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Released: 12/13/2019
Valid until: 12/12/2020
Time needed to complete: 15 Minutes

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Heart-to-Heart Clinical Intensive: Considerations for Rhythm Control Post CABANA

Dr. Miller:
All right, so this is a case of a 58-year-old man. He's hypertensive and has persistent atrial fibrillation and some fatigue. He's had well-controlled hypertension. Atrial fibrillation has been present for a year, and now he comes in and says fatigue. His wife says he snores like a foghorn on steroids. She can't even be in the same room with him. He clearly has markers of sleep apnea. He's drowsy and takes naps during the day. He used to golf a lot and walked with his clubs on the course. Now he only uses the cart due to fatigue, can't make it around that well. He's on ramipril, metoprolol, hydrochlorothiazide, well-controlled hypertension. His BMI is pretty average nowadays. Blood pressure is controlled. Heart rate is 90 and irregular, otherwise reasonably normal physical examination. ECG shows atrial fibrillation with a ventricular response around 95 and some nonspecific ST and T wave abnormalities. He has a lab panel. His BNP and thyroid panel is normal. Liver function test slightly elevated, maybe a little bit of alcohol in the mix. His echocardiogram, not the best windows with his obesity, but his ejection fraction looks pretty good, a little bit of diastolic dysfunction. A little bit more testing, has CPAP [sic] really bad—has sleep apnea, and he's recommended for CPAP.

Now, this guy has I don't know how many choices you want as to why he's fatigued. A lot of our patients come in like this. We have some people who have a zillion different things wrong with them, but they are pinning the tail on the donkey that it's the atrial fibrillation. "If only I weren't in atrial fibrillation I'd feel better." Well, maybe and maybe not. Is it the atrial fib, diastolic heart failure, obstructive sleep apnea, a combination of the above, just that he's older than he used to be?

What should we do about his atrial fibrillation? Well, the first thing is try to figure out why he has it in the first place, if there is some easily reversible thing, mitral stenosis; he drinks too much caffeine, alcohol and so forth; those should be screened out. The second thing is assess the stroke risk in this individual and also their bleeding risk. We're required nowadays to have each of those statements in each of our outpatient characterizations nowadays, and these are not static numbers. CHADS-VASC increases in a (inaudible)*1:15:46 effect. Once you have had a stroke, you don't un-have a stroke, and you get 2 points for that. And once you pass 65 and 75, you don't walk that way back either. So we assess their stroke risk and figure out the best means of prevention for them and then think about a cardioversion as both a therapeutic and a diagnostic tool.

As has been mentioned before, do a cardioversion, see if they feel better. And some patients will have the cardioversion, and, as Dr. Epstein said, they will relapse. Somewhere along the way you'll see them in 6 weeks, 2 months later, and they're back in atrial fibrillation. "How did you feel?" "I don't know. I didn't feel any different." You don't learn much from that unless you have some correlative measure of when they might have relapsed in atrial fibrillation, so the monitor along the way makes some sense with that. And some of us will say, "Well, let's get the most bang for the buck for that cardioversion." Instead of doing a cardioversion off medications, do a cardioversion only to find that they have relapsed and we didn't learn anything from that, so then we'll repeat it after loading with an antiarrhythmic agent. Why didn't we just do that up front? And a good agent to use might be something that's going to have a reasonably good efficacy and reasonably good safety profile—maybe not in that order, safety first—such as dronedarone. For instance, you can initiate it as an outpatient. It's easy to remember the dose. It's not, perhaps, as effective as amiodarone, but it's reasonably effective and easy to take, and you stop it if it doesn't work, no harm no foul.

And if you try antiarrhythmic drugs, which one? How long do you give it to work? How long do you load? Will control of obstructive

sleep apnea be the ticket, or is it just an ingredient that you shouldn't miss the opportunity to treat? And are there other things we ought to deal with?

Dr. Singh:
John, if I may, one of the things you mentioned on the echo was that there were poor echo windows.

Dr. Miller:
Yes.

Dr. Singh:
One thing I would really look for, what the left atrial size is.

Dr. Miller:
Okay.

