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Case and Review

Polyclonal Immunotactoid Glomerulopathy Associated with Monoclonal Gammopathy of IgM Type and Underlying Plasmacellular Disease: Successful Treatment with Rituximab Alone

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Glomerular nephropathy · Immunotactoid · Monoclonal gammopathy · Immunoglobulin M · Rituximab

Abstract
Immunotactoid glomerulopathy (ITG) occurs infrequently and is characterized by organized IgG containing deposits. It most usually manifests as a concomitant disease of a broad spectrum of oncologic entities. We here present an exceptional case of ITG without glomerular light chain restriction secondary to a IgM kappa type monoclonal gammopathy of undetermined significance. Due to nephrotic syndrome and deterioration of kidney function a rituximab monotherapy was initiated without targeting the plasmacellular augmentation, which was confirmed as the underlying process. The treatment led to a long-term improvement of proteinuria and stabilization of glomerular filtration rate. Its therapeutic effect has to be attributed to immunomodulatory capacities and targeting of podocytes rather than to be interpreted

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as directed against a bone marrow or glomerular clone. We conclude that rituximab therapy may be a valuable part of the therapeutic options in ITG irrespective of the underlying oncologic entity.

Introduction

Immunotactoid glomerulopathy (ITG), first reported in 1977, is a rare kidney disorder caused by glomerular deposits organized as microtubules, which contain IgG by fluorescence microscopy [1]. As such, it is often associated with lymphoplasmocytic disease going along with monoclonal gammopathy [2–4], but its etiology may also include hepatitis C virus infection or autoimmune disease [5]. If associated with lymphoplasmocytic disease or monoclonal gammopathy of undetermined significance (MGUS), the underlying hematologic disease per se often does not meet treatment criteria, but treatment is indicated in order to prevent progressive kidney failure. This condition, which can also be found in other renal entities caused by monoclonal gammopathy like AL-amyloidosis or proliferative glomerulonephritis with monoclonal Ig deposits, has been designated monoclonal gammopathy of renal significance [6]. Experience in specific treatment is scarce and contradictory, but a clone-directed therapy has been advocated [7]. Steroids, cytotoxic agents, and plasmapheresis had a variable effect [1, 8]. Rituximab was first administered successfully as maintenance therapy in a case of ITG exhibiting nephrotic syndrome insufficiently controlled with steroids alone in 2018 [9]. In our report, we will present a case of ITG with underlying plasmacellular disease with monoclonal gammopathy of IgM type, successfully treated with rituximab alone.

Case Report/Case Presentation

A 69-year-old woman was admitted to our hospital for refractory arterial hypertension. The findings in clinical examination were unremarkable except for mild lower leg oedema. Laboratory reports showed earlier unknown normocytic anemia with hemoglobin being 8.7 g/dL as well as an elevated creatinine level of 2.4 g/dL, 3.8 g per day albuminuria and microhematuria. Renal function was normal 1 year prior to admission and from then on gradually deteriorated. Previously, hypertension was well controlled with metoprolol 95 mg, 5 mg ramipril, and 12.5 mg hydrochlorothiazide per day. There was no other comorbidity except for ectopic atrial tachycardia which had been successfully treated by catheter ablation 4 years before.

First upper and lower gastrointestinal bleeding had been excluded from differential diagnosis. Antinuclear antibodies and complement factors C3 and C4 were normal as were serologic parameters of hepatitis B and C and HIV. ANCA was positive based on the indirect immunofluorescence procedures, but anti-myeloperoxidase- and anti-proteinase-3-ELISA were negative. Given that serum immunofixation electrophoresis showed IgM kappa monoclonal gammopathy, a bone marrow biopsy was performed. After CD 138 staining, histology revealed slightly increased plasma cells (up to 15% diffuse), partially arranged in aggregates monoclonal for kappa (shown in Fig. 1), with a slight polyclonal background for lambda, but without significant expression of CD56 or of CyclinD1. A MYD88 mutation was ruled out using next generation sequencing and in addition, Congo red staining gave no hint for AL-amyloidosis. Those results were interpreted as border findings between smoldering myeloma and MGUS because eventually it could not be verified whether or not the degree of bone marrow infiltration exceeded 10%. This uncertainty in part was due to presence of reactive, i.e., polyclonal

plasma cells. Later on, the diagnosis could be clarified by follow-up bone marrow biopsy after 30 months, which confirmed the diagnosis of MGUS. The degree of monoclonal plasma cell infiltration then was stable and could be quantified below 10% (Fig. 1).

