

# LEPTOSPIROSIS

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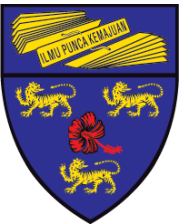
# Leptospirosis

- Epidemiology
- Pathophysiology
- Clinical presentation and Diagnosis
- Management



# Introduction

- Zoonosis caused by spirochetes of the genus, *Leptospira*
- First described by Adolf Weil in 1886
  - **‘an acute infectious disease with enlargement of spleen, jaundice and nephritis’**
- One of the emerging infectious diseases since the late 1990s
- 1995: Nicaragua epidemic of severe pulmonary haemorrhage syndrome (SPHS)
- 1990’s: Leptospirosis among US inner-city homeless population



- 1998: outbreak amongst participants of the Lake Springfield Triathlon
- Recent large outbreaks in several Asian, Central and South American countries
- Becoming an important public health problem, yet it continues to be under recognized by policy makers due to the poor quality of surveillance data



# CDC Update: Outbreak of Acute Febrile Illness Among Athletes Participating in Eco-Challenge-Sabah 2000—Borneo, Malaysia, 2000

“that *Leptospira* were the cause of illness and that water from the Segama River was the primary source of infection. Participants in adventure sports and exotic tourism should be aware of potential exposure to unusual and emerging infectious agents.”



- **MARAN, 10 JULY, 2010:** “Six villagers have died of leptospirosis and melioidosis within a week of joining in the search for a drowning victim in the Lubuk Yu recreational forest near here, Health Minister Datuk Seri Liow Tiong Lai revealed today”.





# **GUIDELINES FOR THE DIAGNOSIS, MANAGEMENT, PREVENTION AND CONTROL OF LEPTOSPIROSIS IN MALAYSIA**

**DISEASE CONTROL DIVISION  
DEPARTMENT OF PUBLIC HEALTH  
MINISTRY OF HEALTH MALAYSIA**

**2011  
1<sup>ST</sup> edition**

# Epidemiology

- No precise estimates of the global burden of human leptospirosis
- Becoming a common public health problem worldwide
- Estimated annual incidence (WHO)
  - 0.1 to 1 per 100 000 per year in temperate climates
  - 10 or more per 100 000 per year in the humid tropics
  - Estimated case-fatality rates in different parts of the world have been reported to range from <5% to 30%
- Seasonal – peak in summer, during rainy/monsoon season

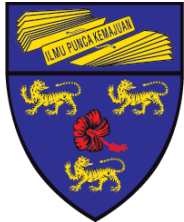




Table 1 showed an apparent increase in leptospirosis cases which could be due to several reasons such as an actual rise in the number of cases, increased awareness and diagnosis, reporting, and laboratory capacity.

**Table 1: CASES OF LEPTOSPIROSIS IN MINISTRY OF HEALTH HOSPITALS, MALAYSIA FROM YEAR 2004 TO 2009**

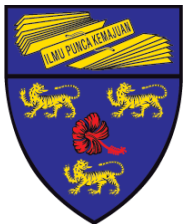
No	State	Year					
		2004	2005	2006	2007	2008	2009
1	Perak	29 (4)	71(4)	93 (9)	149 (3)	289 (16)	280 (19)
2	Selangor	16 (5)	20(3)	37	93	97 (2)	208 (7)
3	Pahang	29 (1)	24	51(3)	184 (3)	198 (7)	184 (5)
4	Kelantan	15 (1)	38 (1)	17 (1)	81(1)	180 (4)	138 (4)
5	Terengganu	7(1)	17	42(1)	55(3)	107 (4)	126 (9)
6	Kedah	15 (1)	27	31	28 (1)	52 (2)	106 (9)
7	N. Sembilan	27(1)	41	24	49	59	91
8	Sarawak	32 (2)	42 (2)	37 (3)	46 (1)	58 (5)	70 (4)
9	Johor	30 (1)	29 (6)	31 (2)	115 (2)	87 (3)	59 (3)
10	WP KL	31	20 (1)	27 (1)	31	45	54
11	Sabah	13 (1)	12 (1)	19	41 (2)	34 (2)	35 (1)
12	P. Pinang	9	25 (1)	28	37	25 (2)	32
13	Melaka	7 (1)	9 (1)	79 (2 )	32 (1)	25	20 (1)
14	WP Putrajaya	1	1	7	3	1	14
15	Perlis	2(1)	2	4	5	6	1
16	WP Labuan	0	0	0	0	0	0
<b>Total</b>		<b>263 (20)</b>	<b>378 (20)</b>	<b>527 (22)</b>	<b>949 (22)</b>	<b>1263 (47)</b>	<b>1418 (62)</b>

( ) Death

Source:

Report of Morbidity and Mortality for Patients For The Year 2004 to 2009, Health Management Information System, Medical Care Subsystem, Health Informatics Centre, Planning and Development Division, Ministry of Health, Malaysia

- Figures are grossly underestimated :  
**Overlooked and underreported**
- Why the lack of recognition?
  - Clinical manifestation wide and varied
  - May mimic many other diseases, e.g. dengue fever and other viral haemorrhagic diseases
  - Diagnostic capabilities are not readily available (especially in countries where the disease is highly endemic) → poor surveillance and reporting of cases



- Occupational hazard
  - miners, sewer workers, farmers, vets, rice field workers, soldiers etc
- Recreational exposures
  - water sports, canoeing, white water rafting



- Spread from traditional rural base → cause of epidemics in poor urban slum communities in developing countries

CLINICAL PRESENTATION AND MEDICAL COMPLICATION  
IN 59 CASES OF LABORATORY-CONFIRMED LEPTOSPIROSIS  
IN BANGKOK

B Ariyapruchya, S Sungkanuparph and S Dumrongkitchaiporn

SOUTHEAST ASIAN J TROP MED PUBLIC HEALTH  
Vol 34 No. 1 March 2003

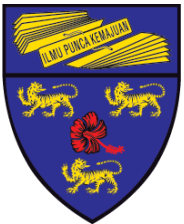
More than half of the cases were Bangkok residents and did not travel outside Bangkok in the preceding 2 weeks. The majority of the cases presented in late rainy season. Leptospirosis in the urban area is common and should be recognized, particularly in rainy season.

- Wild mammals are the maintenance host or primary reservoir for the spirochetes
  - Pathogenic leptospire are maintained in nature in the renal tubules of certain animals
  - Rats/rodents, cats, livestock, raccoons, dogs etc
- Humans become infected through direct contact with the infected urine or indirectly through contact with contaminated water
- Enters through mucous membrane or abraded skin



# Microbiology

- Leptospire are bacteria which can be either pathogenic or saprophytic
- There are over 200 pathogenic serovars divided into 25 serogroups
- Certain serovar may develop a commensal or comparatively mild pathogenic relationship with a certain animal host species
  - Eg. cattle often associated with serovar hardjo, dogs with canicola and rats with icterohaemorrhagiae and copenhageni



## Other ways of classifying leptospire:

- **Species classification:** based on DNA homology

The two classification systems based on the serovar and species concepts are not always in agreement and strains belonging to the same serovar may belong to different *Leptospira species*



Table A9.1 Species and the serogroups most commonly present in them (Brenner et al. 1999).

