Acute pulmonary hypertension in a patient with leptospirosis

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Introduction

Leptospirosis is a zoonosis which is caused by leptospira species, mainly by Leptospira Interrogans. It shows a worldwide distribution mainly in tropics with exclusion of Polar Regions. Natural hosts are various mammals while man is accidentally infected after exposure to the environment contaminated with animal urine.

After an incubation period of 2 - 26 days, it may manifest as a self-limiting infection or a potentially fatal illness accompanied by multi-organ failure. The clinical course may be complicated by acute renal failure, uveitis, hemorrhage, ARDS, myocarditis, meningism and rhabdomyolysis.

We are reporting a clinical case which was complicated by reversible cardiomegaly and severe pulmonary hypertension. To our knowledge a case of this nature has not been reported in the local literature.

Case Report

A 58 year old previously healthy male from Galle was admitted with a history of fever, shortness of breath, chest pain at rest and diarrhoea of 3 day duration.

On admission, the patient was extremely ill and hemodynamically unstable with an elevated Jugular Venous Pressure. He was severely dyspnoeic and had crepitations over both lung fields.

ECG showed curved ST segment elevations in several leads. Arterial blood gas showed a type I respiratory failure. Chest X-ray revealed cardiomegaly and dilated pulmonary arteries suggestive of pulmonary hypertension (Fig 1).

Cardiac 2DEcho showed moderate to severe pulmonary hypertension with marked dilatation of the main pulmonary artery (diameter of 26 mm).

Our presumptive differential diagnoses were Community Acquired Pneumonia with myocarditis and Leptospirosis complicated by pneumonitis and myocarditis.

The patient was started on IV Cefotaxime 1g tds and IV Clarithromycin 500 mg bd empirically to cover both infections.

Initial hematological investigations revealed a neutrophil leucocytosis with a platelet count of 20,000 with a PCV of 37%. He had no bleeding manifestations. Urine full report showed red cells and granular casts. Though his renal and liver function tests remained normal, a high degree of clinical suspicion of leptospirosis was entertained.

As the patient’s respiratory symptoms progressed with worsening of hemodynamic instability, he required ITU care with inotropic support and intubation. After 5 days of ITU care, the patient was transferred back to the ward and antibiotics were continued for a further one week.

Gradually his clinical condition improved. After full recovery a repeat chest X-ray and a 2DEcho was done to assess the improvement. Amazingly, the chest X-ray and the 2DEcho were completely normal. There was no evidence of pulmonary hypertension (Fig 2).

Ultimately clearing our doubts regarding the diagnosis, the leptospira antibody test was positive with a very significant titre of 3,200.
Discussion

As already discussed, leptospirosis is associated with a variable clinical course. Mainly it is an asymptomatic self-limiting infection. Sometimes it may manifest as a potentially fatal infection. Abrupt onset of fever with rigors, myalgia and headache are the main presenting features in 75 - 100% of patients.

Acute renal failure, haemorrhagic manifestations, adult respiratory distress syndrome, myocarditis, liver involvement and rhabdomyolysis are some of the well known complications of leptospirosis. Though known to occur, liver failure usually does not cause death. Vasculitis with necrosis of extremities may be seen in severe cases.

The potential severity of leptospirosis was illustrated in a retrospective study of 60 patients requiring ICU care in India. The total mortality was found to be 52%.

Severe pulmonary manifestations such as hemorrhage and ARDS are well recognized complications. In another study in Peru among 321 patients with serological and clinical evidence of leptospirosis, severe pulmonary manifestations were seen in 3.7% of patients. Five out of seven patients died from pulmonary hemorrhage and ARDS.

Only one case was reported with reversible cardiomegaly with pulmonary hypertension in a study done in North America. This has occurred after penicillin therapy and was attributed to a Jarisch Herxheimer reaction to penicillin.

Leptospirosis is mainly a clinical diagnosis. Full blood count usually reveals a neutrophil leucocytosis with a low platelet count while urinalysis show proteinuria, pyuria, granular casts and microscopic hematuria. Elevated creatine kinase is found in 50% and may be useful as a diagnostic clue. Culture and PCR also assist in the diagnosis but is successful in only 50% of cases.

The gold standard serological test is considered to be microscopic agglutination test. It is most specific when a four fold or greater rise in titre is detected. In our clinical case, the titre was 3200, which is very significant. The convalescent sample also showed the same titre. The titre was normal in the second sample taken one month after obtaining the first convalescent sample.

When we consider the therapy of leptospirosis, penicillins, tetracyclines, chloramphenicol and erythromycin have antileptospiral activity. Currently the widely used antibiotics are penicillin, third generation cephalosporins such as cefotaxime and ceftriaxone and doxycycline.

According to recent studies, the above antibiotics are therapeutically equivalent. Our patient was treated with IV cefotaxime.

The prophylactic use of doxycycline 200 mg / week, 2-3 weeks prior to exposure and a 200mg stat dose after the exposure is a very important preventive method for this potentially fatal illness.
Drowning following a lightening strike

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Case report
A body of a 30 year old male was found floating in the sea around 5.30pm, on a rainy day with thunder and lightning. When he was last seen alive (at 4.30pm), he had been fishing as usual on a rock in the sea, at Ratgama. He was brought to the Teaching Hospital, Karapitiya (THK) and confirmed dead at the OPD.

Examination of clothing - The front of the T-shirt was irregularly torn and the edges were wrinkled from the level of the neck downwards leaving a deficiency of about 30cm x 20cm. The back of the T-shirt was stained with blood. The short trousers were intact. Pockets did not contain mobile phones or any metal objects.

External examination - The face was congested, and whitish fine froth was seen on the nostrils and a bloody discharge was seen at the mouth. There were no petechiae in head or face. There were no metal ornaments.

Examination of the injuries
Injury No. 1 - A linear split laceration 1 x 0.3 cm was situated on the left eye brow with oozed blood. There was no evidence of burning of skin or singeing of hair.

Injury No. 2 - An irregular fresh 1st degree burns associated with singeing and loss of hair was placed on the back of the head with an irregular fresh laceration measuring 3x0.5 cm situated on its centre. An underlying scalp contusion measuring 6x4 cm was seen. There were no injuries involving skull, meninges or brain.

Injury No. 3 - A wide spread continuous 1st and 2nd degree burns were seen over the front of the body from the lower neck and shoulders up to the upper half of both thighs. The upper end of the burn showed some charring. Several other multiple punctate burns ranging from 0.5x0.5cm to 3x2cm were scattered mainly on the upper abdomen and on the right and left flanks. An ash coloured branching discoloration resembling a 1st degree burn was seen in between and surrounding those burns. The burn injuries were associated with marked singeing of hair (Fig 1).

Internal examination - Trachea & bronchi contained frothy fluid mixed with blood, sea sand and water. The lungs were seen distended, but neither filled the thoracic cavity nor covered the pericardial sac markedly. No rib markings were seen. The lungs appeared moderately overinflated and purplish blue in colour. They were less crepitant, and while cutting the trachea

References