

ANBUKARASI Chronic total occlusion of a heart blood vessel is a term we use when the blood vessel is 100% blocked. The 100%
MARAN: blockage can happen all of a sudden, which will lead to a sudden heart attack and it's an emergency. Or it happens over a period of three to four months, or just more than three months. So when it happens so slowly over time, the body has its own innate capacity to form collaterals from the other branches to compensate for this 100% blockage. So all the cardiac catheterizations we do, and we see patients with heart disease, about 15% to 30% of the patients with heart disease can have chronic total occlusions.

AUDIENCE: Great.

ANBUKARASI Think about past, I'm talking about 2000, early 2000s or even before that, before the 2000s it has been only
MARAN: medications. OK? So we put patients on beta blockers, which slow the heart rate. And we put them on calcium channel blockers to open up the collateral blood flow. And we put them on long-acting nitrates to basically dilate the blood vessels which are already there. And that's all we've been doing.

Sometimes we try to open them up with the wires. But they have not been successful. And therefore, most of the time, during a heart cath, if we saw a chronic total occlusion, we will just send the patient for medical management with medications.

The technology has changed. The wires which are available to us are different. And the wires are much more stronger. The wires come with micro-catheters, which can kind of go over the wire and increase the strength of the wire. And devices called CrossBoss and STINGRAY are now in vogue, which help us to go around the blockage.

So basically, if the blockage is right here in the first part of my finger, the CrossBoss can go around the blockage. And we use the STINGRAY balloon to reenter back into the blood vessel. And then we can stent across. So with these technologies, now, we have an algorithm called the hybrid algorithm, which kind of tells us to, based on the anatomy of the patient, we can choose whichever strategy we want to cross these occlusions.

AUDIENCE: OK.

ANBUKARASI Imaging is kind of the cornerstone. We start off with a patient comes to the clinic complaining of a chest pain,
MARAN: shortness of breath on exertion. And most of the time, it's shortness of breath on exertion. And then the cardiologist or the primary care physician can order a nuclear stress test, which may or may not be abnormal. Or most of the times, it's abnormal. Then the patient proceeds for a heart cath. Then we see the chronic total occlusion.

If it is associated with other blockages, if all the three blood vessels have disease, then the patient most of the time is sent for bypass surgery. But if only one or two blood vessels are occluded and one of them is a chronic total, we'll fix the other blockage which is less than 100%, that is, just like a regular blockage, and we'll bring the patient back for a CTO-PCI.

Sometimes, we may need to know information, whether the heart muscle supplied by that blood vessel is viable or not. There are times when chronic total occlusions are there. The collateral flow from the other blood vessel is not good enough and the muscle dies off. In those patients, there's no point fixing the blood vessel, the chronic total blood vessel. So we will do cardiac MRI to look for viability, an echocardiogram to look for contractile reserve.

Sometimes, the blood vessel is closed off and it's 100% closed. So you don't know the direction in which the blood vessel is flowing. We might even need cardiac CT scans, which can delineate the course of the blood vessels so that we can use it during our cardiac cath.

We usually start with access in both groins. Compared to the regular heart cath, these are slightly bigger catheters because we're using more bigger instruments. And we do what is called dual angiogram. In a CTO vessel, you have the native vessel, which is occluded, and you have the opposite side vessel, which is giving bypass.

So you need to take a dual angiogram to get a complete picture of the length of the chronic total occlusion. How is the proximal cap, the initial beak of the chronic occlusion? Are there interventional collaterals, collaterals which we can use to reach the chronic total occlusion?

There are four strategies, antegrade wire escalation, antegrade dissection reentry, retrograde wire escalation, retrograde dissection reentry. These are the four techniques. It is how we do them is where the meat of the matter is.

Antegrade wire escalation we start with a tapered-tip wire called the Fielder wire. We probe and see if we can get through the cap. Sometimes, there are micro-channels kind of traversing through the entire occlusion and we could find a channel.

Then if that doesn't work, we escalate with the push strength of the wire. And we have catheters in the back end to support the push strength. If it doesn't work and if it's in a suitable patient, depending on the anatomy, we can go around, dissection reentry. And we basically go around the blockage within the vessel wall, but around the blockage, and reenter. So that would be antegrade wire escalation and antegrade wire dissection reentry.

If that is not working, but patient has collaterals from the left side or opposite side, whichever it's based on, we can engage the septal collaterals or sometimes, suitable epicardial collaterals, and approach the vessel from the back end and kind of traverse the entire vessel and put stents across. There have been times where we get across. We balloon the wire, the entire vessel, and we allow the body time to heal, and bring the patient back and put stents in, in four to six weeks.

Percutaneous interventions for chronic total occlusions is not for everyone. It's for a select group of patients who have chest pain or shortness of breath on exertion, whose muscle is viable on the imaging test we do, who have been treated on medications and have failed medical therapy. Let me give you an example.