Species	Major serogroups present
<i>L. alexanderi</i> (genomospecies 2)	Hebdomadis, Manhao
<i>L. borgpetersenii</i>	Ballum, Javanica, Sejroe, Tarassovi
<i>L. interrogans</i> sensu stricto	Australis, Autumnalis, Canicola, Icterohaemorrhagiae, Pomona, Pyrogenes, Sejroe
<i>L. kirschneri</i>	Autumnalis, Grippotyphosa, Icterohaemorrhagiae
<i>L. noguchi</i>	Australis, Icterohaemorrhagiae
<i>L. santarosai</i>	Hebdomadis, Mini, Pyrogenes, Sejroe, Tarassovi
<i>L. weilii</i>	Celledoni, Javanica, Tarassovi
<i>L. fainei</i> <sup>a</sup>	Hurstbridge
<i>L. inadai</i> <sup>a</sup>	Lyme, Manhao
<i>L. meyeri</i> <sup>a</sup>	Javanica, Mini, Sejroe
<i>L. biflexa</i> sensu stricto <sup>b</sup>	Andamana
<i>L. wolbachii</i> <sup>b</sup>	Codice, Semarang



# PATHOPHYSIOLOGY

- Infection → leptospire appear in the blood → invade all tissues and organs particularly affecting the liver and kidney → cleared from the body by the host's immune response
- May also settle in the convoluted tubules of the kidneys → shed in the urine for a few weeks to several months or longer
- Subsequently cleared from the kidneys and other organs (may persist in the eyes for much longer)



# PATHOPHYSIOLOGY

- Produces endotoxin → attach onto the endothelial cells → capillary vasculitis (endothelial necrosis and lymphocytic infiltration)
- Vasculitis and leakage → petechiae, intraparenchymal bleeding and bleeding along serosa and mucosa
- Lost of fluids into the third space → hypovolaemic shock and vascular collapse

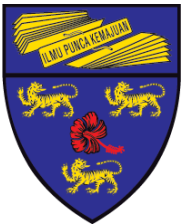


- Humans react to an infection by producing specific anti-*Leptospira* antibodies
- Seroconversion
  - as early as 5–7 days after the onset of disease
  - sometimes only after 10 days or longer
  - IgM appear somewhat earlier than IgG and generally remain detectable for months or even years but at low titre.



# Clinical Presentation and Diagnosis

- Diagnostic challenges
  - early-phase leptospirosis: non-specific presentation
  - frequent cause of undifferentiated febrile illness in developing countries
  - Misdiagnosis in regions where dengue and other infectious diseases with overlapping presentations are endemic
  - Co-infection with other diseases such as scrub typhus, dengue and malaria

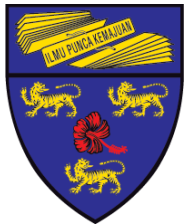


- Timely diagnosis is essential
  - antibiotic therapy provides greatest benefit when initiated early in the course of illness
- Diagnosis of leptospirosis therefore depend on a high index of suspicion among clinicians and the availability of an accurate ‘point-of-care’ test



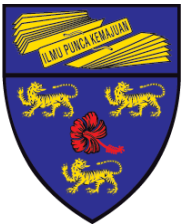
# Clinical Features

- Incubation Period: usually 5-14 days; range: 2-20 days
- Biphasic clinical course
  - Acute or septicemic phase lasting about 1 week
  - Immune phase when antibodies are produced and leptospire are excreted in the urine (6days-4weeks)
- Most complications are associated with localisation of leptospire within the tissue during the immune phase



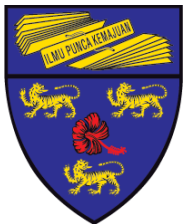
# Clinical syndromes

- Anicteric Leptospirosis
  - Self limiting, symptoms non specific
  - Majority of infection
  - Usually lasts 1-2 weeks
  - Aseptic meningitis seen in 25-50%
  - Mortality almost nil – extremely rare (some reported cases of death due to pulmonary haemorrhage)



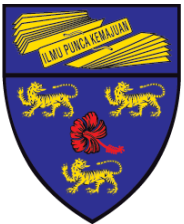
- Icteric Leptospirosis

- Weil's disease
- 5-10% of patients with leptospirosis
- 5-15% mortality
- Biphasic clinical course may be indistinguishable
- Jaundice, renal failure, (pulmonary) haemorrhage
- Other organs – cardiac, ocular, cause for abortion in pregnancy, acalculous cholecystitis, pancreatitis





- Typically, the disease presents in four broad clinical categories:
  - (i) a mild, influenza-like illness;
  - (ii) Weil's syndrome characterized by jaundice, renal failure, haemorrhage and myocarditis with arrhythmias;
  - (iii) meningitis/meningoencephalitis;
  - (iv) pulmonary haemorrhage with respiratory failure.



# Common symptoms and signs

- fever
- severe headache
- myalgias
- conjunctival suffusion
- jaundice
- general malaise
- stiff neck
- chills
- abdominal pain
- joint pain
- anorexia
- nausea
- vomiting
- diarrhoea
- oliguria/anuria
- haemorrhages
- skin rash
- photophobia
- cough
- cardiac arrhythmia
- hypotension
- mental confusion
- psychosis
- Delirium

No presentation of leptospirosis is diagnostic and clinical suspicion must be confirmed by laboratory tests

# Multiorgan involvement

- Ocular
  - Suffusion – dilation of the conjunctival vasculature, subconjunctival haemorrhage, uveitis
  - Icterus scleral with conjunctival suffusion-pathognomic of Weil's disease
- GI
  - Jaundice not associated with hepatocellular necrosis. Bilirubin, ALT, AST will normalise
- Renal
  - Acute tubular necrosis (direct leptospire injury)
  - Interstitial nephritis (relate to Ag-Ab complexes)

- Cardiac
  - Myocarditis, 1<sup>st</sup> degree heart block, coronary arteritis
- Pulmonary
  - Spectrum ranging from cough, dyspnoea, haemoptysis to ARDS
  - Pulmonary haemorrhage may cause death
  - Radiology reveals diffuse small opacities which may disseminate or coalesce – a sign of intra-alveolar and interstitial haemorrhage

# Prognosis

- The virulence factors in leptospirens are poorly understood
- Mortality is dependent on many factors
  - Some serovars generally tend to cause mild disease and others severe disease (icterohaemorrhagiae and copenhageni ). However, there are no serovar specific presentations of infection and any serovar may cause mild or severe disease in different hosts



