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CASE REPORT

Proliferative glomerulonephritis with monoclonal immunoglobulin G deposits in a young woman: A case report

Zi-Gan Xu, Wei-Long Li, Xi Wang, Shu-Yuan Zhang, Ying-Wei Zhang, Xing Wei, Chun-Di Li, Ping Zeng, Shao-Dong Luan

ORCID number: Zi-Gan Xu 0000-0002-3493-8625; Wei-Long Li 0000-0001-8570-4274; Xi Wang 0000-0001-7837-3075; Shu-Yuan Zhang 0000-0003-3075-6709; Ying-Wei Zhang 0000-0001-9736-6583; Xing Wei 0000-0002-5025-3941; Chun-Di Li 0000-0002-1143-2410; Ping Zeng 0000-0002-4022-2551; Shao-Dong Luan 0000-0003-1176-4009.

Author contributions: Xu ZG reviewed the literature and contributed to manuscript drafting; Li WL, Wang X, Zhang SY, Zhang YW, Li CD, and Zeng P collected the information from the patient; Wei X and Luan SD were surgeons for biopsy and revised the manuscript; Luan SD supervised the study; all authors read and approved the final manuscript.

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Zi-Gan Xu, Wei-Long Li, Xi Wang, Shu-Yuan Zhang, Ying-Wei Zhang, Xing Wei, Chun-Di Li, Ping Zeng, Shao-Dong Luan, Department of Nephrology, Shenzhen Longhua District Central Hospital, Shenzhen 518110, Guangdong Province, China

Corresponding author: Shao-Dong Luan, PhD, Chief Doctor, Department of Nephrology, Shenzhen Longhua District Central Hospital, No. 187 Guanlan Road, Shenzhen 518110, Guangdong Province, China. szkidney3@163.com

Abstract

BACKGROUND

Proliferative glomerulonephritis with monoclonal immunoglobulin G (IgG) deposits (PGNMID) is a newly recognized rare disease. The renal pathology is characterized by prominent manifestations of membranous hyperplasia, which are easy to misdiagnose. The clinical symptoms are severe. Massive proteinuria and hypoproteinemia are conspicuous, and most patients are accompanied by renal insufficiency and microscopic hematuria.

CASE SUMMARY

A 27-year-old woman was admitted to a hospital for macroscopic hematuria and proteinuria 4 years prior, and renal biopsy in the hospital suggested moderate-to-severe mesangial proliferating glomerulonephritis (MsPGN). She had taken a glucocorticoid, cyclophosphamide, mycophenolate mofetil, and other treatments and achieved brief partial remission. Recently, the patient visited our hospital due to massive proteinuria. Repeated renal biopsy and re-evaluation of the first biopsy obtained 4 years previously revealed monoclonal immunoglobulin deposition in the glomeruli. A bone marrow examination was performed to exclude hematologic malignancy, and a diagnosis of PGNMID was established. The patient showed remission after four cycles of a bortezomib + cyclophosphamide + dexamethasone scheme.

CONCLUSION

PGNMID is usually misdiagnosed as MsPGN or membranoproliferative glomerulonephritis. Although it often occurs in middle-aged and elderly individuals, it cannot be readily excluded in young people, even when serum immunofixation electrophoresis is negative. IgG subtype and light chain staining are necessary when this disease is highly suspected. An accurate diagnosis at the earliest stage may avoid the overuse of glucocorticoids and immunosuppressants.

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Core Tip: Proliferative glomerulonephritis with monoclonal immunoglobulin G deposits (PGNMID) is a rare renal disease whose pathogenesis is not fully understood. Due to its complexity, this disease is easy to misdiagnose. We report the case of a young female patient with gross hematuria and foamy urine. Renal biopsy in another hospital suggested mesangial proliferating glomerulonephritis (MsPGN). Her condition did not improve significantly after treatment with glucocorticoids and immunosuppressants. Recently, repeated renal biopsy at our hospital suggested PGNMID. This case suggests that although PGNMID often occurs in middle-aged and elderly individuals, it cannot be readily excluded in young people with the pathological type of membranoproliferative glomerulonephritis or MsPGN, even when immunofixation electrophoresis is negative. An accurate diagnosis at the earliest stage may minimize the use of glucocorticoids and immunosuppressants.

