Transcript Details
This is a transcript of an educational program. Details about the program and additional media formats for the program are accessible by visiting: https://reachmd.com/clinical-practice/cardiology/sonata-hcm-whats-on-the-horizon/85675/

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

SONATA-HCM: What's on the Horizon?

Announcer:
You're listening to GLC on ReachMD. This activity, titled SONATA-HCM: What's on the Horizon? is provided by Global Learning Collaborative.

Dr. Masri:
Hello from AHA 2026 here in Chicago. I'm Ahmad Masri, and today I'll discuss the SONATA-HCM trial evaluating sotagliflozin in patients with symptomatic obstructive and non-obstructive hypertrophic cardiomyopathy. Let's consider what the study could mean for the future of HCM management. So the SONATA-HCM trial enrolled patients with both, as we said, obstructive and non-obstructive HCM to evaluate the rationale and the reason to investigate SGLT1 and 2 inhibitor sotagliflozin in these patients.

So as we know from heart failure, SGLT2 inhibitors are one of the main therapies that we use in patients with heart failure with reduced ejection fraction and preserved ejection fraction. SGLT2 itself as a receptor is expressed primarily in the kidneys. However, SGLT1 as a receptor is expressed actually throughout many tissues in the body, including the heart.

We know that there is a metabolic effect of SGLT2 in the heart, but there are lots of effects of SGLT1 in the heart that are in the pro-ischemic, pro-fibrotic, pro-hypertrophy pathways. There have been multiple preclinical studies looking at the SGLT1 and 2 inhibitor sotagliflozin in patients with LVH, heart failure, and hypertrophic cardiomyopathy showing unique advantages of using such a strategy in patients with HCM.

In terms of the design of the trial, this was a unique trial because typically in hypertrophic cardiomyopathy, you divide the population into obstructive and non-obstructive, and you typically run separate individual trials for each. Due to the mechanism of action of sotagliflozin, that enables us to work with one population with hypertrophic cardiomyopathy.

And as such, the inclusion, really, criteria here relied on the fact that you just need to have hypertrophic cardiomyopathy, and then you can be stratified into obstructive and non-obstructive within the trial. The other piece of it is that this trial really looked at how the patients are feeling and used KCCQ as an entry criteria in the trial without having exercise as one of the primary reasons to be in or out of this trial.

In terms of the baseline characteristics, around 540 patients were enrolled in the trial, and they're randomized 1:1 to sotagliflozin versus placebo. Of these patients, about 20% were obstructive, and the rest were non-obstructive. If you look at the baseline characteristics, even though there was no exercise test that is needed to be in the trial, their characteristics are very typical of what you see in a highly symptomatic HCM population, where patients had reduced KCCQ scores at baseline, almost 58 to 60, on average, which is really reduced compared to what we have seen in some other trials.

You have about half and half in terms of females versus males, and you had lots of NYHA class II and III, which is by definition the trial needed you to be symptomatic. And so these characteristics really represent what you would expect to see in such a population of HCM with heavy burden of disease.

As such, these baseline characteristics are very reassuring that the trial enrolled patients with high burden of symptoms in patients with

© 2026 ReachMD

hypertrophic cardiomyopathy, and it will allow us to understand what is the effect of sotagliflozin in this patient population. Looking ahead, based on the timeline of this trial, we expect to hear about these results next year.

There are lots of implications of actually a trial like SONATA-HCM. One is that this is a new, unique mechanism of action in patients with hypertrophic cardiomyopathy. Two, I think—this is a personal opinion—it will unlock the future that we always have thought about, which is in hypertrophic cardiomyopathy, a complex disease will require multiple mechanisms of action to make patients feel better and have better functional capacity as well.

From AHA 2026, I'm Dr. Ahmad Masri, and thank you for watching.

Announcer:
You have been listening to GLC on ReachMD. This activity is provided by Global Learning Collaborative. Thank you for listening!

© 2026 ReachMD