CASE REPORT

Placenta accreta complicated with peripartum cardiomyopathy

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SUMMARY

A 33-year-old G2P1 was referred to our hospital due to placenta accreta. During perioperative preparations, the patient was diagnosed with having a peripartum cardiomyopathy. The patient underwent caesarean hysterectomy at 36 weeks with an associated 2 L blood loss. Haemodynamic maintenance and stabilisation during the operation were challenging, with the combinations of fluid therapy, blood transfusions as well as vasoactive, antifibrinolytic and haemostatic drug. Postoperatively, the patient was managed in the intensive care unit and was subsequently transferred to intermediate care after less than 24 hours' observation. She was stable enough to be moved to the obstetrics ward the next day.

BACKGROUND

Placenta accreta is an abnormal invasion to myometrium. It has a 60% morbidity and 7% mortality rate due to its risk for massive bleeding.¹ In a national case study in UK, the incidence of placenta accreta was 1.7 in 10 000 in all maternities and 577 in 10 000 women had a previous caesarean delivery and placenta praevia.² Prior caesarean delivery, previous history of uterine curettage, placenta praevia, advanced maternal age, multiparity and recurrent pregnancy loss were some of the known risk factors in placenta accreta.^{3 4} Peripartum cardiomyopathy (PPCM) is defined as dilated heart in the last month of pregnancy until 5 months post partum, without any identifiable causes of heart failure, as well as the absence of any evidence of heart disease before the last month of pregnancy. The echocardiography will usually show a left ventricular dysfunction with ejection fraction <45%, fractional shortening <30% or both. The mortality rate for PPCM is 15%–30%.⁵ The combination of both placenta accreta and PPCM is very rare. It may be one of the most nightmare case scenarios that an obstetrician–gynaecologist has to face. Here, we present a 33-year-old parturient with a PPCM and a concomitant massive haemorrhage due to placenta accreta.

CASE PRESENTATION

A 33-year-old G2P1 at 36 weeks was referred from a small community health centre to our hospital due to suspected accreta. The primary obstetrician recommended to proceed with a delivery in tertiary hospital. She did not have any history of vaginal bleeding, nor did she have any systemic complaint

(dyspnoea, arterial hypertension) during pregnancy, which could have potentially limited her work as a nurse. Her previous obstetric history included a caesarean section due to macrosomia 3 years before. She was a non-smoker and took no medications. Her pre-pregnancy body mass index was normal and weight at admission was 83 kg.

INVESTIGATIONS

The obstetrical ultrasound revealed that the internal ostium was completely covered with placenta. The retroplacental hypoechoic zone disappeared and the placenta invaded the myometrium to the level of the uterine serosa. Lacunar vascular spaces were visible within the placenta. There was also thinning of the anterior lower uterine segment. All these signs pointed to an accreta syndrome. Caesarean hysterectomy was recommended because any traction on the placenta would possibly trigger a major obstetrical bleeding.

MRI is a standard diagnostic imaging procedure for placenta accreta; however, we do not perform it routinely in our country. During perioperative examination, diastolic murmur was heard in pulmonary and mitral valve. Blood pressure (BP) was 150/90 mm Hg without any sign of pre-eclampsia. ECG showed a normal sinus rhythm. Echocardiography showed left atrial and left ventricular dilatation, hypertrophy of interventricular septum as well as global hypokinesia. There were also moderate mitral regurgitation, mild pulmonic regurgitation and mild tricuspid regurgitation. Ejection fraction (EF) was only 45%, with fractional shortening of 24% (the values of end diastolic diameter and end systolic diameter were 58.2 mm and 44 mm). Tricuspid annular plane systolic excursion was 28.8 mm, indicating normal right ventricular systolic function. The cardiologist diagnosed class I congestive heart failure due to PPCM. The laboratory results showed haemoglobin of 10.7 g/dL, platelets of $296 \times 10^9/L$ and normal haemostatic profile (prothrombin time 9.4 s and activated partial thromboplastin time 31.2 s) as well as glucose levels. Proteinuria was absent. A multidisciplinary team meeting including obstetricians, gynaec-oncologists, anaesthetists and cardiologists was held. An elective caesarean section continued hysterectomy was proposed. At that time, embolisation or catheterisation by interventional radiologist for placenta accreta had not been established in our centre. We also asked for an urology back-up in case of bladder injury. Postoperatively, the patient was planned to be transferred straight to intensive care unit (ICU).

