ROB SIMARI: Greetings, I'm Rob Simari from the Division of Cardiovascular Diseases at the Mayo Clinic. And I'm joined today by Dr. Rajiv Gulati from the catheterization lab here in Rochester, to discuss a very interesting topic of spontaneous coronary artery dissection. Welcome, Rajiv.

RAJIV GULATI: Thanks, Rob.

ROB SIMARI: You know, it's amazing to me how it can be 2013, and these syndromes that were probably taking place for as long as we can remember are just being identified as sort of unique syndromes in medicine. And I think spontaneous coronary artery dissection is one of those. Where were those cases in the past, Rajiv? Why is the recognition just starting now?

RAJIV GULATI: Thanks, Rob. Well, I think they were always there. I think we've missed them, quite frankly. And as I look back upon my practice, even a few years ago, I suspect I missed some spontaneous coronary dissections. I think the prevalence is probably a lot more than we thought. And I think there are a number of reasons why we are recognizing it more now. I think troponin-- the fact that now young, healthy women with chest pain are actually being recognized as having acute coronary syndromes because of troponin-- and so coming to the lab means we have the opportunity to detect the dissection, increased recognition and awareness of this as a distinct entity, and third, I think the increasing role of intravascular imaging with OCT and IVIS now available in the Cath lab, means that we can address some of the limitations of coronary angiography alone.

What we may previously have called resistant spasm and narrowing in a vessel, or a diffuse disease, sort of irregular tapering of the vessel, we can now evaluate closely. And we're increasingly seeing that what appears like just a narrowing on an angiogram may actually be an intramural hematoma or medial dissection within the vessel wall causing this narrowing.

ROB SIMARI: So for a practitioner who might have a busy practice, but might not be thinking about SCAD as a diagnostic possibility, why is it important to get that in your mind in the Cath lab, or even before you enter the cath lab?

RAJIV GULATI: Yeah, I think it's important for a number of reasons. First, so we don't miss a diagnosis of an acute coronary syndrome. But second, because we're increasingly finding out that the management of patients with SCAD is tricky and may well be different to typical atherosclerotic SCAD.

ROB SIMARI: So what are the things that need to be kept in mind to avoid, rather than the standard treatment of a stenting?

RAJIV GULATI: So we have some data that we published in circulation last year. And we're building on that information. But when we look now, of course, the standard treatment for an atherosclerotic acute coronary syndrome is early catheterization, stent placement where appropriate, and medical therapy with dual antiplatelets and statins. In the SCAD population, we're concerned that PCI may not be the ideal strategy.

Certainly, when there's normal flow at baseline-- and we've seen when we look back over a number of cases, a much higher risk of complication when performing angioplasty for spontaneous dissection compared with plaque. And I think there's mechanical vessel wall reasons for that. There's no plaque in SCAD. I think it's important to recognize that it's not an atherosclerotic condition.

So the goal of mechanical treatment is not to seal plaque. In SCAD, the concern is that you will exacerbate a dissection by placing a stent, or exacerbate and intramural hematoma. And we've sure seen that clinically on looking back through some of the old angioplasty films. So I think recognizing this is a distinct entity has immediate implications for Cath lab therapy. And it may well be that backing off a little and taking a deep breath, and trying to establish a diagnosis prior to performing a PCI is the right way to go.

ROB SIMARI: So you often find yourself leaving these lesions that are present-- I use the term lesion as a narrowing, not as a plaque-- in getting more information or treating conservatively.

RAJIV GULATI: Yeah, if there's one thing that's changed in our practice, it is that. It is when we now suspect when there was a higher risk maybe for risk of spontaneous dissection. So typically, a younger or middle aged female with little, if any, atherosclerotic risk factors, we have an awareness pre-them coming to the lab. If we then see a lesion without threatened MI-- so with normal flow-- we'll often back off an image. And even if there's a significant narrowing from a hematoma, we may well do nothing at all. Because the natural history in the majority is for these to heal.

Now, it's important that not everyone follows that path. And we're learning as we go along as to which patients have a higher risk of not doing well with conservative therapy. And I think this is a real frontier for us now, is how to manage this population who were otherwise extremely fit and well, who are suddenly faced with an MI or a coronary disease diagnosis that they really weren't anticipating.

ROB SIMARI: So when we think about the pathophysiology-- and you mentioned the differences-- your paper in circulation suggested there might be relationships to disease outside of the coronary bed. Can you share with us those insights?

RAJIV GULATI: Yeah. The classic textbook teaching for SCAD was that it was typically seen in postpartum females. And sure, that's true, but it's now emerging that the principal association by some considerable distance is the presence of fibromuscular dysplasia in non-coronary vessels. So we found serendipitously, when we looked back at a whole bunch of angiograms, FMD in the iliac vessels-- when femoral angiograms were done at the time of closure device placement-- and now, other groups have shown that too. And we're screening all our SCAD population and finding a really high prevalence, somewhere between 60% and 90%, depending on which series you look at of FMD and non-coronary vascular beds.

ROB SIMARI: So does that have prognostic or therapeutic information, that relationship? Or is it too early to tell?

RAJIV GULATI: It's too early to tell. That's exactly our next question, and we're looking at that, and hope to present those findings at some stage soon. It would be nice to use that as a risk stratification, a variable to see if we can help define who is going to do better or worse, and who we need to monitor more closely.

It also brings up the intriguing possibility that SCAD is an FMD diagnosis. Maybe this is fibromuscular dysplasia of the coronary arteries that predispose them to dissection.

ROB SIMARI: And has that been borne out by any pathology studies in individuals that have gone to autopsy in SCAD?

RAJIV GULATI: There is autopsy confirmation of the presence of spontaneous coronary dissection and FMD elsewhere. But there's been no precise honing down of FMD in the coronary arteries, because it hasn't really been looked up well. There are some small reports, small case series of imaging for FMD, suggesting that maybe FMD on IVIS or OCT. But really, autopsy is where we need to look at this in more detail.

ROB SIMARI: So with this disorder that is relatively infrequent, and growing information in the field, are there opportunities for registry or sharing information among sites in this field?

RAJIV GULATI: Very much so. And I think, if anything is going to accelerate knowledge in the field, it is consolidation of information. And we're fortunate to have the Mayo Clinic SCAD registry, where we now have hundreds of patients recruited into it. And that will help us define demographics, outcomes, recurrence risk, and potentially unidentified risks for spontaneous dissection.

ROB SIMARI: Getting back to the recurrence risk, what do we tell a patient after-- they're a postpartum female, they've had an episode, we've left them conservatively. What are the chances of recurrence?

RAJIV GULATI: In our first series last year, the risk of recurrence was in females only. And there was a one in six chance of recurrence at some stage of the mean of, I think, 10 years of follow-up. It's a tough one, because it's always a number one question when patients come to us having had a SCAD event. We do say one in six based on our current data. We'll have more information as we develop knowledge from the registry.

It does seem to burn out, so that's an encouraging thing. It's an infrequent event in women over the age of 55, for example. So there's a very high likelihood of this burning out over time. But of course, it's an important question. We need to get more people in the registry to address that.

ROB SIMARI: Well, terrific. Thank you very much, Rajiv, for your insights into an area that I think there's going to be more to come, so to speak. And I'd like to thank the viewers for watching today, and encourage people to join us again for one of the Mayo cardiovascular videos here on the Heart.org. Thank you.