CASE REPORT

Pregnancy in a patient with portal hypertension secondary to liver cirrhosis

Faith Jelagat Lelei-Mailu,¹ Charles Muriuki Mariara²

¹Department of Family Medicine,
AIC Kijabe Hospital, Kijabe,
Kenya

²Department of Obstetrics
and Gynaecology, AIC Kijabe
Hospital, Kenya

Correspondence to
Dr Faith Jelagat Lelei-Mailu,
faithlelei@gmail.com

Accepted 9 January 2018

SUMMARY

This case report is of a 32-year-old woman of African descent on follow-up for pregnancy in the background of portal hypertension due to liver cirrhosis. She had initially been treated for chronic hepatitis B infection with lamivudine and tenofovir, complicated by portal hypertension and variceal bleeding that thrice required banding. Her pregnancy was uneventful until 31 weeks gestation when she presented with dyspnoea. On examination and investigation, she had oedema, bilateral pleural effusions and ascites. Multidisciplinary discussions involving surgery, anaesthesia, obstetrics, neonatology and medicine were held. A consensus outpatient and inpatient management plan was implemented. At 36 weeks, following non-reassuring fetal cardiotocography, she underwent induction of labour. An assisted vacuum delivery was conducted in a controlled setting. She gave birth to a live female infant who had an APGAR score of 9 at 5 min. Both she and the baby had an uneventful postpartum period.

BACKGROUND

Pregnancy in the setting of liver cirrhosis is uncommon due to hypothalamic–pituitary dysfunction with associated disturbances in endocrine metabolism, especially oestrogen.¹ The obstetric outcomes of pregnant patients with liver cirrhosis in Sub-Saharan Africa are not well described. A population-based study reviewing the outcomes of pregnant women with liver cirrhosis found that gestational hypertension, placenta abruption and uterovaginal haemorrhage were more common in this population. The rate of spontaneous miscarriages was significantly higher,² as was the risk of prematurity and perinatal death.³ Hepatic decompensation occurred in 15% of patients, including 11% with ascites and 5% with variceal bleed.² In women with hepatic decompensation, maternal and fetal mortality was 6% and 12%, respectively. Portal hypertension is a major complication of liver cirrhosis. Oesophageal and gastric varices, hepatic decompensation and encephalopathy, spontaneous bacterial peritonitis and postpartum uterine haemorrhage are all potential maternal complications.^{2,3} Termination of pregnancy may be required in instances where the mother gets a variceal bleed or intra-uterine growth restriction.² Due to these potential complications, a multidisciplinary approach is generally recommended.^{1,2}

CASE PRESENTATION

This is a case report of a 32-year-old woman of African descent who presented in her second pregnancy. She had been diagnosed with portal hypertension and oesophageal varices secondary to liver cirrhosis 7 years prior. The cirrhosis was from underlying chronic hepatitis B.

During her initial presentation at a different facility with variceal bleeding, she underwent oesophageal banding and was commenced on tenofovir and lamivudine for the hepatitis B infection. She presented to our facility (AIC Kijabe Hospital, Kijabe) 1 year later during her third variceal bleed as an emergency having discontinued her antiviral therapy after 2 months of treatment. Initial tests suggested that portal hypertension was a result of liver cirrhosis (following hepatitis B infection). The hepatitis B surface antigen (HBsAg) test was still positive but the initial hepatitis B virus (HBV) serological tests were unavailable. She was initiated and maintained on propranolol 40 mg once a day, spironolactone 25 mg once a day, furosemide 40 mg once a day and proguanil 100 mg once a day (for possible malarial splenomegaly).

Her first pregnancy 8 years prior and antecedent to HBV infection was uneventful. She had a normal delivery at term to a live infant weighing 2600 g.