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Signs and symptoms of heart congestion would be managed in collaboration with anaesthesiologists and cardiologists using intra-aortic balloon pump (IABP) and extracorporeal membrane oxygenation (ECMO) as a backup. A diagnosis of gestational hypertension was considered as the cause of hypertension due to late onset of hypertension, with no signs of proteinuria or pre-eclamptic features.

DIFFERENTIAL DIAGNOSIS

Moderate mitral regurgitations, mild pulmonic regurgitation and mild tricuspid regurgitation can be found in late pregnancy without any symptoms. This can be a consequence of lower systemic vascular resistance during pregnancy. Raised blood pressure without proteinuria and sign of pre-eclamptic features at the near-term pregnancy is considered as gestational hypertension. However, a heart-failure-associated hypertension is a chronic process. Genetic testing and familial screening should exclude pre-existing or familial dilated cardiomyopathy. PPCM was an exclusion diagnosis after a thorough medical work-up.

TREATMENT

A classical caesarean section was performed, followed by caesarean hysterectomy preceded by ligation of bilateral hypogastric artery to reduce blood flow to the pelvic area. The placenta invaded the myometrium to the uterine serosa, thus there was placental tear during hysterectomy. Surgery lasted 4 hours, with an estimated blood loss of 2000 mL. There was a hypotensive period (due to blood loss) to 90/60 mm Hg at the middle of procedure. A total of 3000 mL of crystalloids and 1500 mL of colloids were given to ensure adequate intravascular volume during surgery. Dobutamine 3 µg/kg/min and norepinephrine 0.03–0.06 µg/kg/min were also used. As haemoglobin dropped to 5.8 g/dL, 920 mL packed red cells and 600 mL fresh frozen plasma were transfused intraoperatively. For further haemostatic control, 1 g of tranexamic acid and 500 mg of ethamsylate were administered. Cell salvage was not used, as it was unavailable in our facility. Operating room was kept at a temperature of 24°C. To maintain a normal body temperature, a warmer blanket was used and core body temperature was monitored. The patient was transferred to ICU in stable condition. Dopamine and norepinephrine were then continued for the next 5 hours. Tramadol 300 mg/24 hours was given for analgesia. Due to an increased arterial blood pressure of 160/110 mm Hg, we used a combination of ACE inhibitor and angiotensin receptor blocker. The laboratory results after surgery showed haemoglobin of 8.4 g/dL, platelets of $172 \times 10^9/L$ and normal haemostatic profile (prothrombin time 10.7 s and activated partial thromboplastin time 33 s) with clinically stable haemodynamics. The patient was transferred to intermediate care unit after 18 hours' observation in ICU and then moved to obstetric ward the next day.

OUTCOME AND FOLLOW-UP

Postoperative care was unremarkable. The patient was planned to have a repeat echo after delivery; however, due to insurance issues, echocardiography was performed in another hospital, where we were unable to access the echocardiography report. Currently, the patient is asymptomatic and has no complaints during day-to-day activities.

DISCUSSION

PPCM, with an incidence of 1 in 1300 to 4000 live births, contributes to 4% of all cardiomyopathies in pregnancy.^{6 7} PPCM is also responsible for 7% of maternal mortality and 36% of heart failures during delivery period.⁸ Maternal consequences

of dilated cardiomyopathy in pregnancy are heart failure (36%) and arrhythmia. The reported mortality rate in the USA is 0%–19%. Mortality increases with older age, multiparity, decreased myocardial function and delayed diagnosis.⁵

