

Leptospirosis – An Overview

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Abstract

One of the important causes of acute febrile illness in a country where malaria, typhoid and dengue are also not uncommon, leptospirosis, a zoonotic disease spread by rodents, is endemic in Tamil Nadu, Kerala and Andamans; and is now being increasingly reported from other parts of India, perhaps with better facility to diagnose the disease. Disease of profound importance in view of its grave outcome, in its icteric form (Weil's disease), may have a mortality of as high as 40%. Worst prognosticator is the presence of multi-organ failure (MOF), as in any other septicemia. Andaman hemorrhagic fever (AHF), a type peculiar to Andamans, is now being described elsewhere in the country also. IgM ELISA, Dot-ELISA, dip-stick method and slide agglutination test (SAT) are newer screening methods for diagnosis of leptospirosis, but are only genus-specific. Identifying specific serovar is possible by Micro-agglutination test (MAT) and culture method only. Anicteric type of disease, however, is easily treatable with penicillin and has a good prognosis. Oral doxycycline can be used for prophylaxis during the risk of exposure. ©

HISTORY

Leptospirosis is a zoonotic disease caused by pathogenic spirochetes of the genus *Leptospira*. In 1907, Stimson described the micro-organism in renal tubules of a patient who died of so-called yellow fever. The spirochete was first isolated in Japan by Inada and co-workers in 1915, nearly 30 years after Weil described the clinical disease in 1886¹. Its relatively recent discovery belies the long history of leptospirosis, which was probably known much earlier in China and Japan by names such as "rich harvest jaundice" and "autumn fever". The genus *Leptospira* contains both pathogenic and nonpathogenic strains.

SPECIES

Genus *Leptospira* is broadly divided into two species: *Leptospira interrogans* comprising all pathogenic strains, and *Leptospira biflexa*, containing the saprophytic (non-pathogenic) strains isolated from the environment. Species *Leptospira interrogans*, comprises of at least 250 antigenically distinct variants known as serovars belonging to 23 serogroups. Identification and classification of species of *Leptospira* is important because of different host specialities.

This disease has been described as the most common zoonosis affecting many species of wild and domestic

animals such as rodents, livestock, wild mammals, dogs and cats. The most frequent hosts are rodents, especially the common rat (*Rattus norvegicus*). In this and other reservoir species, the organisms persist indefinitely in the convoluted tubules of the kidney without causing apparent disease, and are shed into the urine in massive numbers. Particular serovars are associated with characteristic animal hosts; e.g., *L. icterohemorrhagiae* / *copenhageni* is the classical parasite of rats, *L. grippityphosa* of voles, *L. canicola* of dogs, *L. hardjo* of cattle and *L. pomona* of pigs.²

EPIDEMIOLOGY

Leptospirosis is an infectious disease of worldwide distribution. Human infection can occur either through direct contact with infected animals or, much more commonly through indirect contact with water or soil contaminated by urine of infected rodents or animals.³ Person-to-person transmission is extremely rare since man is a dead-end host for leptospiral dissemination. In contrast, leptospire can survive for long periods in the renal tubules of infected animals without causing illness. Most human infections occur in young adult men and children and result from occupational or environmental exposure.³ Epidemiological studies indicate that infection is commonly associated with certain occupational workers such as farmer, sewage worker, veterinarian, and animal handler. Leptospirosis can also be transmitted during recreational activities such as hiking, picnicking, swimming and canoeing.⁴⁻⁶

Leptospire can survive in untreated water for months or years, but cannot survive desiccation or salt water.

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Only sporadic cases of leptospirosis are seen in arid climates and deserts. In comparison, the disease is endemic in tropical areas, areas with heavy precipitation, and areas with high levels of subsurface water. Hence, China, Southeast Asia, Africa, and South and Central America have immense areas where the disease is endemic. Leptospirosis occurs sporadically throughout the year in these areas, with a peak seasonal incidence in summer. Large epidemics are reported after monsoons and periods of unusually heavy rainfall. In India, Kerala, Tamil Nadu and Andamans are endemic for leptospirosis. But now with better facilities to detect the disease, the disease is being reported from almost all parts of India.⁷⁻¹²

