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Case Report

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Atypical MGRS-associated renal injury in Waldenström macroglobulinemia: occult intraluminal monoclonal κ light chain deposition, a case report

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Abstract

Background Waldenström macroglobulinemia (WM) is a rare, indolent lymphoplasmacytic lymphoma. Renal involvement in WM can be attributed to three mechanisms: monoclonal protein deposition, direct tumor cell infiltration, and indirect injury unrelated to deposition or infiltration, with AL amyloidosis being the most common clinical presentation. We report a case of atypical monoclonal gammopathy of renal significance (MGRS) renal injury associated with WM.

Case presentation A 58-year-old man with a 5-year history of asymptomatic WM was admitted with foamy urine, nocturia, dizziness, and blurred vision. Physical examination revealed pallor, a diffuse purplish-red non-blanching rash over the entire body, and no bilateral lower extremity edema. Laboratory tests showed elevated total protein, globulin, IgG, and IgM; increased serum creatinine; and mildly reduced hemoglobin. Serum protein electrophoresis combined with serum and urine immunofixation electrophoresis identified a monoclonal IgM-κ band. Bone marrow examinations were consistent with the WM disease spectrum, and imaging revealed multiple enlarged extramedullary lymph nodes. Renal biopsy demonstrated occult monoclonal κ light-chain deposition within vascular lumens accompanied by focal glomerular ischemia, confirming the diagnosis of atypical MGRS renal injury associated with WM.

Conclusions Renal injury secondary to WM exhibits substantial pathological heterogeneity, and renal biopsy remains the cornerstone for subtyping and definitive diagnosis. This case may provide a reference for the clinical recognition of this disorder.

Keywords Waldenström macroglobulinemia; Monoclonal gammopathy of renal significance; Light-chain deposition; Renal biopsy

Introduction

Waldenström macroglobulinemia (WM) is a rare indolent lymphoplasmacytic lymphoma characterized by clonal proliferation of lymphoplasmacytic cells in the bone marrow and elevated serum IgM monoclonal immunoglobulin. The disease progresses slowly with an insidious early course. A considerable proportion of patients remain asymptomatic for a long time, with abnormal laboratory findings detected only during routine follow-up. As a systemic clonal hematologic disorder, WM can involve multiple organ systems, among which renal injury represents one of its important complications. WM-associated renal injury exhibits marked pathological heterogeneity, and its pathogenetic mechanisms fall into three categories: injury mediated by monoclonal immunoglobulin deposition, direct renal infiltration by tumor cells, and indirect renal injury without immunoglobulin deposition or tumor infiltration, resulting in a complex

clinicopathological spectrum. At present, AL amyloidosis is regarded as the most common form of renal involvement in WM. Herein, we report a middle-aged and elderly male patient with a 5-year history of asymptomatic WM, whose initial manifestations included foamy urine, nocturia, dizziness and blurred vision. Combined with laboratory tests, serum immunofixation electrophoresis, bone marrow aspiration, imaging examinations and renal biopsy pathology, occult monoclonal κ light-chain deposition within vascular lumens together with focal glomerular ischemia was identified on renal biopsy. The patient was ultimately diagnosed with atypical MGRS renal injury secondary to WM. Cases of this type lack distinctive clinical manifestations and typical pathological changes and may occur in patients with longstanding asymptomatic WM. By systematically summarizing the clinical and pathological features of this case, we aim to further enrich the disease spectrum of renal involvement in WM.

Case report

A 58-year-old male patient presented with a 5-year history of generalized non-pruritic, non-blanchable erythematous rash with progressive brown pigmentation, without blistering, ulceration, or exudation. Previous laboratory examinations revealed elevated serum total protein and globulin with normal baseline renal function and urinalysis. Hematological workup showed increased serum IgG and IgM levels, while bone marrow cytology and biopsy demonstrated mild expansion of plasmacytoid lymphocytes and lymphoproliferation, consistent with a diagnosis of Waldenström macroglobulinemia, for which the patient received no specific treatment. Ten days prior to admission, the patient developed new-onset foamy urine, nocturia (2–4 episodes per night), nocturnal bilateral lower extremity spasms, dizziness, and blurred vision, without gross hematuria, oliguria, fever, or gastrointestinal symptoms. The patient had a prior history of cholecystectomy, benign rectal polyp resection, and chronic prostatitis, but no hypertension, diabetes, or infectious diseases.

