

MODULE 12

LABORATORY TESTS FOR OI'S

LEARNING OBJECTIVES

When you have completed this module you should be able to:

1. List Laboratory tests and correct specimens as well as their proper collection for the diagnosis of specific OI'S in HIV patients?
2. Know the capabilities and limitation of the available Laboratory diagnostic services for OI'S in Zimbabwe and elsewhere.

12.1 INTRODUCTION

One of the early significant advances in the management of HIV/AIDS was the demonstration that Chemoprophylaxis could prevent PCP and thereby improve survival. The *laboratory diagnosis back up* confirmed the clinical presentation. Currently effective prophylactic and treatment agents for this PCP and other common OI'S is the way forward in management of HIV/AIDS patients in countries with poor resources in developing countries such as Zimbabwe. The decision making as to when to use or not to use the prophylactic and treatment agents requires Laboratory based diagnosis which give definitive results. Definitive data can be accumulated for use in policy making for preventative measures such as prophylactic use of certain antibiotic or anti-fungals in prevalent or high risk OIs.

Besides the Laboratory culture or definitive diagnosis and confirmation of the presence or absence of the causative agent of the OI'S the laboratory determine the effectiveness of the agents used for prevention and /or treatment. In cases where antimicrobial or antifungal elements/agents are prescribed, the laboratory facilities monitor the susceptibility or the development of resistance to them. Below are some outlines of the laboratory diagnostic methods available in Zimbabwe. The availability of or the degree of sophistication of the laboratory service is typical of any other developing country in the world. The less complicated (simple) methods can be carried out in provincial and district hospitals laboratories and followed by the most complicated diagnostic methods, addition to the simple methods above, being done in central, private and University Laboratories.

12.2 LABORATORY METHODS FOR FUNGAL INFECTION

Fungal infections are very rare infections in immuno-competent persons.

12.2.1 Candidiasis:

The appearance of mucosal candidiasis is often the first clinical indication of impaired T cell immunity in HIV infected individual. Oral and vaginal thrush are almost ubiquitous but Candida esophagitis is the second most common OI after PCP. Candidemia and tissue disease are rare. Pharyngitis may be asymptomatic while plaques can be easily scraped from the pharynx or buccal mucosa: severe cases will involve the tongue, gums and lips. Vaginitis causes thick white discharge, pruritis and sometimes dyspareunia and has a similar appearance on speculum examination.

Specimens: scrapings from appropriate sites such as oral or high vaginal swabs collected and transported immediately to the laboratory unless transport medium such as stuart's or Amies transport medium is used (24 –48 hrs limits)

Laboratory diagnosis:

Diagnosis is by microscopy (wet and stained smears) and culture of the appropriate specimens on selective media. Anti fungal susceptibility testing can be carried out on request for drugs used for Treatment e.g.: Fluconazole, Itraconazole or Amphotericin B

12.2.2 Cryptococcal Meningitis:

Presents as nothing more than the worst headache of the present life.

Specimen:

- Cerebrospinal fluid (CSF) collected and transported immediately in sterile universal bottles.
- Blood for blood culture
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Laboratory diagnosis is made by:

- Detection of cryptococcal capsular antigen in the cerebrospinal fluid (CSF) which relies upon a positive India ink wet preparation stain that demonstrates the organism's thick capsule.

- Culture of blood or CSF.

12.2.3 Histoplasmosis:

This is caused by a yeast called *Histoplasma capsulatum*. This is widely distributed in dry hot areas (associated with dried bird droppings). It produces a systemic disease with granulomata in the liver or granulomatous disease of the skin and often affects the apex of the lung. The lung appears like tuberculosis with nodular apical lesions and often one or more cavities.