In a study conducted all over the country by National Reference Centre, Regional Medical Research Centre (ICMR), Port Blair during the period 2000–2001, a seropositivity rate ranging from 0 to 46.8 % amongst all cases of fever was observed from various parts of India.¹³ The positivity rate was highest in South India at 25.6%. It was 8.3%, 3.5%, 3.1% and 3.3% in northern, western, eastern and central India respectively.¹⁴ Cases of leptospirosis were reported through out the year at the majority of hospitals. 32% of urban slum children in Mumbai had acute leptospirosis during monsoon in one of the studies.¹⁵

Several outbreaks of leptospirosis have occurred lately. An outbreak emerged in Orissa following super cyclone in 1999.¹²

PATHOLOGY AND PATHOGENESIS

Leptospire (from the Greek leptos, fine and spira, a coil) are finely coiled, filamentous spirochetes measuring 6-20 mm in length and 0.1mm in width with characteristic curved or hooked ends. Human infection occurs by direct contact with urine or blood of an infected rodent or animal, or from water, soil or vegetable contaminated by urine. The organisms can penetrate abraded skin or intact mucous membrane, after which they enter the circulation and rapidly disseminate to various tissues.

There are two distinct phases of leptospiral infection in the body – first, septicemic phase and second, immune phase.

The pathogenesis of leptospirosis is not fully understood. In the septicemic animal models, vascular injury is seen in various organs.¹⁶ Spirochetes can be found in the walls of capillaries and medium- and large sized vessels. The exact mechanism of vascular damage is not clear. A direct toxic effect of the leptospire has been proposed to cause the vascular injury, but no bacterial endotoxin has been demonstrated.

In the immune (second) phase of illness, the host immune response, including immune complex deposition, may play a role in endothelial injury.

During septicemic phase, invading leptospire are

distributed throughout the body. Penetration and invasion of tissues is presumably accomplished through a burrowing motion produced by a pair of axial filaments and release of hyaluronidase.¹⁷ The dissemination and proliferation of spirochetes in various tissues results in a systemic illness, which has a broad spectrum of clinical manifestation. Generalised petechiae and ecchymosis occur in most internal organs in a severe case. Main organs affected are kidney, liver, brain and meninges. Microscopically, a systemic vasculitis with endothelial injury is seen. The damaged endothelial cells usually show different degrees of swelling, necrosis and denudation. The main histopathologic changes are usually found in the liver,¹⁸ kidney, heart and lungs. Hepatic lesions include mild degenerative changes in hepatocytes, prominent hypertrophy and hyperplasia of Kupfer cells, erythrophagocytosis and cholestasis. Focal necrosis with occasional acidophilic bodies may occur, but there is no particular zonal distribution associated with the necrosis. Mild to moderate mononuclear cell infiltrates are present in portal tracts. In the kidney, the main histopathologic feature is diffuse tubulointerstitial inflammation characterized by cellular infiltration with lymphocytes, plasma cells, macrophages and polymorphonuclear leucocytes. Tubular necrosis is also a common finding. Glomeruli show mild hyperplasia of mesangial cells and occasional infiltration with inflammatory cells. Grossly the lungs are heavy and severely congested, with focal areas of hemorrhage. Microscopically the lungs show congestion with foci of intraalveolar hemorrhage. In some cases pulmonary lesions include diffuse alveolar damage and variable degrees of airspace disorganization.

CLINICAL MANIFESTATIONS

In its mild form, leptospirosis may present as an influenza-like illness with headache and myalgia. Severe leptospirosis, characterized by jaundice, renal dysfunction, and hemorrhagic diathesis, is referred to as Weil's syndrome.

Incubation Period: 2-26 days (usually 7-12 days)

In general, clinical manifestation can be divided into two distinct clinical syndromes. 90% of patients present with mild anicteric febrile illness; 10% are severely ill with jaundice and other manifestations (Weil's disease). Both anicteric and icteric leptospirosis may follow a biphasic course (Table 1).^{19,20}

Anicteric leptospirosis is the more common and milder form of the disease, and is often biphasic. In the first or septicemic phase, patients usually present with an abrupt onset of fever, chills, headache, myalgia, skin rash, nausea, vomiting, conjunctival suffusion, and prostration. Leptospire can be isolated from blood, cerebrospinal fluid (CSF), and tissues. The fever may be high and remittent reaching a peak of 40°C before defervescence. Conjunctival suffusion is characteristic

