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Case Report

IgG4-related tubulointerstitial nephritis associated with only lymphadenopathy and without elevated serum IgG4 or renal imaging abnormalities: a case report and literature review

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Abstract: IgG4-related tubulointerstitial nephritis (IgG4-TIN) is the most common renal manifestation of IgG4-related kidney disease (IgG4-RKD) and may cause acute or chronic renal dysfunction. Imaging often shows heterogeneous densities in the kidneys, such as a mass or multiple nodules. Serology usually demonstrates high levels of serum IgG4 and total IgG. Most patients have other organs involvement by IgG4 related disease. Although lymphadenopathy is frequently observed in patients with IgG4-TIN, it is rarely presented as the only extrarenal lesion. Herein, we present a rare case of IgG4-TIN associated with only lymphadenopathy and without elevated serum IgG4 or renal imaging abnormalities. A 61-year-old Chinese man was admitted to our hospital with seven months history of generalized lymphadenopathy and five months history of renal dysfunction. His renal imaging was normal. He had no current or previous clinical, radiographic, and/or histologic evidence of other organ involvement except for the lymphadenopathy. Renal biopsy indicated plasma cell-rich TIN with an increased number of IgG4-positive plasma cells and storiform fibrosis. Repeated lymph nodes biopsy revealed IgG4-related lymphadenopathy. However, he did not have elevated serum IgG4 or total IgG levels. Oral prednisone therapy improved his renal function and lymphadenopathy. These findings supported our final diagnosis of IgG4-TIN. Clinicians should be aware of this condition and steroid therapy should be considered for such patients. An early diagnosis and appropriate therapy can induce remission and preserve renal function.

Keywords: IgG4-related tubulointerstitial nephritis, lymphadenopathy, serum IgG4, renal radiographic lesions

