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HEPATORENAL SYNDROME AND PREGNANCY - A RARE CASE REPORT

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Abstract

Pregnancy in women with liver cirrhosis is relatively uncommon. During pregnancy, liver cirrhosis and portal hypertension may worsen significantly, placing both the mother and fetus at an increased risk of serious morbidity and life-threatening events. With the use of a wide variety of diagnostic tools and considerably improved treatment strategies, many women with liver disease in pregnancies are being diagnosed with significantly improved obstetric outcomes. We present a case of a 33 years female, gravida 2, para 1, live 1 at 31Weeks + 4 days, Previous Lower segment cesarean section with Last child birth – 8 years back came to saveetha Medical College with complaints of decreased perception of fetal movements, vomiting three – four episodes since four hours. Patient had Acute kidney injury secondary to accelerated hypertension/Hepato renal Syndrome. Patient had cirrhosis of liver with splenomegaly with portal hypertension with pancytopenia with hepatorenal syndrome. There was no evidence of hypertensive retinopathy.

Keywords: Hepatorenal Syndrome, Pregnancy, Liver Cirrhosis and Portal Hypertension.

INTRODUCTION

Hepatic pathology occurs in approximately 3% of pregnancies. Liver disease during pregnancy results in an increased risk of maternal and new born complications. Pregnant women with advanced cirrhosis has increased risk of developing bleeding from esophageal varices, liver failure, and hepatorenal syndrome.

Liver cirrhosis is a chronic hepatocyte injury with extensive fibrosis and nodular regeneration.1 Pregnancy with liver cirrhosis is uncommon due to disturbances in endocrine metabolism, especially estrogen.2 With improved therapeutic options for liver disease, more women are presenting for prenatal care with concomitant cirrhosis.3

Pregnant women with advanced cirrhosis are associated with an increased risk of complications such as new-onset or deteriorated ascites for blood volume changes, bleeding from esophageal varices, liver failure, and hepatorenal syndrome.4 Besides the deteriorate complication of liver cirrhosis, adverse events of mothers and fetuses, such as spontaneous abortion, stillbirth, fetal or neonatal demise, placental abruption, preeclampsia, preterm delivery, small-for-gestational-age neonate, and postpartum hemorrhage are at an increased risk in women with cirrhosis.5

For women with liver disease, the principle risks during pregnancy relate to height end portal hypertension leading to decompensation; to the foetus, the risks are of prematurity, low birth weight and considerations of medication exposureinutero. This article will discuss these risks and focus on the management of pre-existing chronic liver disease in pregnancy.

CASE DESCRIPTION

A 33 years female, gravid 2, para1, live1 at 31 Weeks + 4 days, Previous Lower segment cesarean section with Last child birth – 8 years back came to saveetha medical college with complaints of decreased perception of fetal movements, vomiting three to four episodes since four hours. Her First and second trimester was uneventful. In Third trimester she complained decreased perception of fetal movements. Echocardiography showed Rheumatic heart disease (thickened doming anterior mitral leaf let, restricted mobility Posterior mitral lea flet, mild to moderate Mitral regurgitation, thickened aortic valve, mild aortic regurgitation). She had regular menstrual cycles. She is a Known case of chronic liver disease (Non Cirrhotic Portal Fibrosis) (Figure 1 & 2). No surgical interventions.

Her Hemoglobin was 9.5g/dl, Platelet was 48000, WBC was reduced. Her liver function test showed Total bilirubin/Directbilirubin-2.61/0.42, SGOT–65, SGPT–20, ALP–211, TP /ALB–6/2.5. Her Renal function test showed –urea of 43, create of 1.3 and Uric acid of 8.9. Spot PCR was 1.17 (normal is<0.3) and LDH of 527. Dengue IgG IgM was negative. COOMBS TEST and VIRAL HEPATIC MARKERS – were found to be negative.

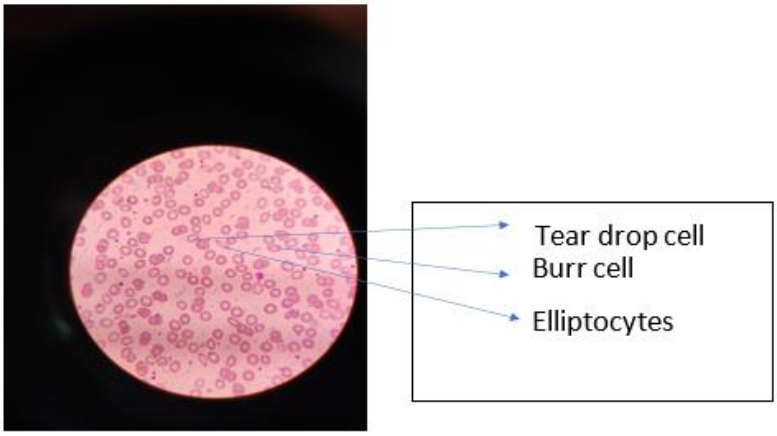

Fig 1: Pheripheral Smear- Showing Severe Microcytic Hypochromic RBC, Few elliptoCytes, Tear Drop Cells and Burr Cells

