

Ferhaan Ahmad:

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Hello. I'm Ferhaan Ahmad. I am an associate professor in the division of Cardiovascular Medicine at the University of Iowa, Carver College of Medicine. I'm also director of the cardiovascular genetics program and the hypertrophic cardiomyopathy center of excellence at the University of Iowa Healthcare System. It is truly an honor for me to have the opportunity to host a podcast today on a really interesting topic. Advanced surgical approaches and emerging medical therapies for HCM. And to help walk us through these topics, we have an exciting panel of experts who will discuss these different areas. And we have today joining us, Dr. Ahmet Kilic, who is a cardiac surgeon and an associate professor of surgery at Johns Hopkins University School of Medicine in Baltimore. He also serves as the director of the heart transplantation and mechanical circulatory support program there.

And Dr. Anjali Tiku Owens, who is a heart failure cardiologist and an assistant professor of medicine at the University of Pennsylvania in Philadelphia. She also serves there as the medical director of the center for inherited cardiac disease. And finally, we have Ms. Ovukeye McCoy, who is a patient with HCM. And unfortunately, a member of a family that's been afflicted by a lot of HCM and underwent a heart transplant in 2018. And she lives in Alabama. The HCM podcast is sponsored by Bristol Myers Squibb. So with that, hello everyone. Thank you for joining us.

I think we'll start by talking a little bit about surgical approaches to HCM beyond the standard, which is surgical septal myectomy for patients that have obstructive forms of HCM. But there's often situations where patients have undergone some kind of septal reduction therapy and they're still running into trouble. And that's often a very challenging problem for us. And there's of course, other patients that are not obstructed, but still are running into issues that are intractable to medical therapy. So, I think I'll probably start with you Dr. Owens and ask you, what are these patients that you end up seeing that you have to start thinking you're going to send to Dr. Kilic?

Anjali Owens:

[02:38](#)

Great. Thank you. So, we frequently see patients with HCM referred at a late stage to our center and then ultimately even to our transplant program. I would say that in the community, the warning signs clinically of patients who are reaching sort of an advanced stage or refractory stage, include increasing burden of atrial and ventricular arrhythmias. So, when you start to see that in your patients be wary that they may be advancing to end-stage, increasing diuretic requirements and intolerance of their negative inotropic agents which they've been on for

many years are other sorts of warning signs that patients may be advancing. And, when we see patients with refractory symptoms despite maximally tolerated medicines, we do start to think about the potential need for heart transplant. And it comes really in three main subsets of patients. The first is patients who have regression of their hypertrophy over years and dilation of their LV cavity over time which results ultimately in systolic dysfunction. Looks like the more classic dilated cardiomyopathy.

Although we really should be starting to think about this when the LVEF drops below 50% in patients with HCM who normally have of course, a normal or even hyperdynamic ejection fraction. The second group is patients who have continued non-obstructive HCM and maintain either a relatively normal or small size LV cavity and hypertrophy, but they remodeled to more of a phenotype of restrictive cardiomyopathy often with by-atrial dilation, refractory heart failure symptoms despite a preserved ejection fraction. And the third subset of patients either develop intractable ventricular arrhythmias or less commonly ischemia.

Ferhaan Ahmad:

[04:33](#)

So for you, 50% ejection fraction is sort of the warning sign that things may have to progress to more advanced therapies?

Anjali Owens:

[04:42](#)

Absolutely. And I don't think you should wait until your patient with HCM, for example, has an LVEF of less than 35% or less than 25% or something that is more akin to what we look for in dilated forms of cardiomyopathy. By the time a hypertrophic heart gets to an LVEF that's consistently lower than 50%, those patients are really looking at trouble. And the next step in picking up how advanced they are is really looking at functional testing, which we frequently use serial VO<sub>2</sub> testing or CPETs to look at their exercise tolerance objectively over time to look for decline or the presence of prognostic features that are poor, like an abnormal or hypotensive blood pressure response to exercise, ventilatory inefficiency, which we can look at with the VE/VCO<sub>2</sub> slope that's elevated above 35 would be concerning. Or other sort of poor prognostic features that you can see on functional testing.

