CASE REPORT

A rare case of placenta praevia percreta of an unscarred uterus in a systemic lupus erythematosus patient

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SUMMARY

Pregnancy in systemic lupus erythematosus (SLE) patients poses higher risks of adverse pregnancy outcomes. Placenta accreta spectrum (PAS) with various degrees of abnormally invasive placentation is associated with severe maternal morbidity and mortality. Pregnancy in SLE with coexisting PAS is indeed a double whammy. This was an unusual case of placenta previa percreta in a nulliparous with unscarred uterus and underlying SLE with grade IV lupus nephritis, in whom a placenta previa major with PAS suspected prenatally, sustained massive obstetric haemorrhage at delivery due to percreta, necessitated multiple blood transfusions and Caesarean hysterectomy. Adjunct MRI found suspicious of percreta. She has been on steroid for past 11 years, and her SLE remained quiescent throughout pregnancy. She underwent bilateral internal iliac artery balloon occlusion, bilateral retrograde pyelogram with ureteric stenting and Caesarean hysterectomy at 37 weeks. Intraoperatively noted bulging hypervascularized uterine lower segment with large vessels diffusely seen on uterine serosa, diffuse thinning of myometrium with areas appeared devoid of myometrium and large placenta occupying almost entire uterus. Histopathological analysis revealed percreta, with diffuse myometrial thinning especially at placental adherent site, and areas of placental invasion breaching uterine serosa.

This case emphasizes the significance of precise prenatal diagnosis of PAS for optimal perioperative preparation to minimise complications. PAS is indeed rare among nulliparous women and unusual on an unscarred uterus. However, preexisting SLE with prolonged steroid consumption may be a rare risk factor for PAS especially in placenta previa.

INTRODUCTION

Systemic lupus erythematosus (SLE) is a multisystemic autoimmune disorder with heterogeneous manifestations that is prevalent in females of reproductive age. Pregnant women with SLE are at higher risks of adverse pregnancy outcomes and complications, including severe preeclampsia, infections, thromboembolic complications, flares during pregnancy, utero-placental insufficiency, fetal growth restriction and even maternal or fetal mortality.

Placenta accreta spectrum (PAS) defined by various degrees of abnormally invasive placentation is associated with severe maternal morbidity and mortality, mainly reflecting massive maternal hemorrhage at delivery, the need for massive blood transfusion, complex surgery and increased intensive care unit admissions. Predisposing factors for PAS include multiparity, placenta praevia, smoking, previous PAS and previous uterine surgeries including endometrial curettage, Caesarean delivery and myomectomy. The increase in the overall rate of Caesarean deliveries with concurrent increase in maternal age at delivery, causes a rise in the incidence of PAS.

Prenatal sonographic findings associated with PAS, especially in placenta praevia cases include the loss of hypoechoic retroplacental sonolucency, myometrial thinning, interruption of utero-vesical plane, placental bulge into the bladder wall, hypervascularity of the utero-vesical interface, multiple placental lacunae ("moth-eaten" appearance of the placenta), hypervascularity of subplacental area, bridging vessels with vascularity extending from the placental bed across the uterine wall into the bladder or adjacent pelvic organs.

CASE PRESENTATION

This is an unusual case of placenta praevia percreta in a young nulliparous patient with unscarred uterus and underlying systemic lupus erythematosus (SLE) with grade IV lupus nephritis, in whom placenta praevia major with placenta accreta spectrum (PAS) suspected prenatally, sustained massive obstetric haemorrhage at delivery due to placenta accreta spectrum, necessitated multiple blood transfusions and Caesarean hysterectomy.

A 29-year-old, primigravida, with underlying SLE and diffuse proliferative lupus nephritis of grade IV since age 18, had a spontaneous conception after 2 years of marriage. She has been on steroid treatment for the past 11 years. Her last flare was at age 25. She was on Prednisolone, Hydroxychloroquine and Azathioprine. She was on low dose Aspirin in pregnancy, and her SLE remained quiescent throughout pregnancy. Her baseline 24-hour urine protein at 18 weeks was 140 mg/day. Laboratory value indices including full blood count, renal function and liver function were within normal limits. Modified oral glucose challenge test at 13 weeks and 28 weeks were normal. Antiphospholipid antibodies and extractable nuclear antigen antibodies were negative, whereas complements level C3 and C4 were normal.