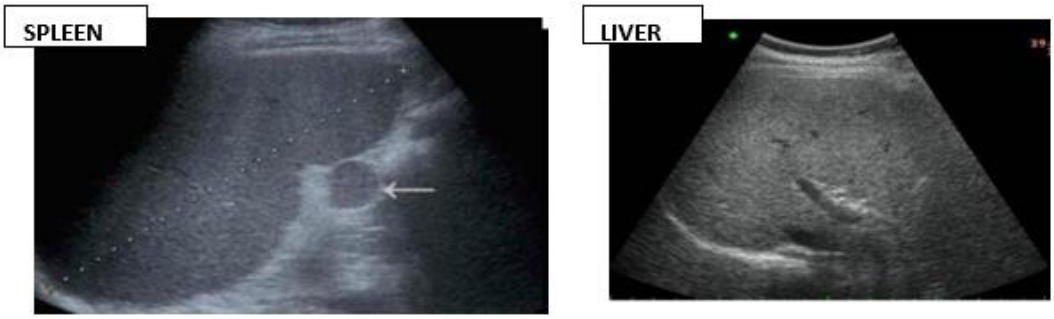

Fig 2: Ultrasonography of Upper Abdomen Showing Fatty liver & Mild spleenomegaly

On general examination she was a febrile, severe pallor present, pedal edema present, mildicterus present, Petechiae all over Abdomen and arms were noted. Blood pressure was180/100mmhg and pulse rate was99/min.

On palpating Liver and Spleen was palpable, nontender, uterus was corresponding to 30-31weeks, cephalic, relaxed Suprapubicscar present, no scar tenderness.On auscultation the fetal heart rate bradycardia was noted, Cervix soft, posterior, un effaced, OS closed.

Case Summary:

Patient had Acute kidney injury secondary to accelerated hypertension/Hepatorenal Syndrome. Patient had cirrhosis of liver with splenomegaly with portal hypertension with pancytopenia with hepatorenal syndrome. There was no evidence of hypertensive retinopathy.

So, the Patient was planned for emergency Lower segment cesarean section in view of previous Lower segment cesarean section with fetal brady cardia and chronic liver disease with hepatorenal syndrome. Intra operatively1unit of packed red blood cells, 2 Units Fresh frozen plasma, 2 units plate lets transfused.

Post operatively patient was shifted to Intensive care unit – vitals monitoring done. Patient was conservatively managed with intravenous antibiotics, intravenous Labetalol, Magnesium sulphate, intravenous analgesics. On post-operative ay 9-Ascitic tapping was done which showed High SAAG, low proteinascites–child pugh 9; class B. On post-operative day 14 Upper gastrointestinal endoscopy was done – oseophagealvarix grade 1, congestive gastropathy noted. Patient was advised for liver biopsy but was not done since patient was not willing.

DISCUSSION

Pregnancy and liver cirrhosis are a high-risk combination. Patients with liver cirrhosis have a significantly lower level of platelet, hemoglobin, prothrombin activity, and a higher level of Alaninetransaminase, total bilirubin, and creatinine.

Maternal mortality was attributed to hemorrhage from gastro intestinal varices, occurring most commonly in these condtrimester and during labor from recent studies, viral hepatitis seems to be the most common cause for cirrhosis.[6]

Child-Pugh-turcottes core and Model of end stage liver diseases core can be used to predict these verity of liver damage.[7,8] Recent studies have shown that compared to women who continue the pregnancy, women who chose to discontinue the pregnancy in the first trimester could avoid the most severe adverse events Compared to women who continue the pregnancy, women who chose to discontinue the pregnancy in the first trimester could avoid the most severe adverse events.[9,10]

So those patients with a higher Child-Pugh-turcottes core should be advised to discontinue the pregnancy in the first trimester. Many of these women will be un-aware of the risks of pregnancy, so we have to ensure that they are well informed about the adverse effects.

CONCLUSION

Close monitoring is very important when pre-existing liver disease patient becomes pregnant. Child-Pugh-turcotte and Model of end stage liver diseases core must be used to assess these verity of liver disease and prompt intervention should be taken a and when necessary. Timely termination of pregnancy during the first trimester may avoid the incidence of those severe adverse events.

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Conflict of interest: None to declare."

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