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Placenta percreta complicated by uterine rupture and thrombotic microangiopathy

Dalia Laužikienė[1],

Saulius Vosylius[2],

Ieva Šiaudinytė[1]*,

Emilis Laužikas[1],

Diana Ramašauskaitė[1],

Daiva Bartkevičienė[1]

[1] *Department of Obstetrics and Gynaecology, Faculty of Medicine, Vilnius University, Vilnius, Lithuania*

[2] *Clinic of Anaesthesiology and Intensive Care, Faculty of Medicine, Vilnius University Vilnius, Lithuania*

Background. Uterine rupture at the site of a previous caesarean scar with abnormal placental penetration through the uterus wall with bladder invasion is a rare and serious pregnancy complication. Our aim was to report a case of uterine rupture with placenta percreta complicated by thrombotic microangiopathy.

Materials and methods. We did a literature review and analysed medical documentation retrospectively.

Results. A patient was admitted with complaints of lower abdominal pain at 21 weeks of gestation. Sonography of the caesarean scar increased suspicion of placental penetration. Anaemia, thrombocytopenia, coagulopathy, and acute kidney injury developed and led to the diagnosis of thrombotic microangiopathy. The termination of pregnancy was required due to severe deterioration in organ functions. The complete uterine rupture with placenta percreta invading the urinary bladder was confirmed, and total hysterectomy was performed to control life-threatening haemorrhage. The patient was treated by blood component transfusions, renal replacement therapy, and plasmapheresis. Good health was confirmed two months later by laboratory and instrumental tests.

Conclusions. It is a rare but very serious condition that increases morbidity of mother and foetus, therefore immediate diagnostics and treatment are required.

Keywords: placenta percreta, thrombotic microangiopathy, uterine rupture, acute kidney injury, blood component transfusion

Introduction

The growing frequency of caesarean deliveries leads to a dramatically increasing rate of morbidly adherent placenta. Prior multiple caesarean deliveries and placenta praevia are well-known risk factors of placenta percreta, which significantly increase the risk of life-threatening haemorrhage. Rarely, pregnancy is also an initiating event for thrombotic microangiopathy, causing haemolytic anaemia, thrombocytopenia, and severe organ system dysfunction. We describe a clinical case of uterine rupture at the site of a previous caesarean section scar and placenta percreta complicated

* Correspondence to: Ieva Šiaudinytė, Department of Obstetrics and Gynaecology, Faculty of Medicine, Vilnius University, Santariškių St. 2, Vilnius 08661, Lithuania. Email: jievai@yahoo.com

by neurologic abnormalities, acute kidney injury, coagulopathy, and massive bleeding with successful maternal outcome of the patient admitted to the hospital in the second trimester of pregnancy. It was a challenging clinical condition that required well-coordinated multidisciplinary teamwork leading to a favourable outcome. A pregnancy complicated by both placenta percreta and thrombotic microangiopathy has not been previously reported.

Case report

A 35-year-old woman, unaccompanied by anybody, was admitted to the hospital after loss of consciousness. She was confused and disorientated, and complained of abdominal pain. Bruises on her face and bite marks on the tongue were detected. The patient denied violence, use of alcohol, drugs, and any medication. She was at 21 weeks of gestation with a history of prior two vaginal and four caesarean deliveries, three surgical abortions, and uterine perforation during the last abortion. The vital signs were within normal range: heart rate 80 bpm, arterial blood pressure 120/79 mmHg, breath rate 14 bpm. The results of the blood test were as follows: white blood cells 21.2 × 10^9/l, hemoglobin 107 g/l, platelets 156 × 10^9/l, C-reactive protein 42.8 mg/l, creatinine 330 μmol/l. The urine test showed proteinuria, erythrocyturia, leucocyturia, and glycosuria.

Sonography of the caesarean scar increased the suspicion of a caesarean scar rupture and placental penetration with invasion into the bladder (Fig. 1). During cystoscopy no pathological changes were found, so placenta did not penetrate the bladder wall completely. Abdominal ultrasound showed enlarged kidneys with increased echogenicity with a small amount of fluid on the right side, increased arterial and venous blood flow. Echocardiography showed increased cardiac output, hyperkinetic left ventricular activity, and moderate dilatation of all cardiac chambers. Chest X-ray demonstrated congestive pulmonary lesions.

