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

Balloon Pulmonary Angioplasty: Issues and Perspectives

Announcer:
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Dr. Schimmel:
We're going to talk about chronic thromboembolic pulmonary hypertension and balloon pulmonary angioplasty. But, although Dr. Haft is going to be talking about the surgical treatment of this disease, I do have to just make reference to it.

Here is a pulmonary angiogram. And you can see on the left, definitively proximal disease, on the right, there's also proximal disease, but also involves some distal disease as well. And so, as you think about balloon pulmonary angioplasty or pulmonary thromboendarterectomy, the surgical treatment for CTEPH, we really need to know about, one, the location of the disease, the experience level of the surgeon, and the experience level of the interventional cardiologist.

Because the PTE, the success is very dependent on the location of the thromboemboli and the center experience, the expertise, you know, historically speaking, surgery can have a high mortality, morbidity in this space, unless you're doing a relatively high volume. When you look at balloon pulmonary angioplasty getting in the distal vasculature, I can only get there if the central area is clear and if it's obstructed. If it's not obstructive, I can snake through there and maybe do something and decrease pulmonary vascular resistance. But this is what it would look like if I was to do a pulmonary angioplasty. There's a catheter that sits kind of coming at the top of the screen injecting some contrast dye, you can see a ring-like lesion there, there's some diffuse and mural thrombi, you can't see them all on the angiogram, you can see kind of mural irregularities in some patients. Sometimes it's more defined. I'm going to show a number of pulmonary angiograms in this 10 minutes, but this would be an example of an angioplasty result in the middle image there, showing dilation of that ring lesion. On the right, coming from a paper by a Ogawa, this is pathologic specimen where you can see what a web might look like where blood would move through those central spaces with the red and webs fibrous tissue, old clot that's become kind of white, hard, and scarred, is creating disrupted flow, higher pressures for the RV and the pulmonary hypertension we experience. When we send our wires through these lesions, we're finding microchannels in some of these patients, and when we do angioplasty, we expand that central lumen, and we get better flow.

Here on the left side, we have some very distal disease. So this patient did not have very central disease. And you can see up there and the left-hand side of the screen, let's see if my pointer works here. Out in this area, there's a deficit of flow. You can't really see a ring lesion here, but this would be very difficult for a surgeon, say, to get out to. We have a fairly inline flow here, we've got good perfusion here, good profusion here. And it's not to say the surgeon goes in and tries to get forceps into this area. Oftentimes they start a dissection plane centrally and then kind of dissect down and pull into as many segments as they can. But if there's not significant disease proximately it's going to be very difficult to get to lesions out in this space.

In the picture on the right here, you can see after balloon angioplasty, a couple of things. One, we have blush of the vessels here, the parenchyma, again nice blush here. And then we see this wonderful venous return of the vessel we didn't see on the prior angiogram.

In addition to angiogram, which isn't perfect, particularly in larger vessels or when the vessels are somewhat irregular; this has been

published in from a couple of different places, most recently Temple using fractional flow wires to kind of look at the pressure distal to a lesion and proximal. Here, there's a bit of a ring lesion this arrow is identifying. It doesn't look particularly significant, but you can see a pressure difference here there's a drop of at least 60% in the pressure pre and post which correlates to flow. Doing angioplasty or the 4-mm balloon and we can see that the difference in pressure has decreased significantly, and angiographically, it looks great, but it's still not what I would consider normal. And although we really don't have an understanding of what normal truly is, at least in the coronary literature, we use a value of 0.8 as a cutoff for FFR. And so, after a larger balloon, a 6-minute balloon, you can see there's not a whole lot of visual difference, but we do get a significant normalization of the pressures pre and post lesion. And so, this can be used as an adjunct as can other coronary tools. Many of the things that we use in balloon pulmonary angioplasty are taken from our coronary space.

