Portal Hypertension and Pregnancy: Three Cases

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ABSTRACT

The occurrence of pregnancy in a woman with an HTP is daunting because of the possibility of maternal and fetal worsening. The character of a high-risk pregnancy is confirmed by the frequency of gastrointestinal bleeding, spontaneous abortions and premature deliveries. Clinical symptomatology is often dominated by the complications, and the ultrasound and endoscopy, can confirm the diagnosis and detecting complications. The management of pregnancies among women with HTP must begin before conception to postpartum. It requires close collaboration between obstetricians, gastroenterologists, anesthetists and neonatologists. Our study concerns three cases of portal hypertension in pregnancy collected in the service of gynecology & obstetrics, and intensive care unit in the University Hospital Ibn Sina of Rabat. In light of these three observations, we discuss mainly, the physiopathological particularity, evolutionary aspects, and principles in the management of these pregnancies at risk.

Keywords: Portal hypertension. Pregnancy. Multidisciplinary management

1. INTRODUCTION

The association of pregnancy with portal hypertension (PHT) is exceptional because of subfertility associated with the pore pressure given the essential role of the liver in the metabolism of sex hormones(1). This uniqueness can also be explained by the intervention of several factors: age of the patient, the etiology of PHT, and endocrine and ovarian disorders(2). The high-risk pregnancy nature is affirmed by the frequency of gastrointestinal bleeding by rupture of esophageal varices in 40% to 50% of cases, a preterm birth rate of 19%, and perinatal mortality estimated at 18%(3).

Currently, any woman with HTP can lead a normal pregnancy to term, and vaginal delivery of a newborn alive futures are common through a strict maternal-fetal monitoring and adequate multidisciplinary management(4). Caesarean section is reserved for purely obstetric indications and anesthesia peri-spinal techniques are not recommended because of the frequency of coagulopathy(3). Maternal prognosis is directly related to the existence of esophageal varices should be sought routinely before conception or early in pregnancy by endoscopy(5,6). Fetal prognosis is related to the search of intrauterine growth restriction and prevention of premature labor(6,7). Endoscopic treatment disrupts care, and surgery appears to improve maternal-fetal prognosis thanks to shunt performed before pregnancy(3). About 3 cases of pregnancy in patients with PHT, we mainly discuss the evolutionary aspects and difficulties in the management of these pregnancies at risk.

2. CLINICAL CASES

Case One: Patient aged 23, G1P1 followed for PHT on post-hepatitis C cirrhosis for two years. An upper gastrointestinal endoscopy performed during the first trimester of pregnancy had objectified esophageal varices stage
III and varicose veins under-cardial why it had been put under propranolol (40mg / day). She was hospitalized, within our service 36 WA for the management of pregnancy at risk. Clinical examination found a patient in good general condition, subictérique with a huge splenomegaly and collateral venous circulation, without hepatomegaly. The obstetrical examination had found a uterine height 28 cm, a positive fetal cardiac activity, a long neck and hind closed cephalic presentation. Obstetrical ultrasound had objectified: a scalable single-fetal pregnancy estimated 33 SA and fundus placenta grade II. Abdominal ultrasound showed: a heterogeneous liver with the presence of a hepatic nodule at the V segment, a trunk door dilated 16mm, splenomegaly, and absence of ascites. Laboratory tests were objectified: anemia microcytic hypochromic 6 g / dl hemoglobin, thrombocytopenia to 85,000 / mm3 total bilirubin to 28 mg / l of bilirubin which 7, 55% prothrombin, and amino -transférases were normal. Support was comprised of a transfusion four packed red blood cells and fresh plasma six units frozen with the administration of vitamin K to the 10mg / day dose. Three weeks after his hospitalization, indicating a caesarean was laid before a premature rupture of membranes with unfavorable Bishop score. The procedure was performed under general anesthesia, after formal requirement, giving birth to a newborn female, with a score Apgar On 5 min 10/10 and a weight of 2kg birth 600. The postoperative course was without complications, and the patient and her baby were allowed to leave the hospital ten days after delivery and was addressed to the consultation gastroenterology.

