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Use of Thromboelastography in Early Detection of Coagulopathy in Amniotic Fluid Embolism

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ABSTRACT

Introduction: Amniotic fluid embolism (AFE) is a rare and life-threatening obstetrics emergency which relies on clinical diagnosis. In this case report, we have used thromboelastography in early detection of AFE and subsequent guidance for primary post-partum hemorrhage secondary to coagulopathy with successful outcome.

Case Presentation: In September 2021, a patient at 39 weeks of gestation presented with sudden cardiovascular collapse and cardiac arrest underwent emergency cesarean section. The case was complicated with primary postpartum hemorrhage. Thromboelastography was used as an early detection of coagulopathy in high suspicion of AFE. Patient was successfully resuscitated under TEG guidance and was discharged on post-operative day 11.

Conclusion: TEG is a useful tool in early detection for coagulopathy associated with AFE and serve as a guidance for intra-operative blood management, thereby reducing high morbidity and mortality associated with AFE.

Keywords: Thromboelastography; Amniotic fluid embolism; Coagulopathy; Primary postpartum hemorrhage; Intraoperative point of care testing

INTRODUCTION

Amniotic fluid embolism (AFE) is a rare but life-threatening pregnancy related complication with significant morbidity and mortality. The disease presents as sudden and unexplained maternal collapse with shock, hypoxaemia, and disseminated intravascular coagulopathy (DIC). The incidence of AFE has been reported as 5.5 per 100,000 maternities and case fatality of 24.8%. AFE was shown to be the fifth direct cause of maternal mortality in recent MBRRACE-UK report.[1] Whilst various risk factors for AFE, such as induction of labour and eclampsia, have been identified, recognition of AFE remains challenging due to its rare incidence.

Thromboelastography (TEG) 6S is a commonly used point-of-care viscoelastic test that assess coagulation status, and has been widely applicable in different clinical settings including trauma, critical care, cardiology and obstetrics. The test is useful in guiding intraoperative transfusion therapy due to its real-time and comprehensive analysis of clot dynamics.[2]

In this case report, we have used TEG-6S in early detection of coagulopathy in patient with AFE who developed sudden maternal collapse undergoing crash caesarean section. TEG-6S was also used as subsequent blood transfusion guidance. Coagulopathy was largely corrected intra-operatively and there were no significant complication or transfusion-related adverse event. This case report demonstrates TEG-6S as a useful tool for early recognition and guidance for management of AFE, therefore avoiding serious morbidity and mortality.

CASE PRESENTATION

Demographic detail and clinical history

Our patient is a 34 year-old Para-1 woman (body weight 65kg) undergoing crash caesarean section for fetal distress. The patient has no known medical history. Her antenatal history was unremarkable except mild anaemia with haemoglobin of 10.4 g/dL on iron sulphate. She was admitted to antenatal ward at 39 weeks of gestation for show and further transferred to labour ward for increased intermittent contraction. She was noted with confusion by midwife upon admission to labour ward and developed subsequent loss of consciousness. Blood pressure (BP) taken at the time was 250/165 mmHg, heart rate (HR) 111 beats/min, oxygen saturation (SpO2) 78% on room air, Glasgow Coma Scale (GCS) of 3 (eye 1, voice 1, movement 1). Patient was given 100% oxygen via non-rebreathing mask immediately. Shortly after, patient developed sudden cardiovascular collapse and maternal cardiac arrest in labour ward. Cardiopulmonary resuscitation (CPR) commenced immediately and rhythm on cardiac monitor was asystole. Patient was given 500 ml normal saline i.v. full rate and had return of spontaneous circulation (ROSC) after 2 minutes of CPR. No noted seizure or per vaginal bleeding. Crash caesarean section was performed for maternal cardiovascular collapse and fetal distress.

