Miguel Leal, MD (00:16):

Hello, and welcome to the Hypertrophic Cardiomyopathy podcast on hypertrophic cardiomyopathy and atrial fibrillation. This is another one in a series of podcasts from the American Heart Association, a hypertrophic cardiomyopathy or HCM initiative, which is sponsored by Bristol Myers Squibb. I am Miguel Leal and I'm an associate professor of medicine at the University of Wisconsin, School of Medicine and Public Health. And I'm honored to be accompanied in this podcast by two colleagues and a patient who will be teaching us and explaining to us their experience when it comes to this complex introduction between atrial fibrillation and hypertrophic cardiomyopathy. The first person I'd like to introduce to our conversation today is Dr. Ethan Rowin. Dr. Rowin is the co-director of the Hypertrophic Cardiomyopathy Center, and also the director of cardiac MRI at Tufts Medical Center, United States. Joining us will also be Dr. Iacopo Olivotto, who is the head of the cardiomyopathy unit at the Careggi University Hospital in Florence, Italy.

Miguel Leal, MD (01:13):

And we also have the honor to be accompanied today by Ross Hadley, who will bring a unique patient perspective. He is a 49-year-old man who lives in Iowa, United States, and he has a diagnosis of both atrial fibrillation and hypertrophic cardiomyopathy. And he will share with us his experience along his journey as a patient, we will start our podcast talking a little bit about atrial fibrillation in terms of what it is, what exactly the natural history of the disease tends to be, and its major and multiple clinical consequences. So let's start with Dr. Rowin, if you could explain to us, what exactly is this so common and so complex arrhythmia called atrial fibrillation?

Ethan Rowin, MD (01:53):

So atrial fibrillation, abnormal heart rhythm, which is common in general as individuals age in the general population, but it is much more common within HCM. In fact, it's the most common sustained arrhythmia in this disease. About one in four individuals will develop a-fib during their lifetime with HCM. And a-fib is about four to six times more higher in HCMs compared to an age and gender match population. While in HCM, a-fib also occurs more commonly as people age. It's notable that it can occur at a young age in HCM patients and about 5% of patients with a-fib and HCM develop a-fib at under the age of 30. So really common in the disease and really important in the disease, as we're going to talk about.

Miguel Leal, MD (02:50):

Thank you, Dr. Rowin. Dr. Olivotto, tell us a little bit about your experience treating atrial fibrillation, in terms of how common it is in your clinical practice and what are common challenges you find when you're trying to individualize care for patients with a-fib?

Iacopo Olivotto, MD (03:01):

Well, first of all, atrial fibrillation, as Ethan was saying, is an extremely common problem. Usually in the context of hypertrophic cardiomyopathy, we see atrial fibrillation in two different clinical contexts. One is in patients with obstructive disease, whereby obstruction also caused by SAM, systolic anterior motion of the mitral valve also causes mitral regurgitation and atrial stretch leading to progressive dilatation of the atrium, which eventually supports and triggers atrial fibrillation, paroxysmal, and then permanent. And that is definitely an issue for treatment as we will probably discussed later on.

Iacopo Olivotto, MD (03:40):

The second scenario is patients who develop progressive disease and fibrosis of the left ventricle and proceed towards the pre-end stage or end stage phase so-called, as this phase where there is a progressive heart failure scenario, which is associated again with refractory. Sometimes there's systolic abnormalities, restrictive feeding patterns, again, high pressure within the atrial stretch. And again, atrial dilatation and atrial fibrillation, as a consequence.

Iacopo Olivotto, MD (04:14):

These two scenarios are very different in terms of management, in terms of the stage at which patients develop these complications and each of these should be targeted in a very specific and different manner. In both cases, however, it does represent a turning point because of course you need anticoagulation because symptoms are very often associated with the arrhythmia, and sometimes because the challenges with the treatment of the arrhythmia become the most important clinical problem for the patients.