So I'm going to show a couple of slides and it looks like these might have been my old slides but that's okay I can do with the old lecture is that there's a - we have a 44-year-old woman with multiple sclerosis and a history of remote DVT admitted for acute-on-chronic dyspnea and newly discovered hypoxia. You can see there on the V/Q And on the right that she's got multiple defects there. She's taken to a hospital has failed catheter thrombolysis and suction embolectomy, which was performed for abnormal vital signs. Sometimes my fellows will say, 'Oh, the patient's stable,' but what they really mean is they're not in shock and hypotensive, and I'd say this patient's not necessarily stable. You know, they have RV findings of severe dilation and dysfunction, and their vital signs are abnormal. She gets transferred to another center where they try and do another catheter-directed therapy, but it's unsuccessful and ultimately gets transferred to Northwestern for evaluation of likely CTEPH.

And she's this young patient. You can see here on a pulmonary angiogram, she has very central defects. There's kind of a cutoff here with a large perfusion defect in the back from this LAO view, you can see large perfusion defects, same thing all across the lungs. This would be one of the situations where I'd say well, you know, even though I can see some vessels out here, this is not an appropriate patient for balloon pulmonary angioplasty for me. She's young, she's a good surgical candidate, very central disease, it's unlikely I'm going to be able to make a huge difference, particularly with that central thrombotic disease. And so she goes for surgery. You can see the large central thrombus that's removed, and the goal for the surgeon is to get these very long tails. But we're not successful in all cases after surgery, getting those long tails, or sometimes the patient isn't a good surgical candidate.

So this is a second case. This is a different person, 80 years old, remote PE, presents with dizziness and recurrent syncope. Her RV is dilated, her pulmonary pressure is very high, of course, and you can see her invasive hemodynamics there with a PVR that ends up being close to 2000. I mean, it's tremendous.

Her cardiac index is 1.3. She's older. And when you take her for a pulmonary angiogram, you can see here that she's very dilated proximally. There's no frank occlusion, there's probably a mural thrombus there. She can't do my typical digital subtraction angiogram because she can't hold her breath, she can't get a nice static image. But you can see here nevertheless, without that perfusion aspect in the imaging, that she has rapid tapering of her vessels into the periphery and not much blush of the parenchyma. And so, it's decided that she's a little small, she's frail, she's older, this might not be a great patient to undergo pulmonary thromboendarterectomy.

And so here you can see a picture of selective engagement of one of her occlusions and trying to get a wire through that occlusion and you can get restoration of flow down the side following the wire here. You know, after four sessions, we were able to drop her RA pressure to 10. Obviously, these aren't normal, a mean PA is still 57 a wedge 11. And we're able to decrease her PVR to 1378 and get a better cardiac output. This obviously is not restoring someone to normal, but it's taking an older patient and making her functional. She no longer had syncope in the bathroom every time she took a shower, and symptomatically did much better.

And the question is always, I mean with balloon pulmonary angioplasty, it's how many sessions do you do? How far do you keep going? At what point is there diminishing returns? And I will say every session for her we had hemoptysis as well. So, you know, there are risk factors for hemoptysis during balloon pulmonary angioplasty, which is the most common complication. And I would say older age frailty, and then angulated vessels has been published as one of the other criteria that result in hemoptysis. And for her, given the recurrent hemoptysis, her age, and the fact that her primary symptom had resolved, we decided to stop here and treat her medically.

BPA has taken off as a focus of interest. I did this kind of PubMed review looking at surgical papers. And this is a combination of 1980 to 2000, you can see lots of surgery papers, there are a couple of BPA papers. And there are some deaths and hemoptysis that were concerning interventionalists. And all sudden, there's not a BPA paper at all until we get to 2012 to 2014. And 2014 is really when a lot of PE papers came out related to catheter-directed lysis the PEITHO trial came out, all of a sudden it resurrected interest within the role of cardiology and pulmonology. And so then we had all these papers come out in BPA.

