ReachMD
Be part of the knowledge.

TRANSCRIPT

Transcript Details

This is a transcript of a continuing medical education (CME) activity. Additional media formats for the activity and full activity details (including sponsor and supporter, disclosures, and instructions for claiming credit) are available by visiting:
https://reachmd.com/programs/cme/the-hearts-aftermath-iris-impact-on-cardiac-morbidity-mortality/26977/

Released: 10/02/2024
Valid until: 10/02/2025
Time needed to complete: 29m

ReachMD

www.reachmd.com
info@reachmd.com
(866) 423-7849

The Heart's Aftermath: IRI's Impact on Cardiac Morbidity & Mortality

Announcer:
Welcome to CME on ReachMD. This episode is part of our MinuteCE curriculum.

Prior to beginning the activity, please be sure to review the faculty and commercial support disclosure statements as well as the learning objectives.

Dr. Patel:
Hello. This is CME on ReachMD, and I'm Dr. Manesh Patel, and I'm here today with a friend and a colleague, Dr. Deepak Bhatt. Deepak, welcome.

Dr. Bhatt:
Great to be here with you.

Dr. Patel:
Well, today we're talking about ischemia-reperfusion injury, and it's a significant contributor to myocardial damage following ST-elevation myocardial infarction, or STEMIs, and often after we do a percutaneous intervention, we think about this. One of the ways we often think about this is understanding the impacts of ischemia-reperfusion injury and the development of our patients, unfortunately, developing heart failure or mortality. Do you mind telling us a little bit about that association, Deepak?

Dr. Bhatt:
Sure. So we've got lots of evidence through the years that primary PCI is the way to treat patients coming in with acute MI. But a number of animal models have shown that when you open up an artery in the setting of infarction, so-called ischemia-reperfusion injury can occur. And as part of that, well, it's hot in every area of medicine, inflammation occurs. And that consists of things like neutrophil migration, oxidative stress, calcium overload, endothelial dysfunction, all the usual bad players that we associate with cardiovascular disease and adverse outcomes. But with ischemia-reperfusion injury, even though we're doing the right thing, opening up an artery quickly, that secondary process of ischemia-reperfusion injury inflammation can lead to myonecrosis, and that myonecrosis can, in turn, lead to dysfunction of myocardium, ie, heart failure. It can also lead to expansion of the infarct, which can also contribute to heart failure, but in its own right, can cause problems as well. So there's been a search for years, for decades, for ways to modulate ischemia-reperfusion injury, lots of things that look good in animals to date, none has actually worked in humans.

Dr. Patel:
Yeah. So these are often ways in which we try to stop this process, as you said, which is important. We've got to open the artery as fast as possible. And we've seen these analyses of patients and animal data initially from Reimer and Jennings and others with the wavefront phenomenon, time is muscle. So we get there to open up the artery, and the muscle's been starving. As the muscle's starving, we get blood flow back, we lead through these processes that might, in fact, lead to infarct size expansion, heart failure, worse outcomes.

We talked a little bit – Deepak, you mentioned some of the things that have been tried, and we talk in another one of our discussions

about some of those things have been tried. Are there newer agents that are being evaluated now that hold promise that we should be on the lookout for?

Dr. Bhatt:
Yes. In the ischemia-reperfusion injury field, you have to keep trying. And those of us that have been interested in this area continue to do so. And indeed, this is something I think we'll discuss in another segment in greater detail.

There is a compound under study, FDY-5301 is the name for it, but it is essentially a way we hope of mitigating ischemia-reperfusion injury. It is a sodium iodide, and the idea is that it provides an ability to reduce reactive oxygen species damage and potentially salvage myocardium. Of course, all this needs to be approved. There's some intriguing early data from preclinical and phase 2 study. But a phase 3 trial, of course, is the only way to know and indeed, we are doing one.

Dr. Patel:
That's fantastic, Deepak. And as a clinician or as a person listening to this, you might be thinking, who are those patients that are at the highest risk with STEMI for ischemia-reperfusion injury? I'm assuming those would be some of the ones that you're also targeting in these phase 3 studies. Maybe give us a sense of who people should be on the lookout when they're taking care of patients with these conditions.

Dr. Bhatt:
Well really, it's the folks that have a big MI that even at the current stage of all the advances in cardiology and interventional cardiology can die or end up with bad heart failure. And these are typically folks with large anterior wall ST-segment elevation MIs. These folks still develop a lot of complications, still are at risk for going into cardiogenic shock, or even if they do find, in the short term, developing heart failure in the intermediate or long term. So these are the sort of patients where myocardial salvage, potentially by mitigating, reducing, eliminating ischemia-reperfusion injury, could make a difference and improve their outcomes. These patients remain ones at very high event rates, and it's an area where I hope we can make a dent.

Dr. Patel:
Yeah, no. Absolutely appreciate your perspective. We've now gone through what I'll call the rapid reperfusion era, where we now get as fast as possible and measure our times to opening these arteries. But unfortunately, as you've highlighted, some of these patients get ischemia reperfusion, larger infarcts, heart failure, and with those infarcts, have worse outcomes.

So thank you for joining me, and we'll continue to search for those therapies with some new therapies hopefully that will help us with ischemia reperfusion. Thank you for listening. And thank you for this discussion on ischemia reperfusion.

Announcer:
You have been listening to CME on ReachMD. This activity is provided by Total CME, LLC and is part of our MinuteCE curriculum.

To receive your free CME credit, or to download this activity, go to ReachMD.com/CME. Thank you for listening.