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MICROBIOLOGY

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Solved Question Papers

Fifth Edition

Singi Yatiraj

Foreword
SB Siddesh

JAYPEE

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MBBS PHASE II EXAMINATION
OCTOBER 2007
(Revised Scheme 2)
Paper II

JAYPEE BROTHERS

LONG ESSAYS

1. List the aroboviruses seen in India. Describe the epidemiology, clinical features and laboratory diagnosis of dengue fever.

For general properties of arboviruses and list of arboviruses.

Refer Question No. 1, Oct. 2001 Paper II.

Dengue Fever

Dengue fever is an arboviral disease caused by dengue virus.

Dengue Virus

Dengue virus is an arbovirus belonging to group B or flaviviruses which is cubic in symmetry with diameter of 40–50 nm. It contains ssRNA of molecular weight. 3.9×10^6.

Pathogenicity

Dengue virus is transmitted by the Aedes aegypti mosquitoes. Man is the only vertebrate host.

The dengue virus causes three clinical manifestations of the dengue.

a. *Classical dengue fever or break bone fever*: It is the predominant type often seen in adults. It presents with a sudden fever, headache, retrobulbar pain, pain in the back and limbs, lymphdenopathy and maculopapular rash. The incubation period is 5–8 days and the fever is typically biphasic and lasts for 5–7 days followed as a rule by recovery.
b. *Dengue hemorrhagic fever and Dengue shock syndrome*: It remains confined among indigenous children of 6–8 years age group in area where multiple dengue viruses cause disease. Hemorrhagic dengue appears to be a hyperimmune response. It is seen in patients previously infected with dengue virus. On reinsertion with a different strain of virus within a period of 2 years of first infection antibodies formed against the first virus reacts with the second subtype forming immune complexes.

   Initial symptoms are like classical dengue fever but hemorrhagic fever is associated with hemorrhagic rash and thrombocytopenia and shock syndrome is associated with shock. The mortality rate is 5–10%.

Laboratory Diagnosis

Specimen

Blood collected during acute phase of illness.

Diagnosis is established by virus isolation and serology.

a. *Virus isolation*: The virus is isolated in tissue culture or eggs. Isolate is identified by hemagglutination inhibition, complement fixation, gel precipitation, immunofluoresence or neutralization with appropriate antisera.

   Virus isolation from the Aedes aegypti and monkeys, squirrels and other forest animal also aids the identification of the dengue virus.

b. *Serology*: Serological diagnosis is made by demonstrating rise in antibody titer in paired serum samples by hemagglutination inhibition, complement fixation or neutralization tests.

2. Name the different plasmodia that infect man. Describe the life cycle in man, clinical features and laboratory diagnosis of *P. falciparum* infection.

Plasmodia infecting humans

i. *Plasmodium vivax*
ii. *Plasmodium malariae*
iii. *Plasmodium falciparum*
iv. *Plasmodium ovale*.

Plasmodium falciparum

It is the parasite of malignant tertian malaria. The name falciparum is derived from its sickle shaped gametocytes.

Life Cycle

The *Plasmodium falciparum* passes its life cycle in two hosts.

In man : Man is the intermediate host where the parasite resides in the liver cells and red blood cells and reproduces asexually.

In female anopheles mosquito : Female anopheles mosquito is the definitive host. The sexual forms or gametocytes developed in the human host are transferred to insect host where they develop further and transformed into sporozoites, the infective forms for human host.

Human Cycle

It starts with the introduction of sporozoites by the bite of an infected female anopheles mosquito.

Human cycle comprises of following stages.

a. *Pre-erythrocytic schizogony*: It occurs inside the parenchyma of hepatocytes. It lasts for 6 days.

The mature pre-erythrocytic schizont measures 69 $\mu \times 30$ μm and contains numerous small merozoites, which are liberated on the 7th day in the circulation.

These merozoites enter the red blood cells.

b. *Erythrocytic schizogony*: *Pl. falciparum* invades both the reticulocytes and erythrocytes. Schizogony occurs inside the capillaries of internal organs. The cycle of schizogony or asexual multiplication is completed in about 36–40 hours. More than one parasite can invade an erythrocyte.

