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Case: 60 yo Male on Factor Xa Inhibitor with Bright Red Vomiting

Announcer:
Welcome to CME on ReachMD. This episode is part of our MinuteCME curriculum.

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Dr. Cash:
So, let me go through a case study briefly. We've got a 60-year-old man, Mr. B, he's brought to the ED. He had an episode of syncope at home. He's had melena for the last five hours and upper abdominal pain for the last three days. He's on anticoagulation for thromboembolism. He's actually had two PEs. The last was two years ago. He also has hypertension. You can see his medications. Rivaroxaban is the one I'm going to highlight. 20 milligrams a day. Blood pressure, he's shocky. He's got a systolic of 94, diastolic is 60, heart rate's 110, and his hemoglobin has fallen by about six points compared to a baseline value two months ago. His BUN is up based on his melena, his presentation, his BUN being elevated, we're suspicious that he's got an upper GI bleed.

So, we calculate his Blatchford score. It's 16, so he is at high risk for mortality. This qualifies as a life-threatening bleed based on the parameters that we just went over from the ACG guidelines. We ascertain his last dose of his DOAC, it was six hours prior to presentation. We admit him to the ICU. We follow our criteria. We get him a couple of large boar IVs. We start resuscitating him with crystalloid and blood products. We start him on an IV PPI. We want to get that pH, that intragastric pH, greater than four ideally to help platelets work. He has witnessed hematemesis in the ICU, and we give him a dose of Reglan to try and promote some gastric emptying, and you can do this with erythromycin or azithromycin if you have that available. Typically, we use Reglan about 30 to 45 minutes prior to endoscopy to try and get that clot out and help our visualization.

So, our critical decision, so we know we're going to scope this gentleman. Our critical decisions now are do we reverse his anticoagulation, or not reverse? Do we just go in and start doing things endoscopically? What other resuscitation does he need? What's his stability with regards to endoscopic evaluation? Can we sedate him? Does he need another study in lieu of endoscopy? Does he need a CTA or therapeutic angiography? Usually, that's going to be done after endoscopy if we're not able to stop bleeding or we can't identify where the patient's bleeding from, and then ultimately surgery.

This is the busy slide that I mentioned. I'm not going to go through what's on the right, because I don't think you can read it, but a very nice article by Milling that was published in a GI journal, "Digestive Diseases and Sciences" last year, kind of going through an algorithm and a recipe for reversal, and you can see, or what I've highlighted here is that there's recommendations for vitamin K reversal, as well as for DOAC reversal based on the dosing as well as the timing of reversal and what agents to use, and it goes along with those guidelines that we just talked about. Generally, four-factor PCC or specific reversal agents based on dosing and timing of the last dose.

So, we decide to reverse this patient because he is critically ill. He's got risk factors for significant mortality, so we give him Andexanet 800 milligram bolus, then eight milligrams per minute for two hours based on his timing of his last dose as well as his actual dose that he's taking. We give him some more blood. We do endoscopy. We find a Forest 1 lesion. You can see that spurting ulcer there in his stomach. That's in the greater curve, and we achieve endoscopic hemostasis with some epinephrine, and we inject some clips around

that, and I'll show you that just so you can see what we do when we do endoscopy.

When we des describe ulcers and stigmata, this patient had a Forest 1 ulcer. You can see, we break up the stigma of bleeding based on whether they're actively bleeding, whether it's a non-bleeding visible vessel adherent clot, pigmented spot, or a clean base. Clean base, we love to see. Those patients can be discharged. We're not worried about them. They're going to heal up nicely. Their risks of re-bleeding is really, really small, but the patients who have active bleeding, those Forest 1 lesions, those are the ones we worry about. The mortality rate's 11%. About one out of three are going to end up going to surgery. Re-bleeding risk, more than 50%. If we don't do anything with endoscopy, so that's without endotherapy, we reduce that risk by about 50% with our endotherapy, with our PPIs, and certainly reversal of anticoagulation is an adjunctive therapy for that. Then the list, you can see that risk going down with the other stigmata, but everything other than a clean base, we're going to intervene with during endoscopy. We're going to inject with epinephrine. We're going to burn those lesions with COAG therapy or we're going to click those lesions.

So, you can see active bleeding in this gentleman. We are going to inject this ulcer and this bleeding lesion around the bleeder, as well as right into the bleeder. You know, it's important to recognize that when we use epinephrine and that is our preferred injection agent, it's one to 10,000. It comes in the little brown boxes. What we're really doing is we're tamponading that vessel. We're not really causing a lot of vasoconstriction. Everybody thinks that the EPI causes the blood vessels to shrink up and stop bleeding. It's really the tamponade. There are some studies showing that you can achieve hemostasis just by injecting saline. Of course, that's not going to stick around that long, so we typically use epinephrine, but that's insufficient as monotherapy. We have to do something else. We need to use some other form of therapy in this patient. What we typically will do is, we'll use COAG therapy, or we'll clip these lesions. You can see this hemostatic clip going right up into that ulcer, and we're going to clip that, and it's a generally simple process, and of course, what we're trying to do is physically close that bleeding lesion and clamp that vessel that's underneath the mucosa causing all of the problems.

All right, so in terms of our case conclusion, Mr. B does well with his endoscopic therapy. He remains in the hospital another three days. His melena clears, his vital signs are stable. We continue his IV PPI drip according to protocol when we have an active bleeder, and we are using IV PPIs in terms of bolus and drip. We continue that for three days after hemostasis has been achieved, and then we advance this diet slowly. We keep him on clears for an extra day just to make sure if we have to go back that we can go back and get good visualization, and then we advance him. He does well. He's discharged on twice-a-day PPI for a couple of weeks to try and heal that ulcer, and then once a day for life. Because he is a high-risk patient, he needs his anticoagulation and so, he needs to be on a PPI forever because we're going to have to restart that anticoagulation. We also found him to be H pylori positive. This is something that's frequently overlooked in the heat of the moment. This is another risk factor that's modifiable. About 10% risk of peptic ulcer disease over a lifetime with H pylori. We took gastric biopsies after we had achieved hemostasis in this patient and we found him to be positive, so we treated that, eradicated that, and then further, hopefully, further reduced his risk for future bleeding, and then we managed him in terms of his anticoagulation traditional anticoagulants for about a week, and then we restarted his DOAC in consultation with his prescribing physician with close monitoring. He's done well since then. So, with that, I will stop. Thank you very much for listening to me and allowing me to present to you and I'll look forward to the Q&A.

Announcer:
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