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/expert-perspectives-on-invictus-trial/14445/

Released: 09/30/2022
Valid until: 09/30/2023
Time needed to complete: 1h 02m

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

Expert Perspectives on INVICTUS Trial

Announcer:
Welcome to CME on ReachMD. This episode is part of our MinuteCME 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:
Hi, my name's Manesh Patel from Duke University, and I'm here for Duke Heart On The Go. We're at the European Society of Cardiology meeting, and we're going to be talking about the INVICTUS trial. I'm joined by a friend, colleague, and co-faculty member, Renato Lopes. Renato, tell us about the INVICTUS trial.

Dr. Lopes:
Great. Thanks, Manesh, for having me. For sure, one of the most important trials of the meeting because no one, including us, I believe, could never have guessed the results of these trials, and I think that illustrates why we do, and why we need randomized trials to guide practice. So, basically, you're right. The INVICTUS trial is study a population that we're not studying, the AFib, the classic pivotal DOAC AFib trials, which is patients with primarily moderate to severe mitral stenosis, so rheumatic disease but primarily mitral stenosis, and comparing DOAC versus Warfarin. This case the DOAC was with Rivaroxaban. And to our surprise, this was to be a non-inferiority trial of Rivaroxaban versus Warfarin. So really expecting that Rivaroxaban would be as good as Warfarin, and maybe better. And what we found is actually the opposite, that Warfarin was better, with about 20% relative risk reduction in the primary endpoint, which was a composite of stroke, bolus MI, and death for vascular causes. And these results were also positive for death alone. So about 20% reduction in all caused death and about 20% reduction in ischemic strokes. So really, really striking findings because nobody could have expected that Warfarin would beat Rivaroxaban in this clinical setting.

Dr. Patel:
Yeah, and tell us a little bit about the population, Renato, because this population of patients with mitral stenosis were in fact not as high risk for stroke as some of our AFib patients but they had a high mortality rate. So there's certainly a disconnect here about some of these patients. Tell me about this population.

Dr. Lopes:
Correct. you're absolutely correct. You're absolutely right. The main driver of the difference was in mortality. And that's the most striking results. And we still don't know the reasons for that. We have some hypothesis, but this patient population is much younger, about 50 years of age, not the 70, 75 that's the typical AFib patients. We had about 72% of women and about 40% were zero to one. So really not a lot of co-morbidities but yet really high mortality rate and a really marked difference in mortality favoring Warfarin compared to Rivaroxaban, so really not expected.

Dr. Patel:
Yeah, and so what are the next steps? How will we tease this out? I guess the big take home message is for patients with mitral

stenosis, rheumatic heart disease, Warfarin is still the thing you have to use these patients cause it seems clearly to be better. So that's message one. And then the message two might be around how will we learn from this so we understand how to do better?

Dr. Lopes:
Correct. Well, I think the, at least some lessons learned. First, we learned that controlling well TTR having good TTRs, cause again the differences in mortality and the primary endpoint in favor of Warfarin started to happen about two years, which is exactly when the TTR was better controlled. So these might have contributed some, not for the entire explanation, but some of this might be explained by that. So good TTRs, if you can tolerate Warfarin, Warfarin works really well. I think that's one of the messages. The second message is that rheumatic heart disease, Manesh, might be a different animal. And we have never studied very well these disease and maybe this is time to do better studies, more studies, including mechanistic studies around rheumatic heart disease because it affects about 40 million people worldwide. So, I think one of the message that we might need to have better studies in this field. So those are two important message and third homework for the investigators. I think they're going to have to come up with a lot of sub analysis to further clarify some of the issues and shed some lights around the mechanism of these findings.

Dr. Patel:
Fantastic, Renato, I love the messages. I love the homework assignments. I love all of it, but it's been fun to be at ESC. We thank you all for tuning in for Duke Heart On The Go from the European Society of Cardiology.

Dr. Lopes:
Thanks guys.

Announcer:
You have been listening to CME on ReachMD. This activity is jointly provided by Global Learning Collaborative (GLC) and TotalCME, Inc. and is part of our MinuteCME curriculum.

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