Ultrasound at 22 weeks found anterior low-lying placenta with thin residual myometrium and multiple placental

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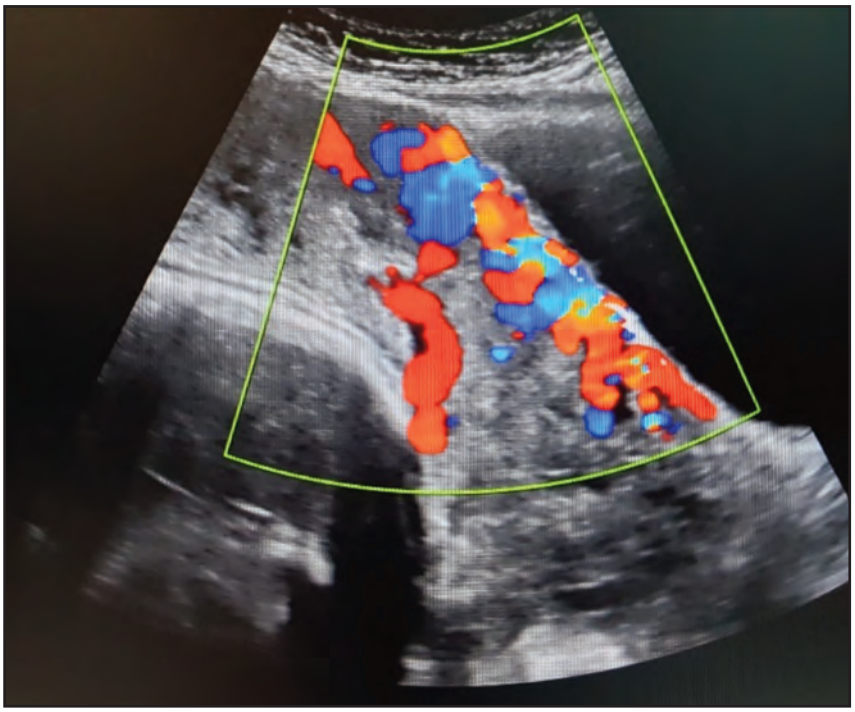

Fig. 1: USG Color Doppler image showing hypervascularity at utero-vesical interface

