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LEPTOSPIROSIS IN PREGNANCY: A MATERNAL NEAR MISS CASE

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ABSTRACT We report the case of a 21 year old primipara presenting with primary postpartum haemorrhage with features of hepatocellular jaundice, later diagnosed to be due to leptospirosis on serology. AFLP and HELLP syndrome being the most common differential diagnosis in case of hepatorenal involvement, were ruled out. There was intrauterine fetal death with PPH occurring post expulsion of the fetus. She was treated with IV ceftriaxone and oral doxycycline along with supportive management. She was discharged well after 20 days of admission. Herein we discuss the patient's clinical progression and management of hepatorenal involvement in leptospirosis and review the recent literature.

KEYWORDS : Leptospirosis, pregnancy, hepatorenal

INTRODUCTION

Leptospirosis is a bacterial zoonosis caused by pathogenic Leptospira species which are transmitted to humans by exposure to water containing the urine of infected mammals, predominantly rodents[1]. The disease occurs worldwide and over 853,000 cases and 48,000 deaths are estimated to occur each year[2]. In India, thirty-eight acute renal failure cases with clinical suspicion of leptospirosis were screened from July to November, 1996 and 27 (71%) seropositive cases were diagnosed by MAT[3]. The disease was considered inconsequential till recently, but it is emerging as an important public health problem during the last decade or so due to sudden upsurge in the number of reported cases and outbreaks. Leptospirosis in pregnancy may mimic hepatorenal failure following PIH or septicaemia or malaria. More than 90% of symptomatic mothers have mild disease and full recovery will occur. Following severe infection, intrauterine fetal death, stillbirth, and congenital leptospirosis may occur[4]. Data on leptospirosis in pregnancy is scant. Most of the reported cases resulted in death of the foetus and is rarely associated with maternal mortality[4-11]. Here we report such a case of leptospirosis in pregnancy resulting in intrauterine fetal death but a complete maternal recovery.

CASE REPORT

The patient was a 21year old with no prior medical or surgical history. She has been married since 2 years, this being her first pregnancy, she had 2 antenatal checkups at 25 and 34 weeks of pregnancy. Both of them were routine checkups without any complaints and was managed conservatively. At 36 weeks gestational age, she attended a nearby ANC clinic with complaints of vomiting, abdominal pain and yellowish discoloration of sclera for 2 days. For this, she was advised investigations and was started on oral ursodeoxycholic acid (300) twice daily. She reported to the clinic after 3 days with the reports and increasing jaundice. She was advised hospitalisation but declined the same. Her investigations revealed s.bilirubin 3.9mg% (total) and 1.3mg% (direct), urine being positive for bile salts and pigments. Malarial parasite was negative. 3 days later, she reported to the local hospital with intensifying pain and jaundice. She delivered a stillborn male baby vaginally the same day. Following delivery, there was atonic PPH for which she received injectable carboprost and syntocinon and a unit of blood transfusion. Intrauterine pressure gauze was given and the she was referred to SCBMCH for further management.

Thereby, she was received in our labour room with the complaints of jaundice since last 8 days and bleeding per vaginum since last 8 hours post-delivery. She had no signs of impending eclampsia like headache or blurring of vision. She also did not have any localising symptoms of systemic infection. On admission, the patient was conscious, oriented and afebrile (temperature-36.6 degree Celsius). She was icteric, with moderate pallor, without any oedema and BP of 110/70 mm of Hg associated with tachycardia (HR- 107/min). Her abdomen was soft, non-tender. Uterus was well contracted with size of around 26weeks. On inspection of vulva, one soaked vaginal gauze pack was in situ, the patient was catheterised and about 200ml of high-coloured urine collected.