And the other piece of it is to consider at an early stage and I think which is probably underutilized in patients with HCM, is an invasive hemodynamic assessment with a right-heart catheterization. That gives you not only the important filling pressures by ventricular filling pressures but also critically assess for pulmonary hypertension. And we often see when disease has progressed significantly that we may be entering the stage

of irreversible pulmonary hypertension which will take them out of candidacy for heart transplant. And so, it's important to get that pulmonary pressure and PVR information and also of course their cardiac output.

Ferhaan Ahmad: [06:24](#) So that's interesting, we have certain standard criteria that we use for most patients that have heart failure with reduced ejection fraction that are going to be potentially candidates for transplant. But you pointed out that there are some differences between both patients and patients with HCM that are starting to have a reduced ejection fraction. And then those patients that actually don't have a normal ejection fraction or even super abnormal ejection fraction but have a restrictive physiology that might be candidates. Do you use the same criteria to assess them for candidacy for transplant or do you modify them in any way?

Anjali Owens: [07:02](#) I think it's really important to tailor your assessment to the patient in front of you. And we see a lot of different forms of HCM that come to end-stage. And so, in your restrictive patient they may not meet the standard ISLT guideline-based criteria. But nonetheless, they have an end stage form of heart failure with very few, if any, other options for definitive treatment and when their quality of life is poor enough that you think it justifies the risk of heart transplant, then we absolutely would consider it.

The other thing I think that's important on the peak VO<sub>2</sub> is that a lot of these patients are younger than your standard transplant patients. And so, to look at their peak VO<sub>2</sub> as a percent predicted for their age rather than just the absolute criteria of cut off a 14 or 12. I think those parameters are really important in young patients and young patients with HCM.

Ferhaan Ahmad: [07:59](#) And so Dr. Kilic, my impression as a non-surgeon is that surgeons look for that sweet spot. You do not want to operate on a patient that is still quite well, but you don't want to wait until they're too sick either. So, I just wanted to get your perspective on what is the right stage to consider a transplant in a patient with HCM?

Ahmet Kilic: [08:25](#) That's a great question. Oftentimes as surgeons we're at the last step, so to speak, of heart failure. So, the tough job of diagnosing genetic testing during the primary preventions, figuring out who needs AICDs are already done by the time that we get referred. And the tough part of figuring out how bad someone's hypertrophic cardiomyopathy is, is based on symptoms. So, these patients that come in don't necessarily

meet the traditional criteria that you would with regards to either ejection fraction or exercise testing. So you really have to rely on the physicians that know them best. So these patients are best served in a center of excellence where you have each of these different specialists that can work to find this fine line between patients that are sliding at what we call burned-out dilated hypertrophy. That's what we say in the medical community.

But what really means is that they're on that edge and any little further pushing, they'll go into intractable arrhythmias or they'll start having significant filling pressures where their pulmonary vascular resistance will be irreversible or worse yet they'll start having end organ damage where their creatin will rise or their liver becomes dysfunctional. And as a result they become not the ideal candidates for an operation. And really it's that identification before you lead into end organ dysfunction that is really important. And that's why establishment of care and cereal either imaging or clinical visits is paramount in these decision-makings

Anjali Owens:

[10:02](#)

Such an important point about end organ dysfunction. And oftentimes we see that providers or cardiologists will look at the renal function, but they forget about the liver function. And congestive hepatopathy is a very bad sign in these patients. Particularly when they've got elevation of right atrial pressures chronically. And so, picking that up early by doing a spot check of the liver and kidney function, spot check, trending over time, the NT-proBNP and the use of cardiac MRI to assess how much fibrosis is developing in the heart. If you have a heart that's more than 30, 40% fibrosis and scar, you know that that heart has very little reserve and it's time to think about what your next step is going to be.

Ferhaan Ahmad:

[10:44](#)

And then you both pointed out that you have to tailor your criteria a little bit for end stage HCM patient as opposed to other patients of heart failure. Sometimes that I think probably makes it even more difficult for them to get a heart compared to some other patients that are more sort of classic like fitting the criteria. So, then you have to worry about, how are you going to bridge them to transplant and on how to keep them well enough to get to a transplant. And that's a collaborative effort between heart failure, cardiologists and cardiac transplant surgeons. And I'd like to ask both of you actually, what are your strategies that you use to keep patients reasonably healthy until they get to the point that they get a heart?