She first presented to the antenatal clinic in this current pregnancy at 13 weeks gestation (calculated from her last menstrual period). She did not have any complaints and her vital signs were normal. Her abdominal examination was significant for splenomegaly whose lower edge was 10 cm below the costal margin and a non-palpable liver. There was neither distended veins nor ascites noted during this initial visit. The rest of her clinical examination was normal. Her antenatal profile test results were as follows: haemoglobin 12.2 g/dL (12–16), Venereal Disease Research Laboratory test (VDRL) and HIV were negative. Her blood group was O+ and HBsAg was negative. There was clinical suspicion of seroconversion but this could not be confirmed by a positive anti-HBs serology due to cost implications. A urinalysis revealed 500 leucocytes though she was asymptomatic. She was treated with cefuroxime 500 mg twice a day for 7 days. Notably, her platelet counts were low at $46 \times 10^9/L$. The thrombocytopenia was present preconception, and persisted through to her postpartum period (table 1).

She had an obstetric ultrasound that showed a viable pregnancy with demise of one twin. The ultrasound estimated the gestation at 14 weeks and 3 days. She was initiated on haematinics.

To cite: Lelei-Mailu FJ, Mariara CM. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-223076

Table 1 Laboratory results

	Hb (11–13 g/dL)	WBC (4000–10 000 cells/mm ³)	Platelets (150 –450 × 10 ⁹ /L)	SGPT (0–31 U/L)	SGOT (0–31 U/L)	PT (s)	INR	Albumin (3.8–5.1)	Creatinine (mg/dL)
Preconception	11.6	2400	28		51				0.83
13 weeks	12.2		46						
25 weeks	11.6	3200	18	46				4.02	0.63
31 weeks	11.0	4600	28	40	38			3.73	1.17
34 weeks				36	34	21.4	1.84	2.98	0.7
36 weeks	11.2	5100	65	40	43	28.8	2.23		0.53
After delivery	11.7	8400	72	45	31				

Hb, haemoglobin; INR, international normalised ratio; PT, prothrombin time; SGPT, serum glutamic pyruvic transaminase; SGOT, serum glutamic oxaloacetic transaminase; WBC, white cell count.

Furosemide, propranolol, proguanil and spironolactone were discontinued due to their potential teratogenic effects.

An anomaly scan at 20 weeks gestation was normal and she continued her antenatal care follow-up at the high-risk clinic. At 31 weeks, she was admitted with lower limb swelling, shortness of breath and worsening ascites. She did not have any obstetric concerns. She was tachypnoeic with an oxygen saturation of 92% on room air. Her abdominal examination revealed flank fullness, ascites and an enlarged spleen 10 cm below the costal margin. The fundal height was assessed to be at 30 cm with a regular fetal heart rate of 148 beats per minute. On auscultation of her chest, she had good air entry bilaterally on the upper zones, but reduced air entry on the lower zones. An abdominal ultrasound confirmed the ascites, and pleural ultrasound confirmed the pleural effusion. The obstetric ultrasound gave an estimated fetal weight of 2011 g (within normal range) with a biophysical profile score of 8/8. During this admission she received antenatal steroids for fetal lung maturity, and required diuresis with furosemide 40 mg intravenously once a day. She also received vitamin K (10 mg intramuscular) to correct her deranged international normalised ratio (INR). A multidisciplinary discussion involving neonatology, medicine, surgery, anaesthesia and obstetrics was held to agree on a treatment plan including delivery. The consensus was to maintain the pregnancy and continue with antenatal care weekly at the high-risk clinic. She was subsequently discharged 2 weeks later at 33 weeks gestation following optimisation of her fluid status.

She was later readmitted 5 days after discharge for similar concerns namely: lower limb swelling, difficulty in breathing, abdominal distension and abdominal discomfort. During this admission, a decision to restart the 40 mg propranolol once a day was made as it was thought the benefits (reduction in portal pressures) outweighed the risks (mainly fetal growth restriction). Furosemide 40 mg once a day was also administered intravenously for diuresis. She was kept inpatient for close observation with the aim of balancing the benefits of extending the pregnancy against maternal safety. Fetal well-being was monitored by weekly ultrasounds and twice weekly non-stress tests (NSTs).

At 35 weeks, following a multidisciplinary discussion, it was decided to have an elective delivery at 36 weeks within a controlled environment. The delivery plans included having fresh cross-matched blood and platelet concentrates. At this time she had a thrombocytopenia of 56 000 cells/mm³, haemoglobin of 11.2 g/dL and an elevated prothrombin time/INR for which she received another stat dose of vitamin K 10 mg intramuscularly.

