CASE REPORT

Disseminated Intravascular Coagulation leading to Fetal Death in Utero and Uterine Infarction

Goh Yin Peng · Lau Kenneth K.

Received: 5 May 2010 / Accepted: 8 August 2011 / Published online: 6 September 2013
© Federation of Obstetric & Gynecological Societies of India 2013

Introduction

Uterine infarction is a rare condition. It is the necrosis of the uterus when the oxygen need exceeds its vascular supply or when there is a complete occlusion of the circulation to the uterus. There have been several non-iatrogenic causes of uterine infarction reported in the medical literature in the past, namely, carneous degeneration of large intracavitary leiomyoma [1], atherosclerosis of uterine vessels secondary to underlying medical conditions [2], uterine torsion [3], and postpartum incomplete uterine inversion [4]. Typically, histological examination demonstrates ulcerated endometrium [2] and extensive coagulative necrosis of the myometrium [4]. Iatrogenic causes of extensive uterine ischemic necrosis have been reported in uterine artery embolization in the management of leiomyomas [5, 6] and

postpartum hemorrhage [7]. We present an unusual case of disseminated intravascular coagulation secondary to gastroenteritis, leading to fetal death in utero, postpartum hemorrhage, and uterine infarction.

Case Report

A 26-year-old previously well Vietnamese primigravida woman presented with gastroenteritis at 35 weeks of gestation with symptoms of fever, nausea, vomiting, and diarrhea after eating unrefrigerated 4-day-old pork. The inflammatory markers, urine test and fetal monitoring at initial presentation were within normal range. Her condition rapidly deteriorated overnight and went into early labor on the following day. The cardiotocograph raised suspicion of fetal death in utero (FDIU), which was confirmed on ultrasound shortly afterward. Repeat blood tests 12 h later showed a platelet count of 89,000/ μ L, activated partial thromboplastin time (APTT) of 104 s, D-dimer of >70 mg/L and fibrinogen of 1.2 g/L. Blood film demonstrated marked neutrophilia with a left shift, severe thrombocytopenia, and fragmented red cells, but no evidence of helmet cells. The blood culture from admission subsequently grew multisensitive streptococcus pyogenes, which was treated with intravenous antibiotics accordingly. The fecal culture and urine culture were otherwise unremarkable. Above findings were consistent with sepsis complicated by disseminated intravascular coagulation (DIC).

Goh Y. P. (✉), Diagnostic Imaging Registrar
Department of Diagnostic Imaging, Monash Medical Centre,
Southern Health, 246, Clayton Road, Clayton,
VIC 3168, Australia
e-mail: yinpeng.goh@gmail.com;
yinpeng.goh@southernhealth.org.au

Lau K. K., Associate Professor
Department of Diagnostic Imaging, Monash Medical Centre,
Southern Health, Clayton, VIC, Australia
e-mail: ken.lau@southernhealth.org.au

Lau K. K., Associate Professor
Faculty of Medicine, Nursing and Health Sciences, Monash
University, Clayton, VIC, Australia

A stillborn female fetus was delivered by ventouse extraction. The delivery was immediately complicated by severe postpartum hemorrhage (PPH) with 5 L of estimated blood loss. She was then transferred to theater, and an intrauterine Bakri Balloon was inserted. Postoperatively, the patient was managed in the intensive care unit. Placental histopathology demonstrated normal vessel and membranes with otherwise unremarkable findings. The patient was given fresh frozen plasma and blood transfusion for the DIC.

The Bakri Balloon was removed 3 days later when the vaginal bleeding was minimal. Her platelet count was persistently low while the APTT was improving. On day 5 post delivery, she developed abdominal pain and fever with elevation of white cell count, and C-reactive protein. At the same time, she developed acute renal failure with a creatinine level of 394 $\mu\text{mol/L}$ and estimated glomerular filtration rate of 43 mL/min/1.73 m². Computed tomography scan was organized to look for a collection, but it demonstrated irregular gas densities within the myometrium of an enlarged postpartum uterus (Fig. 1). Subsequent ultrasound showed no arterial Doppler signal in the myometrium (Fig. 2). These findings were suggestive of uterine infarction. A laparotomy with total abdominal hysterectomy and bilateral salpingo-oophorectomy was then undertaken on the same day. The histopathology of the surgical specimen confirmed widespread arterial and venous thromboembolism within the uterus, cervix, ovaries, and fallopian tubes, associated with tissue infarction and hemorrhage. There were also widespread endometrial, myometrial infarction and stromal hemorrhage with some secondary fungal colonization noted. The right infundibulopelvic veins were also occluded.

Post-operatively, she developed a pelvic collection posterior to the urinary bladder. A surgical communication was made at the vaginal vault to allow drainage of the pelvic collection. The patient continued to improve on

Fig. 2 Pelvic ultrasound showed no Doppler signal within the uterine parenchyma. Blood flow was only detected outside the uterus (arrows)

intravenous antibiotics and was transferred to the renal unit for the management of acute renal failure with hemofiltration. On discharge, her renal functions were normal, and she was subsequently managed at the menopause clinic.

