*ejpmr, 2018,5(9), 355-357*

SJIF Impact Factor 4.897

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Case Report

ISSN 2394-3211

EJPMR

# CASE REPORT ON LEPTOSPIROSIS IN A PEDIATRIC PATIENT

***Shilpa P. P., Niranjana Syam, Akshay T. S., Athira R. S., Sreemanju M. S. and Sarath S. M.**

India.

***Corresponding Author: Shilpa P. P.**

India.

**Article Received on 21/06/2018** **Article Revised on 11/07/2018** **Article Accepted on 31/07/2018**

**ABSTRACT**

Leptospirosis is a bacterial infection spread by contaminated water, urine by infected animals and it affects both human and animals. it is commonly seen in monsoon season and here we report an anicteric case of leptospirosis in a 10 year old female.

**KEYWORDS:** Leptospirosis.

## INTRODUCTION

Leptospirosis is a common zoonosis worldwide that affects mammals, including human beings. Infection is endemic and occurs with greatest frequency in tropical and subtropical regions. It is emerging as an important public health problem in India.[1,3] Both humans and animals can be directly infected through contact with infected tissue or urine or indirectly through contact with contaminated soil and water.[4,5] In humans, typical symptoms can include fever, headaches, chills, vomiting, sore muscles, jaundice, red eyes, abdominal pain, diarrhea, and rashes. Leptospirosis can become considerably dangerous if not treated, potentially leading to kidney damage, meningitis, liver failure, and respiratory problems.[5]

Leptospirosis has been recognized as an important occupational hazard of agriculture manual laborers, sewage workers, animal handlers, forestry workers and other outdoor workers who work in wet conditions, and butchers. The transmission cycle involves interaction between one or more animal hosts harboring leptospires, an environment favorable for its survival, and human beings. Leptospirosis in human occurs in two courses: anicteric or benign (between 85 and 90% of cases) and icteric or serious, also known as Weil's diseases (between 10 and 15% of cases). The wide spectrums of clinical symptoms that characterize leptospirosis make its diagnosis easily confused with other febrile diseases.[6]

## CASE REPORT

A previously healthy 10-year-old female child who had suffered from sore throat,cough, rhinitis, headache(diffuse type), vomiting(non-bilious and non-projectile), high grade intermittent fever, malaise, low abdominal pain and loss of appetite for 3 days and also visited a local clinic where common cold and fever was diagnosed and she was treated accordingly. Her symptoms were temporarily relieved at that time. The symptoms reappeared and the patient was admitted to OP department of Pediatrics on the 12$^{th}$ day and she developed a new symptom ie, congestion of left eye for 2days. Patient lost 2kg in past 2 weeks.

The patient had a history of eating fruit salad from outside. On examination patient's respiratory system, cardiovascular system were normal, tenderness over the abdomen and CNS showed no neck signs throat was congested, halitosis was present. Hematology evaluations showed a peripheral white cell count of 6620/microLitre, DC-N63%, L-33%, M-4%, Hemoglobin 12.9g/dL (normal,13-15g/dl), and a platelet count of 26,0000/cumm (normal, 150,000–450,000). ESR was 21mm/Hr. Blood chemistry showed elevated blood urea 18mg/dl(normal,15-45mg/dl) creatinine (Cr) of 0.4 mg/dL (normal, 0.5–1.4 mg/dL), alanine aminotransferase (ALT) of 17 IU/L (normal, < 40.0 IU/L), CRP: 0.44mg/dl(normal, upto 0.5). Peripheral smear showed few reactive lymphocytes. LeptospiralIgM(ELISA) was equivocal -10.5 and repeated after 3 days to demonstrate the rise in titre and it was positive with value 14. On the day 1 she was treated with infusion of DNS 500ml over 8 hours and 1 ampule of polybion along with it. Inj. Pantoprazole 25mg IV OD, Inj.emest 2.5mg IV SOS, Inj.ceftriaxone 1g IV BD, Syp.oseltamevir 4ml BD, T.Azithromycin 500mg OD, Syp. Acetaminophen (250mg/5ml) 7.5ml Q6H. Syp. ASCORIL LS (ambroxol+guaifenesin+levalbuterol) 5ml TID, Tobramycin 0.3% eye drop 1 drop 4 times in left eye and 1 drop 2 times in right eye and lid hygiene was advised. The same orders were repeated on the day 2,3,4,5 and on day 5 Syp. Oseltamevir was stopped, her vitals were stable, she was symptomatically better and active hence discharged and also advised to continue Tobramycin 0.3% eye drop for 4days and

Syp.Zincovit(multivitamin supplement) for 10days and asked to attend the OPD after week for review.

