Decompensated cirrhosis and pregnancy: a case report

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Summary

The association of decompensated cirrhosis and pregnancy is rare. Portal hypertension exposure to gastrointestinal bleeding from a ruptured esophageal varix may at any time complicate the course of the disease. We report the case of a 24-year-old patient who delivered at 35 weeks/four days of gestation with decompensated cirrhosis secondary to viral hepatitis B; icterus, oedema, and ascites were present. The postpartum course was uneventful despite the biological disorder of coagulation.

Key words: Pregnancy; Decompensated cirrhosis.

Introduction

Liver diseases in pregnancy may be categorized into liver disorders that occur only in the setting of pregnancy and liver diseases that occur coincidentally with pregnancy [1]. The association of decompensated liver cirrhosis and pregnancy is a rare event [2]. It is a high-risk pregnancy. Portal hypertension and gastrointestinal bleeding mostly by rupture of the oesophageal or gastric varix may at any time complicate the course of the disease. These occur mainly in the third trimester of pregnancy and the postpartum period [3, 4]. We report the case of a young woman with decompensated cirrhosis and ascites secondary to viral hepatitis B who gave birth.

Case Report

A 24-year-old housewife, fourth gravida and primipara, has suffered from hepatitis B virus since 2000. Her history showed a vaginal delivery in 2005 of a living child (birth weight 3000 g). She had had two arrested pregnancies at 11 weeks of amenorrhea and 18 weeks of amenorrhea in 2009. The diagnosis of ascites and histological confirmation of cirrhosis was made in 2006 in the Gastroenterology Department of the University Hospital of Cocody. The patient was sent on June 9, 2010 to a peripheral maternity ward due to abundance of ascites associated with evolutive pregnancy. The examination for admission to the maternity ward of the university hospital of Cocody (09-06-2010) showed an ongoing pregnancy of 35 weeks/1 day (by early ultrasound performed at the 9th week), important ascites, oedema and dyspnea at rest. She had conjunctival icterus but no fever or hepatic encephalopathy. Blood pressure was 130/90 mmHg. Ultrasound showed intrauterine growth retardation (IUGR), liver cirrhosis, splenomegaly homogeneous and high abundance ascites. The recording of the foetal heart rate was normal with a basic rhythm to 130 bpm.

Laboratory renal tests were normal (urea = 0.15 g/dl, glucose = 0.78 g/dl, creatinine = 7 mg/µl). There was liver failure (ALAT = 45 IU/l, ASAT = 77 IU/l). Haemostasis was disrupted (platelets = 76000/mm^3; prothrombin rate = 44.6%; fibrinogen = 1.15g/l). Hemoglobin was 11.2 g/dl; serum albumin was 36 g/l and bilirubin equal to 30 µmol/l. The prognostic score of Child-Pugh was grade B 8. Gastroscopy in search of varices was not performed in this patient due to lack of funds. Caesarean section was indicated due to IUGR, which was to be executed after correction of the coagulation disorders.

Dyspnea was resolved after removal of four liters of ascites whose cytology examination was normal. The patient was systematically put under beta-blockers and antibiotics. She received 1545 ml of fresh frozen plasma (FFP), 743 ml of packed red blood cells and 90 mg of vitamin K1. During the correction of disorders of haemostasis on the third day, the patient spontaneously went into labour and gave birth to a hypotrophic newborn (after a quick and easy expulsion) weighing 2150 g, (Apgar score was 7-8 at 1 min and 5 min). Placental stage was determined by controlled cord traction followed by continuous infusion of syntocinon. The immediate postpartum was uneventful. The baby was discharged to neonatology for neonatal distress syndrome and the mother was sent to the hepatogastroenterology unit for the chronic liver disease. US performed in March (15/03/2010) at 23 weeks of amenorrhea showed foetal dimensions, homogenous splenomegaly, a heterogeneous liver and an abundance of ascites (Figure 1 a-c).

Discussion

Although uncommon, women with cirrhosis may become pregnant and may have a relatively benign course of pregnancy [1], even with the exceptional decompensated cirrhosis. The late age of onset of liver damage and hormonal changes inducing hypo-fertility explain this rarity [3]. Note however that the actual frequency of this association remains unclear, and in the literature only a few cases or a few short series of cases have been described [5-8]. Our observation is the second documented case in our country after the first was published in 2007 [9].

The cirrhosis was secondary to viral hepatitis B infection in our patient. Indeed, while the origin of ethyl cirrhosis predominates in developed countries [3], viral hepatitis B – endemic in our region – is the most common etiology of chronic haepathopathies in Black Africa [10, 11]. Liver cirrhosis occurs at a relatively advanced age and the association of cirrhosis and pregnancy occurs mainly between 30 and 35 years [12]. However, cases of young patients like our case have been described [5].