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INTRODUCTION

Proliferative glomerulonephritis with monoclonal immunoglobulin G (IgG) deposits (PGNMID), first reported by Nasr *et al*[1] in 2004, is a newly recognized disease characterized by glomerular monoclonal IgG deposition, and histopathological manifestations of renal biopsy show glomerular proliferative lesions. A total of 56.8% of PGNMID patients have been diagnosed with membranoproliferative glomerulonephritis (MPGN) based on light microscopy, with prominent manifestations of membranous hyperplasia[2]. Due to its complexity, the pathogenesis of PGNMID is not fully understood.

The disease is commonly observed in middle-aged and elderly people, among whom 65% are over 50 years old[2]. The incidence of PGNMID diagnosis by autologous kidney biopsy is approximately 0.07%-0.17%[2,3]. The main clinical manifestations of PGNMID include proteinuria, hematuria, and renal insufficiency.

We report a young female patient who was diagnosed with mesangial proliferating mesangial proliferating glomerulonephritis (MsPGN) due to proteinuria in another hospital, with poor therapeutic effects. She was recently admitted to our hospital, and repeated renal biopsy suggested PGNMID.

CASE PRESENTATION

Chief complaints

A 27-year-old Chinese woman with hematuria and albuminuria for over 4 years was admitted to our nephrology department on December 15, 2019

History of present illness

After 1 mo of gross hematuria and frothy urine, the patient visited her local hospital for treatment in June 2015. Laboratory examinations showed the following: 24 h urine protein (UP) 3.09 g/d; serum albumin, 24.6 g/L; serum creatinine (Scr), 120 μmoL/L; immunofixation electrophoresis (IFE), serum protein electrophoresis (SPEP), and autoimmune markers, including anti-neutrophil cytoplasmic antibody, antinuclear antibody, anti-double stranded DNA, and anti-glomerular basement membrane antibody, that were negative. Histological examination of the renal biopsy at that time

revealed moderate-to-severe MsPGN with crescent formation. The patient was given methylprednisolone injections (500 mg/d, 3 d in total, then 40 mg/d). A few days later, she was discharged, stopped taking the drugs on her own and then went to a private clinic in Hong Kong for further treatment. She had taken prednisone and cyclophosphamide (CTX) for 3 mo; CTX was then replaced by mycophenolate mofetil (MMF) (the doses are unknown). This treatment was applied for 2 years, after which MMF was substituted with tacrolimus. The dosages were gradually reduced and then withdrawn on August 28, 2018, as prescribed. During this time, her 24 h UP fluctuated between 2 and 3 g/d, and her Scr level was in the 80 to 110 μmoL/L range. The nephritic syndrome relapsed in January 2019, and the previous specialist treated her with prednisone (500 mg/d, 3 d in total) and CTX (100 mg/d). CTX was replaced by MMF (1 g/d) 1 mo later because of severe hair loss and irregular menstruation. Prednisone was reduced gradually and then withdrawn completely in October 2019. The patient underwent a checkup on December 15, 2019, and her laboratory data were as follows: Scr, 92 μmoL/L; serum albumin, 38.3 g/L; UP, 3+; urine occult blood, 3+.

History of past illness

The patient had no diagnosed history of metabolic disease, coronary heart disease, or liver disease.

Physical examination

Slight pitting edema was noted in both lower extremities. Other physical features included a mild anemic appearance. No swelling of surface lymph nodes was found.

Laboratory examinations

On admission, laboratory data revealed the following values: Hemoglobin (Hb), 71 g/L; Scr, 94 μmoL/L; serum uric acid, 528 μmoL/L; serum albumin, 24.6 g/L; globulin, 19.7 g/L; calcium, 1.97 mmoL/L; complement C3, 0.84 g/L; C4 0.19 g/L; UP, 2+; urine occult blood, 2+; 24 h UP, 1.1 g/d. Liver function was normal. We detected no anti-DNA, anti-glomerular basement membrane, or anti-neutrophil cytoplasmic antibodies and no hepatitis B virus, hepatitis C virus (HCV), or human immunodeficiency virus. IFE and SPEP did not show any paraproteins, although the urine free light chain ratio (κ/λ) was as high as 6.7974. Computed tomography of the chest, abdomen, and pelvis detected no enlarged lymph nodes.

Imaging examinations

No obvious abnormality.