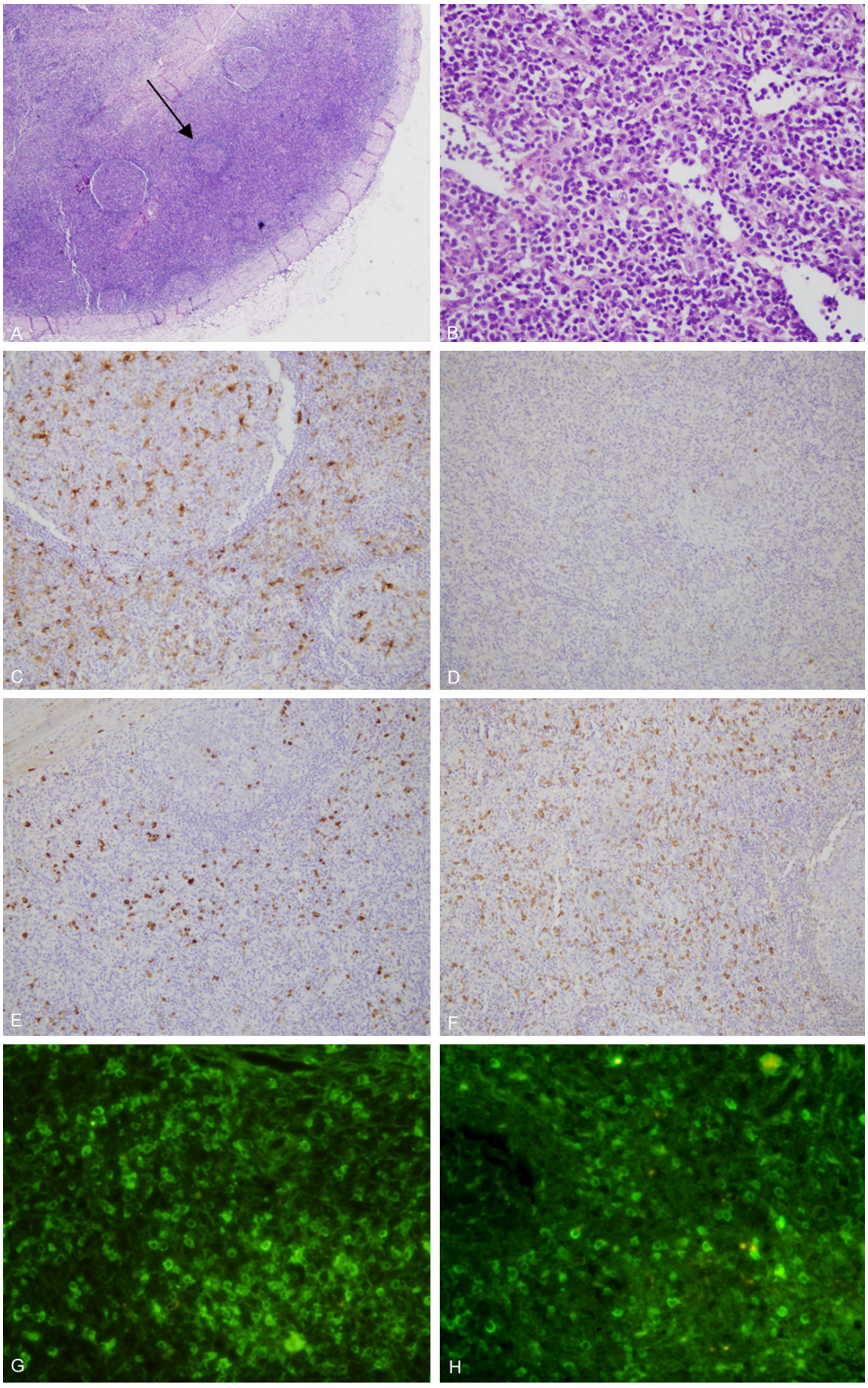
Introduction

IgG4-related disease (IgG4-RD) is a newly recognized systemic fibro-inflammatory disease characterized by infiltration of the organs with IgG4 positive plasma cells along with storiform fibrosis [1]. Renal involvement, termed IgG4-related kidney disease (IgG4-RKD), usually presents as plasma cell-rich tubulointerstitial nephritis (TIN) associated with renal mass lesions [2, 3]. Serology usually demonstrates high levels of serum IgG4 and total IgG [2]. Most patients have accompanying IgG4-related extrarenal lesions such as sialadenitis, lymphadenopathy, or type 1 autoimmune pancreatitis (AIP) [4, 5]. However, IgG4-TIN with only lymphadenopathy has not been well recognized [6]. Here, we present a rare case of IgG4-TIN associated with swelling of many lymph nodes as the only extrarenal lesions and without elevated serum IgG4 level or renal imaging abnormalities. Following the case report we review the diagnosis and management of IgG4-TIN.

Case report

A 61-year-old man was admitted to our hospital with seven months history of generalized lymphadenopathy and five months history of renal dysfunction in May 2013.

Seven months prior to his admission, he noted cubital lymph nodes swelling. He did not take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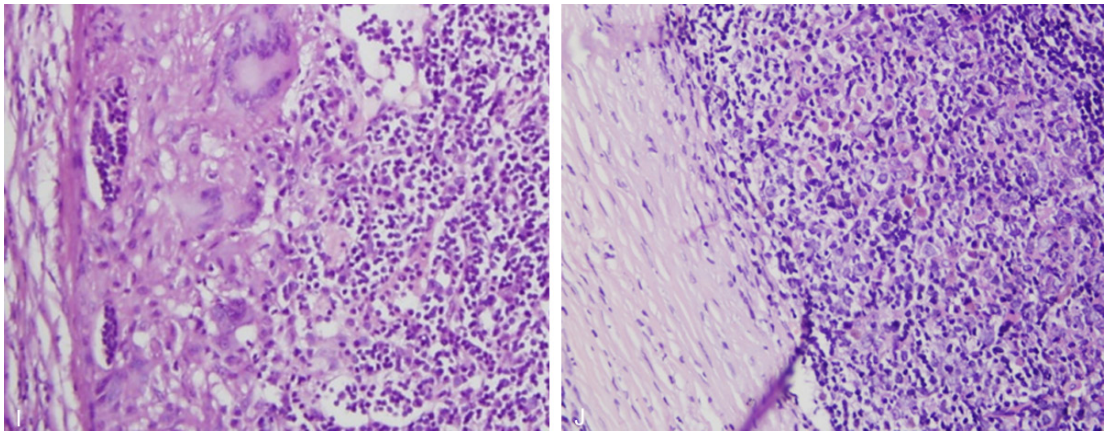