# Prognosis

- Patient factors
  - old age, multiple underlying medical problems, nutritional status
- The infection dose
- Renal failure, HyperK, Plt, cardiac failure with hypotension and arrhythmia, and SPHS may be contributory factors to the mortality rate
- Neurological (e.g. disturbance of consciousness, delirium and stiffness of the neck) and GI symptoms (e.g. GI bleeding, repeated nausea and vomiting, abdominal pain and hiccups)

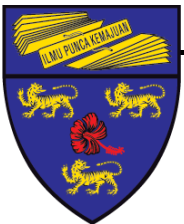


# Prognostic Factors in Leptospirosis: A Study From Kerala, India

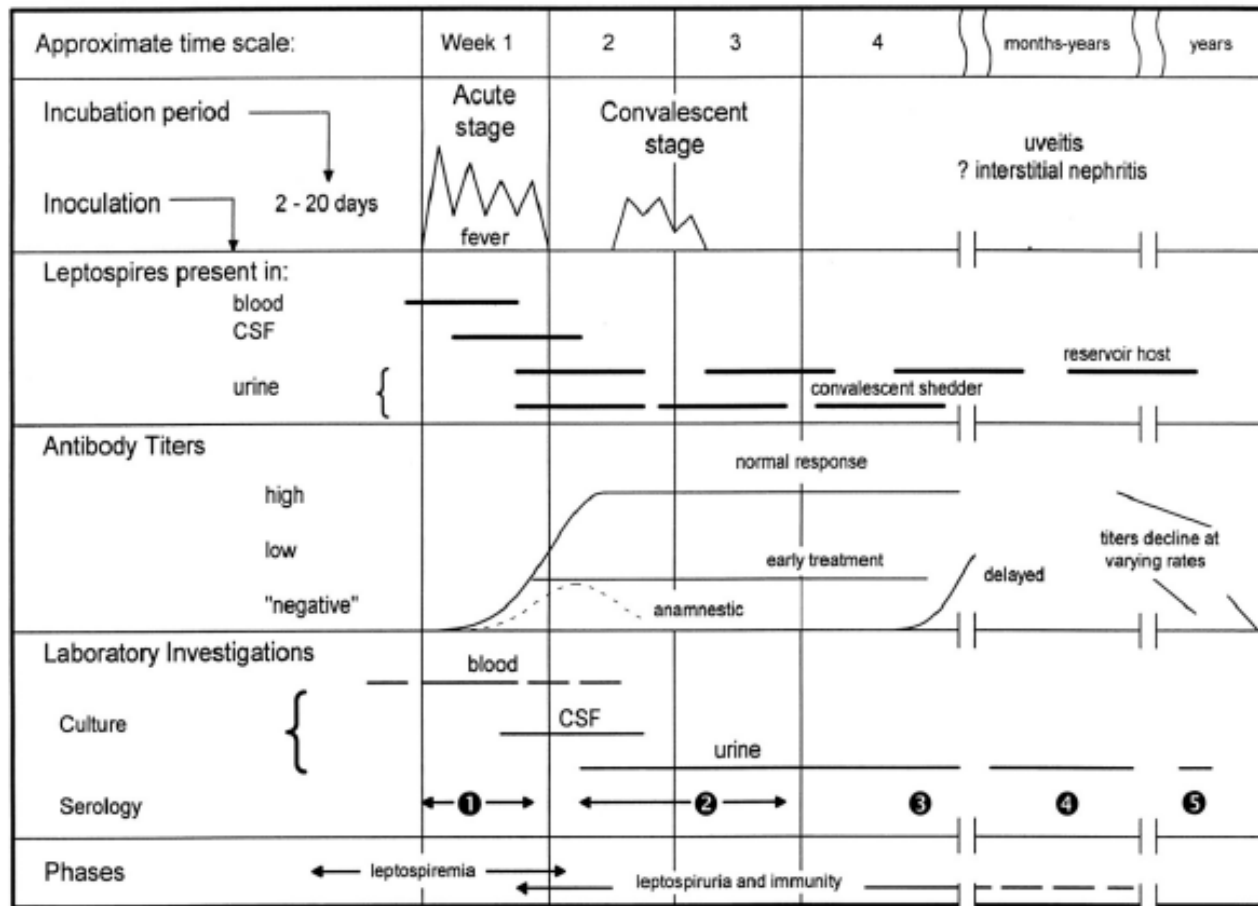
- Moderate and severe disease
- Mortality : 15%, suggests of a higher mortality > 50 yrs
- Gender, at-risk occupation, rodent exposure or interval to penicillin therapy were not significant predictors of mortality
- CPK, Na, K and platelet counts were significantly different between the two groups
- On multivariate analysis, organ dysfunction, hyponatremia, elevated CPK, bleeding manifestations, cardiac or neurologic dysfunction were significant predictors of mortality
- Identification of patients who can be predicted to do poorly helps focus intense treatment to improve outcome

# Laboratory

- Inadequate laboratory test : major barrier for diagnosis and epidemiologic surveillance
- The disease is usually diagnosed by
  - detecting antibodies
  - culturing the bacteria from blood, urine or tissues
  - demonstrating the presence of leptospire in tissues using antibodies labelled with fluorescent markers
  - polymerase chain reaction (PCR)
  - immunostaining







**Figure 1: Leptospiremic phases in conjunction with the laboratory methods of diagnosis (4).**

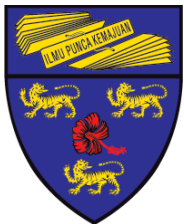
Note: Biphasic nature of leptospirosis and relevant investigations at different stages of disease. Specimens 1 and 2 for serology are acute-phase specimens, 3 is a convalescent-phase sample which may facilitate detection of a delayed immune response, and 4 and 5 are follow-up samples which can provide epidemiological information, such as the presumptive infecting serogroup (4).

