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CASE REPORT

A rare case of Weil's disease in pregnancy - a case report

Anusha Ginjupalli, Naimisha Movva, Rajkumar Owk, Ravi Gowda

Corresponding author: Dr Anusha Ginjupalli, Senior Resident, Department of Obstetrics and Gynaecology, Mamata Medical College, Khammam, Telangana, India; Email – anush617an@gmail.com

ABSTRACT

Leptospirosis is a rare zoonotic disease caused by spirochaete of the genus *Leptospira.* About 5-10% of patients manifest as severe disease known as Weil's disease associated with high fatality. Infection in pregnancy is uncommon and moreover it may mimic viral hepatitis, acute fatty liver in pregnancy (AFLP), pregnancy induced hypertension (PIH) and HELLP (hemolysis, elevated liver enzymes, low platelet) syndrome. A 23 year old, primigravida (34 weeks gestation) who is Rh negative and not in labour was referred to our hospital in view of persistent hypotension and bradycardia. Prior to this, patient had complaints of swelling of lower limbs since 15 days and cough with expectoration, sore throat and dysphagia, fever 1 week ago followed by jaundice, epistaxis and hematochezia. Patient had blood pressure recording of 80/40 mm Hg, pulse -40bpm on admission. On further evaluation she was negative for dengue, malaria but leptospira positive. She was severely anemic with deranged hepatic and renal functions and diagnosed to be disseminated intravascular coagulation (DIC) with decompensatory shock. Preterm lower segment caesarean section (PTLSCS) was done in view of failed induction and a fresh still born baby of weight 2.2 kg was extracted. Patient had severe postpartum hemorrhage (PPH) and due to failed medical management, stepwise devascularisation was performed followed by peripartum hysterectomy and was shifted to ventilator due to persistent hypotension. On the postoperative day 2, patient blood pressure did not pick up and patient died of DIC complications. In pregnant women, early identification of the disease is required to avoid complications as well as fetal and maternal mortality.

Keywords: Leptospirosis, jaundice, disseminated intravascular coagulation (DIC), postpartum hemorrhage (PPH).

Leptospirosis, also known as Weil's disease is the most widespread zoonotic disease in the world caused by bacteria spirochaete of the genus *Leptospira*.[1] Infection occurs through direct or indirect contact and leptospires may enter the body through skin injuries, mucus membrane and conjunctivae exposureor inhalation of microscopic droplets.[2] Upon entering the body, leptospires cross endothelial barrier and cause heamatogenous dissemination by pass through the endothelial cell cytoplasm accounting for broad spectrum of illness. In humans, leptospirosis may cause a wide spectrum of symptoms. Most cases have a biphasic clinical presentation, which begins with the septicemic phase followed by immune manifestations.[3] Severe vascular injury may lead to pulmonary haemorrhage, ischemia of the renal cortex and tubular-epithelial necrosis.[4] Infection in pregnant women is uncommon, however it could lead to severe fetal and maternal mortality. The presentation may mimic other viral, bacterial and parasitic infections, acute fatty liver, pregnancy-induced hypertension, and HELLP syndrome and AFLP (acute fatty liver of pregnancy). Because of its unusual clinical presentation, leptospirosis in pregnancy is often misdiagnosed and under-reported.[5]

Case

A 23 year old woman (primigravida) at 34 weeks gestation with vertex presentation, Rh negative, with decreased fetal movements, not in labour, was transferred

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from a peripheral hospital to our tertiary care centre, in view of severe bradycardia and hypotension. Prior to this, patient had complaints of swelling of lower limbs since 15 days, cough with expectoration, sore throat and dysphagia since 8 days, epistaxis 1 week ago, fever with yellowish discoloration of eyes and urine, and hematochezia since 4 days. She was not a known case of pregnancy induced hypertension (PIH), gestational diabetes mellitus (GDM), thyroid, cardiac, renal abnormalities.

On general and physical examination she was conscious, co-operative, well oriented to time, place and person, found to be febrile with temperature of 100°F, severe icterus with yellowish discoloration of palms and soles, bilateral grade 3 pedal edema and toad skin appearance on whole body. Her pulse rate was 46bpm, feeble, low volume, regularly regular and blood pressure of 80/50 mm hg noted in bilateral upper limbs in sitting position. On systemic examination, uterus 32 weeks size, relaxed, head lower pole, fetal bradycardia with 90-95bpm.Patient attendees have been explained about the grave risk and the need for ICU admission, termination of pregnancy. She was started on noradrenaline iv infusion and immediately blood sample was collected for complete blood picture **(**CBP), serological and biochemical profile.