Figure 1. Histological and immunostaining analyses of lymph nodes. (A) Haematoxylin and eosin staining showed dense capsular fibrosis, reactive follicular hyperplasia (arrows) and marked interfollicular expansion (HE, original magnification ×100). (B) The interstitial infiltrate was composed of plasma cells, histiocytes, and eosinophils (HE, original magnification ×400). (C-F) Immunohistochemical staining for IgG subclasses, IgG1-4 (C: IgG1, D: IgG2, E: IgG3, F: IgG4, original magnification ×200), showed that IgG4-positive plasma cells crowded more than others. (G) Immunofluorescence staining for IgG revealed a marked increase in IgG-positive plasma cells (original magnification ×400). (H) Immunofluorescence staining for IgG4 showed a marked increase in IgG4-positive plasma cells in the infiltrate (original magnification ×400). Lymph node biopsy in Dec 2012 (I) showed granulomatous inflammation, which wasn't observed in the lymph node biopsy in May 2013 (J) (HE, original magnification ×400).

the symptom seriously until he found axillary and inguinal lymph nodes swelling accompanied by polyuria, progressive fatigue, loss of appetite, and a 5-kg weight loss. In Dec 2012 he was admitted to hematology at a local hospital. Contrast-enhanced computed tomography (CT) from chest to pelvis showed normal kidneys and no abnormalities of the pancreas or retroperitoneum, but revealed swollen mediastinum, bilateral hilus pulmonis, bilateral axillary and bilateral inguinal lymph nodes. The cubital lymph node biopsy was conducted with a pathological diagnosis of "chronic granulomatous inflammation" in his local hospital. No definite diagnosis was retained. In May 2013 he was referred to our nephrology division for renal dysfunction accompanied by new-onset hypertension. The highest value of blood pressure measured was 180/110 mmHg. Nifedipine was prescribed and the blood pressure was soon under good control. His serum creatinine (Scr) level had gradually increased from 1.33 mg/dL to 3.45 mg/dL over 5 months. He had never experienced dry eye or dry mouth. Measurement of serum IgG level and urinalysis had not been performed before admission. He had no significant past medical history and no family history of renal disease.

Upon admission, the patient had a temperature of 37.0 degrees Celsius, heart rate of 85 beats per minute, blood pressure of 130/85 mmHg, respiratory rate of 16 breaths per minute. His height was 169 cm, and weight was 49 kg (a 5-kg loss over 7 months). Bilateral axillary, bilateral cubital and bilateral inguinal lymph nodes were palpable without tenderness. He had neither parotid gland nor submandibular gland swelling. An operation scar was seen in right elbow. His cardiac, pulmonary, abdominal, neurologic and musculoskeletal exams were normal. The remaining physical examination was unremarkable.

Laboratory examinations showed the following results: Urinalysis revealed no hematuria or proteinuria. The level of urinary N-acetyl-β-D-glucosaminidase (NAG) was 5.46 U/mmolCr (normal <1.3 U/mmolCr) and that of urinary α1-microglobulin was 16.05 mg/mmolCr (normal <1.2 mg/mmolCr). White blood cell count of 7.49×10^9/L with 13.3% eosinophils (normal 0.5~5%); hemoglobin, 103 g/L; a platelet count of 266×10^9/L; blood urea nitrogen, 43.6 mg/dL; Scr, 3.45 mg/dL; normal electrolytes; aspartate aminotransferase (ALT), 12 U/L; alanine aminotransferase (AST), 12 U/L; alkaline phosphatase (ALP), 49 IU/L; amylase, 70 IU/L; C-reactive protein (CRP), 1.77 mg/L; albumin, 38.4 g/L; globulin, 32 g/L; IgG, 1480 mg/dL; IgA, 78 mg/dL; IgM, 64 mg/dL; IgG subclasses: IgG1, 1040 mg/dL (normal 405 to 1011 mg/

