Cirrhosis with Portal Hypertension in Pregnancy

Dr. R. Sowjanya1 Dr. R. Sudha2
1Assistant Professor, 2Resident
(Department of Obgy, Siddhartha Medical College/ Dr. Ntruhs, India)

Abstract: Here is a case of a G3P1L1A1 with cirrhosis with portal hypertension complicating pregnancy who presented to us with Page 1 Abruption. After delivery, the patient was investigated further with complete liver and coagulation profiles. Ultrasound Abdomen and Pelvis and upper GI endoscopy was done. The management of this case has been discussed.

Keywords: Abruption, Cirrhosis, Portal Hypertension, Splenomegaly, Varices.

I. Introduction

Cirrhosis is an irreversible disease characterised by damage to the liver and fibrosis which eventually leads to blood being shunted away from the liver leading to elevated pressures in the portal system. Complications of cirrhosis during pregnancy are similar to those that can occur in any non pregnant state including variceal bleeding, liver failure, encephalopathy, splenic artery aneurysm, etc. Esophageal variceal bleeding has been in upto 20% - 30% cases with portal hypertension, most commonly during second and third trimesters when maternal blood volume is maximally expanded and the larger fetus causes increased compressions of the inferior vena cava and collateral vasculature. Pregnancy is rare in patients with cirrhosis for two reasons - Firstly advanced liver disease doesnot typically occur until most patients have completed their families and Secondly cirrhosis results in metabolic and hormonal derangements that lead to anovulation and amenorrhoea. Improvements in the treatment of chronic liver disease have resulted in higher conception rates and more successful pregnancy outcomes.

II. Case Report

A 25 years old Mrs ABC, a G3P1L1A1, who was an unbooked case, reported to the labour room with complaints of bleedind per vaginum and labour pains on 20/2/2015. She was a known case of cirrhosis with portal hypertension diagnosed 4 months ago and had interrupted treatment.

On General examination, she was moderately built and moderately nourished, her vitals were stable. The patient was mildly anemic, icteric with B/L wheeze and a loud P2. On Abdominal examination, not corresponding to 30 weeks GA with Splenomegaly palpable upto 12cm below the costal margin. Fetal heart sounds were absent and Uterus tense and tender. On Per vaginum examination, the patient was in latent phase of labour with excessive show present. After ARM and acceleration of labour, patient delivered in 6hrs. After delivery retroplacental clots of about 250gms was expelled - Page 1 Abruption.

Complete investigations were done which cited Hyperbilirubinemia(2.7mg/dl) and Thrombocytopenia(40,000 cells/cumm) as the only abnormal findings. All the other parameters including Liver function tests, Renal function tests, Haemogram were normal. Urine output was adequate.

The patient was shifted to ICU and physician and gastroenterologist were called in for further management. On upper GI endoscopy multiple esophageal varices Grade 1 were noted. Patient was started on Diuretics(Lasix + Spirinolactone) and propranalol 40mg OD. Postnatal period was uneventful and the General condition of the patient improved dramatically within a week. Her Bilirubin levels fell drastically and her Platelet counts improved upto 90,000 cells/cumm. The patient was discharged after two weeks and advised followup.

III. Discussion

Prognosis of portal hypertension during pregnancy depends upon the underlying cause and the extent of derangement of liver function. Maternal prognosis is better with Extra hepatic portal venous obstruction (EHPVO) and Non cirrhotic portal fibrosis (NCPF) and poor with cirrhosis of the liver. Maternal mortality ranges between 2% and 18%; being maximum with cirrhosis. The causes of death are generally hematemesis, hepatic coma or postpartum hemorrhage. Perinatal mortality ranges between 11% and 18%, mainly due to preterm delivery or intrauterine growth restriction (IUGR).

Of the women with cirrhosis, 20 -30% will have hematemesis during pregnancy with the mortality ranging between 50–60%. The incidence of hematemesis in patients with EHPVO and NCPF is around 7 %. The timing and severity of hematemesis, however are unpredictable. Hematemesis is more common in pregnancies
complicated by varices. Hematemesis during pregnancy is contributed to by increased portal pressures during pregnancy, reflux esophagitis and obstruction to the inferior vena cava by the gravid uterus.

Management of portal hypertension in pregnant women is similar to that in non-pregnant patients. Beta blockers are given to reduce portal venous pressures. Surgical management by banding and sclerotherapy have been successfully employed during pregnancy. It is possible to do shunt surgery during the second trimester.

There is a danger of variceal rupture and hematemesis when the patient strains during labor. Patients with EHPVO and NCPF generally tolerate labor well and cesarean section is not mandatory. It is however, necessary to have a Sengstaken- Blackmore tube and adequate amount of cross matched blood readily available if these patients are given a trial of labor. They must not be allowed to bear down and the second stage should be cut short.

Pregnancy is not contraindicated in patients with portal hypertension due to NCPF, EPVOC and compensated cirrhosis. Termination of pregnancy needs to be considered only in patients with decompensated cirrhosis, recurrent hematemesis and deranged liver functions, especially abnormal coagulation profiles. The management of pregnancy with portal hypertension should only be done at tertiary care centres by a multidisciplinary team with backup facilities for intensive care and blood transfusion.

References