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Bringing It All Together: Real-World Data in Anticoagulation Reversal

Announcer:
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Dr. Dobesh:
Alright, so kind of just bring it together, quick summary of what we've discussed. And then we're going to go into Q&A. As we mentioned, okay, several times, now, there are real-world data comparing these agents, but these studies are limited by hemostatic efficacy collected retrospectively in relatively small numbers. We do have some large data, which is available, which might be helpful in this, but once again, this doesn't tell us anything about specific patient. There are, you know, when we - if you are going to implement, when you implement, it's not really an 'are' thing anymore. When you implement reversal protocols for your institution, there's lots of things that need to be addressed. This sets very well with the role of an anticoagulation stewardship program. And so just things that we should try to tackle, and whether that be, you know, regardless of the agent you're using, and obviously, there are some special considerations when considering andexanet alfa in your institution.

But we need the balance. Yes, we do need clinical trial data. But it doesn't answer everything. And it's very, as Mark said, it's deep, but it's not very wide. Then we need real-world evidence. And so we have to basically train ourselves and remember the benefits and disadvantages of each, so that we can make a complete and educated decision on what we plan to do.

And then finally, just remembering that not all bleeding patients are the same. And so there's, you know, we can't sit here and say, okay, this is what you do for everybody. It's going to matter, where's the bleed? How quick do they need to reverse? What's the potential consequences here? How much decision-making time do we have? And so with that, I want to thank everybody very much. We're going to open it up to questions. I'll sit down at the table with Mark.

Dr. Crowther:
Yeah, so the question is, if you're looking at the studies of andexanet alfa, where we've looked at the anti-Xa heparin level, or the anti-Xa rivaroxaban or apixaban levels, they dropped down to essentially nothing as soon as you start the infusion, and they remain low for the duration of the infusion, and then they start to rise thereafter. And so the question is, is that your opportunity to plug the hole? Meaning to stop the bleeding. Or is there an effect that hangs on after that, and that return of the anti-Xa rivaroxaban or apixaban levels, doesn't necessarily imply that the bleeding propensity has come back? And that's the critical question because people say, 'Well, wait a second, this person comes in, rivaroxaban or apixaban, they've got a triple A repair, they're currently in the OR, they're - it's two hours and the andexanet infusion is going to finish - remember, no data on surgical, but triple A's bleed, so it could be bleeding data, you know, do we need to continue the infusion to prevent that Xa level coming back? So Paul, what do you think about that?

Dr. Dobesh:
So actually, I think I wouldn't continue the infusion in that setting. I think, with the - with this line shows there, the thrombin generation, we've jumped - in that timeframe, we have given a time to jumpstart the thrombin generation. Alright? Now Mark and I, even just discussing before, what does thrombin generation actually mean? And there's some debate about that. But you have - given that the, you know, the thrombin generation does not go back down like. The thrombin generation kind of returns to the patient's stable baseline. And so therefore, theoretically, you know, that part of it's been taken care of, and so therefore the up-blip ,per se, in the Xa, I find extremely not - because - not very concerning myself.

The other thing, though, is also in that timeframe, right, we - typically a bleed's going to – you've got to stitch it, cauterize it, or plug it right or put pressure on it. Usually something has to happen in that process. So yes, it does give you a time window to do that. It also

gives a time window just for hemostasis to kind of normalize. So I personally am not concerned about what happens after that.

Dr. Crowther:
Yeah, and I would agree with that completely. What would be not a good thing is that what you don't want to do is say, 'Well, let's start the andexanet and let's see if the bleeding stops. And if at 2 hours, the bleeding's still going on, then we'll talk to the interventional radiology people.' That's not the correct strategy. I have a rules of hematology. And the fourth rule of hematology is that, unless fired from a cannon, warfarin does not cause bleeding. And people say, 'That doesn't make any sense.' I said, I've never seen a case of warfarin causing bleeding.

I've seen lots of holes in blood vessels, where it was a lot easier to see where the bleeding was coming from when the patient was on an anticoagulant. And so the key there is that andexanet is not the - it's part of a strategy. And the most important part of that strategy is identifying where the bleeding is coming from, and doing the intervention required to stop that bleeding. And using the reversal agent, as a bridge to allow you to get the person to the endoscopy suite, and then to allow that definitive clot to firm up before the andexanet is withdrawn and the Xa levels start to rise again. And I would absolutely highlight what Paul said about the endogenous - thrombin generation, it does not drop back down below normal. And so that, to me, is a positive thing. People say, 'oh, you know, the thrombin generation has gone way up. I'm concerned about that.' I say, I'm not concerned about it at all. The person is going to bleed to death. I'd like to have a little extra thrombin floating around myself. And I think that's actually a positive to have a thrombin generation potential that's higher than that before the bleed started.

So yeah, if there's no questions, then I think we'll give you a few extra minutes back of your day. So thanks, everybody, for coming. Hope you're enjoying the meeting. Thanks very much.

Dr. Dobesh:
Thank you.

Announcer:
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