Physical examination on admission: temperature 36.5°C, heart rate 97 beats/min, respiratory rate 20 breaths/min, blood pressure 94/66 mmHg (1 mmHg = 0.133 kPa), BMI 16.68 kg/m^2. The patient presented with anemic features, pale conjunctivae and nail beds. Lung auscultation revealed clear bilateral breath sounds; cardiac rhythm was regular with no pathological murmurs. The abdomen was soft and non-tender, and the liver and spleen were not palpable below the costal margin. Mild bilateral lower-extremity edema was noted. Laboratory findings: complete blood count showed hemoglobin 83 g/L (decreased), red blood cell count 3.17×10^{12}/L (decreased), white blood cell count 5.75×10^{9}/L, and platelet count 319×10^{9}/L. Urinalysis: pH 6.0, specific gravity 1.017, trace protein, negative for red blood cells, white blood cells and

glycosuria. Urinary renal function panel: urinary microalbumin 8.6 mg/L, urinary microalbumin/creatinine ratio 0.57 mg/mol, α1-microglobulin 19.3 mg/L (elevated, reference <12 mg/L), urinary β2-microglobulin 0.255 mg/L (elevated, reference <0.23 mg/L). Twenty-four-hour urinary protein quantification was 120 mg. Serum biochemistry: albumin 24.6 g/L (decreased, reference 40–55 g/L), globulin 49.9 g/L (elevated, reference 20–40 g/L), blood urea nitrogen 4.71 mmol/L, creatinine 265 μmol/L (elevated, reference 52–101 μmol/L), uric acid 363 μmol/L, calcium 2.4 mmol/L. Cardiac injury panel: troponin I 0.002 ng/mL, pro-BNP 235.1 pg/mL. ANCA, anti-GBM antibody and anti-phospholipase A2 receptor antibody were negative; autoantibodies, anti-streptolysin O and rheumatoid factor were negative. Thyroid function and complement levels were normal. Immunoglobulin panel: IgG 13.4 g/L, IgA 0.651 g/L (decreased, reference 1.0–4.2 g/L), IgM 47.1 g/L (elevated, reference 0.3–2.2 g/L). Coagulation tests and cryoglobulin were negative; urine Bence Jones protein was negative. Serum β2-microglobulin 4.71 mg/L (elevated, reference 1.0–2.3 mg/L). Serum free light chain assay: kappa 215 mg/L, lambda 27.3 mg/L, kappa/lambda ratio 7.875 (reference 0.26–1.65). Serum relative viscosity 3.7 (elevated, reference 1.4–1.8). Serum protein electrophoresis demonstrated gamma fraction 45.8% (elevated, reference 1.1–18.8%). Serum immunofixation electrophoresis showed a dense band of IgM-kappa monoclonal immunoglobulin; M-protein percentage 23.8%, M-protein concentration 24.2 g/L. Urine immunofixation electrophoresis revealed a dense band of kappa monoclonal immunoglobulin. Chest CT showed minor fibrotic streaks in the bilateral lower lobes. Abdominal ultrasound demonstrated hepatic cysts, status post cholecystectomy with no dilatation of intrahepatic or extrahepatic bile ducts; the pancreas and spleen were normal in size without abnormalities. Both kidneys were normal in size with no remarkable imaging findings; bilateral ureters and bladder were unremarkable. The prostate was slightly enlarged with prostatic calcification. Electrocardiogram showed normal sinus rhythm. Echocardiography revealed normal dimensions of cardiac chambers and great vessels; left ventricular diastolic dysfunction with preserved systolic function, and no pathological valvular regurgitation on color Doppler flow imaging.

