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Putting It All Together: Coordinating the Multidisciplinary Care Plan for the Patient With VTE

Announcer:
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Dr. Davidson:
I'm glad to be here with my colleague and friend Dr. Vic Tapson to put together a DVT/PE diagnosis and treatment. We'll spend a few minutes in the beginning reviewing what I've already presented, really. And for the last 4 or 5 minutes or so, I'm going to ask Dr. Tapson to elaborate on how to consider PE patients in whom anticoagulation by itself isn't sufficient. And what to do and what to expect and what the courses are there.

So my approach to this, my philosophy to taking care of patients, including these, is to put multiple layers of safety between the patient and a bad outcome. And second, that the very best and the simplest way to treat patients is to treat them all the same: the way you'd want to be treated. And as a consequence, Dr. Tapson and I may actually have some differences, which would be good to surface, which we'll briefly discuss.

First, when it comes to DVT, have high suspicion, get the diagnostic tests. But if there's a delay in getting the tests, start treatment. Oral treatment with a DOAC, rivaroxaban or apixaban, is actually quite safe. You do that for the 2 or 3 days, if necessary, to get your ultrasound and take your next steps. And as for duration, you can look that up. There are lots of rules and recommendations.

Vic, anything to add there?

Dr. Tapson:
I think you covered that part pretty well, Bruce. Yeah. Agree. Get the diagnosis. We still underdiagnose the disease. Make the diagnosis and get started. Get on anticoagulation.

Dr. Davidson:
Okay, so now for pulmonary embolism. Again, critically important to diagnose if there is suspicion. And if there's high enough suspicion, it does not require a D-dimer, get an imaging test. If for some reason you're in some setting where you can't even get the imaging test and the patients appear safe to anticoagulate, do that while you're working them up. I have also emphasized in my slides that what I think has been a mistake amongst what have been consensus patterns and what I've really considered in some cases, unfortunately, more like expert focus groups than expert people, that patient may be under perfusing without having a systolic blood pressure below 90. The normal blood pressure is 120 or more, and somebody who's having acute PE, it's probably higher. So in such patients be very mindful that they are under perfusing their cutaneous tissue, and I have recommended that the first dose in almost everyone should be IV heparin 80 U/kg of absolute body weight. The advantages: they're instantly receiving anticoagulant. You can adjust it if they bleed. You can reverse it with protamine, which you can't do with sub-Q low-molecular-weight heparin. If the patient is quite stable, then absolutely start low-molecular-weight heparin. The IV heparin has a half-life of only an hour. You can give the low-molecular-weight heparin right away if they're stable and know your patient's covered. And then, if they are stable, by all means, low-molecular-weight

heparin by weight twice daily, shift them to a DOAC. But there are plenty of patients who are not rendered hemodynamically normal with that anticoagulation, and for that reason I think we're past the beginning. We're now well into alternative approaches that appear very safe and can help patients.

Now, Dr. Tapson worked on catheter-directed thrombolysis, biochemical lysis of clots for a long time. He did the pioneering studies to find minimum effective dose, and he can correct me, but I think he has now shifted his primary attention to suction thrombectomy, which involves no lytic bleeding risk factor outside of the procedure itself, which can be done very safely, which definitely removes clots and may help the patient in the short term and the long term.

But since a lot of doctors don't know what the catheter-directed biochemical thrombolysis involves for the patient, or what the suction thrombectomy involves, Dr. Tapson, I'd like you to go for our remaining 4 minutes or so.

