3 days
Salmonellosis	Ciprofloxacin 750 mg BD for 10-14 days
Shigellosis	Ciprofloxacin 500 mg BD for 3 days
CMV colitis	Gancyclovir 5 mg/Kg IV q12h for 3-6 weeks
Herpes simplex	Acyclovir 5 mg/kg IV q8h for 5 days

In countries such as ours where diagnostic facilities may not widely be available, empirical trial of therapy for patients with chronic diarrhoea may be indicated. Combinations that have been studied are: (a) Albendazole 800 mg BD for 2 weeks and (b) TMP/SMX DS II BD for 3 weeks with Ciprofloxacin 750 mg BD for 1 week. The latter regimen was studied at CMCH with partial efficacy.

**DIAGNOSTIC ALGORITHM FOR EVALUATION OF DIARRHOEA**



### HEPATIC DISORDERS

Hepatomegaly with or without jaundice and associated abnormalities of liver function tests are frequent finding in AIDS patients. These abnormalities may be due to hepatic parenchymal or biliary diseases.

#### Hepatic parenchymal diseases in AIDS

##### 1. Infections:

Viruses: Hepatitis C, CMV, Hepatitis B and D

Bacteria: Peliosis hepatis, M.Tuberculosis, MAC

Protozoa: Microsporidium

Fungi: Cryptococcus

##### 2. Drug induced hepatitis:

TMP/SMX, AZT, DDI, Azole agents, INAH, Rifampicin, Pyrazinamide, Tetracyclines, Gancyclovir, Nevirapine, Ritonavir, Saquinavir, Nelfinavir, Clarithromycin

##### 3. Tumours:

Lymphoma

#### Drugs and infiltrative diseases

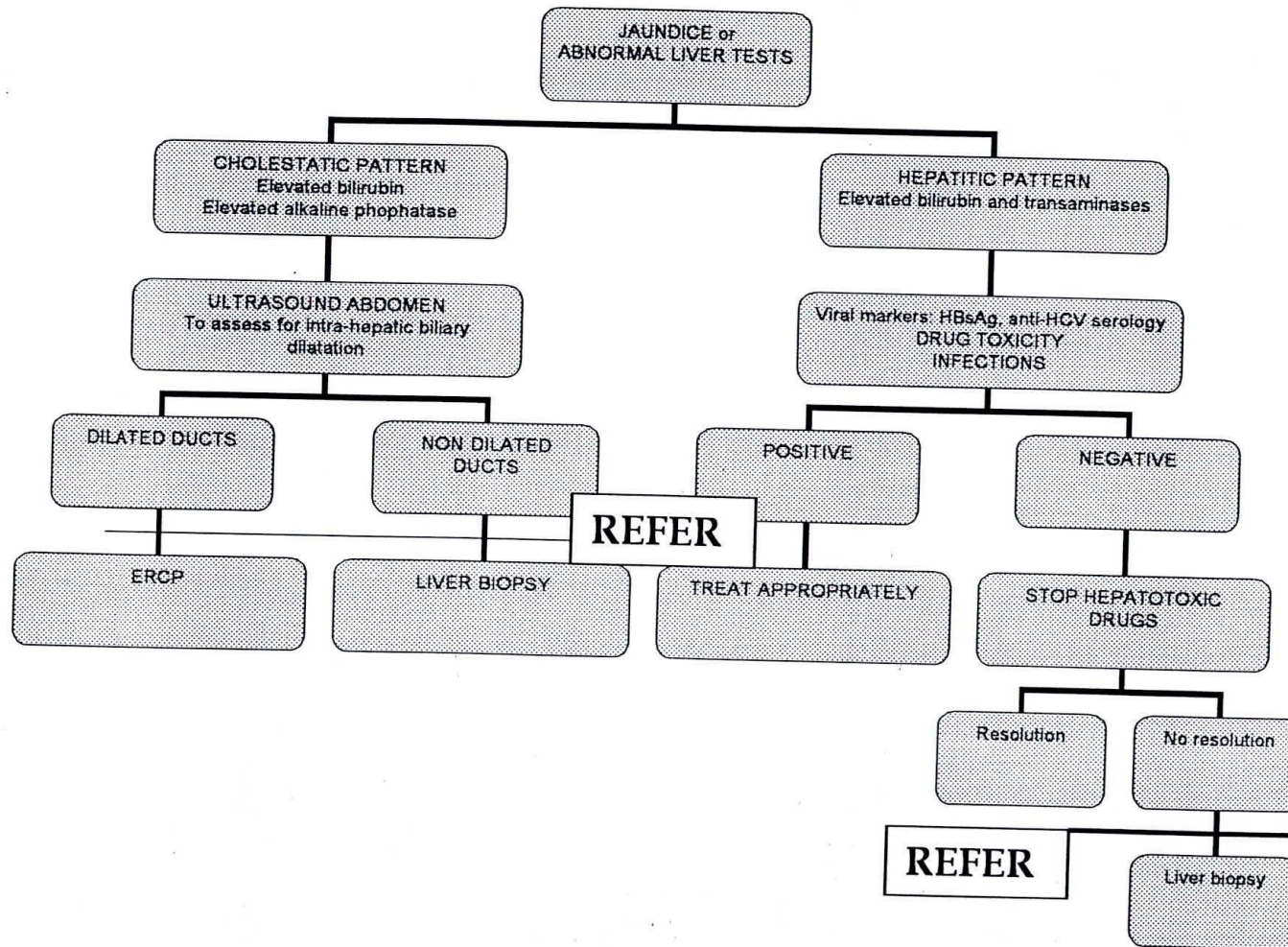
The key diseases that need to be ruled out in AIDS patients are either infective or drug related. Drug toxicity is more common in patients with AIDS and stoppage of hepatotoxic drugs should parallel the evaluation of jaundice in these patients. Diagnosis of infections requires a liver biopsy for obtaining appropriate histopathology and cultures. Treatment is tailored according to the specific opportunistic pathogen.

#### Viral Hepatitis

Hepatitis B and C co-infection are common in HIV infection as their transmission patterns are similar (Hepatitis B by parenteral and sexual route and Hepatitis C by the parenteral route). Clinical manifestations of the

hepatotropic viruses are altered with co-infection. HIV co-infection leads to reappearance of hepatitis B, increased viral replication, elevated levels of viral DNA and increased expression of core antigen in the liver. However, the host response is blunted due to the immunosuppressed state and there is very little inflammation in the liver despite high viral titres. More patients with acute hepatitis B progress on to develop chronic hepatitis (> 6 months). However clinical manifestations of chronic hepatitis B are less severe in HIV patients. On the other hand, in Hepatitis C where the virus is directly cytopathic, co-infection leads to increased viral load and greater hepatic parenchymal injury. The current recommended treatment of chronic Hepatitis B is with Interferon or Lamivudine and for Hepatitis C with Interferon with Ribavirin. These treatments are less effective in patients with HIV infection.

## ALGORITHM FOR JAUNDICE IN HIV INFECTION



### BILIARY DISORDERS

Biliary presentations of gastrointestinal disease are relatively less common. The conditions which fall within this category are the following:-

- 1) Diseases not associated with HIV infection such as gall stone disease and biliary stricture
- 2) Acalculous cholecystitis
- 3) "AIDS cholangiopathy"

### ACALCULOUS CHOLECYSTITIS

Most of the cases of acalculous cholecystitis are isolated reports. The causes of acalculous cholecystitis are: Cryptosporidia, Candida albicans, Microsporidia, Isopora belli, Salmonella enteridis, Campylobacter and CMV infection.

The characteristics of acalculous and calculous cholecystitis are similar: right upper quadrant or epigastric pain, fever, nausea, vomiting and diarrhoea. The serum bilirubin is normal, aminotransferases are minimally elevated and alkalize phosphatase levels are markedly elevated. Ultrasound reveals a thickened gall bladder wall, gall bladder dilatation and thickened biliary ducts and pericholecystic fluid.

Surgical excision of the gall bladder is the definitive treatment of acalculous cholecystitis. Mortality in patients can increase if there is delay as gangrene and perforation can set in. Usually late AIDS patients with malnutrition develop this condition.

### AIDS CHOLANGIOPATHY

This generic term encompasses a range of biliary tract disorders that occur in patients with AIDS. Four distinct entities are as follows: papillary stenosis; sclerosing cholangitis; papillary stenosis and sclerosing cholangitis; and extrahepatic bile duct stricture. The causes of AIDS cholangiopathy are the exact same organisms, which cause acalculous cholecystitis. However in 28-50% of cases no identifiable pathogen or disease can be ascertained. The clinical symptomatology include epigastric and/or right upper quadrant pain, fever, vomiting, diarrhoea, weight loss and pruritus are the common

clinical symptoms. The diagnosis is made by CT scan and ERCP. The treatment of the underlying opportunistic infection forms the basis of management of AIDS cholangiopathy. Endoscopic sphincterotomy and ursodeoxycholic acid have been shown to partially ameliorates symptoms.

**REFERENCES**

1. Dieterich D.T, Poles M.A, Cappell M.S, Lew E.A. (1999) Gastrointestinal manifestations of HIV disease including peritoneum and mesentery. *In* Merigan T.C, Bartlett J.G, Bolognesi D (eds) *Textbook of AIDS Medicine*. Williams and Wilkins.
2. Main J, McNair A, Goldin R, Thomas H.C. (1999). Liver Disease and AIDS *In* Merigan T.C, Bartlett J.G, Bolognesi D (eds) *Textbook of AIDS Medicine*. Williams and Wilkins.
3. Wilcox C.M, Friedman S.L. (1998) Gastrointestinal Manifestations of Acquired Immunodeficiency Syndrome. *In* Feldman M, Scharschmidt B.F, Sleisenger M.H (eds) *Gastrointestinal and Liver Disease*. Saunders.
4. Smith P.D, Wilcox C.M. (1999) Gastrointestinal Complications of the Acquired Immunodeficiency Syndrome. *In* Yamada T, Alpers D.H, Laine L, Owyang C, Powell D.W. (eds) *Textbook of Gastroenterology*. Lippincott Williams and Wilkins.