Low hemoglobin at the time of the first bone marrow biopsy was not regarded due to bone marrow plasma cell proliferation but interpreted as renal anemia. Treatment initiation was thus deemed unnecessary with regard to the plasmacellular disease.

Successful management of arterial hypertension involved 25 mg of torasemide, 10 mg of amlodipine, 10 mg ramipril, 25 mg of spironolactone, and 0.6 mg moxonidine daily. Renal artery stenosis was ruled out.

However, the etiology of acute kidney injury, new proteinuria, and worsened arterial hypertension remained unclear. Therefore, ultrasound guided kidney biopsy was performed in the tertiary university center and ITG was diagnosed. Light microscopy on renal biopsy showed up to twenty-six glomeruli, four of which were sclerosed, and exhibited a membranoproliferative pattern. In the interstitium, in some parts acute tubular injury and more globally slight to moderate tubular atrophy and lymphoplasmacellular inflammation were present (shown in Fig. 2). Congo red staining was faintly positive but without green birefringence under polarized light. In molecular pathological analysis, a significant peak could be demonstrated neither in the T-cell gamma nor in the heavy chain locus. Immunofluorescence was positive for IgG and IgM with coexpression of C1q and C3c and was negative for IgA (shown in Fig. 3). The kappa and lambda light chain ratio was equal. Finally, microtubules with a diameter of approximately forty-eight nanometer arranged in parallel bundles were shown by electron microscopy of separately stored material (shown in Fig. 4).

In terms of specific treatment, we opted solitarily for rituximab administered every 3 weeks although chemotherapy was initially taken into consideration. In total, rituximab alone 375 mg/m^2 was applied in 5 cycles. Treatment was concluded when serum creatinine levels decreased to 2.0 mg/dL and proteinuria to 442 mg per day within 2 months after initiation of rituximab therapy. Follow-up care was conducted every 3 months. Within 32 months, the patient has stable kidney disease with a stage 4 reduction of glomerular filtration rate and persistent proteinuria below 500 mg per day. Arterial hypertension is well controlled with 95 mg of metoprolol, 5 mg of amlodipine, 20 mg of torasemide, and 5 mg of ramipril. Hemoglobin levels have increased constantly to levels above 10 g/dL. So far, IgM-levels always were clearly below 3 g/dL, together with abnormal kappa/lambda ratios indicating MGUS. A transient decrease of IgM-level and the kappa/lambda ratio has been notified after application of rituximab therapy (shown in Fig. 5).

Fig. 1. Light microscopy of a representative trephin shows (**a**, Giemsa stain, original magnification ×200) slightly hyperplastic bone marrow, regular fat cell distribution, slightly hyperplastic and left-shifted granulopoiesis with complete maturation, some small and larger groups of erythropoiesis, scattered megakaryocytes (black arrows) as well as some rare lymphocytes and few plasma cell aggregates (red arrows). CD20 staining (**b**, original magnification ×200) shows randomly distributed B-lymphocytes (yellow arrows), CD138 staining (**c**, original magnification ×200) shows plasma cells both scattered (black arrows) and arranged in very small aggregates (red arrows). **d** (original magnification ×200) shows Kappa restricted plasma cells irregularly scattered and organized in very small clusters (red arrows), polyclonal background. In total the degree of bone marrow, infiltration is approximately 15% (clonal and polyclonal plasma cells together). The lower panels under the black line demonstrate stable findings in a follow-up bone marrow biopsy after 30 months with a bone marrow infiltration of monoclonal plasma cells below 10% (**e**, Giemsa stain, original magnification ×200: small plasma cell aggregates (red arrows). **f** CD20 staining, original magnification ×200: scattered B-cells (yellow arrows). **g** CD138 staining, original magnification ×200: scattered plasma cells (black arrow) and small plasma cell aggregates (red arrows). **h** Kappa stain, original magnification ×200: small plasma cell aggregates (red arrows).

(For figure see next page.)

