Transcript Details

This is a transcript of a continuing medical education (CME) activity. Additional media formats for the activity and full activity details (including sponsor and supporter, disclosures, and instructions for claiming credit) are available by visiting:
https://reachmd.com/programs/cme/hfpef-diagnostic-approach-through-case-review/16515/

Released: 11/30/2023
Valid until: 11/30/2024
Time needed to complete: 4h 49m

ReachMD
www.reachmd.com
info@reachmd.com
(866) 423-7849

HFpEF – Diagnostic Approach Through Case Review

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

Prior to beginning the activity, please be sure to review the faculty and commercial support disclosure statements as well as the learning objectives.

Dr. Raza:
So, we are now going to be moving into focusing on left heart disease in pulmonary hypertension.

Okay, so we're first just going to start off with looking at the diagnostic approach of HFpEF through a case review. Okay, so starting off, we have a 58-year-old female who has a past medical history of prior PE and DVT. Sleep apnea treated with CPAP, obesity, hypertension, and then this reported history of lupus that we know that the patient's stable off of medical therapy, and actually all serologies were negative when we get further collateral information from their rheumatologist. So the patient was diagnosed with, quote unquote, pulmonary hypertension, but this was based on an echocardiogram alone. And we've heard a lot today about how really right heart cath is the gold standard and must be performed to really confirm the diagnosis of pulmonary hypertension, and also understand what's the primary underlying pathophysiologic mechanisms so we can appropriately characterize and treat our patients. We note on the echo report, we don't have images yet, that there is a normal LV/RV size and function, and then there's an estimated PSP of 71 mmHg. So based on this information alone, this patient is treated or started on treatment with sildenafil, and this is back in 2019.

A few months later, the patient undergoes at the same institution a right heart catheterization. We don't have any waveforms sent to us, but per report, there's a mean PA pressure of 64/16 with a mean of 37, a wedge pressure of 15. But then the report notes that the LVEDP is 26 with an index and output that are normal at 2.9 and 6.4. So taking a second to pause here, so per these reported hemodynamics, we've confirmed pulmonary hypertension but haven't really understood whether this is a pre-capillary or post-capillary patient with conflicting information based on the reported wedge pressure versus the LVEDP.

Now fast forward, so it's been about a year and a half, almost 2 years. This patient continues on sildenafil, it's now March 2021, and she's presenting to our clinic for a second opinion on her diagnosis and management of pulmonary hypertension. She notes that she has persistent dyspnea on exertion, has not felt really any symptomatic improvement after being started on sildenafil.

In terms of her exam, you know that her BMI is 39. Her JVP is 6 cm of water just normal, negative HJR, normal S1, S2, no lower extremity edema.

And then for her EKG, we see that she's sinus rhythm. We notably see the absence of any changes significant with right ventricular hypertrophy, right atrial enlargement, right access deviations, just sinus rhythm.

And then moving on to her echo. First starting with the left-hand side with the apical four-chamber view, as has been, you know, alluded to in prior talks throughout the day, the echo is a very, very important screening tool in our patients with pulmonary hypertension, gives us a wealth of information. So what we see here is normal RV size and function. It's a nice normal crescent shape to the RV. Just eyeballing the base to apex ratio, this is less than 1 which is normal, the right atrium appears normal in size. We can also look at the

base to apex ratio, the RV should be a normal sort of crescent-shaped size. So really, based on, at least on this image, we don't see evidence of significant pre-capillary disease or high PVR.

And then on the right hand, it might be a little bit harder to see with the way it's projecting but this is a short axis view at the level of the PAT muscles where we'll want to look at the contour of the interventricular septum and look for any flattening, particularly in systole. And we see a nice normal sort of round doughnut shape to the LV, no evidence of septal flattening.

Moving forward through our assessment of the echocardiogram, we see that based on TAPSE and the tissue Doppler with the S prime, both are normal, which are different components that we look at in terms of assessing RV function. And then this is the RVOT T Doppler on pulse wave and we see a nice normal triangular profile for our patient. And so, this was again also discussed in a previous talk but thinking about what is the importance of the RVOT T Doppler when we're trying to characterize our patients as to whether they have a high PVR state likely or not based on echocardiogram. So this is data that was looking at whether there was the presence of a notch and then whether it was happening in midsystole, or late systole. And so what the notch is, is that there's forward blood flow exiting out of the RVOT branching out into the smaller blood vessels of the pulmonary arterial vascular space. As it does that, there's a normal wave reflection. In a patient who does not have elevated PVR, typically, this wave reflection is small in magnitude, and it happens late. So we really don't see any interruption in the flow through the RVOT Doppler.