Specimens:

- **Deep coughed sputum**
- **Broncho-alveolar lavage**
- **Lung biopsy**
- **Liver biopsy**
- **Blood**

Laboratory Diagnosis method:

- **Demonstration of yeast like organism with a very distinct thick capsule in the sputum.**
- **1 inch stained blood films or Histological sections of tissue (oral yeast-forms seen within mononucleus phagocytes)**
- **Culture or serology (Compliment fixation test)**
- Anti-fungal susceptibility testing can be done and is done routinely in Laboratories where culture facilities are available.
eg.for: Amphotericin or Itraconazole

12.2.5 *Pneumocystis carinii* Pneumonia: (PCP)

Once a protozoan parasite but recently reclassified to be a fungi on the basis of genetic. The classic triad of fever exertional dyspnea and non-productive cough occurs in only half of the cases, although almost all have at least two of the following: fever, cough. PCP may present as spontaneous pneumothorax.

Many diseases may have similar presentations to the *Pneumocystis carinii* [PC] infections such as the viral or bacterial pneumonias, Heart failure, pulmonary and pulmonary emboli and this overshadows definitive diagnosis and thus the significance of definitive Laboratory diagnosis.

Specimens:

- Bronchoalveolar lavage
- Induced sputum
- Deep coughed sputum
- Lung biopsies
- Saliva

Laboratory diagnosis:

This requires appropriate specimens from the affected lung segments to be concentrated and stained for PC organisms (trophozoites and /or Cysts) by:

- Any Romanosky stains (Giemsa stain, Wrights stain or Diff. Quick)
- Toluidine- O- Blue stain
- Grocott methanamine silver stain
- Molecular techniques are available for the diagnosis of *P. jirovecii* in saliva, tissue or the lung washings.(PCR)

EXERCISE. 6.2

For the Fungal opportunistic infections, list specimens with corresponding expected laboratory diagnostic tests. Give examples of specific organism/s (fungal) which is/are being tested for in each of the specimen.

12.3 VIRAL INFECTIONS:

12.3.1 Herpes Zoster infection:

This is caused by the Varicella Zoster-virus (VZV) or Herpes Zoster recently reclassified as Human Herpes virus 3 (HHV-3). This infection usually referred to as Chicken pox, primarily an illness of childhood manifested by a generalised vesicular rash moderate fever and systemic symptoms.

Complications are rare in children but include arthritis, hepatitis, pneumonia, encephalitis and glomerulonephritis. In immunocompromised hosts a progressive varicella infection can develop marked by prolonged fever and continued eruption of vesicles into the second week of illness. Herpes Zoster can be recurrent in a similar fashion as the herpes simplex 1 and 2. Treatment: Acyclovir

Specimens;

VZV is detected in the lesions by culture,

Laboratory diagnosis:

Serology

IFA or PCR

(Tests available in central private and University laboratories)

Treatment: Acyclovir

12. 3. 2 Herpes simplex viruses (HSV 1 & HSV 2)

HIV infected individual may have recurrent genital HSV that can be suppressed with oral antiviral drugs such as Acyclovir. Treatment and prophylaxis of HSV may require higher doses and some instances longer administration.

In men lesions appear as clusters of vesicles located primarily on the penis while in women the lesions can appear on the vulva, perineum buttocks cervix and less frequently the vagina in association with a light vaginal discharge.

Specimens:

- Exudates from the vesicles for IFA, culture-cell
- Blood for the serology tests-RIBA, ELIZA
- CSF
- Urethral swabs
- Vaginal and cervical specimens
- Throat and conjunctiva swabs for culture or IFA
- Exudates for PCR

Specimens:

- Blood for CMV antibody detection but this requires careful interpretation.
- Culture for CMV viral syndrome from urine peripheral blood leukocytes, breast milk, semen and cervical secretions.
- Lung or liver biopsies

Specimens:

- Puncture biopsy
- Blood

Laboratory Diagnosis:

- Histological stains of the section of the punch biopsy showing typical spindle-shaped cells is a definitive diagnosis.
- Blood for the detection of antibody is to the HHV-8 virus.
- PCR of blood or biopsy for HHV8 virus.