Dr. Tapson:
Sure, Bruce. Well, great introduction. I agree with what you've said so far, that blood pressure, 90 systolic, is arbitrary; we got to be careful with that. Sometimes people are hypoperfusing when the blood pressure is greater than 90, so be careful and get them anticoagulated. We divide these patients up. The sicker ones, the high-risk patients, I think decisions there are pretty easy, Bruce. When you're hypotensive and high risk, we know we've got to do something more. And guidelines tell us thrombolytics, based on pretty poor data. Eight-patient study, going back to 1995, systemic thrombolytics. So the sick people, these hypotensive people, we've got to do something more. In general, we can give systemic thrombolytics or ECMO. If we can't move those patients, Bruce, if they can't be moved, they're too sick to move, we got to do something. But in the modern era, like you suggested, we've got other techniques. Lazar Greenfield suggested transvenous pulmonary embolectomy 50 years ago. Didn't have a lot of engineering help, perhaps, back then, may not have caught on, but was kind of a cool idea. And now we have suction embolectomy, aspiration embolectomy, large-bore embolectomy we can use now, and we've got some pretty good data on these, even the high-risk sick patients. In one study called FLAME, aspirating clot with the FlowTriever device with large-bore embolectomy. Lower mortality than in patients that got systemic thrombolytics.

So the high-risk patients, I mean, these are the tough ones, Bruce, are the ones you kind of alluded to. These intermediate-risk cases, they're not hypotensive. Their troponin may be up, their RV may be abnormal, they may have a large clot burden. These are the kind of less sick, but still concerning, intermediate-risk patients. There's a lot of features about these patients that can be concerning: heart rate greater than 100, been shown to be associated with increased mortality; shock index greater than .89, increased mortality; extensive clot burden, increased mortality; contrast reflux on CT down to liver, increased mortality; elevated lactate, increased mortality.

When I see a patient that's intermediate risk, not hypotensive, I think, wow, what can we do here? I'm a little uncomfortable just sitting and waiting when Cecilia Becattini's case, her study published in 2016, suggested if you're intermediate high risk, meaning you're not hypotensive but your RV's abnormal and your tropes abnormal, your mortality rate is 10 times higher than if you're low-risk PE. A lot of our colleagues, Bruce, want to wait for randomized trials for intermediate-risk PE. Do an aggressive therapy versus anticoagulation alone. You know, we don't have the data right now; we don't have a randomized trial. I'm in the camp where we have enough data now, I'd rather not wait. I'd rather consider intervening in someone I'm concerned with that has features of concern, tachycardia, high clot burden, et cetera.

Dr. Davidson:
And if I could interrupt just a moment. Hypotensive is less than normal blood pressure. It's not all the way down to 90. So I think that's exactly what you're saying. 90 is a failure, 90 systolic is shock, and we need to be concerned if anticoagulation alone has not restored normal hemodynamics. Go ahead.

Dr. Tapson:
Sure, absolutely, Bruce. So the idea is, yeah, we see these patients. One example is in this 800-patient large-bore catheter embolectomy registry where we saw less than 1% all-cause mortality in mostly intermediate-risk cases. In that study, 34% of intermediate-risk cases, normotensive cases, had a low cardiac index. So we've got data now in several studies suggesting these patients may, in fact, even though the blood pressure is greater than 90, may have a low index. I think the goal of therapy now should be prevention of shock rather than waiting for rescue therapy if you've not been hypotensive yet. It's like waiting for the ambulance at the bottom of the cliff.

So we've got data now, Bruce, that we can aspirate clot. The FLASH registry and the FLAME study suggest we can aspirate clot. Low mortality, low side effect rate, and we don't have the complications of thrombolytics. Again, I'd rather do something in these patients. There's several large studies, meta-analyses suggesting if you have catheter-related procedure, mortality may actually be lower than for medical therapy. So we don't have randomized trials yet; we're getting there, Bruce. And the data we have so far, I think is pretty convincing. Large observational registries, let's do something with these cases. You can sit and wait and be in that camp and say, I want

a randomized trial, or you can say, I think there's enough data out there that we should be doing something while we wait for randomized trials.

Dr. Davidson:
I agree, we have to do more than one thing at a time rather than wait for a randomized trial, and some of the randomized trials or taking patients and giving them sub-Q low-molecular-weight heparin, which may not be properly absorbed. When we already have this data that shows in patients whose hemodynamics are not normal, suction thrombectomy improves their prognosis and without the hemorrhage risk of lytics.

I think I think that's a good review. Please, listeners, you're free to contact me or Dr. Tapson. Thanks for your interest.

Dr. Tapson:
Thanks folks. We covered a lot in 10 minutes. Thanks, Bruce.

Announcer:
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