In erythrocytic schizogony, the parasite passes through the stages of trophozoite, schizont and merozoite.

- *Trophozoite*: It is the early ring form measuring 1.25 to 1.5 μ in diameter, consists of fine and uniform cytoplasmic ring with a nucleus often projecting beyond the ring or lying outside the ring. The parasite often attaches itself to the margin or edge of the host cell. The pigment granules formed by the granules are dark brown or black in color. As soon as the pigment appears in the cytoplasm of the parasite and there is increase in the size of the ring. The red cell is filtered out by the capillaries of the internal organs.
- *Schizont*: As the growth continues, the nucleus divides into several masses. The cytoplasm also divides forming as many segment arranged around the central pigment mass. The mature schizont occupies two-third of the invaded erythrocyte.
- *Merozoites*: It measures 0.5 to 0.7 mm in diameter. There average number is 18 to 24 per schizont.

c. *Gametogony*: After erythrocytic schizogony for a certain period some of the merozoites gives rise to forms, which are capable of sexual function after leaving the man host. These are called gametocytes.

The gametocytes of the *Pl. falciparum* are sickle shaped and are called crescents. Gametogony occurs inside the capillaries of bone marrow and spleen. With the growth of gametocytes the erythrocytes gets stretched up. There are two types of gametocytes—the microgametocytes and the macrogametocytes.

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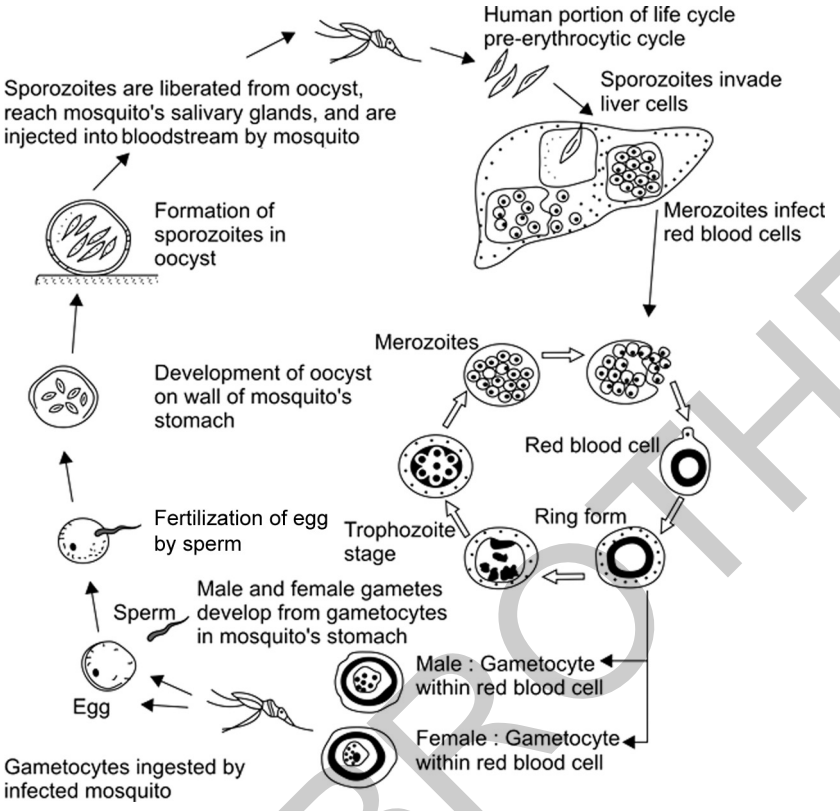

Fig. 1: *Plasmodium falciparum*—life cycle

Microgametocyte is broader; shorter with blunt ends 8–10 μm is size. Cytoplasm staining light blue, nucleus is scattered in fine granules over a wide area, pigments scattered. Macrogametocyte is longer, narrower, pointed ends of 10–12 μ by 2–3 μ, stains deep blue, nucleus condensed into a small compact mass into the center, pigments aggregates like wreath round the nucleus.

