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Case Report **Medicine**

Decompensation of Chronic Liver Disease during Pregnancy, a Case Report

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Abstract

Chronic hepatopathies are cosmopolitan disorders, the most common of which are chronic hepatitis and cirrhosis. Cirrhosis is a pathology of diverse etiologies characterized by diffuse disorganization of the normal hepatic structure due to hepatocyte destruction, with the appearance of regenerative nodules surrounded by fibrosis compressing the hepatic vascularization and causing portal hypertension. We report the case of a patient hospitalized in intensive care for decompensation of chronic hepathopathy during her 4th pregnancy, and we will describe the various consequences of pregnancy on this type of hepathopathy and vice versa via a detailed review of the literature.

Keywords: Pregnancy, Chronic liver disease, Cirrhosis, portal hypertension, ascites, case report.

INTRODUCTION

Chronic hepatopathies are cosmopolitan disorders, the most common of which are chronic hepatitis and cirrhosis.

Cirrhosis is a pathology of diverse etiologies characterized by diffuse disorganization of the normal hepatic structure due to hepatocyte destruction, with the appearance of regenerative nodules surrounded by fibrosis compressing the hepatic vascularization and causing portal hypertension [1, 2].

We report the case of a patient hospitalized in intensive care for decompensation of chronic hepathopathy during her 4th pregnancy, and we will describe the various consequences of pregnancy on this type of hepathopathy and vice versa via a detailed review of the literature.

OBSERVATION

We report the case of a 44-year-old female patient, multiparous with 3 previous pregnancies without incident, vaginal delivery with 3 live children.

She was admitted during her fourth pregnancy at 33 weeks' gestation for jaundice with haematemesis. Initial assessment revealed a conscious and lucid patient, eupneic but slightly tachycardic at 110 beats per minute, with blood pressure at 14/7 cmH2O, soft oedema of both lower limbs, taking the bucket.

The rest of the examination revealed a mucocutaneous uterus with normal uterine height, positive cardio-fetal noises, a long posterior closed cervix on vaginal touch and a progressive monofetal pregnancy on obstetrical ultrasound. Biological workup showed microcytic anemia at 9.4 g/dl, thrombocytopenia at 85,000 elements per mm3, PT at 45%, hypoglycemia at 0.6 g/l, with mild cholestasis at 50 total bilirubin, in the absence of hepatic cytolysis. All serologies were negative and abdominal ultrasonography revealed a chronic liver disease with abundant ascites and portal hypertension. An abdominal CT scan revealed a dysmorphic liver with bumpy contours, hypertrophy of segment 1 and atrophy of segment 4, with no associated nodules. High-calibre portal trunk with partial thrombosis of the latter with perigastric colateral venous circulation and an enlarged spleen, homogeneous in appearance.

Fibroscopy revealed stage 1 esophageal varices with pangastritis. Ascites puncture yielded 3 liters of citrine-yellow fluid, rivalta negative with biochemical protein < 8g/l and bacteriological culture negative.

The evolution was marked by a preterm vaginal delivery, precisely at 34 weeks' amenorrhea, of a hypotrophic female neonate, APGAR 10/10, with a birth weight of 1900g.

The patient subsequently began to deteriorate in GCS with tachycardia, arterial hypotension and respiratory distress. She was transferred to intensive care, conditioned by filling, introduction of vasopressor drugs and orotracheal intubation. Cerebral CT came back without abnormality, and thoracic CT identified inhalation pneumopathy.

Hepatic encephalopathy was considered in view of the altered state of consciousness, with a decrease in PT from 33% to 5% and episodes of hypoglycemia, in the absence of any other metabolic abnormality. The patient was put on antibiotic therapy based on tazocillin + azobactam and amikacin, with transfusion of FFP and packed red blood cells, and sedation was maintained for 48 hours.

The evolution was favorable, with recovery of normal consciousness on discontinuation of sedation, noradrenaline withdrawal and improvement of the exchanger.

The patient was transferred to the gastrohepato-enterology department for further etiological evaluation.

DISCUSSION

Chronic hepatopathies are cosmopolitan disorders, the most common of which are chronic hepatitis and cirrhosis.