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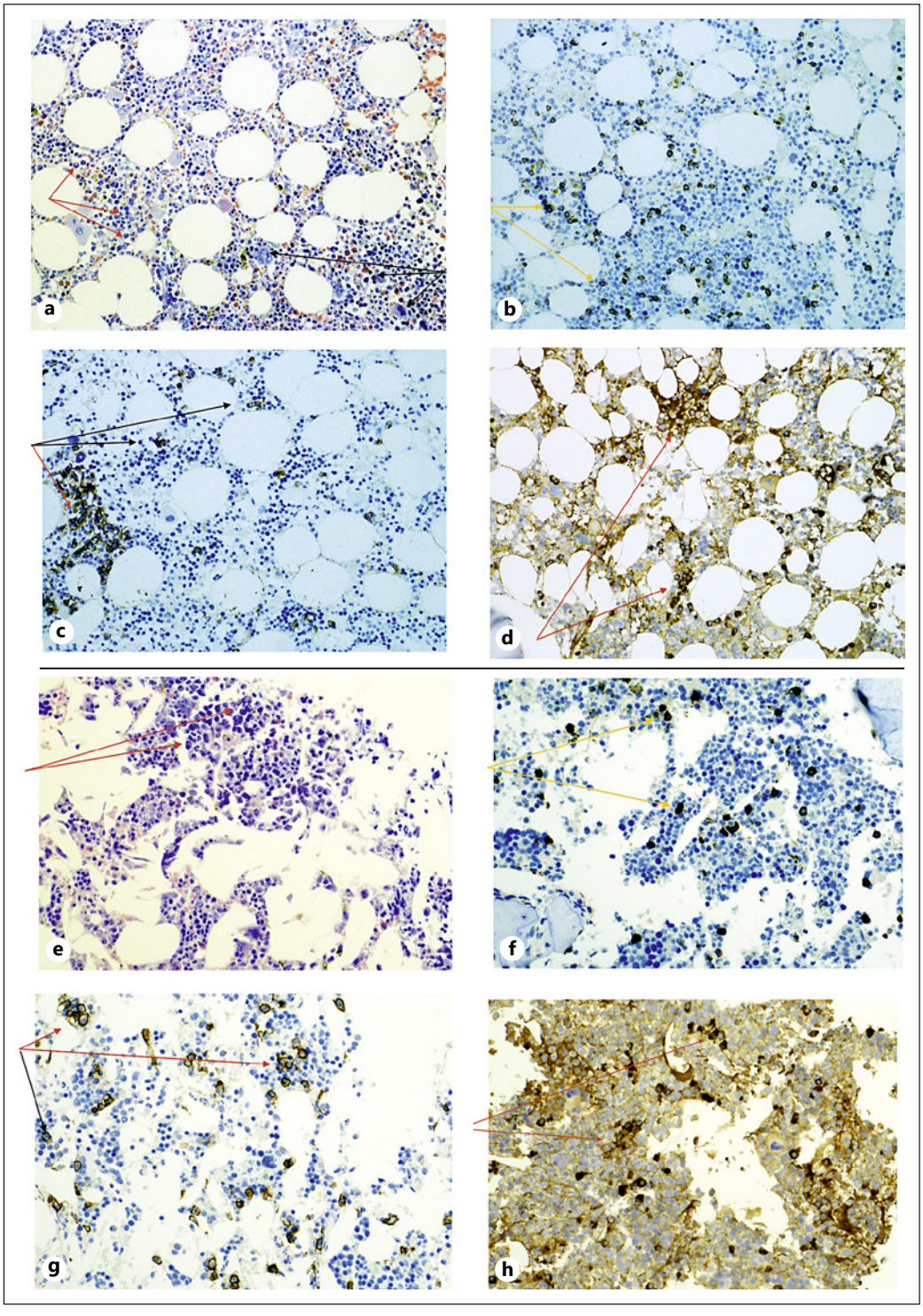

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Discussion/Conclusion

ITG is a rare renal entity [10, 11], which is characterized by a glomerular deposition of microtubules with hollow centers and an average diameter of 10–52 nm [12, 13], sometimes even up to 90 nm [14]. They are typically arranged in parallel arrays and can be found in the

