North Asian International Research Journal of Multidisciplinary

CIF IMPACT FACTOR:4.465

ISSN: 2454-2326 Vol. 4, Issue-1 January-2018

Index Copernicus Value 58.12 Indexed As Per MCI

JAUNDICE IN PREGNANCY

DR.A.CHARMILA*

*MD.,DGO.,DFP.,FICOG.,PGDCR., Director, Aditi Hospital & Parvathy Ayyavoo Fertility Centre, Tiruchirappalli, Tamil Nadu, India

ABSTRACT

Jaundice caused by hepatobiliary disease in pregnancy poses a challenge for the consulting clinician. Diagnostic and therapeutic decisions have to consider the implications for both the mother and the foetus. The types and presentation of hepatobiliary disease during pregnancy are varied. This article reviews three different causes and presentations of jaundice in pregnancy. HELLP syndrome, hepatitis in pregnancy and sepsis in pregnancy presenting with jaundice are discussed in the article. Two patients died because of the severity of the disease at presentation. The third patient presented with hepatic encephalopathy. She survived because of intensive care and monitoring.

***KEYWORDS**: Jaundice, Icterus, HELLP syndrome, Hepatitis, Acute fatty liver, Hepatic Encephalopathy*

BACKGROUND

Jaundice in pregnancy is a serious illness. It can affect both the mother and the foetus. It can be a cause of maternal morbidity and mortality. 3% of all deliveries in a prospective study in Wales showed abnormal liver function tests. The disorders causing the disease were either pregnancy specific disorders or other related conditions. The pregnancy specific disorders were pre-eclampsia, HELLP syndrome, obstetric cholestasis, hyperemesis gravidarum and acute fatty liver of pregnancy (AFLP). The other conditions which can contribute to

the disease are sepsis, drug related conditions, bile duct stones, hepatitis and uncertain etiology [1]. The common causes contributing to the disease are acute fatty liver of pregnancy, intrahepatic cholestasis of pregnancy, hyperemesis gravidarum, necrosis of liver of pregnancy, and hepatorenal syndrome following delivery [2].

CASE REVIEWS

This article reviews three cases of jaundice in pregnancy which were followed up and treated at a private hospital in Tiruchirappalli, Tamilnadu.

The first patient was Mrs.A aged 33 years and was admitted at 38 weeks of pregnancy with diagnosis of jaundice. She was a 4^{th} gravida with previous 2 caesarean deliveries and 1 abortion. She had been posted for an elective repeat caesarean and on routine testing was found to have abnormal liver function tests and low platelets at Pudukkottai, Tamilnadu. She was referred to our hospital. On admission, she was icteric. Her blood pressure was 130/100mm of Hg and pulse rate was 88/minute. Her blood investigations showed that she had a bilirubin level of 11.4 mg/dl. Direct bilirubin was 6.9mg/dl and indirect bilirubin was 4.4mg/dl. Platelet count was 26,000. Liver enzymes were elevated. She had an abnormal coagulation profile. Blood urea was 64 mg/dl and serum creatinine was 2.3mg/dl. Periphrel smear showed normocytic normochromic anaemia with reduced platelets and no parasites were seen. Obstetric ultrasound showed a mild growth restriction of the foetus.

She went into spontaneous labour. She was transfused with whole blood, blood products and platelet transfusions. She delivered an alive male baby of weight 2.4 kg after 4 hours of labour. She developed massive postpartum haemorrhage. In spite of uterotonics, continuous uterine massage and balloon tamponade, the bleeding continued. 38 units of blood products were transfused. Patient developed disseminated intravascular coagulation (DIVC). In spite of all resuscitative measures, she went into haemorrhagic shock and expired 28 hours after delivery. The cause for her multi-organ failure was HELLP syndrome.

HELLP is an acronym that refers to a syndrome characterized by Hemolysis with a microangiopathic blood smear, Elevated Liver enzymes, and a Low Platelet count [3]. It probably represents a severe form of preeclampsia, but the relationship between the two disorders remains controversial. In contrast to preeclampsia, nulliparity is not a risk factor for HELLP syndrome [4]. Half or more of affected patients are multiparous. The complications which are commonly observed are disseminated intravascular coagulation, abruption placentae, acute renal failure, pulmonary oedema, subcapsular liver haematoma and retinal detachment [5].

