Case Report

Case Review of lgG4-Related Disease Patient with Combined Tubulointerstitial Nephritis and Membranous Nephropathy Coexisting with Cholangiocarcinoma: Case Report

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Immunoglobulin G4-related disease (IgG4-RD) has recently been recognized as an autoimmune disorder involving multiple organs. The kidney is a represented organ with a wide range of renal manifestations. The authors report a case of an 83-year-old Thai male with combined IgG4 tubulointerstitial nephritis and membranous nephropathy coexisting with cholangiocarcinoma. The patient presented with proteinuria, acute renal failure, eosinophilia, hypocomplementemia, and high serum IgG4 concentration. The diagnosis was IgG 4-related tubulointerstitial nephritis and membranous nephropathy on renal biopsy, with negative immunohistochemistry for anti-phospholipase A2 receptor antibodies. Magnetic resonance imaging (MRI) abdomen showed two wedge shaped arterial enhancing lesions of liver. Liver biopsy revealed adenocarcinoma, compatible with cholangiocarcinoma. Proteinuria and renal failure were resolved with initial steroid treatment. Meanwhile, IgG4-related membranous nephropathy should be considered in the differential diagnosis for patients with proteinuria. Potentially, IgG4-RD may be rarely associated with carcinoma development. However, further studies are recommended to ratify and confirm the association between IgG4-RD and incidence of malignancies.

Keywords: IgG4-related disease, Membranous nephropathy, Secondary membranous nephropathy, Tubulointerstitial nephritis, Cholangiocarcinoma

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Immunoglobulin G4-related disease (IgG4-RD) is an immune-mediated fibroinflammatory condition characterized by tumerfactive, tissue-destructive lesions or organ failures[1], which potentially involves multiple organs[2,3]. The hallmarks of IgG4-RD in histopathologic features are dense lymphoplasmacytic infiltrations with predominance of IgG4-positive plasma cell, obliterative phlebitis, and storiform fibrosis in the affected tissues[4]. Serum IgG4 levels are elevated (defined as greater than 135 mg/dL) in approximately two-thirds of the patients[5]. However, a minority of the patients maintain normal serum IgG4 concentrations despite the presence of typical histopathologic findings[6,7]. Pancreatic involvement is the most frequent manifestation of IgG4-RD, which can be also found in multiple other tissues including salivary glands, lymph nodes, lungs, prostate, thyroid, and kidney[8]. Importantly, the most common of IgG4-related kidney disease (IgG4-RKD) is tubulointerstitial nephritis (TIN). Nonetheless, few cases have been reported as glomerular lesions in IgG4-RD[9-11]. In addition, there could be a complex relationship between IgG4-RD and cancer. IgG4-RD represents a premalignant state, paraneoplastic condition[12-14] or malignancies complicating IgG4-RD due to chronic inflammation[15-17].

The authors present a cholangiocarcinoma case of combined IgG4-TIN and membranous nephropathy that responded well to the initial steroid treatment.