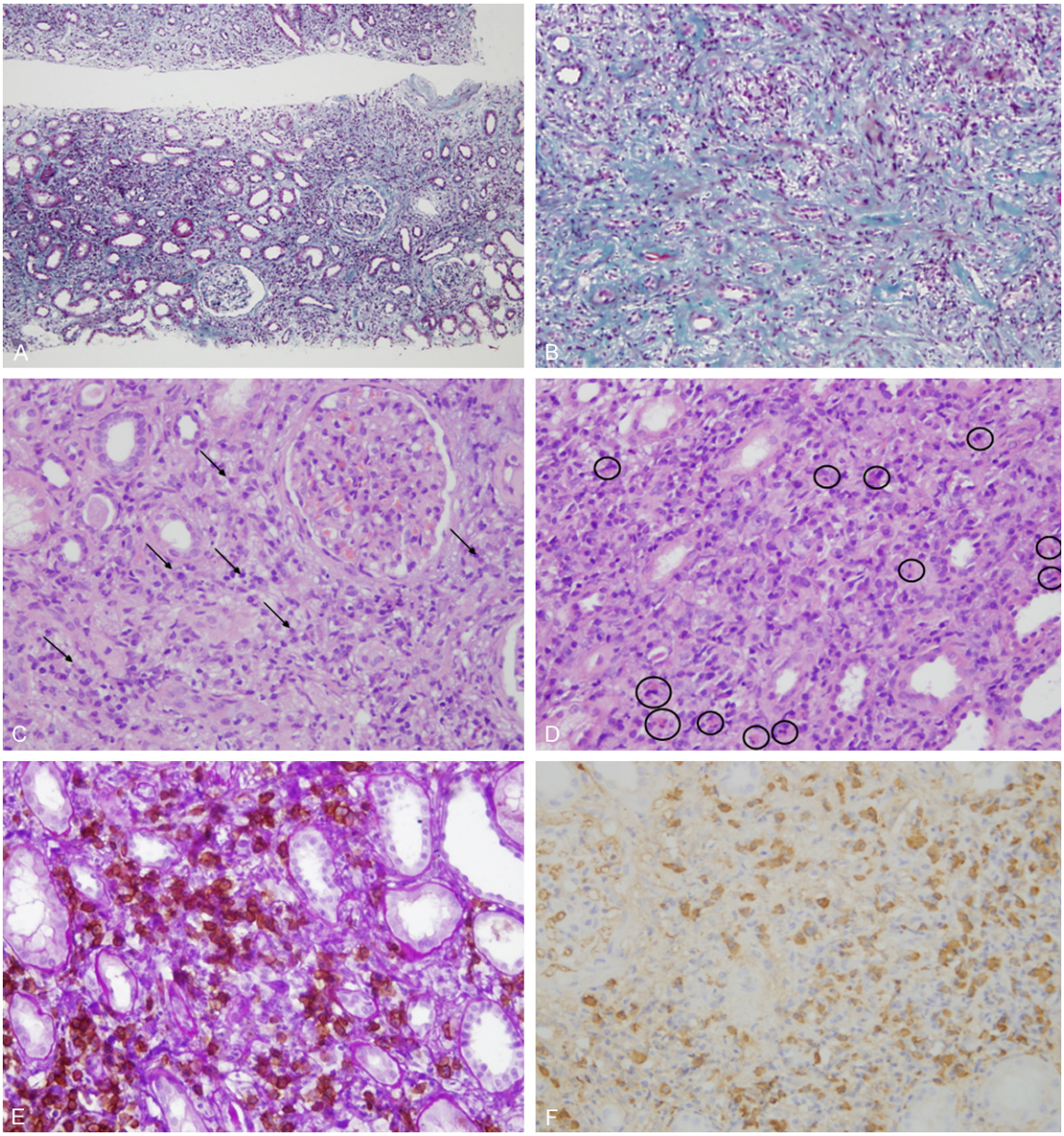

Figure 2. Histological and immunohistochemistry findings in the renal tissue. A. Diffuse interstitial fibrosis and tubule atrophy were observed. Glomeruli were unaffected except for periglomerular fibrosis and global glomerulosclerosis (Masson's trichrome, original magnification ×100). B. Masson's trichrome staining revealed severe tubulointerstitial nephritis with storiform fibrosis (Masson's trichrome, original magnification ×200). C, D. The interstitial infiltrate was composed of mononuclear cells, plasma cells (arrows), and several eosinophils (circles) (HE, original magnification ×400). E. Immunohistochemical staining showed CD38+ plasma cells in the infiltrate (original magnification ×400). F. Immunohistochemical staining for IgG4 showed a marked increase in IgG4+ plasma cells in the infiltrate (original magnification ×400).

dL); IgG2, 298 mg/dL (normal 169 to 789 mg/dL); IgG3, 126 mg/dL (normal 11 to 85 mg/dL); IgG4, 16 mg/dL (normal 3 to 201 mg/dL); C3, 0.37 g/L (normal 0.79 to 1.52 g/L); and C4, 0.03 g/L (normal 0.16 to 0.38 g/L). There was no monoclonal gammopathy. Antinuclear, anti-dsDNA, anti-Ro, anti-La, anti-Sm, anti-RNP, anti-Sc170, anti-SS-A, anti-SS-B, anti-Jo1, anti-neutrophil cytoplasmic antibodies (ANCA, proteinase 3 and myeloperoxidase) and anti-glomerular basement membrane were negative. The results of both tumor marker panels and

hepatitis virus panels were clear. Testing for HIV and human T-lymphotropic viruses were negative. Renal color Doppler ultrasound examination showed normal kidneys.