Dr. Singh:
And if the left atrial size is on the bigger size—and we can get into those details—the potential that this has been going on for a long period of time is there. I mean, the atrium is fairly remodeled, and if the atrium is fairly remodeled, a cardioversion is not going to get them back into normal sinus rhythm for more than a few hours or a day or 2 potentially, so in that circumstance I would be more inclined to actually talk to them about an antiarrhythmic strategy and bringing them in or starting as outpatient, either of the 2, and then cardioverting the patient with that on board, but at the same time work on all the other aspects, as you have said, with the sleep apnea, the weight, the high blood pressure and all the other things.

Dr. Miller:
Yeah.

Dr. Mittal:
John, I'm going to ask you a question about this patient because I actually—I see patients like this, and I find this to be somewhat difficult, and what I'm going to pose for you is the scenario… like many of us, we've cardioverted the patient, and I'm going to be more optimistic than Jag and I'm going to let this guy have a month in sinus rhythm, and he goes a month in sinus rhythm and he feels no different. You're watching him, and 6 or 8 weeks later he comes back to you because now he's back in persistent AFib, and now you're left with a dilemma that you have a 58-year-old male with persistent AFib with a CHADS-VASC of 1. And so the 2 issues that come up are anticoagulation or not—because we see this a lot in our community in the US. I think our European colleagues are much more aggressive about anticoagulation. Our guidelines are still a little bit on the fence, aspirin or anticoagulation, and I know there is this uncertainty about what to do. And also, this whole issue of the decisions you make today, of course, are going to impact this patient for years down the road.

Dr. Miller:
Yes.

Dr. Mittal:
I'm sure you battle similar issues. What's your approach to those 2 issues in a patient like this?

Dr. Miller:
Yeah, there is a lot in there. These are just real-life questions that we have to address in our 20-minute outpatient visits, right? The first question about he has been in sinus rhythm for 4 weeks, doesn't feel any different, then you see him at 6 or 8 weeks and he's back in atrial fib and he still doesn't feel any differently, that person is probably not going to benefit from sinus rhythm, probably not, although we know that the reverse remodeling and the restoration of mechanical function of the atrium can take up to 8 weeks. The longer it's been in atrial fibrillation the longer it seems to take. Although, you have some people that you cardiovert and they wake up and they think, "Wow, this is just fantastic." They are going from the get-go. So this individual has got 1½ strikes against him probably in that scenario, but it's not a definitive answer as to whether he's going to benefit from sinus, and I would say let's give you... If we're at a fork in the road here, pursue sinus rhythm or abandon that hope. At 58 years of age, as far as I'm concerned, this is a young man still, and I don't want to consign him to a life of atrial fibrillation and then some 10 years from now he develops mitral regurg, needs to have his atrial kick. And the horse has left the barn. We had our shot back at 19. We didn't take it, and now he's left with this stuff.

Andy, did you have a comment?

Dr. Epstein:
Yeah, I was going to say 2 things, 1 about anticoagulation. He's got a CHADS score of 1, but we know from the NOAC trials that people who were in those had a benefit for survival and decreased thromboembolic stroke being anticoagulated, so I think a CHADS score of 1

in my mind I anticoagulate these people and recommend that. I don't know what the others feel, but I see nodding.

Dr. Miller:
I sure promote that as well.

Dr. Epstein:
And that's number one. Number 2 is a lot of people talk about HAS-BLED, and in response to that, as the CHADS-VASC score goes up —

Dr. Miller:
Same, same factors.

Dr. Epstein:
—HAS-BLED score goes up. Furthermore, as both go up, there is greater benefit from anticoagulation. And I like to think of the HAS-BLED score not as something that I use to not anticoagulate, but rather it raises my antennae to say I need to be more careful with that patient, pick drugs that don't have interactions, and it's a warning. It doesn't say don't anticoagulate. As I said before, I tilt towards anticoagulation whenever I can.

Now, that being said, when you were talking right before you went back to the podium, you talked about people who feel better when they are in sinus. The converse is, there are people who get AF and feel no different. And there's old work from... One of the device companies made a pacemaker called the AT500 that you may remember, and this was put in for people with atrial fibrillation, and it could pace people out of atrial flutters and some AFs. Everybody who got this put in, because it was a transvenous lead, had highly symptomatic AF, and what was shown in this was that people had their first recurrence of AF after 3 months that could go over 48 hours and be asymptomatic, so I don't ever stop anticoagulation just because somebody says they are feeling better.

