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Case report : *Obstetric management of chronic portal vein thrombosis* in pregnancy

Dr. Eyob Dagnew, Dr. Semere Fisseha, Dr. Seid Arage

Introduction

Portal vein thrombosis (PVT) is characterized by the obstruction of the main portal vein and/or its tributaries. There are wide varieties of etiologies. Occasionally, it is not possible to recognize any overt cause of PVT. In the literatures there are no data about its exact prevalence, etiology, and outcome, and no definite guidelines for the management of this condition during pregnancy are available.
We here describe the case of a pregnant woman who was referred to our department after an incidental diagnosis of chronic PVT was made 8years before pregnancy. The report will emphasize on management of pregnancies complicated by non cirrhotic PVT where the cause is not identified.

Case report

The index case was a 26-year-old, primigravida lady, she is a known chronic portal vein thrombosis patient for the past 8 years. she was on follow up at our medical OPD. It was diagnosed initially with abdominal ultrasound with Doppler then with abdominal CT with contrast. She discontinued follow up by her own 3 years back because her symptoms resolved. Her initial symptoms upon presentation were recurrent dizziness & fainting episodes with associated back pain 8 years back. Again the patient was referred from another hospital at 38week gestation as a case of known chronic portal vein thrombosis & moderate thrombocytopenia. During the hospitalization a routine abdomen scan revealed the presence of Chronic PVT wih cavernous transformation + Splenomegally of 14 centimeter. She was admitted to our department for 4 days, clinically asymptomatic, without signs of hypersplenism or portal cholangiopathy. On the admission the patient was hemodynamically stable with a blood pressure of 110/70 mmHg, pulse rate of 72 beat per minuite and respiratory rate of 20 breaths/minute. Obstetric ultrasound revealed a single fetus with estimated weight of 3.3kg, cephalic presentation & reassuring

biophysical profile. A detailed and focused history taking revealed appendectomy were done 2 years back. Medical history was negative for liver disease. Laboratory investigations revealed **platelet count of=66,000** moderate thrombocytopenia, and liver function tests were otherwise unremarkable. Other CBC parameters, prothrombin time, partial thromboplastin time and INR were within the normal range. Thrombotic risk profile was negative for factor V Leiden, prothrombin gene G20210A, hyperhomocysteinemia, antithrombin III deficiency, protein C or S deficiency, and antiphospholipid antibodies. Viral tests were negative for the major hepatotropic viruses (hepatitis B & hepatitis C). The abdominal examination revealed a nontender abdomen, with no pain and normal bowel sounds, of note a mild splenomegaly was found. No signs of ascites were noted. Abdominal ultrasound found a portal vein thrombosis with cavernomatous transformation. There were no signs of inflammatory abdominal foci were found. There was no evidence of free ascitic fluid within the abdominal cavity. Color Doppler US confirmed the cavernomatous transformation of the portal venous system in the gastric fundus, pancrease & periportal areas. Baseline upper GI endoscopy was negative for esophageal varices. Fetomaternal side were communicated & decided on mode of delivery. On the third day of ward admission labor started spontaneously she was transferred to labor ward second stage of labor were shortened by forceps & she delivered 3.6 kg male alive neonate with out complication. The plan was to put her on longterm anticoagulation.

3.Discussion

Several causes can be involved in the pathogenesis of PVT and frequently more than one coexist. Local and systemic risk factors can play a role in the pathogenesis of this condition, in the 70% and 30%, respectively [6]. The most common local thrombotic risk factor is the presence of inflammatory abdominal foci (such as appendicitis, diverticulitis, inflammatory bowel diseases, pancreatitis, cholecystitis, hepatic abscesses, and cholangitis) or a liver disease (cirrhosis or tumors). The prevalence in patients affected by a liver disease ranges from 1%, at the earlier stages, to 30% in candidates for liver transplantation. In patients with a hepatocellular carcinoma, the incidence of PVT rises to 10%–40% [7]. Other acquired conditions including infections, surgery, myeloproliferative disorders, obesity, oral contraceptive intake, pregnancy, and postpartum period can be involved in the pathogenesis. Aggarwal et al. found that an underlying hypercoagulable and prothrombotic state, as high platelets counts, was present in about 20% of the patients [2].With the cessation of the portal venous blood flow, the liver loses about two-third of its supply. This condition is usually well tolerated, mainly due to a compensatory mechanism of venous rescue consisting of the rapid development