- Methods for the direct detection of leptospire are either slow or of limited reliability → therefore serology is often the most appropriate diagnostic method



# MAT- Microscopic agglutination test

- **"gold standard"** of serodiagnosis
  - unsurpassed diagnostic (serovar/serogroup) specificity in comparison with other available tests
- Sensitivity 92% Specificity 95%
- Unable to differentiate between agglutinating antibodies due to current, recent or past infections

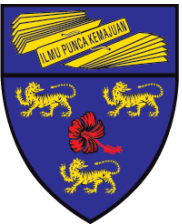


- Two consecutive serum samples should be examined
  - look for seroconversion or a four-fold or greater rise in titre
- Single titre: Different cut-off points. 1:100, 1:200, 1:400 or 1:800 as diagnostic of current or recent leptospirosis



# ELISA

- Popular and several assays available
- Based on a genus-specific antigen
- Detect IgM antibody and sometimes also IgG antibodies in the early phase of the disease
- Some systems are less specific than the MAT
- Weak crossreactions due to the presence of other diseases may be observed
- ELISA results should be confirmed by the MAT



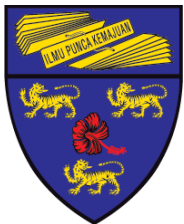
# Direct methods of detecting leptospire

- Culture, dark-field microscopy, inoculation of experimental animals, (immuno)staining and the polymerase chain reaction (PCR)

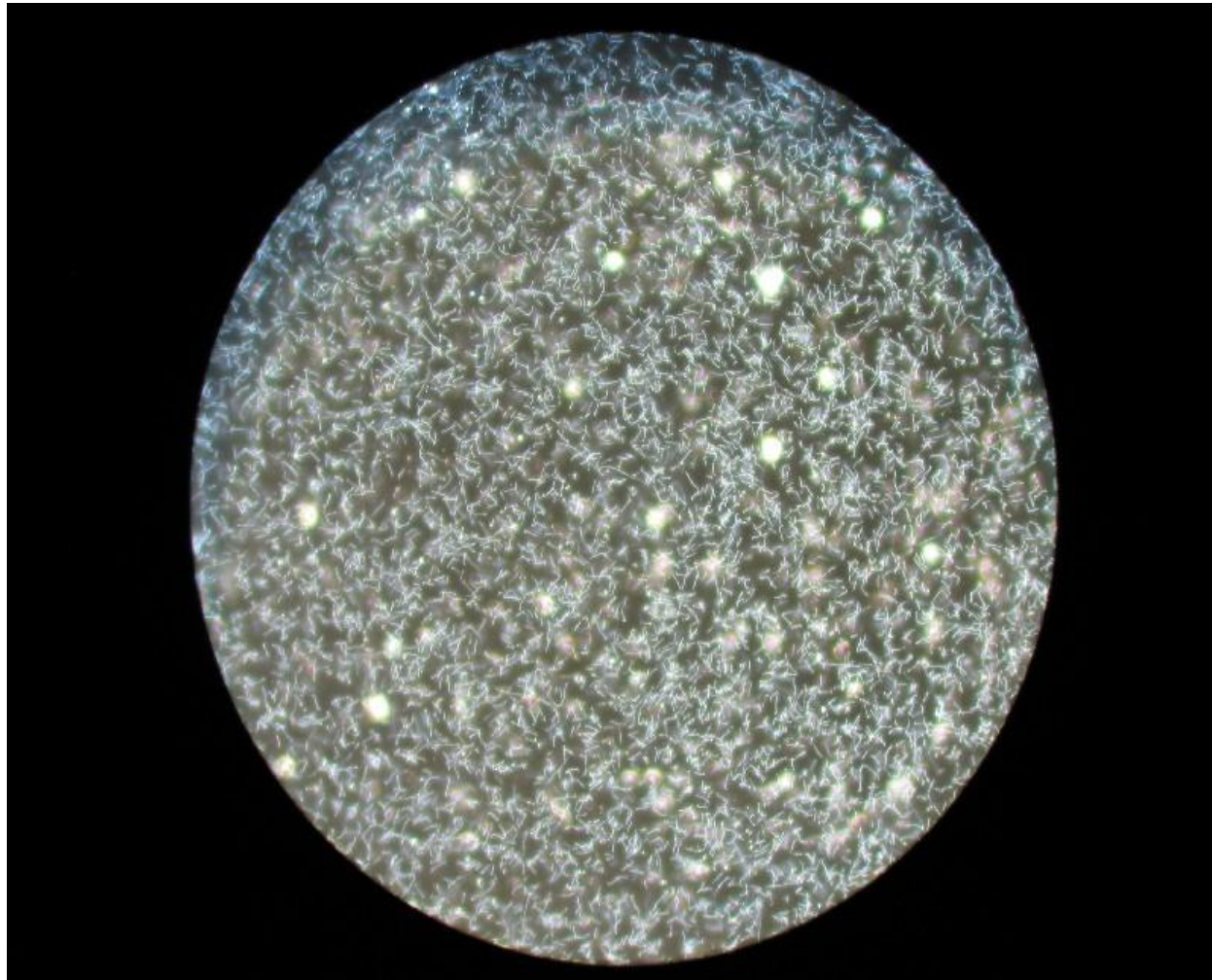


# Dark-field microscopy

- Leptospire are observed as thin, coiled, rapidly moving microorganisms in fluids such as culture medium, blood or urine using dark-field microscopy
- Technically demanding, false-positive and false-negative diagnoses are easily made
- Always confirmed by other tests



# Dark field microscopy





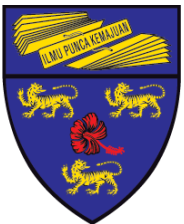
## SEROLOGICAL TECHNIQUES OTHER THAN MAT AND ELISA

Test	Commercially available <sup>a</sup>
Complement-fixation test (CFT)	Antigens
Counterimmunoelectrophoresis (CIE)	–
<u>Dipstick tests:</u>	
– LEPTO Dip-S-Tick	+
– LeptoTek Lateral Flow	+
Dried latex agglutination test (LeptoTek Dri-Dot)	+
Indirect fluorescent antibody test (IFAT)	+
Indirect haemagglutination test (IHA)	+
Latex agglutination test (LA)	–
Macroscopic slide agglutination test (SAT)	Antigens
Microcapsule agglutination test	+
Patoc-slide agglutination test (PSAT)	+
Sensitized erythrocyte lysis test (SEL)	–



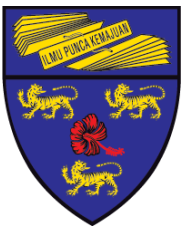
# Treatment

- Appropriate chemotherapy and supportive therapy help reduce the mortality of severe cases
- Early treatment is very important
  - If not treated appropriately within the first 2-3 days, it may progress in severity



# Antibiotics

- Treatment with effective antibiotics should be initiated as soon as the diagnosis of leptospirosis is suspected and preferably before the fifth day after the onset of illness
- The benefit of antibiotics after the fifth day of the disease is controversial - However, most clinicians treat with antibiotics regardless of the date of onset of the illness



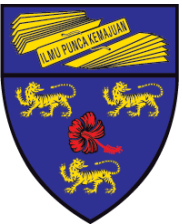
- Antibiotics

- RCT, vs placebo

- Doxycycline shortened illness by two days
    - Penicillin used in severe leptospirosis shorten duration of fever, improved renal function quicker and shorten hospital stay
    - Both prevented shedding of *Leptospira* into urine



