

# Successful Resuscitation of Maternal Cardiac Arrest With Disseminated Intravascular Coagulation Guided by Rotational Thromboelastometry and Transesophageal Echocardiography: A Case Report

Hannah Brown, BMBS, FANZCA,\*† Helen L. Barrett, FRACP, PhD,\*†  
Julie Lee, BPharm, MBBS, FANZCA,\*† Jason M. Pincus, MBBS, FANZCA, FCICM,\*†  
Rebecca M. Kimble, MBBS, FRANZOG,\*† and Victoria A. Eley, MBBS, FANZCA, PhD\*†

We present a case of maternal cardiac arrest during an elective cesarean delivery. Transesophageal echocardiography identified a large pulmonary artery mass, and guided resuscitation efforts. After return of spontaneous circulation, the patient developed disseminated intravascular coagulation with massive hemorrhage. Blood product selection and volume replacement were guided by rotational thromboelastometry and transesophageal echocardiography, respectively. Correction of coagulopathy was observed clinically and confirmed by rotational thromboelastometry. The patient fully recovered without neurological deficit. (A&A Practice. 2018;10:139–43.)

In a healthy parturient, the main causes of cardiac arrest are pulmonary embolism (PE) (19%), hemorrhage (17%), pregnancy-induced hypertension (16%), and infection/sepsis (13%).<sup>1</sup> We present a maternal cardiac arrest caused by a massive PE, complicated by disseminated intravascular coagulation (DIC) at elective cesarean delivery. Point-of-care coagulation testing and intraoperative transesophageal echocardiography (TEE) aided successful resuscitation. The case report was reviewed with the patient and she provided written permission for its publication.

## CASE DESCRIPTION

A 45-year-old (gravidity 5, parity 2) woman with a body mass index of 24 kg/m<sup>2</sup> was admitted because of major placenta previa and antepartum hemorrhage. Apart from 2 previous uneventful spontaneous vaginal deliveries, she had no remarkable medical history. Ten days after admission at 37+1 weeks of gestation, she was scheduled for an elective lower segment cesarean delivery because of continued antepartum hemorrhage and a transverse lie.

The patient consented to a combined spinal-epidural (CSE) block. Two 16-gauge peripheral intravenous (IV) cannulas were placed, and electrocardiogram, noninvasive blood pressure, and oxygen saturation monitoring were established before the block. Initial blood pressure

was 120/80 mm Hg, heart rate 105/min, and peripheral oxygen saturations (SpO<sub>2</sub>) 96%. An uncomplicated CSE was performed with 11.5 mg 0.5% hyperbaric bupivacaine and 15 µg fentanyl. After the CSE, a sensory block to T4 bilaterally was confirmed with a Bromage score of 3. Blood pressure remained stable at 120/75 mm Hg supported by a phenylephrine infusion of 2 mg/h. Heart rate varied between 90 and 120/min and SpO<sub>2</sub> were 96%. After delivery of a live infant (Apgar score 9 at 1 and 5 minutes), the patient complained of nausea and dizziness, followed immediately by bradycardia of 35/min and loss of consciousness. Blood pressure was 70/35 mm Hg. Atropine 600 µg IV was administered to treat the bradycardia and succinylcholine 100 mg to facilitate immediate endotracheal intubation. After intubation, a carotid pulse was absent despite the electrocardiogram demonstrating a sinus rhythm of 100/min and pulseless electrical activity was diagnosed. As per the Australian Resuscitation Council guidelines, cardiopulmonary resuscitation commenced, epinephrine 1 mg was administered IV immediately and after every other pulse check. After endotracheal intubation, chest compressions with asynchronous ventilation were continued. Capnography confirmed endotracheal intubation and effective chest compressions. The lungs were ventilated with 100% oxygen in volume-controlled mode. The obstetricians delivered the placenta, exteriorized the uterus, and monitored effectiveness of thoracic compressions by continuous aortic palpation.

