Disseminated Intravascular Coagulation: An Atypical Presentation of Amniotic Fluid Embolism

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Background

The reported incidence of amniotic fluid embolism (AFE) varies between 1 in 8 000 and 1 in 80 000 pregnancies and is recognised as a significant cause of maternal morbidity and mortality. The classically described picture is one of sudden respiratory distress, haemodynamic instability and confusion. We present an atypical case of AFE which presented without these classical features but as disseminated intravascular coagulation and culminating in a resulting post partum haemorrhage (PPH).

Case Demographics

Age: 28 years
Gravity: 2 Parity: 1 Gestation 39 + 0 weeks
Past Obstetric History: 1 x normal vaginal delivery, 2017 uneventful
Past Medical History: Fit and well
Drug History: No regular medications. NKDA
Family History: Nil significant. No bleeding or coagulation disorders.

Case Report

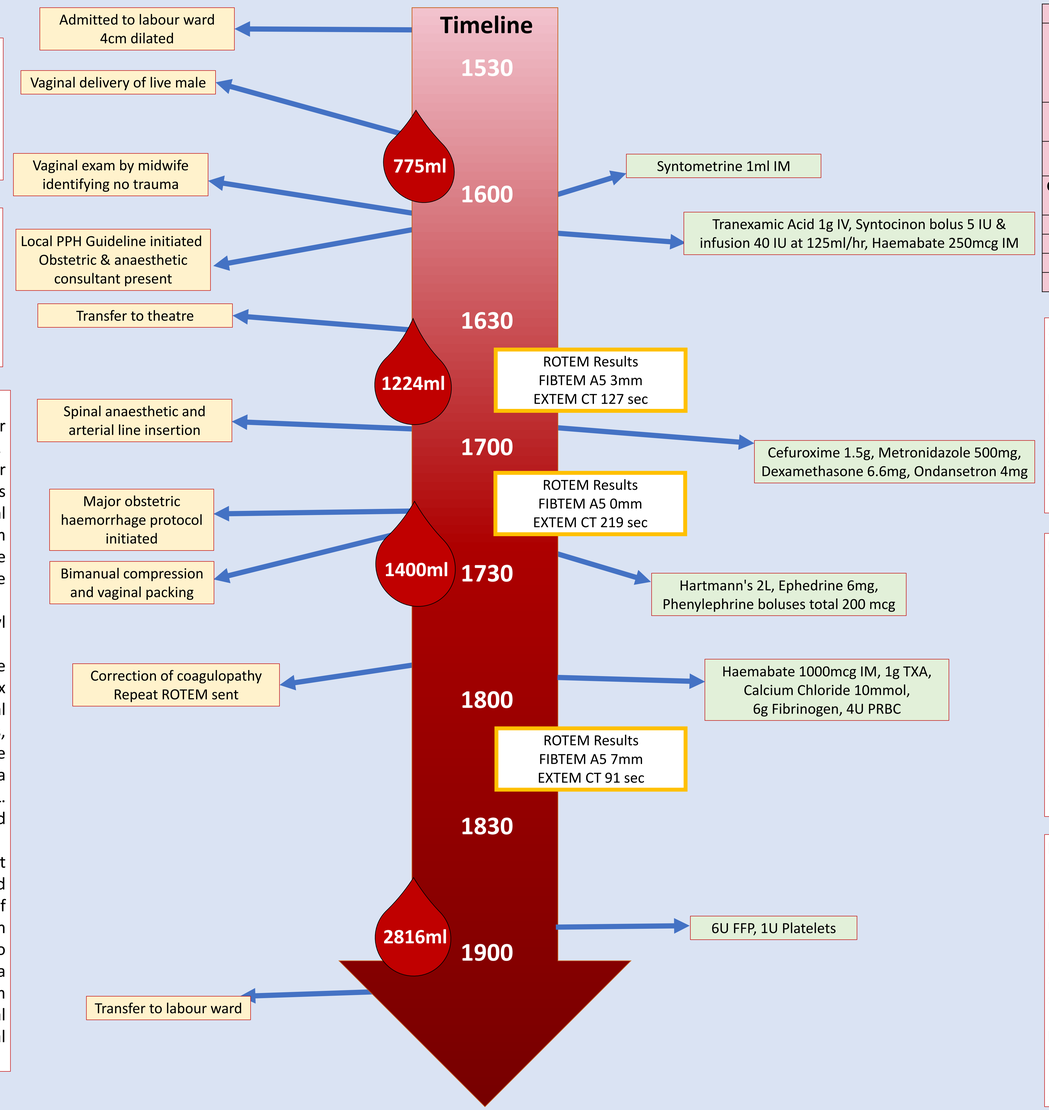
The patient attended at 39 weeks gestation for induction of labour following premature rupture of membranes. Her labour was precipitous progressing from 4cm dilation to vaginal delivery of a healthy live male after only 20 minutes.