Ahmet Kilic:

[11:27](#)

I think early assessment and continued recognition of symptoms is as important as we've already stressed in this podcast. Once patients show any evidence that they're not tolerating medications either through hypotension or an end organ dysfunction or repeat visits into the hospital, that's when they should be assessed for heart transplantation. As many of you probably know, there's been an allocation in the heart transplant policy or patients with restrictive or hypertrophic cardiomyopathy used to be a status 1B now they're status 4.

So even with that they have a fairly long waiting period. So it is very important for us to have strategies to be able to carry patients who are on this edge successfully for an appropriate organ to become available. The one thing that we have not mentioned is just how big of a role AICDs have in preventing what used to be the number one reason for patients not making it to transplant which used to be lethal arrhythmias.

Most of the time, as I stated, when I see patients, they've already had AICDs either as primary, secondary intervention. So those patients that come into the hospital as an inpatient status, when we're very worried, we have to think about different approaches to get them to a heart transplant. Oftentimes they can involve things like inotropes, milrinone. The little balance between that is the fact that this is a pro arrhythmogenic medication. But at the same time, it's a medication that improves your cardiac output and can reduce your pulmonary vascular resistance.

In addition, other agents can be tried. One thing that is having an increasing role in bridging patients to transplantation or temporary and durable mechanical circulatory support devices. This too has an inherent difficulty in that patients that have hypertrophic cardiomyopathy may not have appropriate left ventricular sizes to accommodate some of these devices. Up to half of the patients that we deal with are bridged with some kind of device.

And the things that we look specifically for if we're thinking about this in terms of getting more hemodynamic support to get them to transplantation are specifically just how well the LV cavity looks. The smaller the LV cavity is the more challenging it is. The more hypertrophied the muscle is, the more challenging that it is. The more muscular bands you have near the apex or where are you going to put an inflow cannular, the more challenging it is. And those are some of the interplay we have in trying to decide how we're going to do things successfully. And

the best way once again of doing that is through a multidisciplinary collaboration.

Ferhaan Ahmad: [14:05](#) Dr. Owens you discussed the different subtypes of end stage HCM. Whether it's the reduced ejection fraction or the severe restrictive physiology. And of course it can be combinations of both in the same patient. Are your strategies similar in terms of bridging to transplant? Or do you have different nuances that you have to consider each subtype?

Anjali Owens: [14:28](#) Great question. And yes, we absolutely tailor our bridging strategy based on the patient's cardiac anatomy, their blood type, their expected wait time based on whether or not they're sensitized, their body size. And of course, how sick they are when they're coming in. As Ahmet said, we routinely consider ICD implant for patients who were being bridged to transplant and the vast majority have an ICD at the time that they're transplanted. We implant LVADS, as Ahmet said, in patients who've dilated their LV enough to accommodate the inflow cannula without risking LVAD malfunction, suction events after surgery.

So, this is mostly for us, patients who've dilated enough to have an end diastolic diameter of, I would say, at least five and a half, six centimeters ideally. And most of them have reduced their ejection fraction and have frank systolic dysfunction. Here at our center we frequently use inotropes to support our cardiac patients. Both their cardiac output and as Ahmet said to reduce their pulmonary pressures. We sometimes even add in specific pulmonary vasodilator therapy to target the pulmonary vascular resistance. Particularly if the PVR remains elevated after we've achieved normal perfusion with inotropes and restorative cardiac output and once we've achieved euvoemia.

So, in that situation if they still remain with a high PVR, we'll use pulmonary vasodilators as sort of a targeted therapy. And here we occasionally utilize temporary mechanical support with either a balloon pump or ECMO in our sickest patients. Provided that we think their anatomy and their physiology with HCM will tolerate that after load reduction in the case of an intraaortic balloon pump. So, we've had success with all those strategies but it's really key that you get together as a multidisciplinary team with your surgeon and come up with a good plan for the patient in front of you.

