A case of uneventful early post partum phase of pregnancy with LSCS in a known case of portal hypertension with splenectomy & a delayed peritonitis.

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Abstract:

This is a case of 32 year old multigravida, a known case of portal hypertension with episodes of haemetemesis on and off since last 12 years with history of splenectomy 2.5 years ago with uneventful 1st LSCS presented with pain abdomen and abdominal distension post-LSCS. Blood investigations showed microcytic anaemia corrected by blood transfusion while hypo -proteinemia by FFP transfusion. USG showed portal vein thrombosis with ascites. CT scan was done which confirmed signs of portal hypertension with cavernoma formation and gross ascites, Fluid cytology suggestive of PMN's. Patient was managed by controlled tapping and fluid monitoring along with appropriate antibiotics.

Keywords: Cirrhosis, Peritonitis, Portal Hypertension.

Introduction:

Pregnancy is a hyperdynamic circulatory state. Pregnancy associated with liver diseases is an infrequent situation, but when seen together, presents a complicated clinical situation. Portal hypertension develops as a result of number of etiologies. In the west, cirrhosis is the commonest cause of portal hypertension. In the setting of cirrhotic portal hypertension, pregnancy is very rare due to hepatocellular damage leading to amenorrhea and infertility, the incidence of cirrhosis in pregnancy has been reported as 1 in 5950 pregnancies.[1] Cirrhosis may get exacerbated during pregnancy and has significant adverse effects on the mother and the baby.[2,3,4] In the developing countries, other causes like extrahepatic portal vein obstruction contribute significantly to non-cirrhotic portal hypertension (NCPH). Mostly liver function is much better preserved in women with NCPH and pregnancy is spontaneous in these women. Portal hypertension associated with pregnancy is a high risk situation as both pregnancy and portal hypertension share some of the hemodynamic changes. The physiological changes, in adaptation to the pregnancy and fetal needs, worsen the portal hypertension resulting in potentially life-threatening variceal bleed and other complications. Pregnancy is a potential hazard for occurrence of recurrent variceal bleed due to its hyperdynamic state causing increase in flow to the collaterals.[5,6] Therefore management in pregnancy requires knowledge of both the effects of changes during pregnancy on portal hemodynamics and the effects of portal hypertension and its cause on both mother and

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fetus, hepatotoxicity of the drugs used, management of portal hypertension so as to have an optimal pregnancy outcome.

Case Presentation:

Presenting a case of 32 year old multigravida patient, a known case of portal hypertension since 12 years, presented with pain abdomen and abdominal distension since 10 days while fever with chills and rigors since 5 days, This was after her 2nd LSCS, when patient was asymptomatic for 10 days and developed these symptoms after that. Patient had her episode of haemtemesis, 12 years ago and was diagnosed as portal hypertension 6 years ago when she underwent upper GIscopy and banding for her 3rd episode of haemetemesis. Patient underwent splenectomy 3 years ago for pancytopenia. Her 1st LSCS was uneventful, while in her present pregnancy she had 1 episode of haemtemesis at 4th month and treated with banding. She is poorly built with pallor and pedal edema, with her vitals showing features of fever and hypotension on presentation. Per abdomen was evident of lower abdominal gross distension with horse shoe dullness and fluid thrill. Investigations revealed anaemia treated with blood transfusion , hypoproteniemia with FFP transfusion, USG showed portal vein thrombosis and gross ascites shifting liver downwards due to it, while CT confirmed signs of portal hypertension with Cavernoma formation, no hepatic vein thrombosis found, Spontaneous bacterial peritonitis was confirmed by raised PMN's to 300. Patient's management included controlled ascitic tapping, strict diet and fluid monitoring with higher antibiotics. Aspiration of 1 litre done on 2nd day of admission, 2nd aspiration done after 5 days of 700m1 and on 10th day of admission of 700ml was tapped. Last aspiration fluid was showing normal range of polymorpho -neutrophils. Patient was discharged after she started taking adequate orally. Patient was discharged with propranolol, sorbitrate, lasilactone. After 1 month of follow up, patent is having minimal ascites.

Discussion:

The physiological changes, in adaptation to the pregnancy and fetal needs, worsen the portal hypertension resulting in potentially life- threatening variceal bleed and other complications. Pregnancy is a potential hazard for occurrence of recurrent variceal bleed due to its hyperdynamic state causing increase in flow to the collaterals. Numerous hemodynamic and physiological changes occur during pregnancy as an adaptation to the needs of the growing fetus.[7-8] These changes start as early as six weeks and peak around 32 weeks. These changes are summarized in Table 1.

Table 1 Normal physiological changes during Pregnancy.

1.	Increased maternal blood volume
2.	Increased Maternal heart rate
3.	Increased Systemic vascular resistance and blood pressure
4.	Increased Peripheral vasodilatation & placental bed circulation.