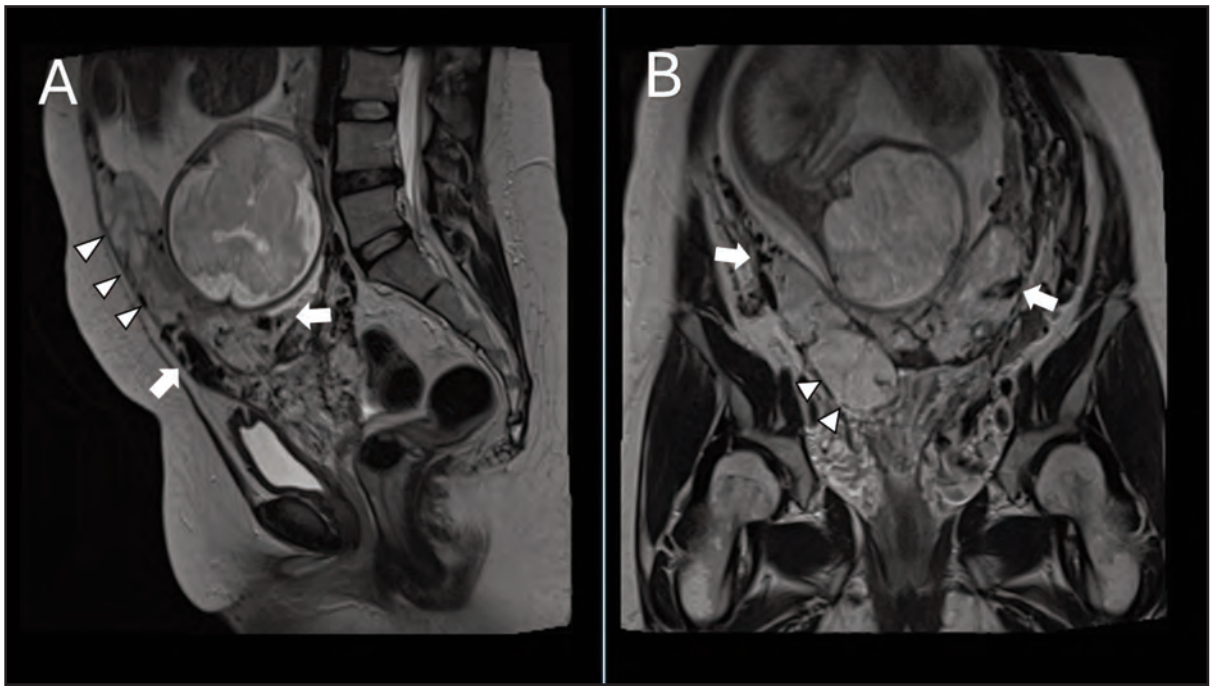

Fig. 2: MRI T2W Sagittal (A) and Coronal view (B): Placenta previa major with abnormal intra-placental vasculature (white arrow in "A" and "B"). Thinned out myometrium with loss of normal hypointense myometrial interface between the placenta and the myometrium (white arrowheads in "A"). Placental bulge at the right lower uterine segment (white arrowheads in "B")

lacunae. Fetus had large omphalocoele with liver content. Couple was not keen for amniocentesis for further evaluation of the fetal omphalocoele. She remained normotensive, whereas fetus was small for gestation, but liquor volume and fetal dopplers were within normal limits throughout pregnancy. Ultrasound at 29 weeks noted placenta praevia major covering internal os, with sonographic features of PAS. Adjunct magnetic resonance imaging (MRI) done to evaluate the extend of placental invasion, revealed loss of normal T2W low signal intensity between uterus and urinary

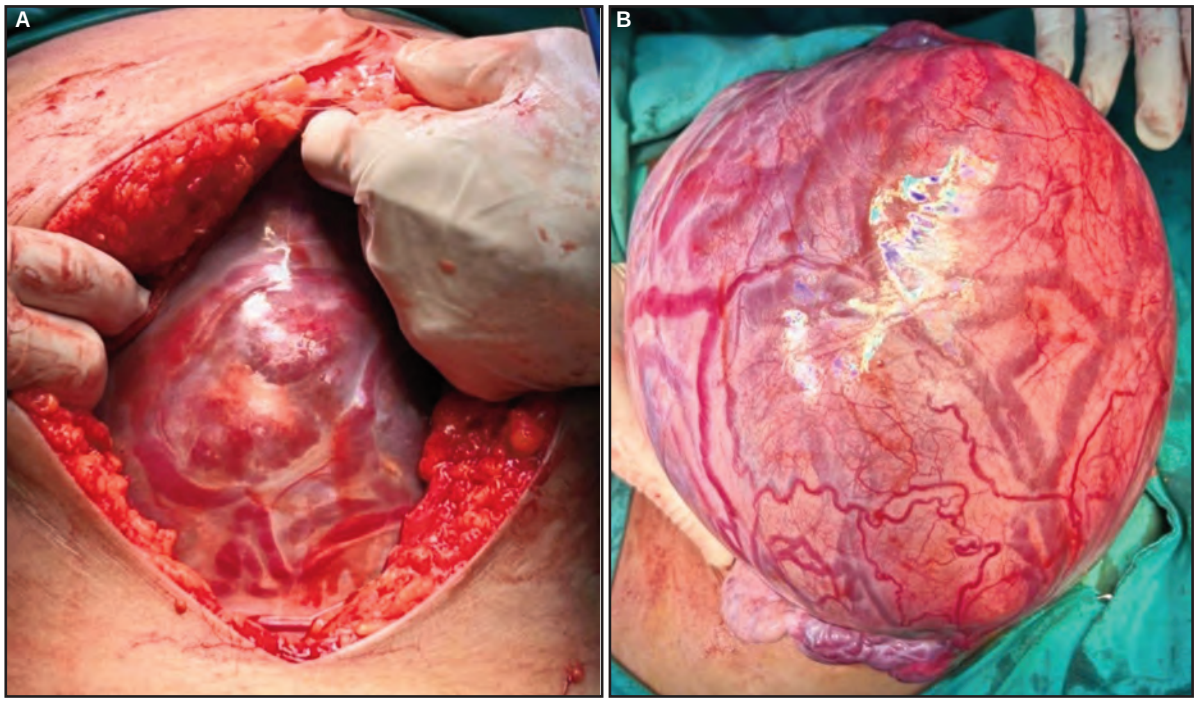

Fig. 3: Bulging hypervascularized anterior lower segment of the uterus (A). Gravid uterus with diffuse vascularization seen through uterine serosa and diffuse thinning of myometrium (B).

bladder, with tented bladder dome, suspicious of placenta praevia percreta.