Mosquito Cycle

A female Anopheles mosquito during its meal from an infected person ingests both the sexual and asexual forms of parasite but it is only the mature sexual forms which are capable of development. The rest die off immediately. For a human host to infect the mosquito the blood of the human carrier must contain at least 12 gametocytes per mm^3 of blood and the number of female gametocytes must be in excess of the number of males.

The first phase of development occurs inside the stomach (mid gut) of the mosquito. From one microgametocyte 5 to 8 threads like filamentous structures, microgametes are developed by process of called as exflagellation. However, from one macrogametocyte only one macrogamete is formed.

Now one of the male gamete attaches to the periphery to the female gamete at the site of a small protrusion and penetrates inside the body. Fusion takes place between the male and female pronuclei and the resulting body is called a zygote. It takes about 20 minutes to 3 hours for formation of zygote after the mosquito's blood meal.

In the next 24 hours, the zygote lengthens and matures into an ookinate. This ookinate enters the mucosal cell of the gut of the mosquito to come to lie against external border of the cell and basement membrane. Here, it develops into an oocyst.

The oocyst is a spherical mass surrounded by a structureless capsule measuring 6–12 μ in diameter. It contains single vesicular nucleus and pigment granules of the macrogamete. The oocyst then matures, increasing in size from 6 to 60 μ and meiotic and mitotic divisions follow to form a large number of haploid sporozoites varying from a few hundred to thousands. When fully mature on about 10th day of infection, the oocyst ruptures releasing these sporozoites. The sporozoites are

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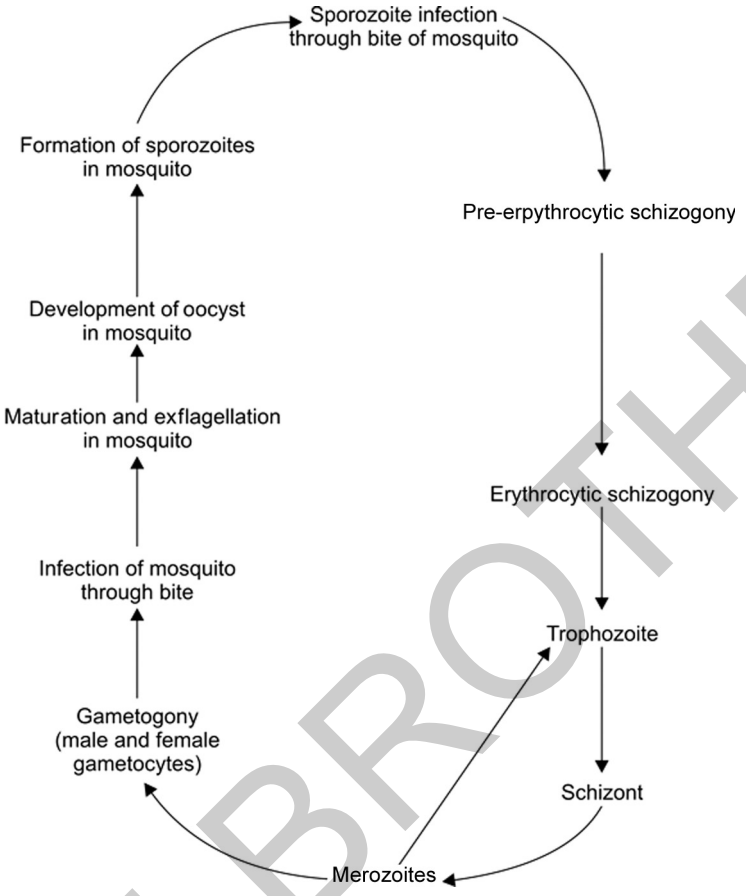

Fig. 2: *Plasmodium falciparum*—life cycle summary

distributed through circulations into various organs and tissue of the mosquito. They have a special predilection towards the salivary gland and ultimately reach a maximum concentration in the ducts. The mosquito at this stage is capable of transmitting infection to man.