Dr. Singh:
One of the things I always do in these patients… Often times patients, when they are symptomatic or asymptomatic, they have adjusted their life around their symptoms and they feel better asymptomatic, so I have a very low threshold to actually put them on a treadmill, do some objective functional evaluation of what their heart rate response is. So the gentleman, you said the heart rate could be between 50 and 90 and the patient is asymptomatic, but you put them on the treadmill and the heart rate now is 180 means you need to do something about that heart rate or something for that patient also. I think having some objective evaluation of functional level is really important. And I think it's really important also to recognize that this gentleman is a 58-year-old, has another 30, 40 years ahead of him, with risk factors that are reversible, and there is a good chance that his atrial fibrillation potentially could be curable or significantly limited, so I would be rather aggressive—unless, of course, the atrial size of the echo as 6.2 centimeters; then I'd take a step back and say, okay, throw our hands up and just rate control him with good anticoagulation on a long-term basis. But if not, I would have a very low threshold to put him on antiarrhythmic, cardiovert him, see how he does, and if he's back in AFib, put him on a treadmill to really see what his functional status is truly and put those things together with lifestyle modification, as you said, before really signing off on the fact that AFib is yours for life.

Dr. Miller:
This is a very different management strategy than it was 15 or 20 years ago. It's very nuanced, very individualized, and I think our patients benefit from that. My mantra is, "when in doubt, thin it out."

Dr. Singh:
I like that, "when in doubt, thin it out."

Dr. Miller:
If you've got any... I look for a reason to anticoagulate.

Dr. Singh:
His BMI was 35, so he needs to thin it out.

Dr. Miller:
True, in many ways. Yeah, to give you a quick anecdote, I had a hematology colleague in Indiana who, after the New England Journal papers came out about aspirin, no benefit in cardiovascular prophylaxis—he was on aspirin, and he was 70 years old, no other risk factors—he wanted my permission to come off aspirin. He e-mailed me one Friday morning. And I said, "Well, I was just thinking of going the opposite direction," because of some recent papers, "putting you on a NOAC," and he said, "Well, I'll think about it." That night he had a stroke. He was reperfused; everything is fine. Sunday night he e-mailed me saying, "I'm out of the hospital on a NOAC."

Dr. Miller:
And so that just reinforces your resolve to... These medications are so safe and so effective nowadays. It's just you really have to look hard for a reason to not do it, I think.

Dr. Epstein:
Could we just also say there is no place for aspirin or antiplatelet drugs as stroke prophylaxis in atrial fib? That is clear now from the literature. And in fact, the whole aspirin story was based on a study in the '70s, SPAF-III. Patients were randomized to aspirin, warfarin and placebo. There were 6 strokes in the warfarin group and 1 in the aspirin group, but this 1 study from 6 strokes drove the entire meta-analysis, which has led to the guidelines, and it is truly a type 2 error that 1 in 20 times you're going to get a mistake in the result. And so aspirin has got no place in this. You anticoagulate with a real anticoagulant.

Dr. Mittal:
So I want to be sensitive... Oh, question.

Dr. Singh
This patient, as you mentioned, had sleep apnea, right?

Dr. Miller:
Yes.

Dr. Singh:
Question was: What first drug you would pick up for this patient if you want to control his rhythm?

Dr. Miller:
That's an excellent question. I don't know that we have good evidence to pick one drug over another. You might want to have something that doesn't have so much rate slowing associated with it. Amiodarone would probably not be your best agent and sotalol would probably not be your best agent because they have decelerations at night as well, and everybody gets pretty upset if they have significant pauses, but I don't know that there's any good evidence to pick one agent over another.

Dr. Epstein:
You mentioned that his LFTs were a little bit up, and I think that certainly plays into the choice of drugs that affect the liver, but I think it also tells us that you have to get a good echo and see what the PA pressures might be, because if they've got pulmonary hypertension and right ventricular failure, you've got a bigger problem, and you need to treat that fluid retention also.

Dr. Mittal:
So, I want to thank Dr. Epstein, Dr. Miller, Dr. Singh in working with me on this presentation. Of course, most importantly, I want to thank you all for taking time out from your busy evenings to attend this presentation. Again, thank you for Sanofi for supporting the program and Medtelligence for handling the logistics so well. I wish you goodnight, safe travels home, and look forward to seeing you all at a future program. Thank you.

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