The investigation reports were as follows, hemoglobin was 6.1 mg/dL, blood urea was 28 mg/dL, serum creatinine was 2.2 mg/dL, total bilirubin was 9.7 mg/dL, direct bilirubin was 8.5 mg/dL, indirect bilirubin 1.2 mg/dL, TC was 9400 cells/mm^3, pH 70,000 cells/mm^3. The partial thromboplastin time (PTT; also known as activated partial thromboplastin time (aPTT)) was >120 sec. AST was 150 IU/L, S. ALT was 170IU/L, ALP-35 IU/L, LDH 280 IU/L, serum sodium 135(mEq/L), serum potassium 4.5(mEq/L), serum chloride 90(mEq/L) serum total protein was 4.5 g/dL, serum albumin was 1.6 g/dL and random blood sugar was120 (mg/dL) (table.1). Urine analysis showed proteinuria (++). Patient was negative for malaria, typhoid, hepatitis and dengue. But, serological test was positive for antibodies (IgM) to leptospira. Ultrasonography (USG) of abdomen and pelvis revealed grade 1 fatty liver and intra uterine fetal demise. Foley's catheter was placed. Patient was administered with injection-pipercillin (4.5gm iv) every 12 hours, injection vitamin K (10 mg) and tablet udoxy (300 mg) 2 times per day. Since the coagulation profile was deranged grossly, blood and blood products transfusion was initiated.2 pints of PCV, 2 pints of fresh frozen plasma (FFP) and 2 pints of cryoprecipitate (cryo) 2 pints RDP (random donor platelets) were transfused. After taking the consent for termination of pregnancy, patient was induced with Foley's catheter followed by syntocinon iv drip.

Table 1: Laboratory results

Variables	On admission	24 hrs after admission	24 hrs after surgery
Hb (g %)	6.1	7.2	5.2
TLC (/cmm)	9400	24000	10680
TPC (/cmm)	70000	43000	19000
Total bilirubin (mg/dL)	9.7	9.2	5.6
Direct bilirubin (mg/dL)	8.5	7.8	4.3
Indirect bilrubin (mg/dL)	1.2	1.4	1.3
AST (IU/L)	150	174	59
ALT (IU/L)	170	113	40
ALP (IU/L)	35	526	62
LDH (IU/L)	280	572	520
Prothrombin time (sec)	>120	39.3	>120
Activated partial thromboplastin time (sec)	>120	74.5	>120
INR	-	3.3	-
Blood urea (mg/dL)	28	25	48
Serum creatinine (mg/dL)	3.0	2.5	3.4
Serum sodium (mEq/L)	135	137	132
Serum potassium (mEq/L)	4.5	4.2	5
Serum chloride (mEq/L)	90	98	98
Serum total protein (g/dL)	4.5	4.3	4.1
Serum albumin (g/dL)	1.6	1.9	2
Random blood sugar (mg/dL)	120	98	130

24hrs after admission patient had complaints of hematemisis, heamoptysis and epistaxis and arrest in progress of labour. Repeat laboratory investigation were performed and results were presented in table 1, and patient was transfused with 2pints of PCV, 4 pints of FFP and 2 pints of RDP. After explaining grave risk and poor prognosis, patient was taken for emergency caesarian section in view of failed induction and severe maternal distress. Emergency LSCS was done and a still born male baby of weight 2.4 kg was extracted.

After the delivery, she had developed severe postpartum haemorrhage. In view of failed medical line of management, stepwise devascularisation was done and, an on table decision was taken to perform peripartum hysterectomy. After achieving complete hemostasis, intraperitoneal drain was kept and abdomen was closed in layers. Patient has been shifted to ICU for further management. On post-operative day 2, patient had persistent hypotension, sub optimal urine output and developed ascites with 24 hr drain output of 1100ml.Her repeat laboratory investigations carried out and results were shown in table.1. She was given 3 pints of PCV, 5 pints of fresh frozen plasma and 5 pints of platelets. Her liver and renal functions, as well as hematological parameters continued to deteriorate. Inspite of best possible resuscitatory efforts, patient succumbed to death after 24 hours of surgery.