**FURTHER READING**

1. Mukhopadhyaya A, Ramakrishna BS, Kang G, Pulimood AB, Mathan MM, Zachariah A, Mathai DC. (1999) Enteric pathogens in southern Indian HIV-infected patients with & without diarrhoea. *Indian J Med Res* 109:85-9.
2. Kelly P, Lungu F, Keane E, Baggaley R, Kazembe F, Pabee J, Farthing M. (1996) Albendazole chemotherapy for treatment of diarrhoea in patients with AIDS in Zambia: a randomised double blind controlled trial. *BMJ* 312(7040):1187-91





Fig. 7-A



Fig. 7-B

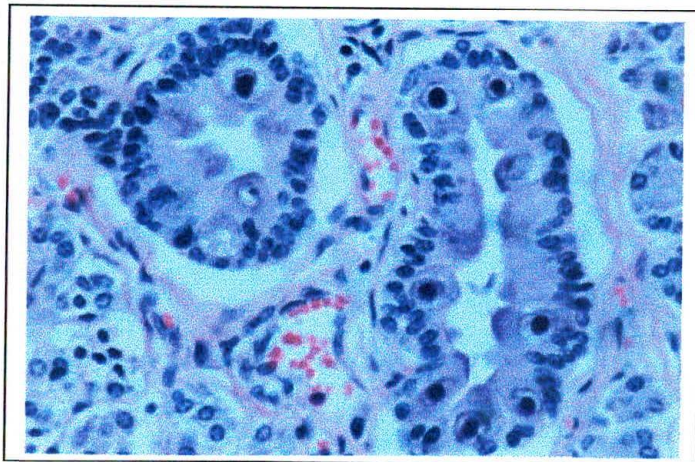


Fig. 7-C

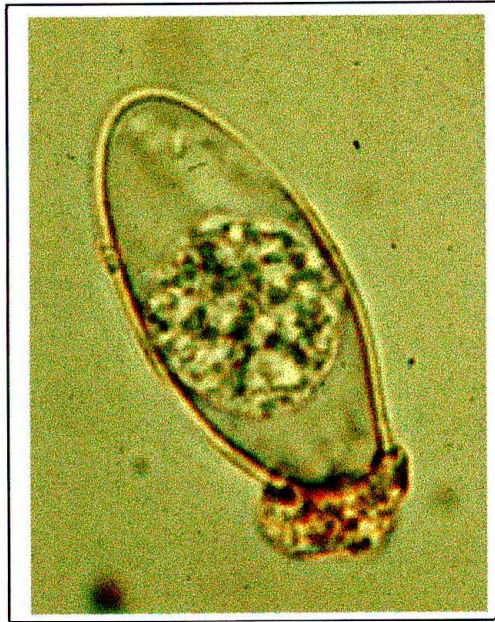


Fig. 7-D

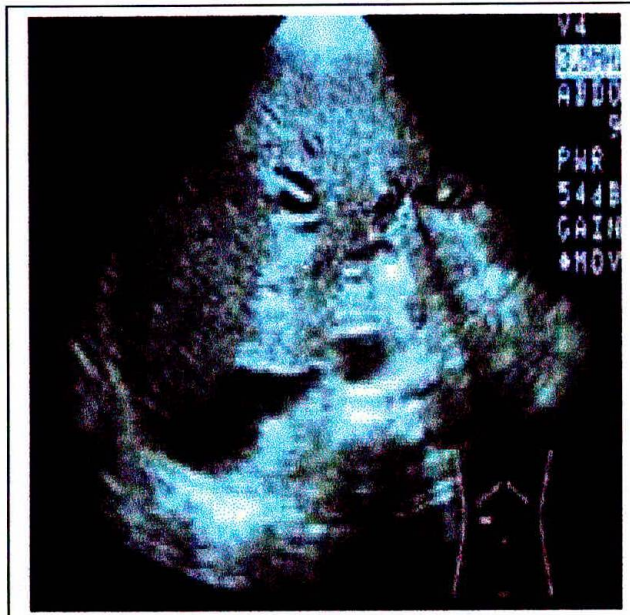


Fig. 7-E

MARY WALTERS

HIV Physician Training Course 2002,  
Christian Medical College, Vellore

DISTANCE LEARNING COURSE

STIs

RTIs

&

HIV

*Author : Susanne Abraham*

Course Organiser : Anand Zachariah

Distance Learning Expert: Janet Grant, Open University, UK

*This course is supported by the European Commission through grant number IND/B76211/1B/1999/0379 provided to the HIV/STI Prevention and Care Research Programme of the Population Council India.*

**MODULE 8**

## OVERVIEW

Sexually transmitted infections (STIs) remain a major public health problem in most parts of the world. Failure to diagnose and treat STIs in early stages may result in serious complications including infertility, foetal wastage, ectopic pregnancy, anogenital cancer, premature death, neonatal and infant infections.

The HIV epidemic has focused greater attention on the control of STI as it is known that STIs enhance the sexual transmission of HIV infection. Both ulcerative and non-ulcerative STIs have been shown to increase the risk of acquiring HIV infection. STIs also increase the infectiousness of HIV-positive persons. Patients with HIV infection have increased morbidity associated with STIs and these STIs may be more difficult to treat.

Effective management of STIs is one of the corner stones of STI control as it prevents development of complications and offers opportunity for targeted education regarding HIV prevention. Therefore appropriate and effective treatment at the first encounter of a patient with STIs is an important public health measure.

STI case management includes correct anti-microbial therapy to reduce infectivity and obtain cure and comprehensive care of the patient's needs for reproductive health. Etiological diagnosis of STIs is problematic in many settings. So syndrome based STI management has been developed and advocated in large number of countries in the developing world.

National demographic characteristics in India reflect an environment that is vulnerable to transmission of sexually transmitted infections (STI): a young population; more men than women, rapid rate of urbanization and severe socio-economic inequality.

STIs are highly prevalent in some areas, but rare in others and differ widely between groups of differing risk of acquiring STIs. In high risk groups overall STI prevalence may be very high, eg. 40% in sex workers and 20 % in truck drivers.

Studies of HIV sero-prevalence have shown increasing rates among sex workers, STI clinic attenders, IV drugs users and in the ante-natal clinic in different parts of India. It is estimated that there are about 4 million HIV positive persons in India. The predominant route of spread is heterosexual transmission. The risk factors for acquiring HIV infection are multiple sexual partners, sex with commercial sex workers, unprotected sexual intercourse, ulcerative and non-ulcerative genital disease, current or past history of STIs, intravenous drug use and lack of circumcision.

This module would help you to improve your skills in management of STI and develop your STI clinical services. Individual risk assessment of acquiring STI and STI oriented examination technique will be dealt with in contact course II.

Amplify  
or HIV

? HIV

<b>OBJECTIVES</b>
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After completion of this module the student should be able to:

1. Recognise the magnitude of STI problem in India.
2. Recognize the role of STIs in the transmission of HIV infection.
3. Understand the principles of syndromic case management.
4. Recognize different STI syndromes and use flow charts in their management.
5. Describe the importance of treatment of partners, risk behaviour reduction and condom promotion in STI management.

<b>CONTENTS</b>
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	<b>Title</b>	<b>Time (minutes)</b>	<b>Page</b>
<b>Activity 8.1</b>	<b>Approach to STI</b>	<b>10</b>	<b>4</b>
Reading	<i>STI agents and symptoms</i>	10	41
	<i>STI &amp; RTI Case management</i>	15	37-40
<b>Activity 8.2</b>	<b>HIV/STI Co-factor effect</b>	<b>10</b>	<b>6</b>
Reading	<i>Public Health Importance of STI and HIV/STI co-factor effect</i>	15	42-43
<b>Activity 8.3</b>	<b>Genital Ulcer-Part I</b>	<b>5</b>	<b>8</b>
Reading	<i>Genital Ulcer Syndrome</i>	10	44-45
	<i>Quality STD care training</i>	10	50
<b>Activity 8.4</b>	<b>Genital Ulcer- Part II</b>	<b>10</b>	<b>10</b>
<b>Activity 8.5</b>	<b>Condom promotion</b>	<b>10</b>	<b>12</b>
Reading	<i>Quality STD care training</i>	20	61-68
<b>Activity 8.6</b>	<b>Behaviour Communication</b>	<b>10</b>	<b>14</b>
Reading	<i>Quality STD care training</i>	20	69-75
<b>Activity 8.7</b>	<b>Inguinal Bubo</b>	<b>10</b>	<b>16</b>
Reading	<i>Quality STD care training</i>	10	53
	<i>Inguinal Bubo</i>	5	46
<b>Activity 8.8</b>	<b>Vaginal discharge</b>	<b>10</b>	<b>18</b>
Reading	<i>Quality STD care training</i>	15	52
	<i>Vaginal discharge</i>	10	46
<b>Activity 8.9</b>	<b>Urethral discharge</b>	<b>10</b>	<b>20</b>
Reading	<i>Quality STD care training</i>	10	51
	<i>Urethral discharge</i>	10	48
<b>Activity 8.10</b>	<b>Scrotal swelling</b>	<b>10</b>	<b>22</b>
Reading	<i>Quality STD care training</i>	10	55
	<i>Scrotal swelling</i>	10	49
<b>Activity 8.11</b>	<b>Lower abdominal pain</b>	<b>10</b>	<b>24</b>
Reading	<i>Quality STD care training</i>	10	54
	<i>Lower abdominal pain</i>	10	50
<b>Activity 8.12</b>	<b>Secondary syphilis</b>	<b>10</b>	<b>26</b>
Reading	<i>Secondary syphilis</i>	10	51
<b>Activity 8.13</b>	<b>Positive VDRL</b>	<b>5</b>	<b>28</b>
Reading	<i>Tests &amp; treatment of syphilis</i>	10	51-52
<b>Activity 8.14</b>	<b>Other STIs</b>	<b>5</b>	<b>30</b>
Reading	<i>Other STIs</i>	5	53
<b>Activity 8.15</b>	<b>Other STIs -treatment</b>	<b>5</b>	<b>32</b>
<b>TMA</b>		<b>40</b>	<b>35-36</b>
<b>Total estimated study time</b>		<b>395</b>	

The following activity will enable you to learn the different approaches to STI management and the causative agents of different STIs syndromes. Read, "STI and RTI Case management" (pg.37-40) and "STI etiological agents and symptoms" (pg.41) in the reader. After reading this you can undertake the following activity.