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Case Report

An 83-year-old Thai male presented with legs swelling, acute kidney injury, and proteinuria. He had history of fatigue with weight loss of three kg over the past two months prior to admission, chronic constipation, and higher sensitivity to cold and dry skin. His medical history showed old ischemic stroke, pseudogout, allergic rhinitis, and benign prostatic hyperplasia for three years. Surgical history revealed inguinal hernia repair and hemorrhoidectomy. Family history was unremarkable. Home medication included aspirin 81 mg/day, atorvastatin 20 mg/day, tamsulosin 0.4 mg/day, and fluticasone furoate nasal spray. Review of system yielded negative skin rash, oral ulcer, arthralgia, dyspnea, abdominal pain, change in bowel habits, and fever. Body temperature was 36.2 Celsius degree, with blood pressure 129/91 mmHg, heart rate 70 bpm, respiratory rate 20 breaths/minutes, oxygen saturation 98 percent, weight 60 kg, height 174 cm, and body mass index (BMI) 19.8 kg/m^2. Physical examination comprised pale conjunctiva, dry tongue, dry skin, cold fingers, and 1+ lower extremity edema. There was no evidence of salivary gland or thyroid enlargement, lymphadenopathy, hepatosplenomegaly, and abdominal tenderness. Laboratory and diagnostic workup were blood urea nitrogen (BUN) 27 mg/dL, creatinine 2.94 mg/dL (baseline creatinine 0.89 mg/dL), estimated glomerular filtration rate (GFR) 18.7 mL/minute/1.73 m^2, white blood cell (WBC) 13.18×10^3/µL (neutrophil 29%, lymphocyte 10%, eosinophil 60%) that elevated the absolute eosinophil count 7,908/µL, hemoglobin 9.3 g/dL, hematocrit 27.6%, Platelets 178×10^3/µL, total protein 8.6 g/dL, albumin 2.4 g/dL, aspartate aminotransferase (AST) 21 U/L, alanine aminotransferase (ALT) 16 U/L, alkaline phosphatase 107 U/L, total bilirubin 1.01 mg/dL, directed bilirubin 0.47 mg/dL, thyroid stimulating hormone (TSH) 7.78 (ref. 0.27 to 4.2), FT3 1.24 (ref. 2 to 4.4), and FT4 0.95 (ref. 0.93 to 1.7). Urinalysis showed proteinuria (2+) quantitated at 1.99 g/day, no WBC, red blood cell (RBC), and cast. Erythrocyte sedimentation rate (ESR) was elevated at 134 mm/hour. Serological evaluation demonstrated positive antinuclear antibodies (ANAs) (1:320 homogenous, 1:160 fine speckle, and 1:640 centromere pattern), positive anti-SSA and anticentromere B antibodies, negative anti-SSB, anti-dsDNA, anti-Sm and anti-RNP antibodies, positive antineutrophilic cytoplasmic antibody (ANCA) (weak positive anti MPO antibody and negative anti PR3 antibody by IBA), low C3 at 0.5 g/L, low C4 at 0.07 g/L. Immunoglobin tests showed the elevation of total IgG at 45.92 mg/mL, IgG1 at 15.89 g/L, IgG2 at 16.61 g/L, IgG3 at 6.36 g/L, IgG4 at 16.97 g/L (1,697 mg/dL), IgG4 at total IgG ratio 36.95%, and IgE 149 IU/mL with normal IgA and IgM quantity. There were no abnormalities on chest X-ray, with kidney ultrasound of right and left kidneys, 13.3×6.1 cm and 13×4.4 cm in size, as well as normal parenchymal echogenicity of both kidneys without stone or hydronephrosis, prostate gland enlarged, measuring about 47 mL.

Ultrasound guided renal biopsy was done and the renal biopsy tissue contained the cortex and medulla, with 10 glomeruli (Figure 1), and none of globally sclerosed. The glomeruli showed thickening of the capillary wall. The mesangium is focal mild increased matrix. The silver staining showed focal spikes. No crescent was seen. The tubules showed injury and focal mild tubulitis, and mild atrophy. The interstitium showed marked lymphoplasmacytic infiltration and moderate fibrosis. The arteries and arterioles were unremarkable. Immunofluorescence (IF) revealed IgG (2+ granular pattern, membrane staining), IgM (negative), IgA (trace), C3 (trace), C1q (negative), Fibrinogen (negative), Kappa (1+ granular pattern, membrane staining), and Lambda (2+ granular pattern, membrane staining) light chains. Electron microscopy illustrated electron dense deposits in subepithelium, intramembrane, and focal mesangium. The podocytes showed partial foot process effacement. Immunoperoxidase staining for IgG4 was positive in glomerular basement membrane of more than 10 IgG4 positive plasma cells per high power field (HPF), and increased IgG4/IgG plasma cell ratio of more than 30%.

Immunohistochemical stain for IgG4 met the current consensus of greater than 10 cells/HPF, with diagnosis of IgG4-RD. IgG4-related membranous nephropathy and IgG4 TIN was diagnosed. Serum IgG4 level was elevated at 1,697 mg/dL, supporting the diagnosis.