Peri-operative management

Upon arrival to operation theatre, BP was 139/109 mmHg with HR of 145 beats per minute. She was breathing spontaneously with SpO2 98% on room air. Pupils size 5mm bilaterally. Noted small amount of fresh blood from oral cavity and petechiae over right shoulder before induction of anaesthesia. Etomidate 10mg i.v. followed by suxamethonium 100 mg i.v. were given as rapid sequence induction. Patient was intubated with size 7.0 cuffed endotracheal tube via oral route. Operation started as soon as patient was intubated with paediatrician stand-by for neonatal resuscitation. Invasive monitoring including arterial line and central line were established simultaneously. A male baby was delivered by forceps 2 minutes after skin incision (23:38), with body weight 3.165 kg, and Apgar Score (AS) of 1 (1 min), 4 (5 min) and 7 (10 min).

Loading dose of magnesium sulphate 4 g i.v. was administered shortly after delivery in view of high blood pressure after ROSC and that possible eclamptic fit could not be ruled out. Carbetocin 100 mcg i.v. infusion and tranexamic acid 1 g i.v. were given immediately after delivery as well to prevent post-partum haemorrhage.

The first set of blood gas done 10 minutes after delivery showed haemoglobin (Hb) of 12.6 g/dL, with metabolic acidosis of pH 7.208 and base excess (BE) -12 mmol/L. Despite firm uterine tone, noted diffuse oozing from raw areas by obstetricians. Shortly after, Hb has dropped to 7.3 g/dL, where patient required increasing dose of inotrope for haemodynamic support. As such, 4 units of packed cell was transfused. The initial central venous

pressure (CVP) was noted to be 11-12 mmHg despite patient was in hypovolaemic shock. After initial fluid resuscitation, CVP has increased to 19-20 mmHg which might indicate presence of right heart strain.

Possible differential diagnosis for maternal collapse would include intracranial haemorrhage (ICH) secondary to ruptured aneurysm, peripartum cardiomyopathy, acute coronary events, massive pulmonary embolism or amniotic fluid embolism. Our team performed a TEG for coagulation assessment. First set of TEG-5000 at 00:36 showed prolonged R time >10 mins, decreased alpha-angle 14.9 degrees and markedly reduced maximal amplitude 23.3mm (Figure 1). Overall picture suggestive of clotting factors, platelets, and fibrinogen deficiency, compatible with a DIC picture. Transthoracic echocardiogram (TTE) was also performed to look for myocardial infarction or right heart strain for possible pulmonary embolism. Parasternal view of TTE showed fair contractility and hyperdynamic circulation, no pericardial effusion and right ventricle not distended, making massive pulmonary embolism less likely. Interim repeated haemocue has further dropped to 5.2 g/dL with estimated blood loss of 2850ml, which indicates primary postpartum hemorrhage. Uterine tone remained normal according to the obstetrician. Second dose of tranexamic acid 1g i.v. infusion and 4g of fibrinogen concentrate were administered. Given sudden maternal collapse pre-operatively, severe anaemia and unexplained coagulopathy, which was disproportional to the extend of bleeding, as shown on TEG, the most likely differential diagnosis would be AFE.

To correct severe anaemia and coagulopathy, on-going blood transfusion was guided by TEG-5000 and i-stat results. A total of 14 units of packed cells, 18 units of platelet, 10 units of fresh frozen plasma, and 8 units of cryoprecipitate were transfused. Prothrombin complex concentrate (Beriplex) 3500 units and further 1g of fibrinogen concentrate was given as per TEG results guidance. Operation theatre temperature was set to 24 degrees Celsius and forced-air warmer was used to avoid hypothermia. Total of 7g calcium chloride IV was given to correct for hypocalcemia secondary to blood transfusion. Clotting profile taken before start of cesarean section was available approximately 1 hour after delivery, with international normal ratio (INR) of 2.28, prolonged prothrombin time (PT) of 24.9 seconds and activated partial thromboplastin time (aPTT) of 77.2 seconds (Table. 1). The results largely corresponded to TEG-5000 results, suggestive of coagulopathy. Second TEG-5000 was performed to re-evaluate patient's coagulation status, and it shows normal R time (5.3 mins), K time (2.3 mins), alpha-angle (62.2 degrees), and MA (58.8 mm). The last set of point-of-care testing showed an Hb of 8.6 g/dL, pH of 7.43 with BE of -1 mmol/L. Anaemia and metabolic acidosis were grossly corrected. Hysterectomy was performed by obstetricians and haemostasis was achieved. No further blood transfusion was required. Total estimated blood loss was 6600 ml with all inotropes weaned off.

