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Dr. Abdellatif:
This is CME on ReachMD. I’m Dr. Abdul Abdellatif. Here with me today is Dr. Richard Johnson.

Richard, what is the association between serum uric acid level and CKD [chronic kidney disease]?

Dr. Johnson:
So as chronic kidney disease worsens, as the kidney function worsens, we accumulate more and more uric acid. Sometimes we refer to this as the uric acid burden. People who have a very high serum uric acid, like 9 or 10, almost inevitably have kidney disease. But not only is it associated with kidney disease, but a rising uric acid is associated with increasing frequencies of obesity, of diabetes, of heart disease, of high blood pressure and of many other metabolic complications. One connecting factor is inflammation. Uric acid crystals are extraordinarily inflammatory, and when they get in the joints, they cause systemic inflammation and fever.

In one study, over 80% of people with gout have uric acid crystals localizable to their blood vessels, like their aorta and coronary arteries. And this has been due to the discovery of a method to look for crystals called the dual energy CT scan, or DECT scan. There’s also been evidence that uric acid, even soluble uric acid, can be associated with increased risk for hypertension and heart disease, and this has been confirmed recently with studies that are called Mendelian randomization studies.

Abdul, what do you think our target serum uric acid should be?

Dr. Abdellatif:
Guidelines recommend that to lower the uric acid below 6 because we know at below 6 the uric acid then is more soluble and is able to be eliminated by the kidney. We also know if the patient is not treated to target of less than 6, that the longer they have a higher uric acid than 6, the faster they can build those crystals and tophi in their different joints and, as you mentioned, in different organs. But also, it’s been shown that the lower the uric acid, the faster you can get rid of those tophi in those patients with tophaceous gout.

In a study of more than 300 patients comparing the current available urate-lowering agents, which is allopurinol, febuxostat, we were only able to control patients to less than 6 using febuxostat in about 53% of the patients. It may rise up to 60% if the dose was higher, as they use in Europe. However, in the United States, our maximum dose is 80, and that’s why they showed about 53% reduction. As for allopurinol, only 21% of those patients actually reached target, which shows that a lot of our patients with chronic gout who need to be at target, they’re actually hard to control if we don’t pursue other alternative therapies for these patients, or more advanced treatment. It’s also been shown that out of those patients diagnosed with gout, which is now close to 10 million adults in the United States, unfortunately, it’s only about 3.3 million adults receiving treatment. And out of those receiving treatment, if they were on maximum dose of therapy, maybe about 60% of them may be less than 6, but unfortunately, if they have chronic kidney disease with GFR [glomerular filtration rate] less than 30, only about 25% of these patients are reaching target of uric acid less than 6 mg/dL.

So it’s very important for us when we treat the patient, we treat the target. We teach the patient that if we’re treating you for gout, it’s a chronic disease, lifelong disease just like diabetes, hypertension. We have a target for our treatment option, which is lowering the uric acid to less than 6. And if you’re still symptomatic, having gout flare, we have to go further lower level. If you still have tophi, we have to...
go even further down. Therefore, alternative therapies may be indicated, such as IV therapies that are available now called pegloticase, that we can actually treat those patients with to get them to target much faster and to get them to base much faster.

Dr. Johnson:
Well, this has been a brief but great discussion. Thanks again, Abdul, for your insights, and that's the end of this session.

Announcer:
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