Second renal biopsy

The histological examination of the renal biopsy showed 27 glomeruli, 11 of which were globally sclerotic and 3 of which were segmentally sclerotic; no crescent was found. Marked mesangial cells and matrix proliferation were observed in the remaining glomeruli, with glomerular lobulation and focal endothelial hyperplasia. Segmental basement membrane thickening, mesangial interposition, and tram track signs were visible. Endocapillary proliferative changes were noted by electron microscopy. The glomerular capillary loops were compressed, and the lumen was stenotic. Segmental mesangial matrix interposition was found. Furthermore, electron-dense deposits were observed in the subendothelial and mesangial areas; at the ultrastructural level, the deposits were not fibrillary or microtubular structures. Part of the interstitial region was infiltrated by inflammatory cells (Figure 1).

Immunohistology revealed positive petaloid deposition of IgG (3+), C3 (2+), IgM (1+), and C1q (+/-) along the capillary loops; IgA and fibrinogen (Fib) were negative. According to additional tests, IgG3 (3+), Kappa (3+), Lambda (1+), IgG1, IgG2, IgG4, phospholipase A2 receptor, thrombospondin type-1 domain-containing 7A, ascorbic acid, HCV, hepatitis B surface antigen, hepatitis B e antigen, hepatitis B core antigen, and Congo red and oxide Congo red staining were positive (Figure 2).

Re-evaluation of the patient's first biopsy

The clinicians who previously treated this patient kindly provided the glass slides of her first renal biopsy specimens. To determine the nature of the deposits, cryosections were freshly prepared for light chain and IgG subclass staining, and subsequent immunofluorescence analyses of these samples confirmed monoclonal deposition of IgG3κ in the glomeruli (Figure 3).

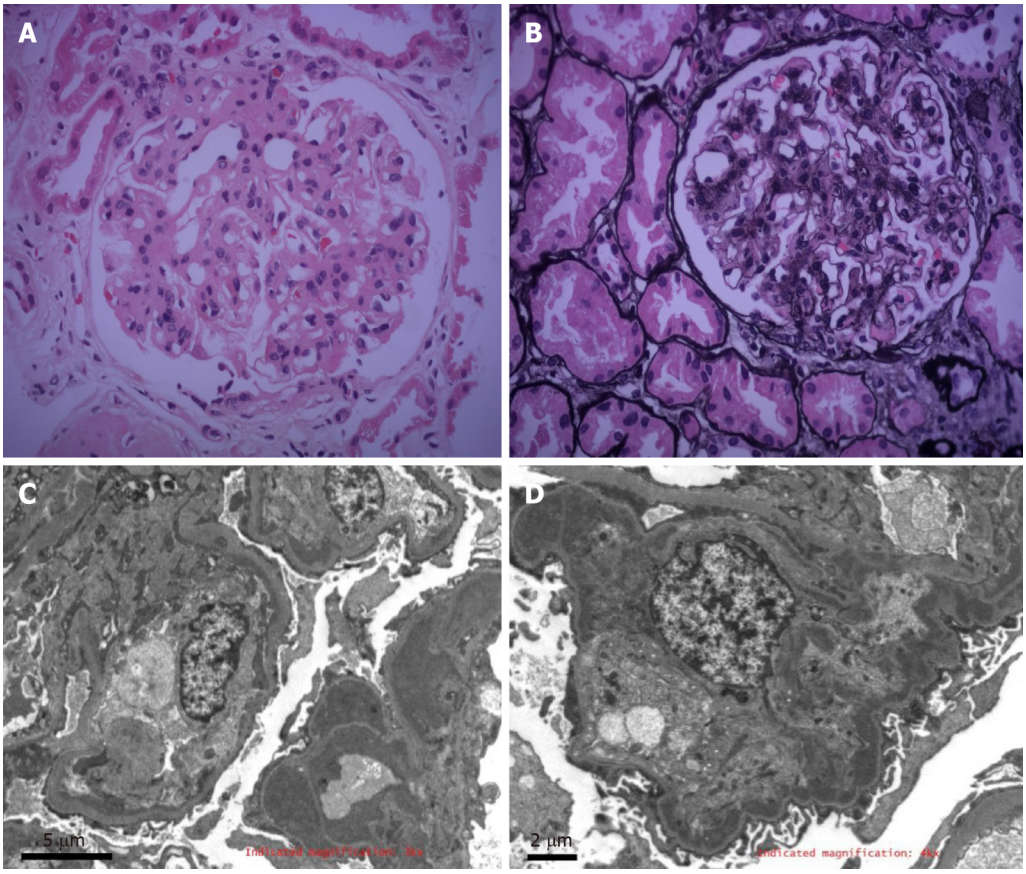

Figure 1 Part of the interstitial region was infiltrated by inflammatory cells. A and B: Section of the kidney obtained at biopsy showed proliferative lesions of the glomeruli (A: Hematoxylin and eosin staining; B: Periodic acid-Schiff-methenamine silver staining). Original magnification × 400; C and D: Electron-dense deposits were observed in the subendothelial and mesangial areas by electron microscopy.