Patient Outcome

Patient was kept intubated to intensive care unit (ICU). Urgent CT Brain and CT pulmonary angiogram (CTPA) was performed post-operatively to rule out ICH and PE. CT Brain showed no gross intra-cranial pathology that accounts for maternal collapse. CTPA on post-operative day (POD) 0 showed no obvious clots in main pulmonary trunk, however the examination was suboptimal in view of images taken at early systemic arterial phase and presence of motion artefact. Bedside TTE in ICU showed thin rim of pericardial effusion of < 1cm without tamponade effect, Left ventricular ejection fraction of 60-65%, no regional wall motion abnormalities,

right ventricle not dilated with good contractility, and IVC around 1.6-1.7cm with 10% distensibility index. Coagulopathy was largely corrected (Table 1) with minimal tubal drain output. Patient was extubated on POD 1 to 2L oxygen via nasal cannula. Arterial blood gas showed satisfactory PaO2 without respiratory distress. However, repeated CTPA on POD 2 revealed pulmonary embolism in right lower lobar artery extending to segmental branches (Figure 2 a&b). As such, heparin infusion was started then converted to low-molecular-weight heparin. Patient had full neurological recovery and remains haemodynamically stable. She was discharged from ICU to general ward on POD 4. Patient remained stable with normalized clotting profile and was discharged on POD 11. Oral anticoagulant for pulmonary embolism was planned to continue for 6 months post-partum. Iron sulphate was also prescribed for mild anaemia. She remained well at post-natal follow-up 2 months after and her Hb returned to a normal level (12 g/dL).

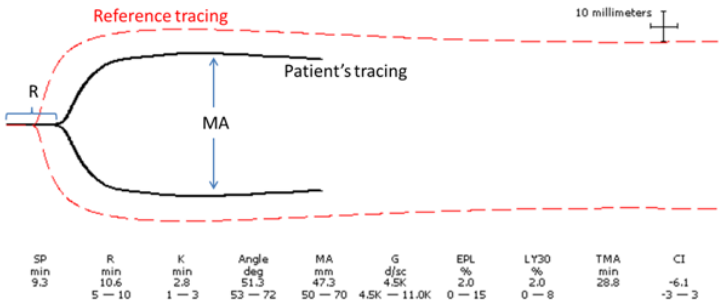

Figure 1: TEG tracing showing prolonged R Time, reduced alpha angle and maximum amplitude, suggestive of disseminated intravascular coagulation.

Table 1: Clotting profile before and after delivery.

	After delivery and post-resuscitation	Before delivery
PT (seconds)	13.1	24.9
INR	1.14	2.28
aPTT (seconds)	38.3	77.2
Fibrinogen (g/L)	1.9	

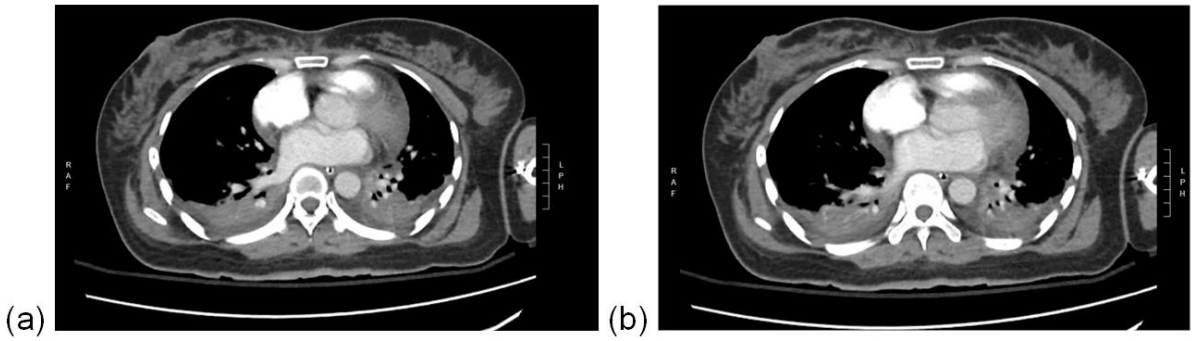

Figure 2 a&b: CT thorax showing small segmental pulmonary embolism at R lower lobe.