Return of spontaneous circulation (ROSC) occurred after 9 minutes. However, the patient suffered 2 further episodes of pulseless electrical activity (sinus rhythm, 140/min), each requiring chest compressions, lasting for approximately 5 minutes and occurring 10 minutes apart. A TEE performed during resuscitation demonstrated a dilated right ventricle (RV) with severely reduced systolic function. The left ventricle (LV) was underfilled with moderate globally reduced systolic function. At 30 minutes, there was relative hemodynamic stability supported by a noradrenaline infusion at 40 µg/min. A well-defined mass was identified in the right pulmonary artery during a detailed TEE examination (Figure 1).

From the \*Royal Brisbane and Women's Hospital, Brisbane, Queensland, Australia; and †The University of Queensland, St. Lucia, Queensland, Australia.

Accepted for publication August 24, 2017.

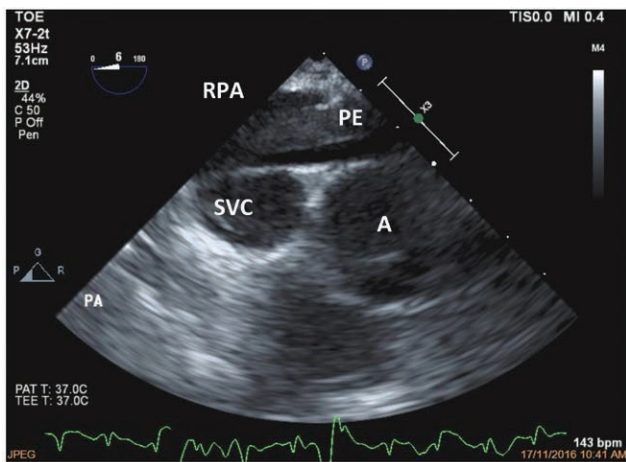
Funding: None.

The authors declare no conflicts of interest.

Address correspondence to Hannah Brown, BMBS, FANZCA, Anesthetic Department, The Royal Brisbane and Women's Hospital, Butterfield St and Bowen Bridge Rd, Herston, Brisbane, QLD 4029, Australia. Address e-mail to hanbrown\_uk@yahoo.co.uk.

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DOI: 10.1213/XAA.0000000000000662



**Figure 1.** Transesophageal echocardiography demonstrating a right pulmonary artery mass. Midesophageal ascending aortic short-axis view. A indicates aorta; PE, pulmonary embolus; RPA, right pulmonary artery; SVC, superior vena cava.

Two abdominal drains, a prophylactic Bakri intrauterine balloon, and vaginal packs were inserted, and the abdomen was closed. However, within 10 minutes of ROSC, bleeding from the vagina, oropharynx, and peripheral access sites occurred. A massive hemorrhage was declared and our institutional massive transfusion protocol was activated. Initial arterial blood gas analysis demonstrated a lactic acidosis and anemia (Table). Before performing rotational thromboelastometry (ROTEM), 2 units of packed red blood cells and 6 units of fresh frozen plasma were administered. In our institution, our ROTEM (Tem International, Munich, Germany) Delta testing module is located in the blood bank and the treating clinician interprets the results.

The initial fibrin formation and polymerization (FIBTEM A5; amplitude at 5 minutes) of 7 mm indicated significant hypofibrinogenemia (Table). The extrinsic coagulation pathway

(EXTEM) clotting time was within normal limits. Progression of the ROTEM trace showed narrowing at 12 minutes and the EXTEM and FIBTEM maximum lysis were >15%. A value of 100% of maximum clot firmness at <20 minutes demonstrated hyperfibrinolysis (Figure 2; Table). Corresponding laboratory blood investigations 30 minutes later confirmed hypofibrinogenemia (Table) and correlated with our suspicions of DIC.