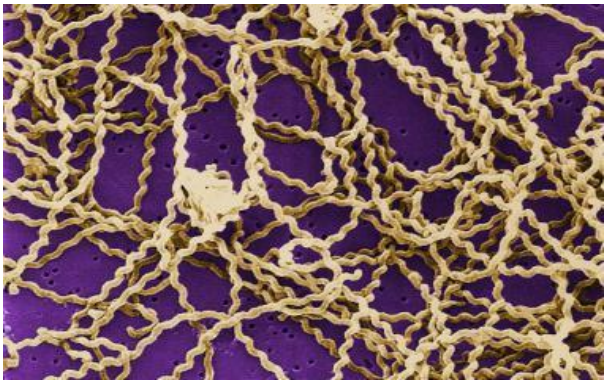
## DISCUSSION

Leptospirosis is distributed worldwide (sparing the polar regions) but is most common in the tropics. Leptospirosis has been recognized as a re-emerging infectious disease among animals and humans.[7] Most leptospiralserovars have a primary reservoir in wild mammals, which continually reinfect domestic populations. The organism affects at least 160 mammalian species and has been recovered from rats, swine, dogs, cats, raccoons, cattle, mongooses, and bandicoots.[8,9]

Urinary shedding of organisms from infected animals is the most important source of these bacterial pathogens. Contact with the organism via infected urine or urine-contaminated media results in human infection. Such media include contaminated water and food, as well as animal bedding, soil, mud, and aborted tissue. Under favorable conditions, leptospires can survive in fresh water for as many as 16 days and in soil for as many as 24 days.[10]

This syndrome, icteric leptospirosis with renal failure, was first reported over 100 years ago by Adolf Weil in Heidelberg.[11] However, an apparently identical syndrome occurring in sewer workers was described several years earlier.[12,13] Earlier descriptions of diseases that were probably leptospirosis were reviewed recently.[14,15] Leptospirosis was certainly recognized as an occupational hazard of rice harvesting in ancient China[15], and the Japanese name akiyami, or autumn fever, persists in modern medicine. With hindsight, clear descriptions of leptospiral jaundice can be recognized as having appeared earlier in the 19th century, some years before the description by Weil.[15]

Leptospires have a typical double membrane structure in common with other spirochetes, in which the cytoplasmic membrane and peptidoglycan cell wall are closely associated and are overlain by an outer membrane.[16] Leptospiral lipopolysaccharide has a composition similar to that of other gram-negative bacteria[17], but has lower endotoxic activity.[18] Leptospires may be stained using carbol fuchsin counterstain.[15]



A scanning electron micrograph depicting Leptospira atop a 0.1-µm polycarbonate filter. (This image is in the public domain and thus free of any copyright restrictions. Courtesy of the Centers for Disease Control/Rob Weyant).

Icteric leptospirosis is a much more severe disease in which the clinical course is often very rapidly progressive. Severe cases often present late in the course of the disease, and this contributes to the high mortality rate, which ranges between 5 and 15%. Between 5 and 10% of all patients with leptospirosis have the icteric form of the disease.[19] The jaundice occurring in leptospirosis is not associated with hepatocellular necrosis, and liver function returns to normal after recovery.[20] Serum bilirubin levels may be high, and many weeks may be required for normalization.[21] The classic form of severe leptospirosis is known as Weil's disease, which is characterized by liver damage (causing jaundice), kidney failure, and bleeding.[22] The infection is often incorrectly diagnosed due to non-specific symptoms.

## CONCLUSION

It is a challenge to diagnose the infection of Leptospira because of the diverse presentations and as it mimic the symptoms of common flu to multi-organ failure. The most common triad is fever, jaundice, and acute renal failure. Early awareness and detection can save the patients. Effective rat control and avoidance of urine contaminated water sources are essential preventive measures against leptospirosis.