Miguel Leal, MD (04:45):

Thank you Doctor Olivotto, that was very insightful as well. And as the AHA likes to do, and it's really one of the hallmarks of these podcasts, we have the pleasure to have a patient here with us today. Again, this is Mr. Ross Hadley, and he'll be talking to us about his experience. Mr. Hadley allowed me to share with us here that he has been diagnosed with atrial fibrillation, has previously undergone a catheter ablation procedure. He is currently on anticoagulation therapy, and some of his co-morbidities in addition to hypertrophic cardiomyopathy includes sleep apnea, for which he is being treated with MICA. So Mr. Hadley, thanks for joining us today. Tell us a little bit about your journey in terms of how these diagnoses were made and what they have represented to you as a patient so far.

Ross Hadley (05:26):

I went into my general practitioner five years ago with a complaints of chest pain and was thinking I was overstressed and probably drinking too much caffeine and whatnot. And he told me just to cut back on caffeine and try to relax, but it didn't work out so well. Ironically, because of my chest pain, I applied for life insurance, a substantial amount of life insurance, and in life insurance physical, they did an EKG and discovered the abnormalities in the EKG. After the EKG was done, the ironic thing is they only noticed t-wave abnormalities, not a-fib, so that I was issued the insurance, which is good. And I was referred back to my physician. He referred me to a cardiologist to undergo examination for atrial fibrillation. And then I was referred to Mayo Clinic, where I went up and was doing the prep work for my ablation and during the transesophageal echo, they discovered that I had apical hypertrophic cardiomyopathy.

Miguel Leal, MD (06:44):

Thank you, Ross. That's an interesting journey you described there, as we all know, atrial fibrillation sometimes can be frankly symptomatic, and sometimes it can be an incidental finding on a routine physical examination with a whole host of variety of presentations in between. Still, I appreciate you giving us a glimpse of your experience so far. So I'm going to turn back to our colleagues here and Dr. Rowin, one might be wondering, why is the AHA deciding or electing to have an entire podcast on a-fib an HCM? So there must be some good reasons for that. So tell us a little bit as somebody who treats both conditions, what makes a-fib, which is such a common arrhythmia as you and Dr. Olivotto said, what makes it so unique in the context of hypertrophic cardiomyopathy?

Ethan Rowin, MD (07:27):

Yeah. So one is patients with HCM and a-fib tend to be very symptomatic and it tends to be recurrent and they feel pretty miserable when they're in a-fib. So it's important in terms of treatment to be aggressive treatment. And for most patients, they really require a rhythm control strategy because of symptoms. And then also patients with HCM and a-fib are at high risk for embolic stroke. And this is not like the general population where CHA_2DS_2-VASc score can identify individuals with higher risk. All HCM patients are at increased risk for embolic stroke, with a-fib. And so a need for, again, treatment with systemic anticoagulation really at the time of the first AF episode.

Miguel Leal, MD (08:20):

Thank you. Dr. Olivotto, tell us a little bit about your experience with rhythm control. It seems like this is a condition that may require a more aggressive strategy in that direction. So what have you tried for your patients that have both a-fib and HCM?

Iacopo Olivotto, MD (08:36):

Absolutely. Of course, Mr. Hadley went for ablation as a first-line therapy as I gathered, if I understood correctly, but of course we do have drugs that have some efficacy on control of rhythm, ambitious with product SysML laser fibrillation. We can use class one agents, such disopyramide, which is commonly used for obstructive HCM, and that has some sort of efficacy in the initial phases, but lacks to achieve real control if the legislature, the modeling, has progressed. The most powerful drug we have now is Amiodarone. And that applies to any patient with atrial fibrillation, even outside of HCM. However, we know well that Amiodarone is a drug we don't use very happily in young patients, such as many HCM patients, because of the long-term consequences on the thyroid and potential toxicities in the eye, the lungs, and others. So I would say that our pharmacological options are limited and therefore to be able to progress to invasive treatment is definitely an attractive options.

Iacopo Olivotto, MD (09:40):

The problem with a catheter ablation in a-fib for HCM is that we have to consider that we have a genetically abnormal substrate in the atrium, as well as in the ventricle. And that the remodeling of the atrium is often very advanced compared to patients with lone atrial fibrillation, for example, or hypertensive patients with a-fib. So that the recurrence of atrial fibrillation, even after a very well-conducted ablation is common, we have to expect a higher rate of failures and higher rate of reduce, as compared to patients with atrial fibrillation outside the context of structural heart disease.