Table 1 : Leptospirosis: Anicteric and Icteric

LEPTOSPIROSIS:	ANICTERIC		ICTERIC	
	First stage 3-7 days (Septicemic)	Second stage 0-30 days (Immune)	First stage 3-7 days (Septicemic)	Second stage 0-30 days (Immune)
Clinical Findings	Fever, myalgia, headache, conjunctival suffusion, abdominal pain, vomiting	Meningitis, uveitis, rash, fever	High fever Jaundice Hemorrhages Renal failure	
Leptospire Present	Blood CSF	Urine	Blood CSF	Urine

and usually appears on the third or fourth day. Myalgias usually involve the muscles in the calf, abdomen, and paraspinal region and can be severe. When present in the neck, myalgias may cause nuchal rigidity reminiscent of meningitis. In the abdomen, myalgia may mimic acute abdomen, leading to confusion with surgical intra-abdominal emergencies. The skin manifestations seen in mild leptospirosis include transient urticarial, macular or maculopapular, erythematous or purpuric rash.²¹ The first phase lasts 3-9 days followed by 2-3 days of defervescence, after which the second or “immune” phase develops.

The immune phase, is characterized by leptospiruria and correlates with the appearance of IgM antibodies in the serum. Leptospira now settle in glomeruli and are eliminated from all sites in the host except eye and perhaps brain, where they may persist for weeks or months. Fever and earlier constitutional symptoms recur in some patients, and signs of meningitis, such as headache, photophobia, and nuchal rigidity may develop. Central nervous system (CNS) involvement in leptospirosis most commonly occurs as aseptic meningitis. Complications such as optic neuritis, uveitis, iridocyclitis, chorioretinitis, and peripheral neuropathy occur more frequently in the immune phase. Prolonged or recurrent uveitis was demonstrated in 2% of patients with onset several months after symptoms of clinical leptospirosis.²² A rare but severe manifestation is hemorrhagic pneumonia. The illness in anicteric leptospirosis may be self-limited, lasting 4-30 days, with complete recovery as a rule.

In icteric leptospirosis (Weil’s syndrome) (usually caused by *L. icterohaemorrhagiae*), persistent high fever and jaundice may obscure the two phases. This is usually associated with hepatic dysfunction, renal insufficiency, hemorrhage and multi-organ failure (MOF). Hemorrhage can occur as petechiae, purpura, conjunctival hemorrhage and gastrointestinal hemorrhage. MOF is associated with a very high mortality. Myocarditis and hemorrhagic pulmonary infiltration are other complications, which may prove fatal.

PULMONARY SYNDROME

An exclusively pulmonary syndrome has long been recognized in the Far East. The syndrome is characterized

by hemoptysis, patchy lung infiltrates on chest X-ray, and respiratory failure. Total bilateral lung consolidation and the ARDS develop in fatal cases. Almost with similar presentation and high mortality, leptospirosis with early pulmonary symptoms including hemoptysis and other hemorrhagic manifestations in Andamans was called as *Andaman hemorrhagic fever*. It was long considered to be a viral hemorrhagic fever, before leptospira as etiological agent was identified in 1993.²³ Severe pulmonary hemorrhage has also been described in China, Korea and Nicaragua, where patients died due to this. We too had several instances of pulmonary involvement, and one with hemorrhagic pleural effusion with ARF, which required hemodialysis.^{13,24}

UNCOMMON MANIFESTATIONS

Less common manifestation of leptospirosis is hemorrhage with no significant renal dysfunction or jaundice, when it may be mistaken for dengue hemorrhagic fever. Other uncommon manifestations are generalized lymphadenopathy, pharyngitis and acalculous cholecystitis.

Table 2 depicts our experience with leptospirosis. An analysis of 33 patients of icteric leptospirosis in a study is presented.

INVESTIGATIONS

Most of the routine laboratory tests show nonspecific findings. The leucocyte count can be low, normal or elevated, but is usually associated with a shift to left. Mild anemia and thrombocytopenia are common; hemolytic anemia and disseminated intravascular coagulopathy (DIC) have been described in severe cases. Thrombocytopenia is found in more than 50% of patients and is significantly associated with renal failure. Liver, kidney and central nervous system involvement may be present in any combination. Liver involvement may be mild, or severe with bilirubin levels reaching very high.