- Severe cases of leptospirosis - high doses IV penicillin
- Less severe cases - oral antibiotics eg. amoxicillin, ampicillin, doxycycline, erythromycin, azithromycin
- As effective - Third-generation cephalosporins, such as ceftriaxone and cefotaxime, and quinolone

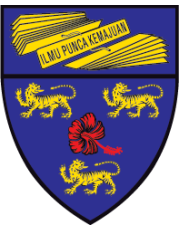


- RCT, penicillin vs ceftriaxone and penicillin vs cefotaxime vs doxycycline
  - All were therapeutically equivalent
  - Doxycycline or ceftriaxone/cefotaxime recommended in severely ill patient when the diagnosis is in doubt especially in areas endemic for rickettsial infection



# Prophylaxis

- Studies from the late 1970s of US soldiers in Panama
  - Doxycycline 200 mg per week prevented leptospirosis
- A randomized, placebo-controlled study of the efficacy of doxycycline 200 mg/week in the Andaman Islands
  - No protective efficacy among indigenous populations in terms of acquiring leptospiral infection
  - But significant effect in reducing clinical illness and mortality
- Difficult to recommend to people with continued ongoing exposures
- The need for leptospirosis vaccines to prevent disease in those at highest risk should be emphasized



# Steroids

- Case series analysing the benefits of methylprednisolone in pulmonary leptospirosis
- 30 pts, 17pts received steroids (methylprednisolone 1g/day for three days followed by 'o' prednisolone 1mg/kg for 7 days), 13pts did not receive steroids
- 18% with steroids died ,62% without steroids died
- Acute lung injury mortality : 37% - from the steroids group, 89% - without steroids

**Steroids affects outcome by reducing mortality, if given within 12 hours of manifestation of pulmonary manifestation**



Postgraduate Med. Journal Sept. 2006, 82(971):602-06





- 'Pre-MP period' vs 'MP period'
- Clinical score 0-6 applied to assess severity
- A score  $\geq 2$  = severe
- MP period: 149 cases, 72 severe cases, 62 received MP 500 mg intravenously for 3 days, followed by oral 8 mg for 5 days (10 did not receive MP- terminally ill)
- Pre-MP period: 78 cases, 60 had a severe score



High efficacy of bolus methylprednisolone in severe leptospirosis: a descriptive study in Sri Lanka  
Senanayake A M, Postgrad Med J (2010)

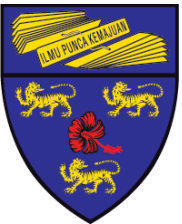


**Table 1** Clinical score (to develop score categories, 0–6)

No	Parameter	Score
1	Stable general condition, no complications	0
2	Poor general condition: ill look with dehydration, inability to keep head up, faintish and malaise	1
3	Haematological: thrombocytopenia (platelet count $<100 \times 10^9/l$ ), bleeding tendency	1
4	Pulmonary: dyspnoea, hypoxaemia, diffuse fluffy shadows in chest radiograph	1
5	Cardiac: tachycardia, hypotension, T inversions in ECG, low EF*	1
6	Renal: oliguria, high blood urea and serum creatinine, microscopic haematuria, and cast in urine sediments	1
7	Hepatic: deep icterus, 10-fold rise of alanine and aspartate transaminases, features of acute liver failure	1
Total clinical score		6

\*EF, ejection fraction in two dimensional echocardiogram.

- Mortality: 17 Pre MP (21.8%) and 16 MP (10.7%), (p=0.025)
- Survival rate at score 4 in the MP period severe group was 100% (16 of 16), compared to 38% (5 of 13) in pre-MP period severe group, (p<0.001)
- Six patients who died despite MP therapy had a clinical score of 5 or 6; four were alcohol consumers, and two had heart disease and hypertension.



**Table 2** Distribution of total clinical score category (0–6) between two periods (before and after 25 May 2008)

Total score category	Pre-MP period		MP period			
	No. of cases	No. of deaths (%)	MP given		MP not given	
			No. of cases	No. of deaths (%)	No. of cases	No. of deaths (%)
00	5	0	0	—	22	0
01	13	0	0	—	55	0
02	27	0	16	0	0	—
03	11	0	18	0	0	—
04	13	8 (62)	16	0	0	—
05	6	6 (100)	10	5 (50)	5	5 (100)
06	3	3 (100)	2	1 (50)	5	5 (100)
Total	78	17 (22)	62	6 (10)	87	10 (11)

MP, methylprednisolone.

- MP may reduce mortality in patients with severe leptospirosis, except in cases with established multiple organ dysfunction and comorbidities
- Early administration of MP seems advisable



- Steroids

- 8 patients – 7 out of 8 resumed normal life after treatment with a combination of steroids and noninvasive ventilation was life saving

**Ind. Journal of Critical care medicine 2005, Vol9(3) Pg. 133-136**



# Ventilation

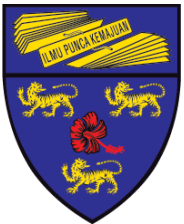
- Similar to ARDS, prone ventilation improves oxygenation
- Case report: Patient with severe hypoxaemia from diffuse alveolar haemorrhage caused by vasculitis and +ve ANCA
- Prone ventilation improved oxygenation by 70%, improvement even occurring at first hour
- Improved VQ matching by changes in inflation, ventilation, recruitment and perfusion of alveoli
- Removes secretions and blood
- Supine-blood drains posteriorly blocking dependent airways and cause hypoventilation of the dorsal lung units

British Journal of Anaesthesia 2004(Vol. 92), no.5, Pg 754-57



# Plasma exchange

- Case reports
- Clin, Diag. Lab Imm. 2002, Mac, 9(2), Pg 482-484
  - Plasma exchange should be considered as an adjunctive therapy for patients with severe icteric leptospirosis (severe hyperbilirubinaemia) complicated by ARF who have not shown rapid clinical response to conventional treatment (antibiotics and haemodiafiltration)





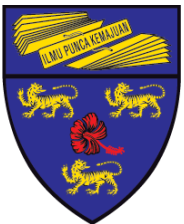
# Conclusion

- Clinicians must be more aware of Leptospirosis
  - Nonspecific clinical presentation
  - Difficult laboratory diagnosis
- Consider Leptospirosis as a differential diagnosis for any undifferentiated febrile illness
- Early detection leads to early treatment with antibiotics, steroids and other supportive measures including respiratory support



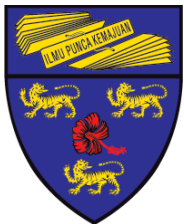
# CASE HISTORY

- JS, 17, Male
- Presented with 4 days of
  - Fever - sudden onset, high grade
  - Diarrhoea and Vomiting
  - Giddiness. Headache and Retroorbital pain
  - Generalised myalgia
  - Nonproductive cough
- Nil significant PMH/PSH/FH/DH
- No jungle trekking/travel



