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Right from the Onset: PE Diagnosis, Management

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

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Dr. Davidson:
Let's talk about pulmonary embolism, acute PE diagnosis and management.

History: Use the simplified Geneva score, Wells score, or you can even use clinical impression. The D-dimer as with DVT, unless it's high probability impression, then you skip the D-dimer and go straight to imaging. So what are the imaging tests? A CT pulmonary angiogram. Look at the PE, but not merely the presence, the severity, additional risks for bad outcome. Does contrast reflux at all down the IVC or to the liver? Is there a large RV? It should be smaller, not equal to, certainly not larger than the size of the LV. Is there emphysema, fibrosis, pleural effusion? Does the heart appear normal? Is there ascites, hepatosplenomegaly, which you can see in the lower slices of the CT. Any of these give you evidence for a more difficult course. And if the patient has renal insufficiency when you're planning the CT pulmonary angiogram, if you can give them additional normal saline over 3 to 12 hours, if tolerated, that's been shown to have some kidney protection from the dye. If you can't do a CT pulmonary angiogram, do a VQ scan, or even simply a perfusion scan compared to a chest X-ray. If the patient has multiple, more than one, segmental or larger perfusion defects where the chest X-ray is normal, that counts as diagnostic. And if it's a pregnant woman, then definitely put in a urinary catheter so the radioactivity from the perfusion scan, when it drips down to the bladder, doesn't irradiate the fetus, but rather goes right out in the urine. You can do a duplex ultrasound of extremities if you can't do any of the above, but a negative doesn't help you at all. A positive gives you a reason to anticoagulate. And obtain an ECG in every patient so you can see what the baseline heart looks like. Hopefully normal, but if there's any strain on the right ventricle, that's a warning to you.

All right, further assessment. Blood pressure, systolic, is it lower than normal? Not as low as 90, frank shock, which some people push for. Is it lower than normal? Is there tachycardia? Is the rhythm normal? Is there an A-a gradient, for which you'll need a blood gas not merely O_2 saturation? If you're able to do lab studies, the BNP or NT-proBNP is more responsive than troponin. It's not influenced the same way troponin is by underlying renal insufficiency. Dual lactate, if it's elevated there is under perfusion. If you know about a circulation time, it's very simple; it's mag sulfate 50% 2 mLs. Read about it. That's easy to do at the bedside and safe. Do an echocardiogram if you have that luxury available.

What about the clot management? If it's safe, anticoagulate IV with a bolus of 80 units unfractionated heparin/kg actual body weight, not ideal. The half-life at that dose is 60 minutes. Most patients will be in range during that time and at least you have anticoagulated the patient. It's been shown that – and I'll get to this shortly – that low-molecular-weight heparin, you barely get there in normal volunteers in an hour, and that's not what we want. If the hemodynamics are normal, you can follow that bolus with low-molecular-weight heparin 1 mg/kg actual body weight every 12 hours, but they have to be normal. If somebody's having a QP, their blood pressure is at least 125, 130. If the blood pressure is less than that, that is not normal. If they're not normal, continue the heparin IV 18 U/kg actual body weight

per hour. Continued instability but not shock? Consider action, adding suction thrombectomy if that's available to you. Shock? Systemic IV thrombolysis or surgical embolectomy.

This is what I wanted to show you. During COVID, our colleagues in Europe did many studies looking at the kinetics in ICUs of sick patients on full anticoagulation. And this is after 4 days of weight-adjusted, creatinine-adjusted, twice-daily low-molecular-weight heparin. And what you see here are 4 or 5 of the 25 patients who had subtherapeutic levels despite 4 days of twice-daily dosing by hours 8 or 9. This is a dreadful result. And the other thing you see here, if you see numbers above the green box of so many patients who had excess anticoagulation making them prone to bleeding. So low-molecular-weight heparin, any subcutaneous therapy, not even insulin, we don't do that in patients who are quite ill, whether they're in the emergency department, the step down, or the ICU. We should stop doing that, don't do it anymore. Use IV heparin.

Further patient management. Oxygenate, monitor the venous return, provide adequate arterial perfusion with pressors, and use VA-ECMO if it's available. If the BP, heart rate normal, everything's normalized and they're stable with stair climbing, ask yourself that question. Is there pleuritic pain? You need not touch a narcotic. Oral indomethacin – not ibuprofen – oral indomethacin 50 mg every 8 hours is highly effective. And if there's stable normal perfusion after IV heparin bolus and sub-Q low-molecular-weight heparin or IV heparin infusion, you can switch to oral.Thank you for your attention and feel free to contact me for questions.

Announcer:
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