Based on the clinical examination, she was diagnosed a case pf primary postpartum haemorrhage with hepatocellular jaundice. All routine investigations were sent, she was started on IV fluids with syntocinon drip and exploration of the uterine cavity was done. Three vaginal packs were removed, uterine cavity was explored and products of conception was found. Right cervical angle tear with mild bleeding was present which was repaired. Multiple oozing sites were identified for which a pressure gauze was given and was removed after 24 hours. Post exploration, the patient was transfused with one unit of blood, was kept on oxytocics for next 6 hours and IV ceftriaxone was started empirically. She was continued with ursodeoxycholic acid and, syrup lactulose and tablet rifaximin were added up.

Table 1: Investigations done post exploration (PPD2)

VARIABLE	RESULT
Hb (g %)	9.6
TLC (/cmm)	10800
TPC (lacs/cmm)	1.8
Neutrophil (%)	70
Total bilirubin (mg/dl)	14.6 ↑
Direct bilirubin (mg/dl)	11.7 ↑
AST (IU/L)	96
ALT (IU/L)	35
ALP (IU/L)	1012
Prothrombin time (sec)	20.1
Activated partial thromboplastin time (sec)	53.2 ↑
INR	1.81
S. urea (mg/dl)	59 ↑
S. creatinine (mg/dl)	3.1 ↑
S. sodium (mEq/l)	138
S. potassium (mEq/l)	4.6
S. protein (g/dl)	4.6
S. albumin (g/dl)	2.3
Random blood sugar (mg/dl)	82

On the first puerperal day, she was transfused with 4 units of fresh frozen plasma and intramuscular injection of vitamin K 10mg once daily was initiated. Serum fibrinogen was 59.5mg/dl and fibrinogen degradation product was positive by latex agglutination method. Liver function deteriorated further as revealed in the graph on the second puerperal day. Viral serology was negative.

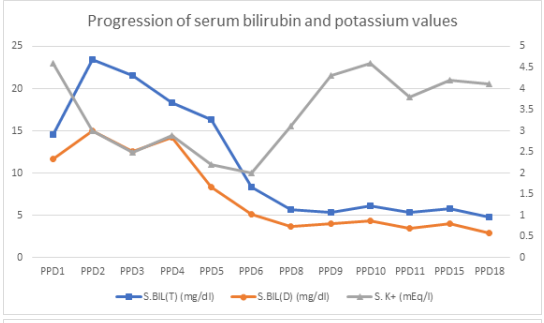
On the third puerperal day, the patient developed mild fever (temperature- 100.6degree F) and abdominal distension with sluggish

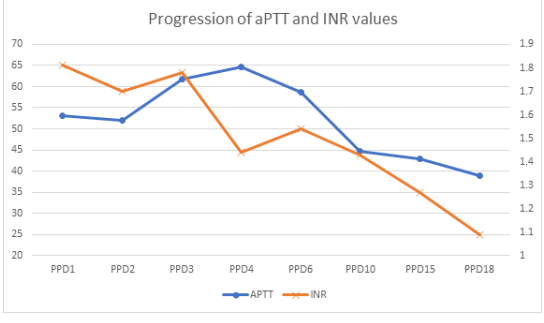
bowel sounds. Though urine output was maintained (≈100ml/ hour), renal function tests were deranged with raised urea and creatinine (s. urea= 60mg/dl, s creatinine- 2mg/dl) and reduced potassium levels (s potassium- 2.5 mEq/l). Medicine, hepatology and nephrology consultations were done. Serial investigations for liver and renal function were planned. Investigations for pyrexia came out negative. Ultrasonography was done which ruled out any retained products of conception, further revealing moderate ascitis, hypoechoic liver shadow (suggestive of hepatitis). Leptospirosis serology was done in view of fever with hepatorenal involvement. The immunoglobulin M levels were raised by enzyme linked immunosorbent assay method, revealing the cause of infection. Intravenous ceftriaxone was continued along with oral doxycycline. For hepatic involvement, intravenous glutathione twice daily was started and vitamin K injections continued. 2 units of fresh frozen plasma each day was infused till normalisation of INR values. Potassium chloride infusion was given with cardiac monitoring by daily ECG for 2 days, then changed to oral form.