She had an initial measured blood loss of 750ml and a post-partum haemorrhage was declared. Senior anaesthetic and obstetric teams were present and the all-Wales OBSCymru post-partum haemorrhage protocol was initiated. She received her first dose of IM syntometrine at this time. Within the first 30 min and at 1200ml measured blood loss, point-of-care venous blood gas and rotational thromboelastometry (ROTEM®, Tem International, Munich, Germany) were taken alongside routine blood tests. The patient was transferred to theatre and initial management included 1g of tranexamic acid, 40 I.U. oxytocin infusion, 250mcg Carboprost, and routine intravenous antibiotic therapy.

A spinal anaesthetic was subsequently performed with 2.5ml 0.5% heavy bupivicaine and 20mcg fentanyl achieving a sensory block T4 – S1 and dense motor block).

Two obstetric consultants performed vaginal examinations and found the vagina and perineum was intact, the uterine cavity was empty. The uterus did not appear to be atonic and there were no signs of abruption. The cervix was ragged and friable, with bleeding from multiple areas although no distinct trauma. At this point the initial ROTEM report taken at 1200ml blood loss became available revealing a FIBTEM A5 of 3mm and EXTEM CT of 127s, usually >10mm and < 85s respectively. This result was confirmed by repeat ROTEM at 2500ml blood loss and one hour post-delivery showing a FIBTEM A5 of 0mm and EXTEM CT of 219s. Formal lab results also showed a Haemoglobin of 116 g/L, platelets of 113 x 10^9/ L, prolonged PT of 19s APTT of 69s and fibrinogen of only 0.9 g/L. The D-dimer was extremely high measuring above the standard laboratory range of a maximum 20 000. She had otherwise normal renal and liver function.

Immediate haematology advice was sought whilst appropriate resuscitation continued. Her management involved ongoing uterotonics in the form of Carboprost and oxytocin as per protocol, bimanual compression and vaginal packing, as well as correction of her coagulopathy including four units of packed red blood cells, one gram of tranexamic acid, 10mmol of calcium chloride, and six grams of fibrinogen concentrate. Hospital stores of fibrinogen were exhausted, and haematology advised a further six units of fresh frozen plasma, and one unit of platelets to good effect. Remarkably despite an ultimate total measured blood loss of 2800ml she had only a mild tachycardia (heart rate 90 -110 beats per min), her blood pressure was well maintained throughout (blood pressure ranging from 135/70mmHg – 90/40mmHg), and she had no respiratory embarrassment (SpO2 98% on room air) or neurological deficit. She required a further two units of packed red blood cells but remained stable following vaginal pack removal at 24hrs.



	RESULT						
Time & Date Sample Collected	20/07/20 Pre admission	28/9/20 1630	28/9/20 2050	29/09/20 1020	30/9/20 0745	1/10/20	15/10/20
PT (sec) (12-15.5)		19.6	15.8	14.9	13.4	12.7	
APTT (sec)(25-36)		69.8	43.1	37.6	35.4	33.3	
Clauss fibrinogen g/L (2-4)		0.9	1.7	2.7	4.7	5.2	
WCC	6.0	10.4	19.3	18.5	16.3	12.2	5.4
Hb (g/L)	113	116	96	62	87	90	125
Plt	199	143	52	66	71	94	498
Ddimer			>20000				

Conclusion

The patient had a profound isolated coagulopathy at the outset with only 1200ml measured blood loss. Repeated obstetric examinations excluded retained tissue, uterine atony and trauma as sources of bleeding. Subsequent haematology follow up excluded congenital bleeding disorders with normal factor eight levels and Von Willebrand factor antigen. The extraordinarily high D-dimer and rapid evolution of her DIC suggests an atypical presentation of AFE without the classical features of cardiorespiratory insult.

Discussion

Whilst disordered coagulation is a sequelae of amniotic fluid embolism there are few reported incidences of an isolated immediate DIC, particularly without ever developing the accompanying traditional cardiovascular and neurological clinical signs. This case may suggest an under diagnosis of AFE as a cause for post-partum haemorrhage. Clinicians should be alerted to this atypical presentation as the rapidity of DIC appears to be out of keeping with the blood loss. Furthermore, the mainstay of treatment for these individual's rests on rapid replacement of clotting factors and fibrinogen as opposed to uterotonic therapies and obstetric interventions. Early point of care testing such as with ROTEM, particularly indicators of fibrinogen levels and D dimers may improve diagnosis of this rare condition and improve successful outcomes.

References

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