During its blood meal, the mosquito releases the sporozoites into the human circulation thus completing the life cycle of *Pl. falciparum*.

Pathogenesis

The average incubation period of falciparum is 12 days. The typical picture of malaria consists of periodic bouts of fever with rigor followed by anemia and splenomegaly.

The febrile paroxysm comprises three successive stages. In the cold stage lasting for 15–60 minutes, the patient experiences intense cold and uncontrollable shivering. This is followed by the hot stage lasting for 2–6 hours when the patients feel intensely hot. The fever mounts to 41°C or higher. Severe headache, nausea and vomiting are common. This is followed by the sweating stage when the patient is drenched in profuse sweat. The temperature drops rapidly and the patient usually falls into deep sleep to wake up refreshed. This paroxysm usually begins in the early afternoon and lasts for 8–12 hours.

All these clinical manifestations of malaria are due to products of erythrocytic schizogony and the host's reaction to them. The febrile paroxysm follows the completion of erythrocytic schizogony when the mature schizont ruptures releasing red cell fragments, merozoites, malarial pigment and other parasitic debris. Macrophages and polymorphs phagocytose these and release large quantities of endogenous pyrogens leading to elevation of temperature cytokines such as tumor necrosis factor and interleukin 1 may play a pivotal role in the pathogenesis of malarial fever.

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Laboratory Diagnosis: (Pernicious Malaria)

a. *Blood examination*: The most important method for diagnosis of malaria is demonstration of parasite in the blood.

Two types of blood films are prepared for examination the thick and the thin films. These films are examined under oil immersion microscope for detecting the parasites or their gametocytes.

A slide is rated quantitatively as follows:

1–10 parasites per 100 thick film fields	+
11–100 parasites per 100 thick film fields	++
1–10 parasites per each thick film fields	+++
More the 10 parasites per each thick film fields	++++

About 200 oil immersion fields should be examined before declaring a thick film negative.

The thin film is used for identification of the species as it preserves the morphology of the parasite.

b. *Serological tests*: These are employed for the diagnosis in the acute phase of disease but for serological surveys. The commonly employed tests are Henry's melanin flocculation test, complement fixation test, passive hemagglutination tests, immunofluoresence test, ELISA, RIA, etc.

SHORT ESSAYS

3. Specific tests for HIV infections.

Refer Question No. 1, April 2007 (RS2) Paper II.

4. Viral transformation of cells.

- Transformation is the introduction of inheritable changes in a cell causing changes in the growth phenotype and immortalization
- Viral transformation of cells is responsible for about 15% of human cancers.

Examples

	Virus	*Cancer*
i.	Human papilloma virus	Cervical, vulvar, penile cancers Squamous cell carcinoma
ii.	Ebstein-Barr virus	Nasopharyngeal carcinoma African Burkitt's lymphoma
iii.	Herpes simplex virus type 2	B cell lymphoma Cervical lymphoma
iv.	Hepatitis B virus	Hepatocellular carcinoma
v.	Hepatitis C virus	Hepatocellular carcinoma
vi.	HTL virus	Adult T cell leukemia

Mechanism of Viral Transformation

- The frequency of infection by agents such as EBV, HBV or HTLV-1 are far greater than the incidence of the cancers with which they are linked
- Viruses may act in concert with other factors like genetic, immunological or environmental in the evolution of cancer
- Cell transformation by viruses is accompanied by the persistence of all or a part of the viral genome and the continual expression of a limited number of viral genes
- Viral oncogenes disrupt the normal cellular gene expression and signal transduction pathways
- The signal transduction pathway is responsible for altering cellular gene expression in response to a wide range of external and internal signals
- Viral oncogenes disrupt the normal functioning by virtue of altered structures or overproduction. The end result is an altered transcriptional profile, which favors cell division.

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- This can be divided this into 4 broad groups, with many known examples of oncogenes for each grouping:
  i. Cell surface where receptors interact with growth factors and components of the extracellular matrix.
  ii. Transmembrane signaling apparatus including the cytoplasmic domain of receptors and submembranous components that are functionally linked to surface receptors conveying signals from the outside of the cell to the interior.
  iii. Cytosolic elements, i.e. soluble proteins and second messengers.
  iv. Nuclear proteins including DNA binding proteins and factors which interact directly and indirectly in gene regulation and replication.

