

A Study of Multi - Organ Involvement in Leptospirosis in paediatric patients.

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Abstract

Background: Awareness about the infection is scarce mainly in the growing nations as a consequence of which, it is not often thinking of in the differential analysis of febrile illnesses. Failure to diagnose leptospirosis is especially unfortunate: severely ill sufferers regularly recover completely with prompt remedy but if therapy is delayed or no longer given, death or renal are probable to make sure. **Methods:** A whole 50 patients who were admitted with fever at FIMS hospital, medication branch over a period of 2 years that is at some stage in August 2005 to August 2007 who have been diagnosed to have leptospirosis satisfying the inclusion and exclusion criteria. Present study sketch is a prospective learn about at some point of which affected person will be followed up throughout clinic stay, healing and one month after discharge. **Results:** In our find out about out of 5 sufferers sixteen sufferers (32%) had no complications, 34 sufferers (68%) have developed a range of complications. 4 patients (8%) developed ARF, 7 patients (14%) developed hepatitis, 17 patients (34%) ARF with Hepatitis, 2 sufferers (4%) Hepatitis with altered sensorium, 1 patient (2%) developed Myocarditis; Hepatitis; ARF, 1 affected person (2%) developed ARF; ARDS; Hepatitis; Myocarditis, 2 patients (4%) ARDS with ARF and Hepatitis. **Conclusion:** Early analysis & initiation of remedy of leptospirosis prevents mortality and morbidity.

Keywords: Nonalcoholic Fatty Liver Disease, Non-alcoholic steatohepatitis, Diabetes.

INTRODUCTION

Leptospirosis is an acute febrile illness common in many parts of the world. Most cases are mild or asymptomatic but the most severe illness, known as Weil's disease, may be associated with death through renal failure. Leptospirosis is a worldwide zoonotic infection. It is known by many different local names (e.g. mud, swamp, sugarcane, fort Bragg, Japanese autumnal fevers). The major reservoir is rodents and the organism is passes in their urine for moths and can survive in fresh but not brackish water. Man is infected by contact with rodent urine or with the urine of other infected animals such as dogs and domestic farm animals, or with meat contaminated by urine.[1-7]

Awareness about the infection is scarce especially in the developing countries as a consequence of which, it is rarely thought of in the differential diagnosis of febrile illnesses.

Failure to diagnose leptospirosis is particularly unfortunate: severely ill patients often recover completely with prompt treatment but if therapy is delayed or not given, death or renal are likely to ensure.[8]

As leptospirosis can be treated by antibiotics especially penicillin group treating physicians should have high clinical suspicion of the disease especially in tropical countries like India and do appropriate investigation to confirm it.[9]

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Out of 704 patients presenting with acute febrile illness in J.S.S Hospital, from 1-1-2004 to 31-12-2004 (1year) 126 patients (17.86%) were found to have leptospirosis.

MATERIAL AND METHODS

Source of data: A total 50 patients who were admitted with fever at FIMS hospital, medicine department over a period of 2 years that is during August 2005 to August 2007 who have been diagnosed to have leptospirosis fulfilling the inclusion and exclusion criteria.

Present study design is a prospective study during which patient will be followed up during hospital stay, recovery and one month after discharge.

Method of collection of data: Data for the proposed study was collected in a pre-tested proforma which includes various socioeconomic parameters like age, sex, occupation etc. 50 cases have been selected on the basis of simple random sampling.

Clinical, biochemical and hematological profile of these patients was followed up from the date of admission, during the hospital stay and one month after discharge.

Statistical Analysis: The analysis of the data was made on the basis of important statistical parameters like the Mean, Standard deviation, Standard error, T-test and proportion test where applicable. All values are compared at 5% or 0.05 and 1% or 0.01 levels of significance for the corresponding degree of freedom to arrive at conclusion regarding the objectives of the study.

Inclusion criteria: All the patients diagnosed to have leptospirosis on the basis of scoring criteria devised by Faine (score is more than 26), All the patients tested positive for anti-leptospiral IGM antibodies, Organism is demonstrated by dark ground microscopy.

Exclusion criteria: All known patients with leptospirosis, who were also positive for acute febrile illness like malaria, typhoid, dengue, brucella, viral hepatitis, rickettsiae.

Investigations: The following investigations are done to assess the study: Complete blood picture, routine urine analysis, random blood sugar, blood urea, serum creatinine, chest x-ray pa view, HIV-antibodies defecated by ELISA, dark ground microscopy for leptospirosis blood and urine, IGM antibodies to leptospira, QBC for MP, LFT, platelet count, ultrasound abdomen, Widal test, IGM antibodies to dengue, LP CSF analysis (if necessary), kidney biopsy (if necessary).