### ACTIVITY 8.1

#### APPROACH TO STI (TIME: 10 MIN.)

1. List the advantages and disadvantages of syndromic approach to management of STIs.

ADVANTAGES	DISADVANTAGES
<ol style="list-style-type: none"> <li>1. - Treatment is over</li> <li>2. - Can be decentralized to less trained staff</li> <li>3. - Greater results possible</li> <li>4. - A must for reducing AIDS epidemic</li> <li>5.</li> </ol>	<ol style="list-style-type: none"> <li>1. - Needs to be based on local pathogens &amp; sensitivities</li> <li>2. - hence reference lab</li> <li>3. - Trends to overtreat</li> <li>4. - Over treatment of two partners</li> <li>5.</li> </ol>

2. What are the common causative agents of the different STIs syndromes?

STIs SYNDROME	DISEASES
1. Genital ulcer - Syphilis,	Herpes simplex, chancroid
2. Urethral discharge - Gonorrhoea, chlamydia	
3. Inguinal bubo - <del>G. G. I</del> , Syphilitic bubo, ? L. G. V, chancroid	
4. Vaginal discharge - Bacteriosis, Trichomonas, candida ? Gonorrhoea & chlamydia	
5. Scrotal swelling - Gonorrhoea, chlamydia	
6. Lower abdominal pain - P.I.D	



### FEEDBACK 8.1

1. List the advantages and disadvantages of syndromic approach to management of STIs.

ADVANTAGES	DISADVANTAGES
<ol style="list-style-type: none"> <li>1. Treatment at the first visit.</li> <li>2. Cost saving.</li> <li>3. No loss to follow up.</li> <li>4. Effective in mixed infection.</li> <li>5. Applicable in settings without laboratory facilities.</li> <li>6. Reduces risk of STI and HIV transmission.</li> <li>7. Can be applied by paramedical workers.</li> </ol>	<ol style="list-style-type: none"> <li>1. Over-treatment</li> <li>2. False positive diagnosis.</li> <li>3. Social problems related to STI misdiagnosis.</li> <li>4. Over-treatment of sex partner based on syndromic diagnosis.</li> </ol>

2. What are the common diseases which produce the different STIs syndromes?

STI SYNDROME	DISEASES
1. Genital ulcer	Syphilis, Chancroid, Genital herpes, Donovanosis (Granuloma inguinale), Lymphgranuloma venereum
2. Urethral discharge	Gonococcal and non-gonococcal urethritis
3. Inguinal bubo	Lymphgranuloma venereum, Chancroid
4. Vaginal discharge Cervical discharge appearing as vaginal discharge	Vulvovaginal candidiasis, Trichomoniasis, Bacterial vaginosis Gonococcal and Chlamydial cervicitis
5. Scrotal swelling	Lymphgranuloma venereum and Gonorrhoea
6. Lower abdominal pain	Pelvic inflammatory disease caused by gonorrhoe or chlamydia.



This activity aims to help you learn about the STI/HIV co-factor effect. Please read, "Public health importance of STIs and STI/HIV co-factor effect" (pg.42-43) of the reader. After completing the reading you can proceed to the next activity.



## ACTIVITY 8.2

### HIV/STI CO-FACTOR EFFECT (TIME: 10 MIN.)

1. Which are the STIs that increase the susceptibility of HIV-negative individuals to HIV infection? What are the mechanisms underlying the increased HIV susceptibility?

Ulcerative lesions & non-ulcerative inflammatory lesions  
 - Increased transport of virus through macrophage  
 and ↑ infectivity of HIV positive STD.

2. Why do STIs increase the infectiousness of HIV positive individuals?

↑ viruses around the lesions

3. In which stage of an HIV epidemic is the control of STIs an effective strategy for reducing HIV transmission?

Early stage where it is driven by high risk groups - cutting down STIs will reduce transmission

4. What are important strategies for reducing STIs in the community?

Condoms / Syndromic mgmt / Single partner



## FEEDBACK 8.2

1. Which are the STIs that increase the susceptibility of HIV-negative individuals to HIV infection? What are the mechanisms underlying the increased HIV susceptibility?

Genital ulcers caused by syphilis, chancroid and genital herpes and gonorrhoea, chlamydia and trichomoniasis increase susceptibility to HIV infection. The basis of the increased susceptibility is:

- a. STIs interrupt the integrity of the genital epithelium
- b. STIs attract inflammatory cells which increase access of HIV virus to CD4 receptors.

2. Why do STIs increase the infectiousness of HIV positive individuals?

The concentration of HIV virus in semen is increased in patients with urethritis. HIV virus can be detected in genital ulcers. HIV shedding is increased in HIV infected women with gonococcal cervicitis. STIs are hypothesised to upregulate the viral load which increases concentration of virus in genital fluids.

3. In which stage of an HIV epidemic is the control of STIs an effective strategy for reducing HIV transmission?

STIs treatment is an effective strategy for HIV control in the early and moderately advanced stages of an HIV epidemic when the prevalence is rising. Once the epidemic becomes generalised then the contribution of STIs to the epidemic seems to steadily decrease.

4. What are important strategies for reducing STIs in the community?

Training of GPs in syndromic case management of STIs.

Training of health workers to screen and detect STIs in the community.

Education of the community regarding symptoms of STIs and to improve health seeking behavior regarding STIs.

Sexual behaviour change and condom promotion.

This activity aims to help you learn about the management of a patient with genital ulcer. Please read, "Genital Ulcer Syndrome" (pg.44-45) of the reader and Quality STD Care Training Module page 50 Genital Ulcer - treatment flow chart. After completing the reading you can proceed to the next activity.



### ACTIVITY 8.3

#### GENITAL ULCER – PART I (TIME: 5 MIN.)

30 year old Mr. Venkatappa, comes with complaints of of painless ulcer of the penis of 2 weeks duration. On examination there were three mild tender 0.5 cm sized non-indurated ulcers on the coronal sulcus. There was no past history of genital ulcer.

1. List the STIs which can cause genital ulcers.

1. Syphilis	3. LGV
2. Herpes	4. LGI
5. Chancroid	

2. How would you manage this patient?

- Edu for risk reduction, partner notification  
- ? Pen. shot + Doxy + cipro

3. What serological tests to screen for sexually transmitted diseases would you like to do on this patient?

VDRL Titri

4. How would you follow-up this patient?

What would you do if the patient failed to respond to treatment?

How should the patient's partner be screened?



### FEEDBACK 8.3

1. List the STIs which can cause genital ulcers in this patient:

1. Herpes Simplex
2. Syphilis
3. Chancroid
4. Donovanosis
5. LGV

2. How would you manage this patient?

Inj. Benzathine Penicillin 24 million units in 2 injections (give one injection in each buttock).  
 + Erythromycin 500 mg QID for 7 days  
 or Ciprofloxacin 500 mg as a single dose.

3. What serological tests to screen for sexually transmitted diseases would you like to do on this patient?

VDRL/RPR, HIV- ELISA, HBsAg after pre-test counselling (as in all cases of patients with STIs).

4. How would you follow-up this patient? What would you do if the patient failed to respond to treatment?  
 What should the patient's partner be screened for?

Review after 7 days. If the ulcer has not healed then you should assess compliance with medications. If the patient has been compliant then he should be referred to a higher centre for further evaluation.

Follow up RPR/VDRL results and other serological tests HIV ELISA and HBsAg.

The partner should be screened according to syndromic case management.



## ACTIVITY 8.4

## GENITAL ULCER - PART II (TIME: 10 MIN.)

Twenty-five year old Mr. Kuppan comes to your clinic. He has been working as a lorry driver for the last 4 years. During his trips he has been in the habit of visiting commercial sex workers and does not use condoms. He complains of a sore on the penis since 9 days. On questioning, he gives a history of recurrent episodes of painful lesions starting as tiny vesicles on the prepuce and glans penis, which then ulcerate. The lesions heal in 4 to 5 days. These lesions occur at a frequency of about once a month. Examine Fig. 8-A (in cover)

1. What is the most likely diagnosis in this patient?

*Herpetic lesions*

2. How would you manage this patient?

*Local antibiotic cream.  
Acyclovir 400mg tid x 7-10 days.*

3. What are the other components of treatment that we should institute in this case?

*Education - risk reduction & partner notification*

4. What treatment can be given to prevent recurrence of the disease?

*Acyclovir 400mg bid*

5. The patient's HIV ELISA test was positive. What do you think are factors that led to this patient acquiring HIV infection?

*High risk behavior, Ulcerative STD.*



## FEEDBACK 8.4

1. What is the most likely diagnosis in this patient?

- Herpes Genitalis

Genital HSV infection is mostly diagnosed on clinical grounds.

2. How would you manage this patient?

Herpetic genital ulcers are usually self-limiting. They require symptomatic treatment with dilute potassium permanganate soaks. However since this patient's lesions are lasting for a longer duration, it would be advisable to start on Acyclovir 400 mg tid for 5-7 days.

3. What are the other components of treatment that we should institute in this case?

**Risk reduction counseling**

**Condom promotion**

4. What treatment can be prevent recurrence of the disease?

Suppressive treatment with Tab. Acyclovir 400 mg twice a day is indicated as the patient has > 6 episodes per year.

5. The patient's HIV ELISA test was positive. What do you think are factors that led to this patient acquiring HIV infection?

The patient has history of unprotected sex, sexual partners were CSW's and has had multiple partners all of which will increase the risk of acquiring HIV infection. Any genital ulcerative disease will increase the risk of acquiring HIV. The risk of acquiring HIV infection is in the order of 20-100 fold per sexual act. The patient was not circumcised. The absence of circumcision also increases the risk of acquiring HIV infection.

