Acute interstitial nephritis induced by glucosamine

Sir,

Acute tubulointerstitial nephritis (TIN) is an important cause of acute renal dysfunction resulting from immune-mediated tubulointerstitial injury. The commonest causes of TIN include drugs and infection. Acute interstitial nephritis accounts for up to 15% of patients hospitalized for acute renal dysfunction. Glucosamine is a relatively new alternative therapy for the treatment of osteoarthritis (OA). We present a case of possible glucosamine-induced TIN.

A 75-year-old man was admitted with a history of difficulty in passing urine, urgency, nocturia and hesitancy. There was no history of fever, rash or arthralgia. Past history was uneventful; he denied known drug allergy and the only medication he had been exposed to had been glucosamine (2–3 months) used for treatment of his osteoarthritis. General and systemic examination was unremarkable except for dehydration. Investigation revealed haemoglobin 10.3 g/dl; white cell count 15.1 x 10^9 (neutrophils 13.98, eosinophils 0.02), platelets 179 x 10^9, sodium 140 mmol/l, potassium 4.3 mmol/l, urea 45.8 mmol/l, creatinine 97 µmol/l, bicarbonate 19 mmol/l, bilirubin 2 µmol/l, alanine transferase 36 IU/l, alkaline phosphatase 183 IU/l, albumin 26 g/l and CRP 221 mg/l. He was initially fluid-resuscitated aggressively and catheterized, draining approximately 2000 ml of urine. Despite the above, his renal functions deteriorated. During the same period, he also had a series of blood tests which included normal complement and negative serum electrophoresis, auto-immune screen, ANCA and anti-GBM. Ultrasound demonstrated normal sized kidneys with good cortical thickness and a simple cyst in the left kidney. Prostatic volume was mildly increased at 48 cm^3 and prostatic specific antigen was within normal limits. Renal biopsy demonstrated a heavy mixed inflammatory cell infiltrate within the interstitium, suggestive of acute TIN. A minor degree of age-related atherosclerosis, involving the small and slightly larger blood vessels, was also noted. Glucosamine was discontinued and the patient received haemodialysis along with a short course of steroids. His symptoms improved significantly and he was dialysis-independent on discharge.

Drug-induced acute TIN is an inflammatory process involving the tubules and the space between the tubules and the glomeruli. It is mediated by T cell hypersensitivity reaction and cytotoxic T cell injury. Renal biopsy is the gold standard for diagnosis. Stopping the suspected medication forms the main component of treatment, with most patients recovering rapidly on withdrawal of the offending drug. Corticosteroid and immunosuppressants, in cases where there is no significant improvement in the renal function, may be of value. Recovery is more rapid in those individuals who have been exposed to the drug for less than 2 weeks, in comparison with those who have taken the suspected medication for more than 3 weeks.

Glucosamine is a commonly used alternative therapy in OA. Glucosamine is an aminosaccharide derived from chitin that takes part in the synthesis of glycosaminoglycans and proteoglycans by chondrocytes. It serves as a substrate for the biosynthesis of chondroitin sulfate, hyaluronic acid and other macromolecules located in the cartilage matrix.

Experience with the use of glucosamine in OA is limited. Commonly known documented side effects of glucosamine, non-specific gastrointestinal tract symptoms lead the list, followed by worsening insulin resistance in diabetics. There have been no reports of glucosamine-induced TIN. In this case, there was no obvious precipitating factor other than glucosamine for the histological changes and impaired renal function. We therefore believe that glucosamine contributed to the pathogenesis of TIN in this case.

The results presented in this article, have not been published previously in whole or in part.

Conflict of interest statement. There is no conflict of interest to be reported by the authors.


Transhepatic venous access as an alternative for Tesio catheter in the case of a patient on haemodialysis with antiphospholipid syndrome

Sir,

We report on a case of a patient with antiphospholipid syndrome with exhaustion of vascular accesses for haemodialysis. The transhepatic route through the suprahepatic vein