As pointed out by Dr Schiffl, there is a time lag between the fall in glomerular filtration rate and the rise in serum creatinine, which delays the diagnosis of AKI. A key goal in AKI management is early detection and intervention. The hourly measurement of urine volume provides the opportunity to treat urine flow as a continuous physiological variable, providing more time points for early detection of AKI than serum creatinine assessed once or twice a day. Urine output changes raise red flags to the physicians for an ongoing injury to the kidneys possibly leading to an early intervention. In a recent review, Goldstein and Chawla proposed the concept of 'renal angina' to trigger additional attention to the kidney [2]. Oliguria is one of the three objective criteria along with any increase in serum creatinine and fluid overload that should prompt the concern of evolving AKI.

Despite Dr Schiffl's concerns we are confident that our findings will not “muddy the waters” for AKI. Rather, we have proposed a new methodology to standardize the urine output criteria in situations where an hourly urine flow measurement may not be available. Although the clinical relevance of the hourly assessment of the urine flow is not questioned, the 6-h block interval may be the only available information in some scenarios. This parameter is also relevant in retrospective studies, when often the hourly information is not available. We believe our paper has raised some awareness about the importance of timely diagnosis of AKI and the use of urine output as an early biomarker. Furthermore, our data provide support that urine output is an important biomarker of renal function, associated with major outcomes. Despite the limitations in the use of urine output as an early biomarker, its changes provide a sensitive and easy mean to identify patients with early AKI. We acknowledge that there is a requirement of additional large multicentre trials capturing refined data on fluid balance, diuretics and other factors affecting the urine flow, to explore further the use of urine output as a biomarker.

Conflict of interest statement. None declared.

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doi: 10.1093/ndt/gfq578

Advance Access publication 21 September 2010

Renal injury due to hepatic hydatid disease

Dear Editor,
I read with great interest the article by Altay et al. [1]. The authors made an association between hepatic hydatid disease and different glomerulonephritides including IgA nephropathy and MPGN. The article is important because hydatid disease is endemic in many parts of the world. However, a number of limitations, none of which was mentioned in the study, deserve attention.

First, and in my opinion the most important one, is that the authors did not use a control group matched for age, gender and other major aetiologic factors for renal disease. Thus, we cannot be sure whether haematuria and proteinuria in the study population are products of simple chance or because of hepatic hydatid disease. These results do not show an increased frequency of glomerulonephritides in patients with hydatid cyst disease.

Second, it seems paradoxical that none of the patients who had renal cysts (5% of the study population) had evidence of renal injury. How do the authors explain this finding? If it is said that the mechanism of injury is immune-mediated, the same immune mechanisms should also be the same for the patients with renal cysts.

Third, did the authors exclude patients with hypertension? We know that hypertension, with diabetes mellitus, is one of the major causes of renal disease. And again, there is no mention about the status of angiotensin-converting enzyme inhibitor and/or angiotensin receptor blocker use. Thus, a number of aetiologic factors such as hypertension, analgesic use, etc., which may be responsible for impaired kidney function, have not been appropriately controlled. IgA nephropathy is fairly common among renal biopsies. The frequency of IgA nephropathy reaches up to 35% of all renal biopsies [2].

Fourth, two of the biopsy-proven IgA nephropathy patients improved after treatment for the underlying hydatid cyst disease. Two other cases progressed despite therapy. We also know that spontaneous remission rates are fairly high in IgA nephritis and membranous nephropathy even in the absence of immunosuppressive therapy [3,4]. Thus, the lack of a consistent response to hydatid disease treatment in half of the patients in the absence of a control group and the lack of data about antihypertensive treatment modalities make it hard to draw definitive causal relations between hydatid disease and glomerulonephritis.

And finally, one should be cautious in using the term ‘hydatid nephropathy’, until an unequivocal causal relationship was proved.

Conflict of interest statement. None declared.

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doi: 10.1093/ndt/gfq583

Advance Access publication 21 September 2010

Reply

Sir,

We thank Dr Solak for his comments about our article.

Glomerulonephritis is a rare disorder, and various reports tell us that the incidence of glomerulonephritis is 12.5–47 per million population [1,2]. However, there are not enough data about the prevalence of glomerulonephritis. For this reason, we did not use a control group. Another reason was that we had a small patient population for an epidemiological study. So, in our opinion, using a control group would not increase the power of our study. Furthermore, we suggest that >10% frequency of glomerulopathy in this group is not because of simple chance. We also excluded the patients having hypertension or any other systemic diseases, or those who were using antihypertensives or analgesics which may be responsible for impaired kidney function to avoid interference in our study.

As we wrote in the introduction part of our article [3], parasitic nephropathies occur in three forms by three mechanisms. Renal cyst formation usually occurs with physical invasion, not by immunological mechanisms [4]. Furthermore, in the literature, there is no evidence of glomerulopathy in kidneys involved by hydatid cysts, such as in our study [5,6]. So, it is not paradoxical that none of the patients who had renal cysts (5% of the study population) had evidence of renal injury.

Although various factors such as type and process (acute or chronic) of glomerular disease and accompanying other renal injuries (tubulointerstitial nephritis, amyloidosis, etc.) can affect the response to the therapy, and sometimes patients may have spontaneous remission, it is not rational to say that glomerulopathy could not be related to hydatid disease in the absence of a consistent response to treatment in patients by lack of a control group. As we have seen in our cases, the presence of hydatid disease as the only aetiological factor for glomerulopathy and the remission after a treatment that is targeted at only hydatid disease suggest that there is a causal connection between renal injury and hydatid disease.

Conflict of interest statement. None declared.

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doi: 10.1093/ndt/gfq584

Advance Access publication 4 October 2010

Gastric bypass in patients with chronic kidney disease

Sir,

We read with great interest the article entitled ‘Bariatric surgery and renal function: a precarious balance between benefit and harm’ [1]. As prospective data increase, Roux-en-Y gastric bypass (RYGB) surgery does appear to cause moderate range hyperoxaluria (40–80 mg/day) in a significant number of RYGB patients, not just stone formers. While we agree with the authors’ final conclusion that further research is needed to determine the best means of treating renal complications of bariatric surgery, we disagree with the authors’ conclusions about the effect of bariatric surgery on patients with pre-existing renal disease. In particular, the authors reference Alexander et al. [2], a group that retrospectively examined renal outcomes of 45 patients with varying degrees of chronic kidney disease (CKD) who underwent RYGB surgery. The authors highlight 9 out of 23 patients with CKD not requiring dialysis whose disease stabilized over a short period of time. Implied but not discussed in the article of Alexander et al is progression of the other 14 patients (61%) [2]. No information regarding oxalate levels or cause of kidney disease progression is given by the authors, but a 61% progression rate, even in advanced CKD, would be considered extremely high. In contrast, Keith et al. followed up >11 000 non-obese patients with CKD for 5 years and reported that only 1.3% of those with stage 3 CKD and <20% of those with stage 4 CKD progressed to ESRD requiring dialysis or transplant [3].

There is little doubt that bariatric surgery is the most effective and sustained form of weight loss for morbidly obese patients, reversing associated renal co-morbidities such as diabetes and hypertension. Logically, it makes sense that renal function would follow suit. However, as physicians, we must carefully review and report studies accurately to our patients. Besides a handful of case reports and limited case series, there are little prospective data to