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/programs/living-rheum/gout-epidemiology-and-pathogenesis/13355/

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

Gout Epidemiology and Pathogenesis

Announcer:

You're listening to ReachMD. This episode of *Living Rheum*, titled "Gout Epidemiology and Pathogenesis" is sponsored by Novartis US Clinical Development and Medical Affairs. The speakers have been compensated for their time. This program is intended for healthcare professionals. Here's your host, Dr Jason Liebowitz.

Dr Liebowitz:

Gout has been around for centuries, but we have just recently begun to understand its pathogenesis. With the prevalence of gout on the rise, what can we learn about its epidemiology and its causes?

This is ReachMD, and I'm Dr Jason Leibowitz. Joining me to discuss the epidemiology and pathogenesis of gout is Dr Sara Tedeschi. Dr Tedeschi is an Assistant Professor of Medicine at Harvard Medical School and Associate Physician at Brigham and Women's Hospital. She also serves on the American College of Rheumatology Committee on Quality of Care. Dr Tedeschi, thanks for joining us.

Dr Tedeschi:

It's a pleasure to be here.

Dr Liebowitz:

Let's begin with some background. Dr Tedeschi, what is the epidemiology of gout in the United States? And how does it compare to the rest of the world?

Dr Tedeschi:

Hyperuricemia, which is a prerequisite for developing gout, has a prevalence of 6 to 8% in healthy adults, and a prevalence of 1 in 3 adults who have uncontrolled hypertension or other cardiovascular risk factors. In the United States, the prevalence of gout has more than doubled between the 1960s and the 1990s. And it's now estimated that 3.9% of U.S. adults have gout, which amounts to about 8.3 million adults. Gout is more common in men than women, from anywhere, from a ratio of about 3 to 1 and up to 10 to 1. Gout incidence and prevalence increase with older age, and gout prevalence is also increasing worldwide, especially in racial minorities in the United States, in the New Zealand Māori, in Han Chinese, and in some ethnic groups in Asia who have a higher prevalence of gout.

Dr Liebowitz:

Can you give us a sense of our current understanding of the pathogenesis of gout, especially as it pertains to disease flares?

Dr Tedeschi:

Sure. Gout is caused by inflammatory responses to the deposition of monosodium urate, or MSU for short, monosodium urate crystals, which form in the presence of increased serum urate concentrations. MSU can deposit in other tissues, but deposits in the joints is what results in inflammation leading to clinical gout. MSU crystals trigger a strong inflammatory response by activating macrophages in tissues and promoting the collection of neutrophils in tissues or organs.

Dr Liebowitz:

Thank you very much for describing that pathogenesis. What would you say accounts for the self-limiting nature of gout flares?

Dr Tedeschi:

The self-limiting nature of a gout flare may relate to several mechanisms that include neutrophil extracellular traps and negative regulators of the inflammasome and toll-like receptor signaling, including anti-inflammatory cytokines.

Dr Liebowitz:

And if we take a look at gout in the context of an autoinflammatory disease, can you share some recent findings that would support the classification of gout as an autoinflammatory disease?

Dr Tedeschi:

The fact that MSU crystals trigger the NLRP3 inflammasome is really what has sparked this idea. NLRP3 is an intracellular sensor that can detect a range of microbial motifs, such as endogenous danger signals and environmental irritants. And this leads to the formation and activation of the NLRP3 inflammasome.

Dr Liebowitz:

And do you have any final takeaways to share with our audience with regards to gout epidemiology and pathogenesis?

Dr Tedeschi:

There's still a lot we don't know about gout, such as the relationship between gout and the increased risk of having adverse cardiovascular outcomes. It's still unclear whether it's gout itself, or hyperuricemia, or inflammation resulting from gout that results in the increased risk of heart disease. We also still need to understand why some patients with significant hyperuricemia don't develop gout. And as globalization of medical data increases, as more and more EHR data are shared across groups, I think that we'll be able to more accurately assess the incidence and the prevalence of gout worldwide.

I think also that our increased understanding of genetics and epigenetics of gout will continue to shed light on mechanisms that are at play in this disease. There have been lots of advances in genetic findings in the past 5 to 10 years, but most of these findings don't currently change the management of patients. And I think, finally, that our continued deeper understanding of the pathophysiology of gout will help us to develop additional treatments that we can help our patients with.

Dr Liebowitz:

Wonderful, thank you so much for your discussion of this interesting and evolving field. I want to thank my guest for helping us better understand the epidemiology and pathogenesis of gout. Dr Tedeschi, it was a great pleasure speaking with you today.

Dr Tedeschi:

It's been great to be here.

Announcer:

This industry podcast was sponsored by Novartis US Clinical Development and Medical Affairs. If you missed any part of this discussion or to find others in this series, visit reach-M-D-dot-com-slash-living-rheum. This is ReachMD. Be part of the knowledge.