Contrast that with a patient who does have elevated pulmonary vascular resistance, blood is going to be flowing forward, and we get this early and larger in magnitude reflected wave that's like, basically impacting or colliding with the forward flow. This leads to a transient stop and flow or that notch pattern. And then also important to realize is that where we see the notch within that RVOT Doppler profile can also suggest whether the PVR is actually greater than 5 potentially with a good specificity and positive predictive value, or over 3 if it's a later systolic notch. So basically thinking that the earlier the notch is happening, midsystole versus late systole, suggesting a higher PVR state.

So this is a measure of the tricuspid regurgitant velocity. And so, this patient does have a nice envelope. But again, as was discussed earlier today, as well, many of our patients, unfortunately, do not have good estimations of the tricuspid regurgitation velocity envelope. When you're traditionally estimating PASP, we're plugging this into the modified Bernoulli equation, 4V2, and then you're adding that to the estimated right atrial pressure. And so, you can see now understanding that equation that any small errors in measurement can lead to over or underestimation. And this has been well documented in studies comparing estimated PASP on echo versus invasively confirmed hemodynamics on the order of 10 or 20 mmHg. So I think it was a great move for the guidelines to move away from estimate PASP and really focus also just on the TR absolute velocity itself, when you're using an echo assessment of whether a patient has PH, so you can get rid of that potential to amplify error in measurement.

And then what we see here is the mitral inflow, and then we see tissue Doppler. And so, when we look at that E/E prime ratio, that's a non-invasive way that we can assess elevations in left atrial pressure. And so we see a value of between 9 and 10 for our patient.

So taking all of this data into account, so we have a patient who started off, you know, presenting as many of our patients do, with potential risk factors for a wide variety of etiologies of pulmonary hypertension. They had a PE and DVT history, have sleep disordered breathing on CPAP, obesity, hypertension, and then this initial questionable diagnosis of connective tissue disease that seem to not be accurate. We then have our echo which shows normal RV size and function, normal right atrial size, no septal flattening, normal RVOT Doppler. So, so far, all of our imaging is suggesting that this patient perhaps does not have a high PVR state, does not actually have significant pre-capillary disease. What we do want to think about is could they have left-sided heart disease that is leading to their symptoms, and perhaps suggesting why they have not improved on sildenafil?

So this is one risk score that we can use to estimate the likelihood non-invasively of whether a patient has HFpEF. And so, this includes variables such as elevated BMI, whether they need hypertension with multiple medications – hypertension treatment, excuse me, with multiple medications. They do look at the Doppler estimated PASP, and then again, assessment of filling pressure along with age and whether atrial fibrillation is present or absent.

Our patient was noted to have a HFpEF score of 5, suggesting a higher likelihood. But again with a patient who was especially already put on sildenafil, given this diagnosis of pulmonary hypertension, it's important as always, to confirm what are the actual invasive hemodynamics. We saw that the right atrial pressure was 9, the PA pressure was 18 for a mean A wave with a V wave up to 30, normal PA Sat index and output, and we see here a normal PVR at 1.3.

So at this point, we conclude that the patient's diagnosis is in fact HFpEF, and is not someone who we think is going to benefit from sildenafil. So sildenafil discontinued. She's referred to our HFpEF clinic. She's continued on spironolactone, which had already been started by the prior institution. We started her on SGLT2 inhibitor therapy, which we'll hear about more in the next talk about the effects that it has in terms of reducing heart failure hospitalization for our patients and having some benefits with decongestion. And then also,

there's a lot of different clinical trials happening in the HFpEF space.

At that time, she was referred for assessment, whether she could be included in the STEP-HFpEF trial, looking at semaglutide in HFpEF, knowing that the obesity phenotype is a really large driver of HFpEF in a lot of our patients.

And so, take home points, our echo is a very powerful tool in terms of assessment of pulmonary hypertension, including patients with HFpEF PH. But we want to make sure again, as was emphasized earlier in this conference, that we're really moving beyond just the estimated PASP, looking at a lot of these other markers as highlighted here. And it was introduced in the, you know, the 2022 European guidelines, saying, does this patient really have evidence of significant pre-capillary disease? Or perhaps do they have evidence of significant left heart disease? And thinking that, you know, the hemodynamics that we get should really fit the echo picture. So making sure are we getting accurate assessments of our left-sided filling pressure is super important in our patients, especially with pulmonary hypertension. And then again, right heart cath remains the gold standard, not always needed if someone clearly has, you know, HFpEF, HFrEF, severe MR, and you know that the focus is really going to be on treatment of group 2 disease. But if we're unsure our patient has multiple predisposing risk factors based on echo and non-invasive testing alone, we don't know, we must always confirm what their hemodynamic profile is. And then generally speaking, pulmonary vasodilator therapy lacks benefit and may lead to worsening actually in our patients with group 2 disease.

Announcer:
You have been listening to CME on ReachMD. This activity is jointly provided by Global Learning Collaborative (GLC) and TotalCME, LLC. and is part of our MinuteCE curriculum.

To receive your free CME credit, or to download this activity, go to ReachMD.com/CME. Thank you for listening.