### PULMONARY EMBOLISM

As per our critical bleeding ROTEM algorithm, EXTEM A10 of 23 mm (range, 43–65 mm) in combination with FIBTEM A10 <12 mm indicated the need for platelet and fibrinogen administration and maximum lysis 100% indicated the need for tranexamic acid. Following these results, the patient received 3 g fibrinogen concentrate, 2 units of platelets, 8 units of cryoprecipitate, and 1 g of tranexamic acid. Our institutional protocol did not call for the off-label use of recombinant activated factor VIIa.

Under TEE-guided volume resuscitation, 2 L of balanced electrolyte solution and 1.5 L of 4% albumin were administered. Two doses of 10 mL of 10% calcium gluconate were administered prophylactically during resuscitation.

An hour later, ROTEM showed improvement. However, reduced FIBTEM A5 of 6 mm indicated continued hypofibrinogenemia (Table). Guided by these results, another 3 g of fibrinogen concentrate was administered.

Two hours after the initial arrest, the uterus contracted, core temperature was 35.5°C, and hemodynamic stability maintained with a noradrenaline infusion at 17 µg/min and the patient was transferred to the intensive care unit. After consultation of cardiologists and cardiac surgeons, plans were made to transfer the patient to the nearby cardiothoracic center for potential extracorporeal membrane oxygenation and surgical pulmonary embolectomy.

A transthoracic echocardiography performed in the intensive care unit demonstrated improved LV contractility

**Table. Serial ROTEM, Coagulation, and Arterial Blood Gas Results After Maternal Cardiac Arrest**

	09.30 <sup>a</sup>	10.00 <sup>b</sup>	10.30 <sup>c</sup>	11.50 <sup>d</sup>	13.10 <sup>e</sup>	15.50 <sup>f</sup>
<b>ROTEM</b>						
FIBTEM A5 (range, 6–22 mm)	7	-	6	10	11	11
EXTEM A10 (range, 43–65 mm)	23	-	41	48	51	56
EXTEM CT (range, 38–79 s)	61	-	83	77	65	61
EXTEM ML (range, <15%)	100	-	0	0	0	0
<b>Coagulation</b>						
Platelets (range, 140–400 × 10 <sup>9</sup> /L)	-	83	-	164	-	212
INR (range, 0.9–1.2)	-	1.3	-	1.6	-	1.5
Fibrinogen (range, 2.0–4.5 g/L)	-	1.2	-	1.9	-	3.6
PT (range, 9–13 s)	-	13	-	16	-	16
aPTT (range, 24–39 s)	-	51	-	47	-	34
<b>Arterial blood gas</b>						
Hb (range, 115–161 g/L)	78	-	82	59	79	98
pH (range, 7.32–7.43)	7.26	-	7.18	7.28	7.36	7.45
Lactate (range, 0.5–2.2 mm/L)	14.3	-	4.8	2.6	3.3	2.1
Base excess (range, -2.0 to 3.0 mmol/L)	-14	-	-9.2	-6.4	-5.8	-2.8

Abbreviations: aPTT, activated partial thromboplastin time; CT, computerized tomography; EXTEM ML, extrinsic coagulation pathway maximum lysis; FIBTEM, fibrin formation and polymerization; Hb, hemoglobin; INR, international normalized ratio; PT, prothrombin time; ROTEM, rotational thromboelastometry.

<sup>a</sup>Time (h) following the return of spontaneous circulation and administration of 6 units of fresh frozen plasma.

<sup>b</sup>Time (h) following administration of 3g fibrinogen concentrate.

<sup>c</sup>Time (h) following the administration of further blood products, calcium, and tranexamic acid.

<sup>d</sup>Time (h) in the intensive care unit.

<sup>e</sup>Time (h) during hysterectomy.

<sup>f</sup>Time (h) post hysterectomy.

