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Case Report: Amniotic fluid embolism, a description of coagulopathy

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Introduction

Coagulopathy is a marked feature of amniotic fluid embolus (AFE) yet the pathophysiology of the coagulation failure is not well described. It is widely reported to cause consumption of clotting factors, fibrinogen and platelets. However, there is little evidence in the literature of the level of individual clotting factors having been measured.[1, 2]

In this case report, AFE occurred at elective caesarean section. Sequential blood samples were taken during resuscitation and extended tests of coagulation were performed, measuring levels of clotting factors (CFs) VIII, II, IX, V, VII, X, XI, XII, thrombin generation, D-dimer and fibrinogen (table 1).

Case report

A woman with polyhydramnios and BMI 56 kg/m^2 had a fourth caesarean section under combined spinal epidural. During the difficult delivery of her baby, she reported sudden difficulty breathing followed by cardiac arrest. Return of spontaneous circulation occurred after one cycle of ALS guided resuscitation and intubation.

On suspicion of AFE, tranexamic acid 1g was given, then bloods taken. ROTEM showed hypofibrinogenaemia (Fibtem A5 of 7). She had early fibrinogen concentrate 6g and repeat ROTEM tests guided further management. In total she had fibrinogen concentrate 10g, blood 2 units, FFP 4 units and tranexamic acid 2g. Total blood loss was 2400mL.

She was transferred to ITU and bedside ECHO showed a dilated right ventricle evidencing an embolic event. She had no end-organ damage, was extubated and returned to delivery suite the following day. Bloods taken for routine coagulation screens were stored and retrospective consent taken for D-dimer, thrombin generation and clotting factors VIII, II, IX, V, VII, X, XI, XII, XIII measurements.

Key points

- Evidence of massive fibrinogen consumption and fibrinolysis
- No consumption of clotting factors

Hypothesis: Fibrinogen consumption is the initial pathology.
Early tranexamic acid and fibrinogen replacement halted the progression of consumptive coagulopathy

References

1. Metodiev, Y, Ramasamy P and Tuffnell D. Amniotic fluid embolism. BJA Education 2018; 18(8):234-238
2. Sultan P, Seligman K and Carvalho B. Amniotic fluid embolism: update and review. Curr Opin Anesthsiol 2016; 29:288-296
3. Schroder L, Hellmund A, Gembruch U et al. Amniotic fluid embolism associated coagulopathy: a single center obserational study. Arch Gynecol Obstet 2020; 301: 923-9.

Table 1: A summary of the patient's blood loss, products given and tests of coagulation

Sample time from event	+ 30mins	+55 mins	+110mins
Measured blood loss (mls)	1310	1620	2300
Products pre-sample	1gTXA	6g fibrinogen 1g TXA, 1 unit RBC	4g fibrinogen 4 FFP, 1 unit RBC
ROTEM	FIBTEM A5 7 EXTEM CT 91	FIBTEM A5 10 EXTEM CT 67	FIBTEM A5 13 EXTEM CT 58
PT (s)	13.4	14.0	13.4
APTT (s)	41.8	35	32.9
Fibrinogen (g/L)	1.6	2.1	2.4
Hb g/dL	115	101	104
Platelets (x10^9/L)	92	90	130
D-dimer (ng/ml) Normal range: 500-1000	282,110	267,955	220,610
Factor V (% of normal)	50%	50%	50%
Clotting factors VIII, II, IX, V, VII, X, XI, XII	Normal	Normal	Normal
Thrombin generation	High	High	High

Discussion

This case of AFE shows early fibrinogen deficiency with massively elevated D-dimers (normal 500-1000ng/mL) indicating hyperfibrinolysis. All other clotting factors were normal except Factor V, which although reduced, was adequate for haemostasis. Thrombin generation (a functional measure of haemostasis excluding fibrinogen) was high. The extreme elevation of D-dimer, with preserved clotting factors, in this case of AFE is almost unique. One other report measuring a limited number of clotting factors showed similar results.[3]

This case challenges the assumption that in AFE there is early widespread consumption of all clotting factors and suggests primary fibrinogen consumption and hyperfibrinolysis as the initial pathology. Early targeted administration of tranexamic acid and fibrinogen may halt the progression of coagulopathy and improve outcome.