RESULTS

Out of 50 patients in 23 patients (46%) blood urea levels are within normal limit (i.e. 20-40 mg/dl), 27 patients (54%) have levels of blood urea elevated (i.e. >40mg/dl) in these, 14 patients have blood urea level of 41-80mg/dl, 2 patients 81-100mg/dl, 9 patients 101-200mg/dl, 2 patients 201-250mg/dl.

Out of 50 patients, 16 patients (32%) creatinine level is elevated (i.e. >1.4).

34 patients (68%) creatinine is within limits (0.8-1.4 mg/dl). Out of 16 patients 6 patients creatinine is >4.1, 4 patients 1.41-2.0mg/dl, 3 patients 2.1-3.0mg/dl, 1 patient 3.1-4mg/dl [Table 1].

Out of 50 patients in 18 patients (36%) T.bilirubin is within normal limit (i.e. below 1.1 mg/dl). 32 patients (64%) T.bilirubin is elevated (> 1.1 mg/dl), among these 32 patients 14 patients T.bilirubin is 5.1 – 16.5 mg/dl, 9 patients 1.1-2.0 mg/dl, 4 patients 4.1-5.0 mg/dl, 3 patients 2.1-3.0 mg/dl, 2 patients 3.1-4.0 mg/dl. Maximum total bilirubin observed in this study is 16.50mg/dl.

Out of 50 patients 10 patients (20%) have D.bilirubin within normal limit i.e. below 0.2 mg/dl). 40 patients (80%) D.bilirubin is elevated (>0.2mg/dl) among these 40 patients 14 patients D.bilirubin is in between 3.1-10.8mg/dl, 11 patients 0.2-0.9mg/dl, 10 patients 0.91-2.0mg/dl, 5 patients 2.01-3.0mg/dl. Maximum D.bilirubin observed in this study is 10.8mg/dl.

In our study 16 patients (32%) SGOT level is within normal limit (20-40u/l). 34 patients (68%) SGOT level is elevated (>40u/l) among these 34 patients 12 patients SGOT level is 41-100u/l, 10 patients 101-150u/l, 9 patients >200u/l, 3 patients 151-200u/l. in this study maximum SGOT observed is 900u/l.

16 Patients (32%) SGPT levels are within normal limits (i.e. 20-30u/l). 34 patients (68%) SGPT is elevated (>40u/l) among these 34 patients 16 patients SGPT level is 41-100u/l, 9 patients >200u/l, 7 patients 101-150 u/l, 2 patients 151-200 u/l. in this study maximum SGPT level observed is 920 u/l.

15 patients (30%) ALP is elevated (i.e. >147u/l). 35 patients (70%) ALP is within normal limit (i.e. 37-147u/l). Maximum ALP observed in this study is 540u/l [Table 2].

In our study out of 5 patients 16 patients (32%) had no complications, 34 patients (68%) have developed various complications. 4 patients (8%) developed ARF, 7 patients (14%) developed hepatitis, 17 patients (34%) ARF with Hepatitis, 2 patients (4%) Hepatitis with altered sensorium, 1 patient (2%) developed Myocarditis; Hepatitis; ARF, 1 patient (2%) developed ARF; ARDS; Hepatitis; Myocarditis, 2 patients (4%) ARDS with ARF and Hepatitis [Table 3].

Table 1: Cases depending on renal parameters

Renal parameters	No. of patients	%
Urea (>40mg/dl)	27	54
Creatinine (>1.4 mg/dl)	16	32

Table 2: Liver function tests

LFT	No. of patients	%	P-value	Inference
T.Bilirubin(>1.1mg/dl)	32	64	< 0.05	H.S
D.Bilirubin (>0.2mg/dl)	40	80	< 0.05	H.S
SGOT (> 40u/L)	34	68	< 0.05	H.S
SGPT (> 40u/L)	34	68	< 0.05	H.S
ALP (> 147u/L)	15	30	> 0.05	N.S

DISCUSSION

In spite of major successes against infectious diseases in 20th century, new infections have emerged and old ones re-emerged in recent decades. Leptospirosis appears to be on the increase in Karnataka, Kerala, Tamil Nadu and Andaman during last two decades probably due to increased farming and inadequate rodent control.[10, 11] In this study 80% of the cases had abnormal liver function tests. Most of the elevation of SGOT & SGPT mild to moderate ranging from 40-250u/l. T.bilirubin > 1.1mg% was seen in 64% highest recorded value in this study is 16.50mg%. D.bilirubin >2.0mg% is seen in 80% of the cases. Highest recorded value of D.bilirubin in this study is 10.8g%. SGOT is >40u/l in 68% of the cases. Highest recorded value of SGOT in this study 900u/l. SGPT is elevated > 40u/l in 68% of the cases. Highest recorded value of SGPT in this study is 920u/l. Alp is elevated > 147u/l in 30% of the cases. In Madras study 84% of the cases had high bilirubin (>2mg%) and abnormal SGOT & SGPT (>50u/l).