## REFERENCES

1. S. C. Sehgal, "Leptospirosis on the horizon," The National Medical Journal of India, 2000; 13(5): 228–230.
2. "Leptospirosis in India—report of the investigation of a post-cyclone outbreak in Orissa, November 1999," Weekly Epidemiological Record, 2000; 75: 217–223.
3. S. Ratnam, "Leptospirosis: an Indian perspective," Indian Journal of Medical Microbiology, 1994; 12: 228–239.
4. E. S. Faine, A Brief Overview of the Disease, Leptospirosis, CRC Press, Boca Raton, Fla, USA, 1994.
5. R. W. Farr, "Leptospirosis," Clinical Infectious Diseases, 1995; 21(1): 1–8.
6. P. Perolat and P. A. Reeve, "First evidence of leptospirosis in Vanuatu," Transactions of the Royal Society of Tropical Medicine and Hygiene, 1992; 86(5): 557–559.
7. Yang CW. Leptospirosis in Taiwan--an underestimated infectious disease. Chang Gung Med J., 2007 Mar-Apr.; 30(2): 109-15. [Medline].
8. GILLESPIE RW, RYNO J. Epidemiology of leptospirosis. Am J Public Health Nations Health, 1963 Jun.; 53: 950-5.[Medline].

9. GB, Nimmanitya S, Karnchanachetanee C, Tingpalapong M, Samransamruajkit S, Hansukjariya P, et al. Epidemiology and characterization of leptospirosis at an urban and provincial site in Thailand. Southeast Asian J Trop Med Public Health, 1988 Jun.; 19(2): 317-22. [Medline].
10. SMITH DJ, SELF HR. Observations on the survival of Leptospira australis A in soil and water. J Hyg (Lond)., 1955 Dec.; 53(4): 436-44.[Medline].
11. Weil A. Ueber eine eigentümliche, mit Milztumor, Icterus und Nephritis einhergehende akute Infektionskrankheit. Dtsche Arch Klin Med., 1886; 39: 209–232.
12. Landouzy L T J. Fièvre bilieuse ou hépatique. Gaz Hôpital, 1883; 56: 809. [Ref list].
13. Landouzy L T J. Typhus hépatique. Gaz Hôpital, 1883; 56: 913.
14. Everard J D. Leptospirosis. In: Cox F E G, editor. The Wellcome Trust illustrated history of tropical diseases. The Wellcome Trust, London, U.K., 1996. pp. 111–119., 416–418. [Ref list].
15. Faine S. Leptospira and leptospirosis. Boca Raton, Fla: CRC Press, 1994.
16. Haake D A. Spirochaetal lipoproteins and pathogenesis. Microbiology, 2000; 146: 1491–1504.
17. Vinh T, Adler B, Faine S. Ultrastructure and chemical composition of lipopolysaccharide extracted from Leptospira interrogans serovar copenhageni. J Gen Microbiol, 1986; 132: 103–109. [PubMed].
18. Shimizu T, Matsusaka E, Takayanagi K, masuzawa T, Iwamoto Y, Morita T, Mifuchi I, Yanagihara Y. Biological activities of lipopolysaccharide-like substance (LLS) extracted from Leptospira interrogans serovar canicola strain Moulton. Microbiol Immunol, 1987; 31: 727–735. [PubMed] [Ref list].
19. Heath C W, Alexander A D, Galton M M. Leptospirosis in the United States: 1949–1961. N Engl J Med., 1965; 273: 857–864., 915–922.
20. Ramos-Morales F, Díaz-Rivera R S, Cintrón-Rivera A A, Rullán J A, Benenson A S, Acosta-Matienzo J. The pathogenesis of leptospiral jaundice. Ann Intern Med., 1959; 51: 861–878.
21. Edwards C N, Nicholson G D, Hassell T A, Everard C O R, Callender J. Leptospirosis in Barbados: a clinical study. West Indian Med J., 1990; 39: 27–34.
22. Ko AI, Goarant C, Picardeau M (October 2009). "Leptospira: the dawn of the molecular genetics era for an emerging zoonotic pathogen". Nature Reviews Microbiology, October 2009; 7(10): 736–47. doi:10.1038/nrmicro2208. PMC 3384523 .PMID 19756012.