Ferhaan Ahmad: [16:27](#) So it sounds like having a heart team is extremely important and Dr. Kilic, I presume you have one at Hopkins as well as you have in Iowa?

- Ahmet Kilic: [16:38](#) Absolutely. And we meet every day to discuss each one of these different options and any given plan can change minute to minute. And that's why it's so important to have plans and active discussions and have multiple people looking at things
- Ferhaan Ahmad: [16:51](#) All right. So, I'd like to actually also talk a little bit about another problem that I face a lot which is patients, particularly patients with apical variant form of HCM and many of them are at risk for acute develop apical aneurysms. And as you know recent data suggests that these patients are at high risk of arrhythmias and also at high risk of thromboembolic phenomena. This is probably particularly relevant or appropriate for you Dr. Kilic. What do you see a role for aneurysm resections and situations like this?
- Ahmet Kilic: [17:25](#) Great question and one where we don't have much data, to be honest, to help guide us. We know that aneurysms can occur in probably about 5% of all hypertrophic cardiomyopathy patients. When they do they're at increased risk of having issues with regards to mortality or morbidity. Most of these are related to either a clot formation with subsequent embolization of this clot or from it serving as a foci for arrhythmogenic activity.
- So, it becomes a really tough thing because our usual criteria or indications for an operation on an aneurysm are those three things. Thromboembolic disease, arrhythmias and heart failure. And oftentimes it's tough to say which is driving the boat in patients that have hypertrophic cardiomyopathy. If it's really their underlying genetic predisposition or if it truly is an aneurysm. The tough thing is because not all of them are massively dilated any operation we do to resect this aneurysm leads to a reduction in the size of the left ventricle which in itself can become an issue.
- So once again, this is one of those things where we look at it from a multidisciplinary fashion, if it's something that we fear with regards to an arrhythmia, I'll have my electrophysiologist involved. I have them make sure that we map it, and we make sure that the scar where this aneurysm is, is truly the foci of the arrhythmias. Last thing you want to do is do a big operation and have it not be the area of concern. Obviously thromboembolic phenomenon, if it's isolated, you can do things like anticoagulation. And I would say in this time and age, I try to reserve aneurysm surgery for those who have recurrent disease or in those where the aneurysm is growing under surveillance and causing rapid issues.

- Ferhaan Ahmad: [19:15](#) That's an interesting dilemma, isn't it? You're making a relatively small ventricle or ventricular cavity even smaller. Really in the absence of a lot of good data, do you have any way of sort of predicting how someone's going to respond to aneurysm resection?
- Ahmet Kilic: [19:32](#) Unfortunately we don't. And that's why we as surgeons are so hesitant to perform these type of procedures. The other thing that puts a little wrench in the works is that there is no real correlation between aneurysm size itself and symptoms. So it can't go based on criteria level like you would for, let's say an aortic aneurysm and base things on size. And it really has to be the clinical course in the clinical sequelae of this aneurysm.
- Ferhaan Ahmad: [19:58](#) Yeah. So obviously there's a lot more data to acquire, to be on firmer footing, to determine how we should be managing these patients. I'm going to turn over the podcast now to serve the second half or second third I should say our discussion today, which is emerging medical approaches to treating these patients who fail conventional medical therapy. And I like to joke that I'm hoping to put people like Dr. Kilic out of business by improving our medical options.
- So I'd like to start with actually a really exciting development, which is mavacamten, which some of us have experienced in both pre-clinical and in clinical studies. And Dr. Owen do you want to talk a little bit about what your thoughts are? What your experience has been so far and what your thoughts are for the future in terms of the benefit of mavacamten and what kinds of patients might benefit?
- Anjali Owens: [20:52](#) I'd be happy to. I think it's one of the most exciting aspects of HCM management that has come up and hopefully we'll be part of the future for our patients with HCM and hopefully something that will have a very positive, meaningful place in the treatment of this disease. I hope that will be the case. My experience thus far with mavacamten has been serving as our site PI for clinical trials here and we have been involved in both the early phase two open-label study, which was PIONEER-HCM.
- And then on to MAVERICK-HCM which of course was a phase two randomized controlled trial in non-obstructive HCM patients. And the larger phase three randomized control trial EXPLORER-HCM, which was the pivotal trial in obstructive HCM. And we're currently participating in VALOR-HCM, which is a phase three randomized trial in patients who are eligible for septal reduction therapy.