Bone marrow biopsy report: markedly hypercellular bone marrow with diffuse interstitial proliferation of lymphocytes, predominantly mature-stage lymphocytes that are relatively small in size and regular in morphology; plasma cells are mildly increased, distributed scatteredly or in clusters, with binucleated plasma cells visible. Interstitial edema is present, with focal proliferation of fibrous tissue. Gomori stain: MF-1. Bone marrow cell examination report: plasmacytoid lymphocytes account for 11.2%. Lymph node ultrasound: enlarged lymph nodes in the bilateral inguinal regions, bilateral axillae, and left cervical level IV; no obvious enlarged lymph nodes are seen in the right

paratracheal great vessel region, bilateral submandibular regions, or submental region.

After admission, renal puncture biopsy was performed (Figure 1): Routine frozen immunofluorescence (4G): IgG, IgA, IgM, C3, C4, C1q, κ, λ, AA, IgG1–IgG4, and PLA2R were all negative. Immunofluorescence after paraffin antigen retrieval: diffuse monoclonal κ light chain-positive deposits were observed in the glomerular capillary loops and renal interstitial capillary lumina, while λ light chain was negative; IgG, IgA, IgM, C3, and C1q were all negative. Light microscopy: 37 glomeruli were identified, of which 7 showed ischemic sclerosis, while the remaining glomeruli showed no obvious morphological abnormalities; renal interstitial capillaries were dilated, with no obvious lymphoplasmacytic infiltration, tubular atrophy, or interstitial fibrosis, and no pseudothrombi-like material in the glomerular lumina. Electron microscopy: no characteristic ultrastructural abnormalities such as amyloid fibrils, microtubules, crystals, or dense deposits in the mesangium or basement membrane were observed. Pathological diagnosis: Waldenström macroglobulinemia-related atypical MGRS renal injury: cryptic monoclonal κ light chain deposition within vascular lumina, accompanied by focal glomerular ischemic injury.