Hematologic examinations

Bone marrow smears showed hyperplasia of the granulocyte series, erythron series, and megakaryocytic series. Biopsy revealed active bone marrow proliferation, without any obvious mutant cells. A bone marrow flow cytometry immunofluorescence assay indicated no immunophenotypic abnormal evidence of multiple myeloma, acute leukemia, plasma cells, non-Hodgkin's lymphoma, or high-risk myelodysplastic syndrome. Multiple myeloma-associated gene mutation analysis, karyotype analysis of bone marrow chromosomes, and fluorescence *in situ* hybridization, including Vysis TP53/CEP17, cytocell RB1(13q14), Vysis IGH, and cytocell CKS1B/CDKN2C(P18), were all negative.

FINAL DIAGNOSIS

The patient was diagnosed with PGNMID in accordance with the monoclonal pattern of IgG3κ deposition found in both the first and second renal biopsy specimens.

TREATMENT

We corrected the previous diagnosis result to PGNMID and immediately initiated four cycles of a bortezomib (B) + cyclophosphamide (C) + dexamethasone (D) (BCD) scheme within 5 mo. The specific formula was as follows: B (1.3 mg/m^2) days 1, 4, 8, and 11 + C (0.3 g) days 1-4 + D (20 mg) days 1, 2, 4, 5, 8, 9, 11, and 12.