Serum alkaline phosphatase, lactate dehydrogenase, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) may all be elevated, but serum AST is only mildly or moderately elevated (unlike in viral hepatitis). Hyperamylasemia occurs frequently in severe disease, but pancreatitis is rare. Serum creatinine phosphokinase (CPK) is usually raised due to muscular

Table 2 : Icteric Leptospirosis - Analysis of 33 patients

<p>Bleeding Manifestations Major bleed (melena, hematuria, retinal hemorrhage) - 3 Minor bleed (subconjunctival bleed) - 6 Isolated hemoptysis - 1</p> <p>Hematological Thrombocytopenia - 20 Pancytopenia - 2 Prothrombin time ↑ - 14 Coomb's +ve Hemolytic Anemia - 1 Disseminated intravascular coagulopathy (DIC) - 1</p> <p>Central Nervous System Altered sensorium - 22 Neck stiffness - 12 Deep coma - 4 Left sided hemiplegia and anisocoria - 1 Generalized tonic & clonic seizure - 1 Subarachnoid hemorrhage - 1 Aphemia (speech apraxia) - 1</p>	<p>Pulmonary involvement Pleural effusion - 4 ARDS - 1 Empyema - 1 Pneumonia and hemoptysis - 1</p> <p>Major hepatic findings Bilirubin - <i>Total</i> : 5.7mg/dl (mean) (highest - 27 mg/dl) - <i>Direct</i> : 2.8 mg/dl (mean) (highest - 11 mg/dl) Isolated hepatomegaly - 7 Liver span ↓ - 4 Flapping tremor - 3 Ascites - 2 MOF - 20</p> <p>DEATH - 13</p>
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involvement.¹⁹

Proteinuria, pyuria, hematuria, and hyaline or granular casts are common findings on urine analysis even in the absence of renal dysfunction. Renal functional impairment is primarily a result of tubular damage; however, hypovolemia may play a critical role in the subsequent development of renal insufficiency.

In the cerebrospinal fluid (CSF), features are those of aseptic meningitis. The CSF protein level may be normal or elevated upto 300 mg/dl, while the glucose concentration is normal. Although abnormal CSF findings are reported in as many as 80% of leptospirosis cases, only half of the patients are symptomatic.

Various electrocardiographic and chest X-ray abnormalities are observed in patients with leptospirosis. Arrhythmias due to significant cardiac irritability have been documented. The arrhythmias observed were atrial fibrillation, atrial flutter, atrial tachycardia and premature ventricular contractions. Echocardiograms may reveal pericarditis and small pericardial effusion, in few cases.

The chest radiographic abnormalities can include pulmonary edema, diffuse pneumonitis, nonsegmental or basal linear opacities and pleural effusions.

Specific Diagnosis: (Table 3)

Definitive diagnosis of leptospirosis depends on:^{19,20}

- (i) Isolation of organism
- (ii) Serological tests
- (iii) Detection of specific DNA

Isolation of organism

- (i) **Blood** : The organism may be identified by dark field examination of the patient's blood or by culture on a semisolid medium (eg. Fletcher's EMJH i.e. Ellinghausen-McCullough-Johnson-Harris), if taken

Table 3 : Diagnosis of Leptospirosis

<p>Isolation of organism</p> <p>1. Before tenth day of illness:</p> <p>Blood -</p> <ul style="list-style-type: none"> i. Dark field examination of the patient's blood ii. Culture on a semisolid medium (eg. Fletcher's EMJH) <p>2. After tenth day of illness:</p> <p>Urine -</p> <ul style="list-style-type: none"> i. Dark field examination of the patient's urine ii. Culture of urine (for several months in untreated patient) <p>Serology</p> <p>Agglutination tests : Paired sera (fourfold or greater rise in titer)</p> <ul style="list-style-type: none"> i. Microscopic, using live organisms (MAT) ii. Macroscopic, using killed antigen <p>ELISA IgM and Slide agglutination tests (SAT) :</p> <ul style="list-style-type: none"> - Measure IgM antibodies - Single sample adequate - The ELISA IgM test helpful for early diagnosis (positive 2 days into illness) <p>Dot-ELISA and dip-stick methods:</p> <ul style="list-style-type: none"> - Newer screening methods (for detecting IgM antibodies) <p>PCR test</p> <p>Leptospiral DNA:</p> <ul style="list-style-type: none"> - Detected in blood, urine, CSF, and aqueous humor
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• Specific serovar is detected only by microscopic agglutination test (MAT) and culture isolation

before the tenth day of illness. Cultures take 1-6 weeks to become positive.

- (ii) **Urine** : The organism may be isolated from the urine on darkground microscopy tenth day onwards, and in untreated patient, may be recovered on urine culture for several months.

Serology

Diagnosis is usually made by means of serologic tests, of which several are available.