Cirrhosis is a pathology of diverse etiologies characterized by a diffuse disorganization of the normal hepatic structure due to hepatocyte destruction, with the appearance of regenerative nodules surrounded by fibrosis compressing the hepatic vascularization and causing portal hypertension.

The most frequent cause is chronic viral hepatitis, but other pathologies have also been incriminated, such as BUDD CHIARI syndrome, WILSON disease, GAUCHER disease, primary biliary cirrhosis and autoimmune cirrhosis [1, 2]. During pregnancy, several changes occur in the hemodynamic system. As far as the liver is concerned, there is an increase in hepatic cholesterol synthesis, with increased coagulation factors and alkaline phosphatases [3].

The association of cirrhosis and pregnancy is rare. Hepatic cirrhosis generally leads to metabolic and endocrine disorders responsible for infertility. Non-cirrhotic portal hypertension is also associated with hypofertility, the mechanism of which remains unclear [4].

Hepatic cirrhosis occurs at a relatively advanced age, and cirrhotic women are generally no longer of childbearing age [5]. Pregnancy, while quite rare in hepatic cirrhosis, appears to be more frequent in post-hepatic forms than in alcoholic forms, given the greater number of women in this category who are still fertile. Caroli and Chevrel [6] believe that such a difference in distribution is due to the fact that alcoholic cirrhosis, when it occurs in still-young subjects, generally presents an acute, febrile course that is much more severe than that presented by common cirrhosis in middle age.

In the case of biliary cirrhosis or Bantian cirrhosis, hepatocellular function is compromised only in the most advanced phases of the disease, which explains why patients have tolerated childbirth relatively well [7].

Wilson's disease is always in its early stages, and liver function is well preserved. Pregnancy will cause a worsening of nervous disorders in these patients, which will last during the breast-feeding period; this would be due not so much to the physiological increase in ceruleoplasmin that pregnancy provokes, as to a mobilization by the fetus' needs of the excess copper in maternal deposits [8].

In fact, the main causes of pregnancy termination in women with chronic liver disease during the 1st trimester are spontaneous abortion in 18 to 20% of cases, and therapeutic abortion. During the 2nd and 3rd trimesters, digestive haemorrhage due to ruptured varices is the main cause of pregnancy termination.

It has been established that patients who had their esophageal varices treated prior to pregnancy had fewer complications during delivery, and a lower fetal mortality rate. However, there are no data on the complications of vaginal delivery compared with vaginal delivery [2].

Prematurity occurs between the 2nd and 37th week of amenorrhea, in 20.5% of a series of 117 pregnancies in cirrhotic women, and in 18.75% in a series of 32 women with PH due to extrahepatic obstruction.

According to a study by CHENG, perinatal death accounted for 17.85% of cirrhotic patients and 11.5% of parturients with PH [9]. Pregnancy in cirrhotic women also entails numerous dangers, the most important of which are possible terminal hepatic coma, possible digestive haemorrhage caused by the action of hypervolaemia on oesophageal varices, and progressive worsening of pre-existing hepatic insufficiency in the post-partum period [7].

Hemorrhage due to rupture of esophageal varices is the major risk in women with PH during

pregnancy. In some studies, it may account for up to 44% of cases.

In cases of hematemesis resistant to medical resuscitation and Blackmore catheterization, surgical anastomosis can be performed during pregnancy.

Concerning maternal mortality, a total of 19 deaths in cirrhotic parturients have been reported in the literature. Causes of death include: hematemesis, hepatic coma, peritonitis, splenic aneurysm rupture [9, 10].

CONCLUSION

The occurrence of pregnancy in cirrhosis is rare, difficult to manage, and the prognosis is severe. It is a high-risk pregnancy, since pregnancy increases the risk of cirrhosis decompensation and vice versa.

This calls for a thorough understanding of the pathophysiology of both pregnancy and cirrhosis, and coordination between the obstetrician, hepatologist, neonatologist and intensive care anesthetist, with appropriate management and rigorous monitoring before, during and after pregnancy, so that any parturient can bring her pregnancy to term with the least possible risk of complications.

Conflict of Interest: No

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