A patient has known chronic total occlusion. He or she has been tried on beta blockers, calcium channel blockers, long-acting nitrates, but the patient is unable to move from point A to point B. Some patients just make their life smaller. That is, instead of taking 20 minutes to go from point A to point B, they take 35 minutes or 40 minutes. Or they just move to point A point 5, just walk shorter distances.

Some patients are just not able to do anything. And their life becomes shorter, smaller and smaller. And because of that, they get physically deconditioned and lose their stamina and their quality of life is extremely poor. It's those patients this treatment can be helpful. The CTO-PCI right now has not shown any evidence that it helps you live longer or even it prevents future heart attacks or future hospitalizations. All we know from stents is that stents can help with symptoms of chest pain and shortness of breath.

Patients have definitely shown an improvement in their dyspnea on exertion or shortness of breath on exertion. I've had a patient who was on disability and was using 15 nitroglycerin tablets per day to get through his day, who's now off any nitroglycerin and has actually gone back to the workforce because of resolution of his symptoms of chest pain. And there have been patients whose ejection fraction, which is the strength of the heart squeeze, has been down because of the CTO of the LAD, or the front wall blood vessel. And once we fix that, the ejection fraction is improved. And his symptoms of heart failure have also come down.

So the normal heart cath or the normal stenting procedure, the risk of complication, any complication, such as bleeding, stroke, heart attack, or death is about 1 in 1,000, so less than 1%. CTO-CPI, because we use stiffer wires, stronger push force, and micro-catheters, and we are kind of going in the architecture of the vessel and not in the lumen of the blood vessel, the complication rates are higher. It's about up to 5%, which includes bleeding, stroke, need for emergency surgery, injury to the kidneys because of the amount of contrast and radiation.

CTO-PCI, it's very exciting for me because these patients are usually deemed hopeless or there's nothing more we can do for them. And then they come to me with-- like, literally, there are patients who are in tears because they just cannot do anything more. They can't go shopping with their wives. They just cannot take their run around with their kids.

They can sit and watch. They can walk around in their own pace. But they're just not having a full life. And when you tell them there is something I can do where the chance of success is anywhere between 85% to 90%, it's like the look on their face is amazing. It's unbelievable. And when they come back to me for follow-up and they say that how they feel. They have a new outlook towards their life. That is an amazing feeling that we were able to do that for them.

AUDIENCE: OK.

ANBUKARASI
MARAN: So if you have a patient who has a known chronic total occlusion and is on more than two drugs for angina, it's not the end of the road. And if they are still continuing to have symptoms, it is not the end of the road. They should be offered CTO-PCI. And that would be the most important step to know that, that just because they have a chronic occlusion that we don't treat them further. There is yet another step of treatment which can be offered right now. And the Medical University of South Carolina offers it.

The first frame basically shows two catheters, one in the right coronary artery and one the left coronary artery. The dual program, again, defines the anatomy and tells us how long the lesion is. It also helps to score the severity of the CTO.

Most commonly used is the Japanese CTO score. It tells you about the cap, the length of the lesion, amount of calcification present, any tortuosity in the lesion. And based on that, we can plan for a procedure. So this is the first picture which shows the dual angiogram and delineates the anatomy of the lesion for us.

The second picture here shows me going in with the micro-catheter. And I have a Fielder XT wire, which is knuckled in. And I am in the subintimal space, or within the architecture of the vessel, but not in the true lumen.

The third picture I have come back with what is called the STINGRAY balloon. The STINGRAY balloon has two dots, which indicates two balloon with two exit ports, which are placed at 90 degrees of each other. So the wire can exit upwards where the vessel is, as it is in this picture. Or the wire can exit downwards into the myocardium. And this picture is basically shows appropriate position of the STINGRAY balloon.

The fourth picture here shows successful reentry into the true lumen using the STINGRAY balloon. We went across the lesion or around the lesion into the subintimal space. That is, we dissected the vessel and reentered the true lumen of the vessel using the STINGRAY balloon.

This picture shows our wire in the distal PD branch within the true lumen, which is basically confirming our wire position. We then proceed to balloon dilate and stent as needed. And here is the final angiogram of the right coronary artery fully revascularized.

First picture demonstrates a dual angiogram. Picture number two is another view of the dual angiogram. Basically, a LAO cranial view demonstrates the septal collaterals and gives us a better idea of which septal artery to engage. Picture three is basically septal surfing. I have a micro-catheter in the left anterior descending artery. I have a regular workhouse wire in the septal artery.

Once I have chosen the septal artery, I will continue with my micro-catheter and switch out for one of the dedicated septal surfing wires, such as the Fielder FC or the Si on. In this, I have got a Fielder FC in the septal artery. And you can see me traverse the septal artery and engage the collateral and get into the distal PDA and back into the distal RCA.

And now, I have followed it with my micro-catheter. And we have externalized the wire and stented the vessel. And here is the final angiogram showing PD and PL branches, which are intact.