- (i) **Agglutination tests** (microscopic, using live

organisms; and macroscopic, using killed antigen) become positive after 7-10 days of illness, peak at 3-4 weeks, and may persist at high levels for many years. Thus, to make a diagnosis, a fourfold or greater rise in titer must be documented. The agglutination tests are cumbersome to perform and require trained personnel.

- (ii) ELISA IgM and slide agglutination tests (SAT) are also available.

As ELISA IgM and Slide agglutination tests (SAT) are simple, sensitive tests which measure IgM antibodies, they are used to diagnose current leptospirosis at a very early stage and a single sample is adequate. The IgM ELISA test is particularly useful in making an early diagnosis, as it is positive as early as 2 days into illness, a time when the clinical manifestation may be nonspecific. It was found to be 100% sensitive and 93% specific in one study.⁸ Dot-ELISA and dip-stick methods for detecting IgM antibodies are newer screening methods.

PCR test

PCR methods (presently investigational) appear to be sensitive, specific, positive early in disease, and able to detect leptospiral DNA in blood, urine, cerebrospinal fluid (CSF) and aqueous humor. Currently major disadvantage with these tests is that these are genus-specific, not serovar specific.

Specific Serovar

The investigations of choice to identify specific serovar is microscopic agglutination test (MAT) and culture isolation. However, culture growth may take several weeks.

WHO GUIDELINES: Faine's Criteria For Diagnosis Of Leptospirosis

Faine had evolved a criteria for diagnosis of leptospirosis on the basis of clinical, epidemiological and laboratory data (Parts A, B and C respectively)³ (Table 4).

A presumptive diagnosis of leptospirosis may be made if:

- (i) Parts A and B score = 26 or more (Part C laboratory report is usually not available before fifth day of illness; thus it is mainly a clinical and epidemiologic diagnosis during early part of disease)
- (ii) Part A+B+C = 25 or more

A score between 20 and 25 : Suggests a possible but unconfirmed diagnosis of leptospirosis

Shivakumar *et al* from Chennai have suggested modification on Faine's criteria to include local factor (like rainfall) and newer investigations in the total scoring. As per this, epidemiological and laboratory criteria (Parts B and C) are modified only; no modification is made in the clinical criteria (Part A) (Table 4).²⁵

Table 4

Faine's Criteria ³		Modified Faine's Criteria ²⁵	
Part A: Clinical data	Score	Part A: Clinical data	Score
Headache	2	Headache	2
Fever	2	Fever	2
If fever, temperature 39°C or more	2	If fever, temperature 39°C or more	2
Conjunctival suffusion (bilateral)	4	Conjunctival suffusion (bilateral)	4
Meningism	4	Meningism	4
Muscle pain (especially calf muscle)	4	Muscle pain (especially calf muscle)	4
Conjunctival suffusion + Meningism + Muscle pain	10	Conjunctival suffusion + Meningism + Muscle pain	10
Jaundice	1	Jaundice	1
Albuminuria or Nitrogen retention	2	Albuminuria or Nitrogen retention	2
Part B: Epidemiological factors		Part B: Epidemiological factors	
Contact with animals or contact with known contaminated water	10	Rainfall	5
		Contact with contaminated environment	4
		Animal contact	1
		Total	10
Part C: Bacteriological and Lab. findings		Part C: Bacteriological and Lab. findings	
Isolation of leptospire in culture – diagnosis certain		Isolation of leptospire in culture – diagnosis certain	
Positive serology (MAT)		Positive serology	
Leptospirosis endemic		ELISA IgM positive*	15
Single positive low titre	2	SAT positive*	15
Single positive high titre	10		
Leptospirosis non-endemic		MAT single high titre*	15
Single positive low titre	5	MAT rising titre (paired sera)	25
Single positive high titre	15		
Rising titre (paired sera)	25		
Total Score		Total Score	

*Any one of the tests only should be scored

The reasons for modification suggested are as follows:

Most of the leptospirosis is reported in monsoon and post-monsoon period. Therefore they have suggested rainfall separately to be adjusted in the scoring criteria of Part B.

Microscopic agglutination test (MAT) is the Gold Standard test, but it is complicated and less sensitive compared to newer tests like ELISA IgM and SAT. ELISA IgM and Slide agglutination tests (SAT) are simple, sensitive tests and can be used to diagnose current leptospirosis. Thus, they have been included with appropriate score.