# Examination

- Alert, dehydrated, no rash
- Temperature 39.5 C, BP 85/46 mm/Hg, PR 108/min, SpO<sub>2</sub> 95% ↓room air
- CVS and Respiratory system – NAD
- Abdomen – No hepatosplenomegaly, generalised tenderness, no rebound or guarding



# Investigations

- Hb 14.7
- WBC 12.9
- Plt  $132 \times 10^9$
- Hct 0.44
- Na 137 mmol/L
- K 4.1 mmol/L
- U 7.3 mmol/L
- Cr 102  $\mu\text{mol/L}$
- TBil 41  $\mu\text{mol/L}$
- ALT 31 IU/L
- AST 53 IU/L
- Amylase 35 IU/L

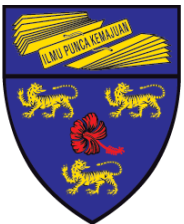


# D2 of admission

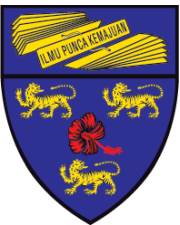
- One episode of haemoptysis
  - Reduced urine output overnight.
  - Haematuria upon insertion of CBD
  - On further questioning.....
- 
- Had been to Hulu Langat waterfall for a swim with friends two weeks prior to admission.....



- T38 C, RR 30/min,
- SpO<sub>2</sub> 78% ↓ room air, 99% ↓ 15L/min O<sub>2</sub> via HFM
- BP 98/50 mm/Hg with Dopamine infusion
- PR 115/min, bounding pulse
- Respiratory – Bibasal crepitations

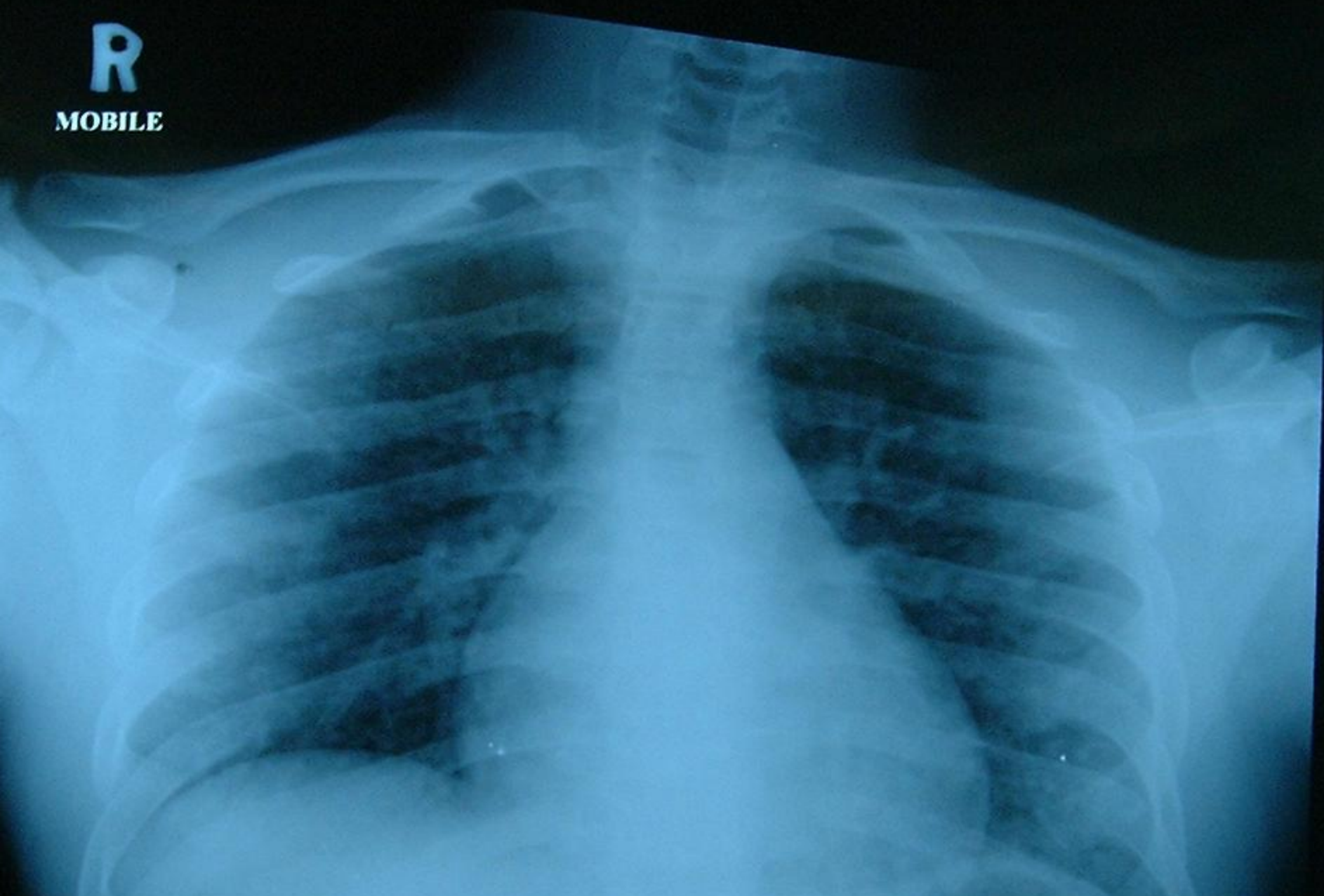


- Hb 12.3 ↓
- HCT 0.36 ↓
- Plt  $91 \times 10^9$  ↓
- Urea 13.8 mmol/L ↑
- Cr 136 mmol/L ↑
- ABG (room air)
  - pH 7.42, pCO<sub>2</sub> 4.7 kPa, pO<sub>2</sub> 6.8kPa, HCO<sub>3</sub>  
22mmol/L, BE -2 mmol/L
- AST/ALT normal



