International Journal of Obstetric Anesthesia (2019) **38**, 146–150

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CORRESPONDENCE

Rotational thromboelastometry as a tool in the diagnosis and management of amniotic fluid embolism

Rotational thromboelastometry (ROTEM®, Tem International, Munich, Germany) is a visco-elastometric method used to explore plasma coagulation. Compared with standard haemostasis testing (such as prothrombin time (PT) or activated thrombin time (TTa)) it has the advantage of providing results at the patient's bedside within approximately 10 minutes. Amniotic fluid embolism (AFE) is a rare and severe complication of delivery that can present with a wide range of clinical features.[1,2] In a multicenter prospective study including 45 patients, high plasma levels of insulin-like growth factor-binding protein-1 (IGFBP-1) were found to be a specific biomarker of amniotic fluid passage into the maternal circulation and might, therefore, be used to help confirm the diagnosis a posteriori.[3] Serum IGFBP-1 was higher in a case of AFE compared to a patient with compromised hemodynamic status (PPH, pulmonary embolism) or a normal pregnancy. We managed a 41-year-old primipara in labour who was using epidural analgesia, who then had an emergency caesarean delivery because of fetal heart rate abnormalities. Fifteen mL of lidocaine (20 mg/mL) plus epinephrine (5 μg/mL) were added over 5 minutes epidurally to provide effective anaesthesia. Ten minutes after the birth, the parturient presented a brief episode (less than five minutes) of severe arterial hypotension (systolic blood pressure 60 mmHg) associated with dyspnoea and agitation. Ephedrine 9 mg was injected, followed by a total dose of phenylephrine 500 μg combined with crystalloid 1 L, to restore an adequate blood pressure. In conjunction with a complete biological assessment including a search for IGFBP-1, we performed ROTEM®. The initial EXTEM plot showed a normal clotting time (CT) of 70 s (normal <100 s), whereas the initial FIBTEM A5 was 6 mm (normal >12 mm). This value subsequently matched with the plasma fibrinogen laboratory value of 1.9 g/L.[4] Postpartum haemorrhage (PPH) started shortly after the ROTEM® analysis was performed and rapidly reached 900 mL, so tranexamic acid 1 g, 3 g of fibrinogen concentrate, and sulprostone 500 μg over one hour were administered. A second ROTEM® analysis performed 60 min after fibrinogen supplementation showed an improvement of the FIBTEM A5 (to 9 mm) which correlates with plasma fibrinogen of 2.6 g/L. The patient did not receive any other vasopressor therapy or blood products.

The patient's first blood sample was analysed the next day: serum tryptase was negative whereas IGFBP-1 was significantly present in maternal serum (semi-quantitative technique with detection threshold >25 μg/L), which almost ruled out anaphylaxis and was strongly suggestive of the AFE diagnosis. The chronology of clinical signs with respect to the timing of the caesarean section and the fact that PPH occurred after the coagulopathy was diagnosed were also in favour of that diagnosis. It appeared unlikely that hypofibrinogenemia was due to dilution with crystalloids as only 500 mL of Ringer Lactate had been administered at the time of blood analysis. It was also unlikely that hypofibrinogenemia had started to develop prior to delivery as the fibrinogen concentration was 4.9 g/L at the onset of labour and no placental abruption was noted at the time of delivery. Anaphylaxis and high spinal block were ruled out as alternative aetiologies for the patient's severe hypotension. The absence of low oxygen saturation and the coagulopathy were against a diagnosis of pulmonary embolism. Loughran et al. have just reported a case of ROTEM®-assisted diagnosis and management of AFE that presented as a cardiac arrest.[5]

In our case, hypofibrinogenemia was detected with ROTEM® approximately one hour before laboratory results were available, which aided an early diagnosis of AFE and allowed us to anticipate subsequent severe PPH, initiating good venous access; extra manpower; and the early administration of fibrinogen concentrate, tranexamic acid and prostaglandins when PPH started. In the absence of a ROTEM® analysis, fibrinogen concentrate would have been administered only when PPH had reached 1500–2000 mL or when laboratory results revealed the low plasma fibrinogen. Given the pathophysiology of AFE, a wait-and-see attitude might have increased the chance of disseminated intravascular coagulation, massive blood loss and blood transfusion. We suggest that ROTEM® is a useful tool for the diagnosis and management of AFE before massive PPH occurs.