This activity will help you learn about how to educate patients regarding condom use. Read **Quality STD Care Module, page 61-68 Session 6 Condom Promotion** and then proceed to the activity.



### ACTIVITY 8.5

### CONDOM PROMOTION (TIME: 10 MIN.)

1. What are common misconceptions regarding condom use?

- Pleasure ↓  
-

2. What are common errors in the use of condoms?

- slipping  
- Touching wrong surface  
- reuse.

3. In what ways can a doctor promote condoms in his/her clinic?

4. How will a person know if a condom is of good quality?

- Seal not broken  
- soft

**FEEDBACK 8.5**

1. What are common misconceptions regarding condom use?

Condoms reduce sexual pleasure

Women do not like using condoms

Condoms tear during sexual intercourse

2. What are common errors in the use of condoms?

1. Condom packet opened and applied before penis is erect.
2. Condom unrolled before application.
3. Tip of condom not squeezed while applying it.
4. Penis not withdrawn immediately after ejaculation.
5. Reservoir tip not facing downward while slipping off condom.
6. Condom not disposed properly.

3. In what ways can a doctor promote condoms in their clinic?

Distribute condoms.

Display condom information in the clinic.

Explain and demonstrate condom use.

4. How will a person know if a condom is of good quality?

Check expiry date on packet.

If condom dried or sticky or changed in colour or uneven in texture, it may be damaged.





This activity will help you learn about counselling and behaviour change communication in the context of STIs management. Read **Quality STD Care Module, page 69-75 Session 7 "Counselling and Behavioural Change Communication"** and then proceed to the activity.



### ACTIVITY 8.6

## BEHAVIOR CHANGE COMMUNICATION

(TIME: 10 MIN.)

1. What are important tips in interviewing technique to facilitate behaviour change?

Focus on patient's need - understand  
 Provide info on consequences.  
 - Provide alternative behavior options

2. Who are the patients who need special attention to initiate behaviour change communication?

- High risk behavior group  
 - Adolescents  
 - CSW

3. What are the most important messages to convey in behaviour change communication?

Single partner, protected sex every time  
 Seek medical care at the earliest for STI

**FEEDBACK 8.6**

1. What are important tips in interviewing technique to facilitate behaviour change?

A listening and understanding approach may build the confidence of the patient.

Give appropriate information.

Clarify doubts.

Encourage and support patients to change risky behaviour.

2. Who are the patients who need special attention to initiate behaviour change communication?

Patients who come for treatment for STIs the first time.

Patients who come for treatment for STIs the second time.

Persons whose lifestyle and job may promote risky behaviour.

Women whose husbands may have risky behaviour.

3. What are the most important messages to convey in behaviour change communication?

Basic information on STDs and HIV infection.

Emphasise need for compliance to drug regimen.

Importance of safe sexual behaviour:

Abstaining from sex till cure, not having penetrating sex, using condom correctly and consistently.

Need for concurrent partner treatment.

Emphasise the need for follow-up.

This activity aims to help you learn about the management of a patient with inguinal bubo. Please read, "Inguinal Bubo" (pg.46) of the reader and Quality STD Care Training Module page 53 Inguinal Bubo - treatment flow chart. After completing the reading you can proceed to the next activity.



### ACTIVITY 8.7

#### INGUINAL BUBO (TIME: 10 MIN.)

Thirty-five year old Mr. Ramachandran, a labourer, comes with history of swelling in the left inguinal area of 20 days' duration. He has been diagnosed to have HIV infection since 2 years.

On examination, there were no lesions on the external genitalia. However, there was left-sided inguinal swelling with overlying erythema.

Examine Fig. 8-B (in cover).

1. List the sexually transmitted diseases which can present as inguinal lymph node swelling.

- |             |    |
|-------------|----|
| 1. Chancres | 3. |
| 2. L.C.V    | 4. |

2. What is the clinical sign in the picture?

3. How would you manage this patient?

**FEEDBACK 8.7**

1. List the sexually transmitted diseases which can present as inguinal LN swelling.

- |                             |                  |
|-----------------------------|------------------|
| 1. Lymphogranuloma venereum | 3. Syphilis      |
| 2. Chancroid                | 4. HIV infection |

2. What is the clinical sign in the picture?

Groove sign. This sign is seen only in about 20% of cases with LGV.

3. How would you manage this patient?

Doxycycline 100 mg twice a day for 14 days.

Bubo may have to be aspirated. Incision and drainage is not recommended.

Since the patient is HIV positive, the treatment may have to be prolonged.

This activity aims to help you learn about the management of a patient with vaginal discharge. Please read, "Vaginal Discharge" (pg.46-47) of the reader and Quality STD Care Training Module, page 52 Vaginal Discharge - treatment flow chart. After completing the reading you can proceed to the next activity.



### ACTIVITY 8.8

## APPROACH TO VAGINAL DISCHARGE

(TIME: 10 MIN.)

Twenty-five year old Mrs. Vijaya, who is a housewife from a nearby town, has history of vaginal discharge since 4 weeks. On speculum examination there was profuse yellowish vaginal discharge. The risk assessment was negative.

1. List the RTI's that can cause vaginal discharge in this patient.

Trichomonas V, Candidiasis, Chlamydiae T, Gonorrhoea  
Bacterial vaginosis

2. What laboratory tests could be done on the vaginal discharge?

Wet prep, KOH & Gram stain

3. How would you manage this patient?

- Metronidazole 400mg tid x 7-10 (no risk hist)  
- Edox - against risk, Partner Dx

4. What serological test to screen for STIs would you do in this patient?

VDRL ? HIV Elisa & Hb Ag



## FEEDBACK 8.8

1. List the RTI's that can cause vaginal discharge in this patient.

1. Trichomoniasis
2. Vulvo vaginal candidiasis
3. Bacterial vaginosis

One should keep in mind that infections that cause such as gonorrhoea and Chlamydia infection, whilst usually asymptomatic, may cause cervical discharge which presents as vaginal discharge

2. What laboratory tests could be done at the time of the vaginal examination?

KOH, saline mount, Gram staining

3. How would you manage this patient?

T. Metronidazole 400 mg TID for 7 days

Examine partner and institute treatment with Metronidazole.

4. What serological test to screen for STIs would you do in this patient?

VDRL/RPR, HIV ELISA, HBsAg



This activity aims to help you learn about the management of a patient with urethral discharge. Please read, "Urethral discharge" (pg.48) of the reader and Quality STD Care Training Module page 51 Urethral Discharge - treatment flow chart. After completing the reading you can proceed to the next activity.



### ACTIVITY 8.9

### URETHRAL DISCHARGE (TIME: 10 MIN.)

Twenty-nine year old Sadasivam is unmarried, and comes with history of discharge per urethra since 2 days. On questioning, he admits to having had multiple partners in the past 3 years. On examination discharge per urethra was confirmed.

1. Please list the likely pathogens that cause discharge per urethra in this patient.

*A. Gonorrhoea, Chlamydiae, Trichomonas,*

2. What test would you do for presumptive diagnosis?

*Discharge test, Gram stain*

3. The gram stain showed gram-negative intra-cellular diplococci. How would you manage the patient?

*? Cipro. 500mg stat / + 200mg 1000mg bid x 7 days*

*1) Co-wife*

4. On follow up, his HIV ELISA was positive. Do you have to change his treatment regime?

*They need No*

5. However, his symptoms of dysuria were persisting. What are the possibilities to be considered?

*1) Abstinence, Stricture, Ur. Infection/Epididymitis  
Compliance, reinfection, other organism mixed*



### FEEDBACK 8.9

1. Please list the likely pathogens that cause discharge per urethra in this patient.

Neisseria gonorrhoea  
Chlamydia trachomatis

2. What test would you do for presumptive diagnosis?

Gram stain of discharge.

3. The gram stain showed gram-negative intra-cellular diplococci. How would you manage the patient?

Cap. Doxycycline 100 mg bd x 7 days.  
T. Ciprofloxacin 500 mg stat or Inj. Ceftriaxone 250 mg IM stat.  
To abstain from sexual intercourse until 7 days after therapy is initiated.  
To come back for evaluation after 1 week.  
To bring partner for examination and treatment.  
Condom usage whenever he has sexual relationships.  
This patient is being treated for both gonorrhoea and chlamydial infection because of possibility of co-infection and difficulty of excluding diagnosis of NGU. The same treatment could be administered if gram stain was not available.

4. On follow up, his HIV ELISA was positive. Do you have to change his treatment regime?

No

5. However, his symptoms of dysuria were persisting. What are the possibilities to be considered?

Lack of treatment compliance  
Treatment failure  
Other etiologies such as Trichomoniasis  
Re-infection  
  
Re-treat with initial regime, if he did not comply with the initial treatment regime or was re-exposed to untreated sex partner(s).  
Treat with Metronidazole to treat for possible trichomonas urethritis.  
Dose- Metronidazole 400 mg TID for 7 days.



This activity aims to help you learn about the management of a patient with scrotal swelling. Please read, "Scrotal Swelling" (pg.49) of the reader and Quality STD Care Training Module page 55 Scrotal Swelling- treatment flow chart. After completing the reading you can proceed to the next activity.



### ACTIVITY 8.10

### SCROTAL SWELLING (TIME: 10 MIN.)

A 25 year old man presents to the clinic with symptoms of pain in the scrotum for 3 days. On examination he has a tender and warm unilateral scrotal swelling. The epididymes was tender and thickened. There is no inguinal adenopathy. On milking the urethra there is minimal discharge per urethra.

1. What additional history and examination will you evaluate for?

*Acute Orchitis / filarial exposure / Trauma & torsion //*

2. What is the likely diagnosis? What differential diagnosis do you need to take into account?

*N. Gonorrhoea - D/D Chlamydia, Bacterial (mixed)*

3. What treatment will you initiate?

*Ciprofloxacin 500mg x 3 days stat, Doxy. 100mg qd x 7*

*- Health Educ.*



### FEEDBACK 8.10

1. What additional history and examination will you evaluate for?

History of trauma, onset of pain (acute onset may suggest torsion), loin to groin pain (may suggest torsion testis), previous similar episodes (may suggest filarial epididymo-orchitis), recent urethral discharge and history of high risk behaviour, past history of tuberculosis.

