

Letter

Thrombelastography and sepsis

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We read with great interest the recent report by Gonano *et al.* [1] describing the effects of antithrombin versus placebo in the coagulation profile of a group of patients with severe sepsis. However, we thought that several important issues needed to be addressed in regards to hypercoagulability and thrombelastography (TEG). First, hypercoagulability was not clearly defined in the study. Second, neither Coagulation Index (CI) nor Thrombodynamic Potential Index (an alternative TEG index of hypercoagulability) [2] were calculated or reported. Third, conventional studies of hypercoagulability were not performed in parallel. Fourth, Table 2 in the paper shows that some patients had low fibrinogen, platelets, and prolonged activated partial thromboplastin time with a probably 'hypocoagulable' thrombelastogram (CI lower than -3) profile.

Contrary to the authors' statements, there are previously described studies of hypercoagulability in sepsis using TEG.

A decade ago, Grant and Hadley [3] described the coagulation changes of 27 neonates with sepsis and found a 'hypocoagulability' profile in 85% of the neonates studied. These findings are also supported in animal data for which our lab has shown similar hypocoagulable patterns in a porcine model of sepsis using TEG [4].

Hypercoagulability is defined in thromboelastography as a CI greater than +3 [2]. This means that patients with reaction time (*r*), coagulation time (*k*), alpha angle, or maximum amplitude on the 'hypercoagulable side' may still fall within a normal CI range and not meet criteria to be called 'hypercoagulable'. We believe that it would have been of great interest if the authors had provided the actual CI values, as well as the percentage of patients with hypercoagulable and hypocoagulable profiles.

Authors' response

Stephan C Kettner and Christopher Gonano

We appreciate the interest of Dr Puana and Dr Nates in our article and their comments. Standard TEG parameters have been described as they are most commonly used by clinicians. Calculated indexes, however, add limited information. No patient with severely altered coagulation was included in our study, due to the complex inclusion and exclusion criteria of the KyberSept trial. We actually excluded patients with severe coagulation abnormalities, who might have benefited most from antithrombin therapy. A recent analysis of the KyberSept trial showed that high-dose antithrombin in septic patients with disseminated intravascular coagulation resulted in decreased mortality rates [5]. We agree that hypercoagulation may not be present in septic patients with disseminated intravascular coagulation, as hyperfibrinolysis may cause straight line TEG.

We knowingly did not cite the study by Grant and Hadley [3], for several reasons. First, the coagulation system in neonates is profoundly different compared to adults [6], and normal values for TEG have not been established in neonates [7]. Second, causes and incidence of sepsis in neonates are completely different compared to adults [8]. Third, the immune system in neonates is immature [9]. Fourth, we feel Grant's interpretation that TEG has a 96% sensitivity of and a 96% specificity for sepsis is inappropriate, as if every abnormal TEG in neonates should be associated with sepsis. Of course there are other causes for abnormal TEGs in neonates.

Competing interests

The authors declare that they have no competing interests.

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