The difficulties in utilizing MAT are due to the following reasons:

- (a) The antibody titres rise and peak only in second or third week. Thus, paired sera are required to demonstrate 4-fold rise of titre.
- (b) The test is complicated requiring dark-field microscopy and cultures of various serovars, which may not be available in smaller laboratories.

The advantage of including simple diagnostic tests (ELISA IgM or SAT) in modified Faine's criteria is that it helps in diagnosing milder forms of leptospirosis which are associated with low clinical score (Part A).

Suggestion of modification of existing Faine's criteria appears justified;⁷ however further evaluation is required.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of leptospirosis depends on the epidemiology of acute febrile illnesses in the particular area. A high index of suspicion is needed in endemic areas, and leptospirosis must be considered when a patient presents with acute onset of fever, headache and myalgia. However, in locations where dengue fever or malaria is also present, the differentiation may be very difficult because of similar clinical manifestations. Laboratory confirmation is crucial, especially when these diseases are occurring simultaneously during the rainy season. Other conditions to be considered in the differential diagnosis include influenza, meningitis (or encephalitis), viral hepatitis, rickettsiosis, typhoid fever, septicemia, toxoplasmosis, and Legionnaire's disease.

When the patient presents with jaundice during or after an acute febrile illness, leptospirosis must be differentiated from other causes of febrile jaundice such as malaria, septicemia, alcoholic hepatitis and typhoid hepatitis.

The high bilirubin level seen in Weil's disease with mild to modest elevation of transaminases assists in differentiating it from viral hepatitis, which has usually a much higher elevation of transaminases. Further, a high serum creatinine phosphokinase (CPK)

concentration or thrombocytopenia also favors a diagnosis of leptospirosis.¹⁹ Serum CPK is usually found normal in viral hepatitis.

TREATMENT

Various antimicrobial drugs, including penicillin and tetracyclines, show antileptospiral activity. Penicillin (e.g. 6 million units daily intravenously) is the drug of choice in severe leptospirosis and is especially effective if started within first four days of illness. Jarisch-Herxheimer reactions may occur. Total duration of therapy should be 10-14 days.¹⁹ Amoxycillin and erythromycin have also been found effective in severe leptospirosis. Patient should be observed for evidence of renal failure, and treated, if necessary, with hemodialysis. Patients with Weil's disease having hemorrhagic manifestation may require whole blood or platelet transfusion. Patients with MOF require to be observed in intensive care unit.²

Besides penicillin, doxycycline in a dosage of 100 mg twice daily for 7 days is effective in treatment of mild and moderate leptospirosis.

Effective prophylaxis consists of doxycycline, 200 mg orally once weekly, during the risk of exposure.

PROGNOSIS

The prognosis of leptospirosis depends on the severity of the disease and the associated complications. Anicteric leptospirosis usually has a good prognosis. Without jaundice the disease is almost never fatal; however, fatal pulmonary hemorrhage and myocarditis have been reported occasionally in anicteric cases. The case fatality rate for Weil's disease is 15-40%, and is higher for patients over 60 years of age.²⁶ We observed a mortality of 39.3% (13 patients) in one of our studies of 33 patients of icteric leptospirosis.

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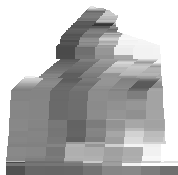
DNB Seats in Global Hospitals, Hyderabad

The **Global Hospitals, Hyderabad** is a 200 bedded Super Specialty Hospital, a dedicated facility for Multi Organ Transplantations. It is a pioneer and dedicated in Liver Transplantations. The Hospital performed FIRST Bone marrow, Liver, Cadaver Kidney, Bi-lateral Kidney, Heart transplantation etc. The department of Medical & Surgical Gastroenterology, Laparoscopic Surgery, Hepatology, Cardiology & CT Surgery, Nephrology & Urology, Internal Medicine & Critical Care, Anaesthesia & Pain management etc are very well developed. The hospital is having 6 Ultra modern OTs, State of art ICUs with hi-tech equipment, backed by modern laboratories and blood bank with components facility.

The hospital is accredited with **National Board of Examinations, New Delhi**. Applications are invited from interested candidates to join the DNB courses in (2 seats each in)

- Nephrology
- Medical Gastroenterology
- Cardiology

Eligibility: MD/ DNB (General Medicine) or MD/ DNB (Paediatrics). Attractive Stipend will be paid.



The candidates are requested to contact:
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