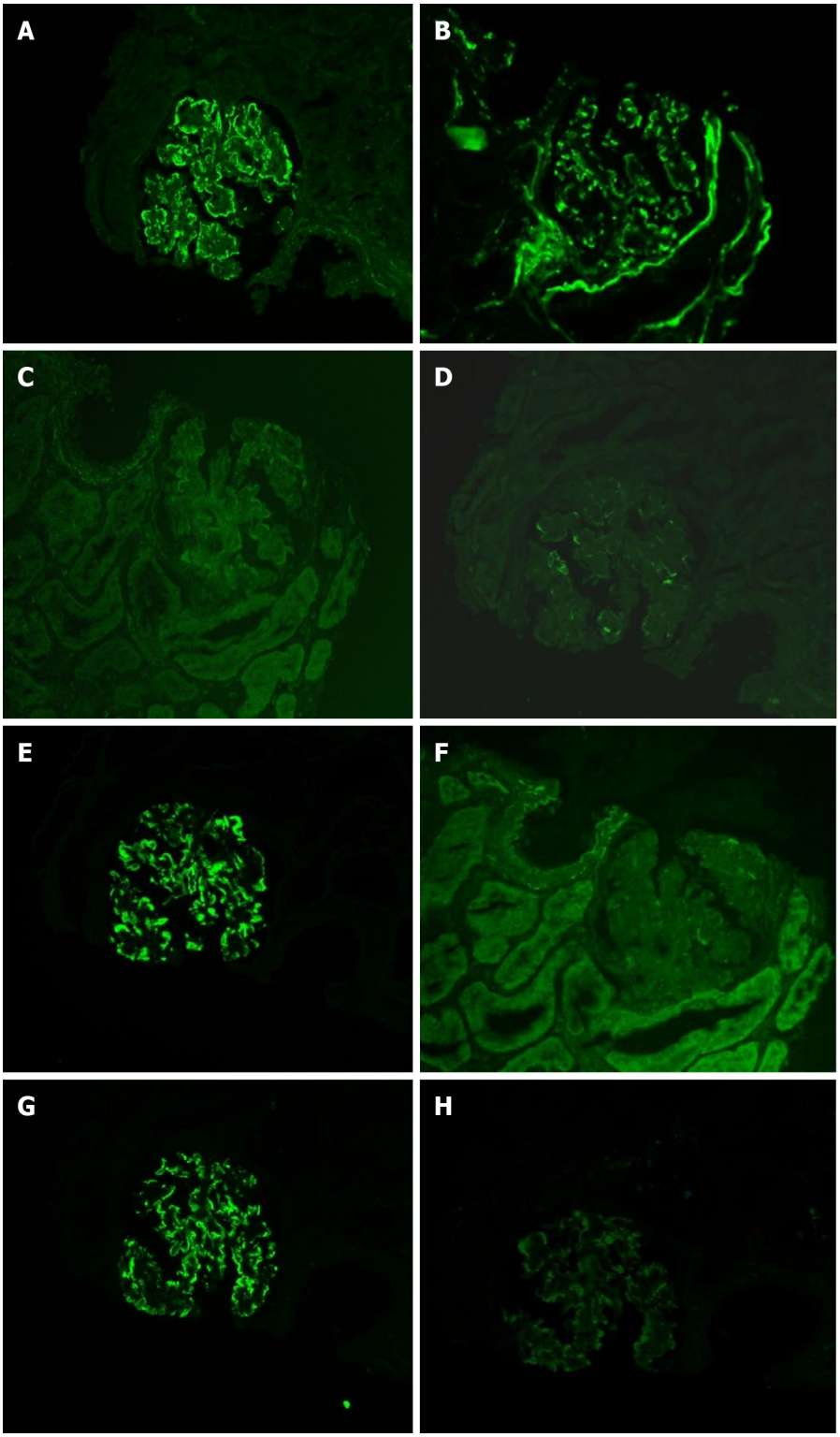

Figure 2 Immunofluorescence staining. A and B: Immunofluorescence staining revealed positive petaloid deposition of (A) immunoglobulin G (IgG) and (B) C3; C-F: Immunofluorescence staining for IgG subclasses showed intense positivity for (E) IgG3 and negative staining for (C) IgG1, (D) IgG2, and (F) IgG4; G and H: Strong glomerular staining for κ light chain (G) and weak staining for λ light chain (H) were observed. Original magnification, × 400.

OUTCOME AND FOLLOW-UP

The patient was followed for over 200 d. No specific discomfort was reported during the period. Her condition improved after BCD treatment. At the last follow-up, her urine protein-to-creatinine ratio was 1.4 g/g, Scr was stable at 111 μmoL/L, complement was normal, and the urine free light chain ratio decreased from 5.0217 to 2.6894. In addition, her Hb was stable at 112 g/L, and the serum albumin level increased to 38.3 g/L (Figure 4).

