528

An Unusual Infiltrative Cause of Acute-on-Chronic Kidney Injury

Dr Hugh Leonard[1], Dr Stephen Sampson[2], Dr Andrew Laurie[3], Dr Bhriguraj Sood[4], Dr Subash Somalanka[4]
[1]Research Fellow, Renal Medicine, Guys Hospital, London, United Kingdom, [2]Department of Histopathology, St Helier Hospital, Carshalton, United Kingdom, [3]Department of Haematology, St Peters Hospital, Chertsey, United Kingdom, [4]Department of Nephrology, St Helier Hospital, Carshalton, United Kingdom

Introduction: Acute kidney injury (AKI) is an important cause of morbidity and mortality. It is seen in up to 43% of lymphomas. AKI may occur by several mechanisms such as obstruction, infection, therapy and direct or indirect effects of the lymphoma. We present an unusual case of AKI on chronic kidney disease stage G3 in a patient with a complex medical background. The renal biopsy was the key in informing the decision to treat an otherwise indolent process without any other indicators.

Case: A 76-year-old man was referred to nephrology with a rise in his creatinine from a baseline of 140 µmol/L to 250 µmol/L over two months. His background included a transitional cell carcinoma (TCC) of the bladder and a marginal zone non-Hodgkin lymphoma (MZL) that was under haematology surveillance with stable haematopoiesis, lymphadenopathy and IgG kappa paraprotein over the last 4 years. TCC had been treated in 2013 with a radical cystectomy and ileal conduit formation with follow-up imaging demonstrating chronic moderate bilateral hydronephrosis. On examination he was euvolaemic and normotensive. Investigations demonstrated nephrotic-range proteinuria (urine protein: creatinine ratio 412mg/mmol) without haematuria, serum albumin of 29g/L and a serum IgG kappa paraprotein of 50g/L, having risen from 45g/L over the last 12 months. Serum autoimmune screen was negative. He was not taking any nephrotoxic medications. Imaging demonstrated mild bilateral hydronephrosis with subsequent MAG-3 renogram and loopogram ruling out an obstruction. With stable haematopoiesis and lack of B symptoms, there was no indication to commence cytotoxic chemotherapy for his lymphoma. A renal biopsy was performed due to a rise in creatinine. It demonstrated a dense lymphoid cell infiltrate without any evidence of paraprotein-mediated glomerular or tubular pathology, with the infiltrate extending beyond the capsule of the kidney. Very mild interstitial fibrosis and tubular atrophy were noted. Immunohistochemistry demonstrated CD20 positivity and kappa light chain restriction, in keeping with direct lymphomatous infiltration of the kidney. He was commenced on rituximab and bendamustine chemotherapy for marginal zone lymphoma [MZL]. Renal function improved to a previous baseline, alongside marked improvements in proteinuria and paraprotein.

Discussion:

Renal parenchymal infiltration is seen in up to 34% of lymphoma patients but rarely causes an acute kidney injury. AKI is seen alongside lymphoproliferative disorders through several mechanisms including obstruction, therapy-related, glomerulonephritis, paraprotein-related and, rarely (<1%), due to direct infiltration. MZL is an indolent B-cell non-Hodgkin lymphoma accounting for 8% of lymphomas and can occur in extranodal areas in 70% of cases (in which case it is known as mucosa-associated lymphoid tissue lymphoma), the spleen in up to 20%, and isolated nodal disease is seen in 10% of cases. The kidney is a rare primary site of MZL (≤ 1%).

This case highlights the diagnostic utility of the renal biopsy in informing a decision to commence potentially harmful therapy in cases where the benefit is unclear. The past history of urological cancer, with concerns about ongoing obstruction further complicated and delayed this decision. The renal biopsy, in this case, confirmed a progressive, infiltrative disease process causing organ dysfunction which led to the commencement of chemotherapy and consequent improvement in renal function.