Case Two: Patient aged 22, G1P1 followed for PHT on cirrhosis of unknown origin for three years. An upper gastrointestinal endoscopy performed before pregnancy showed esophageal varices stage I. The patient was admitted to our service in order to follow the chronic disease. She was admitted to the intensive care unit at the Souissi maternity hospital in Rabat 20 WA for hemorrhagic syndrome due to gingival bleeding, epistaxis, diffuse petechial purpura and gastrointestinal bleeding type melaena without genital bleeding. The clinical examination had found an obsessed patient, dyspnea, and jaundice with a massive splenomegaly. The obstetrical examination found a uterine size corresponds to the term with a subsequent long neck closed without bleeding. Obstetrical ultrasound had objectified fetal death in utero. Abdominal ultrasound showed: a liver cirrhosis, portal vein dilated, a huge splenomegaly, an abundance of ascites. Laboratory tests were objectified: hypochromic microcytic anemia in 4.2 g / dl hemoglobin, thrombocytopenia 3500 / mm3, TP 22% TCA twice the indicator, the presence of fibrin degradation products (FDP) in moderate amounts, AST 4 times the normal, ALAT to 3 times normal and a total bilirubin 77mg / l. After a condition for symptomatic treatment and (Oxygen, intravenous fluids, the contribution of red blood cells, PFC and vitamin K), fetal extraction was performed vaginally. The evolution of the mother was marked by persistent bleeding despite treatment, and death occurred after 48 hours of hospitalization.

3. DISCUSSION

PHT is defined by increased pressure in the portal venous territory above 10mmHg, or a pressure gradient between the portal venous territories and cellar than five mmHg\(^8\). The pressure in the portal system depends on both the portal blood flow and intrahepatic vascular resistance, correspondent Ohm's Law\(^{5,9} \), The increase of one or other of these factors or both generates PHT. PHT is characterized by hyperkinetic syndrome secondary to systemic
arteriolar dilation that installs vasopressors despite activation systems\(^\text{10}\). During normal pregnancy, there are more hemodynamic changes that predispose to increased portal pressure, the occurrence of transient esophageal varices and rupture of these varices\(^\text{7}\). Pregnancy is also accompanied by anatomical and physiological changes that alter the results of physical examinations and biochemical liver tests\(^\text{11}\). However, normal pregnancy does not interfere, to a significant degree metabolism or liver function\(^\text{11,12}\). In late pregnancy, the gravid uterus compresses the inferior vena cava, and may even directly compressing the portal vein. Venous return will be through the gastroesophageal system increasing the risk of developing esophageal varices (OV). These veins appear from a hepatic venous pressure gradient exceeding 12mmHg. At this pressure, the veins in the collateral circulation dilate and become twisted thereby forming varices. More varicose veins have a high pressure, a large radius, and a thin wall, the more they are susceptible to breakage.

The incidence of PHT association and pregnancy is different\(^\text{6}\). It is variable depending on the etiology of liver disease\(^\text{6}\). Aggarwal et al. report a series of 30 pregnant patients with PHT: 83.2% were non-cirrhotic, whereas 16.8% had cirrhosis\(^\text{4,22}\). Liver cirrhosis occurs at a relatively advanced age compared to The child-bearing age\(^\text{4,6,19,26}\). The prevalence of cirrhosis in women of reproductive age was 0.45 cases / 1000\(^\text{18}\). The maternal mortality rate varies, depending on the group of patients studied, 10 to 60%\(^\text{5,18}\). It seems related to the initial liver condition of the patient\(^\text{2}\). The causes of death include hematemesis, hepatic coma, peritonitis, splenic aneurysm\(^\text{4}\). In our study, age was between 22 to 35 years. A single case of maternal death was observed the persistence of postpartum bleeding disorder.

The impact of pregnancy on the PHT is discussed. Cheng et al. consider that pregnancy affects the prognosis of patients suffering from cirrhosis PHT: The hepatic coma increases with pregnancy due to the increase of estrogen that is toxic to the liver\(^\text{3}\). Borhanmanesh when has it, found that pregnancy has no damaging effect on the prognosis of maternal cirrhosis\(^\text{13}\). However, the complications of PHT during pregnancy appear more in cirrhotic patients. The main complications are represented by a possible hepatic encephalopathy, gastrointestinal bleeding caused by the action of hyper blood volume on esophageal varices, and a gradual worsening of preexisting liver failure in the postpartum period\(^\text{4,14}\). However, a healthy liver, breast prognosis is good, and pregnancy has no effect on liver function. Maternal mortality varies, depending on the group of patients studied, 10 to 60%\(^\text{1,12}\).