On the second day of hospital stay, the blood test showed anaemia (hemoglobin 82 g/l), thrombocytopenia (platelets 87 × 10^9/l), low fibrinogen (1.61 g/l), and increased fibrin degradation products (D-dimers 39880 μg/l), slightly prolonged activated partial thromboplastin time (APTT 46 s).

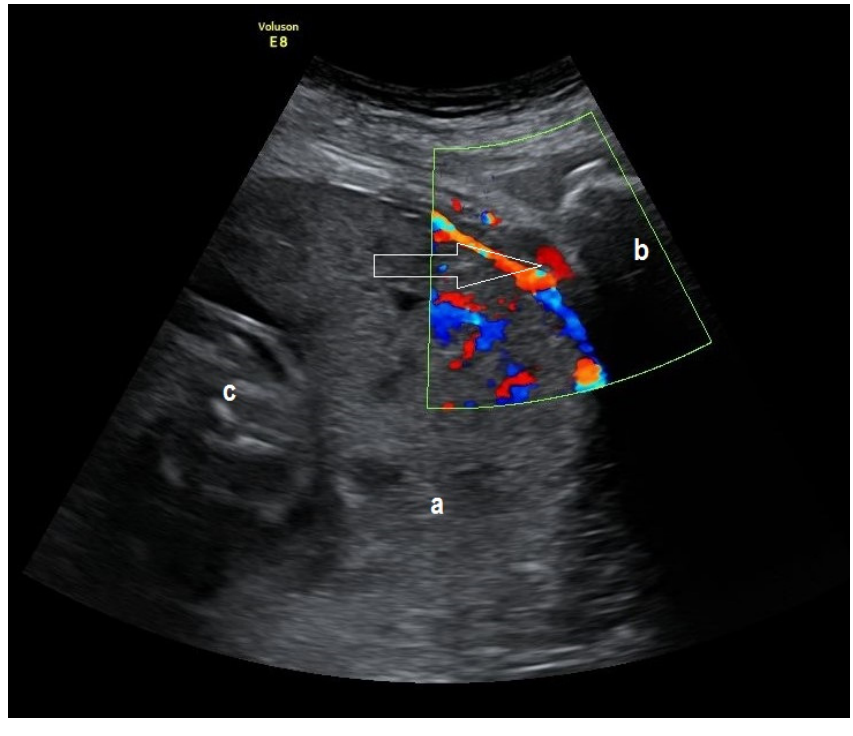

Fig. 1. Ultrasound examination showed features of placenta infiltrating the bladder base (arrow): thin retroplacental myometrial, irregularity of the uterine-bladder interface, and presence of blood flow on the bladder; a – placenta, b – bladder, c – foetus

Thrombotic microangiopathy was suspected due to the presence of renal, neurological, and blood test abnormalities. Reticulocyte count was 0.03%, schistocytes were not found, slightly elevated lactatodehydrogenase (496 U/L), and decreased haptoglobin (0.2 g/l). Direct and indirect Coombs reactions were negative. ADAMTS 13 activity and complement C3, C4 level were within the normal range. Treatment of thrombotic microangiopathy with steroids, fresh frozen plasma transfusion, and plasmapheresis was started.

On the third day, the patient's blood pressure rapidly dropped to 70/40 mmHg with sinus bradycardia (42 bpm). Anaemia, thrombocytopenia, and uraemia progressed, and anuria developed. The development of multiple organ dysfunction led to the decision to terminate the pregnancy by emergency laparotomy and caesarean section. Haemoperitoneum and anterior uterine wall defect in the caesarean scar were found. The broad-based placenta protruded through the uterine serosa completely and penetrated the full thickness of the lower uterine segment reaching the pelvic wall on the right side. The diagnosis of complete uterine rupture with placenta percreta invading the urinary bladder was confirmed. Due to abundant life-threatening haemorrhage, total hysterectomy was performed with right salpingo-oophorectomy and the infiltrated posterior bladder wall was repaired. The estimated total blood loss was 4000 ml; during the operation the patient was transfused six units of red blood cells and six units of fresh frozen plasma. After surgery, the circulatory shock and respiratory failure continued, so mechanical ventilation and norepinephrine infusion lasted for two days. Continuous renal replacement therapy with citrate anticoagulation was implemented and continued for nine days due to anuria, fluid overload, and uremia. Five procedures of plasmapheresis were performed until the platelet count rose to normal values. The pathological examination confirmed the diagnosis of placenta percreta (Fig. 2). The length of the hospital stay was 14 days. Two months later, the patient did not have any complaints: laboratory and instrumental tests showed normal renal function and confirmed uneventful recovery.

