CSL

Bleeding news

Point-of-care visco-elastic testing for postpartum haemorrhage: A narrative review

Hans Vandersmissen, Eva Roofthooft, Sarah Devroe, Steven Thiessen, Hendrik Stragier

PMID: 41084872 DOI: 10.1097/EJA.0000000000002293

Author of the comment: *Dra. Raquel Ferrandis Comes.* *Anaesthesiology and Reanimation. Hospital Universitari i Politècnic La Fe, Valencia. Member of the SEDAR-COVID working group.*

The authors carry out a narrative review on the use of viscoelastic testing (VET), primarily ROTEM, in postpartum haemorrhage (PPH), with a particular focus on the indication of fibrinogen and tranexamic acid (TXA). Its main limitation is the lack of a systematic literature review, as the authors themselves acknowledge the selection bias in the articles.

It is important no note that coagulopathy is rare at the onset of PPH and will develop as blood loss increases. Thus, with around 1,200 ml of blood loss, only a minority have a Clauss <2 g/L. In this context, **fibrinogen is the key marker:** A Clauss value <2 g/L or a low FIBTEM A5 predict progression to severe PPH.

VET in PPH have made it possible to avoid unnecessary plasma transfusions and to optimise fibrinogen replacement, which, particularly in pregnant women, should be carried out using fibrinogen concentrate. The ROTEM model should be considered, since the authors suggest that with ROTEM Sigma, and the change of cartridges, the replacement threshold becomes FIBTEM A5 ≤8 mm, equivalent to the previous ≤11–12 mm (ROTEM Delta), corresponding to the 2 g/l threshold according to Clauss. The use of VET in the context of PPH has resulted in less plasma transfusions, less TACO and less blood products in large cohorts (particularly the OBS Cymru programme in Wales). However, RCTs are small or show negative results; it should be assessed whether this is due to an inappropriate endpoint: OBS-2 reported fibrinogen with FIBTEM <15 mm and showed no benefit, and only the subgroup with A5 ≤12 mm or Clauss ≤2 g/L suggested an effect.

With regard to tranexamic acid, it should be administered as soon as possible following the PPH diagnosis. However, routine prophylaxis is not supported and should be reserved for high-risk patients. VET results cannot be trusted either, since they are not very sensitive to hyperfibrinolysis. Other limitations of VET include the fact that the platelet function is poorly assessed (the role of PLTEM in PPH is unclear), and the devices and their thresholds are not interchangeable.

A different scenario is acute obstetric coagulopathy (AOC), which occurs as a distinct condition (≈1/1000 births), associated with placenta abruption and amniotic fluid embolism. It combines hypo-/dysfibrinogenemia with massive hyperfibrinolysis (very high PAP levels, elevated aPC, and consumption of FV/FVIII/FXIII). The treatment should be early fibrinogen plus TXA.

As key points, I would highlight the indication of VET to guide fibrinogen (with the right threshold depending on the cartridge) and to *avoid* plasma transfusion. Conversely, the information from VET cannot be regarded as reliable on platelets or fibrinolysis. Therefore, early TXA should always be administered, without waiting for the tracing. In case of abruption or amniotic fluid embolism, a completely different scenario must be considered, that of AOC, and an aggressive early treatment with fibrinogen must be applied.