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Discussion of: “The hypercoagulability paradox of chronic kidney disease: The role of fibrinogen”

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Discussant

DR. TAM HUYNH (Houston, TX): Dr. Nunns and his group has shown that patients with severe chronic kidney disease indeed show a pro-thrombotic state based on the TEG parameters, namely increased clot formation, increased clot strength and decreased clot breakdown. In addition, the authors also show a moderate to strong correlation between the final clot strength and the fibrinogen level.

We know from previous studies, increased clot strength is also associated with increased risk of thromboembolic complications, so that others conclude that hypercoagulability in patients with chronic kidney disease must be mediated, at least in part, by fibrinogen level.

Indeed the paradox of hypercoagulability and increased bleeding risks described in these patients remains poorly understood. Research such as this study by Nunns, Moore and co-authors is definitely much needed.

I have three questions for Dr. Nunns' group.

First, chronic anemia and thrombocytosis are two common characteristics of patients with chronic kidney disease, and those two factors have been shown to be associated with increased final clot strength on TEG parameters. Did your group look at those two specific factors?

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Second, in the current cost-conscious environment, would you and your co-authors recommend that we must draw TEG assay and fibrinogen level in all patients who undergo hemodialysis access surgery?

And lastly, based on the results of your study, what should be the next study with regard to studying hypercoagulability in patients with kidney disease?

Closing discussant

DR. GEOFFREY NUNNS: With regard to the first question, in this study, we unfortunately didn't look at either hematocrit or platelet count. However, we feel the other important thing more than platelet count to look at is also platelet function assays given that we know there's a wide degree of platelet dysfunction in this population, and we really need to look at how the platelets work, not just how many of them there are.

As for the second question of cost effectiveness, we do actually use TEG clinically in these patients. Patients that have hypercoagulability manifested on TEG, generally with an increased MA, they do receive low dose of heparin intraoperatively with clamping of the artery, and then they receive postoperative aspirin to try to prevent thrombotic complications.

Fibrinogen at this point, it's really just for research purposes. We can't get any outcomes with that. In all honesty, given that TEG MAs is the best test of clot form, I think fibrinogen is probably inferior to MA.

With regard to future studies, we do have ongoing prospective studies investigating end stage renal disease patients to determine additional factors involved in hypercoagulability or bleeding risks.

To this end, we are including proteinase as well as platelet function assays and then collect thrombotic complications to see if we can make some correlations between TEG variables or other hemostatic parameters. With this, we'll include pre- and post-dialysis samples for these patients to see what dialysis does to their clotting factors, to their platelet function, and eventually the goal is to perform randomized control trials to see if we can identify certain patients that would benefit from antiplatelet therapy and anti-fibrinogen therapy to prevent thrombotic complications.