The second patient who was admitted with jaundice in pregnancy was Mrs.K aged 30 years. She was a third gravida and was 12 weeks pregnant. Her previous delivery was a caesarean delivery. She had had one MTP done before in her first trimester. She was admitted with the complaints of fever of one month duration. She gave a history of jaundice and facial distortion of one week duration. On examination, she was icteric. She had a

slurred speech. Her pulse rate was 170/minute. Her BP was 150/90 mm of Hg. Her liver was palpable and she had 9th, 10th and 12th nerve palsy on neurological examination. On investigating her, she was found to be hepatitis B positive. Her total bilirubin was 13.2 mg/dl. Direct bilirubin was 9.72 and indirect bilirubin was 3.48 mg/dl. Her platelet count was 1.32 lakhs, blood urea was 44 mg/dl and serum creatinine was 1.7 mg/dl. She was treated with intravenous fluids, antibiotics, lactulose, sorbilene. She developed hepatic encephalopathy the next day. She was shifted to ICU. She was febrile from the time of admission. In spite of all methods of resuscitation, she expired 2 days after admission. The cause for her death was septicaemia with fulminant hepatic failure.

Acute viral hepatitis can complicate pregnancy [6]. Although vaccination may prevent acute infection with hepatitis A and hepatitis B, a large pool of unvaccinated, non-immune women worldwide remains at risk for developing these diseases during pregnancy. The course of hepatitis A, B and C is similar to that of non-pregnant patients. By contrast, hepatitis E is more severe during pregnancy. Pregnant women with jaundice and acute viral hepatitis caused by hepatitis E virus infection appear to have worse obstetric and foetal outcomes compared with pregnant women with jaundice and acute viral hepatitis due to other causes [7]. Because of this risk, such women should carefully consider the risk of travel to endemic areas.

The third patient was Mrs. S who was 28 years old. She was a third gravida. She had delivered three children vaginally. She had delivered her third child normally in a nearby town 15 days back. The child was referred for neonatal sepsis to our hospital and was admitted in the NICU. The mother suddenly developed fever. 2 days later, she had reduced urine output and became drowsy. On admission, she was febrile and unconscious. Pulse rate was 130/minute. BP was 140/90 mm of Hg. She responded only to deep painful stimulus. She was icteric and had diffuse purpuric rash of the lower limbs. She had pedal oedema. Blood investigations showed serum bilirubin level of 8 mg/dl. Her liver enzymes were grossly elevated. Platelet count was 69,000. Coagulation parameters were elevated. Blood urea was 88 mg/dl and serum creatinine was 2.7 mg/dl. Infectious profile for hepatitis was normal. Ultrasound showed hepatomegaly with ascites. CT scan of the brain showed a haemorrhagic infarct in her right posterior frontal lobe. She was started on higher end antibiotics. She was transfused with 7 units of packed cells, 18 units of FFP, 34 units of platelets and 3 units of cryoprecipitate. She underwent one session of haemodialysis. In the next few days, her sensorium detiorated and bleeding diasthesis became more severe. She developed seizures and was put on anti-convulsants. She was intensely treated and monitored in the intensive care unit. She gradually recovered but her platelet dysfunction was persistent. She was then treated with intravenous immunoglobulin after which she recovered. The provisional diagnosis for her disease was hepatitis due to cause leading on to sepsis and DIVC.

Acute liver failure is characterized by acute liver injury, hepatic encephalopathy, and an elevated prothrombin time/international normalized ratio (INR). It has also been referred to as fulminant hepatic failure,

acute hepatic necrosis, fulminant hepatic necrosis, and fulminant hepatitis. Untreated, the prognosis is poor, so timely recognition and management of patients with acute liver failure is crucial [8]. Whenever possible, patients with acute liver failure should be managed in an intensive care unit at a facility capable of performing liver transplantation.

REFERENCES

1. Ch'ng CL, Morgan M, Hainsworth I, Kingham JG (2002) ; Prospective study of liver dysfunction in pregnancy in Southwest Wales; Gut.; 51(6): 876-80

2. Ellington SR, Flowers Legardy-Williams JK, Jamieson DJ, Kourtis AP (2015); Recent trends in hepatic diseases during pregnancy in the United States, 2002-2010; Am J Obstet Gynecol. 2015;212(4):524.e1. Epub 2014 Oct 30.

3. Stone JH (1998), HELLP syndrome: hemolysis, elevated liver enzymes, and low platelets;;JAMA.1998; 280(6):559-62.

4. Audibert F, Friedman SA, Frangieh AY, Sibai BM (1996), Clinical utility of strict diagnostic criteria for the HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome; Am J Obstet Gynecol.;175(2):460-4.

5. Sibai BM, Ramadan MK, Usta I, Salama M, Mercer BM, Friedman SA (1993); Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome); Am J Obstet Gynecol;169(4):1000-6

6. American College of Obstetricians and Gynecologists. Viral hepatitis in pregnancy. ACOG Educational Bulletin number 248. Washington, DC: ACOG 1998.

7. Patra S, Kumar A, Trivedi SS, Puri M, Sarin SK (2007), Maternal and fetal outcomes in pregnant women with acute hepatitis E virus infection; Ann Intern Med.;147(1):28.

8. Lee WM, Squires RH Jr, Nyberg SL, Doo E, Hoofnagle JH (2008), Acute liver failure: Summary of a workshop, Hepatology. 2008;47(4):1401-15.