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Fig. 1a

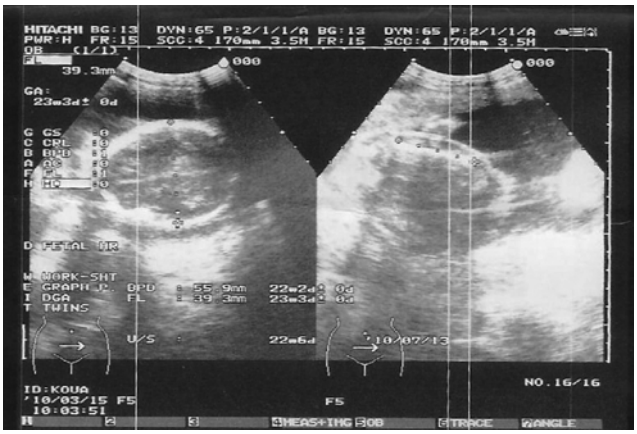

Fig. 1b

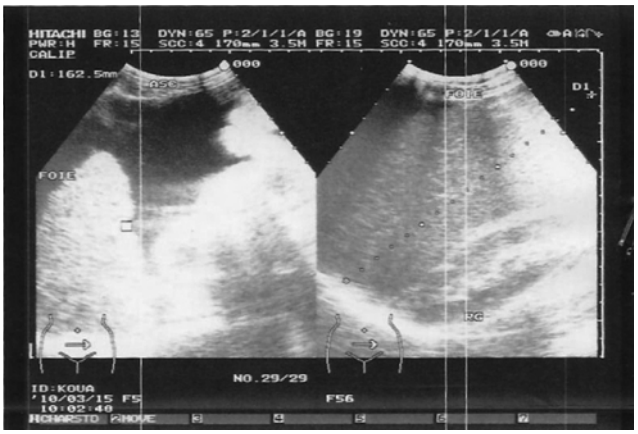

Fig. 1c

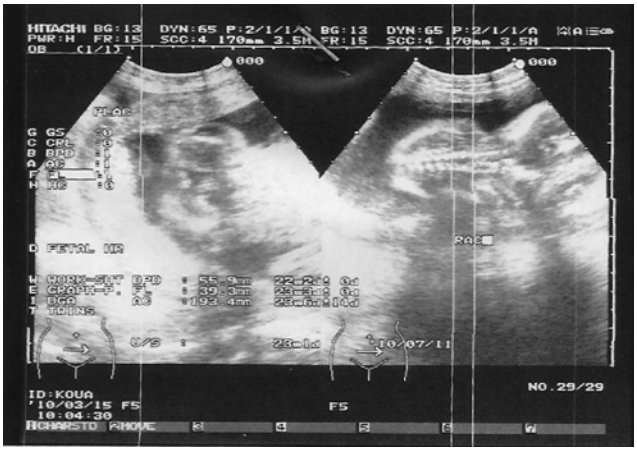

Figure 1. — Ultrasonography.

Figure 1a. — Foetal development at the 23rd weeks of gestation.

Figure 1b. — Heterogeneous liver and abundance of ascites.

Figure 1c. — Dimensions of the foetus with homogeneous splenomegaly.

Our patient had two pregnancies which were arrested at the stage of decompensated cirrhosis. She then gave birth to a child at the 36th week that was hypotrophic but otherwise healthy. Indeed, at that stage of cirrhosis, foetal risks are increased and there is more frequent foetal death [5]. Risks also exist even without any real decompensation of liver function such as in our patient [5]. Cases of miscarriage and pregnancies carried to live term birth in the same women with cirrhosis have been published [4, 7].

In general, the delivery route of choice is not influenced by the existence of liver disease; caesarean section always maintaining its traditional obstetric indications [13, 14]. Some argue that in case of acute liver failure, the emergency retrieval of a premature foetus even seems justified [15]. The rate of caesarean section is higher among pregnant women with cirrhosis compared with general obstetric patients [16].

An indication for caesarean section was assumed in our patient due to IUGR. However the caesarean was not performed immediately because of the major disorders of haemostasis. Faced with these major disorders (prothrombin rate = 44.6%, fibrinogen = 1.15 g/l, platelets = 76000/mm³) our patient needed priority of delivery in order to prevent haemorrhage at that placental stage. The patient received fresh frozen plasma, red cell concentrate and vitamin K1. Fibrinogen is not easily available in our pharmacies. Indeed, the vascular filling pre- and intraoperatively must be associated with the transfusion of FFP and/or platelets in case of biological abnormalities that warrant it. Platelet transfusion is recommended immediately prior to caesarean cases of thrombocytopenia less than 50000/mm³ and immediately prior to vaginal delivery in cases of thrombocytopenia less than 30000/mm³ [17]. FFP transfusion in obstetrics is recommended in case of bleeding associated with disseminated intravascular coagulation syndrome and in patients with an impaired haepatocellular condition; FFP transfusion is also indicated in case of bleeding or an invasive procedure [17]. In our case, the transfusion of FFP was justified by the pre-existing coagulation abnormalities and caesarean section indicated due to IUGR. Her platelet count did not justify a platelet transfusion.

The woman gave birth spontaneously vaginally with a quick and easy expulsion. The instrumental delivery might be justified in difficult cases of expulsion to minimise the patient's expulsive efforts that could increase the risk of bleeding [5]. The placental stage was made by cord traction control followed by continuous infusion of

syntocinon. The postpartum course was uneventful. However, it is worth noting that during the postpartum period maternal risk remains dominated by major postpartum haemorrhage due to the cumulative effect of thrombocytopenia because of hypersplenism and bleeding disorders resulting from alterations in hepatic synthesis of certain coagulation factors [4]. It is an almost constant risk that must be rigorously prevented.

Conclusion

The association of cirrhosis and pregnancy is a high-risk combination. However, cirrhosis is not a contraindication, as pregnancy may be tolerated if cirrhosis is well compensated. The maternal risk is directly related to the existence of portal hypertension and oesophageal varix which should be systematically investigated in early pregnancy. In the postpartum period, the fear of postpartum haemorrhage requires close monitoring of blood coagulation.

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