Runners in Ultramarathon Increase AKI Risk

Ibuprofen May and Mortality Risks May Predict ESRD

Filtration Markers Consistently Associated with Incidence and Progression of Kidney Disease

Filtration Markers and Their Association with 1-Year Change in Measured (mGFR) and Estimated (eGFR) GFR. The study included 317 patients from the Modification of Diet in Renal Disease study and 373 patients from the African American Study of Kidney Disease and Hypertension (AASK).

At 12- and 24-month follow-up visits, patients underwent measurement of creatinine, cystatin C, β-trace protein (BTP), and β2-microglobulin (B2M), along with mGFR. Associations with ESRD and all-cause mortality per 30% decline in mGFR or eGFR were analyzed for individual markers and for the average of four markers.

In both groups of patients, 1-year declines in mGFR, eGFR based on creatinine, and eGFR based on BTP were significantly associated with incident ESRD. The average of all four markers was also associated with ESRD. The only filtration marker more strongly associated with ESRD risk in both studies was decline in eGFRBTP.

Decline in eGFRCr was associated with all-cause mortality only in AASK; incidence rate ratio 4.17 per 30% decline. This was not significantly different from the association observed in the Modification of Diet in Renal Disease study.

Findings

Ibuprofen May Increase AKI Risk in Ultramarathon Runners

Acute kidney injury (AKI) may be more frequent in ultramarathon runners who take ibuprofen, according to a randomized controlled trial in Emergency Medicine Journal.

The study included 91 athletes participating in 50-mile ultramarathon races in desert environments. Runners were randomly assigned to take ibuprofen 400 mg or placebo tablets every 4 hours during their race. Incidence of AKI was compared between groups: “risk” was defined as a 1.5-fold increase in creatinine and “injury” as a twofold increase. Runner characteristics were similar between groups; in the ibuprofen group, average total dose was 1200 mg.

Overall AKI incidence was 44%. On intention-to-treat analysis, AKI occurred in 52% of runners taking ibuprofen versus 34% taking placebo. The 18% difference exceeded the 15% noninferiority threshold. However, the number needed to harm was 5.5 ibuprofen-treated runners to cause 1 additional case of AKI.

Both categories of AKI were more frequent with ibuprofen: 58% versus 26% for “injury” (nonsignificant) and 14% versus 9% for “risk” (significant). Slower finishers were less likely to develop AKI: odds ratio (OR) 0.67. Greater weight loss was associated with a higher risk of AKI: OR 1.2 at a 1.5% reduction in body weight.

Studies have reported 34% to 85% rates of AKI in ultramarathoners. Although it has been suggested that nonsteroidal anti-inflammatory drugs (NSAIDs) might contribute to these rates, studies have not evaluated the risk for runners who still take NSAIDs during races. The evidence for and causal nature of this association are unclear.

Despite the lack of statistical significance, this trial suggests an increased risk of AKI in ultramarathon runners who use ibuprofen. Taking NSAIDs during endurance running “could exacerbate renal injury,” the researchers write. They note that associations of AKI with finishing time and weight loss suggest a role of dehydration (Lipman GS, et al. Ibuprofen versus placebo effect on acute kidney injury in ultramarathons: a randomised controlled trial. Emerg Med 2017; doi: 10.1136/ emermed-2016-206353).

Filtration Markers May Predict ESRD and Mortality Risks

Concentrations of four markers of filtration, individually and in combination, are consistently associated with the risk of progression to end stage renal disease (ESRD), reports a study in the American Journal of Kidney Diseases.

Members of the Chronic Kidney Disease Biomarkers Consortium analyzed filtration markers and their association with 1-year change in measured (mGFR) and estimated (eGFR) glomerular filtration rate. The study included observational data on 317 patients from the Modification of Diet in Renal Disease study and 373 patients from the African American Study of Kidney Disease and Hypertension (AASK).

At 12- and 24-month follow-up visits, patients underwent measurement of creatinine, cystatin C, β-trace protein (BTP), and β2-microglobulin (B2M), along with mGFR. Associations with ESRD and all-cause mortality per 30% decline in mGFR or eGFR were analyzed for individual markers and for the average of four markers.

In both groups of patients, 1-year declines in mGFR, eGFR based on creatinine, and eGFR based on BTP were significantly associated with incident ESRD. The average of all four markers was also associated with ESRD. The only filtration marker more strongly associated with ESRD risk in both studies was decline in eGFRBTP.

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Iron-deficiency anemia in CKD is different.

Is it time for a new school of thought?

In CKD, progressive loss of renal function along with chronic inflammation leads to:

• High concentrations and reduced clearance of hepcidin
  • Impaired intestinal iron absorption
  • Restricted release of iron from storage

Can different thinking help us address these challenges for iron-deficiency anemia in CKD?