The inguinal lymph node biopsy exhibited enlarged size. The essential structure of the lymph node was conserved. The capsule was intact, however, fibrotic thickening. Reactive follicular hyperplasia and interfollicular expansions were predominant (**Figure 1A**). There were an increased number of cells, consisting of plasma cells, histiocytes, and eosinophils, in the area surrounding the follicles (**Figure 1B**). Immunofluorescence staining demonstrated that most plasma cells were positive for IgG (**Figure 1G**). Immunohistochemical staining for IgG subclasses, IgG1-4 (**Figure 1C-F**), showed that IgG4-positive plasma cells crowded more than others. To confirm the results, immunofluorescence stains for IgG4 were performed and revealed that IgG4-positive plasma cells >50/hpf (**Figure 1H**). The ratio of IgG4-positive to IgG-positive plasma cells (IgG4+/IgG+) was approximately 50%.

In light of this finding, the prior cubital lymph node biopsy slides were reviewed and immunohistochemical stains were performed. The results were also consistent with IgG4-related lymphadenopathy (**Figure 1I**, **1J**).

We performed renal biopsy under ultrasound guidance. The specimen for light microscopy contained 30 glomeruli with 2 global glomerulosclerosis. Other glomeruli showed only minor abnormalities. There was no obvious endocapillary or extracapillary proliferation and no capillary wall thickening. Atrophic tubules and widespread interstitial fibrosis, typical fibrosis so-called storiform appearance, were observed (**Figure 2A**, **2B**). Diffused interstitial infiltration of plasma cells and mononuclear cells with eosinophils was also seen (**Figure 2C**, **2D**). Immunohistochemistry was performed for CD38 (plasma cell maker) and IgG4 on paraffin tissue. It revealed that numerous plasma cells had infiltrated the interstitium (**Figure 2E**). Immunohistochemistry for IgG4 showed that IgG4-positive cells >50/hpf (**Figure 2F**). Importantly, we did not find any granuloma, the most prominent feature of sarcoidosis, in the kidney specimen. The morphologic and immunostaining findings were diagnostic of an IgG4-TIN.