Discussion

In the setting of a thrombotic event associated with sepsis during pregnancy, the distinction between thrombotic thrombocytopenic purpura-hemolytic uremic syndrome (TTP-HUS), and DIC is important as this will alter the treatment modality accordingly. DIC is an inappropriate activation of the clotting system with deposition of thrombi and emboli in the microvasculature [8]. They can manifest as end-organ dysfunction or infarction in severe cases. Endotoxin induces the expression of tissue factor on the surface of monocytes and endothelial cells, which in turn

Fig. 1 Post contrast CT scan of the pelvis in axial plane (a) and sagittal plane (b) demonstrated irregular hypodensity in an enlarged postpartum uterus which contained multiple locules of air (arrows) in the myometrium

accelerates coagulation reactions. The consumptions of procoagulant and secondary fibrinolysis promote hemorrhage as a result of deficiency of coagulation factor and platelet depletion. TTP-HUS, on the other hand, results from primary platelet activation or primary endothelial injury that causes microangiopathic hemolytic anemia. Unlike DIC, it does not usually cause consumption of clotting factors [9].

Common laboratory findings of DIC and TTP-HUS include thrombocytopenia [9]. However, other laboratory markers, such as plasma clotting times, D-dimer, and fibrinogen can distinguish between the two. The laboratory findings of DIC include prolonged clotting time, high D-dimer, and low fibrinogen level. The reverse is true in TTP-HUS [9]. Fragmented red cells (schistocytes) can be seen in the blood films of patients with DIC or TTP-HUS; however, it is usually less marked in TTP-HUS. The presence of helmet cell fragments on blood film is also more characteristic of TTP-HUS [10].

In our patient, DIC is the more favorable diagnosis as this disease process is usually seen in the typical clinical setting of bacteraemia sepsis, whereas TTP-HUS is normally associated with enterotoxin produced by *Escherichia coli* [10]. Subsequent correlation with the biochemical markers and blood film confirmed the diagnosis of DIC in our patient.

The branches of the uterine and ovarian arteries form anastomoses that supply the ovary, fallopian tube, uterus, and cervix. The uterus becomes a highly vascularized organ during pregnancy to allow for adequate perfusion to the placenta for exchange of wastes and nutrients. Uterine infarction is extremely rare during peripartum period given its immense vascularity. In DIC, the circulating thrombin as a result of fibrin generation and platelet activation can lead to small vessel thrombosis in several organ systems, namely the kidney, lung, central nervous system, and skin. However, it is very unusual that in our patient, the reproductive system was the only organ system affected by this systemic process. This has resulted in the occlusion of the uterine and ovarian arterial branches, and the infundibulopelvic veins, confirmed on the histopathology of the surgical specimen. The PPH would be a consequence of the marked depletion of coagulation factors with DIC. At the same time, PPH by itself may also serve as a perpetuating factor that may worsen the DIC in this case.

Other reported etiologies of uterine infarctions in postpartum women include complications secondary to the management of PPH. These can result from the insertion of Bakri balloon, uterine artery embolization [7], placement of compression suture after the cesarean section [11, 12], and iatrogenic arterial ligation from multiple sutures to

achieve hemostasis during the cesarean delivery [13]. Based on our literature search, this is the first case report of DIC resulting in uterine infarction during the peripartum period.

Summary

This is a unique case report in the medical literature in which DIC is a causative factor for FDIU and subsequent uterine infarction. It is important to recognize early signs of sepsis in pregnant women to minimize this rare but important condition, which may lead to FDIU, PPH, and uterine infarction. Accurate diagnosis based on clinical history, laboratory results, and imaging findings is crucial in the management of this life-threatening disease and the recognition of its associated complications.

Conflict of Interest None

References

1. Johnson KF. Uterine infarction with endotoxic shock: a case report. *Am J Obstet Gynecol.* 1984;149(2):227–8.
2. Tan LK, Busmanis I. Spontaneous uterine perforation from uterine infarction: a rare case of acute abdomen. *Aust N Z J Obstet Gynaecol.* 2000;40(2):210–2.
3. Daw E, Saleh N. Massive infarction of the uterus and appendages caused by torsion. *Postgrad Med J.* 1980;56(654):297–8.
4. Romo MS, Grimes DA, Strassle PO. Infarction of the uterus from subacute incomplete inversion. *Am J Obstet Gynecol.* 1992;166(3):878–9.
5. Aungst M, Wilson M, Vournas K, et al. Necrotic leiomyoma and gram-negative sepsis eight weeks after uterine artery embolization. *Obstet Gynecol.* 2004;104(5 Pt 2):1161–4.
6. Godfrey CD, Zbella EA. Uterine necrosis after uterine artery embolization for leiomyoma. *Obstet Gynecol.* 2001;98:950–2.
7. Cottier JP, Fignon A, Tranquart F, et al. Uterine necrosis after arterial embolization for postpartum hemorrhage. *Obstet Gynecol.* 2002;100:1074–7.
8. Kasper D, Braunwald E, Fauci A, et al. *Harrison's principles of internal medicine.* 16th ed. USA: McGraw-Hill; 2004:683–685.
9. Rocha E, Paramo JA, Montes R, et al. Acute generalised, widespread bleeding. Diagnosis and management. *Haematologica.* 1998;83:1024–37.
10. Crowther MA, George JN. Thrombotic thrombocytopenic purpura: 2008 update. *Cleve Clin J Med.* 2008;75(5):369–75.
11. Gottlieb AG, Pandipati S, Davis KM, et al. Uterine necrosis: a complication of uterine compression sutures. *Obstet Gynecol.* 2008;112:429–31.
12. Reyftmann L, Nguyen A, Ristic V, et al. Partial uterine wall necrosis following Cho hemostatic sutures for the treatment of postpartum hemorrhage. *Gynecol Obstet Fertil.* 2009;37(6):579–82.
13. Pretorius ES, Siegelman ES, Tureck R, et al. Uterine necrosis after cesarean delivery seen on MR imaging. *AJR.* 1997;168(3):849–50.