DISCUSSION

Amniotic fluid embolism (AFE) is a rare but serious obstetric emergency characterized by the sudden cardiovascular collapse, respiratory distress, and disseminated intravascular coagulation (DIC) during labour, delivery, or immediately postpartum. The pathophysiology of AFE is complex and not fully understood, but it involves several critical processes starting with the entry of amniotic fluid into maternal circulation. The maternal immune system reacts to foreign amniotic fluid components as allergens, leading to systemic inflammatory response. This is followed by cardiovascular collapse, through pulmonary vasoconstriction leading to acute pulmonary hypertension and right ventricular failure, myocardial depression leading to left ventricular failure, and subsequent hypotension. AFE often presents with acute respiratory distress due to pulmonary oedema, ventilation-perfusion mismatch resulting in hypoxaemia.[3]

Disseminated intravascular coagulation (DIC) is often a sequelae of AFE, resulting in thromboembolism as well as consumptive coagulopathy and fibrinolysis, leading to bleeding tendency. Patients with AFE typically present with rapid onset of shock and respiratory distress, seizure or cardiac arrest.Management of AFE is primarily supportive, including cardiopulmonary resuscitation, oxygenation and ventilation, haemodynamic support with inotropes and vasopressor, and managing DIC and haemorrhage.[1,3]

Diagnosis of AFE is challenging due to its sudden onset and non-specific symptoms. AFE diagnosis remains primarily clinical and its criteria, according to the AFE foundation, include sudden cardiac arrest or shock with respiratory compromise, absence of fever, overt DIC. Other symptoms include seizure or altered mental status. Onset of the above symptoms occur during labour or within 30 minutes postpartum. Important different diagnosis to exclude would be pulmonary embolism, anaphylaxis, sepsis or cardiogenic shock.

To-date, there is no single definitive test for AFE. Laboratory findings could support the diagnosis of AFE such as low platelet count, prolonged thrombin time and activated partial thromboplastin, low fibrinogen levels, elevated D-dimer indicative of DIC; blood gas showing hypoxaemia and metabolic acidosis. Additional diagnostic tool include transoesophageal echocardiography (TEE) which might be useful in detecting embolism in pulmonary arteries. Recently, there has been suggestion using blood detection of insulin-like growth factor binding protein 1 for early diagnosis of AFE; however the result showed a low sensitivity (16%), low positive and negative predictive value (58 and 50%), which suggested a questionable use of accurate diagnosis in AFE.[4] In fatal cases, post-mortem histological examination can confirm AFE by the presence of fetal squamous cells or other amniotic fluid components in the pulmonary vasculature.

Thromboelastography (TEG) is a diagnostic tool used to assess the haemostatic function of blood by measuring the viscoelastic properties of clot formation and dissolution. The key parameters in TEG include R Time (reaction time), K Time, Alpha-angle, Maximum Amplitude (MA), and LY30 (Lysis at 30 minutes).[5] R Time indicates coagulation factor function, prolonged R time indicated clotting factor deficiency. K time indicates time from initial fibrin formation till clot reaches a fixed strength, prolonged K time suggests fibrinogen deficiency or platelet dysfunction. Alpha-angle is the rate of clot formation, indicating fibrinogen level and function. Maximum amplitude measures the maximum clot strength and indicates platelet function or count. LY30 is the percentage of clot lysis in 30 minutes after reaching MA; high LY30 suggests hyperfibrinolysis.[5]

