CASE REPORT

Bleeding from oesophageal varices in pregnancy

Marta Sobral,¹ Carla Granja,² Margarida Sampaio,² Fernando Guerreiro²¹Department of Serviço de Ginecologia, Hospital de Faro, EPE, Faro, Portugal²Department of Obstetrics and Gynecology, Centro Hospitalar do Barlavento Algarvio, Portimão, Portugal**Correspondence to**Dr Marta Sobral,
martasobral04@yahoo.com.br**SUMMARY**

Bleeding from oesophageal varices is the most fearful event in pregnant women with cirrhosis and is the main cause of death in this group. Its approach and treatment are controversial, with ligation, sclerosis and β -blockers as possible choices. Follow-up must be multidisciplinary and delivery programmed because of the high risk of bleeding. Delivery route is also controversial. We present a case of a pregnant woman, with alcoholic cirrhosis and upper tract bleeding during the second trimester of pregnancy. She was submitted to an oesophagogastroduodenoscopy (OGD), which revealed varices grade II/III in the lower third of the oesophagus. She was transfused and was haemodynamically stable. It was decided not to start β -blockers. The pregnancy went well, with regular follow-up in obstetrics and hepatology. A deficiency of α -1 antitrypsin was detected. Delivery was programmed at 39 weeks of gestation, and a caesarean section was performed, with good maternal and fetal outcome.

BACKGROUND

Pregnancy is a rare event in patients with cirrhosis, because advanced liver disease does not typically occur until well after most patients have completed their reproductive years, and because cirrhosis results in metabolic and hormonal derangements that lead to anovulation and amenorrhoea.¹

Liver cirrhosis is also related to an increase in the risk of preterm delivery and perinatal mortality, and to maternal complications such as anaemia, pre-eclampsia, postpartum haemorrhage and oesophageal varices bleeding. Oesophageal variceal bleeding is one of the most fearful events in pregnant women with cirrhosis and is the main cause of maternal mortality, reported to be as high as 10.5%.^{1 2}

The case we present highlights the importance of the multidisciplinary follow-up of pregnant women with cirrhosis and shows the challenge of the management of bleeding from oesophageal varices in pregnancy, in a woman with ethanol-induced cirrhosis and with α -1 antitrypsin deficiency. This case shows that the optimal management of pregnancy with cirrhosis and portal hypertension in modern obstetrics is undefined.

CASE PRESENTATION

Our patient was a 35-year-old woman, whose occupation was as a waitress. Her menarche was at 12 years, with regular cycles of 28 days and lasting 4 days. She had had a spontaneous abortion at 6 weeks of gestation, without complications, and a previous normal pregnancy with a vaginal delivery at 39 weeks of gestation in 2004, with a healthy

newborn weighing 2400 g. She was a smoker and had a history of excessive alcohol consumption since she was 17 years old, and was on follow-up in hepatology because of liver cirrhosis.

This pregnancy was unplanned and was followed by her family doctor until 23 weeks of gestation. In the first trimester of pregnancy, liver biochemistry was normal and the obstetric ultrasound revealed fetus of 12 weeks and 4 days, nuchal translucency in the 50th centile for the crown-rump length and the nasal bone was present. Free β -human chorionic gonadotropin (B-HCG) and pregnancy associated plasma protein A (PAPP-A) in maternal blood were not determined. Haemoglobin was 11.9 mg/dL, and she was tested negative for hepatitis C, hepatitis B, HIV 1, HIV 2 and syphilis. She reported no discomfort and she denied alcohol consumption in the past 10 months.

In the second trimester ultrasound, the fetus was growing according to expectations and no malformations were detected. At 24 weeks and 3 days of gestation, she was admitted in the emergency room with haematemesis and melena. She was haemodynamically stable and fetal well-being was confirmed. Physical examination was normal.

INVESTIGATIONS

An urgent OGD was performed, which revealed grade II–III varices in the lower two-third of the oesophagus, and a small clot, in one of the varices in the gastro-oesophagic junction, was detected. The liver biochemistry was alanine transaminase (ALT) 97 IU/L and aspartate aminotransferase (AST) 107 IU/L, γ -glutamyl transpeptidase (GGT) 112 IU/L. We investigated α -1 antitrypsin, which was 75 mg/dL, below expected (reference range 90–200 mg/dL), mainly because this protein rises during pregnancy. The autoimmunity study was negative and ceruloplasmin was normal.

TREATMENT

Haemoglobin was 7.6 g/dL, and she was transfused with one unit of red blood cells. The gastroenterologist decided not to perform band ligation or sclerotherapy, as she was haemodynamically stable, and there was no active bleeding.

She stayed in observation for 3 days and later she was followed in hepatology and high-risk pregnancy appointments. She was started on iron and folic acid supplement. It was decided not to initiate β -blockers, as she was clinically and biochemically stable.

OUTCOME AND FOLLOW-UP

There were no other complications during pregnancy. The fetus was growing accordingly to what

To cite: Sobral M, Granja C, Sampaio M, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-009653

was expected and the amniotic fluid and umbilical artery Doppler were normal.