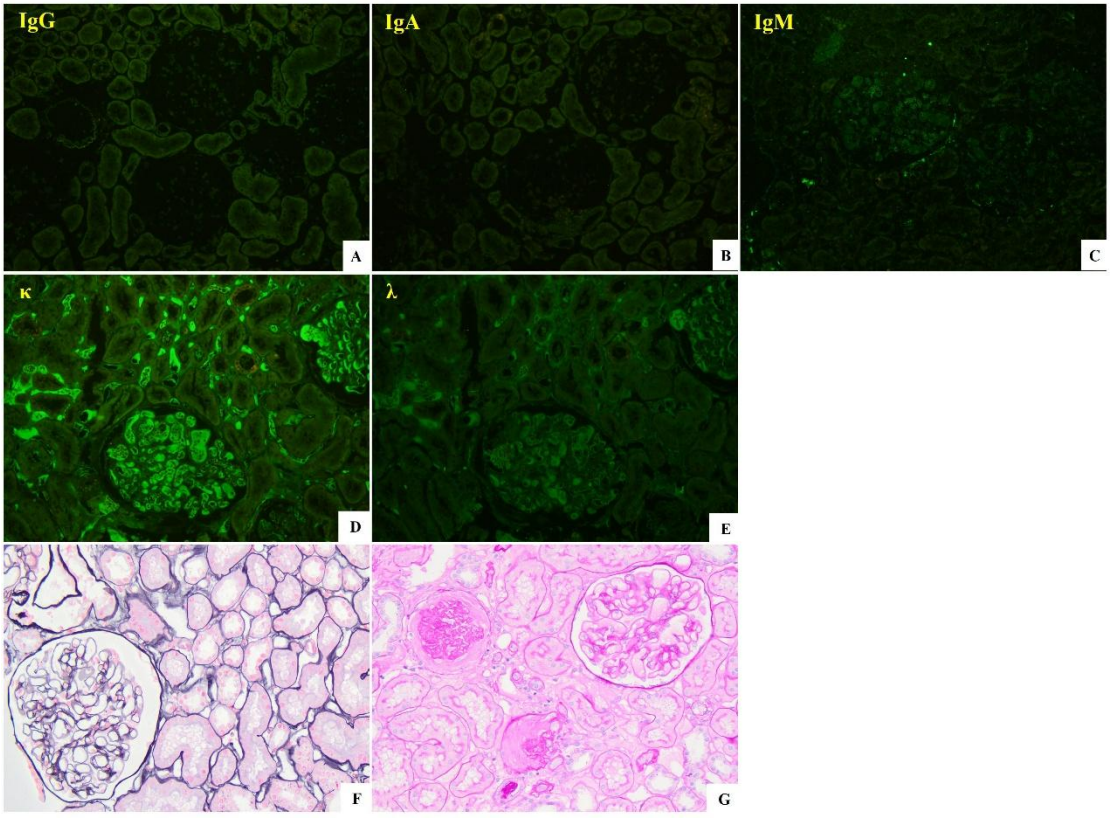


Figure 1. Renal pathological findings: A–E, immunofluorescence after paraffin antigen retrieval showing diffuse deposition of monoclonal κ light chain within glomerular capillary loops and renal interstitial capillaries; IgG, IgA, IgM and λ light chain were negative (IF, ×200). F, No intracapillary pseudothrombi were observed in glomeruli, whereas dilatation of renal interstitial capillaries was

present (PASM-Masson, ×200). G, Focal ischemic sclerosis was seen in one glomerulus, and the remaining glomeruli were largely unremarkable (PAS, ×200).

The patient had serum monoclonal IgM gammopathy, bone marrow pathology consistent with a lymphoplasmacytic neoplasm spectrum, and extramedullary involvement manifested by generalized superficial lymphadenopathy in multiple regions, confirming the diagnosis of Waldenström macroglobulinemia. With regard to renal disease, the patient presented with insidious renal function decline without heavy proteinuria. Renal biopsy revealed focal ischemic renal injury, and paraffin antigen-retrieved immunofluorescence demonstrated diffuse occult monoclonal κ light-chain deposition within vascular lumens. These findings did not fulfill the diagnostic criteria for monoclonal immunoglobulin deposition disease (MIDD). Final diagnoses were as follows: 1. Waldenström macroglobulinemia (IgM-κ type, lymphoplasmacytic lymphoma); 2. WM-associated renal injury: (i) intravascular occult monoclonal κ light-chain deposition; (ii) focal glomerular ischemic changes. During hospitalization, rituximab (1000 mg once every two weeks) was administered after discussion with the patient and consideration of his preference. Supportive treatments including anemia correction, renal function preservation, and prophylaxis with co-trimoxazole for infection prevention were also initiated. The patient returned to our department for follow-up two weeks later for the second rituximab infusion. Repeat laboratory tests showed that urinary α1-microglobulin increased to 34.7 mg/L, serum creatinine rose to 352 μmol/L, albumin was 31.9 g/L, globulin 68 g/L, and IgM 51 g/L. Total B cells accounted for 2%. Serum immunofixation electrophoresis showed a dense band of IgM-kappa monoclonal immunoglobulin, with an M-protein percentage of 28.6% and an M-protein concentration of 28.57 g/L. Monotherapy with rituximab yielded a poor response, with no hematologic response and elevated monoclonal IgM as well as serum creatinine. Digital droplet PCR (ddPCR) testing detected a positive MYD88 L265P mutation. The patient declined plasmapheresis. In consultation with the hematology team, the regimen was switched to zanubrutinib 160 mg twice daily, and regular follow-up at the hematology outpatient clinic was recommended.

Discussion

Waldenström macroglobulinaemia (WM) is a rare, indolent lymphoplasmacytic lymphoma characterized by bone marrow infiltration by lymphoplasmacytic cells and a serum monoclonal IgM paraprotein, and is clinically classified into asymptomatic and symptomatic disease [1,2]. Asymptomatic WM is defined by the presence of monoclonal IgM and bone marrow involvement without disease-related organ dysfunction, and does not require immediate systemic therapy. Symptomatic WM is diagnosed when tumour infiltration causes lymphadenopathy, hepatosplenomegaly or

cytopenias, or when the monoclonal IgM causes hyperviscosity, amyloidosis or other organ damage [3,4]. MYD88 L265P, the most common molecular abnormality (approximately 90%–95% of patients), is useful for diagnosis and differential diagnosis and may also help predict response to Bruton tyrosine kinase (BTK) inhibitors, particularly when interpreted together with CXCR4 mutation status [5–7].

