Disseminated intravascular coagulation intrapartum following late intrauterine fetal death requiring hysterectomy

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CASE

A 36-year-old primiparous woman presented at 37 weeks gestation with significant reduction in fetal movements over the preceding 17 hours. Her antenatal period was otherwise uneventful, with protective serology, a low risk combined first trimester screen and normal morphology ultrasound. She had no past medical history or medication use of note. On examination, she was haemodynamically stable and abdomen was soft and non-tender. A clinician performed ultrasound revealed no fetal heart motion and formal obstetric ultrasound confirmed intra uterine fetal demise with no obvious radiological indication of cause.

Induction of labour with Misoprostol was commenced the same day, followed by an epidural block (EDB), artificial rupture of membranes with blood stained liquor, and commencement of a Syntocinon infusion. Four hours following EDB insertion, the patient reported dyspnea, pruritis and nausea. She was noted to have a blood pressure of 80/50 and frank haematuria, but otherwise stable with no blood on aspiration of the epidural catheter and anaesthesia to T11 bilaterally. She was managed effectively with cessation of the epidural infusion, intravenous metaraminol 0.5 mg and ondansetron. A vaginal examination at this time revealed full cervical dilatation and 1-hour of passive descent was commenced. On commencement of pushing, frank bleeding and haematoma formation were noted at the epidural site. A diagnosis of disseminated intravascular coagulopathy (DIC) was confirmed by blood sampled at this time: platelets 119 x 10^9/L, PT >120 seconds, INR >10, aPTT 107.5 seconds and fibrinogen <0.5 g/L. On transfer to the operating theatre for anticipated haemorrhage, a stillborn male infant was delivered vaginally, followed by active management of the third stage with significant blood loss following delivery of the placenta.

A general anaesthetic was administered, followed by the administration of several uterotonics including Syntocinon, Ergometrine and Carboprost. Multiple attempts of Bakri balloon insertion and vaginal packing were thwarted by ongoing massive haemorrhage. Simultaneously, the massive transfusion protocol was activated with total administration of 17 units of packed red blood cells, 8 units of fresh frozen plasma, 20 units of cryoprecipitate, 4 units of platelets and 2 doses of activated factor VIIa 7g. In the context of over 6 litres of blood loss and following liaison with haematologists, vascular surgeons and the obstetric and gynaecology team, the decision was made for a laparotomy and total abdominal hysterectomy.

She was admitted to the ICU for 3 days for stabilisation. The post-operative period was complicated by acute ischaemic tubular necrosis with a maximal Creatinine of 257 on day 7, pleural effusions, hyperphosphataemia and hyperkalaemia. She was appropriately fluid resuscitated with strict fluid balance and commenced on potassium binders and phosphate chelators, with subsequent resolution. Of the investigations into the cause of FDIU, the only finding of note was that of delayed villous maturation of the placenta.

DISCUSSION

The pathophysiology of DIC in the context of IUFD is not clearly understood. Classically, DIC is known to complicate prolonged cases of undiagnosed IUFD, with the literature reflecting that DIC is rare before 4 weeks of fetal demise (1). Other more common obstetric causes include placental abruption severe pre-eclampsia, amniotic fluid embolism, septic miscarriage and postpartum haemorrhage. The resultant release of tissue factor stimulates a cascade of cytokines from the endothelium, leading to activation of the coagulation pathway and eventual consumptive coagulopathy (2). Early recognition of the obstetric patient at risk of DIC and its clinical presentation is integral to stem the potentially devastating sequelae of this disorder.

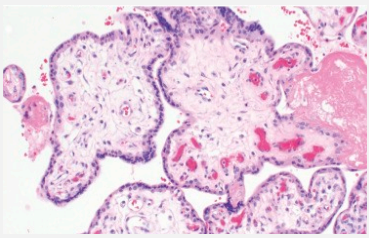

Image 1. Histology of delayed villous maturation with large, immature-appearing villi with few syncytial knots

In this case, the only noted pathology with potential contribution to IUFD was placental delayed villous maturation (image 1). It is characterized by decreased tertiary villus formation, reduced vasculosyncytial membrane formation, and increased large bullous villi. This is a clinically silent event with no prenatal or ultrasound markers and is associated with increased perinatal and early neonatal mortality of approximately 8% (3).

REFERENCES

1. Pritchard J. Haematological problems associated with delivery, placental abruption, retained dead fetus and amniotic fluid embolism. Clin Haematol. 1973;2(3):563-86.
2. Cunningham F, Leveno K, Bloom S, Spong CY, Dashe J. Williams obstetrics, 24e: Mcgraw-hill; 2014.
3. Higgins M, McAuliffe FM, Mooney EE. Clinical associations with a placental diagnosis of delayed villous maturation: a retrospective study. Pediatric and Developmental Pathology. 2011;14(4):273-9.