12.3.5 Human Papiloma virus (HPV):

This is the etiological agent for genital warts or condylomata accumulata a sexually transmitted disease. HPV infected lesions are located in areas of sexual contact the perineum genitalia in folds, anus or internally on the cervix and urethra. These lesions are potentially carcinogenic in nature. The HPV infection and genital warts are more common in HIV infected individual correlated with level of immunosuppression.

Diagnosis is usually clinically but laboratory diagnosis can be done in specialised laboratories using advanced techniques. (Treatment: Podophyllin)

Specimens: Paps smear,

biopsy of cervix and site of infection

exudates from wart site/s

Laboratory Diagnosis:

- Cytology and Histology methods for Koilocytosis demonstration
- Electron Microscopy
- Immunofluorescent stain by detecting specific antigens of the virus or antibodies to the virus.
- Molecular techniques such as PCR

12.3.6 Molluscum contagiosum

This infection is caused by the molluscum contagiosum. This virus is a member of the pox-virus family and is increasingly observed in sexually active adults and HIV infected individuals.

The presentation of the MCV lesions is a benign popular lesion of the skin and mucous membranes. They are mostly self-limited lesions developing as tiny pin point papules which group into a giant molluscum. They appear with translucent or light yellow in colour and may be found on thighs, inguinal region, buttocks and lower abdominal wall. Diagnosis clinically mainly and this is on the basis of the MC characteristic pearly umbilicated papule with the caseous center.

Specimens: Biospsies

Exudates from the site of infection or lesions

Laboratory diagnosis:

Diagnosis is basically histological, cytological demonstration of the pathogenic enlarged epithelial cells with intracytoplasmic molluscum bodies.

Thinly spread smears of material expressed from the lesions cores stained

by Wright stain will demonstrate sheets infected cells or

by Gram stain will also demonstrate sheets infected cells.

by IFA detection of the MCV antigens.

12. 3. 7 Hepatitis B

A DNA virus and a member of the hepadnaviridae virus family. Three important mechanisms of transmission prevail:

- Contact transmission via bodily secretions (blood semen and vaginal fluids)
- Maternal-infant transmissions across the placenta or during delivery.
- transmission at risk groups includes parental drug abusers and health care workers involved in needle sticks injuries. Infection via blood products if not screen prior.
- HBV incubation period of 6 weeks-6months is followed by symptoms associated with immune complex disease occur, rarely vasculitis glomerulonephritis.

Complications include liver failure and chronic hepatitis. Chronic HBV infection develops in 1-6% of persons who are infected as adults and these are at risk of chronic liver disease including cirrhosis and hepatocellular carcinoma. The presence of HB e Ag includes active viral replication and increased infective hepatitis. HIV infection increases risk of chronic HBV infection. No treatment is available but reduction of alcohol intake, which may act as a co-factor in the development of cirrhosis (liver function tests must be monitored).

Specimens: Blood specimens

Laboratory Diagnosis:

- Three HBV antigens and the corresponding antibodies are used in the diagnosis of acute and chronic HBV infection.
- Hepatitis B surface antigen (HbsAg) is the first serological marker of acute HBV infection appearing several weeks before symptoms.
- Hepatitis B antigen) HbeAG) appears soon after HbsAG and is the first antigen to disappear in patients who are recovering from hepatitis B. Its presence is associated with increased infectivity.
- IgM antibodies to the hepatitis b core antigen are a useful marker of acute HBV infection in patients presenting after surface antigen is no longer detectable.
- Presence of antibody to HbsAg the last serological marker to appear indicates past infection with HBV or previous vicinal.
- Chronic Hepatitis b infection is diagnosed based on failure to clear Hbs antigen. Hbe antigen may also persist and indicate continued viral replication and increased infectivity.