The patient in the present case initially had low-burden, indolent asymptomatic WM and was managed with observation. During long-term follow-up, the proportion of plasmacytoid lymphocytes in the bone marrow increased to 11.2%, and new renal impairment developed in the context of a positive MYD88 L265P mutation and a marked increase in serum IgM, indicating progression to symptomatic disease. Importantly, the transition to symptomatic disease was not accompanied by a dramatic increase in bone marrow tumour burden; instead, renal dysfunction and symptoms suggestive of hyperviscosity syndrome became the clinically dominant manifestations. Renal involvement in WM is uncommon but clinically important. In a retrospective Dana-Farber cohort of 1391 patients with WM, biopsy-confirmed WM-related nephropathy occurred in 44 patients, with an estimated cumulative incidence of 5.1% at 15 years, and was associated with shorter median overall survival, particularly when renal function failed to stabilise or improve after treatment [8]. More recently, a nationwide Italian multicentre study of symptomatic WM reported renal dysfunction, defined as a creatinine clearance below 60 mL/min/1.73 m^2, in 29.6% of patients at diagnosis, associated with inferior overall and progression-free survival [9]. Taken together, these data indicate that renal impairment is not merely a comorbidity in WM but may represent an adverse prognostic feature and a clinically relevant marker of active disease.

WM-related renal disease is pathologically heterogeneous, and the main mechanisms include (i) glomerular injury caused by deposition or precipitation of monoclonal immunoglobulins; (ii) direct renal infiltration by lymphoplasmacytic lymphoma cells; and (iii) indirect injury related to haemodynamic or paraneoplastic mechanisms [8,10–13]. Deposition-related lesions include AL or AH amyloidosis, monoclonal immunoglobulin deposition disease, immunotactoid or fibrillary glomerulopathy, proliferative glomerulonephritis with monoclonal immunoglobulin deposits, and cryoglobulinaemic or non-cryoglobulinaemic membranoproliferative glomerulonephritis [10–13]; among these, amyloidosis is a frequent pathological pattern and commonly presents with nephrotic-range proteinuria [8,10,13]. Direct

tumour-cell infiltration may involve the renal interstitium and result in interstitial inflammation, tubular injury and progressive renal dysfunction [14,15]. Indirect mechanisms include reduced renal perfusion in the setting of hyperviscosity, thrombotic microangiopathy and paraneoplastic glomerular disease [8,16]. These mechanisms are not mutually exclusive and may coexist in the same patient, and clinical or serological findings alone cannot reliably determine the underlying renal lesion. Renal biopsy therefore remains essential when the cause of renal impairment is uncertain, particularly in patients with unexplained acute or progressive kidney dysfunction, significant proteinuria or suspected monoclonal immunoglobulin-related disease [8,17].

The present case illustrates a combined and atypical pattern of WM-associated renal injury. The patient developed a marked increase in serum IgM together with dizziness and cutaneous petechiae, findings clinically consistent with hyperviscosity-related manifestations. Renal biopsy demonstrated occult deposition of monoclonal κ light chains within glomerular capillary loops and the lumina of interstitial capillaries, accompanied by focal ischaemic glomerular injury; no typical pattern of widespread interstitial tumour infiltration or conventional tubular cast nephropathy was identified. These pathological findings cannot be adequately explained by either isolated monoclonal light-chain deposition or isolated renal ischaemia. A more coherent interpretation is that hyperviscosity-related impairment of the renal microcirculation was superimposed on direct endothelial and microvascular injury caused by monoclonal κ light chains. IgM is a high-molecular-weight pentameric immunoglobulin that can increase plasma viscosity and impair microvascular blood flow [16]. The focal ischaemic glomerular changes provide morphological support for a haemodynamic component, while the accumulation of monoclonal light chains within the renal microcirculation may have caused endothelial injury and further disturbed local blood flow. These processes may reinforce one another, resulting in a self-perpetuating cycle of microvascular stasis, ischaemia and progressive renal dysfunction.