(Ref: http://www.tulane.edu/~dmsander/WWW/224/Trans224.html on 18th Jan 2008)

5. Type C hepatitis.

- Type C hepatitis is also known as parenterally transmitted non-A, non-B hepatitis and non-B transfusion associated hepatitis.

Morphology

- Hepatitis C virus is a small virus with a linear single stranded RNA enclosed within a core and surrounded by an envelope, carrying glycoprotein spikes
- The virus core particle measures 33 nm in diameter and intact virion is about 55–65 nm with spike like projections
- The genome of the virus encodes for four structural proteins namely core, E1, E2 type A and E2 type B and six nonstructural proteins.

Mode of Infection

- Most common methods of transmission are percutaneous exposure to blood and plasma derivatives, use of contaminated needles and syringes, needle stick injury, sharing of needles by IV drug abusers, tattooing and skin piercing.

Pathogenesis

- The average incubation period is about 150–160 days with mean of 50 days after infection
- The acute illness is usually mild and anicteric with fatigue as only symptom
- A common feature of HCV infection is episodic, fluctuating pattern of SGPT activity
- The striking feature of hepatitis C infection is its tendency towards chronicity and slowly progressive liver damage
- The disease remains clinically silent for years or even decades
- 50–80% patients develop chronic hepatitis with some developing cirrhosis and hepatocellular carcinoma
- Factors determining progression of chronic hepatitis to cirrhosis
  - Patients age at exposure
  - Duration of liver damage
  - Level of liver damage at initial exposure
  - Host immunity
  - Alcohol intake
  - Concomitant infection with HIV, hepatitis B or D virus.

Laboratory Diagnosis

Serological and virological markers for HCV infection are:

a. *Anti-HCV antibodies*: Three generations of anti-HCV IgG assays are available:
   i. Antibodies to C100-3 region: It appears 1 to 3 months after infection.
   ii. Antibodies to C33c region: It appears one month earlier than antibodies to C100-3 region.
   iii. Antibodies to NS-5 region: It is detected even earlier.

b. *HCV/RNA*: HCV infection is confirmed by HCV/RNA employing PCR technique which can be detected within a few days after exposure to HCV infection much before appearance of anti-HCV and persists for the duration of HCV infection.

c. *Serum enzymes*: Serum transaminases, SGPT in particular are raised in the early stage of the disease to 100–2,000 IU/L.

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6. Clinical features and prevention of varicella.

Refer Question No. 4, April 2008 (RS2) Paper II.

7. Serological tests for toxoplasmosis.

Refer Question No. 2, April 2011 (RS2) Paper II.

8. Clinical features and diagnosis of cysticercus cellulosae.

Refer Question No. 2, Oct. 2014 (RS2) Paper II.

9. Pathogenesis of intestinal amebiasis.

Refer Question No. 1, Oct. 2010 (RS2) Paper II.

10. Larva migrans.

Larva migrans describes a parasitic disease involving migration of immature (larval) worms in various parts of the body.

Types

i. Cutaneous larva migrans: When the worms migrate through the skin of the host.
ii. Visceral larva migrans: If the worm larvae migrate through various internal organs of the host.
iii. Ocular larva migrans: Sometimes the worm larvae may invade the eye of the host.

a. *Cutaneous Larva Migrans*
- Cutaneous larva migrans or creeping eruptions or ground itch is a skin disease in humans.

Cause
- It is caused by the larvae of various nematode parasites, most common parasite is Ancylostoma.

Pathogenesis
- These parasites are usually found in dog and cat feces and although they are able to infect the deeper tissues of these animals (through to the lungs and then the intestinal tract), in humans they are only able to penetrate the outer layers of the skin and thus create the typical wormlike burrows visible underneath the skin.
- The parasites apparently lack the collagenase enzymes required to penetrate through the basement membrane deeper into the skin.