**R**

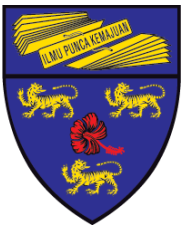
**MOBILE**

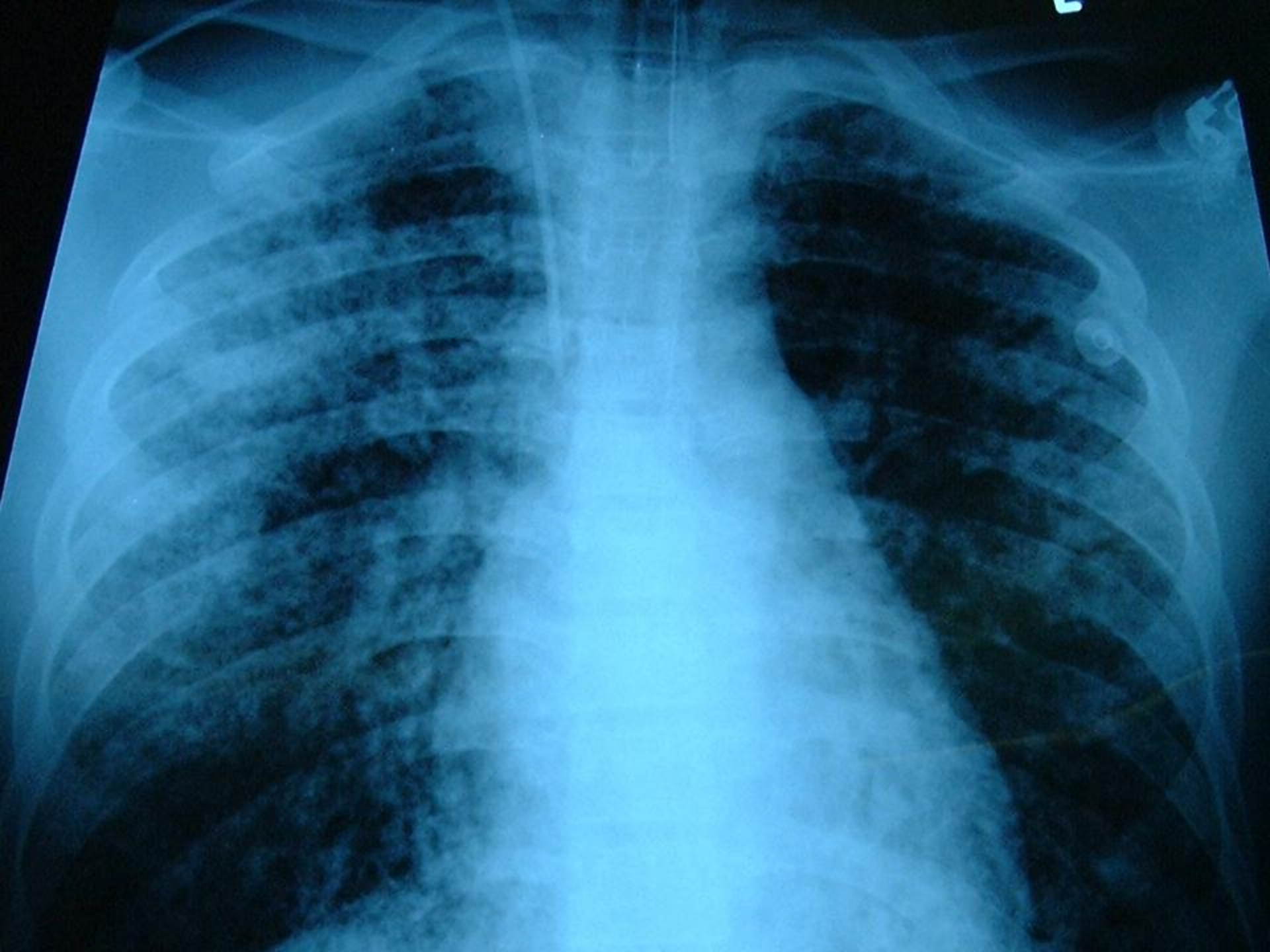




# Diagnosis

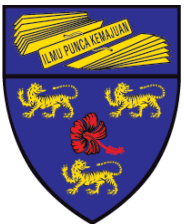
- Leptospirosis with
  - Renal failure
  - Thrombocytopenia
  - Pulmonary haemorrhage with type 1 resp failure
  - Septicaemic shock
  - Metabolic acidosis





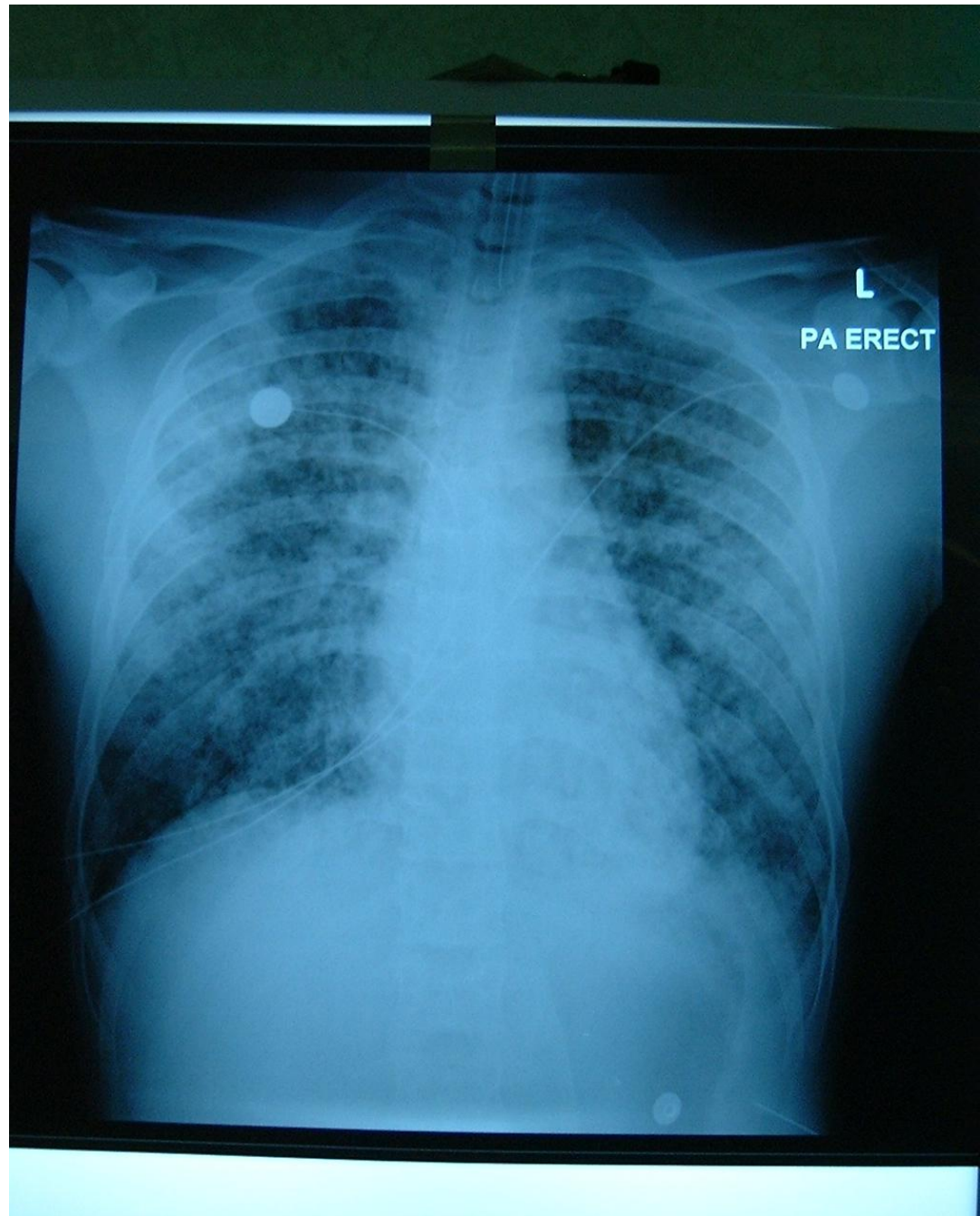
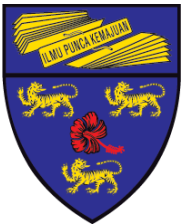
# Management