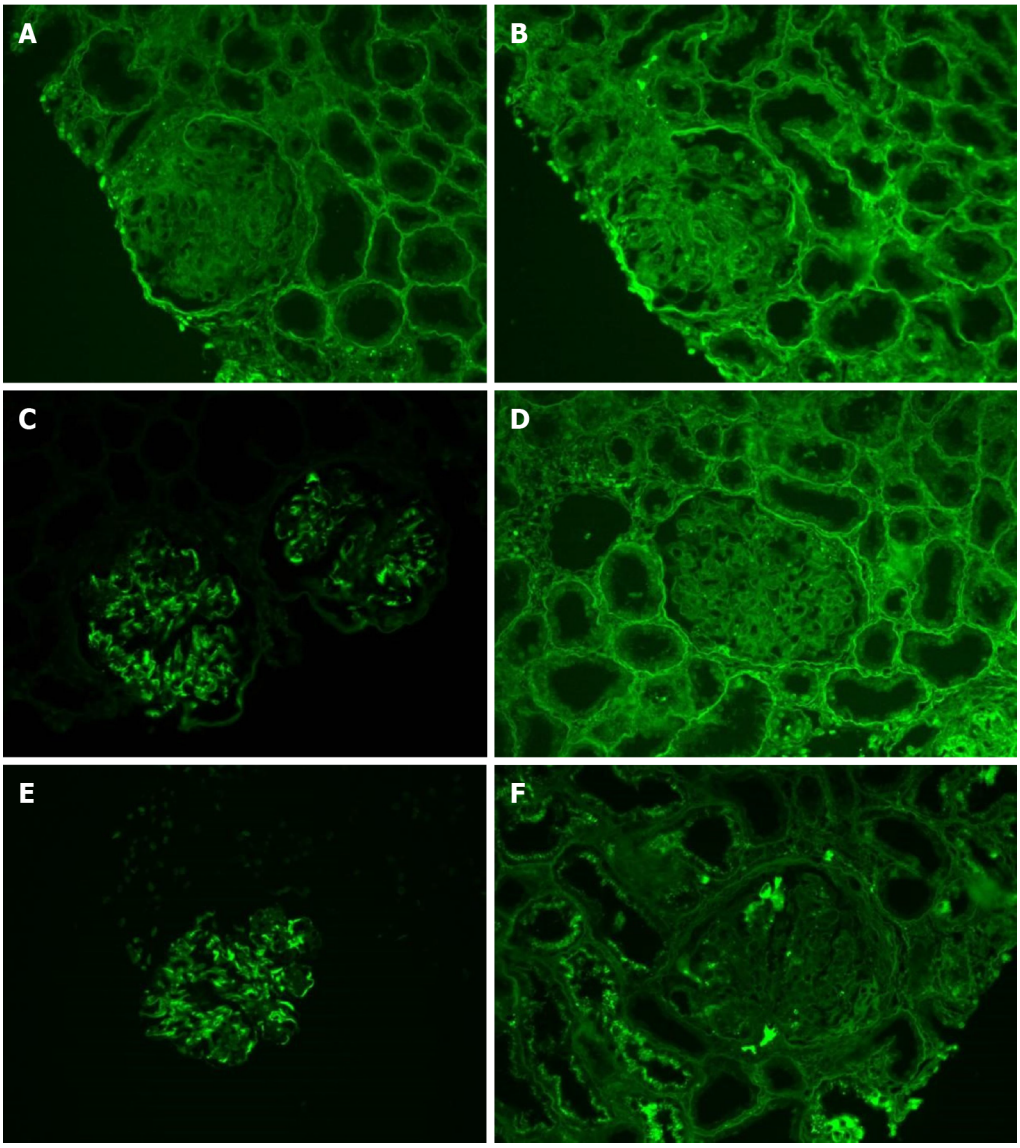

Figure 3 Immunofluorescence analyses. A-D: Immunofluorescence staining for immunoglobulin G (IgG) subclasses shows intense positivity for (C) IgG3 and negative staining for (A) IgG1, (B) IgG2, and (D) IgG4; E and F: Strong glomerular staining for κ light chain (E) and weak staining for λ light chain (F) were observed. Original magnification, × 400.

DISCUSSION

As illustrated in our patient, PGNMID is an important phenotype of monoclonal gammopathy of renal significance. It has a dual nature of blood and kidney disease. Because of the complexity of its pathogenesis, the exact causes of PGNMID are still not fully understood. It is believed that the disease is caused by the deposition of intact immunoglobulins produced by clonally proliferating plasma cells or B cells in the glomeruli[4]. Preud'homme *et al*[5] reported that the clustering of hydrophobic amino acids in the complementarity determining region 1 in monoclonal immunoglobulin (MIg) creates a hydrophobic zone that might promote interactions favoring light chain aggregation and tissue precipitation[5]. For a definitive diagnosis, a complete examination including serum and urine immunofixation, protein electrophoresis, free light chain assay, and complete renal pathology is necessary. In 2009, Nasr *et al*[2] retrospectively identified 37 patients; according to IFE, 7 patients had a monoclonal spike (M-spike) in both serum and urine, and 4 patients had an M-spike detectable in the serum only, but no patient had an M-spike detectable in the urine only. To our knowledge, this is the first report of a PGNMID patient who had monoclonal protein in the urine only. The mechanism needs to be studied further.

There is no effective method to inhibit the deposition of MIg in tissues or directly remove the MIg deposited thus far. Some cases had achieved clinical complete recovery or partial recovery for the treatment of abnormal cloned cells[2]. Given that

