The utility of ultrasound may be limited for at-risk individuals aged 30 yr. In contrast, the study by Pei et al.7 of both patients with PKD1 and with PKD2 suggests the utility of ultrasound may be limited for at-risk individuals who are younger than 30 yr and have a negative or indeterminate scan and unknown genotype. For these individuals, repeat ultrasound scanning or even magnetic resonance imaging scanning4 may be useful to detect new cysts. According to the Ravine criteria for patients with PKD1, the finding of fewer than two cysts in each kidney is enough for disease exclusion in at-risk individuals aged ≥30 yr. In the contrast, the study by Pei et al.7 of both patients with PKD1 and with PKD2 suggests the utility of ultrasound may be limited for at-risk individuals who are younger than 30 yr and have a negative or indeterminate scan and unknown genotype. For these individuals, repeat ultrasound scanning or even magnetic resonance imaging scanning4 may be useful to detect new cysts. According to the new unified criteria presented by Pei et al.,7 the presence of fewer than two renal cysts has a negative predictive value of 100% and is enough to exclude the disease in at-risk individuals who are aged ≥40 yr.

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DISCLOSURES

None.

REFERENCES


See related article, “Unified Criteria for the Ultrasonographic Diagnosis of ADPKD,” on pages 205–212.

Yin and Yang: Acute Kidney Injury and Chronic Kidney Disease

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Patients with chronic kidney disease (CKD) have high rates of acute kidney injury (AKI). In the other direction, some AKI does not resolve and a hospitalized patient may be left with CKD. However, most of the time an AKI episode is self-limited, and kidney function returns to a level that predates the acute injury. Two questions then surround the natural history of this latter type of AKI when a patient is discharged from the hospital: Did the AKI identify a group of patients who failed a “stress test,” patients predestined to have high rates of mortality and morbidity including CKD? Did the injury damage the kidney in such a way that there will be a faster rate of decline toward advanced CKD?

Both questions are important for clinical care. The former question guides patient counseling and follow-up and emphasizes the outpatient testing and use of interventions proven to prevent CKD. The latter question also emphasizes the importance of preventing AKI and has partic-
ular implications for any intervention that prevents AKI at a high cost or with a possible risk of toxicity. In the current issue of JASN, Ishani and colleagues examine the yin and yang of AKI and CKD. They characterize the association between AKI and subsequent CKD in a group of elderly Medicare beneficiaries in the United States.

In brief, Ishani studied just over 230,000 patients, of which approximately 7000 had AKI. Baseline characteristics at the time of hospital discharge were ascertained using health administrative databases: the mean age was 79 yr, 27% were diabetic, and 12% had CKD. The data confirm that patients with CKD are more likely to be diagnosed with AKI during a hospital admission than those without CKD. When one considers the outcomes after hospital discharge, the 2-yr risk of CKD requiring dialysis was 43 per 1000 persons who developed AKI, and 4 per 1000 persons without AKI. The risk of CKD requiring dialysis after AKI remains evident after accounting for age, sex, previous CKD, diabetes, and other factors. With this work, Ishani extends knowledge about the epidemiology of CKD after AKI. Although the incidence of CKD after AKI has been described in over 20 previous studies (summarized in systematic review), Ishani’s is among the first to characterize the association in which groups of patients without AKI were also considered. Newsome et al. published the other recent study in which groups of patients with and without AKI were concurrently followed for the development of CKD requiring dialysis after hospitalization for myocardial infarction. The sampling in Ishani’s study is population based and can be expected to generalize well to elderly persons in the United States. Finally, the observed association is supported by biology. Failure to restore renal structure and function adequately after an episode of AKI may upregulate inflammatory and fibrotic signaling pathways, leading to progressive structural kidney damage that manifests as a more rapid decline in the GFR.

Yet, as the philosopher who struggles with the concept of “unity in duality,” Ishani’s data leave the reader with more questions than simple answers. There were no serum creatinine data available, and the AKI could not be described according to modern staging systems. We remain uncertain as to what degree the AKI resolved other than by the absence of dialysis. Administrative database codes for CKD, AKI, and other comorbidities are less than perfect as acknowledged by the authors. Codes may not equally identify all patients within a spectrum of disease, and it remains likely that only those patients with the most severe form of illness were identified with AKI and CKD. When the ICD9-CM AKI codes used by Ishani were validated against a 100% change in serum creatinine from nadir to peak during a hospitalization by Waiker et al., it is true the specificity was 97.7% despite a sensitivity of only 35.4% (ICD9-CM: International Classification of Disease 9th Edition, Clinical Modification). However, if the true prevalence of nondialysis-dependent AKI was 3% (as seen in Ishani’s study), this would mean 67% of the patients identified with AKI in the study did not meet the reference standard definition of AKI (i.e., they were false positives). Similarly, when various ICD9-CM CKD codes were validated against a first estimated GFR during a hospitalization of less than 60 ml/min/1.73 m², the sensitivity was no more 27%. Although one could argue the association seen by Ishani in the setting of random chaos speaks to an even stronger signal, it also remains possible that differential misclassification of AKI and comorbidities cloud the observed association. Finally, the study was observational in nature; outside of an interventional trial it remains unknown to what degree preventing or reducing AKI prevents CKD.

Looking to the future, prospective studies should follow up with serial measurements of renal function, especially in those with milder self-limited AKI, to confirm the association described by Ishani. Better information about the AKI would help, including aspects of its duration, recovery, treatment, and associated features. Similar knowledge of the renal function predating the AKI could also be of benefit, including the natural course and treatment of any CKD before the AKI. Only then will we perhaps become truly enlightened as to how much AKI and CKD transform each other, in the dynamic balance of all things nephrologic.

DISCLOSURES

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