Urine examination showed albumin present in 48% of the cases. RBCs present in 38% of the cases. Cells present in 66% of the cases. Casts seen in 16% of the cases. All the patients were seen positive for IgM antibodies to leptospira, of these 30% of the patients showed 11-12 PBU, 70% of the patients showed > 12 PBU. Highest recorded is 61 PBU. Normal PBU value less than 9, equivocal 9-11 PBU, more than 12 PBU consider leptospira positive.

Only 4 patients (8%) have shown abnormalities in chest X-Ray. 1 patient (2%) showed features of CCF. 3 patients (6%) showed features of ARDS. ABG was done showing 6% of patients with metabolic acidosis. 2 patients (4%) had features of Myocarditis.

LP CSF analysis was done; it shows mild elevation of proteins, normal glucose and normal cell count in most of the cases.

In our study of 50 patients all of them had fever of various duration as such they were given paracetamol and intravenous fluids.

98% of the patients received Crystalline penicillin for >5 days, 2% of the patients received ciprofloxacin. 76% were given steroids and platelet rich plasma in these patients platelet count improved within 5 days.

8% of the patients received blood transfusion.

In our study of 50 patients 68% developed complications 32% of patients had no completions. Of these 68% of the patients 8% patients had acute renal failure, 14% had

Hepatic damage, 34% had both acute renal failure and Hepatic damage, 4% developed Hepatic damage & Altered sensorium, 2% had Myocarditis, Hepatic damage with Acute renal failure, 4% had ARDS, ARF & Hepatic damage. 2% developed ARF, ARDS< Myocarditis, Hepatic damage.

Out of 50 patients 46% of the patients developed multi organ damage (2 or more organs are involved). 2 patients (4%) died due to ARDS, ARF & Hepatic damage. 80% of the patients stayed at the hospital for less than 10 days.

CONCLUSION

84% of the patients had Hepatomegaly, 58% patients had Splenomegaly. 80% of the patients had Thrombocytopenia. 50% of the patients had acute renal failure. 60% of the patients had Hepatic damage. 4% of the patients had altered sensorium, 6% of the patients had ARDS. 46% of the patients developed Multi organ damage. Crystalline penicillin is the drug of choice in leptospirosis. 4% of the patients died due to ARF, ARDS& Hepatic damage. 80% of the patients stayed at hospital for less than 10 days. Early diagnosis & initiation of treatment of leptospirosis prevents mortality and morbidity.

REFERENCES

1. William. A. Petri Jr. XXII Infectious diseases. Chapter 396. Harcourt, Asia: Cecil Textbook of Medicine, 21st Edition, W.B. Saunders Company; 1761-2.
2. Watt G, Tuazon ML. Placebo controlled trial of intravenous penicillin for severe and late leptospirosis. *Lancet* 1998;20:433-5.
3. Martone WJ, Kaufman AF. Leptospirosis in human in the United States. *J Infect Dis* 1979;43:1020-2.
4. Torten M, Shenberg E, Geichter CB. A new leptospiral vaccine for use in man clinical and serologic evaluation of a field trial of volunteers. *J Infect Dis* 1973;56:647-51.
5. Merian F, Perolat P. Public health importance in south pacific a five year study in New Caledonia. *Am J Trop Med Hyg* Aug 1996;23:174-8.
6. Wesley F. State of the art—Clinical article on leptospirosis. *Clinical Infectious disease*, 1995;15:1-8.
7. Johnson WD, Silva IC, Rocha H. Serum creatinine phosphokinase in leptospirosis. *JAMA* 1975;63:981-2.
8. De Brito T, Morais CF, Yasuda PH. Cardiovascular involvement in human and experimental leptospirosis: Pathologic findings and immunohisto-chemical detection of leptospiral antigen. *Ann Trop Med Parasitol*. 1987;79:207-4.
9. Faine S. Guidelines for the control of leptospirosis. Offset publication 67, Geneva, World Health Organization; 1982.
10. Sitprija V, Evans H. The kidney in human leptospirosis. *Am. J. Med.* 1970;19:780-8.
11. Rajiv C, Manjuran RJ. Cardiovascular involvement in leptospirosis. *Indian Heart Journal* Nov-Dec 1998;29:691-4.