DISCUSSION

This case report presents an uncommon combination of a complete uterine rupture and placenta percreta with bladder invasion complicated by thrombotic microangiopathy in the second trimester of pregnancy. Placenta percreta is a rare form of morbidly adherent placenta with penetration through the myometrium reaching serosa and even adjacent pelvic organs. The incidence of abnormal placentation has been on the rise because of the increasing number of caesarean deliveries (1). The scarring process after surgery can cause abnormal vascularization, leading to the disruption of the decidualisation process and excessive invasion of trophoblast (2).

The patient had several risk factors, including former interventions (caesarean sections, surgical abortions, and uterine perforation), multiparity, older maternal age, and placenta praevia (3).

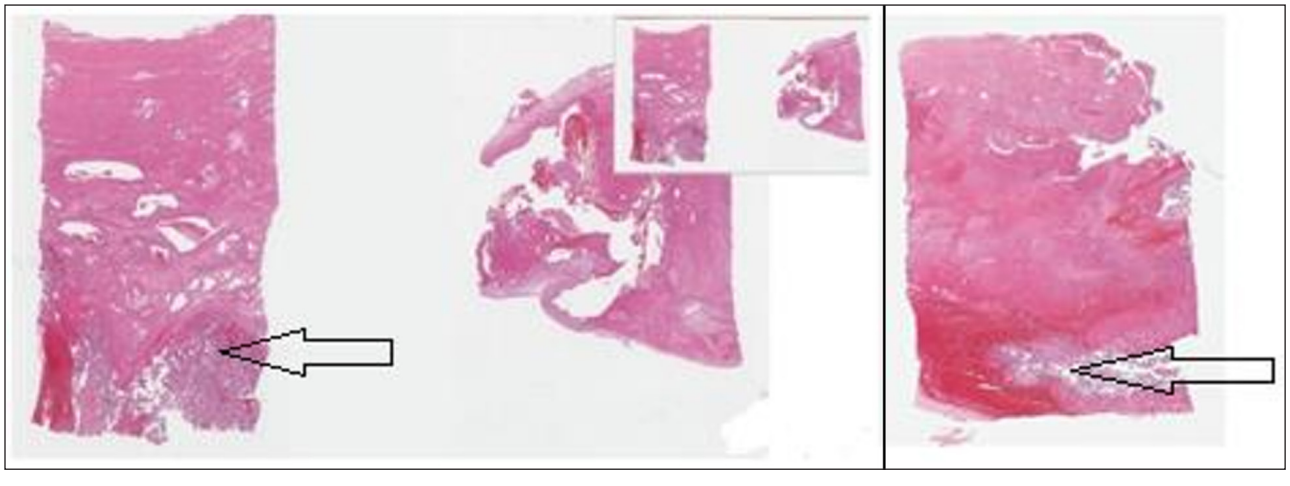

Fig. 2. Histological examination: chorionic villi invaded the myometrium of the uterus (arrows)

Hematuria, pain in the lower part of the abdomen, and the presence of risk factors led us to the suspicion of placenta percreta; however, these symptoms are not specific (4). Sonography of the caesarean scar showed placenta praevia located on the uterine scar area, thinning retroplacental myometrial zone, an unclear uterine-bladder interface, and blood flow (colour Doppler) on the bladder (5).

These findings confirmed the diagnosis of placenta percreta, which precluded the use of MRI. Although placenta percreta can be treated conservatively, the most common option is caesarean hysterectomy. The general condition of the patient and the organ failure deteriorated rapidly so the decision to terminate the pregnancy by urgent hysterectomy was chosen for better control of the obstetric haemorrhage.

In this case we suspected that pregnancy could be a precipitating factor for some type of thrombotic microangiopathy because of the presence of impaired consciousness, acute kidney injury, rapidly developing anaemia, thrombocytopenia, and coagulopathy. The manifestations of the thrombotic microangiopathy were not typical and the differential diagnosis was challenging due to non-specific clinical presentation and borderline changes in specific blood tests. It is recognized that pregnancy can trigger acquired thrombotic thrombocytopenic purpura or atypical haemolytic uremic syndrome (6). Thrombotic thrombocytopenic purpura is a rare condition in the second and third trimesters of pregnancy caused by an acquired or congenital lack of von Willebrand factor-cleaving protease (ADAMTS-13) (7). Autoantibodies to ADAMTS-13 induce vascular thrombosis and damage to endothelial cells. The clinical signs are anaemia, thrombocytopenia, neurological abnormalities, and less severe renal damage. The clinical features of pregnancy-related atypical haemolytic uremic syndrome are haemolytic anaemia, thrombocytopenia, and severe acute kidney injury requiring renal replacement therapy (8). It usually occurs postpartum and is determined by hyperactivity of the alternative complement pathway and regulators dysfunction resulting in endothelial damage and platelet activation (9).