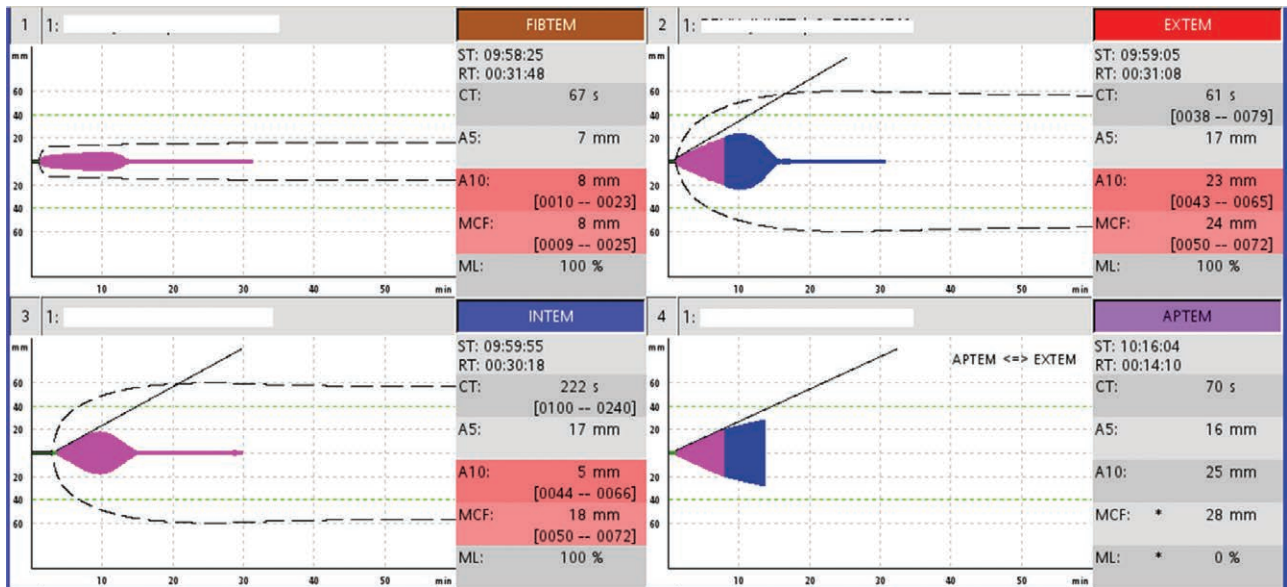


Figure 2. Initial rotational thromboelastometry result at the onset of bleeding.