Clinical Features
- The infection causes a red, intense itching eruption. The itching can become very painful and if scratched may allow a secondary bacterial infection to develop.

Treatment
- Albendazole and ivermectin orally
- Thiabendazole topically or taken by mouth
- Ethyl chloride or liquid nitrogen, topical freezing agents, applied locally can freeze and kill the larvae.

b. *Visceral Larva Migrans*
- Visceral larva migrans is a condition in children caused by the migratory larvae of nematodes.

Cause
- It is caused by nonhuman species of nematodes like Neoascaris, Toxocara, etc.

Pathogenesis
- These nematodes cannot reproduce in humans, thus they wander throughout the body invading the heart, and the CNS inducing granulomatous lesion causing local damage.

Clinical Features
- Fever, hepatomegaly, pneumonitis, hyperglobulinemia and pica are common findings
- Neurological disturbances and endophthalmitis may develop as complications
- Marked leukocytosis occurs with high eosinophilia.

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c. *Ocular Larva Migrans*
   – Ocular larva migrans is the ocular form of larva migrans.

   Cause
   – Toxocara canis

   Pathogenesis
   – When Toxocara canis invade other body tissues, it may also invade the eye producing ocular larva migrans.

   Clinical Features
   – Unilateral visual disturbances, strabismus and eye pain
   – Eosinophilic granulomatous mass developing around the entrapped larva, most commonly in posterior pole of retina.

Ref:
http://www.cfsph.iastate.edu/FastFacts/pdfs/larvamigrans_F.pdf 28th July 2008
http://en.wikipedia.org/wiki/Cutaneous_larva_migrans 28th July 2008
http://en.wikipedia.org/wiki/Visceral_larva_migrans 28th July 2008
http://en.wikipedia.org/wiki/Ocular_larva_migrans 28th July 2008.

11. Fungi imperfecti.

- Fungi imperfecti or deuteromycota are the fungi which have lost their ability to reproduce sexually or their sexual state is not discovered
- These fungi do not fit into the commonly established taxonomic classifications of fungi that are based on biological species concepts or morphological characteristics of sexual structures, because their sexual form of reproduction has never been observed; hence the name "imperfect fungi"
- Only their asexual form of reproduction is known
- This group of fungus produces their spores asexually
- This is a temporary phylum and the fungi place in this phylum whose sexual sporulation is discovered, is transferred to a suitable position in any of the phyla.

Classification (Kendrick – 1981)

- They are further classified on the basis of morphology and ontogeny of the conidia and conidiogenous cells.
- Thus, there are three classes:

a. Class Hyphomycetes—lacking fruiting bodies
   i. Order Moniliales (producing spores on simple conidiophores)
   ii. Order Stilbeliales (producing spores on synemmata)
   iii. Order Tuberculariales (producing spores in sporodochia).
b. Class Coelomycetes—spores produced in fruiting bodies
   i. Order Melanconiales (producing spores in acervuli)
   ii. Order Sphaeropsidales (producing spores in pycnidia).
c. Class Agonomycetales—lacking spores.

Importance

- Most fungi of medical importance belong to this group.

(Ref: http://en.wikipedia.org/wiki/Deuteromycota 28th July 2008).

12. Lepromin test.

Lepromin test is a skin test for detecting delayed hypersensitivity in cases of leprosy.

It was described by Mitsuda in 1919 and till recently was only method for studying the immunity.

The antigen injected originally was lepromin, the oiled, emulsified, lepramatous tissue rich in lepra bacilli. The modern antigens are standardized according to their lepra bacilli content (4×10^7 lepra bacilli per mL) and are prepared from armadillo derived lepra bacilli (lepromin-A).

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About 0.1 mL of lepromin is injected intradermally to an individual. The response to the lepromin is typically biphasic, consisting of two separate events, early and delayed reaction.

a. *Early reaction of Fernandez*: It is the erythema and induration at the site of inoculation developing in 24–48 hours and usually remaining for 3–5 days. It is analogous to the tuberculin reaction, is poorly defined and carries little significance.
b. *Late or delayed reaction of Mitsuda*: It consists of an indurated skin nodule of 3–5 mm diameter which may ulcerate, at the site of inoculation. It starts in 1–2 weeks, reaching a peak in 4 weeks and gradually subsiding in the next few weeks. Histologically, there is infiltration of lymphocytes, epithelioid cells and giant cells.