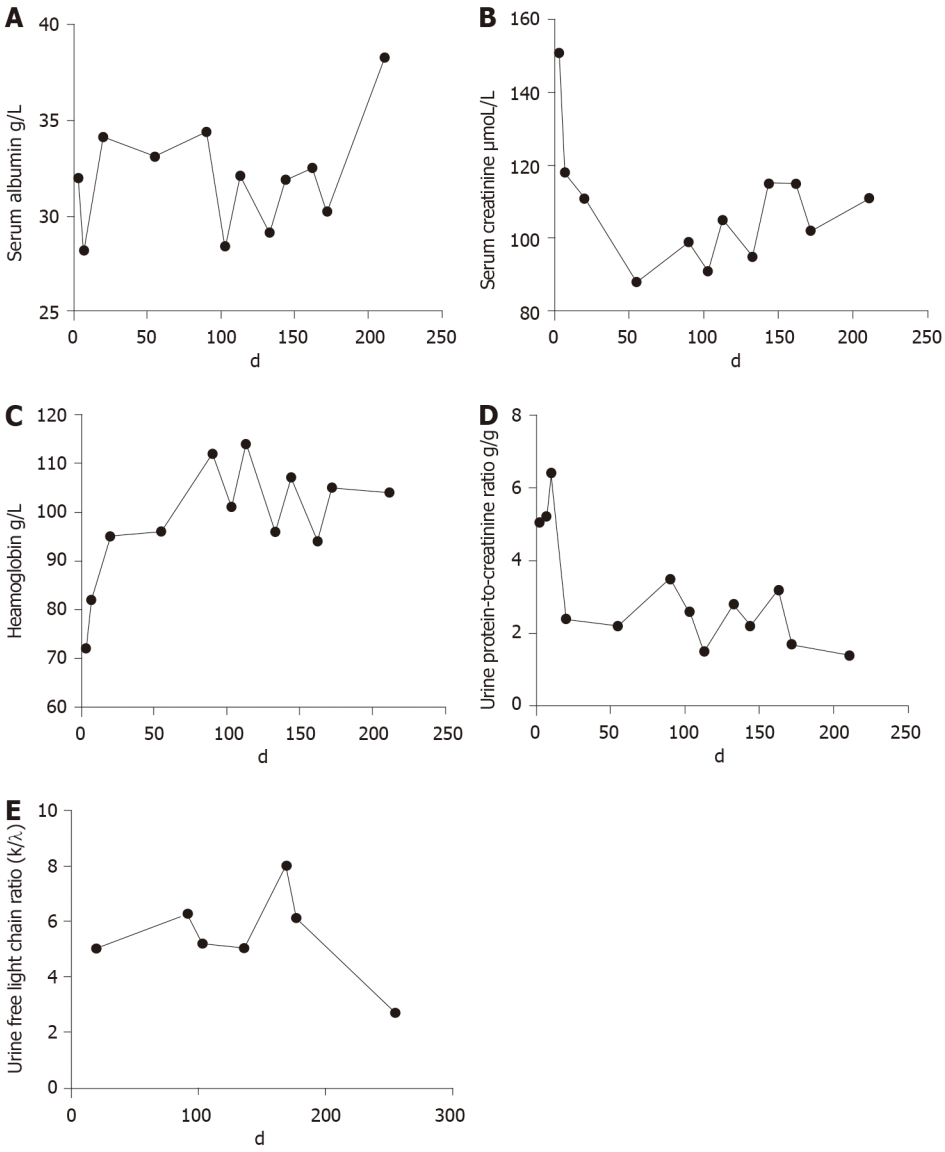

Figure 4 Trends of the urine protein-to-creatinine ratio, serum creatinine, hemoglobin, serum albumin levels, and urine free light chain ratio since the first day of initiation of the bortezomib (B) + cyclophosphamide (C) + dexamethasone (D) scheme. A: Serum albumin; B: Serum creatinine; C: Heamoglobin; D: Urine protein-to-creatinine ratio; E: Urine free light chain ratio.

more than 50% of the glomeruli were sclerotic, we think that the PGNMID is irreversible in this case.

The treatment options mainly refer to the clinical experience in the therapies of hematologic malignancies such as multiple myeloma and amyloidosis. Andréau *et al*[6] believed that BCD scheme is a common regimen for the treatment of monoclonal gammopathies. Cell proliferation and cell cycle progression can be inhibited by dexamethasone in B lymphocytes[6]. Similarly, the activation/proliferation sequence and the differentiation phase of the B cell maturation sequence are suppressed by cyclophosphamide[7]. Bortezomib is a proteasome inhibitor that is regarded as a first-line drug for the treatment of plasma cell disease. It induces apoptosis of monoclonal plasma cells and inhibits renal fibrosis[8]. It is important to note that bortezomib may also induce acute interstitial nephritis[9]. Therefore, the renal function should be followed closely during medication.