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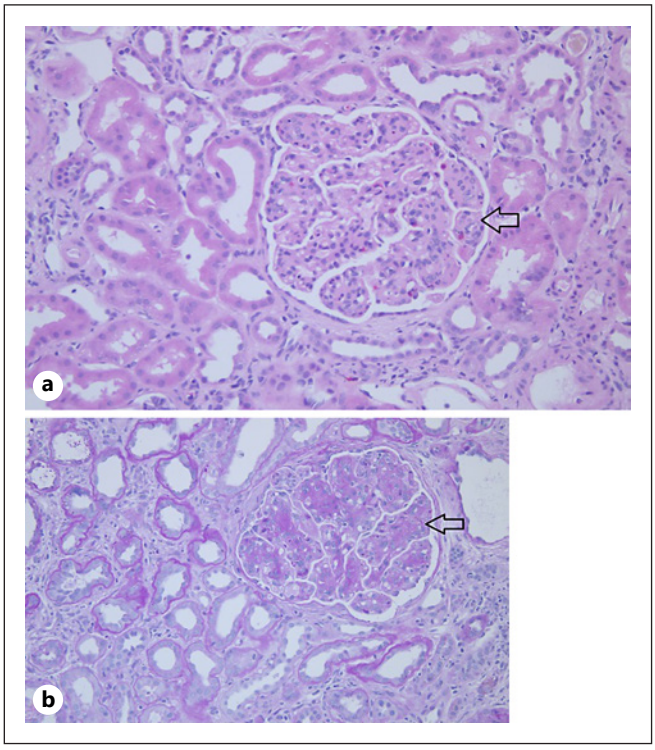

Fig. 2. Renal biopsy: Light microscopy (**a**: Hematoxylin-eosin-stain and **b**: Periodic acid-Schiff stain; original magnification, ×200): The glomeruli show typical changes with mesangial proliferation, luminal cells and basal membrane splitting (see arrow marks). Tubulointerstitium with a focal tubulusatrophy, fibrosis, and a lymphocytic infiltration.

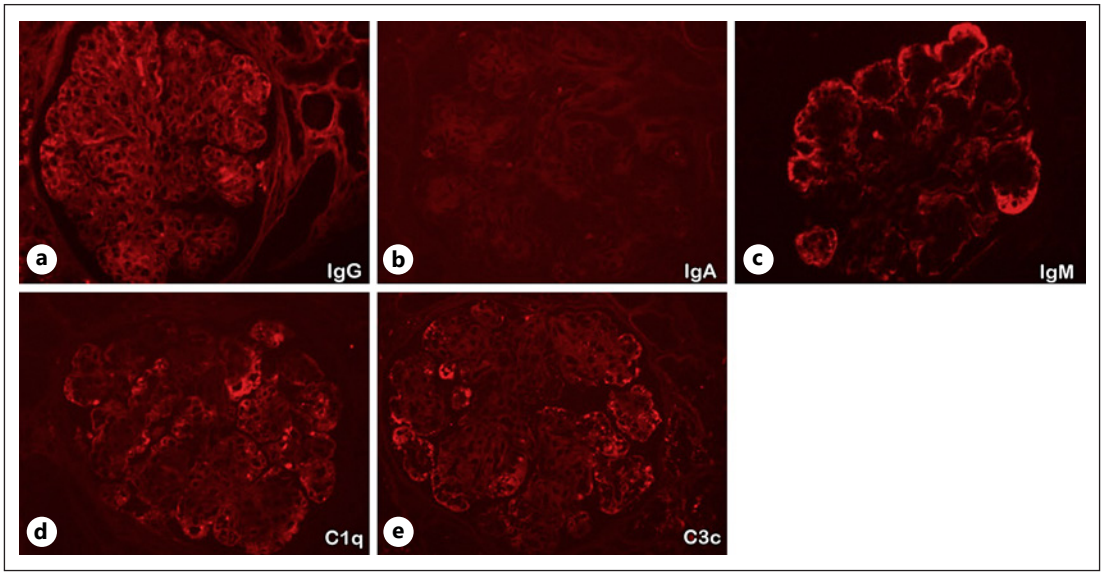

Fig. 3. Renal biopsy: Immunofluorescence analysis with predominant positivity for IgG (**a**) with lesser IgA (**b**) and IgM (**c**) by coexpression of C1q (**d**) and C3c (**e**). (Original magnification, ×200).

mesangium, the subendothelial, and subepithelial spaces. In light microscopy, an endocapillary proliferative, a membranoproliferative or a membranous pattern are most typical. The characteristic immunofluorescence and electron microscopy findings are essential for diagnosis [15]. The term "immunotactoid" has been introduced by Schwartz by analogy to the linear crystallization of hemoglobin S that forms elongated tactoids in red blood cells during

