Correspondence

Letter by George Regarding Article, “Uric Acid-Lowering Treatment With Benzbromarone in Patients With Heart Failure: A Double-Blind Placebo-Controlled Cross-Over Preliminary Study”

To the Editor:

I read with interest the study by Ogino et al1 about the effects of the uricosuric agent benzbromarone in patients with chronic heart failure. It is a well thought out study that further adds to the current body of evidence on the role of urate.

In our studies using probenecid in this same cohort, there was also no improvement in endothelial function using forearm venous occlusion plethysmography despite a 46% reduction in urate using probenecid (1000 mg/d) compared with a 59% improvement in forearm blood flow seen with allopurinol (300 mg/d), which reduced urate by 44%.² Perhaps, to strengthen this finding, I wonder if the authors have assessed endothelial function in these patients to demonstrate lack of effect when lowering urate without interfering with the xanthine oxidase system compared with previously published data with xanthine oxidase inhibitors such as allopurinol.²,³

Second, if, as it is becoming increasingly clear, xanthine oxidase is the culprit, were other measures of oxidative stress such as plasma F2-isoprostanes or urinary 8-epi-prostaglandin F (2alpha) measured? This may be extremely useful given the superoxide anion and hydrogen peroxide generation with xanthine oxidase’s catalytic activity.

Finally, it is worth noting that there have been studies²,³ that have shown improvement in parameters of vascular biology, such as endothelial function, in patients with normal urate levels and that urate may not be the ideal marker of xanthine oxidase activity due to the extremely complex regulation of urate and the different extracellular versus intracellular role that it may play.⁴,⁵

Disclosures

None.

Jacob George, MBChB, MRCP, MD
Centre for Cardiovascular & Lung Biology
Division of Medical Sciences
University of Dundee, Ninewells Hospital & Medical School Dundee
Scotland, UK
E-mail: j.george@dundee.ac.uk

References

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Jacob George

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