Iacopo Olivotto, MD (10:19):

And then of course we have surgical options. If the patient has to undergo myectomy or other surgical procedures, one may add surgical maze for control of rhythm. That seems to work in good hands. If you do the real maze procedure, does seem, I mean, there are these different tasks and maybe Ethan can expand on that, that seem to be very promising. Although not many surgeons actually do the procedure as it should be done. So ultimately, we do have weapons to achieve rhythm control. Again, provided the left atrium has not remodeled too extensively to allow any of this.

Miguel Leal, MD (10:57):

Dr. Rowin, how about your thoughts on this topic?

Ethan Rowin, MD (10:58):

Yeah, I agree with pretty much everything Dr. Olivotto had said. I will say that in our experience we do use Sotalol pretty commonly for our obstructive patients. We do require them to be admitted as an inpatient for it, for initiation, but we've had very low rates of adverse events in that scenario, but without obvious diligent monitoring for QT prolongation. And the Cleveland clinic has this series where they also looked at using Dofetilide and have shown that in a small subset of patients, pretty safe as well. But other than that, besides Disopyramide and Amiodarone as Dr. Olivotto said, that's really what you're limited to from an antirrhythmic perspective.

Ethan Rowin, MD (11:47):

Myectomy maze procedure, which our surgeon, Dr. Hassan Rastagar routinely does for our patients with a-fib, either paroxysmal or persistent at the time of surgical myectomy, does seem to really offer patients a benefit in terms of about three quarters remain free from a-fib at five years after that procedure. So it really can impact the natural history of a-fib while also improving heart failure symptoms for obstruction. But we are aggressive in treatment, meaning we do the procedure early again with the goal of avoiding extensive left atrial or modeling long-term.

Miguel Leal, MD (12:29):

Thank you both. I think a very significant important theme that we're getting from today's discussion is that that is a time sensitive components of treating a-fib and HCM, perhaps more than the average a-fib patient out there. And we all know that once you cross the net or you start a-fib from paroxysmal disease to persistence, and then to long-standing persistent, the probability of cure or adequate control goes down significantly over time.

Miguel Leal, MD (12:54):

So along those lines, Dr. Olivotto, in your experience when you have to pull the catheter ablation trigger, when you tried medical therapy and the patient has proven refractory to a medication or another, do you have a specific strategy in mind such as radio frequency ablation or cryoballoon, or is there anything about the procedure that you manage differently? Or do treat these patients as you would treat your traditional a-fib patient?

Iacopo Olivotto, MD (13:19):

First of all, we usually have a threshold of approximately 350 cc's for left, non-index, left atrial dimension as the, this due to our retrospective analysis of our series. About that threshold, we consider the procedure to be quite unlikely to succeed. Below that threshold, we have used both. We can use cryo, or you can use radio frequency. We have also tried in the early days to do additional lines besides the inserting of Armani arteries in the veins, also add lines to the roof of the atrium. That has not really turned out to be very useful. So, we now stick to the strickling or the permanently veins. It's usually done by razor frequency, but we can do is cryo as well. I don't think that the results are terribly different. In very few cases, and really it's talking about very few cases where patients who are really rendered very sick from a-fib, young, active patients, we have also resolved to hybrid procedures, including catheter ablation, plus thoracic surgery for AB cardio, or sort of completion of the ablation. But then again, that is quite a rare and aggressive indication.

Miguel Leal, MD (14:32):

Thank you. And Dr. Rowin, tell us a little bit about your anticoagulation strategies. You were mentioning that there's this specific combination where you cannot unfortunately use your CHA_2DS_2-VASc score as

your helper here because we're treating a very different entity. So we have Warfarin, we have the direct oral anticoagulants, and now we also have left atrial appendage closure. So how do you go about that choice?

Ethan Rowin, MD (14:54):

You know, for most of our patients, they really choose the novel DOACs for treatment. Rarely will we use Coumadin or Warfarin at this stage. And the data really seems to be that the DOACs are very effective in preventing stroke in HCM patients. Definitely have some concern in terms of left atrial appendage occlusion as a sole mechanism for preventing stroke in HCM. I don't believe there's any data specific to HCM and I'm interested certainly in Dr. Olivotto's opinion as well. However, in my opinion, there's probably an underlying left atrial myopathy in this disease, and I'm concerned about source of stroke outside of the left atrial appendage. So we really don't use that as a strategy for our HCM patients. The very few times I've recommended it was when bleeding risks was super, super high when it was really that versus nothing.

