



A Case of Myeloma Kidney With Glomerular C3 Deposition

Asif Khan

Shaare Zedek Medical Center, Israel.

Abstract:

C3 glomerulonephritis is rare form of membranoproliferative glomerulonephritis, which result from defects in complement regulatory proteins that promotes excessive activation of alternative complement pathway. Kidney disease is common complication of multiple myeloma (MM). Most common renal complications in MM include monoclonal immunoglobulin deposition disease and myeloma cast nephropathy. Moreover, monoclonal Ig, through the interference of the complement alternative pathway has been shown to play the synergistic role towards renal damage. Up to 50% of MM patients present with renal impairment at diagnosis, 20% may present with acute kidney injury, and 10% require dialysis. In this case report, we describe a case of MM with cast nephropathy with mesangial staining for C3 consistent with C3 glomerulopathy, and the interrelationship between MM and complement system that leads to C3 glomerulopathy. A 59-year-old Trinidadian man with a 2-year history of hypertension presented with nausea and vomiting associated with a generalized weakness for the past 3 months. On admission laboratory results were as follows: hemoglobin 9.1 g/dL; red blood count $1.45 \times 10^6/\text{mm}^3$; white cell count $3.2 \times 10^3/\text{mm}^3$; platelet count $94 \times 10^3/\text{mm}^3$; blood urea nitrogen 94 mg/dL; serum creatinine 10.47 mg/dL (patient had a baseline creatinine level of 1.5 mg/dL); sodium 130 mEq/L; potassium 6.2 mEq/L; bicarbonate 14 mEq/L; total protein 7.0 g/dL; albumin 2.9 g/dL; alkaline phosphatase 57; AST and ALT normal; lipase 102 U/L; urine analysis showed 2+ protein with bland urine sediment and microscopic hematuria (3 -6/HPF). 24-h urine protein was 2 g/day. Renal ultrasound was significant for a right renal 0.6 cm cyst. The patient was admitted to ICU and was subsequently hemodialyzed due to worsening hyperkalemia and acute kidney injury. Serologies were notable for positive anti-dsDNA antibody and low levels of C3 (46 mg/dL) with normal C4 were observed. Immunofixation by electrophoresis showed free lambda. Serum plasma electrophoresis showed two M-spikes: Lambda light chains and IgG Lambda. A renal



biopsy was performed and cast nephropathy was identified with mesangial staining for C3. Bone marrow biopsy was performed and showed CD 56-positive plasma cell myeloma. Patient was treated with Velcade, Cytosin, and dexamethasone. The patient was subsequently discharged on chemotherapy and intermittent hemodialysis therapy. Follow-up evaluation of the alternative complement pathway showed normal activity level. This case illustrated myeloma kidney associated with mesangial C3 deposition in glomeruli and C3 hypocomplementemia. Our hypothesis is that in monoclonal gammopathy induced C3 glomerulopathy; paraprotein itself is acting as a trigger that excessively activates and dysregulates the AC pathway systemically. Thus, it is highly feasible to tailor the treatment to reduce the amount of paraproteins in C3 glomerulopathy associated with myeloma kidney, as opposed to conventional treatment for C3 glomerulopathy such as plasma exchange, rituximab/eculizumab, etc. Recognizing of the association between C3 GN and MM is important because it can be used as potential marker for hematological malignancy as well as the potential effective treatment for C3 G associated MM.

Biography:

Asif Khan is a pediatric Nephrologist from Shaare Zedek Medical Center, Israel

Recent Publications:

1. J Ped. Nephrology 2017;5(3)

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