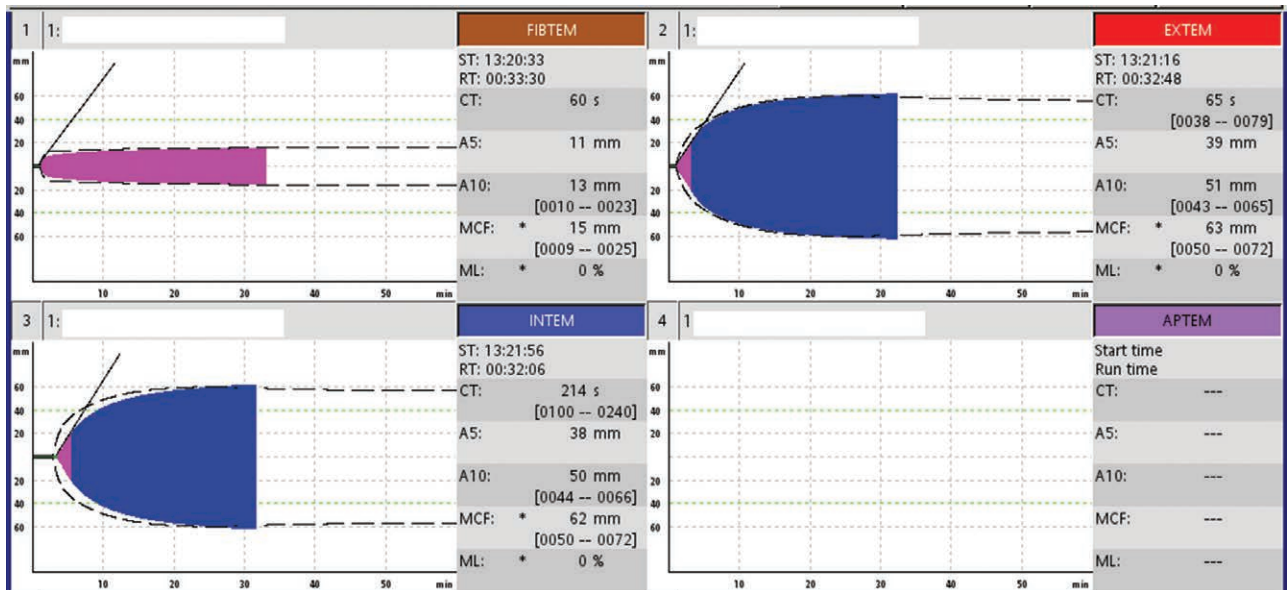


Figure 3. Rotational thromboelastometry results during emergency hysterectomy.

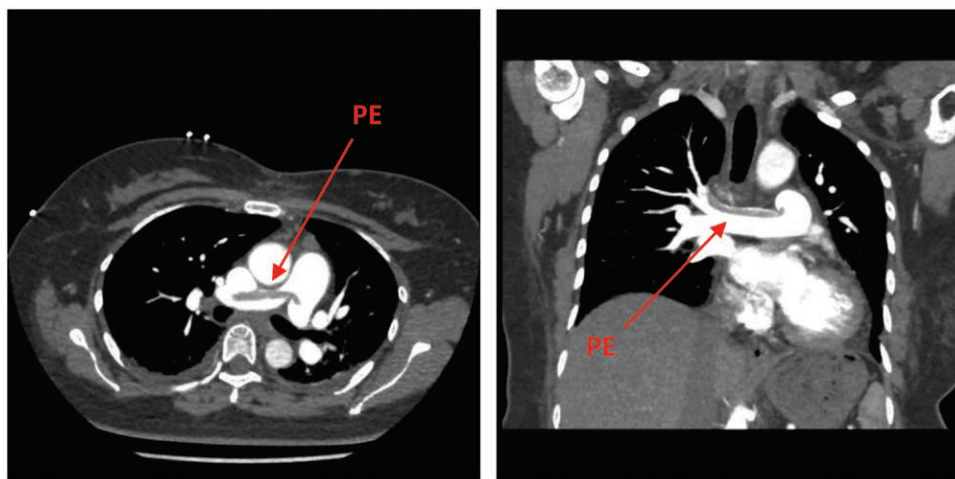
(ejection fraction 0.55–0.60), with a severely dilated RV and impaired systolic function. There was mild pulmonary hypertension (RV systolic pressure 32 mm Hg, range 12–16 mm Hg). Milrinone was commenced to support RV contractility and decrease pulmonary vascular resistance and vasopressin to support systemic vasoconstriction. Further ROTEM results revealed improvements in the coagulopathy (Table); however, there was ongoing uterine bleeding with a further decrease in hemoglobin to 59 g/L (range, 115–161 g/L; Table). Because of the continuing uterine bleeding, anemia, and the planned transfer of the patient to a hospital without obstetric facilities, the patient underwent a hysterectomy. This occurred 4 hours after the initial arrest with further 4 units of packed red blood cells being administered. No further coagulation correction was required,

because ROTEM had normalized (Figure 3; Table) and clinically the coagulopathy had resolved.

Following hysterectomy, formal laboratory tests confirmed a resolved coagulopathy with stabilized hemoglobin concentration (Table) and the interhospital transfer occurred. Further interventions were not necessary because the patient stabilized and was extubated the following day.

A computerized tomography pulmonary angiogram (Figure 4) performed following extubation demonstrated a saddle embolus extending into the segmental branches of the upper and middle lobes. Ultrasound of the lower limbs excluded deep venous thrombosis. Abdominal ultrasound investigation suggested a possible right ovarian thrombosis.

