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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.

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
demonstrate that RYGB surgery reverses or even stabilizes renal function in patients with pre-existing renal disease. Therefore, the enthusiasm for this procedure in CKD patients should be tempered. When we review the literature, a dichotomy is evident: urologists detail oxalate levels without measuring global renal function, whereas nephrologists tend to relate markers of renal function without reporting urinary oxalate. In future studies, we urge urologists, bariatric surgeons, and nephrologists to consider measurements of all these labs, as perhaps oxalate is the missing link in this literature. Only by carefully designed, prospective studies can we better counsel our patients on this complex topic.

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Reply

Bariatric surgery and chronic kidney disease: an intriguing relationship

We read with interest the response to our brief review by Canales et al. who highlight the evidence that there are limited prospective data to demonstrate that RYGB surgery reverses or even stabilizes renal function in patients with pre-existing renal disease. The authors also refer to the dichotomy in focus between nephrologists and urologists stating that "urologists detail oxalate levels without measuring global renal function whereas nephrologists tend to relate markers of renal function without reporting urinary oxalate". Both we and Canales et al. agree that more research in this field is urgently needed to help identify who exactly will benefit from surgery and who will not.

We acknowledge that bariatric surgery per se may be associated with a little benefit and can even be associated with increased risk of acute kidney injury. For example, Sharma et al. showed that the incidence of acute renal failure was 2.3% among 1800 individuals who underwent bariatric surgery [1]. However, we consider that the balance of evidence is suggesting a benefit of marked weight loss in this group of patients. For example, a recent systemic review showed that interventional weight loss was also associated with decreased proteinuria and microalbuminuria [2]. We acknowledge that different studies show different effects of bariatric surgery on chronic kidney disease (CKD). For instance, Navaneethan and Yehnert showed that in 25 obese individuals with stage 3 CKD who underwent bariatric surgery, there was significant improvement in glomerular filtration rate from 47.9 to 61.6 mL/min/1.73 m² [3], and we have summarized the current evidence showing both the harm and benefit of bariatric surgery on renal function [4].

Interestingly, emerging evidence shows that bariatric surgery can be safely performed on patients on dialysis and also on transplant patients [5]. Another important question is whether the presence of associated co-morbidities (hypertension and diabetes) affects deterioration of renal function after undertaking bariatric surgery. Crucially, it is also important to assess the role of the pre-operative care, intra-operative procedure and postoperative care in renal function in these obese individuals. It is also not yet clear whether the deficiency of some micronutrients associated with bariatric surgery treatment may contribute to exacerbation of CKD. Therefore, we agree with Canales et al. that there is an urgent need for further studies to assess the benefit and harm of bariatric surgery in CKD. Currently, it is accepted that bariatric surgery can treat important risk factors for CKD (e.g. diabetes, hypertension and gross obesity). However, it is plausible that the effects of surgery per se may have an unfavourable impact on progression of CKD and affect renal function in those with prior normal renal function leading to acute kidney injury.

Taking all of these factors into consideration, it is rational to suggest that in every individual who undergoes bariatric surgery, monitoring of all parameters of renal function is the best way to proceed to try and ensure patient safety until a consensus is established about how best to select patients who are likely to benefit most from surgery.

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