She underwent bilateral internal iliac artery balloon occlusion, bilateral retrograde pyelogram with ureteric stenting and Caesarean hysterectomy at 37 weeks. Midline laparotomy with Classical uterine incision done followed by delivery of the baby. Intraoperatively noted bulging hypervascularized uterine lower segment with large vessels diffusely seen on uterine serosa, diffuse thinning of myometrium with areas appeared devoid of myometrium but with diffuse neovascularisation, and a large placenta occupying almost the entire anterior uterine wall. Massive obstetric haemorrhage due to placenta accreta spectrum with hypervascularisation and uterine atony complicated with approximately 8000 mL of blood loss, necessitated immediate multiple blood products replacement, of total 10 units of packed red blood cells, 8 units of fresh frozen plasma and 4 units of pooled platelet, and resort to total hysterectomy. Baby girl with birth weight of 2800 grams and major omphalocoele with liver and bowel content, required endotracheal intubation and NICU admission upon delivery. Postoperative recovery of mother was uneventful and she was discharged home on postpartum day 7. Histopathological analysis revealed placenta praevia major with percreta. Sections of uterus with placenta show focal areas displaying villi adhered directly to the thinned-out myometrium of less than 1 mm, without intervening decidua, and focal areas of placental uterine serosal breach and with presence of fibrin and blood.

DISCUSSION

Placenta accreta spectrum (PAS) commonly occurs among patients with previous cesarean scars, with the presence of placenta previa in the index pregnancy, and the likelihood of PAS increases proportionally with the number of previous cesarean deliveries. Other risk factors include increased maternal age, multiparity, other prior uterine surgery, prior uterine curettage, uterine irradiation, endometrial ablation, uterine synechiae, uterine leiomyomata, uterine anomalies, hypertensive disorders of pregnancy and smoking. Placenta accreta spectrum (PAS), formerly known as morbidly adherent placenta, refers to the range of pathologic adherence of the placenta, including placenta accreta, placenta increta and placenta percreta. The accepted hypothesis regarding the etiology of PAS is the defect of endometrial–myometrial interface leading to failure of normal decidualization especially in the area of a uterine scar, which facilitates abnormally deep placental anchoring villi and trophoblast infiltration.

Systemic lupus erythematosus (SLE) is an autoimmune disorder that can cause chronic inflammation, vasculitis, and diffuse thinning of the uterine myometrium, which may predispose patients to placental invasion even without prior cesarean or uterine scars. Emerging reports has shown SLE is linked to an elevated risk of abnormal placentation, including PAS.[1-5] This risk is heightened by long-term steroid therapy, which is hypothesized to cause muscle degeneration and diffuse uterine myometrial thinning. Furthermore, from the immunological perspective, autoimmune activity in SLE, can cause abnormal trophoblast invasion and poor placental anchoring. These conditions significantly increase the peril of severe obstetric hemorrhage, spontaneous uterine rupture, with maternal and neonatal morbidity and mortality.

This case illustrates the unusual complication of SLE, likely related to the peculiar condition of estrogen-mediated immune system, affecting uterine wall echo-structure.

Previous case reports hypothesized few theories including; chronic steroid exposure and SLE-induced antiestrogenic effect contributing to uterine wall thinning by inhibiting myometrial hypertrophy and endothelial proliferation, whereas complement cascade activation causes uterine microvasculature damage, and chronic inflammation contribute to endo-myometrial layer damage.[1-5]

Nevertheless the pathophysiology of the association between SLE and extensive PAS remains unclear, it is plausible that myometrial thinning resulting from chronic steroid exposure may contribute to this rare occurrence.

CONCLUSION

Pregnancy in SLE, which is a multisystemic autoimmune disorder with heterogeneous manifestations, poses higher risks of adverse pregnancy outcomes. PAS with various degrees of abnormally invasive placentation is associated with severe maternal morbidity and mortality.

SLE in pregnancy with coexisting PAS elicits great challenge in managing. Thus, a precise prenatal diagnosis of PAS is crucial for optimal perioperative optimization and preparation involving multidisciplinary approach to minimize complications to both, mother and baby.

PAS is rare among nulliparous women and unusual on an unscarred uterus. However, preexisting SLE with prolonged steroid consumption may be a rare risk factor for PAS especially in placenta previa, which necessitates a comprehensive prenatal evaluation and diagnosis.

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DECLARATION

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