# Renal Consult

## Is "Runner's Kidney" a Thing?

**Q Many of my patients are athletes. I recall reading something about kidney disease in marathon runners. Am I remembering correctly?**

Although data on acute kidney injury (AKI) in marathon runners are limited, two recent studies have added to our knowledge. In 2017, Mansour et al studied 22 marathon runners, collecting urine and blood samples 24 hours before, immediately after, and 24 hours after a race. The results showed that in 82% of the subjects, serum creatinine increased to a level correlated with stage 1 or 2 AKI (as defined by the Acute Kidney Injury Network criteria).[1]

Based on urine microscopy results, as well as serum creatinine and novel biomarker levels, the researchers concluded that the runners' AKI was caused by acute tubular injury—likely induced by ischemia. However, the subjects did not show any evidence of chronic kidney disease (CKD), despite years of running and intensive training. One theory: Habitual running might condition the kidneys to transient ischemic conditions—in other words, they build tolerance to repetitive injury over time.[1]

The other recent study examined use of NSAIDs by ultramarathon runners (ie, those who run more than 26.219 miles). In an intention-to-treat analysis, 52% of runners taking ibuprofen developed AKI, compared with 34% of those receiving placebo; the number needed to treat was 5.5. AKI was also more severe in NSAID users than in placebo users. The results were not statistically significant due to an underpowered study (N = 89). However, the authors also observed that slower runners were *less* likely to develop AKI, and those who lost the most weight during the race were *more* likely to develop AKI—suggesting that lower intensity running and adequate hydration may help prevent kidney injury.[2]

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In summary: While marathon runners are prone to AKI, the injury seems to be transient and does not progress to CKD. Furthermore, use of NSAIDs during endurance running may contribute to AKI development, so patients should be advised to use caution with these analgesics. Finally, remind your endurance runners to stay hydrated, since it may help to limit kidney damage. As for the casual runner? The impact on the kidney remains unclear and needs further investigation. **—DSW**

### REFERENCES


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## Can African-American Patients Take Metoprolol?

**Q One of the physicians in my practice won't use metoprolol in African-American patients. He says it causes kidney disease. Is he right, or is this an old wives' tale?**

There are multiple concerns with the use of metoprolol specifically—this does not ap-

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The National Kidney Foundation Council of Advanced Practitioners' (NKF-CAP) mission is to serve as an advisory resource for the NKF, nurse practitioners, physician assistants, clinical nurse specialists, and the community in advancing the care, treatment, and education of patients with kidney disease and their families. CAP is an advocate for professional development, research, and health policies that impact the delivery of patient care and professional practice. For more information on NKF-CAP, visit **www.kidney.org/CAP**.

ply to all ß-blockers—in the African-American population. The main concerns are

- Lack of effective blood pressure control, compared to angiotensin-converting enzyme (ACE) inhibitors and calcium channel blockers (CCBs)
- No observable reduction in proteinuria
- The possibility of a significant increase in uric acid.

Most of the evidence-based guidelines for care of hypertensive nephrosclerosis in the African-American population were derived from the African-American Study of Kidney Disease and Hypertension (AASK) trial. This large-scale, multicenter, randomized, double-blinded study from the National Institute of Health had multiple arms to compare an ACE inhibitor (ramipril) to a CCB (amlodipine) or a ß-blocker (metoprolol) in the nondiabetic African-American population.[1]

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In a subgroup analysis, more than 1,000 subjects with hypertensive nephrosclerosis were followed for four years, with serial glomerular filtration rate (GFR) measurements taken. Treatment with ACE inhibitors was shown to be superior to CCB and ß-blockers for hypertension and proteinuria control.[1]

One important takeaway from the AASK trial has been that strict blood pressure control is not enough to improve kidney outcomes. Proteinuria (albuminuria) must also be controlled.[1]

In a subsequent secondary analysis of data from the AASK study, Juraschek et al showed that metoprolol significantly increased serum uric acid in African-American adults.[2] It is known that hyperuricemia (> 6 mg/dL) can cause a decline in kidney function.[3]

Furthermore, uric acid may be a strong prognostic factor for chronic kidney disease (CKD) progression. (This association, however, remains controversial. One recent study showed that, while hyperuricemia is associated with higher risk for kidney failure, the relationship was not parallel in CKD stage 3 or 4 [GFR ≤ 60 mL/min]).[4] In fact, taking uric acid–lowering medications did not slow progression of kidney disease.

In other words, your colleague seems to believe that since A (metoprolol) leads to B (hyperuricemia) and B (hyperuricemia) leads to C (kidney disease), then A leads to C. While the theory is undoubtedly logical, we have no proof that metoprolol causes increased kidney disease in African-American patients.

What we do know, thanks to AASK, is that an African-American patient with kidney disease should be treated with a diuretic and/or an ACE inhibitor as initial therapy. Furthermore, we have a blood pressure goal: < 130/80 mm Hg. And we know that CCBs are most effective for African-American patients who do *not* have kidney disease.[5] **—BWM** **CR**

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1. Toto RD. Lessons from the African-American Study of Kidney Disease and Hypertension: an update. *Curr Hypertens Rep.* 2006;8(5):409-412.
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