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References

1. McBride AM. Clinical presentation and treatment of amniotic fluid embolism. *AACN Adv Crit Care* 2018;**29**:336–42.

2. Helviz Y, Einav S, Hersch M, Shapiro H, Ioscovich A. Thromboelastography and amniotic fluid embolism. *Perinat Med* 2014;**3**:97–101.
3. Legrand M, Rossignol M, Dreux S, et al. Diagnostic accuracy of insulin-like growth factor binding protein-1 for amniotic fluid embolism. *Crit Care Med* 2012;**40**:2059–63.
4. Huissoud C, Carrabin N, Audibert F, et al. Bedside assessment of fibrinogen level in postpartum haemorrhage by thromboelastometry. *BJOG* 2009;**116**:1097–102.
5. Loughran JA, Kitchen TL, Sindhakar S. Rotational thromboelastometry (ROTEM®)-guided diagnosis and management of amniotic fluid embolism. *Int J Obstet Anesth* 2019;**38**:127–30.

0959-289X/$ - see front matter

https://doi.org/10.1016/j.ijoa.2019.02.005

Optic nerve ultrasonography for evaluating increased intracranial pressure in severe preeclampsia

We were very interested in the article by Brzan Simenc et al. regarding the use of the ocular ultrasonography for diagnosing increased intracranial pressure in patients with severe preeclampsia.[1]

We would like to congratulate the authors for their paper, however we wish to comment on the methods used to measure the optic nerve sheath diameter (ONSD). First, the authors used the B-Scan technique, an examination broadly utilized to diagnose ocular diseases. This technique has limited sensitivity for measuring orbital structures, as it is affected by the so-called 'blooming effect'.[2] This is related to the lack of a standard sensitivity setting in performing a B-Scan and should not be confused with the Doppler-associated blooming effect. In the case of the B-Scan, it means that if ONSD is measured at a lower sensitivity setting, bigger dimensions will be seen in comparison to those obtained at an increased sensitivity setting. This effect could be misleading if a difference of less than 0.5 mm is being considered, as happens when we evaluate ONSD, but is less important when considering larger lesions.[3]

Another issue is the use of the probe through the closed eyelids, making detection of the direction of gaze, and consequently the exact probe position, difficult. In ophthalmology, during the ultrasound examination, the B-scan probe is routinely used with open lids, using methylcellulose and anesthetic drops. This allows visualization of the eye, making the probe orientation much more reliable.[4]

Due to these aforementioned limitations, in future studies we suggest utilizing the standardized A-Scan technique.[5] With this technique it is not only possible to measure the ONSD with more precision, because the interface between the arachnoid and subarachnoid fluid gives high reflective spikes that allow an objective way of performing such measurements,[6] but also with this method there is no blooming effect.[7–9]

For this reason, the A-Scan technique provides a more accurate reference range for ONSD that can be widely used and replicated without the need for laboratory-related reference settings. Finally, we strongly disagree with the authors when they state that "ultrasonography is an easy-to-learn technique". Because of the blooming effect and the difficulties in the exact placement of the probe, skill is required to get reproducible measurements.[10]

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References

1. Brzan Simenc G, Ambrozic J, Prokselj K, et al. Ocular ultrasonography for diagnosing increased intracranial pressure in patients with severe preeclampsia. *Int J Obstet Anesth* 2018;**36**:49–55.
2. Rosa N, Lanza M, Borrelli M, et al. Low intraocular pressure resulting from ciliary body detachment in patients with myotonic dystrophy. *Ophthalmology* 2011;**118**:260–4.
3. De Bernardo M, Rosa N. Measuring optic nerve sheath diameter as a proxy for intracranial pressure. *JAMA Ophthalmol* 2018;**136**:1309–10.
4. Tenuta M, De Bernardo M, Rosa N. Comments on "Neuromuscular ultrasonography of cranial nerves". *J Clin Neurol* 2017;**13**:212–3.
5. Ossoinig KC. Standardized echography of the optic nerve. In: Till P, editor. *Documenta Ophthalmologica Proceedings Series, Ophthalmic Echography 13*, 55. Dordrecht: Springer Netherlands; 1990. p. 3–99.
6. Ossoinig KC, Cennamo G, Frazier-Byrne S. Echographic differential diagnosis of optic nerve lesions. In: Thijssen JM, Verbeek AM, editors. *Documenta Ophthalmologica Proceedings Series Ultrasonography in Ophthalmology*, 29. The Hague: Springer Netherlands; 1981. p. 327–32.
7. De Bernardo M, Rosa N. Clarification on using ultrasonography to detect intracranial pressure. *JAMA Ophthalmol* 2017;**135**:1004–5.
8. De Bernardo M, Rosa N. Transbulbar B-Mode sonography in multiple sclerosis: Clinical and biological relevance. *Ultrasound Med Biol* 2018;**44**:508.
9. Rosa N, De Bernardo M. Measurement of the optic nerve in a resource-limited setting. *J Neur Rural Prac* 2017;**8**:310–1.
10. Iaconetta G, De Bernardo M, Rosa N. Coronal axis measurements of the optic nerve sheath diameter. *J US Med* 2017;**36**:1073.

0959-289X/$ - see front matter

https://doi.org/10.1016/j.ijoa.2018.12.010