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A CASE OF LEPTOSPIROSIS WITHOUT RENAL INVOLVEMENT

General Medicine

Dr Amandeep Singh Kaloti Professor Medicine.

ABSTRACT

Leptospirosis is caused by spirochaete bacteria belonging to the genus *Leptospira*. 21 species of *Leptospira* have been identified. 13 species cause disease or have been detected in human cases. Signs and symptoms can range from none to mild such as headaches, muscle pains, and fevers; to severe with bleeding from the lungs or meningitis. If the infection causes the person to turn yellow, have kidney failure. Severe manifestations include extreme fatigue, hearing loss, respiratory distress, and azotemia. Here I report a case of leptospirosis which did not have any renal involvement and responded to the treatment.

KEYWORDS

INTRODUCTION:-

Leptospirosis is an infection caused by corkscrew-shaped bacteria called *Leptospira*. Signs and symptoms can range from none to mild such as headaches, muscle pains, and fevers; to severe with bleeding from the lungs or meningitis. If the infection causes the person to turn yellow, have kidney failure and bleeding, it is then known as **Weil's disease**. If it also causes bleeding into the lungs then it is known as **severe pulmonary hemorrhage syndrome.** It is often transmitted by animal urine or by water or soil containing animal urine coming into contact with breaks in the skin, eyes, mouth, or nose. In the developing world the disease most commonly occurs in farmers and low-income people who live in cities. In the developed world it most commonly occurs in those involved in outdoor activities in warm and wet areas of the world. Diagnosis is typically by looking for antibodies against the bacterium or finding its DNA in the blood.[1]

Case Report:-

The patient, a female aged 21 years, married, presented in the emergency department with chief complaint of –

Fever for the past 06 days,
Yellowish discoloration of eyes for 06 days,
Dyspnoea for the past 04 days,
Cough for the past 04 days,
Generalized weakness for 04 days.

The patient was alright about 06 days back from admission, when she started experience fever which was of moderate to high grade and was associated with chills and rigors. It was continous in nature with no history of any evening rise of temperature. Patient also had h/o yellowish discolouration of the eyes which was gradual in onset and was progressive in nature. There was history of loss of appetite. There was no history of any altered sensorium. Then later the patient started experiencing dyspnoea for the past 04 days before admission. Patient used to get breathless even on doing her routine activities. It was progressive in nature. Patient also had a history of cough which was productive in nature, with production of yellow coloured sputum which was small to moderate in amount,. There was no history of haemoptysis. Patient complained of generalized weakness which occurred during the illness. She felt extreme prostration in doing her daily activities.

There was no history of similar complaints in the past. There was no previous history of diabetes mellitus, hypertension, coronary heart disease, bronchial asthma or tuberculosis. There was no family history of diabetes mellitus, hypertension, coronary heart disease, bronchial asthma and there was no history of similar complaints in the family. The patient was vegetarian and had no addictions, bowel and bladder were normal. The menstural history of the patient was normal. The socio-economic history showed that the patient belonged to the lower income class.

On general physical examination, the patient was conscious, cooperative and lying comfortably in the bed. Patient was in poor general condition and was febrile with a temperature of 101^{0}F. The pulse rate was 108 per minute, regular, normal volume, normal character. The respiratory rate was 28 per minute and was thoracoabdominal in nature. The BP was 130/80 mm Hg in the right arm in the supine position. Patient had pallor, icterus and had conjunctival congestion and suffusion. There was no evidence of any ecchymosis or haemorrhages. On respiratory system examination, diminished breath sounds in the right infrascapular, infraaxillary regions. Crepitations were also present in these regions. On per abdomen examination, the patient was found to have hepatomegaly of 1 finger below the right costal margin in midclavicular line. Other system examination were normal. Later on, the patient started complaining of diplopia and developed dysarthria, palsy of the right VII, IX, X CNs and the GCS was 12. Patient had developed a rash which was macular/morbilliform all over.

Investigation done showed :-

PT(INR):-1.44
Dengue Serology :- Negative
WHWR WVR
Blood Grouping, Rh typing: B +ve
Viral Markers – Negative
Typi Dot:- Negative

ABG done showed evidence of hypoxia with a low oxygen saturation. Later-on, the ABG was normal.