On the 6th puerperal day, diagnosis of ascitic fluid revealed sparse cellularity with occasional lymphocytes and mesothelial cells, the Gram stain showing few pus cells but no bacteria. The same treatment was continued and the patient vitals improved though the distension did not subside. Urine and blood cultures showed no growth.

On the 10th puerperal day, the fever spiked to 101 degrees F with abdominal distension (abdominal girth-94cm) and bilateral crepitations while her liver and renal function were returning to normal. On pulmonary medicine consultation, Intravenous meropenem injection was added to her antibiotic regime of ceftriaxone and doxycycline. Ultrasonography of whole abdomen and pelvis revealed puerperal uterus, mild pleural effusion and moderate to gross ascitis. Along with the antibiotics, supportive treatment was continued and the patient improved by 17th puerperal day.

The patient was discharged on the 20th puerperal day, after receiving meropenem for 7 days, IV ceftriaxone and oral doxycycline for 14 days, 2 units of human albumin infusion, 16 units of FFP, and 3 units of blood transfusion. As the baby was stillborn, she was given cabergoline(0.5mg) tablets to control lactation. The patient was advised to continue ursodeoxycolic acid tablets and silymarin capsules for further 2 weeks and to review in hepatlogy department on OPD basis.





DISCUSSION

Most of the leptospiral infections are subclinical or mild and patients recover without any complications. In few cases it may manifest as multiorgan failure leading to maternal mortality and morbidity. This patient contacted the disease in third trimester which resulted in intrauterine death and maternal morbidity.

As in nonpregnant patients, leptospirosis in pregnant women is a biphasic illness. This patient presented to the hospital in the second phase with characteristic liver, kidney and vascular dysfunction. The same features in the 3rd trimester are more consistent with HELLP syndrome and AFLP, which are more common, thus making diagnosis difficult. Gaspari et al. reports a case of leptospirosis without fever during the late stage of pregnancy, mimicking the clinical pattern of HELLP syndrome or AFLP.[12]

In our context, history of poor hygienic conditions, leucocytosis with neutrophilia, significant increase in bilirubin (essentially conjugated) out of proportion to transaminses, and the chronological evolution were all suggestive of leptospirosis.

Treatment with antibiotics remain the primary intervention. In our case, we started the treatment with injectable ceftriaxone and oral doxycycline. As secondary infection was suspected later, because of rise in fever, injectable ceftriaxone was changed to meropenem for 5 more days. Intensive care management is needed to manage severe cases. Close monitoring of fluid electrolyte balance, dialysis for renal impairment, transfusions to control bleeding tendencies and hepatic support are usually required in severe disease. A few cases in the literature have reported that plasma exchange, corticosteroids, and intravenous immunoglobulin may be beneficial in selected patients in whom conventional therapy does not elicit a response.[13,14]

Variable perinatal outcomes are possible including intrauterine fetal death, abortion, healthy newborn or newborn showing signs of active infection. Similar to our case, Baytur et al. reported a case of preterm delivery with fetal loss[6]. Gainder et al. present a case in a pregnant patient who presented with fever, jaundice, coagulopathy, and intrauterine fetal demise[15]. Hicham et al. reported a case where urgent caesearan section was done to save the baby, the baby did not develop signs of active infection[16]. Shaked et al reported leptospirosis in a woman in the second trimester of pregnancy who delivered a healthy baby[11]. These authors also reviewed 15 reported cases of leptospirosis in pregnancy, and found that eight women had abortions, four delivered babies with signs of active infection, two delivered babies that were healthy, and the outcome of one was not stated. They concluded that spontaneous abortion was more likely if the infection occurred in the earlier months of pregnancy, but that termination of pregnancy should not be a consideration in pregnant women with leptospirosis, as congenital infection is rare.

We report a case of leptospirosis in pregnancy mimicking features of HELLP syndrome which is a more common condition. But as cases of leptospirosis are increasing and the disease is still underreported in most parts of our country, it should be considered a differential diagnosis in pregnant women presenting with hepatorenal syndrome.

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