The patient's significant weight loss and negative anti-phospholipase A2 (PLA2) receptor antibody were consistent with a secondary etiology for membranous nephropathy, such as malignancy. Bone marrow aspiration and biopsy were normal. Computed tomography (CT) chest without contrast showed no abnormalities, with magnetic resonance imaging (MRI) whole abdomen of 4.2×4.1 cm wedge shaped arterial enhancing lesion in medial segment of left hepatic lobe, 2.8×1.7 cm wedge shape arterial enhancing lesion in hepatic segment V, and multiple enlarged lymph nodes along para-aortic, aortocaval regions, measuring up to 1.2 cm in short axis. Liver

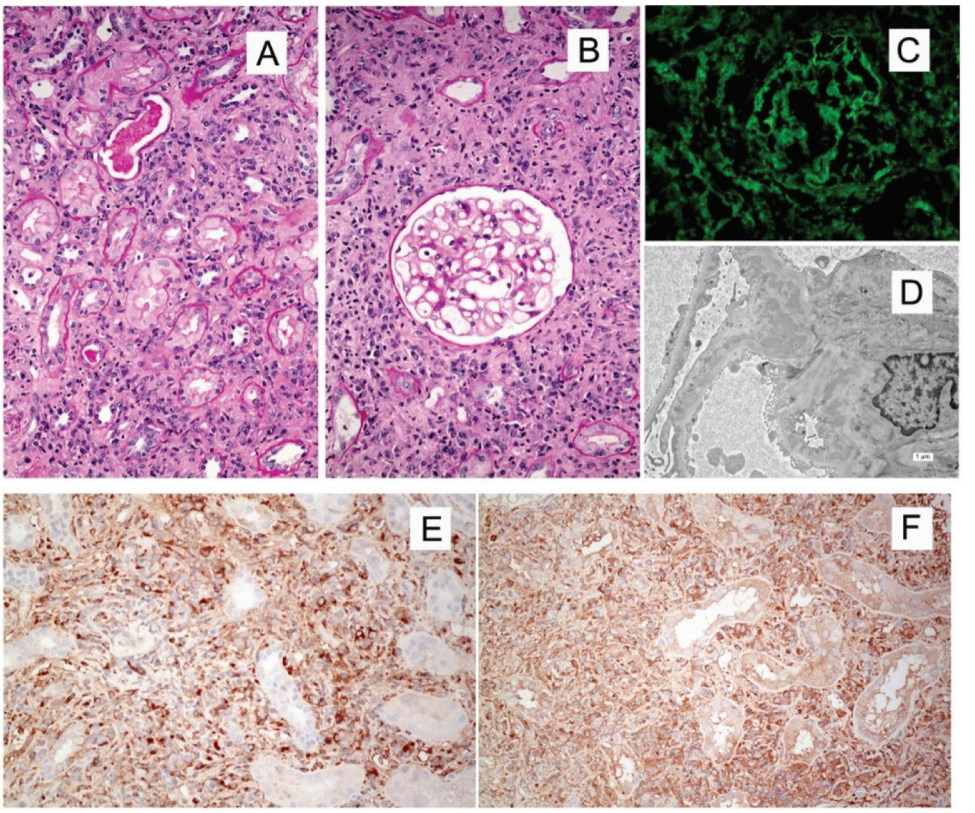

Figure 1. The kidney biopsy shows (A, B) diffuse lymphoplasmacytic infiltration in the interstitium with foci of tubulitis and mild thickening of the capillary wall. (PAS x400), (C) The immunofluorescent study shows granular IgG deposits in the GBM, mesangium and extraglomerular cells. (FITC x400), (D) The electron micrograph shows electron dense deposits in subepithelium and mesangium with diffuse foot process effacement. (x4,000) The immunoperoxidase staining for IgG (E) and IgG4 (F) show IgG4/IgG plasma cell ratio >30%.

biopsy revealed adenocarcinoma, compatible to cholangiocarcinoma.

The patient started on methylprednisolone 250 mg IV daily for three days before switching to prednisolone 40 mg PO daily (0.6 mg/kg/day) for four weeks and slowly tapering down to 10 mg PO daily due to steroid induced myopathy. His proteinuria was improved, seen with urine protein-creatinine ratio that decreased from 1.99 to 0.46, with lowered ESR from 134 to 14 mm/hour, and reduced serum creatinine level from 2.94 to 1.01 mg/dL. After two months of tapered prednisolone dose, proteinuria recurred (1.24 g/day). Mycophenolated mofetil (2 g/day) was then added to the regimen to resolve proteinuria. The patient was followed for 12 months after initial diagnosis, and until now, there was no recurrence of TIN, membranous nephropathy, extrarenal manifestations of IgG4-RD, and decreased symptoms of dry mouth that may be IgG 4-related salivary gland. Thyroid hormone replacement was administered with levothyroxine for hypothyroid that may be IgG4-RD. He denied thyroid and salivary gland biopsy, as well as treatment for cholangiocarcinoma.