The patient was discharged home 3 weeks after the cesarean delivery for continued outpatient care. A repeat



**Figure 4.** Computerized tomography pulmonary angiogram demonstrating a large saddle pulmonary embolus (PE). Axial and sagittal views.

transthoracic echocardiography demonstrated a normal LV size with low normal systolic function, ejection fraction 0.54, and a normal RV with low normal systolic function. The patient had no significant neurophysiological deficits and formal neuropsychology review revealed intact cognition. She continued to be on anticoagulation treatment under the guidance of the hematologists.

## DISCUSSION

Thrombosis and thromboembolism remains the leading cause of direct maternal death (11% maternal mortality).<sup>1,2</sup> Massive pulmonary thromboembolism constitutes 10% of all presentations of venous thromboembolism.<sup>3</sup>

Sudden maternal cardiopulmonary collapse followed by coagulopathy is often attributed to amniotic fluid embolism.<sup>4</sup> TEE findings after ROSC suggested a massive PE, confirmed in the subsequent computerized tomography pulmonary angiogram. We hypothesize that a thrombosis of the proximal veins in the pelvis was dislodged at the time of delivery of the baby.

This patient had multiple antepartum risk factors for thromboembolism, including advanced maternal age,<sup>5,6</sup> parity,<sup>5,6</sup> antepartum hemorrhage,<sup>6</sup> and hospitalization for non-delivery reasons for >3 days.<sup>7</sup> Prophylactic anticoagulation was withheld due to ongoing antepartum hemorrhage, known placenta previa, and the potential for emergent delivery.

DIC after massive PE has been reported in both obstetric and nonobstetric patients.<sup>8–10</sup> Up to 9% of patients who suffer a cardiopulmonary arrest secondary to a PE fulfill overt DIC criteria.<sup>11</sup> It is proposed that the presence of a large PE results in insufficiently contained thrombin generation at the clot surface causing an overflow of active coagulation factors into the systemic circulation. Hyperfibrinolysis has also been demonstrated using ROTEM after presumed amniotic fluid embolism.<sup>12</sup>

ROTEM is a point-of-care test of coagulation, used in trauma, maternity, hepatic, and cardiac surgery.<sup>13</sup> It rapidly analyzes the entire process of coagulation, facilitating the targeted correction of coagulopathy. The standard panel comprises EXTEM, intrinsic coagulation pathway, and FIBTEM.

Using TEE during resuscitation facilitated the early diagnosis of the pulmonary arterial mass and guided

fluid management. Coupled with ROTEM to select blood products, we avoided overtransfusion and RV strain.<sup>13–15</sup> Early detection and aggressive correction of the hypofibrinogenemia was key to the successful management of the coagulopathy.

Obtaining a formal TEE during stabilization of the patient provided vital diagnostic information. Continuous consultation between our intensivists and the cardiothoracic center permitted patient transfer as soon as it was feasible.

This is the first published case report of maternal cardiac arrest with DIC successfully managed using ROTEM and TEE. This patient had a definitive diagnosis of a massive PE causing cardiopulmonary collapse with DIC during an elective cesarean delivery. Effective interdisciplinary communication was essential in our tertiary hospital, particularly because of the lack of an on-site cardiac surgery service. The use of TEE and ROTEM throughout resuscitation allowed targeted therapy and may well have contributed to the positive outcome. ■