CSF :- Colour – Pale Yellow
Deposits :- Clear, No clot
TLC :- 16 cells/mcl,
few lymphocytes and few RBCs
Protien :- 149 mg%
Glucose :- 57 mg%
ADA :- WNL

Leptospira :- IgM :- 28.66 (neg <15) U/ml
IgG :- 4.97 (neg <9) U/ml
RBS - 123 mg%
ECG :- showed evidence of sinus tachycardia.

Chest X-ray:- showed evidence of non-homogenous opacities in the right lower zone and blunting of the right CP angle.

Patient was started on treatment and was given IV fluids, Inj ceftriaxone 2 gm IV BD,

Inj Doxycycline 100 mg IV BD, Inj Pantoprazole 40 mg IV BD, Inj Ondansetron 4 mg IV BD, Inj Ofloxacin 200 mg IV BD, Syp Lactulose 3 tsf BD, Lactulose enema,

Tab Shelcal Tab Rifaximin Cap Becosule
O2 inhalation
Tab Livogen Blood Transfusion

The patient gradually responded to the treatment and improved over the next few days with the treatment given. The sepsis and the hepatitis part of the infection improved. The multiple CN palsies also improved

and the patient was discharged in good condition and was ambulant and doing all her routine activities independently.

DISCUSSION:-

Leptospiral infection in humans causes a range of symptoms, and some infected persons may have no symptoms at all. Leptospirosis is a biphasic disease that begins suddenly with fever accompanied by chills, intense headache, severe myalgia (muscle ache), abdominal pain, conjunctival suffusion (red eye), and occasionally a skin rash. The symptoms appear after an incubation period of 7–12 days. The first phase (acute or septic phase) ends after 3–7 days of illness. The disappearance of symptoms coincides with the appearance of antibodies against *Leptospira* and the disappearance of all the bacteria from the bloodstream. The patient is asymptomatic for 3–4 days until the second phase begins with another episode of fever. The hallmark of the second phase is meningitis (inflammation of the membranes covering the brain).

Ninety percent of cases of the disease are mild leptospirosis. The rest experience severe disease, which develops during the second stage or occurs as a single progressive illness. The classic form of severe leptospirosis is known as Weil's disease, which is characterized by liver damage (causing jaundice), kidney failure, and bleeding. Additionally, the heart and brain can be affected, meningitis of the outer layer of the brain, encephalitis of brain tissue with same signs and symptoms; and lung affected as the most serious and life-threatening of all leptospirosis complications. The infection is often incorrectly diagnosed due to the nonspecific symptoms.

Other severe manifestations include extreme fatigue, hearing loss, respiratory distress, and azotemia. On infection the microorganism can be found in blood and cerebrospinal fluid (CSF) for the first 7 to 10 days (invoking serologically identifiable reactions) and then moving to the kidneys. After 7 to 10 days the microorganism can be found in fresh urine. Hence, early diagnostic efforts include testing a serum or blood sample serologically with a panel of different strains. [2]

Kidney function tests (blood urea nitrogen and creatinine) as well as blood tests for liver functions are performed. The latter reveal a moderate elevation of transaminases. Brief elevations of aspartate aminotransferase (AST), alanine aminotransferase (ALT), and gamma-glutamyltransferase (GGT) levels are relatively mild. These levels may be normal, even in children with jaundice.

Diagnosis of leptospirosis is confirmed with tests such as enzyme-linked immunosorbent assay (ELISA) and polymerase chain reaction (PCR). The MAT (microscopic agglutination test), a serological test, is considered the gold standard in diagnosing leptospirosis. As a large panel of different leptospira must be subcultured frequently, which is both laborious and expensive, it is underused, especially in developing countries.

Differential diagnosis list for leptospirosis is very large due to diverse symptoms. For forms with middle to high severity, the list includes dengue fever and other hemorrhagic fevers, hepatitis of various causes, viral meningitis, malaria, and typhoid fever. Light forms should be distinguished from influenza and other related viral diseases. Specific tests are a must for proper diagnosis of leptospirosis.

Under circumstances of limited access (e.g., developing countries) to specific diagnostic means, close attention must be paid to the medical history of the patient. Factors such as certain dwelling areas, seasonality, contact with stagnant contaminated water (bathing, swimming, working on flooded meadows, etc.) or rodents in the medical history support the leptospirosis hypothesis and serve as indications for specific tests (if available). [3]

CONCLUSION:-

Leptospirosis is a life threatening disease with many complications. The clinical manifestations vary in each and every person. Some of these patients may not have the renal involvement. This should be recognised as the diagnosis may be missed in these cases of leptospirosis which is otherwise a treatable disease.

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