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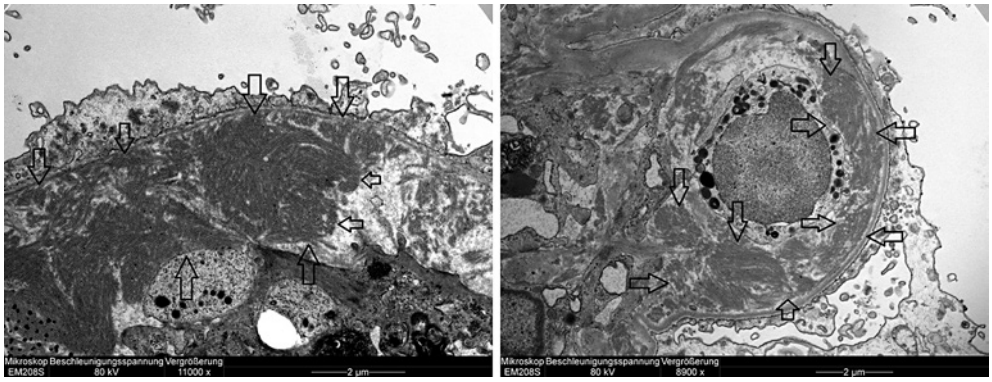

Fig. 4. Electron microscopy: Dense microtubular deposits in parallel arrays from 30 to 50 nm as specific criteria for the diagnosis of ITG (see arrows marks) (Original magnification, ×11,000 in the left panel and ×8,900 in the right panel).

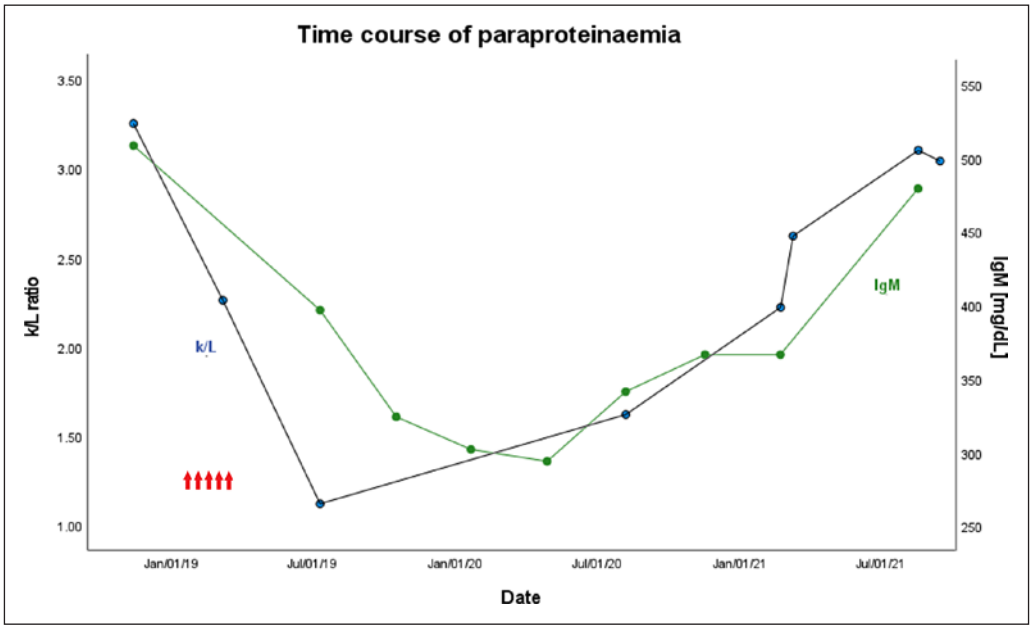

Fig. 5. Time course of paraproteinemia by the kappa/lambda ratio of the free light chains (in blue, left vertical axis) and IgM-level (in green, right vertical axis). The 5 red arrows indicate the application of rituximab.

sickle cell crisis. The immunotactoid deposits contain immunoglobulins and complement as principal components [1]. Cryoglobulinemia can cause glomerular lesions that resemble ITG and should be ruled out [16], although a definitive exclusion of cryoglobulinemia may be difficult due to histologic overlap and the insensitivity of serologic testing [13]. In our case, cryoglobulinemia is unlikely because of lack of light chain restriction in the kidney biopsy despite monoclonal gammopathy and the lack of systemic signs of cryoglobulinemia. The clinical presentation of ITG typically involves hypertension, proteinuria, hematuria, and renal insufficiency [17]. As stated above, ITG commonly goes along with hematologic disorders or MGUS and might even occur years before a hematologic diagnosis can be made [18, 19].