And mavacamten as many of you know, is a new medication with a novel mechanism of action. It's a myosin inhibitor so it modulates contractility. And the thought behind the drug is that many of our patients with HCM have normal ejection fraction or hypercontractility or hyperactive contraction. And that we're hoping to modulate that and it's a reversible inhibitor. And so if you withdraw the drug, we get the baseline ejection fraction and contractility comes back. But when it's active we hope that it will improve both the contractility to make it more normal and also improve diastolic function. Which of course is something that is very elusive with all of the medicines that we have today. We're not able to really target diastolic function or normalize relaxation.

So that is the hope. And in the clinical trials we have seen benefit. In particular, the EXPLORER-HCM trial, which was for patients with obstructive symptomatic HCM, who were on standard background therapy with beta blockers, calcium channel blockers. Patients who were on digoxin were excluded from that trial. But the vast majority of patients were on beta blockers and calcium blockers. And what we saw in the group that received mavacamten compared to placebo, was an improvement in the three things you want to see. Objective exercise, capacity symptoms and reduction in LVOT gradient.

And so, again, that's exactly what you want to see in your patients who have obstructive HCM and that's what we saw. In addition, we saw improvement in biomarkers. And in the MRI sub study, we saw improvement in markers of remodeling. So improvement in left atrial volume and reduction in left ventricular mass. Now that's a pretty short term, eight months of drugs. So more data to come on the long-term extension but again, very promising. I think a bigger question is, what's going to be the role for myosin inhibitors for non-obstructive HCM.

And we have the MAVERICK trial that showed us that compared to placebo, we saw marked reductions in markers of stress, NTproBNP and troponin levels in patients who received mavacamten versus placebo. And we're awaiting and putting together data from the long-term extension of these patients who had open access drug, open-label drug for a much longer time. And that data should come out soon at the upcoming national meetings. And I should mention the second in class agent that is under clinical trial investigation. And that is CK-274 and that is currently the REDWOOD-HCM trial, which is a phase two randomized control trial. Again, a second same class agent, also a myosin inhibitor that's being trialed for obstructive HCM. And

we hope to get a readout on those results hopefully by the end of the year.

Ferhaan Ahmad:

[25:21](#)

So I'm really excited about this potentially being a game changer especially with regard actually even more than obstructive HCM, where we do have people like Dr. Kilic can help us out. The non-obstructive forms, short of transplant, .you don't have really great therapies and about half our patients fit that particular category. And as you're saying, Dr. Owen potentially these agents are going to be beneficial for most subsets of patients. And I just like to add that there's a lot of pre-clinical data as well in different models, some of which has been published, some which is unpublished at this point, that suggests that everything you hope to see long-term in these patients, you can see in these animal models as well.

And I have had the privilege of working on genetically engineered pig models as well, .and we're seeing really amazing benefits in that model system, so more to come, but we're very, very excited, I think all of us in the HCM community about this. Now, there's other trials also underway for both obstructive and non-obstructive. There's some trials underway for non-obstructive first, you can probably talk a little bit about. Did you want to mention some of these Dr. Owens?

Anjali Owens:

[26:38](#)

There is a multi-center trial that I believe is being conducted mainly in Europe, that's randomizing patients to sacubitril/valsartan or Entresto or a lifestyle intervention which includes exercise, physical activity and also a dietary supplement of an inorganic nitrate or optimal standard medical therapy. And the main end point there is to assess for a change in functional capacity by peak oxygen consumption. This trial is still enrolling. And so, we will look for results in the coming year or two. Of course, with COVID it's been hard to enroll some of the clinical trials, so there may be some delays.

But also, I think an interesting concept of using Entresto for non-obstructive HCM. Patients with non-obstructive HCM as we've talked about, are a heterogeneous group and what benefits one subset of non-obstructive HCM, depending on the path of physiology, may not benefit another subset. And so, for example, in patients with very small cavities and thick hearts, I'm not sure that the vasodilatory effects of sacubitril/valsartan may or may not be beneficial as opposed to a heart that is perhaps a bit more dilated with documented, elevated, filling pressures and a reducing ejection fraction even though it's not quite abnormal. That may be a person whose heart may benefit more from the vasodilating properties would get to be seen.