At 35+5 weeks, serial fetal NSTs were non-reassuring with minimal variability and variable decelerations. A decision was

made to induce her. She underwent mechanical induction with an extra-amniotic catheter placed in the cervix and inflated with 50 mL of normal saline. The Foley fell off after 8 hours and the cervix was 5 cm dilated and favourable. A membrane sweep and augmentation with oxytocin titrated to achieve adequate contractions was done.

When her cervix was 8 cm dilated, she was transferred to theatre where the multidisciplinary team (neonatology, surgery, anaesthesia and obstetrics) was present and ready for the delivery. Fresh cross-matched blood products and platelet concentrates were also available on standby. An assisted vaginal delivery with a vacuum was conducted with second stage lasting 20 min.

TREATMENT

Preconception, she was on furosemide, spironolactone, propranolol and proguanil, which were discontinued when she first presented at 13 weeks and haematinics initiated. At 31 weeks, she received antenatal steroids for fetal lung maturity. Furosemide was restarted at 31 weeks and continued through to her post partum. Spironolactone was restarted post partum. She also received a stat dose of vitamin K (10 mg intramuscular) to correct her deranged INR at 31 weeks and a repeat at 35 weeks.

Propranolol 40 mg once daily was reinitiated at 33 weeks, and continued through to the postpartum period.

OUTCOME AND FOLLOW-UP

The outcome was a live female infant whose birth weight was 2200 g and scored an APGAR score of 8 at 1 min and 9 at 5 min. Active management of third stage of labour was done with an estimated blood loss of 450 mL. The infant received hepatitis B vaccine at birth. The patient was clinically stable with no immediate postpartum complications. She received ciprofloxacin 500 mg twice a day for 7 days post delivery for prophylaxis against spontaneous bacterial peritonitis and was discharged home on furosemide 40 mg once a day, spironolactone 12.5 mg once a day and propranolol 40 mg once a day for 2 weeks. These drugs and doses were to be reviewed at her second postpartum week visit.

She had an uneventful postpartum period as noted in her second and sixth week postpartum follow-up visits and was referred to the physician for follow-up. Contraceptive advice was given including counselling against a future pregnancy. She opted for a copper intrauterine device. The baby's weight was appropriate at 4200 g at 6 weeks on exclusive breastfeeding.

DISCUSSION

The incidence of hepatitis B seroconversion is not known in the Sub-Saharan region. Boyd *et al*⁴ found that seroclearance rates are not common in patients from Sub-Saharan Africa treated with antiretrovirals. Chu and Liaw⁵ reported an annual incidence of HBsAg clearance of 1%–2% among Caucasian and Asian carriers.

Pregnancy predisposes a woman with liver cirrhosis and portal hypertension to a higher risk of oesophageal bleeding. Among those with varices, up to 78% may have gastrointestinal bleeding during pregnancy with an associated mortality of 18%–50%. In addition, these patients are at a higher risk of fetal loss.^{6,7} The risk of variceal bleeding is attributed to increased fluid retention and cardiac output in pregnancy, which is highest during second stage.³ It is for this reason that an assisted vacuum delivery in a controlled environment to shorten second stage is recommended.⁸

Thrombocytopenia in portal hypertension due to liver cirrhosis is as a result of the sequestration and destruction in the spleen.⁹ Our patient had thrombocytopenia but was asymptomatic and hence watchful waiting was considered appropriate. The recommended mode of delivery is vaginal with caesarean delivery under general anaesthesia reserved for obstetric indications.² Literature reports that these patients are at high risk of postpartum haemorrhage, which occurs in 7%–10% of cases.² This is attributable to a coagulopathy as a result of liver dysfunction and thrombocytopenia due to hypersplenism associated with portal hypertension or cirrhosis. In preparation for delivery, correction of coagulation status and preparation of the requisite blood products is recommended.^{2,3}

The routine management of portal hypertension needs to be considered alongside the potential risks of the medication and interventions on the fetus and pregnancy outcomes. Below we consider some of the common management strategies and look at the considerations required in pregnancy.