## DISCLOSURES

**Name:** Hannah Brown, BMBS, FANZCA.

**Contribution:** This author helped care for the patient and write the manuscript.

**Name:** Helen L. Barrett, FRACP, PhD.

**Contribution:** This author helped provide expert opinion and edit the manuscript.

**Name:** Julie Lee, BPharm, MBBS, FANZCA.

**Contribution:** This author helped provide expert opinion and write the manuscript.

**Name:** Jason M. Pincus, MBBS, FANZCA, FCICM.

**Contribution:** This author helped care for the patient and edit the manuscript.

**Name:** Rebecca M. Kimble, MBBS, FRANZOG.

**Contribution:** This author helped care for the patient and write the manuscript.

**Name:** Victoria A. Eley, MBBS, FANZCA, PhD.

**Contribution:** This author helped care for the patient, and write and edit the manuscript.

**This manuscript was handled by:** Hans-Joachim Priebe, MD, FRCA, FCAI.

## REFERENCES

1. Saving Lives, Improving Mothers' Care. National Perinatal Epidemiology Unit Website. Available at: <https://www.npeu.ox.ac.uk/mbrace-uk.htm>. Published December 17, 2016. Accessed February 22, 2017.

2. Humphrey MD, Bonello MR, Chughtai A, Macaldowie A, Harris K, Chambers GM. *Maternal Deaths in Australia 2008–2012*. Canberra, Australia: Australian Institute of Health and Welfare. 2015.
3. Jaff MR, McMurtry MS, Archer SL, et al; American Heart Association Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation; American Heart Association Council on Peripheral Vascular Disease; American Heart Association Council on Arteriosclerosis, Thrombosis and Vascular Biology. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: a scientific statement from the American Heart Association. *Circulation*. 2011;123:1788–1830.
4. Helviz Y, Einav S, Hersch M, Shapiro H, Ioscovich A. Thromboelastography as a part of management of amniotic fluid embolism. *Case Rep Perinatal Med*. 2014;3:97–101.
5. James AH, Jamison MG, Brancazio LR, Myers ER. Venous thromboembolism during pregnancy and the postpartum period: incidence, risk factors, and mortality. *Am J Obstet Gynecol*. 2006;194:1311–1315.
6. Abdul Sultan A, West J, Tata LJ, Fleming KM, Nelson-Piercy C, Grainge MJ. Risk of first venous thromboembolism in pregnant women in hospital: population based cohort study from England. *BMJ*. 2013;347:f6099.
7. Solomon C, Collis RE, Collins PW. Haemostatic monitoring during postpartum haemorrhage and implications for management. *Br J Anaesth*. 2012;109:851–863.
8. Chaturvedi S, Sharma R. Intraoperative cardiac arrest during cesarean section – a case report. *Gulf Med J*. 2014; 3:S50–S58.
9. Hart WT, Hallenborg CP. Massive pulmonary embolism presenting as disseminated intravascular coagulation. *Hawaii Med J*. 1992;51:121, 125, 134.
10. Choi SS, Pang SY, Mak WP, Ko A. Pulmonary embolism presenting as disseminated intravascular coagulation. *Hong Kong Med J*. 2002;8:142–143.
11. Leitner JM, Jilma B, Spiel AO, Sterz F, Laggner AN, Janata KM. Massive pulmonary embolism leading to cardiac arrest is associated with consumptive coagulopathy presenting as disseminated intravascular coagulation. *J Thromb Haemost*. 2010;8:1477–1482.
12. Collins NF, Bloor M, McDonnell NJ. Hyperfibrinolysis diagnosed by rotational thromboelastometry in a case of suspected amniotic fluid embolism. *Int J Obstet Anesth*. 2013;22:71–76.
13. Wegner J, Popovsky MA. Clinical utility of thromboelastography: one size does not fit all. *Semin Thromb Hemost*. 2010;36:699–706.
14. Mallaiah S, Barclay P, Harrod I, Chevannes C, Bhalla A. Introduction of an algorithm for ROTEM-guided fibrinogen concentrate administration in major obstetric haemorrhage. *Anaesthesia*. 2015;70:166–175.
15. Snegovskikh D, Walton Z, Souzdalnitski D. Point-of-care thromboelastometry in the management of acute obstetric hemorrhage. *Curr Obstet Gynecol Rep*. 2016; 5:244–249.