The bleeding by rupture of esophageal varices is the most serious complication, with a serious impact on both the mother and the fetus\(^\text{15}\). PHT is a VO bleeding risk factors during and after pregnancy\(^\text{16}\). The estimate of this risk parturients reached PHT is very important that during a normal pregnancy\(^\text{6,17}\). Its incidence varies according to the series from 62% to 78\%\(^\text{1,5,18,19}\). Byrd et al. reported 50 pregnancies in 27 cirrhotic and non-cirrhotic patients; bleeding VO occurred in 34% of cases\(^\text{20}\). Cheng relates bleeding by rupture of VO in 44% of cases of 32 cirrhotic and non-cirrhotic pregnancies\(^\text{21}\). The original version of rupture risk is variable depending on the etiology of PHT, it is higher in the case of extra PHT liver, occurring in over 43% of cases, while it is 23% in patients with cirrhosis\(^\text{22}\). Pregnancy does not appear to alter liver function in case of extra-hepatic hypotension\(^\text{23}\). However, changes in liver function in cirrhotic patients during pregnancy is described as unpredictable. Indeed, it can either remain stable or worsen\(^\text{24,25}\).

The influence of PHT on the motherboard can be represented by the occurrence of preeclampsia\(^\text{6}\). Its incidence is higher in patients carrying extrahepatic PHT with a rate of 9.37%, compared to patients with cirrhosis 7.62\%\(^\text{13,6,21,23}\). Postpartum hemorrhage is secondary to bleeding disorders, mainly deficiency of factor V and VII, and thrombocytopenia due to hypersplenism\(^\text{18,19}\). The fetal complications if maternal PHT arrives in 30 to 40% of cases. They are dominated mainly by spontaneous abortions, premature birth and perinatal mortality\(^\text{18}\). Fetal mortality remains high; it oscillates between 11% and 18%\(^\text{3}\). In our study, there was only one case of fetal death in utero.

The optimal management of women in labor abuses of PHT requires a coordinated approach by a multidisciplinary team including an obstetrician, a hepatologist performance in endoscopy, a resuscitator and a neonatologist\(^\text{19,26,27}\). The patient and family must always be informed of the risks of this pregnancy. Maternal prognosis is directly related to the existence of VO and the risk of impact on liver function, which warrants support in a level 3 maternity\(^\text{19,24}\). The risk of intrauterine growth restriction and necessary prematurity support in neonatal intensive care units\(^\text{7}\). The care of these patients begins with a pre-conceptional consultation where liver damage must be assessed, and
programming of pregnancy may be performed when the disease is stable\(^\text{40}\). It would then be closely monitored throughout pregnancy, labor and delivery, and postpartum to prevent and detect complications.

4. CONCLUSION

The occurrence of pregnancy in a patient with portal hypertension is an unusual situation for the clinician. Although the majority of portal hypertension during pregnancy is not fully demonstrated, there is an undeniable on-morbidity, especially in patients with cirrhosis, which justifies the notion of pregnancy risk and requires a multidisciplinary approach. The bleeding by rupture of esophageal varices is the most serious complication. The means of preventing or fighting against this bleeding are little different from those available outside of pregnancy. Pregnancy must always be programmed with a pre-conceptional care and close monitoring. Research and management of portal hypertension and its complications before or early in pregnancy may reduce the risk of miscarriage, premature delivery, intrauterine growth retardation and maternal complications essentially maternal mortality. The risks associated with the underlying disease should also be considered, whether a liver failure in cirrhosis or thromboembolic risk for vascular liver diseases. It is currently permitted to most women with portal hypertension, to lead a normal pregnancy, subject to adequate care from conception to postpartum. The vaginal delivery, as early and assisted analgesia started an instrumental delivery, should be preferred to cesarean section, the indications must remain in order obstetrical.

REFERENCES

