VOLUME-7, ISSUE-9, SEPTEMBER-2018 • PRINT ISSN No 2277 - 8160

Original Research Paper

Gynaecology

SUCCESSFUL OUTCOME OF NON-CIRRHOTIC PORTAL HYPERTENSION IN PREGNANCY – A CASE REPORT

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ABSTRACT This is a case of a Primigravida with seven months of gestation who landed up in non-cirrhotic portal hypertension during her antepartum period and went into preterm labor & was managed effectively in a tertiary care hospital. Portal hypertension during pregnancy is a rare phenomenon but it carries great feto-maternal risk & a multidisciplinary approach is required for its management.

KEYWORDS : Portal hypertension, Non-cirrhosis, Pregnancy.

Introduction

During pregnancy, the liver metabolic, synthetic, and excretory functions are affected by the increased serum estrogen and progesterone (1). Serum albumin concentration decreases during pregnancy and reaches a nadir towards the end of the pregnancy, secondary to increase in plasma volume (2). Alkaline phosphatase activity is increased during the third trimester both because of leakage of placental alkaline phosphatase into the maternal circulation and because of increased maternal bone turnover (3). Increase in transaminases levels was found during labor and is, most probably, secondary to leakage from the contracting uterine muscle (4). Total and free bilirubin serum levels are lower in pregnancy, both because of hemodilution and the low albumin concentration (3). Incidence of pregnancy with portal hypertension with cirrhosis is 1in 5980 pregnancies& that with non-cirrhotic is 1 in 1077 pregnancies (5). Pregnancy is a potential hazard for occurrence of recurrent variceal bleed due to increased blood volume and cardiac output increasing the portal flow, which increases flow in the collaterals & aggravates portal hypertension (6). NCPF is a syndrome of unknown etiology, characterized by obliterative portovenopathy leading to portal hypertension, massive splenomegaly and variceal bleed, with preserved liver function (7) Portal hypertension is characterized by an increase in portal pressure (>10mmHg) due to cirrhosis of the liver or of non-cirrhotic disease (8). Non-cirrhotic portal hypertension (NCPH) comprises diseases having an increase in portal pressure secondary to intrahepatic or prehepatic lesions, in the absence of cirrhosis. NCPH accounts for 30% of the variceal bleeding (7).

Case report

28 year, female, married since 2 years, Primigravida7 months gestation came with complaints of pedal edema & abdominal distension since 2 months, yellowish discoloration of body and urine & itching since 10 days.

M/H LMP ? EDD? B/D ? B/S 30.4 weeks. PaMC 3-4 D/30 D/ RMPL. O/H Primigravida married since 2 years. Registered & immunized. Past History- h/o Jaundice 10 years back. On examination, general condition was moderate, emaciated afebrile, Pulse- 110/min high volume, regular, Blood Pressure 120/60 mmHg. Pallor++, Icterus +, B/L pedal edema+. Respiratory system & cardiovascular system normal. Central nervous system-conscious and oriented. Per abdomen-Distended, gross ascites++Fluid thrill+, splenomegaly+, spleen palpable 7 cm below costal margin(grade II). Uterus 28 weeks, FHS+, regular, relaxed. Per speculum-cervix vagina healthy. Per vaginum -os 1cm, tubular, uneffaced, station high. BT 30 sec, CT 2 min 35 sec All investigations were done. Blood group O+. CBC Hb 5, TLC 2200, Plt 1.2 lacs. Sr bili- 2.2 Sr Creat 0.7 SGOT 27.6, SGPT 34.9 Arterial ammonia 106.3 µmol/L (10-40) PT 18.3 INR 1.31 aPTT 29.4 D dimer 1.8 (<0.5) HIV NR, HBsAg NR, HCV NR, HAV NR, HEV NR. Peripheral smear-Pancytopenia, microcytic hypochromic anemia. Retic count 1.3% (0.5-1%). Dengue NS1-Neg. Vit B12- >2000 pg/ml Sr Iron 27.83 µg/dL (70-180) TIBC 570 µg/dL (250-400). % Transferrin saturation 4.88 (20-50), Sr Ferritin 8 ng/ml (50-150), LDH 584 IU/L (230-460) HVS- s/o bacterial vaginosis. USG Obs- SLIUG 29 weeks with normal doppler study. USG Abdomen- altered hepatic echotexture, dilated portal vein (19 mm), moderate splenomegaly with dilated splenic vein and multiple collaterals, gross ascites. Chest X ray s/o cardiomegaly. 2 D ECHO- LVEF 60%. 1 pint blood was transfused. Ascitic fluid- pale yellow colored fluid, total nucleated cells 10-15/mm3, RBC 5-7 /HPF, ADA 0.3 (30-45). Hematology reference was done- UGIscopy, Inj iron, T protein, albumin was advised. Gastroenterology reference was done- Expert porto-splenic doppler, HCV, HEV, HAV, stool for occult blood, ANA, Ceruloplasmin, Endoscopic variceal ligation with Portal Hypertensive gastropathy was advised. Portal vein doppler- dilated measuring 19 mm. 1 pint blood transfused. Repeat investigations were done-CBC Hb 9.1, TLC 2900, Plt 1.93 lacs. Sr bili- 2.3 LKM1 Ab Neg, ASmAb Neg, ANA Neg, ANA blot Neg GGT 15 Ceruloplasmin 40.90 (2-50 mg/dL) Hb electrophoresis- normal. OGDscopy was done s/o 3 large varices seen and endoscopic variceal ligation was done. Pt was started on higher antibiotics, 1 pint blood transfused, lasilactone continued, fluid restriction was kept, I/O charting was done 3 Albumin infusion given. Daily fetal monitoring was done. Pt went into active labour and delivered a male baby 1.5 kg by vaginal delivery and was admitted in NICU for LBW. On discharge, investigations were- Hb 9.4 TLC 6200 Plt 44000 Sr bili 0.7 SGOT 67 SGPT 40 PT 20 INR 1.68 Sr creat 0.7 Urea 21, BUN 9.8 Na 144 K 3.2. 1 pint PCV and 8 pint FFP transfused. 2D ECHO LVEF 60%, Mild MR. Pt was discharged along with baby.

Discussion

Pregnancy is not contraindicated in patients with portal hypertension due to NCPF, EHPVO and compensated cirrhosis. Prognosis of portal hypertension during pregnancy depends upon the underlying cause and the extent of derangement of liver function Maternal mortality ranges between 2% and 18%; being maximum with cirrhosis (9). 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 (9). 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 (9). They must not be allowed to bear down, and the second stage should be cut short. The management of pregnancy with portal hypertension should only be done at tertiary care centers by a multidisciplinary team with backup facilities for intensive care and blood transfusion.

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