Ferhaan Ahmad: [28:15](#) And so the other category of patients that I'm thinking about are those that are an emerging class of patients. With our improvements in genetic testing technologies, we're now doing clinical genetic testing on families with HCM. We often do find patients that are carriers of the pathogenic variant that we know is can cause HCM and yet they don't have phenotype or they have a very mild phenotype and they have no symptoms. And at this point my approach and I think most people's approach is we just follow them over time. But I think it would be much more beneficial if you could identify agents that could actually prevent the onset of disease. And I'm just going to ask you what your thoughts were Dr. Owens about any new trials underway to look at that particular issue?

Anjali Owens: [29:08](#) Well, we did of course have the VANISH trial, which was a phase two NIH funded, multi-center randomized, placebo controlled trial looking at valsartan. Just as you said, in younger patients who are genotype positive and either a mild phenotype or no hypertrophy. And the vast majority of patients that were enrolled in this trial were asymptomatic. I think over 90% or so are class one. And so really the test was to see if Valsartan could do just as you said. Attenuate onset or progression of disease.

And we have finished enrolling for this trial and we are anxiously awaiting the results. So I would hope that we'll get some word hopefully later this year but I think many of us are anxiously awaiting and it is a very uncertain population. You know that your gene is high positive, you're looking at them, you're watching their heart year to year. And if there was something you could do, ideally we would want to do it. And I'd love your thoughts on whether or not you think that one day we'll get to myosin inhibitors in pre-disease States or whether that's premature.

Ferhaan Ahmad: [30:24](#) I think that that's a very exciting possibility at this point. We have pre-clinical data, we have animal model data in a variety of different species that are genetically engineered to carry human HCM variant that amvacamten can at least partially reverse established disease, established topography. And potentially also prevent the onset of disease in models that are treated right early on before they've actually developed a significant phenotype. Of course, you can't necessarily translate all these models systems to human patients.

And so time will tell what we see in these animal models will also apply in human patients, but the data are fairly consistent across many different models now. So it's certainly a very distinct possibility that we will have this option in the future. It

won't happen right away; we do things step by step and at this point we hope that the FDA will approve the use of amvacamten or eventually other similar agents in patients that are of established disease.

First starting with obstructive then maybe non-obstructive HCM. And then of course the next step will be to see whether we can use these in patients that either have mild phenotypes or no phenotypes. And you have to remember that the bar is going to be much higher because you are going to be administering a potentially very tricky medication in terms of side effects and dosing, to patients long-term over lifetime potentially, who don't currently have over disease and are not overtly symptomatic.

So, that will always be an issue. But I think long-term, yes, we may have a way of actually preventing or attenuating disease. It's a very exciting time, indeed. All right. Well, I'm going to bring in Ms. McCoy into the discussion a little bit. Ms. McCoy has had a heart transplant not too many years ago and her mom actually also had a transplant not too long ago either. So now she has a unique perspective to bring to this discussion, having actually undergone a transplant.

So I'm wondering to start off by asking you Ms McCoy, you've had actually a pretty tragic family history. You've had many loved ones who passed away from HCM including two brothers, I believe. And then two maternal uncles who've also passed away. That has to be an enormous burden emotionally to you and to your family. And I wanted to ask you how this experience in your family has affected the decisions that you have made in terms of your own health and your loved one's health?

Ovuke' McCoy:

[33:05](#)

Thank you for having me. First of all, it's totally affected the way that I live for as long as I can remember. My mom has taken me to the doctor like I said, for as long as I can remember, it's affected every aspect of my life since I was nine years old. Participating in sports or different activities or what I can and when I could not do or taking medication. And I lived a pretty normal life making sure watching what I ate, living pretty healthy, having to participate in music and arts and letting my friends know, "Well, I can't do that, or I didn't run, or I did not participate in that particular activity." So there were certain things that I didn't do but it just wasn't as... it wasn't bad. So I lived a pretty normal high school kid type of life. My mom told me about my uncles. I was pretty young that I knew that they had passed, but I never knew that my brothers, being that I was the oldest, that they would be taken away from this disease.