We observed severe coagulopathy even before massive bleeding occurred during surgery that required blood component therapy and a postoperative support of vital functions in the intensive care unit. Abnormalities identified by the laboratory tests (fibrinogen, D-dimer, prothrombin time, and platelets count) did not allow confirming classical disseminated intravascular coagulopathy. As it was not directly related to the blood loss, possible subclinical course of uterine type amniotic fluid embolism was suspected (10). Substantial release of a large amount of inflammatory mediators, activation of coagulation cascade, consumption of coagulation factors, and activation of the systemic inflammatory response can occur due to the release of tissue factor to the maternal circulation following the separation of the membranes and the placenta.

CONCLUSIONS

This uncommon case was an extraordinary task to a multispecialty team, which led to a favourable outcome and complete recovery of the patient. Placenta percreta significantly increases the perinatal risk for both mother and foetus. Antepartum diagnosis of invasion of the placenta into the urinary bladder and related perioperative complications are challenging. This case confirmed that previous caesarean deliveries can cause uncommon perinatal complications, which could be associated with significant maternal and foetal morbidity and mortality.

CONFLICT OF INTEREST

The authors declare no conflicts of interest or financial relationships related to this study. The authors alone are responsible for the content and writing of the paper.

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Dalia Laužikienė, Saulius Vosylius, Ieva Šiaudinytė, Emilis Laužikas, Diana Ramašauskaitė, Daiva Bartkevičienė

PERAUGUSI PLACENTA, KOMPLIKUOTA SAVAIMINIO GIMDOS PLYŠIMO IR TROMBINĖS MIKROANGIOPATIJOS

Santrauka

Įvadas. Peraugusi placenta su invazija į šlapimo pūslę ir savaiminis gimdos plyšimas yra ypač reta ir grėsminga akušerinė patologija. Tikslas – pristatyti peraugusios placentos ir savaiminio gimdos plyšimo klinikinį atvejį, kurį komplikavo trombinė mikroangiopatija.

Metodai. Atlikta retrospektyvi medicininės dokumentacijos analizė ir literatūros apžvalga.

Rezultatai. 35 metų nėščioji atvyko į ligoninę dėl pilvo skausmų 21-ą nėštumo savaitę. Pacientės anamnezėje du savaiminiai gimdymai, keturios cezario pjūvio operacijos ir trys nėštumo nutraukimai bei viena gimdos perforacija. Ultragarsinio tyrimo metu įtarta peraugusi placenta buvusių cezario pjūvio operacijų rando srityje. Greitai besivystanti anemija, trombocitopenija, koagulopatija ir ūminis inkstų nepakankamumas leido įtarti trombinės mikroangiopatijos diagnozę. Progresuojant organų funkcijos nepakankamumui, nutarta skubiai užbaigti nėštumą – atlikti cezario pjūvio operaciją. Operacijos metu patvirtinta gimdos plyšimo ir peraugusios placentos su invazija į šlapimo pūslę diagnozė. Per operaciją prasidėjus gausiam kraujavimui atlikta totalinė histerektomija. Po operacijos skirtos kraujo komponentų transfuzijos, pakeičiamoji inkstų terapija bei plazmaferezės. Galutinis sveikatos būklės ir organų funkcijos atsistatymas buvo patvirtintas po dviejų mėnesių, atlikus laboratorinius ir instrumentinius tyrimus.

Išvados. Tai retos ir labai pavojingos akušerinės patologijos, itin didinančios moters bei vaisiaus sergamumo ir mirtingumo rodiklius. Ankstyva diagnostika, tinkamas, laiku parinktas gydymas ir darnus multidisciplininės komandos darbas yra būtini norint pasiekti gerus gydymo rezultatus.

Raktažodžiai: peraugusi placenta, trombinė mikroangiopatija, gimdos plyšimas, ūminis inkstų nepakankamumas, kraujo komponentų transfuzija