There are several advantages in using TEG in context of diagnosing and managing coagulopathy in AFE. Firstly, it allows rapid assessment of coagulation status, especially when coagulopathy can develop quickly and requires immediate correction in AFE. In contrast to standard coagulation test (i.e. PT, aPTT, INR) which provides only static snapshot, TEG measures clotting over time and offers dynamic and comprehensive picture of haemostasis. In addition, it offers an early detection of DIC. The identification of hyperfibrinolysis, which is common in AFE and contributes to severe bleeding, allows timely administration of fibrinogen and antifibrinolytic agents such as tranexamic acid. While TEG may not provide definitive diagnosis of AFE, the prompt detection of rapidly evolving coagulopathy in context of sudden cardiovascular collapse and respiratory distress, in our case maternal cardiac arrest, may support clinical diagnosis of AFE. It provides guidance on blood product therapy (e.g. platelet, cryoprecipitate, fresh frozen plasma) by assessing specific deficiencies in clot formation and strength. As such, TEG helps reducing unnecessary blood transfusion, and avoids transfusion-related complications when compared to employing massive transfusion protocol upon severe bleeding in AFE. As point of care testing, TEG also allows monitoring response to blood products, antifibrinolytics, or coagulation factor concentrates, allowing adjustment in therapy based on real-time coagulation status.[6-9]

While TEG-5000 detects abnormal coagulation, it offers limited specificity and sensitivity to diagnose AFE, such that there could be overlapping patterns with other conditions including sepsis-induced coagulopathy. Although TEG can provide information on platelet count through MA, it is not specific as other platelet function test such as platelet aggregometry.[9] In addition, the results are susceptible to interference and variability. Improper sample handling, storage and preparation could impact the results. Presence of air bubbles or clots in the sample could also lead to erroneous results.[2] Accuracy also relies on proper instrument calibration and maintenance. Last but not least, this is a single case report, therefore lacks statistical evidence and offers limited generalisability and validity. However, there were cases reported using TEG as a guidance in transfusion management in obstetrics patient developing primary postpartum haemorrhage due to AFE. We believed that using TEG could offer early clinical detection in AFE and allowing early correction of coagulopathy.

CONCLUSION

This case study described the presentation, early detection of coagulopathy and subsequent intra-operative management under TEG-5000 guidance in a case of AFE with sudden deterioration requiring emergency caesarean session complicated with PPH. TEG is particularly valuable in managing the coagulopathy associated with AFE, providing real-time, comprehensive insights into the patient's coagulation status. Therefore, TEG could serve as a useful tool in early clinical diagnosis and guidance for coagulopathy management in AFE, and potentially avoiding significant morbidities and mortality associated with AFE.

REFERENCES

1. Y Metodiev, P Ramasamy, D Tuffnell. Amniotic fluid embolism. BJA Education. 2018;18(8):234–238.
2. Hartmann J, Hermelin D, Levy JH. Viscoelastic testing: an illustrated review of technology and clinical applications. Res Pract Thromb Haemost. 2022;7(1):100031.

3. Tamura N, Farhana M, Oda T, Itoh H, Kanayama N. Amniotic fluid embolism: Pathophysiology from the perspective of pathology. J Obstet Gynaecol Res. 2017;43(4):627-632.
4. Bouvet L, Gariel C, Charvet A, Boisson-Gaudin C, Chassard D. Contribution of blood detection of insulin-like growth factor binding protein-1 for the diagnosis of amniotic-fluid embolism: a retrospective multicentre cohort study. BJOG. 202;128(12):1966-1973.
5. Hartmann J., Mason D., Achneck H. Thromboelastography (TEG) point-of-care diagnostic for hemostasis management. Point Care J Near Patient Test Technol. 2018;17:15–22.
6. Da Luz LT, Nascimento B, Shankarakutty AK, Rizoli S, Adhikari NK. Effect of thromboelastography (TEG®) and rotational thromboelastometry (ROTEM®) on diagnosis of coagulopathy, transfusion guidance and mortality in trauma: descriptive systematic review. Crit Care. 2014;18(5):518.
7. M J P Harnett, D L Hepner, S Datta, B S Kodali. Effect of amniotic fluid on coagulation and platelet function in pregnancy: an evaluation using thromboelastography. Anaesthesia . 2005;60(11):1068-72.
8. O Karlsson, A Jeppsson 2, M Hellgren. Major obstetric haemorrhage: monitoring with thromboelastography, laboratory analyses or both?. Int J Obstet Anesth . 2014;23(1):10-7.
9. Denis Snegovskikh, Dmitri Souza, Zachary Walton, Feng Dai, Rachel Rachler, Angelique Garay, et al. Point-of-care viscoelastic testing improves the outcome of pregnancies complicated by severe postpartum hemorrhage. J Clin Anesth. 2018;44:50-56.