This lesion also raises an important issue concerning the spectrum of monoclonal gammopathy of renal significance (MGRS). MGRS was originally proposed to describe renal diseases caused by nephrotoxic monoclonal immunoglobulins produced by a B-cell or plasma-cell clone that does not otherwise meet criteria for overt haematological malignancy requiring treatment [18,19]. The defining clinical principle is that the monoclonal gammopathy is directly responsible for renal injury and that clone-directed therapy may be required to preserve renal function [18,19]. Classical MGRS lesions are generally classified according to the type and location of monoclonal immunoglobulin deposition, including glomerular, tubular, interstitial and vascular patterns. In the present case, however, the monoclonal κ light chains were inconspicuously located

within capillary lumina and glomerular capillary loops, without a conventional pattern of extensive interstitial deposition or tubular cast formation, a phenotype difficult to place within the usual categories of monoclonal immunoglobulin-related renal disease. Although the patient had symptomatic WM rather than a small, otherwise indolent clone meeting the traditional conceptual framework of MGRS, the case shares the central pathophysiological principle of monoclonal immunoglobulin-mediated kidney injury. It may therefore be more appropriate to regard this lesion as an atypical WM-associated monoclonal immunoglobulin-related microvascular injury rather than force it into a single conventional MGRS category.

The clinical course also has implications for the surveillance of asymptomatic WM. Observation without immediate treatment remains an accepted strategy for patients without disease-related organ dysfunction [2,4,20]. However, disease progression does not necessarily parallel a substantial increase in marrow tumour burden, as organ injury may be an earlier indicator of clinically meaningful progression than the absolute change in marrow infiltration. Routine follow-up of patients with asymptomatic WM should therefore include not only serum IgM and haematological parameters but also renal function and urinary indices, including serum creatinine, estimated glomerular filtration rate, urinalysis, urinary protein quantification, serum protein electrophoresis, immunofixation and serum free light-chain measurements; patients with suspected cryoglobulinaemia, amyloidosis or other monoclonal immunoglobulin-mediated complications should undergo appropriate targeted testing [10,13,19]. When renal impairment is otherwise unexplained, early kidney biopsy should be considered because the histological diagnosis may directly influence the choice and urgency of clone-directed treatment [17].

In conclusion, WM-associated renal injury is uncommon but is associated with adverse clinical outcomes. Its pathogenesis is heterogeneous and may involve monoclonal immunoglobulin deposition, direct lymphoplasmacytic infiltration, hyperviscosity-related haemodynamic injury, thrombotic microangiopathy and paraneoplastic mechanisms, which may coexist. The present case, characterized by occult intravascular monoclonal κ light-chain deposition together with focal ischaemic glomerular injury, represents an atypical form of WM-associated renal disease. It highlights the need to integrate serum monoclonal protein burden, rheological manifestations, renal function and renal histopathology rather than attributing kidney injury to a single mechanism. Renal impairment and proteinuria should also be regarded as potential early warning signs of progression from asymptomatic to symptomatic WM, supporting their incorporation into routine long-term surveillance.

Declarations

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Ethics approval and consent to participate

Ethical approval was obtained from the Medical Ethics Committee of the First Affiliated Hospital of the Air Force Medical University. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Disclosure Statement

The authors declare that they have no conflict of interest.

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Author Contributions

Di Wang was involved in the diagnosis and clinical management of the patient, collected and analyzed the clinical data, prepared the figures, and drafted the manuscript. Xiayin Li and Guoshuang Xu contributed to the patient's clinical management and data collection. Shiren Sun and Xiujuan Tian supervised the diagnosis and treatment, and revised the manuscript critically for important intellectual content. All authors read and approved the final manuscript.

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