At 39 weeks of gestation, an elective caesarean section was performed under spinal anaesthesia, because of the risk of variceal haemorrhage. A healthy baby was born, with Apgar score of 10 at first and fifth minute, with 3100 g. A tube ligation was performed on patient's choice. The placental histological evaluation was normal.

The postpartum was without complications, and the newborn was breastfed. Hepatic function remained stable and there was no other episode of bleeding from oesophageal varices.

The patient was followed in hepatology, and a second determination of α -1 antitrypsin (62 mg/dL) was made 6 months after the delivery, which was also below the expected, confirming α -1 antitrypsin deficiency. The genetic study revealed that the patient is heterozygotic for the mutation Gln264Val in α -1 antitrypsin gene. This result corresponds to the phenotype PI*MS.

She was also submitted to two elective sessions of endoscopic band ligation and so far, no more bleeding from oesophageal varices was registered.

DISCUSSION

The ideal orientation and treatment of the pregnant women with cirrhosis with portal hypertension is unclear in the modern obstetrics. Although it is difficult to distinguish the effects of pregnancy from the natural history of cirrhosis, maternal complications have been described in nearly half of pregnancies affected by cirrhosis and portal hypertension, largely as a result of variceal haemorrhage and liver failure, and there is also an increased risk of fetal loss.^{1 3 4}

The main maternal risk is bleeding from oesophageal varices, reported in 18–32% of the cases and in up to 50% of those with known portal hypertension, especially in the second and third trimester, when maternal blood volume is maximally expanded and the larger fetus causes increased compression of the inferior vena cava and collateral vasculature, or during delivery.^{1 2}

In cases of known oesophageal varices, the prophylactic treatment of varices in pregnant women remains controversial. Some authors recommend therapeutic measures before pregnancy, such as endoscopic therapy, shunt surgery or hepatic transplant.

In addition, all patients, even if there are no varices before pregnancy, should undergo OGD for assessment of varices in the second trimester and, if large varices are present, then β -blocker therapy is introduced despite occasional fetal effects.

Upper endoscopy in general appears to be safe during pregnancy, with the main risk being fetal hypoxia from sedative drugs or positioning.⁵

Acute variceal bleeding is managed endoscopically, as in the non-pregnant, with band ligation or sclerotherapy, though vasopressin is contraindicated because of arteriolar vasospasm and an increased risk of placental abruption, myocardial infarction, peripheral ischaemia and hypertension. Octreotide, category B by the Food and Drug Administration (FDA), is often used to treat acute variceal bleeding, although its safety has not been well studied in pregnant patients.

To decrease portal hypertension, propranolol can be used (FDA category C), taking into account the risk of fetal growth

restriction—some authors use it only as a primary prophylaxis in high-risk women.^{1 2 6}

Concerning the α -1 antitrypsin deficiency associated to PI*MS, which is common in the Portuguese population, is not associated with an increased risk for hepatic disease and does not result in protein accumulation in the hepatocytes, contrary to what happens in the PI*ZZ and PI*MZ phenotypes. The increase in the risk of progression of the hepatic disease when combined with alcohol abuse and α -1 antitrypsin is not demonstrated.^{7 8}

Delivery in these women is controversial, but should be programmed because of the risk of bleeding.⁶ The rise of the abdominal pressure in the Valsalva manoeuvres during delivery can increase portal hypertension and cause bleeding from varices. Some authors defend performing an elective caesarean section or an assisted delivery with vacuum or forceps, with extradural anaesthesia.^{1 2 6}

After delivery, effective contraception is important. Oral contraceptives should not be used.

Learning points

- ▶ Pregnancy in women with cirrhosis requires multidisciplinary approach.
- ▶ The ideal orientation and treatment of the pregnant women with cirrhosis with portal hypertension is unclear.
- ▶ Bleeding from oesophageal varices is related to high maternal mortality.
- ▶ Ligation or sclerosis can be used to treat oesophageal varices, and propranolol is an option to decrease portal hypertension.
- ▶ Delivery is also controversial, with elective caesarean or assisted delivery as possible options, to decrease the risk of haemorrhage from varices with the rise of pressure during delivery.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Tan J, Surti B. Pregnancy and cirrhosis. *Liver Transpl* 2008;14:1081–91.
- 2 Russel MA, Graigo SD. Cirrhosis and portal hypertension in pregnancy. *Semin Perinatol* 1998;22:156–65.
- 3 Hay JE. Liver disease in pregnancy. *Hepatology* 2008;47:1067–76.
- 4 Lee NM, Brady CW. Liver disease in pregnancy. *World J Gastroenterol* 2009;15:897–906.
- 5 O'Mahony S. Endoscopy in pregnancy. *Best Pract Res Clin Gastroenterol* 2007;21:893–9.
- 6 Heriot JA, Steven CM, Sattin RS. Elective forceps delivery and extradural anaesthesia in a primigravida with portal hypertension and esophageal varices. *Br J Anaesth* 1996;76:325–7.
- 7 Fairbanks KD, Tavill AS. Liver disease in alpha 1-antitrypsin deficiency: a review. *Am J Gastroenterol* 2008;103:2136.
- 8 Bowlus CL, Willner I, Zern MA, et al. Factors associated with advanced liver disease in adults with alpha1-antitrypsin deficiency. *Clin Gastroenterol Hepatol* 2005;3:390–6.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow