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Hyperfibrinolysis diagnosed by rotational thromboelastometry in a case of suspected amniotic fluid embolism

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ABSTRACT
Rotational thromboelastometry is a viscoelastomeric, point-of-care method for testing haemostasis in whole blood which can be visualised rapidly, in real time, in the operating theatre. Advantages over traditional coagulation tests relate to the rapid feedback of results and the ability to visualise hyperfibrinolysis. We present a case of suspected amniotic fluid embolism that presented with sudden respiratory arrest associated with haemodynamic compromise during a non-elective caesarean delivery. Soon after the collapse, coagulopathy developed. Rotational thromboelastometry showed hyperfibrinolysis and hypofibrinogenaemia, which allowed targeted coagulation factor replacement therapy and the use of tranexamic acid. Hyperfibrinolysis may be a contributor to the coagulopathy associated with amniotic fluid embolism but has been infrequently reported, perhaps due to limited diagnosis with traditional coagulation tests. Treatment of the coagulopathy associated with a suspected amniotic fluid embolism with antifibrinolytic agents may deserve greater consideration.

Keywords: Amniotic fluid embolism; Antifibrinolytics; Tranexamic acid; Coagulation tests; Blood; Thromboelastometry

Introduction

Rotation thromboelastometry (ROTEM, Tem International, Munich, Germany) is a viscoelastometric method for monitoring the coagulation of whole blood. Although ROTEM and thromboelastography (TEG) have been used for several years in anaesthesia, particularly in cardiac, liver and trauma surgery,[1] it is not widely used in obstetric haemorrhage. Both ROTEM and TEG have advantages over traditional coagulation tests such as the international normalised ratio (INR) and the activated partial thromboplastin time (aPTT), particularly in speed of results and the potential to target blood component therapy in trauma and haemorrhage more accurately, which may reduce the use of blood products.[2,3] In view of the hypercoagulable state of pregnancy it has been recommended to use pregnancy-adjusted reference ranges.[4]

Amniotic fluid embolism (AFE) is a rare and potentially catastrophic complication of pregnancy associated with significant morbidity and mortality.[5,6] The diagnosis of AFE is generally one of exclusion associated with the acute onset of cardiorespiratory compromise (including hypotension, respiratory and/or cardiac arrest and pulmonary oedema) in conjunction with a coagulopathy.[7] The aetiology of the coagulopathy is not well understood but development is often rapid and may be severe, requiring significant volumes of blood and blood products.[5,6]

Our institution functions as a stand-alone tertiary referral women's hospital that recently introduced ROTEM coagulation monitoring primarily to assist with the management of complex obstetric haemorrhage.

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In this case report we describe the use of ROTEM in the management of a suspected episode of AFE. Patient permission for publication of the report has been provided.

Case report

A 34-year-old, G3P2 woman weighing 61 kg presented to our institution at 29 weeks of gestation with suspected preterm labour. She was previously well with no significant past medical history, taking no regular medications and reporting no allergies. The current pregnancy had been uneventful. On examination the cervix was found to be fully dilated, with active uterine contractions and the fetus in a transverse position with intact membranes. The potential for cord prolapse prompted an emergency caesarean delivery.

In the operating room, a single-shot spinal anaesthetic using 0.5% hyperbaric bupivacaine 2.5 mL, fentanyl 15 μg and preservative-free morphine 100 μg, was administered using a 27-gauge spinal needle with the patient sitting. Haemodynamic stability was maintained with a titrated metaraminol infusion (10 mg in 20 mL saline, infusion range between 10–30 mL/h). After a bilateral sensory block to ice to the T2 dermatome had been demonstrated, surgery commenced.

A classical uterine incision was performed and clear liquor was noted. Immediately before delivery the patient was alert and comfortable with normal vital signs: oxygen saturation (SpO_2) was 97% on room air, heart rate 80 beats/min and blood pressure 100/50 mmHg. A female neonate was delivered and an oxytocin bolus of 2 IU was given followed by an infusion of 7.5 IU/h. Approximately 2 min after delivery of the placenta, the patient complained of nausea and promptly became unresponsive and apnoeic. Bag and mask ventilation was started immediately, but her SpO2 fell rapidly to 60%. The trachea was intubated after administration of intravenous succinylcholine 100 mg; her SpO_2 rapidly improved. Her blood pressure was 70/40 mmHg with a heart rate of 80 beats/min; hypotension responded to metaraminol (0.5 mg bolus and infusion at 10 mg/h). A BIS monitor showed a reading of 70 before administration of sedative or general anaesthetic agents. Anaesthesia was maintained with sevoflurane in an air/oxygen mixture (end-tidal sevoflurane concentration between 1.2–1.4%).

Approximately 2 min after intubation she had two self-terminating episodes of ventricular tachycardia, the longer lasting approximately 10 s. A slow 5-g bolus of intravenous magnesium sulphate was given, defibrillation pads were applied, a radial arterial cannula was inserted and blood was sent for a full blood count, electrolytes, coagulation screen, ROTEM and blood gas analysis. The differential diagnosis consisted of a high spinal block, pulmonary embolism, air embolism or amniotic fluid embolism.

Over 10 min spontaneous ventilation resumed and ooze in the surgical field, consistent with the development of a coagulopathy, was noted. Preparations were made to obtain fresh frozen plasma and cryoprecipitate, and empirical treatment with tranexamic acid (1 g slow intravenous bolus) given. At this point the ROTEM trace became available. The initial EXTEM trace showed a clotting time (CT) of 69 s (normal <100), a clot amplitude at 10 min (A10) of 35 mm (normal >40) with the FIBTEM A10 also abnormal (5 mm, normal >10), suggesting fibrinogen replacement was required (Fig. 1). As the ROTEM trace progressed, severe hyperfibrinolysis was evident by 16 min on the EXTEM trace, demonstrated by the narrowing of the trace. The APTEM trace, which has aprotinin (an antifibrinolytic) added, was relatively normal suggesting that the appropriate therapy would be replacement of fibrinogen and inhibition of fibrinolysis. In consultation with the on-call haematologist 8 units of cryoprecipitate were given. Coagulation studies before the administration of tranexamic acid and cryoprecipitate are shown in Table 1.

After correction of the initial haemodynamic instability arterial blood gas analysis showed good gas exchange (PaO_2 29.7 kPa – FiO_2 65%, $PaCO_2$ 5.07 kPa, pH 7.30, base excess −7 mmol/L, lactate 2.4 mmol/L). Following haemostasis and completion of surgery, estimated blood loss was 1 L. The patient was woken and her trachea extubated in the operating theatre, after which no neurologic deficit was noted. She was transferred to a high-dependency unit, where she required a phenylephrine infusion for blood pressure support for 12 h. Repeat ROTEM analysis after the administration of tranexamic acid and cryoprecipitate showed resolution of the hyperfibrinolysis and hypofibrinogeneamia (Fig. 2). Although the FIBTEM A10 remained below the normal range, since there was no ongoing bleeding this was not further corrected. Repeat laboratory studies are shown in Table 1. After transfer to the postnatal ward the next morning, she made an uncomplicated recovery with no recollection of perioperative events following her complaint of nausea.

Discussion

In this case, monitoring coagulation with ROTEM rapidly identified hyperfibrinolysis and relative fibrinogen deficiency associated with maternal collapse. This aided the differential diagnosis since there are few causes of collapse associated with the rapid development of coagulopathy. In addition, the ROTEM findings were able to guide precise, targeted coagulation therapy, which may have reduced the need for additional blood products and their associated complication and cost.

The hyperfibrinolysis demonstrated by ROTEM is of particular interest since it has not been well documented in previous cases of suspected AFE, with only one re-