Although ITG appears to have been diagnosed more frequently in the past decades, lack of specific treatment is still a major issue, especially with regard to 40% of all cases progressing

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to end stage renal disease [20]. The largest series of ITG has been published by Nasr et al. [13, 15]. From 1993 to 2011, 16 patients have been registered. Underlying hematologic malignancy was present in 7 cases, comprising 5 small B-cell lymphocytic lymphoma and chronic lymphocytic leukemia as well as 2 cases of myeloma. Monoclonal gammopathy was detected in 10 patients and did not comprise monoclonal IgM in any case. Interestingly, 50% had remission, 33% had persistent renal dysfunction, and 17% progressed to end stage renal disease. Those progressing to end stage renal disease had not received immunomodulatory therapy [13]. In contrast to our case, all patients in follow-up (10 in total) were treated with steroids. Rituximab was added together with cyclophosphamide in 2 patients with chronic lymphocytic leukemia, one of whom having complete and one of whom having partial remission. One of the 2 patients with myeloma was treated with peripheral blood stem cell transplant, but kidney disease progressed. The other myeloma patient received lenalidomide and steroids and had a partial remission. Recently, Nasr et al. [15] published a comprehensive extension of the discussed case series comprising 73 patients. Serum protein electrophoresis with immunofixation proved monoclonal gammopathy in 40%, which was IgM lambda only in 1 patient with chronic lymphocytic leukemia who had IgG kappa deposits in glomeruli. Although a hematologic condition was present in 66% of patients, only 6% suffered from myeloma. Nasr et al. [15] were able to discriminate a monoclonal and polyclonal variant of ITG as defined by glomerular immunofluorescence light chain restriction (67% and 33% of the cases, respectively). Hematologic disorders and monoclonal immunofixation in serum protein electrophoresis were more common in the monoclonal variant (82% and 50% of the cases, respectively) but also present in the polyclonal variant (26% and 16% of the cases, respectively). The monoclonal variant was more commonly treated with clone-directed therapy, which was associated with better outcome, and patients with a monoclonal variant had a better outcome than patients with the polyclonal variant. In terms of glomerular light chain restriction, as suggested by Nasr et al. [15], our case can be classified as polyclonal.

IgM type paraprotein usually is a feature of Waldenstrom's Macroglobulinemia. In a retrospective series of 57 patients with kidney involvement of Waldenstrom's Macroglobulinemia and further IgM-producing hematologic malignancies, there was no case of ITG. There were 9 patients with monoclonal gammopathy of renal significance (16%), but no patient with myeloma. Chemotherapy regimens varied remarkably. Outcome was better in nonamyloid-related than in amyloid-related glomerulopathy (improvement or stabilization in 65% of all nonamyloid-related cases) [21]. A smaller series included 14 patients suffering from IgM-secreting proliferations and concomitant renal involvement. One patient suffered from myeloma and AL-amyloidosis and three from distinct hematologic disorders and unspecified membranoproliferative glomerulonephritis. Twelve patients received various chemotherapies that resulted in significant improvement of kidney function [22]. A case report describes ITG associated with Waldenstrom's Macroglobulinemia and fatal outcome [18].

Waldenstrom's Macroglobulinemia or a precursor entity are unlikely in the presented case because of negative MYD88 mutation and the plasmacellular origin of MGUS has been confirmed immunohistochemically. Given that transitions from MGUS to multiple myeloma are smooth [23], it is noteworthy that in patients with multiple myeloma only 0.5% of paraproteins are of IgM type [24] and that ITG is an exceptional kidney lesion of myeloma patients [7].