Discussion

IgG4-RD is a multiorgan immune mediated condition that mimics many malignant, infectious, and inflammatory disorders[18,19]. In addition, autoimmune disorders such as Sjogren's syndrome, granulomatosis with polyangiitis, and idiopathic membranous nephropathy can also be difficult to distinguish from IgG4-RD[20]. Awareness of IgG4-RD is essential because it generally responds well to treatment.

IgG4, normally the least abundant of all immunoglobulin subclasses, accounts for approximately 4% of total immunoglobulins among healthy individuals. Many patients with IgG4-RD have the striking elevation of serum IgG4 concentrations and often have elevations of other IgG subclasses (particularly IgG1, IgG2, and IgG3)[7].

IgG4 demonstrates weak binding to C1q and Fc-gamma receptors, critically with few amino acid differences in its CH2 domain. As a result, the ability of IgG4 to activate the classic complement pathway and participate in antibody-dependent cell-mediated cytotoxicity[21,22]. The hallmark pathologic features of IgG4-RD include a lymphoplasmacytic infiltrate with a high percentage of plasma cells staining for IgG4, storiform fibrosis, obliterative phlebitis, and mild to moderate tissue eosinophilia[20].

Typical patients with IgG4-RD can be either middle-aged or elderly males, with male to female ratio of 3:2[7,23]. The presentation of IgG4-RD is typical subacute. The most frequently affected tissues are pancreas, major salivary glands, orbits, lymph nodes, and retroperitoneum[20]. The kidney is one of those organs, with various lesions collectively referred as IgG4-RKD[24]. TIN is the most common IgG4-RKD, named as IgG4-related TIN (IgG4-TIN). Approximately 15% to 24.6% of IgG4-RD patients are IgG4-TIN[10,25]. IgG4-TIN is often distinguished from other organ manifestations of IgG4-RD by the profound hypocomplementemia that be found in the authors' patient. The basis of this hypocomplementemia remains poorly understood[20]. Advanced renal dysfunction and even end stage renal disease can result. Proteinuria can develop, but the levels are usually subnephrotic.

The second commonest kind of renal parenchyma involvement is membranous nephropathy secondary to IgG4-RD. There have been reports of membranous nephropathy with and without IgG4-TIN. Alexander et al[26] reported on the largest series of IgG4-related membranous nephropathy cases, 55% of which (five out of nine patients) had concurrent IgG4-related TIN. Nonetheless, the etiopathogenesis of IgG4-related membranous nephropathy remains unclear.

Diagnosis of IgG4-related TIN and membranous nephropathy in the authors' patient can be supported by the following diagnostic criteria[24]:

1. Presence of subnephrotic-range proteinuria with renal dysfunction and elevated serum IgG4, IgE with hypocomplementemia.

2. Elevated serum IgG4 level (greater than 135 mg/dL).

3. Histological evidence of TIN and membranous nephropathy with dense lymphoplasmacytic infiltration greater than 10 IgG4-positive plasma cells/HPF with interstitial fibrosis.

The anti-PLA2 receptor antibody now known to cause "idiopathic" membranous nephropathy is not associated with IgG4-related membranous nephropathy[27]. Hence, anti-PLA2 receptor antibody is a useful marker for differentiating primarily from secondary membranous nephropathy, which seems negative in the authors' patient as expected in a case of secondary membranous nephropathy. Infections, autoimmune disease, drugs, and malignancy are main causes of secondary membranous nephropathy. Work-up on the authors' patient revealed the evidence of malignancy from cholangiocarcinoma. It was another cause of secondary membranous nephropathy other than IgG4-RD in the authors' patient. Recently, a series of published literature suggested that IgG4-RD is a cause of secondary membranous nephropathy[26-30].