The detection rate of circulating MIg in PGNMID patients is low. Nasr *et al*[2] reported that MIg can be detected in only 30% of patients and that abnormal plasma cells usually comprise less than 10% of bone marrow. Bhutani *et al*[10] found that 60% of kidney-related monoclonal diseases originate from plasma cell clones. Therefore, in the absence of circulating or bone marrow monoclonal evidence, bortezomib-based treatment is acceptable. However, due to the limited number of cases, no randomized clinical trial has been published. Previously, Noto *et al*[11] treated an elderly PGNMID case with dual therapy bortezomib and dexamethasone and achieved a satisfactory

result. There is no doubt that BD scheme and BCD scheme[12] are both treatment options for PGNMID. In view of the fact that our case was a young female who was better able to withstand the side effects of the drugs, we chose the BCD scheme in order to stable her renal function as much as possible. After BCD chemotherapy, remission was achieved during follow-up, suggesting that this scheme may be an appropriate option for PGNMID. Nonetheless, further evidence-based research is still needed.

CONCLUSION

Although PGNMID is a rare disease that often occurs in middle-aged and elderly individuals, it cannot be easily excluded in young people, especially in those whose pathological type is MPGN or MsPGN, even when serum IFE is negative. Renal biopsy should be repeated if necessary. An accurate diagnosis at the earliest stage may avoid the overuse of glucocorticoids and immunosuppressants as well as any subsequent serious side effects (Figure 5).

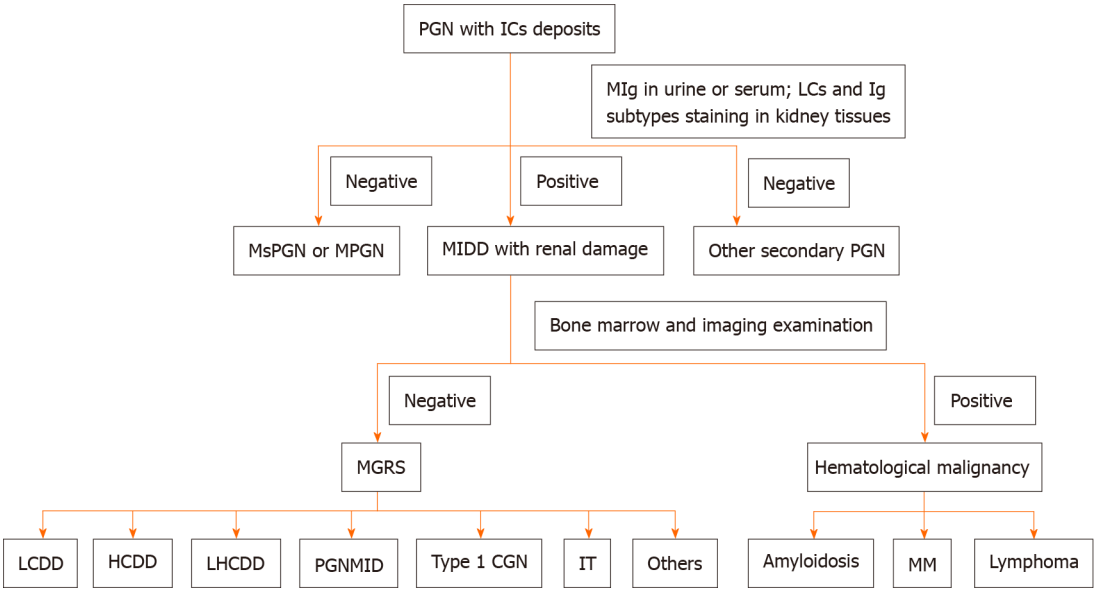

Figure 5 Diagnosis algorithm of proliferative glomerulonephritis with monoclonal immunoglobulin G deposits. PGN: Proliferative glomerulonephritis; IC: Immune complexes; LCs: Light chains; MPGN: Membranoproliferative glomerulonephritis; MIDD: Monoclonal immunoglobulin deposition disease; MGRS: Monoclonal gammopathy of renal significance; LCDD: Light-chain deposition disease; HCDD: Heavy-chain deposition disease; LHCDD: Light-and heavy-chain deposition disease; PGNMID: Proliferative glomerulonephritis with monoclonal immunoglobulin G deposits; Type 1 CGN: Type 1 cryoglobulinemic glomerulonephritis; IT: Immunotactoid glomerulonephritis; MM: Multiple myeloma.

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