Miguel Leal, MD (15:53):

Thank you. Dr. Olivotto, how about your thoughts on this topic?

Iacopo Olivotto, MD (15:56):

I agree entirely with Dr. Rowin. I have occasionally used left atrial appendage closure in patients who had stroke, despite, because unfortunately you do see that in HCM, you can have embolic stroke in patients with optimal anticoagulation. That is because of the issues they have with this. So in that case, I have recommended left atrial closure, but I have also continued anticoagulation. So into two procedure schools, therapists combined, and I would be very concerned in relying on that tissue appendage closure alone. Again, the only exception being when you're forced to it because of high bleeding risk.

Miguel Leal, MD (16:36):

Perfect. Thank you both. So Mr. Hadley, let me bring you back here. So we've heard a lot about treatment options about the adequate candidacy of a patient for one pathway or another. So tell us, where did you fit all of this? What kind of treatments have you received so far?

Ross Hadley (16:51):

Right out of the box, I did take Coumadin. It was difficult to control my INR. After a period of about six months, I did move to Apixaban. Unfortunately, as a solution, with my ablation not being successful, I am in a permanent a-fib now, which does have some effects of quality of life.

Miguel Leal, MD (17:12):

I also understand from your history that you have been diagnosed with sleep apnea, which is known to be a concomitant comorbidity that often is associated with atrial arrhythmias in general. And you have received treatment for that as well, right Mr. Hadley?

Ross Hadley (17:24):

Correct. I started with a CPAP and have moved up to a BiPap, which I find to be a real quality of life improvement.

Miguel Leal, MD (17:30):

Thank you for sharing that with us. So our intent today was essentially to bring an outline of what is the latest and the greatest ways to treat patients with atrial fibrillation and hypertrophic cardiomyopathy. It is important to acknowledge that there are medical therapy options available as both Dr. Olivotto And Dr. Rowin mentioned, and that are invasive management strategies, which involve both percutaneous and surgical procedures as well, which we discussed today. I would like to ask our faculty and Mr. Hadley to share any final thoughts that they may have before we put our closing statement on.

Ross Hadley (18:03):

Thank you for sharing the knowledge that you all have with the medical and patient community just to better everyone's lives. I certainly appreciate it.

Iacopo Olivotto, MD (18:12):

I think that this podcast highlights major unmet needs in this disease, including the development of better anti-arrhythmic agents. We're still using agents that are incredibly old, are still effective, but not as effective as we would like. And we still don't know how to tackle cases as Mr. Hadley's case where you can't get rid of atrial fibrillation, you have to adjust for a rate control. And this is often associated with, again, worsening of quality of life. And we need to understand how to improve our patient's life when this occurs. So thank you again.

Ethan Rowin, MD (18:50):

Thank you also for having me. Completely agree with Dr. Olivotto that really one of the unmet needs in HCM is refractory a-fib are current treatments. You know, for most patients though, with a-fib and HCM, we really do have pretty good treatment options to control a-fib and quality of life. I think in the future, early detection of a-fib is going to even further improve our treatment options with early treatment. On that note, we recently created a risk scoring tool to identify patients who were at risk for a-fib and potentially with the use wearable technology, we can identify HCM patients with a-fib earlier in their course.

Miguel Leal, MD (19:37):

Thank you again. This podcast is a part of the American Heart Association HCM initiative, which is sponsored by Bristol Myers Squibb. In closing, I'd like to remind all of us who are listening to encourage our patients to play an important and active role in your medical care, by advocating for themselves and their family members. Just like we heard today from Mr. Hadley, it is important sometimes to share your personal journey so that we all can learn and hopefully do better. As we just understood, there are many gaps that hopefully scientific research will advance our fund of knowledge as time goes by. You can also visit the American Heart Association hypertrophic cardiomyopathy patient website for more education. Thanks again everyone. And until the next one.