In addition, the issue that IgG4-RD is associated with an increased incidence of total malignancies has not been clarified. Two recent studies have evaluated the SIR support for the association between IgG4-RD and total malignancies[14,17]. Yamamota et al reported a SIR for total malignancies of 3.8[17], while Shiokawa et al yielded a SIR of 2.7[14]. However, other studies showed that IgG4-RD was not associated with the increased incidence of total malignancies[31]. These studies were different at some points of inclusion criteria, especially the period between the onset of IgG4-RD and the occurrence of malignancy. Two opposite conclusions were that "IgG4-RD causes malignancy" and "malignancy causes IgG4-RD". On the other hand, it was difficult to determine the exact time of onset of IgG4-RD or malignancy in many cases, particularly in patients without any symptoms. Interestingly, no cases of IgG4-RD involved the organs previously affected by cancer[32]. IgG4-RD may develop as a paraneoplastic syndrome in some patients. The immunoreactions to cancer may induce this disease because cancer could trigger autoantigen expression, leading to IgG4-RD[32]. However, there appears to be inadequate evidence to support this at the present time. Malignancies complicating IgG4-RD can be classified as lymphoma and non-lymphoid tumors. Lymphoma could be a consequence of chronic inflammation in IgG4-RD[15]. In the present patient, the chronology of IgG4-RD and cholangiocarcinoma may be possibly that IgG4-RD was either a paraneoplastic syndrome or an independent disease.

Current approaches for management are based on observational case series. No randomized clinical trials have been performed to evaluate the comparative effectiveness of different treatment regimens. Glucocorticoids are currently the first-line treatment for IgG4-RD[33]. Most data on the use of glucocorticoids are derived from treatment of IgG4-related pancreatitis (type 1 AIP). A consensus

statement from Japan suggested an initial dose of 0.6 mg/kg/day for two to four weeks, followed by three to six months of tapering period to 5 mg/day, and continued 2.5 to 5 mg/day for up to three years[34]. In IgG4-RKD, the initial dose of prednisolone should be up to 40 to 60 mg/day[19,35]. Saeki et al[10] and Raisson et al[11] demonstrated a good, rapid response rate to prednisolone in patients with elevated serum creatinine. The authors' patient received prednisolone 40 mg PO daily for four weeks and improved significantly after four weeks of treatment. Glucocorticoids were slowly tapered down to 10 mg PO daily after four months due to steroids induced myopathy. Glucocorticoids sparing therapy including azathioprine and mycophenolated mofetil can be effective in the absence of concurrent glucocorticoids[36]. The present patient received mycophenolated mofetil (2 g/day) as maintenance medications after glucocorticoid-induced remissions. If the above treatment dose did not suffice, or patients had poor prognostic features including advanced renal insufficiency, B cell depletion therapy with rituximab should be considered[35,37,38]. Development of therapies targeting B cell and T cell lineage has been proposed. The pathophysiology of IgG4-RD such as plasmablast-directed therapy with a CD19 monoclonal antibody is in clinical trials[39].

A disease responder index is a tool designed to detect any changes in disease activity and identify improvement and worsening in the same or in different organ systems. An IgG4-RD Responder Index has been developed using clinical investigations and laboratory-based data[40]. In addition, recent multispecialty study has validated the Responder Index that the IgG4-RD Responder Index is a valid and reliable disease activity assessment tool, which can be used to measure the response to the therapy[41].

Conclusion

The authors described a case of IgG4-RKD with combined TIN and membranous nephropathy with favorable response to the combination of prednisolone and mycophenolated mofetil. However, the patient refused treatment of cholangiocarcinoma. IgG4-RKD and cholangiocarcinoma could be either a paraneoplastic syndrome or an independent disease.

What is already known on this topic?

IgG4-RD is a multiorgan immune mediated condition. Few cases have been reported as glomerular lesions in IgG4-RD. In addition, there could be a complex relationship between IgG4-RD and cancer.

What this study adds?

This report showed combined IgG4 tubulo-interstitial nephritis and membranous nephropathy coexisting with cholangiocarcinoma that presented with glomerular disorders. Awareness of IgG4-RKD is essential because it generally responds well to treatment. Furthermore, this case found malignancy that may be associated with IgG4-RD.

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Conflicts of interest

The authors declare that there is no conflict of interests regarding the publication of this paper.

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