12.3.8 Hepatitis C (HCV)

Hepatitis c is an RNA virus related to the pestivirus genus of flavivirus family. This was previously known as the non-A none-B hepatitis or non HBV transfusion-related hepatitis. Transmission is similar manner to HBV (blood products, intravenous drug abuse, sexual transmission) Incubation period ranges from 2-6months. The hepatitis is often mild but frequently progresses to chronic active hepatitis and cirrhosis after many years (>25years) with an increased risk of developing liver carcinoma. HIV infection increases risk of chronic HCV infection. Impair response to vaccines against HBv and hepatitis A (HAV) HCV-infected individual are at risk of actual of hepatitis A in pregnancy or in maternal co-infection with HIV and HCV there is an increased risk often perinatal HIV transmission.

Specimen: Blood

Laboratory diagnosis:

Use of serology for the presence of antibody to HCV and in well equipped laboratories polymerase chain reaction is applicable for the detection of HCV-RNA.

EXERCISE. 6.3

For the Viral opportunistic infections, list specimens with corresponding expected laboratory diagnostic tests. Give examples of specific organism/s (viral) which is/are being tested for in each of the specimen.

12.4. PARASITIC OPPORTUNIST INFECTIONS:

12.4.1 Toxoplasmosis:

Is an infection caused by a protozoan called toxoplasma gondii whose definitive host are cats. Humans become infected via ingestion of oocysts from contact with cat faeces or from ingestion of meat contaminated with trophozoites. Maternal infection is often sub-clinical as it presents as a mild influenza like illness.

Clinical features include stillbirths choroidoretinitis, microcephaly, convulsions intracerebral calcification with resultant hydrocephalus hepatosplenomegaly and thrombocytopenia.

Symptoms include fever chills malaise lymph-adenopathy myalgia and headache. *Toxoplasma gondii* often presents as a central nervous system infection with multiple space occupying lesions.

HIV infection is now the common predisposition factor for toxoplasma infection other than HIV conditions which predisposes to toxoplasmosis include Hodgkin's disease, cardiac transplants and acute leukaemia and thus the need for definitive Laboratory diagnosis. (Treatment: Combination of thiamine and Sulphadiazine Dapsone.)

Specimen: Blood

Laboratory Diagnosis:

- Serology-Detection of toxoplasma specific IgM antibodies in the mother during pregnancy or in the neonate.
- Testing for IgG antibodies to toxoplasma is recommended for all HIV infected individuals soon after the diagnosis of HIV is made.
- Prenatal testing in HIV positive pregnant women.

12.4. 2. Cryptosporidiosis

Aetiological agent is the *Cryptosporidium parvum*. A very difficult infection to manage. Acute self-limiting diarrhoea which is profuse and watery develops in immunocompromised infected children and adults but when there is a deficit in T-cell mediated immunity i.e. HIV infection resolution does not occur. Treatment:

C. parvum is naturally resistant to a wide range of disinfectants and antibiotics. Use Azithromycin, Paromomycin, and Spiramycin

Specimen: Stool

Laboratory Diagnosis:

- Modified ZN and examine for acid fast oocysts (red-stained oocysts)
- Stool concentration

12.4.3 Strongyloids stercoralis Infection:

Strongyloids stercoralis a nematode infection which is acquired by direct penetration of infective larvae through intact skin followed by invasion of the small bowel by adults which

produce further larvae perpetuating the infection. Infection can remain largely asymptomatic for more than 40 years. When cellular immunity is reduced uncontrolled multiplication of the parasite can develop. This is known as the hyper infection syndrome which may be complicated by Gram –ve septicaemia pneumonia or meningitis as larvae deposit bacteria in the tissues. Treatment:

Thiabendazole supported with adequate sanitation and AIDS prevention.

Specimen: stool/blood

Laboratory Diagnosis:

Stool microscopy (wet prep). Stool concentration and culture techniques for larvae.