Finally, we made a diagnosis of IgG4-TIN and lymphadenopathy. Oral prednisone at an initial dose of 30 mg/day was administered from May 2013. Four months after therapy, his serum creatinine had decreased to 1.8 mg/dL, complement concentrations (C3, C4) increased to normal and proportion of peripheral eosinophils decreased to 2.6%. Lymph nodes swelling were gradually improved. Symptoms of polyuria, fatigue and anorexia alleviated. The dose of prednisone was gradually tapered and he was receiving 10 mg prednisone daily with a steady Scr level of 1.58 mg/dL as of June 2015.

Discussion

IgG4-RKD is a comprehensive term for renal lesions associated with IgG4-RD. Although, occasionally accompanied by glomerular diseases such as membranous nephropathy, mesangial proliferative glomerulonephritis, IgA nephropathy, membranoproliferative glomerulonephritis and endocapillary proliferative glomerulonephritis, TIN is the most common histopathologic pattern of IgG4-RKD [2, 3, 6-10].

The common clinical features of IgG4-RD include elderly onset, male predominance, and hypergammaglobulinemia [11]. Therefore, in patients with TIN who have these clinical features, IgG4-TIN should be considered in the differential diagnosis. Till now, two papers have proposed diagnostic criteria for IgG4-TIN [6, 12]. Both of these put forward similar criteria that include histology, serology, other organ involvement, and radiographic features. Our patient met both criteria in that his renal histology and immunostaining revealed plasma cell-rich tubulointerstitial nephritis with >10 IgG4-positive plasma cells/hpf and IgG4+/IgG+ plasma cell ratio of >40% and characteristic storiform fibrosis, and he had histologic evidence of IgG4-related lymphadenopathy. However, this patient displayed some unusual features for IgG4-TIN. First, he had no current or previous clinical, radiographic, and/or histologic evidence of other organ involvement except for the lymphadenopathy. Second, he did not have elevated serum IgG4 or total IgG levels, but IgG1 and IgG3 were slightly elevated. Third, the patient did not have distinct renal imaging abnormalities radiographically.

As a component of IgG4-RD, most IgG4-TIN may be associated with other organ involve-

ment such as AIP, sialadenitis, and sclerosing cholangitis [4, 5]. According to Raissian and colleagues, other organs were involved by IgG4-RD in 80% of patients, either concurrently or before the recognition IgG4-TIN [6]. Although lymphadenopathy is frequently observed in patients with IgG4-TIN, it is rarely presented as the only extrarenal lesion [6]. The present case presented with lymphadenopathy as the first manifestation and chief complaint, while lacked clinical or radiographic features of other organ involvement. He was clinically suspected to be multicentric Castleman's disease (MCD), malignant lymphoma and/or sarcoidosis. Lymph nodes morphological and immunostaining findings showed predominant IgG4-positive cells and eosinophilic infiltration, the characteristics of IgG4-related lymphadenopathy. These pathological findings are important clues to rule out the diagnosis of MCD, malignant lymphoma and sarcoidosis.

Elevated total IgG or IgG4 serum levels are predominant serum characteristics of IgG4-TIN. Although about 30% of patients with IgG4-RD have normal serum IgG4 concentrations, almost 80% of IgG4-TIN patients had elevated serum total IgG or IgG4 levels [6]. That is to say, serum total IgG or IgG4 levels can be within the normal range in about 20% of such group of patients. The present patient did not have elevated serum total IgG or IgG4 level. Thus, serum IgG4 or total IgG level cannot, by itself, include or exclude the diagnosis of IgG4-TIN [13]. Of note, our patient had peripheral blood hypocomplementemia and eosinophilia, which are other common laboratory features of IgG4-TIN and can be helpful in making the diagnosis [3, 6, 14, 15].