Karasawa et al. [9] are the first working group to describe the use of Rituximab to achieve a complete remission of ITG. There was no M-protein spike in the electrophoretic profiles of serum and urine and no evidence of hematologic malignancy, but in the kidney biopsy, immunofluorescence findings indicated that κ-light chain was predominantly positive compared with λ-light chain. Initial treatment was started with steroids followed by rituximab every 6 months for 4 years, thus allowing the steroids to be tapered [9]. This seems comprehensible since the authors believed that the glomerular monoclonal deposits were caused by a dangerous,

undetectable B-cell clone. The therapeutic effect of rituximab in a recent case reported by Adilovic et al. [25] possibly can be attributed to antiproliferative properties since the patient suffered from diffuse large B-cell lymphoma, which itself is being treated with rituximab.

From a mechanistic point of view it seems unlikely that our patient mainly benefitted from the antiproliferative properties of rituximab for three reasons. First, rituximab usually is not directed against plasma cells, which is in line with a trial in which rituximab directed against CD20 positive plasma cells demonstrated some effects to a minority of patients but from a clinical point of view has turned out to be unsuccessful in non-pre-treated, refractory, and relapsed multiple myeloma [26]. Second, as discussed above, histologically and molecular pathologically there was no proof of monoclonal glomerular immunotactoid deposits. Especially in case of chronic lymphocytic leukemia, an association of ITG with an intrarenal infiltration by a malignant cellular clone is common [13], but the polyclonal nature of the intrarenal lymphoplasmacellular infiltrate has been confirmed by elaborate pathological workup in our case. Third, IgM levels to a large extent have been unaffected. Decreasing levels mentioned above may be attributed to the afore mentioned minor antiproliferative effects, but from our point of view, it has to be considered that those findings stand in contrast to long-term stable kidney disease independent of transiently falling or rising levels of kappa/lambda ratio and IgM (Fig. 5). Taken together, a direct immunomodulatory impact on the kidney disorder and a non-clone directed rituximab effect has to be postulated at least partly. Possibly, the (polyclonal) intrarenal lymphoplasmacellular inflammation has been targeted. Also, direct effects of rituximab on podocytes may have to be considered [27].

Thus, it can be stated that rituximab alone administered every 3 weeks may be a feasible approach for the treatment of polyclonal ITG. A response to this drug usually is slow ranging up to 18 months [28], and the appropriate treatment duration still has to be defined [9, 13]. Depending on the future course of our case, the question of adequate therapeutic regimens may raise anew. In general, successful therapy of an underlying hematologic malignancy may lead to complete remission of (the secondary) ITG [19], whereas on the other hand specific treatment has been deemed ineffective by others [29]. In a case of ITG associated with serum monoclonal IgM and a small, insignificant, monoclonal B-cell population Kuang et al. [30] reported, that rituximab alone improved renal function and hypertension, but nephrotic syndrome only could be controlled by additional treatment with bortezomib and dexamethasone. Also, the long-term evolution of the glomerular disease has to be considered since it has been reported that a primarily polyclonal glomerular deposit may transform to a monoclonal pattern [31] and that there may be monoclonal recurrence of former polyclonal ITG after kidney transplantation [15].

Conclusion

We present a rare case of monoclonal gammopathy comprising polyclonal ITG associated with a MGUS of IgM type, which has been successfully managed with a mainly not clone-directed rituximab monotherapy. Future treatment targets depend on the course of both interconnected pathological processes. Immunomodulatory and antiproliferative aspects then have to be considered.

Acknowledgment

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Statement of Ethics

We complied with the guidelines for human studies and declare that the research was conducted ethically in accordance with the Declaration of Helsinki. Written informed consent was obtained from the patient for publication of this case report and any accompanying images, and there were no more subjects involved in this case report. By local and national guidelines, an ethics approval was not required since all treatment decisions were merely based on clinical grounds. The decision to publish the case was taken after opting for rituximab treatment.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Funding Sources

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Author Contributions

Wolfgang Neukirchen and Veit Busch performed the literature review and formulated the manuscript including the figures. Anne Oesterling, Dirk Oliver Wennmann, Barbara Heitplatz, Peter Ritter, and Merz contributed to the manuscript text. Barbara Heitplatz and Prof. Harmut Merz provided the images for the Figures 1–4 and assisted in creating their illustration and legends. Wolfgang Neukirchen, Anne Oesterling, Dirk Oliver Wennmann, Barbara Heitplatz, Peter Ritter, Harmut Merz, and Veit Busch reviewed the manuscript.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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