Serology

12.4.4 Isospora belli: infection

Isospora belli is a coccidian parasite which causes diarrhoea and mal-absorption in immunocompromised patients. Treatment: Isospora bell is susceptible to antimicrobial therapy such as; Cotrimoxazole and Metronidazole , Notrofurantoin, Fansidar (Sulphadoxine pyrimethamine)

Specimen: stool

Laboratory Diagnosis:

Stool for the oocysts both direct wet preparation or after concentration Technique (Formal ether concentration)

12.4.5. Microsporidiosis

Microsporidia are protozoa that cause were recognised in electron microscopic studies in AIDS patients with diarrhoea and mal-absorption. These are identified as several small non-invasive parasites infecting the gastrointestinal mucosa. Enterocytoan bieneusi and protozoa can cause debilitating diarrhoea and weight loss in patients with advanced HIV disease. Patients may develop severe dehydration due to voluminous watery diarrhoea. The infection is self-limiting in individuals with immunocompetent defence system. Treatment: Supportive efforts of volume replacement and slowly or stopping diarrhoea. Albendazole.

Specimen: Stool or watery stool.

Diagnosis: Special strains in specialised laboratory. e.g.. IFA after concentration Techniques.

12.4.6. Scabies

Caused by the mite sarcoptes scabies which burrows in the epidermis the female mite laying eggs along their burrow's tracks. The condition is infectious by direct skin contact. Males attracted by warmth emerges into the area contact and burrows into the adjacent epidermis. Normal transmitted sexual those who share beds or those who care for infected.

Infectious asymptomatic but hypersensitivity to the males, their eggs or surface protein eventually causes severe itchy and leads to scratchy and excoriation of affected skin. Scabies can cause wide spread chronic lesions in immunosuppressed HIV patients. Diagnosis is syndromically by the typically distributed itchy marks.

Specimens: Squeezed material /fluid from the nodule

Laboratory diagnosis: Demonstration of the mite.

EXERCISE.6.4

For the Parasitic opportunistic infections, list specimens with corresponding expected Laboratory diagnostic tests. Give examples of specific organism/s (Parasitic) being tested for in each of the specimen.)

12.5 BACTERIAL INFECTIONS:

12.5.1 Introduction

Many clinical bacterial organisms can overcome infections in immunocompetent individuals. In individuals with reduced immune defence systems the infections are more frequent and often severe.

Bacteremia is a common presentation in the HIV associated immunosuppression. The principal bacteria implicated in these patients are Gram negative rods but Gram positive cocci are also important. The frequency with which these organisms cause infection changes with developments in the treatment and antimicrobial prophylaxis.

Enterobacteriaceae and *Pseudomonas* spp are the most commonly isolated gram-negative rods. They are usually derived from the patient's own intestinal flora gaining access to the circulation when the rapidly multiplying intestinal epithelium is damaged by some agent. The introduction of antimicrobial prophylaxis and improvements in drug treatment of these pathogens may reduce both incidence and mortality associated with them.

Hospital patients are susceptible to colonisation with resistant organisms because of both the administration of antibiotics and the effects of serious underlying disease. Hospital organisms may be transmitted on the hands of medical and nursing attendants or ingested food, notably washed vegetables.

Although Enterobacteriaceae and *P. aeruginosa* remain the commonest Gram negative bacilli isolated other organisms such as *Klebsella* spp, *Serratia* spp and *Enterobacter* sp have been noted elsewhere (Europe US) as playing a significant role in pathogens.

Gram positive organisms may include *Staphylococcus epidermis* the oral streptococci (*Streptococci mitis*) and *S. oralis*, *Enterococcus* spp, *Staphylococcus aureus* and *Corynebacterium Jerkeium*. Methicillin resistance *S aureus* (MRSA) strains are cell increasing problems in hospitals where organisms have established themselves. Some examples of infections based on site are as follows:

12.5.2 Bacterial Respiratory Infections:

6.5.2.1 Mycobacterial Tuberculosis:

An acid fast bacilli which is obligate aerobic non-capsulated non-motile and grow slowly a specialised media cell walls have large lipid content belongs to the genus mycobacteria which includes *M. bovis* logeler causing tuberculosis, *M. leprae* (causing) and also atypical mycobacteria *M. avium* intracellular *M Kansais* and *M. Marinum*.