The pregnant woman has a 20-27% chance of esophageal bleed which increases markedly in case she has demonstrable varices. The first known mechanism of portal hypertension is an increase in intrahepatic resistance to blood flow. Hepatic damage thus caused results in shunting of hepatic blood, development of extrahepatic collaterals and

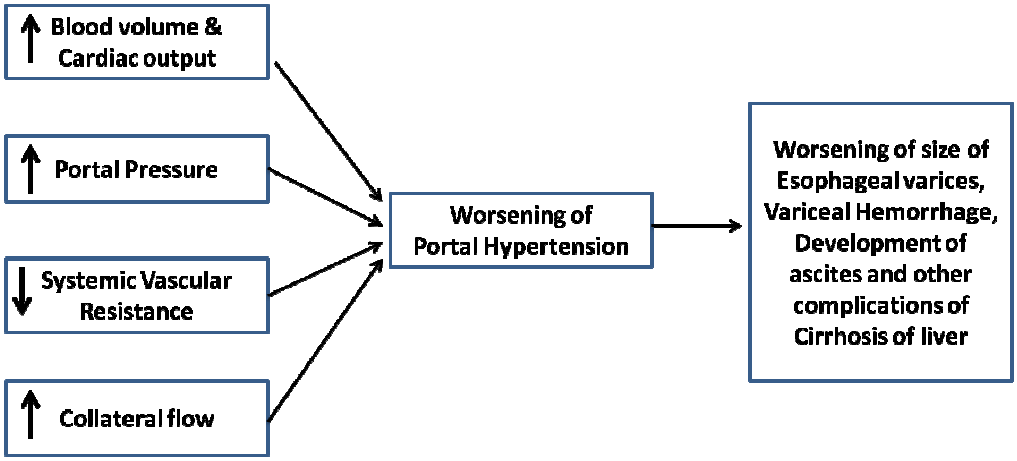
elevated pressure in the portal venous system. Normal values of HVPG are between 1 and 5 mmHg, portal hypertension is defined as the pathologic increase in portal pressure expressed HVPG>10 mm Hg is needed for development of esophageal varices and HVPG >12 mmHg for them to bleed.

Table 2 Pathophysiological effects of Portal Hypertension.

HVPG (mm Hg)	Clinical features	Stage of Cirrhosis
1-5 mm Hg	Normal, non-cirrhotic	-
6-10 mm Hg	Compensated cirrhosis	1
>10 mm Hg	Compensated cirrhosis with development of varices	2
>12 mm Hg	Decompensated cirrhosis with ascites, variceal blood, hepatic encephalopathy	3-4

Variceal bleeding, ascites, encephalopathy and hepatorenal syndrome are the various clinical manifestations of the portal hypertension. Esophageal varices are seen in > 40% of patients with liver cirrhosis at the time of diagnosis. Other manifestations of portal hypertension are splenomegaly and hypersplenism

Image 1: Effect of Pregnancy hemodynamics on Portal Hypertension



In pregnancies with portal hypertension 30-50% of pregnancy suffer from portal hypertension associated complications, resulting mainly because of variceal bleed and hepatic failure. Gastro-intestinal haemorrhage remains the most catastrophic complication of portal hypertension during pregnancy. Variceal bleed has been reported in 18-32% of pregnant patients with cirrhosis and in 50% with a known portal hypertension, about 75% of patients with varices bleed during pregnancy which is one of the most serious consequences. Patients with portal hypertension associated with liver cirrhosis have worst: prognosis, with mortality rate of 18-50% whereas those with primary biliary cirrhosis have the best outcome. Pregnant women with NCPH fare better with mortality rates between 2 and 6%. Approximately 7-9% of patients with portal hypertension suffer from symptomatic anaemia irrespective of the cirrhotic state. The mainstay of treatment remains endoscopic variceal ligation. Pregnant patient with cirrhosis may develop liver dysfunction in the form of jaundice, ascites, and/or hepatic encephalopathy .In 24%, Hepatic decompensation may occur during all stages of

pregnancy but often occurs after episodes of variceal bleeding. It is recommended that patient with portal hypertension and pregnancy should underwent upper GI scopy and banding during pregnancy if there is anaemia, haematemesis, gastropathy or melena. If such patients are developing increase in ascites during antenatal, or post natal period, they should be admitted and further investigated. It may be due to spontaneous bacterial peritonitis, increased portal vein thrombosis or due to hepatic vein thrombosis. In bacterial peritonitis, higher antibiotic has a role over surgery. Few patients who attend hospital early may respond to conservative treatment.

Conclusion:

Even though pregnancy is rare with cirrhosis and advanced liver disease, but it may co-exist in the setting of noncirrhotic portal hypertension as liver function is preserved but whenever encountered together is a complex clinical dilemma. Pregnancy in a patient with portal hypertension presents a special challenge to the obstetrician as so-called physiological hemodynamic changes associated with pregnancy, needed for meeting demands of the growing fetus, worsen the portal hypertension thereby putting mother at risk of potentially life-threatening complications like: variceal haemorrhage. Risks of variceal bleed and hepatic decompensation increase many fold during pregnancy. Optimal management revolves round managing the portal hypertension and its complications. Thus management of such cases requires multi-speciality approach involving obstetricians experienced in dealing with high risk cases, hepatologists, anaesthetists and neonatologists. With advancement in medical field, pregnancy is not contra-indicated in these women, as was previously believed. This paper focuses on the different aspects of pregnancy with portal hypertension, it is recommended that patient with portal hypertension and pregnancy should underwent upper GI scopy and banding during pregnancy, if there is anaemia, haematemesis, gastropathy or melena. If such patients are developing increase in ascites during antenatal, or post natal period, they should be admitted and further investigated. It may be due to spontaneous bacterial peritonitis, portal vein thrombosis or due to hepatic vein thrombosis. In bacterial peritonitis, higher antibiotic has a role over surgery. Few patients who attend hospital early may respond to conservative treatment. Based on extensive review of literature, management from pre-conceptional period to postpartum is outlined in order to have optimal maternal and perinatal outcomes.

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