- Antibiotics – iv Ceftriaxone +Doxycycline
- Airway support – BIPAP then intubation  
Needed high ventilator setting  
– FiO<sub>2</sub> 100%, PEEP 23
- Haemodynamic – inotropic support with dopamine and noradrenaline infusion + iv fluid



# Progress

- Heamorrhagic fluid from ETT



- SpO<sub>2</sub> 85-90%
- Diagnosis- Pulmonary haemorrhage/ ARDS

***Started on iv Methylprednisolone 1g/od for 3 days***

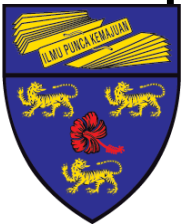
***Transfused 4 units FFP, 4 units Platelet, 2 pints PC***

***Ventilation via prone position***



# Progress

- No further bleeding
- Jaundiced
- Bilateral subconjunctival haemorrhage
- Leptospira serology < 1:80
- Haemodynamically more stable, able to slowly wean off inotropes
- SpO<sub>2</sub> maintained above 95%



- Extubated D11 of admission
- Discharged after 13 days of admission
- Bilateral subconjunctival haemorrhage



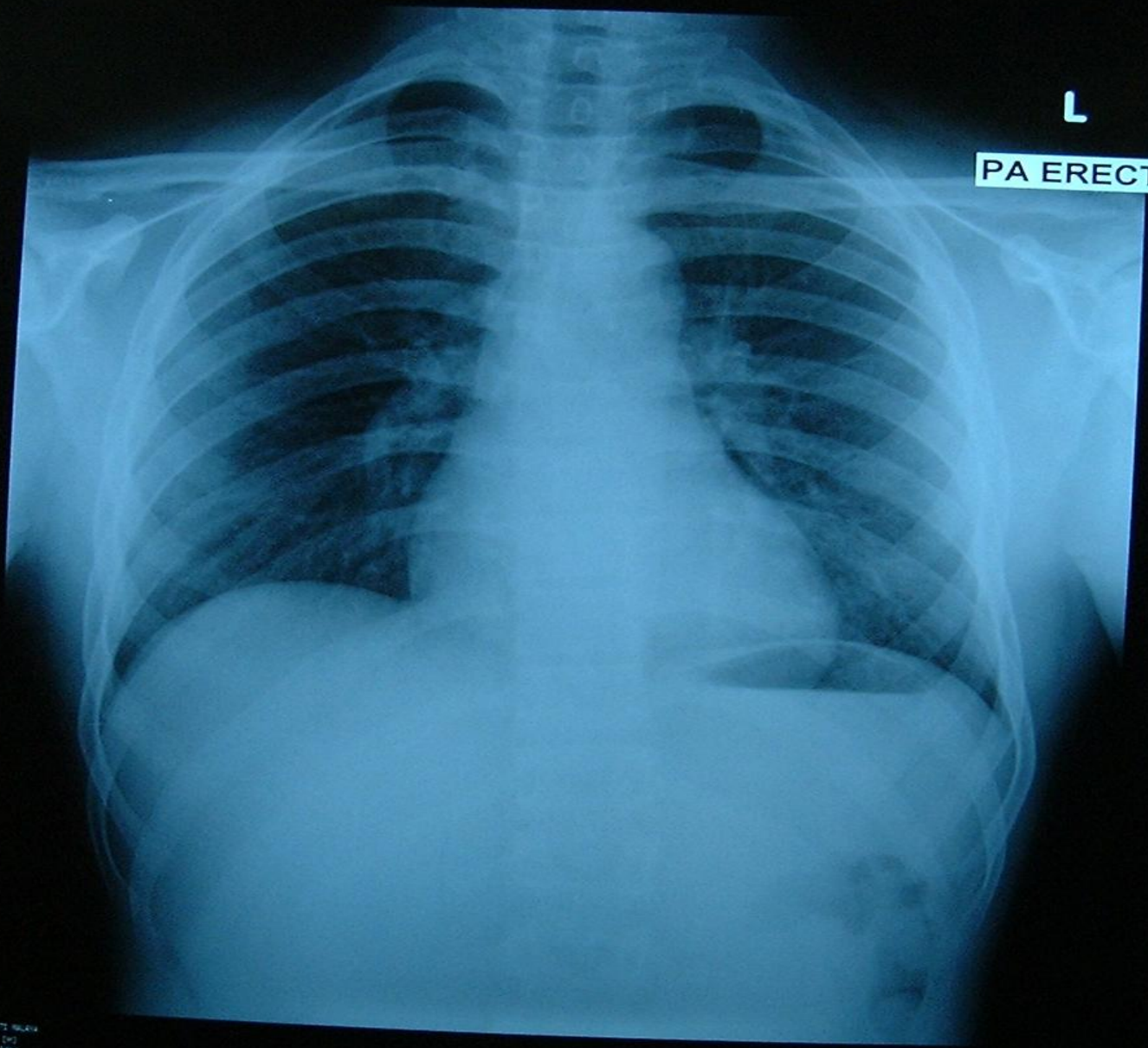
# Results

	4/3	5/3	6/3	7/3	8/3	9/3	12/3	18/3	8/4
Hb	14.7	12.3	10.7	11.1	11.3	11.8	11.3	11.9	
HCT	0.44	0.36	0.32	0.33	0.34	0.36	0.34	0.36	
WCC	12.9	8.7	12.3	8.7	12.6	8.8	8.6	13.8	
PLT	132	91	30	60	47	110	184	802	
Ur	7.3	13.8	15.8	15	11.5	9.4	7.8	7.4	
Cr	102	136	183	165	94	81	42	53	
Bil	41	33	155/139	213/171	260/226	247/204	257/210	123	41/28
AST	53	62	64	71	49	70	88	147	31
ALT	31	30	30	37	39	62	117	394	87



- Paired *Leptospira* serology
  - D5 of illness <1:80
  - D12 of illness 1:1280





L

PA ERECT

150-09271 BALAN  
MURRAY, D  
DOB  
2002 [11:35]  
RT 01

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THANK YOU

