Abstract

Background

Oxalate is produced both endogenously via metabolism of amino acids in the liver and exogenously via the intake of oxalate-rich food. It is primarily excreted via the kidney and excessive oxalate can lead to the precipitation of calcium oxalate in the renal tubules leading to kidney injury. Hyperoxaluria exists as primary and secondary hyperoxaluria and the latter is more common and is due to excessive oxalate in diet, increased intestinal absorption or decreased oxalate degradation.

Case presentation

A 54-year-old, previously well patient presented with an insidious onset abdominal distention and bilateral lower limb oedema associated with constitutional symptoms. Although he was maintaining an adequate urine output, he had an elevated serum creatinine of 8.2mg/dL. In the course of evaluating for a cause for the renal injury, the presence of calcium oxalate crystals in his urine full report, high serum calcium and bilateral nephrocalcinosis on imaging prompted us towards a potential diagnosis of oxalate nephropathy which was later confirmed by the presence of oxalate crystals in the renal biopsy. Later, on further questioning it was revealed that the consumption of local medicinal herbs, star fruit and vitamin c supplements contributed towards the development of oxalate nephropathy.

Conclusion

Oxalate nephropathy is a rare condition and the diagnosis of secondary forms of oxalate nephropathy can be challenging due to the unfamiliarity of the condition. Therefore, clinicians should have a high suspicion for this disorder

and frequently revisit the history to identify risk factors in the absence of findings for more commoner renal disorders.