Examination- Inspection (scrotal oedema), palpation (enlarged tender testis which is elevated and has transverse lie and pain not relieved on elevation of the scrotum is suggestive of torsion testis), epididymal thickening and warmth.

2. What is the likely diagnosis? What differential diagnosis do you need to take into account?

Epididymo-orchitis eg. due to *Chlamydia trachomatis* or *Neisseria gonorrhoea* (since the onset is not acute, there is urethral discharge and the scrotum is warmth).  
The differential diagnosis includes epididymo-orchitis of other causes, bacterial, tuberculous, and filarial, trauma, torsion testis and testicular tumour.

3. What treatment will you initiate?

Cap. Doxycycline 100 mg bd x 7 days

T. Ciprofloxacin 500 mg stat or Inj. Ceftriaxone 250 mg IM stat

Elevation of the scrotum with scrotal support.

Analgesics

To abstain from sexual intercourse until 7 days after therapy is initiated.

To come back for evaluation after 1 week.

To bring partner for examination and treatment.

Condom usage whenever he has sexual relationships

This activity aims to help you learn about the management of a lady presenting with abdominal pain. Please read, "Lower abdominal pain" (pg.50) of the reader and Quality STD Care Training Module page 54 Lower abdominal pain - treatment flow chart. After completing the reading you can proceed to the next activity.



### ACTIVITY 8.11

#### LOWER ABDOMINAL PAIN (TIME: 10 MIN.)

A 22 year old young lady presented to the outpatient clinic with complaints of lower abdominal pain and fever for 4 days. On examination: Temperature - 101° F, Iliac fossa tenderness present. Pelvic examination- cervical motion tenderness. Speculum examination- Copper-T in situ. Scanty mucopurulent discharge from the cervix. No adnexal masses felt on bimanual examination.

1. What additional history will you ask for?

- Risk assessment including partner symptoms
- Possibility of ectopic interventions - ectopic abortion etc.
- Correlation with menstrual history

2. What additional findings will you look for on abdominal examination?

Guarding & rebound tenderness - present

3. What treatment will you initiate?

Doxycycline      Cipro

**FEEDBACK 8.11**

1. What additional history will you ask for?

History of missed periods, recent delivery or abortion, menorrhagia or metrorrhagia, history of sexual behaviour of patient and partner.

2. What findings will you look for on abdominal examination?

Rebound tenderness or guarding. The presence of these would suggest a surgical cause of abdominal pain such as acute appendicitis that requires immediate referral.

3. What treatment will you initiate?

Remove copper-T after 2-4 days.  
Ciprofloxacin 500 mg stat  
Doxycycline 100 mg BD for 14 days  
Metronidazole 400 mg BD for 14 days

This activity aims to help you learn about the management of a patient with features of secondary syphilis. Please read, "Secondary Syphilis" (pg.51) of the reader. After completing the reading you can proceed to the next activity.



### ACTIVITY 8.12

#### SECONDARY SYPHILIS (TIME: 10 MIN.)

Forty-year old Mr. Dhanasekar, who is a clerk in a bank, comes with a history of a non-pruritic rash on the trunk and palms since 1 week. Examine Fig. 8-D (in cover).

1. What is the differential diagnosis of this symptom?

*Sec. syphilis, drug; viral prodrome, ? collagen.*

2. What additional history would you ask for to help you diagnose this patient's disease?

*to primary syphilis, drugs, fever & general symptoms*

3. What diagnostic test will you order?

*VDRL/RPR & Treponema tests (confirmatory)*

**FEEDBACK 8.12**

1. What is the differential diagnosis of this symptoms?

Drug rash  
Exanthematous fever  
Secondary syphilis  
Primary skin disease

2. What additional history would you ask for to help you diagnose this patient's disease?

1. Fever
2. Malaise
3. Headache
4. Drug intake prior to the onset of the rash.
5. Prior history of skin rash and genital ulcer.
6. History of mucosal lesions.
7. Assess risk of acquiring STIs.

Genital examination is also essential to look for a healing ulcer and inguinal adenopathy.

3. What diagnostic test will you order?

VDRL or RPR

This activity aims to help you learn about the management of a patient with a positive VDRL report. Please read, "Serological tests for syphilis and Treatment of Syphilis" (pg.51-52) of the reader. After completing the reading you can proceed to the next activity.



### ACTIVITY 8.13

#### POSITIVE VDRL TEST (TIME: 5 MIN.)

In the past history of Mr. Dhanasekar (the patient in Activity 8.12), there was a positive history of a single painless genital ulcer 6 months ago which healed with topical ointments. His VDRL was reactive in 1:64 dilutions, and his HIV ELISA was positive.

1. What is your diagnosis?

Secondary Syphilis - ? special consideration for HIV status.

2. What treatment would you institute in this case? Does it differ since patient is HIV positive?

2.4 million units Benzathine penicillin

3. When should the serological test be repeated and how would you follow up the patient?

3; 6, 9, 12 upto 2 years.

**FEEDBACK 8.13**

1. What is your diagnosis?

Secondary syphilis.

2. What treatment would you institute in this case?  
Does it differ since patient is HIV positive?

Benzathine penicillin G 2.4 million units IM in a single dose

The treatment does not differ and is the same in HIV infected individuals. However patient needs to be on close follow up.

3. When should the serological test be repeated and how would you follow up the patient?

Patient should be re examined clinically and serologically 3, 6, 9 and 12 months following treatment





This activity aims to help you learn about the clinical recognition of patients with other STIs. Please read, "Other STIs" (pg.53) of the reader. After completing the reading you can proceed to the next activity.



### ACTIVITY 8.14

#### OTHER STI (TIME: 5 MIN.)

Study the photographs 8-E and 8-F in attached cover.

No	Photo	Diagnosis
1	Fig. 8- E	
2	Fig. 8-F	

**FEEDBACK 8.14**

N o	Photo	Diagnosis
1	Fig. 8-E	Genital wart
2	Fig. 8-F	Molluscum contagiosum



*This activity aims to help you learn about the treatment of a patients with other STIs.*

**ACTIVITY 8.15****OTHER STI – TREATMENT (TIME: 5 MIN.)**

How would you treat the respective patients in photographs Fig. 8-E and 8-F?

N o	Diagnosis	Treatment
1		
2		



## FEEDBACK 8.15

N o	Diagnosis	Treatment
1	Genital warts	Treatment options: 1. Podophyllin applications 2. Cryotherapy 3. Cautery 4. Excision
2	Molluscum contagiosum	Treatment options: 1. Needling 2. Cryotherapy 3. TCA/MCA application 4. Cautery



**NOTES**



## READINGS

### STI and RTI Case Management

(Excerpt from: The management and control of sexually transmitted infection, and their implications for AIDS control in South-east Asia Heiner Grosskurth, Gurumurthy Rangaiyan to be published in Journal of Health Management)

Appropriate case management for STIs and RTIs comprise the following steps: a brief history, physical examination, correct diagnosis, early and effective treatment, health education to achieve good treatment compliance and sustainable risk reduction, and effective partner notification (Adler et al 1998, WHO 2001).

### Approaches to STI/RTI diagnosis

There are three distinct approaches to arrive at an STI diagnosis: clinical, laboratory based and syndromic diagnosis.

(i) The clinical approach attempts to arrive at a specific diagnosis based on clinical examination, and to treat the assumed aetiology. This traditional approach has been widely used by care providers without access to laboratory services. However, many studies have demonstrated that the sensitivity and specificity of this strategy is low even in the hands of experienced providers (Holmes and Ryan 1999). For example in a study from South Africa, clinicians diagnosed correctly only about one third of men with chancroid and ten percent of patients with mixed aetiologies (Dangor 1990). In a similar observation from China, 12 of 106 cases of syphilis were incorrectly classified as herpes genitalis, and did not receive the correct treatment (Wang et al 2002). The clinical approach should therefore be abandoned.

(ii) The laboratory based aetiological approach tries to identify the organism responsible for the symptoms with which a patient presents. A number of obstacles make this approach largely inappropriate for many areas in Southeast Asia: Sufficiently equipped laboratories do not exist in rural communities and many smaller towns, and even in urban areas where they exist, quality control systems are often insufficient (WHO 2001). For some STIs, such as *Chlamydia trachomatis* infection, available tests are expensive, sophisticated or too insensitive.

(iii) The syndromic approach is based on the diagnosis of the syndrome, and deliberately does not attempt to identify the underlying aetiology. A syndrome is defined as a combination of symptoms and easily recognisable signs. Important STI/RTI syndromes are the genital ulcer syndrome in men and women, the urethral discharge syndrome in men, the syndrome of

painful testicular swelling in men, the vaginal discharge syndrome in women, the lower abdominal pain syndrome in women, and the inguinal adenopathy syndrome (buboe) in men and women. Each STI/RTI syndrome can be caused by a variety of aetiological agents. The occurrence of these aetiologies, and their proportional distribution may differ between regions and countries. Not all aetiological agents are sexually transmitted. For example, vaginal discharge may be caused by STIs (due to trichomoniasis and occasionally to gonococcal or chlamydial cervicitis) as well as by endogenous RTIs (bacterial vaginosis and candidiasis).

The syndromic approach to case management implies that all major likely aetiological causes of the presenting syndrome are treated simultaneously at the place and time of first contact of the patient with the health sector. This approach has been promoted by the World Health Organization for more than a decade (WHO 1991).

#### Advantages of syndromic STI/RTI case management

Delays in the initiation of treatment are avoided, as patients do not need to wait for laboratory results. Secondly, because all major possible causes of STI/RTI syndromes are covered, cure is usually achieved early on. These advantages are important from a public health perspective: onward transmission is reduced, complications are prevented, clients are satisfied and their confidence in the health system strengthened.