M.tuberculosis and *M bovis* can pulmonary tuberculosis and extrapulmonary tuberculosis including meningitis osteomyelitis military tuberculosis cervical and mesenteric lymphadenopathy abdominal and renal tuberculosis. *M bovis* infection is typical localised to bone marrow and cervical or mesenteric lymph nodes.

There is a bi-directional interaction between mycobacterium tuberculosis and HIV. Each facilitates acquisition of the other. TB is virulent enough to cause disease in patients with intact immune system. It may occur in HIV infected individuals who still have high CD4 cell counts. TB becomes especially virulent in HIV seropositive individuals. Aspects of this virulence include the high frequency of positive blood cultures and have disseminated (miliary) infections. It is essential to provide directly observed therapy to ensure an adequate course of treatment and conversion of positive sputum cultures to negative. Treatment is with combination of antimycobacteria drugs (Isoniazid, rifampicin and Pyrazinamide.

Specimens:

- Biopsy or aspiration
- Urine
- Sputum X 3
- Blood culture

Laboratory Diagnosis:

- Microscopy of smears of sputum urine or tissue by Ziehl Neelsen stain (Zn) stained bacilli appear as thin bacilli with beads.
- A fluorescent rhodamine auramine dye can also be used.
- Culture on Lowenstein Jensen medium for 12 weeks
- Blood cultures for *M. tuberculosis* recovery or detection.
- PCR

12.5.2.2 Bartonellosis Infection:

This is a lymphocutaneous disease caused by *Bartonella henslie* of cat scratch or cat exposure. After 5-10 days swelling appears at the site of the scratch and may discharge a little pus. The local draining lymph nodes enlarge and become tender, occasionally suppurating and discharging.

In immunosuppressed individuals the Bartonella organisms can cause systemic and bacteraemic infection. Diagnosis is usually clinical. Treatment is usually self-limiting but lasts for 3 or more weeks. Oral Tetracycline or Erythromycin may shorten the course of the disease.

Specimen:

- Blood for serology and culture on special media
- Biopsy

Laboratory Diagnosis:

- Blood for serology, molecular techniques (PCR) and culture on special media.
- Silver staining of the lymph node-biopsy material may illustrate silver stain small bacteria which is diagnostic

12.5.2. 3. Lower Respiratory Tract Infections:

12.5.2 .3.1 Bacteremia and Pneumonia in the Immunosuppressed.

Classic chest pathology in this population may be due to *S. pneumonia*, *Mycoplasma pneumonia*, *Klebsiella pneumonia*, *Pseudomonas aeruginosa* and/or *Staphylococcus aureus*.

Specimens:

- Sputum BAL
- Blood cultures

Laboratory Diagnosis

Culture and antibiotic susceptibility testing for:

- *S.pneumonia*-Routine culture of blood culture and sputum culture.
Direct detection of pneumonia and specimens of urine or CSF
Treatment penicillin or erythromycin
- *Staphylococcus aureus*-sputum and blood cultures
Treatment: Penicillins ,Cephalosporins ,Vncomycin
- *Mycoplasma pneumonia* This organism can also be detected by serology also
Treatment: Erythromycin
- *Haemophilus influenza*
Laboratory diagnosis by sputum cultures and blood cultures
Treatment according to antibiotics susceptibility to drugs.
- *Pseudomonas aeruginosa*
Laboratory diagnosis is by culture of sputum or the relevant body sites.

isolation of *P. aeruginosa* and identification and characterisation by pyocin typing serotyping or molecular biology typing.

Treatment: Aminoglycosides or Broad spectrum penicillin (piperacillin)

Third generation cephalosporins (Ceftazidime) Quinolones (Ciprofloxacin)

12.5.2 .3.2 Klebsiella Pneumonia:

The genus *Klebsiella* contains a number of species including: *K. pneumoniae*, *K. aerogenes* and *K. oxytoca*. Pathogenicity is associated with capsule production infections are often opportunistic and associated with hospitalisation. Treatment: is with any Cephalosporins or the Aminoglycosides

Laboratory diagnosis:

Culture of relevant body sites: such as sputum and blood culture (as well as carrying out antibiotic susceptibility testing)

12.5.3 Bacterial Gastrointestinal Infections

Gastrointestinal infections are commonly encountered in persons HIV infected. Infection of the gastrointestinal tract may involve lips the mouth oesophagus small and large intestine and anus.

