

Not All Casts Belong to Myeloma: An Unusual Renal Face of Waldenström Macroglobulinemia

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Waldenström macroglobulinemia (WM) is an indolent lymphoplasmacytic lymphoma characterized by bone marrow infiltration associated with an IgM monoclonal gammopathy. Renal involvement is rare, affecting approximately 3% of patients, and displays significant histological heterogeneity (1). The most frequently reported renal lesions include intracapillary IgM deposits (pseudothrombi), cryoglobulinemia-associated membranoproliferative glomerulonephritis, and AL amyloidosis (2). In contrast, cast nephropathy, which is classically associated with multiple myeloma (MM), is an exceptional manifestation of WM. Here, we present the case of an 80-year-old patient who develops an acute kidney injury related to a light chain nephropathy associated with WM.

The patient was referred to the emergency department following the discovery of acute kidney dysfunction during a work-up for asthenia and anorexia without any other associated symptoms. Serum creatinine level was 3.37 mg/dL, compared to 0.90 mg/dL one year earlier. Laboratory investigations revealed a mild inflammatory syndrome (CRP level of 18 mg/L), normocytic non-regenerative anemia (hemoglobin level of 11.8 g/dL), and normal LDH levels. Serum protein electrophoresis revealed an IgM kappa monoclonal gammopathy of 7.6 g/L, with serum kappa free light chain at 711 mg/L and a κ/λ ratio of 33.89.

Urine analysis showed proteinuria at 830 mg/L with a protein-to-creatinine ratio of 1.025 g/g, albuminuria at 140.4 mg/L with an albumin-to-creatinine ratio of 175.5 mg/g. Additionally, there was a significant increase in urinary β 2-microglobulin levels (>4000 mg/L), and immunofixation identified a monoclonal component consisting of large amounts of free kappa light chains.

Positron emission tomography-computed tomography (PET-CT) showed no lytic bone lesions, lymphadenopathy, or organomegaly.

Bone marrow aspirate showed a hypercellular marrow infiltrated by approximately 37% of a lymphoplasmacytic population, with on immunophenotyping, a monoclonal B-cell population restricted to kappa light chains, expressing CD19, CD20, CD79, CD5, FMC7, CD43, CD38, CD24, CD25, CD27, CD148, and CD200, with no expression of CD23, CD10, CD11c, CD103, CD123, or CD180. Plasma cells accounted for approximately 4% of the bone marrow cellularity.

Because of the unusual presentation of the acute renal failure, a kidney biopsy was performed which demonstrated cast nephropathy, a condition characterized by intratubular kappa light chain casts associated.

Initial management included intravenous hydration, which resulted in a rapid improvement of the renal function, evidence by a decrease in serum creatinine from 3.37 to 1.91 mg/dL within one week. Dexamethasone pulse therapy (20 mg daily for 4 days) was followed by a bortezomib-dexamethasone-rituximab regimen (2–3). The advantage of bortezomib is that it does not require dose adjustment in patients with renal impairment. After the first treatment cycle, we note a significant improvement of the renal function with serum creatinine decreasing to 1.53 mg/dl. Parallely, proteinuria decreased to 200 mg/L, with a protein-to-creatinine ratio of 0.351 g/g. We expect a similar reduction in the free light chain component, but the results are pending.

This case highlights the importance of considering light chain cast nephropathy in patients with rapidly progressive kidney dysfunction and monoclonal gammopathy, including IgM monoclonal disorders such as Waldenström macroglobulinemia. Kidney biopsy remains essential for diagnosis and therapeutic guidance. Prompt treatment of the underlying hematological disorder is crucial to reduce monoclonal protein production and improve renal outcomes.

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