The increased costs due to overtreatment of aetiological agents (that are not present in a particular patient) are outweighed by savings on laboratory costs. In a study of 1500 hypothetical STI patients with different syndromes using decision-theory analysis, it has been demonstrated that both clinical and laboratory based case management would cost two to three times as much as the syndromic based approach (WHO 1993).

The treatment of STIs can be standardised with the help of algorithms, thus enabling paramedical staff to treat STIs effectively in areas where physicians are not available. WHO has published a complete set of syndromic management algorithms and recommendations for the selection of drugs (WHO 2001). Syndromic case management thus allows STI treatment services to be integrated within the existing primary care system. The validity and the operational feasibility of this approach and its cost-effectiveness in reducing new HIV infection has been demonstrated in studies in Asia and elsewhere (Djajakusumah et al 1998, Hong et al 2002, Chilongozi et al 1996, Grosskurth et al 2000ii).

The effectiveness of specific algorithms depends on the correct choice of drugs to be included in the algorithms. WHO has emphasised that the

recommended algorithms should not be applied blindly but have to be adapted to the specific local epidemiological and antimicrobial sensitivity pattern (WHO 2001). It is therefore essential that countries monitor these patterns and that algorithms are constructed that are based on sound surveillance data. Each country should therefore have at least one reference clinic and laboratory that have access to primary care STI/RTI patients, and large countries require more than one such sentinel site. Unfortunately this essential requisite is often lacking.

A study conducted at rural child health/family planning clinics in Bangladesh has recently demonstrated the importance of this principle. Women who presented with vaginal discharge were examined to identify the causative agents of their complaints. Only about 30% of the 320 participating women had detectable infections, most of which were caused by bacterial vaginosis and candidiasis. Cervical infections due to *Neisseria gonorrhoeae* and *Chlamydia trachomatis* were found in only 3 women. The application of the unadapted WHO algorithm that is presently in use in Bangladesh would lead to a high rate of overtreatment, and almost 90% of programme costs would be spent on uninfected women. In addition, women diagnosed as having an 'STI' are potentially exposed to matrimonial conflicts or even violence (Hawkes et al 1999). Clearly, local adaptation of the algorithm is required, and in this particular case should take other causes for abnormal vaginal discharge such as potential side-effects of contraceptive use into consideration.

#### Disadvantages of the syndromic approach

Patients are frequently treated for infections which they do not have, thus exposing them to possible side effects unnecessarily.

Not rarely, physicians who are familiar with the classical diagnostic principle whereby treatment should always be based on a precise diagnosis, have difficulties in accepting a method which they perceive as 'unscientific' (Kumar et al 1995) or 'third world medicine'. However, few procedures recommended in the context of disease management in developing countries are based on so much careful research as the syndromic management of STIs (Chilongoti et al 1996, Dallabetta et al 1998, Harrison et al 2000, Mayaud et al 1997, Grosskurth et al 2000ii).

The syndromic management of vaginal infections has generally worked very well. The approach has occasionally been criticised because vaginal discharge often reappears within some months following initial improvement. However, this problem does usually not originate from the kind of diagnostic approach used, but from the high recurrence rate of bacterial vaginosis.



Between 50% and 70% of patients with proven and appropriately treated bacterial vaginosis experience recurrence of symptoms (Boris et al 1997).

The main problem, however, lies in the management of cervical infections due to *Neisseria gonorrhoeae* (NG) and *Chlamydia trachomatis* (CT). Because NG, and CT are the most dangerous infective agents of the female reproductive tract, and because they occasionally present as vaginal discharge, their treatment is often included in algorithms for the management of the vaginal discharge syndrome. Unfortunately, these infections are usually asymptomatic, and seem to cause clinical symptoms in less than 15% (Korenromp et al 2002). Such algorithms have a high sensitivity because all genuine cervical infections will be treated, but a very low specificity, as many women will be treated unnecessarily; for example 97% in a study from Bangladesh (Bogaerts et al 1999). On the other hand, the sensitivity would be zero if the treatment of NG and CT were not included in the algorithm.

An attempt to overcome this problem has been made through the introduction of a risk assessment step into algorithms for the management of the vaginal discharge syndrome, hoping that this would increase the algorithms' specificity for NG and CT infection without losing much sensitivity (Mayaud et al 1998, WHO 2001). The risk assessment refers to parameters known to be associated with an increased risk of cervical infection. Questions are for example asked about the age of the patient (e.g. less than 21 years), about the marital status (unmarried; new partner in the previous three months), about the number of partners during the recent past, recent use of condoms, and whether the partner has himself a discharge or ulcer. The validity of this approach has been evaluated in a variety of settings but the results are not at all encouraging, unfortunately. Whilst its specificity of this approach is often higher than 90%, its sensitivity in detecting cervical infections ranged only from 5% to 46% in studies from India, Myanmar and Tanzania (Mayaud et al 1998; Vishwanath et al 2000; Department of Health, Myanmar and Population Council, Thailand, 2002). As can be imagined, the strategy is particularly ineffective and leads to much overtreatment in populations with low prevalences of cervical infections such as that in the study from Bangladesh mentioned above (Hawkes et al 1999).

During a recent technical meeting at WHO it has therefore been decided to revise the existing recommendations for the case management of vaginal discharge: risk assessment to identify women with cervical infections as part of the management of vaginal discharge should in future only be used in populations with a proven high prevalence of NG and CT infections. The algorithms for vaginal discharge should focus on vaginal infections.

## STIs- agents and symptoms

STI	Etiological agent	Common symptom
Genital herpes	Herpes simplex virus Type 2	Vesicles, genital ulcer
Syphilis	Treponema pallidum	Genital ulcer, bubo, <u>condylomata and skin rash</u>
Chancroid	Haemophilus ducreyii	Genital ulcer, bubo
Lymphogranuloma venereum	Chlamydia trachomatis (L1-3 serovars)	Genital ulcer, bubo
Donovanosis (granuloma inguinale)	Calymmatobacterium granulomatis	Genital ulcer
Gonorrhoea	Neisseria gonorrhoeae	In men: discharge per urethra, dysuria, scrotal swelling. In women: often asymptomatic, sometimes cervical mucopus presenting as vaginal discharge.
Bacterial vaginosis	Gardnerella vaginalis Bacteriodes sp.	Vaginal discharge
Chlamydial genital tract infection	Chlamydia trachomatis*	In men: discharge per urethra, dysuria, scrotal swelling In women: often asymptomatic, sometimes cervical mucopus presenting as vaginal discharge
Trichomoniasis	Trichomonas vaginalis	In women: vaginal discharge In men: urethral discharge, dysuria
Vulvovaginal candidiasis	Candida albicans	In women: Vaginal discharge In men: Candidal balanitis
Pelvic inflammatory disease	Neisseria gonorrhoeae Chlamydia trachomatis Vaginal anaerobes	Lower abdominal pain or tenderness

\*Other less common agents of NGU are Ureaplasma urealyticum, Mycoplasma genitalium

Public health importance of STIs and the STI/HIV co-factor effect  
Heiner Grosskurth, Gurumurthy Rangaiyan

Sexually transmitted infections (STIs) represent a major public health problem in developing countries. Nearly 150 million sexually transmitted infections occur annually in Southeast Asian countries. According to the World Development Report 1993, the burden of disease in women of child bearing age caused by STIs (without HIV infection) and RTIs is the second highest of all groups of diseases, surpassed only by maternity related disorders. It is their complications and long term consequences rather than the acute infections that make STIs such an important public health problem for women and their offspring: acute and chronic pelvic inflammatory disease, infertility, puerperal sepsis, ectopic pregnancy, miscarriage, stillbirth, preterm delivery, low birthweight and severe congenital infections.

However, the public health importance of STIs has risen even more since it is known that STIs enhance the sexual transmission of HIV infection.

At the individual level, STIs increase the susceptibility of HIV-negative individuals for HIV infection because they interrupt the integrity of the genital epithelium and because they attract inflammatory cells that in turn increase access of HIV to CD4 receptors. Genital ulcers caused by syphilis, chancroid and genital herpes, but also gonorrhoea, chlamydial infection and trichomoniasis are all known to increase HIV susceptibility. There is some evidence that this may also be the case for bacterial vaginosis.

STIs also increase the infectiousness of HIV-positive persons. The concentration of HIV in semen has been found to be six times higher among HIV-positive men who have urethritis compared to those who have not. Similarly, HIV shedding is increased in HIV infected women who suffer from gonococcal infection of the cervix, for example due to gonorrhoea. HIV can also be detected in genital ulcers. It has been hypothesised that STIs upregulate the viral load of HIV in the blood, which in turn enhances the concentration of HIV in genital fluids.

Most epidemiological studies that investigated the cofactor effect of STIs on HIV transmission reported relative risks in the order of 2 to 8, suggesting an up to 8-fold increased risk of HIV acquisition in the presence of STIs (Fleming and Wasserheit 1999). However, such

studies underestimate the per exposure risk of HIV transmission in the presence of STIs. For example for genital ulcers, data are consistent with a 10 - 50 fold increase in the probability for male to female HIV transmission per sexual act, and a 50 - 300 fold increase for female to male transmission.

At the population level, STIs seem to be one of the key factors that drive the HIV pandemic in developing countries. The proportion of new HIV infections in a population due to bacterial STIs is particularly high in early and moderately advanced HIV epidemics when HIV prevalences are still rising. There is evidence from computer based simulation studies that the HIV epidemic could not have taken off in some countries without the facilitation by other STIs. However, once the epidemic approaches a generalised stage, the contribution of STIs to the epidemic seems to steadily decrease. Thereafter, most transmissions occur within stable partnerships even in the absence of STIs, and viral load becomes the main determinant of HIV transmission.

The effective control of STIs in early HIV epidemics, particularly among high risk behaviour groups and bridging populations where STI prevalences are high, has the potential to prevent the generalisation of these epidemics, and is therefore of paramount importance. This is precisely the epidemiological situation that currently still prevails in most parts of Asia, including India.