Discussion

Leptospirosis is a rare disease in pregnancy. Rodents are the reservoir and humans are not commonly infected. Human infections can occur either by direct contact with urine or faeces of an infected animal or indirectly through contaminated water, soil or vegetation. In majority of cases, leptospiral infections are subclinical or very mild, however a small proportion develops various complications due to multiorgan involvement.[6] Infection in pregnant women is uncommon, but may lead to severe fetal and maternal morbidity and mortality.[2,3] Around 90% of the cases are mild and often present with non-specific symptoms. This patient contracted the disease during the third trimester. Leptospirosis manifests as a biphasic illness; the initial phaseis associated with presence of high fever, headache, chills, myalgia, particularly in lumbar and calf muscles, arthralgia, pharyngitis, non-productive cough, abdominal pain, nausea and vomiting. The fever abates and returns at the start of the second phase. In the next phase present with lymphadenopathy, rash and hepatosplenomegaly associated with circulating IgM antibodies. Around 5-10 % cases with severe haemorrhage leads to icteric leptospirosis/Weil's disease, in which the second phase is characterised by jaundice, meningitis, renal failure, pulmonary haemorrhage, myocarditis and rhabdomyolysis, associated with circulating IgM antibodies with a relatively high fatality rate 20 – 40%. [5,7,8]

This patient contracted the disease in third trimester presented in second phase with classical features of severe leptospirosis: coagulopathy, acute kidney injury, liver dysfunction. The same features in third trimester are more common with HELLP syndrome and AFLP, thus making diagnosis difficult. In our context, h/o poor hygienic condition, fever, significant increase in bilirubin out of proportion to hepatic transaminases, and the chronological evolution were all suggestive of leptospirosis. Normal to slightly elevated serum transmainases even with severe jaundice differentiates it from viral hepatitis. Specific serological tests or PCR (polymerase chain reaction) are necessary to make definite and quicker diagnosis of the disease. The presence of oliguria, hyperkalemia, pulmonary rales, hypotension on admission in patients with leptospirosis indicates high risk of death.[9] Diagnosis of the disease is much important. Treatments with antibiotics remain the primary intervention. In our case, we started the treatment with iv piperacillin and oral doxycycline. ICU management is needed to manage severe cases. Close monitoring of electrolyte balance, transfusions to control bleeding manifestations are required in severe disease. Mechanical ventilation is indicated in cases with respiratory distress. A few cases in the literature have been reported that plasma exchange, corticosteroids and intravenous immunoglobulins may be beneficial in selected patients in whom conventional therapy does not elicit a response. [9,10]

Emergency cesarean section is necessary to save the mother and fetus. Variable perinatal outcomes are possible including abortion, IUFD, healthy newborn or newborn with active infections. Preterm delivery with fetal loss[11] and intra uterine fetal demise were also reported in patients who had severe hepatorenal failure.[12] We report a case of leptospirosis in pregnancy mimicking HELLP and AFLP with both maternal and perinatal mortality. As this disease is still underreported in many parts of our country, it should be considered as differential diagnosis in pregnant women with hepatorenal syndrome.

Clinical significance: Leptospirosis is a rare disease, and due to its nonspecific presentation, is often difficult to diagnose. Hence, a high suspicion index is important for the early diagnosis and treatment in order to avoid complications, especially in pregnant women.

Conclusion

Public awareness about leptospirosis including hygienic methods such as avoidance of direct and indirect human contact with animal urine as well as rodent control is necessary for intervention and control of the disease. Since the disease often has symptoms that overlap with the clinical features of viral hepatitis, obstetric cholestasis, therefore these conditions may be considered in the differential

diagnosis of leptospirosis. In the third trimester of pregnancy, abdominal pain associated with jaundice, hemolysis, increased transaminases, and coagulation abnormalities, HELLP syndrome and AFLP (acute fatty liver of pregnancy) are the probable differentials.

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Anusha Ginjupalli [1], Naimisha Movva [2], Rajkumar Owk [3], Ravi Gowda [4]

[1] Senior Resident in the department of OBG, Mamata Medical college, Khammam, Telangana, India; [2] Associate Professor, Department of OBG, Mamata Medical college, Khammam, Telangana, India; [3] Assistant Professor, Department of Medicine, Mamata Medical college, Khammam, Telangana, India; [4] Professor, Department of OBG, JJM Medical College, Davanagere.