12.5.3 .1 Salmonella and non-typhoid salmonellas and serotypes

S. typhi and *paratyphi* causing enteric fever (typhoid and paratyphoid). Other salmonella cause enteritis.

Treatment: enteric fever-Chloramphenicol and Ciprofloxacin Other enterocolitis: Self limiting antibiotics Ciprofloxacin.

Specimen and laboratory diagnosis:

- Stool sample culture of stool samples identification of salmonella by biochemical and agglutination tests, phagotyping and susceptibility tests.
- For enteric fever isolation of *S. typhi* or *S. paratyphi* and other salmonella specimen from blood cultures (1st week of infection) Urine (2nd week) or from faeces (1st week on onwards) or the use of the serology (Widal test)
- Antimicrobial testing

6.5.3 .2 Shigellosis:

Main pathogenic specimens are *S. sonnei*, *S. boydi*, *S. dysenteriae* and *S. flexi* (mainly distinguished by biochemical reactions and antigenic characteristics (O antigen))

Pathogenics:

Pathogenicity: Disease caused by invasion and destruction of the colonic mucosa. They also produce an exo-toxin. Associated infections include dysentery (diarrhoea with blood and pus) Septicaemia complications are rare. Treatment: Often self limiting antibiotics (e.g. Trimethoprim, Chloramphenicol, Ciprofloxacin).

Laboratory Diagnosis:

Stool for culture on selective media DCA incubation and ID of organisms.
and antibiotic susceptibility testing.

12.5.3 .3 Campylobacter infection

Infection caused by a curved gram negative bacilli motile with a polar flagellum, non sporing capsulated sure specimens show optimal growth at 42°C require microaerophilus atmosphere plus 10% CO₂. Campylobacter cell walls contain exotoxin. Cytopathic extracellular toxins and enterotoxin have been demonstrated. Symptoms include acute enteritis rarely complicated by septicaemia. Treatment: antibiotics often required erythromycin for severe cases especially immunosuppressed.

Specimens: Stool and blood

Laboratory Diagnosis:

- Culture of stool sample on selective media at 42°C for 42-72 hours.
- Antibiotic susceptibility testing
- Serology for detection of serum antibodies to Campylobacter

12.5.3 .4 Clostridial infections:

Clostridium pathogens spore forming ensures the organisms survival in many environments. *C. difficile* causes pseudomembranous colitis especially in cases in species of antibiotic therapy complications and HIV co-infection. This organism produces two toxins A

acts on the gut mucosa whilst B results in a cytopathic effect on tissues culture cells.

Treatment: Metronidazole Vancomycin

Specimen: Stool

Laboratory Diagnosis:

Culture Isolation Identification of C difficult from stool using anaerobic culture and selective media. Toxin production done by Diagnosis can be made by direct detection of toxins in faeces.

REFERENCES:

1. Tom Elliot. Mark Hastings and Urrich Delsseibergar: Lecture notes on Medical Microbiology: 1978 (3rd edition) Blackwell Sciences Oxford: pp 1-343.
2. Sexually Transmitted Diseases epidemiology Pathology Diagnosis and Treatment: Kenneth A Borchardt Michael, A noble (Editors)1997 CRC press CLC New York pp 1-349.
3. Barbara A, Bannister, Norman T. Begg and Stephen H Gillespine Infectious Diseases: 1996 Blackwell Science. Victoria pp 1-484
4. A Guide to the clinical Care of women with HIV: Jean R Anderson (Editor) 2001 Edition USA Dept of Health Resources Rockville p 1-510)