CHET RIHAL: Hi. This is Dr. Chet Rihal. Today, my guest is Dr. Amir Lerman, who's a vice chair for cardiovascular research in our division of cardiology. Amongst Amir's many interests has been the pathogenesis of acute coronary syndromes. And Amir, good morning.

AMIR LERMAN: Good morning, Chet. Thank you for having me.

CHET RIHAL: You're very welcome. Listen, Amir, tell us about the pathogenesis of ACS, particularly this concept of the vulnerable plaque.

AMIR LERMAN: So the concept of the vulnerable plaque came from observation that we have-- not only us, but other people-- that most of the patients that present with coronary syndrome actually do not have significant obstructive coronary disease. They draw our attention more on the structure of the plaque, rather the amount of the plaque. And we learn a lot from pathology, from the studies by [INAUDIBLE] who taught us a lot from autopsy, that apparently, the acute coronary syndrome is actually a result from abnormalities in the plaque structure in the constitute of the plaque. And mainly, the enlarged necrotic core, the thin cap-- fiberatheroma-- and a lot of constitute in the plaque from inflammation, blood vessel with no vascularization, if at some point, have a plaque eruption and creating a thrombus leading to that acute coronary syndrome.

CHET RIHAL: So what you're saying is that mild plaques can be so-called vulnerable under certain conditions. Now, can these vulnerable plaques be detected in vivo? Or is this just an autopsy phenomenon?

AMIR LERMAN: No. So that's a very good question. The plaque can be detected in vivo with more sophisticated novel imaging modality, such as, say, intravascular ultrasound with the addition under virtual histology component. With new images such as OCT and lipid scan, we can essentially detect the histology-- close to histology-- of the plaque, determine if the plaque has a lot of necrotic core, a lot of lipid volume. We can measure the thickness of the fibroatheroma with the shoulders that are covering the plaque. And in the future, we'll probably be able also to look at the amount of neurovascularization that may lead to plaque hemorrhage and acute coronary syndrome.

CHET RIHAL: You were heavily involved in the prospect study. Can you give us a brief synopsis of that, and tell us how it should change our practice, if so?

AMIR LERMAN: So the prospect study just briefly was a prospective study in about 700 patients, that we looked at with the direction of Greg Stone-- who was the principal investigator-- on the natural history of vulnerable plaque as detectable virtual histology over three years.

And what was found out, that about 20% of the patients have event. And the interesting part is that half of them-- almost 11% to 12% have event not in the core occlusion that underwent a PCI-- indicating again, that when they look at that meta-analysis, the presence of necrotic core was one of the major components that was leading to cardiovascular event.

However, in the study-- which was relatively small, if you look in that event-- the majority of the event were not actually acute coronary syndrome or sudden death. The majority of the event were actually leading the progression of the disease over time at the site where you have necrotic core, and more plaque volume, and less lumen.

So I think that it taught us a lot-- that the coronary artery is a heterogeneous component. It's a dynamic process. Some necrotic core can undergo healing, some area without healing can undergo a rupture. And we are still in the phase of learning, that taught us a lot that we need to look not only at the lumen and a degree of stenosis, but rather, to be in depth of disease atherosclerosis, to the disease of the vascular world, not of the lumen. And learn how to look at the pathogenesis of the disease that's leading to acute coronary syndrome.

CHET RIHAL: So Amir, can these vulnerable plaques be detected in vivo? Or are these just autopsy phenomena?

AMIR LERMAN: Excellent question. We have now currently, more advanced and sophisticated intravascular imaging modality that allow us to look at the structure of the plan, including an ultrasound with virtual histology, including OCT that can tell us in exact-- not only the constitution of the plaque, but can allow us to accurately measure the thickness of the cap. And there are new modalities using spectroscopy to look at the content of the lipid content of the plaque.

CHET RIHAL: One of the neat things you've done, Amir, is you've developed a movie that illustrates really nicely, this concept of the vulnerable plaque and the events that lead to the rupture and then the occlusion of the vessel. We're going to make this video available to our audience members. It can be downloaded right underneath this video segment. Tell us what this movie shows.

AMIR LERMAN: So this movie was actually done by putting together multiple histology slides from autopsies, creating the concept of the plaque that is growing, creating a positive remodeling. And not obstructing the lumen, creating a mild obstruction of the lumen, about 40%. Their protocol is increasing, the cap became thinner. And at some point, in the shoulder of the plaque, there is a rupture, exposing the circulation to thrombogenic surface, and creating a occlusive thrombus.

CHET RIHAL: Why does the shoulder rupture? Why does it occur at the shoulder?

AMIR LERMAN: The concept the shoulder's rupture-- first of all, there's the physical instability there. But also, we found out that there was a lot of inflammation, macrophages. And that created a lot of weakness of the tissue, mainly, the shoulder.

CHET RIHAL: My guest today has been Dr. Amir Lerman, who's the vice chair for cardiovascular research here at Mayo Clinic. He talked to us today about the pathogenesis of acute coronary syndromes, specifically as it relates to the role of the vulnerable plaque. And he has put together a wonderful movie that I'm sure you will find interesting. You're free to download it and use it in your presentations, if it'll be useful. So thank you for joining us today. Thank you, Amir.

AMIR LERMAN: Thanks for having me.