of collaterals to bypass the obstruction. This vascular neoformation, called "cavernomatous transformation," has been shown to form within the first 6 to 20 days after acute thrombosis of the portal vein and to be complete within 3 to 5 weeks [8, 9]. As a result the thrombosed portal vein is replaced by a network of collateral vessels, the portal cavernoma [10]. It occurs much more frequently in patients without underlying liver disease, but often leads to portal hypertension because the collateral veins are not able to adequately handle the splenic and mesenteric inflow. In cirrhosis, cavernomatous transformation of the portal vein is rare because stasis of portal venous flow prevents the formation of collateral channels in and around the portal venous thrombus. In this condition hemodynamic changes are present in hepatic and splanchnic circulation that are responsible for a partial impairment in liver function, in absence of an overt liver disease, or can precipitate a preexistent clinical status in cirrhotic patients. PVT might indirectly affect other abdominal organs, causing intestinal ischemia and infarction, or predisposition to vascular neoformation and gastrointestinal bleeding.

In a multicentric study on maternal and fetal management and outcome, Hoekstra et al. found that in pregnant PVT patients treated with anticoagulant on an individual basis, the rate of miscarriage and preterm birth appears to beincreased (38%). However, fetal and maternal outcomes are favorable for most pregnancies reaching 20 weeks of gestation[1]. Hoekstra et al. reported that the incidence of abortion,preterm deliveries, and still births in a large series of pregnancies evaluated in a tertiary centre of Northern India was of 20%, 15.4%, and 7.7%, respectively [1]. In a retrospective analysis Sumana et al. found that perinatal loss was around 25% and even lower in patients diagnosed prior to pregnancy[11].

Hoekstra et al. concluded that pregnancy should not be contraindicated in stable PVT patients[1]. In contrast to acute PVT, chronic PVT can be nearly asymptomatic as in our index case, except for the presence of thrombocytopenia & hepatomegally [12],with medical history apparently negative for previous trigger events or diseases. Data in the literature suggest that the most common presenting event in patients with chronic PVT is hematemesis. The exact incidence of variceal bleeding in pregnant patients with noncirrhotic portal hypertension(NCPH) is still unknown with reports ranging from an incidence of 20%–40% in a multicentric study [13] to an incidence of 34% in a prospective-retrospective analysis [1].It is advisable to screen endoscopically all the patients once the diagnosis of PVT is confirmed to rule out the presence of esophageal varices [3]. Other patients can present with thrombosis, abdominal pain, and jaundice or incidental splenomegaly. Hypersplenism and the consequent pancytopenia can be present in chronic PVT [4], but if one branch of the portal vein is preserved and the portal pressure is normal (7 mmHg), they may even be absent with normal white cells, red cells,

and platelets count. The presence of an underlying liver disease influences the prognosis [4]. The overall mortality is less than 10% in PVT chronic onset, and it is higher, around 26%, in patients with malignancy or cirrhosis [5].

4.Conclusion

PVT is relatively uncommon in the general population and it is considered a rare pathology complicating pregnancy.Pregnancy outcome is expected to be successful in women with extra hepatic portal vein obstruction(EHPVO) if disease is diagnosed and adequately controlled prior to pregnancy [1]. In the literature there are no sufficient data regarding the management and the prognosis of the cases of portal vein thrombosis diagnosed before pregnancy.

There is no role of elective termination of pregnancy or caesarean section and these procedures should be reserved only for obstetric indications. Second stage of labor does not exacerbate upper gastrointestinal bleeding and in patients who are at high risk of variceal bleeding, it may be safely cut short by the operative vaginal delivery. Pregnancy in women with non cirrhotic portal hypertension overall has a good outcome. There is no substitute for prepregnancy diagnosis and counseling and prophylactic treatment of esophageal varices before pregnancy. In our case, after a thorough literature review the patient was informed about all the maternal and fetal risks related to thrombocytopenia, and hypersplenism.The patient was aware that her life was not threatened by this condition; labor started spontaneously & she delivered via forceps assisted delivery with out any maternal & fetal complication.

Conflict of Interests

No conflict of interests.

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