Endoscopies ideally should wait until the postpartum period unless there is a strong or life-threatening indication.⁷ If done in pregnancy, then it is safest to be done in the second trimester, as there is a risk of fetal loss in first trimester and inducing preterm labour in the third trimester.^{7,10} Our patient may have benefited from endoscopy to determine the size of her varices and maybe assessed her risk for a potentially fatal variceal bleed. There is a role for prophylactic variceal banding, which if necessary, would best be done in second trimester.⁷

Propranolol is a useful drug for prophylaxis against variceal bleeding. The benefit propranolol confers is to reduce portal pressures hence reduce the risk of a gastrointestinal bleed by 42%–43%.² However, its potential side effects include premature labour, fetal growth restriction, neonatal bradycardia, apnoea and hypoglycaemia. The risks of propranolol have to be carefully weighed against the benefits of using it before prescribing it to the patient. If used, proper fetal monitoring for bradycardia and fetal growth restriction ought to be instituted.

Diuretic use in pregnancy ought to be judicious as there is a risk of reducing the plasma volume with potential although

theoretical risk of decreasing placental perfusion. Its use has to outweigh the benefits. However, a meta-analysis on diuretic use (all diuretics) in pregnancy did not find an increased risk of adverse effects such as birth defects, fetal growth restriction, thrombocytopenia or diabetes among neonates exposed to diuretics in utero.¹¹ In the postpartum period, there is a risk of suppressing lactation with diuretics. The neonate ought to be followed up to ensure proper lactation and weight gain.

Learning points

- Despite limited information and challenges on the management of pregnant women with liver cirrhosis and portal hypertension, a multidisciplinary team can take care of such women with successful outcomes.
- The risks and benefits of all decisions and interventions have to be weighed, and decisions made in consultation with all parties involved, including the patient.
- The team has to constantly anticipate potential maternal and fetal complications (usually in an inpatient setting) and make necessary adjustments to ensure safety of both the mother and the baby.

Contributors FJL-M: contributed to the conception of the case report, collected and analysed the data, drafted the case report, gave final approval for the version to be published and agrees to be accountable for all aspects of the work. CMM: contributed to the conception of the case report, critically revised for important intellectual content and gave final approval for the version to be published. He also agrees to be accountable for all aspects of the work.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

© BMJ Publishing Group Ltd (unless otherwise stated in the text of the article) 2018. All rights reserved. No commercial use is permitted unless otherwise expressly granted.

REFERENCES

- 1 Shaheen AA, Myers RP. The outcomes of pregnancy in patients with cirrhosis: a population-based study. *Liver Int* 2010;30:275–83.
- 2 Aggarwal N, Negi N, Aggarwal A, *et al*. Pregnancy with portal hypertension. *J Clin Exp Hepatol* 2014;4:163–71.
- 3 Tan J, Surti B, Saab S. Pregnancy and cirrhosis. *Liver Transpl* 2008;14:1081–91.
- 4 Boyd A, Maylin S, Moh R, *et al*. Hepatitis B surface antigen quantification as a predictor of seroclearance during treatment in HIV-hepatitis B virus coinfecting patients from Sub-Saharan Africa. *J Gastroenterol Hepatol* 2016;31:634–44.
- 5 Chu CM, Liaw YF. Hepatitis B surface antigen seroclearance during chronic HBV infection. *Antivir Ther* 2010;15:133–43.
- 6 Tran TT, Ahn J, Reau NS. ACG clinical guideline: liver disease and pregnancy. *Am J Gastroenterol* 2016;111:176–94.
- 7 Savas N. Gastrointestinal endoscopy in pregnancy. *World J Gastroenterol* 2014;20:15241–52.
- 8 Westbrook RH, Dusheiko G, Williamson C. Pregnancy and liver disease. *J Hepatol* 2016;64:933–45.
- 9 Mitchell O, Feldman DM, Diakow M, *et al*. The pathophysiology of thrombocytopenia in chronic liver disease. *Hepat Med* 2016;8:39–50.
- 10 Cappell MS. Risks versus benefits of gastrointestinal endoscopy during pregnancy. *Nat Rev Gastroenterol Hepatol* 2011;8:610–34.
- 11 Collins R, Yusuf S, Peto R. Overview of randomised trials of diuretics in pregnancy. *Br Med J* 1985;290:17–23.

Copyright 2018 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