Ferhaan Ahmad: [34:45](#) As you mentioned, you were diagnosed very early on as a child. Tell us a little bit about the course that your health took over the next decades to the point that you actually needed a transplant.

Ovuke' McCoy: [34:59](#) I would say it really didn't start to affect me that much just pretty much medication therapy that I had. I went off to college able to just do my thing. I was pretty much independent. Didn't want to be bothered, normal teenager, one or two party. I just wanted to do my own thing. So, it really did not really start to affect me until my late 30s, early forties. I noticed the biggest changes. Fatigue, passing out spells.

So, I got my first ICD in late 20s and I had to have my battery replaced around my early thirties. And I thought that was going to be the fix for me because I didn't want the transplant at all because I knew my mom had it. And I just was ignorant about transplant, even though my mom had it she did so well. I just didn't want it. I just wanted to go about my life and be independent.

And as close to my early 40s. I started to have more trouble as my heart started to thicken a little bit more. I had to take more medication, the fatigue started to come on, breathing fluid around my heart. The symptoms started to come on a little bit more close to three weeks, close to when I was about to get married. Three weeks after I got married, I was placed on a heart transplant list. I started to get those arrhythmias and there was a lot of fluid around my heart.

Ferhaan Ahmad: [36:48](#) So basically you were forced into this against your will. Completely against your will.

Ovuke' McCoy: [36:54](#) Yeah, I was going to the doctor for an appointment and I've been watched by the heart failure team for probably since about 2002 and going for appointment. And they just pulled me into the exam room, and they said, "Hey, we're going to put you on a heart transplant." I just had a biopsy probably about 20 minutes before. And they just said, "We're putting you on a heart transplant list." And they looked at my blood types, said I had O negative blood, it's going to take about two years. And they just didn't want to wait any longer. They said, referring to Dr. Owen. She said something, "You don't want to be too sick and you don't want to be too healthy." So, I was right in that area where I was, so they didn't want to get me to the point where I was going to be too sick to need a heart transplant.

Ferhaan Ahmad: [37:51](#) So tell us a little bit about the transplant process and then how your life has been after the transplant.

Ovuke' McCoy: [37:58](#) Well, the transplant process, I was still working at the time. I was still going to work. I was an academic advisor at the University where I worked at. So, it was still challenging for me to go to work every day. And so basically, I was just getting up in the morning, dragging myself to work, coming home, laying down. So, it was just a challenge and I was just like, "Ooh, if they could just get me that heart." It was a challenge. But anybody that's on a heart transplant list waiting you just want the heart because I just wanted to feel better. I can just recall going to the grocery store and just having to get a grocery cart. And I just need the grocery cart to walk. I didn't need it to put my groceries in because it was it hard for me to walk.

And I went into my doctor. I was like, "Can you just give me a handicap place card? I just want to park closer to the front of the store because that's how hard for me was to walk." And at 42, it was difficult to me to accept those types of things for myself to do that. So even doing... Because I work with college students. I had to do freshmen orientation. So even thought back in the day it was easier for me to walk a couple blocks, but I couldn't walk through a couple of blocks.

So I had to apply for a service where I had to catch a bus so I could make it to do my job. So those are some of the things that I had to get adjusted to and I could tell my body was getting... excuse me, I'm getting a little emotional because I think about it, because it was a very emotional time. As I think about it, the things that I had to do just to make it. My heart, because sometimes I felt that I was not going to make it through the summer of 2018 if I did not get a heart. And I continued to work and it was the week of 4th of July and I went into work and I didn't feel well.

I was walking in real slow, and I was looking for my co-worker and I said, "Oh, I wish my co-worker could take me to the emergency room." Because I wasn't feeling well at all. But I went into work. I said, "I'm going to have the week off." So, I went in and I was supposed to get fluid drained off because I was going on vacation. So, I said, "Ooh, at least I get to rest." And I talked with my pre-transplant coordinator and I went to the emergency room that night before and I just had to leave because it was so many people and I just couldn't stand to be there.