So this is that same patient that I showed earlier, they had the very distal disease that might not be accessible to a surgeon. So case 1 was a good surgical case. Case 2 is a patient is older, not a good surgical candidate, although might have been good anatomically for surgery, and this is our patient who is a young patient but the disease may not be accessible to a surgeon.

And the fourth case I just want to show is that we can get pretty adventurous in the coronary arteries. This is a relatively younger patient who underwent pulmonary thromboendarterectomy but still had a total occlusion of her of one of her segments in her right lower lobe. And we can use tools just like we do, so I do chronic totally coronary occlusion work, and we can take our tools over into the pulmonary space and use different types of wires and microcatheters in order to try and find the vessels. Now in this picture, my wires found a relatively small vessel and I've used a microcatheter to do a light injection. I don't want to take a balloon, big balloon into that space, but we can use microcatheters that have dual lumens in order to try and find the appropriate space. We can do even bifurcation angioplasty and then grab large segments of vasculature back that otherwise were occluded. And so case 4 is a patient who had PTE, but had residual distal disease, and that too, is a patient who may be best treated with balloon pulmonary angioplasty.

What are the mechanics? Using fairly cheap and standard tools. We can use a short sheath from the groin. I tend to use kind of a 9-French short sheath with the long 70- to 90-cm sheath telescoped through it. We use typical angioplasty guides that we use in the coronaries. I use a lot of DSA and fluoro. And I'll mix my contrast with 50/50 to keep my contrast levels down. And then we kind of minimize time off of anticoagulation, I resume it very quickly. If there's been no hemoptysis and it's a venous stick, a lot of times I just restart anticoagulation after the procedure.

I brought up to hemoptysis a couple of times now, and hemoptysis is one of the feared complications in the literature. They talk about reperfusion injury being as high as 30%, using CT data to kind of look screening wise. But clinically, it's probably, at least for us, it's been about 5% of our patients, and it may be 5 to 10 depending on the center.

This is, you know, published out of French single-center angioplasty outcomes showing that we can have patients before angioplasty who sit in the Class III/IV heart failure range, and then get ourselves down into Class I to II range. And so we can get a lot of benefit and symptoms increase in our 6-minute walk test, and we can keep our mortality less than 4, and after doing just under 200 angioplasties, I can say that our mortality rate has been 0. But again, our hemoptysis rate is about 5%.

BPA after PTE, like the last patient that I showed, can be a nice accessory to the patient after PTE, if they have residual pulmonary hypertension, or disease. And even though all these BPA papers have come out and more centers are doing them, there really hasn't been a decrease in PTE. If anything, it's bringing more people to the table to be evaluated properly by a surgeon so they can undergo potential curative therapy.

And I'll kind of scoop through this real quick. But essentially, it's saying is this patient technically operable? Are they a good surgical candidate? And if they are PTE is a standard therapy. Where I struggle a little bit is with the targeted drug therapy for people who aren't surgical, should they go on to riociguat and sit and see what their symptoms are like? Or should we go on to balloon pulmonary angioplasty first? Or pull the trigger on both at the same time?

A couple outstanding questions in the universe for CTEPH with distal disease: Is BPA a treatment pathway for patients with CTEPH who do not have pulmonary hypertension? That CTED that Ruben talked about earlier? If you have 1 to 2 segmental disease with a CPET that shows a proven vascular limitation but they don't have, you know, high pulmonary pressures, maybe that's a patient who does better with balloon pulmonary angioplasty. Or maybe if they have multiple segments, even though they don't have CTEPH, maybe they do better with surgery. And then should medicine before angioplasty always be the case? Does it decrease the risk of the angioplasty? Or should they be initiated together? And are there situations where BPA should be done to decrease PVR to potentially decrease the risk of PTE for distal disease?

And so, those are some of the outstanding questions that I have. And I look forward to Dr. Haft's lecture on surgery and maybe we can discuss these questions afterwards. Thank you.

Announcer:

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