

An Atypical Amniotic Fluid Embolism Presenting Primarily with Coagulopathy: A Case Report

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RANZCOG *Regional*
SYMPOSIUM
2026 *Swan Valley*

INTRODUCTION

Amniotic fluid embolism (AFE) is an uncommon obstetric emergency characterised by sudden maternal deterioration, typically involving cardiorespiratory compromise and coagulopathy. Disseminated intravascular coagulation (DIC) may be the primary presenting feature of an atypical AFE.

CASE

QP, a G3P1 at 40+6 with a low-risk antenatal pregnancy, underwent induction for post-dates. Admission vitals were normal. During cervical ripening, she developed acute instability following spontaneous rupture of membranes with thick meconium, becoming hypotensive (85/40) in the absence of tachycardia, with mild hypoxia (SpO₂ 91–94% on room air). CTG showed reduced-to-absent variability with complex variable decelerations. Examination revealed a firm abdomen, 3-cm dilation, and no vaginal bleeding. Terbutaline was given for hyperstimulation, with CTG improvement. She progressed rapidly to vaginal birth of a well neonate, immediately complicated by severe postpartum haemorrhage.

MANAGEMENT AND RESULTS

She developed brisk vaginal bleeding immediately post delivery with poor lower segment uterine tone, along with hypotension and hypoxia (SpO₂ 88%). Massive transfusion protocol was activated. QP received uterotonics, tranexamic acid, ephedrine, crystalloid IVT, O₂ via Hudson mask and was transferred to theatre for EUA and insertion of a uterine Bakri balloon. Estimated blood loss was 2 L; there was no retained products or cervical tear. Investigations showed severe coagulopathy with abnormal ROTEM, fibrinolysis, INR 2.7, APTT 84, thrombocytopenia (97), and D-dimer >80, requiring packed red cells and cryoprecipitate. She had transient hypoxia and oliguria, with biochemical end-organ hypoperfusion, improving with supportive care. Post-transfusion Hb was 82 with persistent thrombocytopenia, low fibrinogen, and elevated D-dimer. She recovered well.

Placental histology revealed high-grade chronic villitis and features of placental abruption.

DISCUSSION

Despite the absence of cardiorespiratory arrest, the early and marked severity of DIC, disproportionate to blood loss, raised concern for an atypical AFE-like syndrome, potentially precipitated by membrane rupture or placental abruption. Timely recognition, early fibrinogen replacement, and prompt mobilisation of a multidisciplinary team with activation of a massive transfusion protocol are critical to effective management.