### Genital Ulcer Syndrome

*[Adapted from (WHO) Guidelines for Management of Sexually Transmitted Infection. WHO/HIV\_AIDS/2001.01WHO/RHR/01.10.]*

The relative prevalence of causative organisms for Genital ulcer syndrome varies in different parts of the world and may change over time. Clinical differential diagnosis of genital ulcers is inaccurate, particularly in settings where several aetiologies are common. Clinical manifestations and patterns of Genital ulcer syndrome may be further altered in the presence of HIV infection. After examination to confirm the presence of genital ulceration, treatment appropriate to local aetiologies and antibiotic sensitivity patterns should be given. For example, in areas where both syphilis and chancroid are prevalent, patients with genital ulcers should be treated for both conditions. In many parts of the world, genital herpes is the most frequent cause of Genital ulcer syndrome. Where HIV infection is prevalent, an increasing proportion of cases of Genital ulcer syndrome is likely to harbour herpes simplex virus. Laboratory-assisted differential diagnosis is rarely helpful at the initial visit, as mixed infections are common. In addition, in areas of high syphilis prevalence, a reactive serological test may reflect a previous infection and give a misleading picture of the patient's present condition. Not all genital ulcers are caused by sexually transmitted infections and other causes include traumatic ulcer, tuberculosis, amoebiasis, aphthous ulcer, erythema multiforme and rarely malignancy.

HIV testing should be performed in the management of patients who have genital ulcers. Since hepatitis B can also be also sexually acquired, a screening HBsAg should also be done.

The clinical picture of ulcer disease may be severe and prolonged with HIV infection requiring longer course of therapy. Recurrence rates after treatment may be higher. The presence of ulcerative genital disease increases both risk of acquiring and transmitting HIV infection. Careful clinical follow-up is required to ensure cure of the ulcer disease and prevention of recurrence.

GENITAL ULCER SYNDROME

Disease	Chancroid	Granuloma Inguinale (Donovanosis)	Genital herpes	Lymphgranuloma venereum (LGV)	Syphilis
Agent	Haemophilus ducreyi	Calymmatobacterium granulomatis	Herpes simplex type 2 and 1 (HSV-2 and HSV-1)	Chlamydia trachomatis serovars L1, L2 and L3	Treponema pallidum
Clinical features	Ulcers multiple, painful, irregular with undermined edges, and not indurated (soft chancre). Unilateral painful bubo (Suppuration may occur).	Painless, progressive ulcer ("beefy red appearance")  No regional lymphadenopathy	Multiple painful grouped vesicles; vesicles ulcerate and coalesce  Bilateral adenopathy in primary infection Recurrent herpes genitalis common	Transient ulcer  Unilateral tender inguinal adenopathy (Groove sign seen in 20% of patients)	Painless single ulcer indurated with clean base.  Firm bilateral adenopathy
Treatment	Erythromycin 500 mg QID for 7 days. OR Ciprofloxacin 500 mg stat. OR Ceftriaxone 250 mg intramuscularly (IM) in a single dose.	Doxycycline 100 mg orally twice a day for at least 21 days. OR Trimethoprim-sulfamethoxazole one double-strength (800mg/160mg) tablet orally twice a day for at least 3 weeks. OR Azithromycin 1 g orally in a single dose.	Primary: Acyclovir 400 mg orally three times a day for 7-10 days. OR Acyclovir 200 mg orally five times a day for 7-10 days.  Recurrent Herpes: Initiate treatment < 48 hours of onset for 5 days  Suppressive therapy: (>6 recurrences per year) Acyclovir 400 mg orally twice a day for a few years.	Aspiration of bubo*. Doxycycline 100 mg orally twice a day for 14 days. Alternative Regimen: Erythromycin base 500 mg orally four times a day for 14 days.	Benzathine penicillin G, 2.4 million units IM. Penicillin allergy:** Tetracycline, 500 mg PO, four times daily, for 15 days. (OR) Doxycycline, 100 mg PO, twice daily for 15 days.

\*If in doubt about the underlying cause, buboes must not be incised and drained but should be aspirated with the needle inserted through healthy tissue.

\*\* In pregnant women allergic to penicillin desensitise to Penicillin.

### Inguinal bubo

[Adapted from *(WHO) Guidelines for Management of Sexually Transmitted Infection. WHO/HIV\_AIDS/2001.01WHO/RHR/01.10.*]

Inguinal and femoral buboes are localised enlargements of the lymph nodes in the groin area, which are painful and may be fluctuant. They are frequently associated with lymphogranuloma venereum and chancroid. In many cases of chancroid an associated genital ulcer is visible, but occasionally may not be. Non-sexually transmitted local and systemic infections (e.g. infections of the lower limb) can also cause swelling of inguinal lymph nodes.

See Lymphogranuloma venereum and Chancroid (page 45).

### Vaginal discharge

[Adapted from *(WHO) Guidelines for Management of Sexually Transmitted Infection. WHO/HIV\_AIDS/2001.01WHO/RHR/01.10.*]

A spontaneous complaint of abnormal vaginal discharge (abnormal in terms of quantity colour or odour) is most commonly due to a vaginal infection. Rarely, it may be the result of muco-purulent STI-related cervicitis. *T. vaginalis*, *C. albicans* and bacterial vaginosis are the commonest causes of vaginal infection and *N. gonorrhoeae* and *C. trachomatis* cause cervical infection. The clinical detection of cervical infection is difficult because a large proportion of women with gonococcal or chlamydial cervical infection are asymptomatic. The symptom of abnormal vaginal discharge is highly indicative of vaginal infection, but poorly predictive for cervical infection. Thus, all women presenting with vaginal discharge should receive treatment for trichomoniasis and bacterial vaginosis. Among women presenting with discharge, one can attempt to identify those with an increased likelihood of being infected with *N. gonorrhoeae* and/or *C. trachomatis*. Microscopy adds little to the diagnosis of cervical infection and is not recommended. To identify women at greater risk of cervical infection, an assessment of a woman's risk status is useful, especially when risk factors are adapted to the local situation. Women who are positive on risk assessment, have a higher likelihood of cervical infection than those who are risk negative. Women with vaginal discharge and a positive risk assessment could therefore be offered treatment for gonococcal and chlamydia cervicitis. Where resources permit, one could consider the use of laboratory tests to screen women with vaginal discharge including saline mount, Gram stain and KOH preparation. Such screening could be applied to all women with discharge or selectively to those with discharge and a positive risk assessment. Most patients with Chlamydia and Gonococcal infection are asymptomatic and require screening and treatment. Treatment of both these agents does not differ in HIV positive individuals.

## VAGINAL DISCHARGE

	Candidial vaginitis	Trichomonas vaginitis	Bacterial vaginosis
<b>Etiology</b>	Candida albicans and other Candida sp.	Trichomonas Vaginalis	Gardnerella vaginalis, anaerobic bacteria, Mycoplasma genitalium
<b>Clinical Features</b>			
<b>Symptoms</b>	Vaginal discharge, vulval itching, burning	Vaginal discharge	Malodorous, Discharge
<b>Discharge</b>	Curdy white discharge	Profuse Yellow frothy Discharge	White or grey Homogenous discharge
<b>Genital examination</b>	Erythema of introitus and vaginal wall, vulvar dermatitis	Erythema of vagina Strawberry cervix	None
<b>Microscopy.</b>	KOH - hyphae seen	Motile Trichomonas vaginalis on wet mount	Clue cells (squamous cells covered by bacterial rods) on wet mount
<b>Treatment</b>	Miconazole or Clotrimazole pessaries daily for 3 days. Or Fluconazole 150 mg stat.	Metronidazole* 2 gm stat. Tinidazole 2 G stat or Metronidazole 400 mg tid for 7 days.	Metronidazole* 400 mg tid for 7 days or Metronidazole 2 G stat.**

\* Patients taking the imidazoles should be cautioned against taking alcohol for upto 24 hours after taking the last dose.

\*\* Treatment of sex partner in bacterial vaginosis is not indicated.

Patients with vaginal discharge need to be treated for Gonorrhoea and Chlamydia infection if risk assessment is positive or if there is mucopurulent cervicitis.

Gonorrhoea - T. Ciprofloxacin 500 mg stat

Chlamydial infection - Doxycycline 100 mg BD for 7 days.



### Urethral Discharge

[Adapted from (WHO) Guidelines for Management of Sexually Transmitted Infection. WHO/HIV/AIDS/2001.01WHO/RHR/01.10.]

Male patients complaining of urethral discharge and/or dysuria should be examined for evidence of discharge. If none is seen, the urethra should be gently massaged from the ventral part of the penis towards the meatus. If microscopy is available, examination of the urethral smear may show an increased number of polymorphonuclear leukocytes and a gram stain may demonstrate the presence of gonococci. In the male, more than 5 polymorphonuclear leukocytes per high power field ( $\times 1000$ ) are indicative of urethritis. If the patient complains of dysuria and there is no discharge on examination, a positive leucocyte esterase test or  $> 10$  PMNs/HPF of first voided urine or after holding urine for 4 hours also confirms the diagnosis of urethritis. The major pathogens causing urethral discharge are *N. gonorrhoeae* and *Chlamydia trachomatis* (*C. trachomatis*). In the syndromic management, treatment of a patient with urethral discharge should adequately cover these two organisms.

Persistent or recurrent symptoms of urethritis may be due to drug resistance, poor compliance or re-infection. In some cases there may be infection with *Trichomonas vaginalis* (TV). There is new evidence suggesting high prevalence of TV in men with urethral discharge in some geographical settings. Where symptoms persist or recur after adequate treatment for gonorrhoea and chlamydia in index patient and partner(s), the patient should be treated for TV. If the symptoms still persist at follow up the patient must be referred. Patients who have urethritis and also are infected with HIV should receive the same treatment regimen as those who are HIV-negative.

