score.[3] Simultaneous kidney and liver transplantation (SLK) is advised for patients with either (a,b) chronic kidney disease and GFR $<30$ ml/min (including ESRD), (c) acute kidney injury (presumed HRS) and dialysis over 8 weeks duration, or (d) more than 30% glomerulosclerosis or interstitial fibrosis on kidney biopsy.[4]

The data from van Slambrouck *et al.* provide evidence for a potentially significant contribution of BCN to the renal prognosis of patients after liver transplantation. Although data are lacking, reversal of hyperbilirubinemia through liver transplantation will probably coincide with favorable renal prognosis, parallel to other pigment-derived tubulopathies.[5] Identification of patients with BCN instead of classic HRS through renal biopsy before organ allocation could prove to be an organ-sparing strategy. A study comparing outcome of pretransplant BCN with other causes of renal failure is required to determine the exact role of BCN. Therefore, clinical awareness of this neglected but prevalent diagnosis in any jaundiced patient is urgently needed.


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*Kidney International* (2014) **85,** 713–714; doi:10.1038/ki.2013.480

# Urinary angiotensinogen can be useful for screening patients with polycystic kidney disease

**To the Editor:** We have read with great interest the article by Parikh *et al.*,[1] concerning the urine biomarkers of kidney injury in autosomal dominant polycystic kidney disease (ADPKD). They concluded that urinary neutrophil gelatinase-associated lipocalin (NGAL) and interleukin 18 (IL-18) excretions were elevated in ADPKD, changed over time, but did not correlate with worsening in kidney function or increase in total kidney volume (TKV). It is plausible to find an applicable biomarker to predict the clinical course of the ADPKD due to its progressive pattern. Thus, we have a proposal for the authors to address this issue based upon our recent study in ADPKD patients.

After the clinical availability of urinary angiotensinogen (UANG), a specific index of intrarenal renin–angiotensin system (RAS) status, its role in hypertension and chronic kidney disease has been well established.[2] Early occurrence of hypertension and progression to chronic kidney disease without any intervention are the prominent features of ADPKD. Thus, the role of intrarenal RAS independently from systemic RAS has become a target for the investigators recently in ADPKD. Furthermore, it has been shown that ADPKD kidneys can synthesize all components of RAS.[3] Recently, we have found that UANG levels were increased in both hypertensive and normotensive ADPKD patients with normal renal function compared to matched healthy subjects in our cross-sectional study.[4] UANG may be a harbinger for the future clinical course of ADPKD. Therefore, further randomized prospective controlled trials are needed to clarify this proposal.


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*Kidney International* (2014) **85,** 714; doi:10.1038/ki.2013.515