Most IgG4-TIN patients have renal imaging abnormalities [6, 16, 17]. The most common CT, ultrasonographic or magnetic resonance imaging findings of renal involvement are single or multiple low-attenuation lesions followed by bilateral diffuse kidney enlargement [6, 17]. Image lesions of IgG4-TIN are best visualized on contrast-enhanced CT scan [3]. Our patient did not have parenchymal lesions detected by contrast-enhanced CT when he had already had polyuria and his Scr had reached 1.33 mg/dL. Since the color Doppler ultrasound renal image was normal when he was referred to our division, the patient didn't receive contrast-enhanced CT again considering the higher risk of medium-induced nephropathy (his Scr level had increased to 3.45 mg/dL). So, imaging findings are not specific for the disease either.

As the clinical, laboratory and renal imaging features are atypical in some patients, renal biopsy is often required to make a definitive diagnosis of IgG4-TIN. Histopathology and immunohistochemical or immunofluorescence staining for IgG4 are the most valuable diagnostic tools in IgG4-TIN [6]. The characteristic findings of IgG4-TIN are lymphoplasmacytic infiltrate, storiform fibrosis, and mild to moderate tissue eosinophilia [6, 18]. Immunohistochemical or immunofluorescence staining for IgG4 and IgG are often used to quantify the amount of IgG4-positive cells in the affected tissues [6, 14, 18]. Our patient fulfilled the suggested diagnostic criteria of IgG4-positive plasma cells >10/hpf. It is noteworthy that no granuloma was found in the kidney specimen, which ruled out the diagnosis of sarcoidosis. Renal histopathological findings, together with lymph nodes morphological and immunostaining manifestations, prompted the diagnosis of definite IgG4-TIN.

There is no standard medical therapy for IgG4-TIN till now. Steroid therapy is widely accepted to be the mainstay of IgG4-TIN treatment [2, 19]. Of note, even patients with markedly increased Scr and patients with diffuse interstitial fibrosis on biopsy show a response to therapy [20]. The initial daily dose of prednisone is 30-60 mg/d and treated for the first one to two months and then taper off to a maintenance dose within six to 12 months [20-22]. In the present patient, the Scr level had elevated to 3.45 mg/dL and renal histopathology showed diffuse fibrosis at the time of renal biopsy, however, prednisone therapy improved renal function, lymph nodes swelling, serum complements levels and eosinophil count. This may reflect steroid responsiveness of these lesions. On the other hand, the long-term prognosis of renal damage in IgG4-TIN is unclear, because the observation periods presented in articles are short. Although there are some case reports in which renal function has been stable for several years, their numbers are limited [20]. From the viewpoint of the manifestation of the IgG4-RD, the relapse rate in IgG4-TIN patients is about 20% [7]. It is also unclear whether or not

a small amount of steroid is necessary as maintenance therapy in IgG4-TIN. Patients who have been making satisfactory progress over the long term with maintenance therapy and developed TIN after discontinuance of steroid therapy support the concept that maintenance therapy is necessary in IgG4-TIN [20]. Indeed, our patient has been making satisfactory progress with 10 mg prednisone daily to date. Careful observation is necessary over the long term, regardless of maintenance therapy. Mycophenolate mofetil and rituximab has been used, however, the data regarding their efficacy are limited to small retrospective case series and case reports [6, 22].

In conclusion, to the best of our knowledge, this is the first case of IgG4-TIN associated with only lymphadenopathy and without elevated serum IgG or IgG4 levels or renal imaging abnormalities. IgG4-TIN is a still mysterious disease entity. Cases of IgG4-TIN are rarely reported in China, which might be attributed to the poor recognition of IgG4-RD. This case highlights the need for renal biopsy in renal insufficiency patients with unknown cause. It is necessary to accumulate cases of IgG4-TIN with detailed clinical and laboratory manifestations. Early diagnosis and steroid treatment are of great important in IgG4-TIN.

Disclosure of conflict of interest

None.

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