	Non-gonococcal urethritis	Gonorrhoea
<b>Etiology</b>	Chlamydia trachomatis	Neisseria gonorrhoea
<b>Clinical Features</b>		
Onset	Gradual	Abrupt
Dysuria	Mild	Severe
Discharge		
Quality	Mucoid	Purulent
Quantity	Less	More
<b>Microscopy.</b>	Gram negative intra-cellular diplococci	Urethral smear $> 5$ PMNs/HPF
<b>Treatment</b>	Azithromycin 1 g orally in a single dose OR Doxycycline 100 mg orally twice a day for 7 days. Alternative Regimens Erythromycin base 500 mg orally four times a day for 7 days	Ciprofloxacin 500 mg stat Or Azithromycin 2 G stat Or Ceftriaxone 250 mg IM stat

### Scrotal swelling

[Adapted from (WHO) Guidelines for Management of Sexually Transmitted Infection. WHO/HIV\_AIDS/2001.01WHO/RHR/01.10.]

Inflammation of the epididymis (epididymitis) usually manifests itself by acute onset of unilateral testicular pain and swelling, often with tenderness of the epididymis and vas deferens and occasionally with erythema and oedema of the overlying skin. In men under 35 years of age, this is more frequently due to sexually transmitted organisms than in those over 35 years of age. When the epididymitis is accompanied by urethral discharge, it should be presumed to be of sexually transmitted origin, commonly gonococcal and/or chlamydial in nature. The adjacent testis is often also inflamed (orchitis), giving rise to epididymo-orchitis. In older men, where there may have been no risk of a sexually transmitted infection, other general infections may be responsible, for example, *Escherichia coli*, *Klebsiella* sp. or *Pseudomonas aeruginosa*. A tuberculous orchitis, generally accompanied by an epididymitis, is always secondary to lesions elsewhere, especially in the lungs or bones. It is important to consider other non-infectious causes of scrotal swelling, such as trauma, testicular torsion and tumour. Filarial lymphadenitis is another prevalent cause of scrotal swelling. Testicular torsion, which should be suspected when onset of scrotal pain is sudden, is a surgical emergency that needs urgent referral. If not effectively treated, STI-related epididymitis may lead to infertility.

#### Treatment

Treatment of Gonorrhoea- T. Ciprofloxacin 500 mg stat or Inj. Ceftriaxone 250 mg IM stat.

Treatment of Chlamydia- T. Doxycycline 100 mg bd for 10 days.

### Lower abdominal pain

[Adapted from (WHO) *Guidelines for Management of Sexually Transmitted Infection. WHO/HIV\_AIDS/2001.01WHO/RHR/01.10.*]

All sexually active women presenting with lower abdominal pain should be carefully evaluated for the presence of salpingitis and/or endometritis - pelvic inflammatory disease (PID). In addition, routine bimanual and abdominal examinations should be carried out on all women with a suspected STI since some women with PID or endometritis will not complain of lower abdominal pain. Women with endometritis may present with complaints of vaginal discharge and/or bleeding and/or uterine tenderness on pelvic examination. Symptoms suggestive of PID include abdominal pain, dyspareunia, vaginal discharge, menometrorrhagia, dysuria, pain associated with menses, fever, and sometimes nausea and vomiting.

PID is difficult to diagnose because clinical manifestations are varied. PID becomes highly probable when one or more of the above symptoms are seen in a woman with adnexal tenderness, evidence of lower genital tract infection, and cervical motion tenderness. Enlargement or induration of one or both fallopian tubes, a tender pelvic mass, and direct or rebound tenderness may also be present. The patient's temperature may be elevated but is normal in many cases. In general, clinicians should err on the side of over-diagnosing and treating suspected cases. Hospitalisation of patients with acute pelvic inflammatory disease should be seriously considered when: the diagnosis is uncertain; surgical emergencies such as appendicitis and ectopic pregnancy can not be excluded; a pelvic abscess is suspected; severe illness precludes management on an outpatient basis; the patient is pregnant; the patient is unable to follow or tolerate an outpatient regimen; or the patient has failed to respond to outpatient therapy. Many experts recommend that all patients with acute PID should be admitted to hospital for treatment. Etiological agents include *N. gonorrhoeae*, *C. trachomatis*, anaerobic bacteria (*Bacteroides* spp. And Gram-positive cocci). Facultative Gram-negative rods and *Mycoplasma genitalium* have also been implicated. As it is impossible to differentiate between these clinically, and a precise microbiological diagnosis is difficult, the treatment regimens must be effective against this broad range of pathogens. The regimens recommended in the syndromic case management for abdominal pain are based on this principle.

### Secondary syphilis

#### Clinical findings:

Rash: macular, papular, pustular, combination; usually nonpruritic; may involve palms and soles in 60%. Generalized lymphadenopathy: 86%. Mucous patches (5-30%): flat patches involving mouth, pharynx, larynx, genitals. Condylomata lata (5-25%): heaped, wart-like, papules that enlarge in warm intertriginous areas (gluteal folds, nasolabial folds, axillae, between toes, under breasts, perineum and peri-anal etc.); teeming with treponemes and are highly infectious. Constitutional symptoms: malaise, headache, pharyngitis, slight fever, myalgia; liver and kidney involvement, patchy alopecia.

### Serologic Tests for Syphilis

*Adapted from: (CDC) Sexually Transmitted Diseases Treatment Guidelines 2002 MMWR 2002; 51: 1-80.*

#### a) VDRL and RPR

Titers usually correlate with disease activity; results should be reported quantitatively. A fourfold change in titer, (e.g., from 1:16 to 1:4 or from 1:8 to 1:32) is necessary to demonstrate clinical response to treatment using the same testing method and by the same laboratory. Results from the two tests cannot be compared because RPR titers are slightly higher than VDRL titers.

Titers usually become nonreactive with time after treatment; antibodies can persist at a low titer for a long period of time, sometimes for the life of the patient. This response is referred to as the "serofast reaction." VDRL and RPR are non-specific tests that may sometimes give false-positive results and ideally should be confirmed using a treponemal test.

#### b) Treponemal tests

Fluorescent treponemal antibody absorbed [FTA-ABS], T pallidum haemagglutination [TPHA] will have reactive tests for the remainder of their lives, regardless of treatment or disease activity. Antibody titers correlate poorly with disease activity and should not be used to assess treatment response.

#### c) HIV infection

Some HIV-infected patients can have atypical serologic test results (unusually high, unusually low, or fluctuating titers). When serologic tests and clinical syndromes suggestive of early syphilis do not correspond with one another, use of other tests (e.g., biopsy and direct microscopy) should be considered. However, for most HIV-infected patients, serologic tests are accurate and reliable for the diagnosis of syphilis and for following the response to treatment.

### Treatment of Syphilis

Secondary syphilis:

Benzathine penicillin G, 2.4 million units IM.

Penicillin allergic: Tetracycline, 500 mg PO, for 4 weeks. (OR)

Doxycycline, 100 mg PO, twice daily for 4 weeks.

Pregnant women sensitive to penicillin should be treated with:

Erythromycin 500 mg QID for 15 days.

Follow-Up:

1. Early syphilis: quantitative VDRL or RPR at 6, 12 months.
2. Late latent, tertiary: quantitative VDRL or RPR at 3, 6, 12, 18, 24 months.
3. Neurosyphilis: serological testing as above, with repeat CSF examination at six-month intervals for 3 years or until normal.
4. HIV-infected patients: 3, 6, 9, 12 months for early syphilis, adding 18 and 24 months for syphilis of >1 year duration.

OTHER STIs1. *Genital Warts*

Etiology: Human Papilloma virus

Clinical findings: Warty excrescences in the following areas:

Men- penis, scrotum, urethral meatus, perianal region

Women- Introitus, vulva, perineum, cervix and vagina

HIV infection:

The warts tend to become very large and there is risk of malignant transformation into squamous cell carcinoma

Treatment-

Podophyllin application

80% TCA or MCA

Curretage, cryosurgery, electrocautery and excision

If complete clearance of warts is not achieved in a period of 6 weeks, it is advised to use a different treatment modality or referral to a specialist is recommended.

2. *Molluscum conatgiosum*

Etiology: Pox virus family.

Clinical findings: Flesh coloured smooth, firm and dome shaped papules with central umbilication.

In HIV infection they develop widespread lesions over trunk and the face and giant lesions tend to occur.

Treatment:

Needling, cryotherapy, cautery, TCA, Silver nitrate application

## REFERENCES

1. (APAC) Prevention and Control Quality STD Care Module for private practitioners. *AIDS Prevention and Control Project, Voluntary Health Services* 1998.
2. (CDC) Sexually Transmitted Diseases Treatment Guidelines 2002 *MMWR* 2002; 51: 1-80.
3. (WHO) Guidelines for Management of Sexually Transmitted Infection. WHO/HIV\_AIDS/2001.01WHO/RHR/01.10.
4. (NACO) Practical considerations in diagnosis and treatment of STDs.
5. (Grosskurth H, Rangaiyan G) The management and control of sexually transmitted infection, and their implications for AIDS control in South-east Asia (to be published in *Journal of Health Management*).

## FURTHER READING

1. Hawkes S, Santhya K.G. Diverse realities: sexually transmitted infections and HIV in India. *Sex. Transm. Infect.* 2002; 78 (Suppl I): 131-39.
2. Rodrigues J.J, Mehendale S.M et al. Risk factors for HIV infection in people attending clinics for sexually transmitted diseases in India. *BMJ* 1995; 311:283-286.
3. Pedhambkar R.B, Pedhambkar B.S, Kura M.M. Study of risk factors associated with HIV seropositivity in STIs patients at Mumbai, India. *Sexually Transmitted Infections* 2001; 77:388-389.
4. Thomas K, Thyagarajan S.P et al. Community based prevalence of sexually transmitted diseases and human immunodeficiency virus infection in Tamil Nadu: a probability proportional to size cluster survey. *National Medical Journal of India* 2002; 15: 135-40.
5. Grosskurth H, Gray R et al. Control of sexually transmitted diseases for HIV-1 prevention: understanding the implications of the Mwanza and Rakai trials. *Lancet* 2000; 355: 1981-7.

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