The pathophysiology of PPCM involves oxidative stress and cleavage of the prolactin molecule. The oxidative stress also promotes the release of matrix metalloproteinase and activation of cathepsin D. These substances convert the nursing hormone, 23 kDa prolactin, into 16 kDa prolactin, which will cause vasoconstriction, apoptosis, inflammation and dissociation of capillary structure. In the ventricle, it will cause a decrease in cardiomyocyte metabolism and a decrease in contractility and chamber dilatation.⁹ Based on the hypothesis above, antiprolactin agents such as bromocriptine, immunoglobulin and pentoxifylin were seen as promising treatments for PPCM. Although some cases were reported, the published evidence relating to those treatments refer to a small number of subjects. Further large-scale studies are needed for them to be recommended as the treatment for PPCM.^{5 10 11}

PPCM and EFs are known as independent risk factors for the outcome in PPCM. Subject with preserved EFs have a better outcome, while a decrease of EF below 40% increases mortality. If the EF is below 35%, the risk of thromboembolism increases and anticoagulation is needed. Prediction of cardiovascular outcome is dependent on left ventricular (LV) function measurements and also pre-existing cardiovascular symptoms. The outcome of asymptomatic heart failure present in majority of cases is still unknown. In case of symptomatic LV dysfunctions, 30-day cardiovascular morbidity is significantly increased.¹² The major challenge for the case above was how to maintain the optimal fluid balance, thus avoiding intraoperative myocardial depression. Most patients

Patient's perspective

Translated from Bahasa Indonesia

Before I performed the surgery, I was really worried about how it would be my life after this. I worried about the risks of placental invasion and heart disease I had. What if I didn't have any womb and I couldn't have another child. Both of my children were girl. A baby boy is very important in my ethnic. However, the team convinced me and my family that the most important at the time was my life. Now, I don't have any symptom of heart disease but sometimes I feel incomplete while another woman has regular menstruation but I don't. However, I still feel lucky because of what I already faced was dangerous moment but now I still can be with my family.

Learning points

- ▶ Peripartum cardiomyopathy and accreta syndrome are potentially life-threatening obstetric conditions that require a multidisciplinary approach to management.
- ▶ Guidelines for invasive placentation are effective once diagnosis is made, but diagnosis is sometimes difficult and relies on a high degree of alertness by obstetricians.
- ▶ Key point in management is to be prepared, to be aware of both conditions, to avoid cardiac depression due to hypovolaemia or anaemia, to avoid dilution of coagulation factors, acidosis and hypothermia, and to be able to perform some form of cardiac backup with the pre-emptive placement of intra-aortic balloon pump or extracorporeal membrane oxygenation catheters.

will remain stable and have normal LV functions 6 months after delivery. In the perioperative setting, the therapeutic escalation used in regard to failing inotropic support are mechanical support with IABP, ECMO or LV assist devices with or without bridging to cardiac transplant.^{9 12 13}

Elective caesarean section with caesarean hysterectomy at 36 weeks' gestation was chosen to prevent unplanned labour and emergency procedures that can cause higher maternal and neonatal morbidity.^{14–16} Hypogastric artery ligation was performed before caesarean hysterectomy to reduce arterial flow to the pelvis, as bilateral hypogastric artery ligation is responsible for an 88% decrease in pelvic pulse pressure.^{17 18} General anaesthesia was chosen due to the patient's risk of haemodynamic instability. Epidural anaesthesia could be performed with low threshold of converting to general anaesthesia if needed. An important aspect was the preparation of blood products and ICU for perioperative management.^{16 19 20} The haemorrhagic shock was corrected with fluid administrations, blood transfusions as well as inotropic and vasoactive agents.²¹ The unwanted side effects of iatrogenic hypervolaemia during massive fluid therapy have not been reported. However, in experimental studies, hypervolaemia might cause alveolar and endothelial injury.^{22 23} To decrease preload, diuretics can be used if there is evidence of pulmonary oedema and congestion. Intraoperative cell salvage may be considered in patients with placenta accreta. Nevertheless, cell salvage in obstetrics is more challenging due to amnion-mixed blood and the possibility of maternal alloimmunisation by the fetal blood. Recent trials on cell salvage revealed rare adverse outcomes in patients undergoing obstetric procedures.²⁴

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