And my pre-transplant coordinator, she said, "Why did you leave?" I said, well, I just got tired of being there. She said, "Well, we're going to get you a bed." And I'm like, "Okay, well great." And she called me that afternoon and she said, "Well, we'll get you a bed so we can get the fluid off you. And that was July 3rd and I was still in the hospital July 3rd. And you July 4th, 4th of July, I was still in the hospital and I'm like, "Well, I'm supposed to be out of here. I thought you was going to drain the fluid off me?" And then the transplant person came. The heart transplant. She said, "Well, we want to move you to the transplant floor." And I'm like, "Why you move to the transplant floor? I don't understand this. I'm supposed to be going on vacation."

And then she said, "Well, we going to do something in the morning." So the surgeon came in, the heart transplants surgeon. So she said, "We have a heart for you." And I'm like, "What? So?" And I'm like, "Wow." He said, "We think it's going to work. And we're going to start surgery at 5:30 that afternoon. So that is how I ended up getting my heart. And I just went to praising God. That is how my transplant story started. And I was in the hospital for two weeks and I was released, and I started rehab and that's how I got my heart transplant.

Ferhaan Ahmad: [42:40](#)

Well, that's an amazing story. How's life been since then?

Ovuke' McCoy: [42:44](#)

Life has been really good. I ended up going back to work five months after that but I ended up having a virus. I caught a virus and I had to be in the hospital for several weeks after that. And I ended up having to leave work and that worked... it was fine and I had to get my health back. But after that I've been doing great. And I started a full-time job actually a month ago. So it's been great. Currently right now I started back volunteering. Of course, I'm a 2021 class of American Heart Association, Real Woman. I work with my sorority Alpha Kappa Alpha Sorority, Inc.

I currently do volunteer work with the junior league of Birmingham. I also work with my mom. We're trying to start a non-profit in memory of my brothers that will start this fall. Dealing with African American young men that deal with HCM. So, if we will hopefully get that story as well as helping people with resources. So, it's been great. I've had no issues. It's been a wonderful opportunity. I've had the opportunity to meet my organ donor's family as well.

Ferhaan Ahmad: [43:59](#)

That's amazing. That's good to know that things have improved so much now and you're paying it forward too.

Ovuke' McCoy: [44:07](#) Yes.

Ferhaan Ahmad: [44:07](#) You touched on something that I want to finish up with which is you've mentioned in the past that members of the black community often face a lot of hurdles in accessing healthcare. Can you tell us a little bit about those barriers and then tell us as healthcare providers, what we can do or what we should be cognizant of to help eliminate those barriers?

Ovuke' McCoy: [44:30](#) I can only speak from my experience and so for... sometimes the Black community and there can be stigma for as of not trusting healthcare professionals or of not seeing someone that looks like them in the healthcare community or not trusting someone that looks like them. And that can be that case as well and I can go back 26 years to my mom's case of heart transplant, being that no one in my family wanted my mom to have a heart transplant. Because we didn't know nothing about heart transplant. We didn't trust heart transplant because we thought she was going to die. No one could tell us about it. We didn't trust it. We just didn't trust it because of the healthcare system. My mom was there. She believed and she went forward with it but to tell us and people in her family about it. We told her, "No, you shouldn't go through with it. We don't trust it. We don't trust healthcare. You shouldn't do it."

So I think it's a matter of putting that knowledge out there for the black community and for them to be able to trust the healthcare provider. So that was just one example. But even though that was 26 years in the past, things are a little bit better now, but that was just an example that I can use based on from my mom because I was one of the ones that didn't want my mom to have the heart transplant because I was ignorant on that. But since we have so much knowledge now, that's a little bit different. But still there are some people who are not aware of that and don't have that information, still don't trust healthcare providers.

Ferhaan Ahmad: [46:26](#) I am glad though that at least you have two happy endings here. Well, thank you all so much for participating in this podcast today. This podcast is part of the American heart association HCM initiative, sponsored by Bristol-Myers Squibb. And in closing, I'd like to remind everyone listening, to encourage your patients to play an active role in their medical care by advocating for themselves and their family members. They can also go to the AHA hypertrophic cardiomyopathy patient website for more education. Thank you.