Mitsuda late reaction is a measure of the cell mediated immunity induced by the injected lepromin itself, i.e. it indicates resistance to leprosy. The test is negative in lepramatous leprosy but positive in tuberculoid leprosy. Thus, it distinguishes between person who can mount a cell mediated immunity response against the lepra bacillus antigens and those who cannot.

Significance of Lepromin Test

The lepromin test is not used for diagnosing leprosy nor does it indicate prior contact with the lepra bacillus. In nonendemic areas, a healthy person with no chance of contact with the bacillus may give a positive lepromin test.

The test is employed

i. *To classify the lesions of leprosy patients*: The lepromin test is positive in tuberculoid, negative in lepramatous and variable in dimorphous and indeterminate type of diseases.
ii. *To access the prognosis and response to treatment*: A positive reaction indicates good prognosis and a negative one bad prognosis. During treatment, conversion to lepromin positivity is evidence of improvement.
iii. *To access the resistance of individual to leprosy*: It is desirable to recruit only lepromin positive persons for work in leprosy asylums.
iv. To verify the identity of candidate lepra bacilli.

SHORT ANSWERS

13. Tissue culture.

Refer Question No. 1, Oct. 2006 Paper II.

14. Phage typing.

Refer Question No. 8, April 2009 (RS2) Paper II.

15. Serological tests for infectious mononucleosis.

Refer Question No. 4, April 2009 (RS2) Paper II.

16. Congenital rubella syndrome.

Refer Question No. 9, April 2003 Paper II.

17. Primary amebic meningoencephalitis.

Refer Question No. 9, Oct. 2013 (RS2) Paper II.

18. Cyst of *Giardia intestinalis.*

Refer Question No. 2, April 2013 (RS2) Paper II.

19. Occult filariasis.

Refer Question No. 8 April 2013 Paper II.

20. Parasitic infections and malignancy.

Several parasites have been linked to certain malignancies through mechanisms similar to that in chronic bacterial inflammation.

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- *Clonorchi sinensis* and *Opisthorchis viverrini* have been linked to cholangiocarcinoma
- *Fasciola hepatica* also demonstrated an enhanced mutagenic effect when infection occurred in the presence of exogenous carcinogens
- Many eggs of schistosomes do not escape in the feces but form granulomas in the intestine, liver, lung, and urinary bladder. Their egg antigen extracts have transforming carcinogenic properties
- *Schistosoma haematobium* is linked to squamous cell carcinoma of the bladder and cancer of the venous plexus
- *S. japonicum* may induce hepatocellular carcinoma.
  (Ref: http://www.medscape.com/viewarticle/417716_3 on 27th July 2008).

21. Sporotrichosis.

Refer Question No. 4, April 2012 (RS2) Paper II.

22. Athlete's foot.

- Athlete's foot or Tinea pedis is the ringworm infection of foot particularly of the interdigital webs and soles
- It is common in temperate countries where every one wears footwear and is rare in tropical countries where people walk barefeet.

Causative Agents

- *Trichophyton rubrum*
- *Epidermophyton floccosum*.

Clinical Features

- Web scaling, fissuring, maceration and erythema of foot may be associated with an itching or burning sensation
- The small vesicles rupture and discharge a thin fluid
- Due to maceration, peeling, cracks are prone to secondary bacterial infection
- In chronic infection, sole becomes hyperkeratotic and often covered with fine scales.

Laboratory Diagnosis

- Direct microscopic examination of skin scrapping from lesions in KOH wet mounts shows few long pencil shaped macroconidia (*T. rubrum*) or club shaped macroconidia in clusters (*E. floccosum*)
- Culture on Sabouraud's medium incubated at room temperature shows velvety colonies (*T. rubrum*) or yellowish green powdery colonies (*E. floccosum*).

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