When Crohn's disease meets IgA nephropathy: What do you think?

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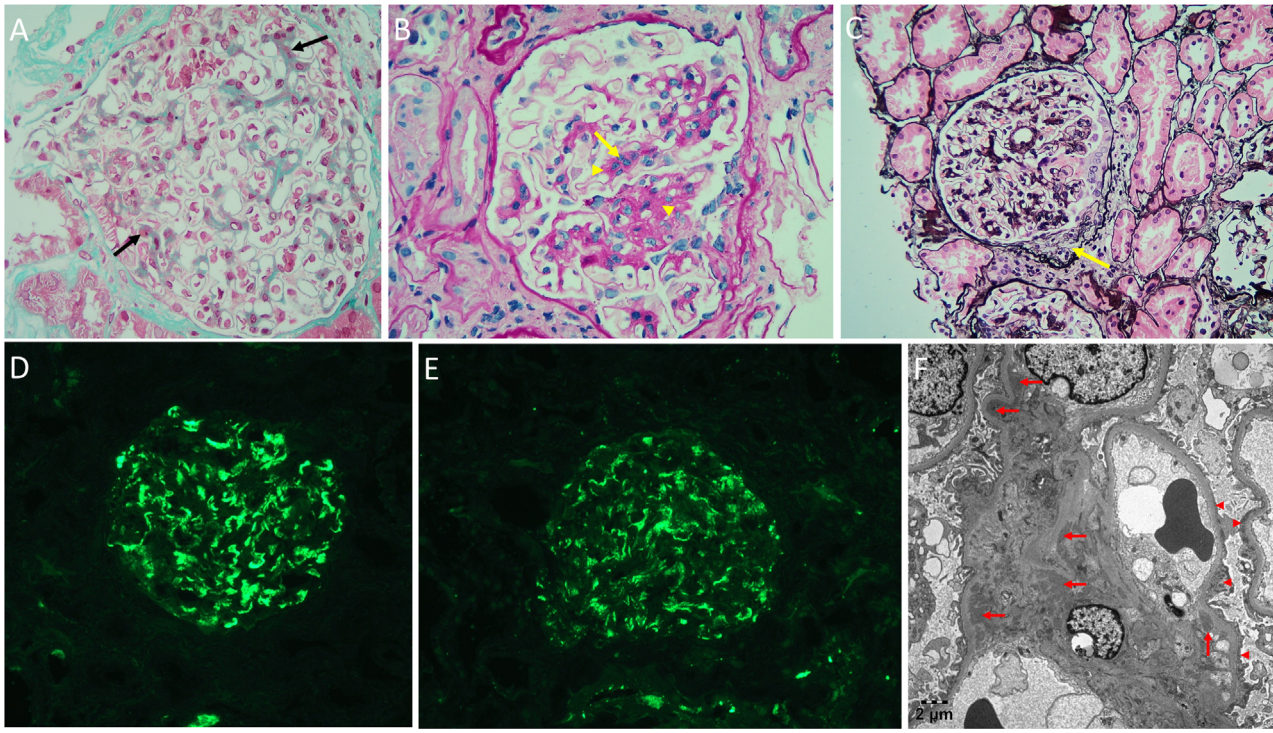

FIGURE 1.

CASE PRESENTATION

A 40-year-old man was admitted to our hospital with a 1-year history of persistent hematuria and proteinuria, accompanied by elevated serum creatinine. He had previously been diagnosed with Crohn's disease (CD) via postoperative pathologic examination three years earlier and treated with azathioprine to prevent postoperative recurrence. He and his family had no history of kidney diseases. He was afebrile and had no gross hematuria, foamy urine, lumbago, diarrhea, abdominal pain, or malaise. Physical examination was unremarkable. Urinalysis revealed hematuria (+++) and proteinuria (++). Blood tests showed mildly elevated serum creatinine (1.31 mg/dl) and immunoglobulin A (IgA, 4.85 g/L). There were no obvious abnormalities in liver function, thyroid function, autoimmune disease indicators, and tumor indicators.

To our knowledge, azathioprine has not been associated with increased risk for renal insufficiency. So, what reasons would that be?

In order to clarify the etiology for renal insufficiency, renal biopsy was performed and revealed deposition of eosinophilic material in mesangial regions (Fig. 1A, arrows), mild/moderate mesangial matrix increase (Fig. 1B, arrowheads) and mesangial cell proliferation (Fig. 1B, arrow), the presence of cellular crescents (Fig. 1C, arrow) as well as glomerular adhesions (Oxford classification: M1 E0 S1 T0 C1). Immunofluorescence examination of glomeruli demonstrated IgA (3+) (Fig. 1D), C3 (3+) (Fig. 1E) and IgM (+) deposits in the mesangium. Electron microscopy revealed the presence of prominent electron-dense deposits in mesangial areas (Fig. 1F, arrows) and foot process fusions (Fig. 1F, arrowheads). The above-mentioned findings of examinations were consistent with the diagnosis of focal proliferative IgA nephropathy (IgAN) and Lee grading III was presented. Subsequently, he began treatment with prednisone 30 mg once daily, hydroxychloroquine 100 mg twice daily, mycophenolate mofetil 500 mg twice daily and

candesartan 4 mg once daily. After a period of medical therapy, the renal function improved gradually. The patient remains under close follow-up.

A wide range of extraintestinal manifestations of inflammatory bowel diseases (IBD) occur in both CD and ulcerative colitis (UC) patients, and they can affect any system, including the hepato-pancreatobiliary, renal, musculoskeletal, ocular, dermatologic, and pulmonary systems. Although urologic complications (mainly for urinary calculi, enterovesical fistulas, and ureteral obstruction) were found in around 4–23% of patients with IBD, the concomitant presence of CD and IgAN is still rare.[1] Many evidences argue that a lot of con-founding factors may play an important role in coexisting CD and IgAN, such as the increased permeability of intestinal mucosa, increased number of the mucosal IgA, dysfunction of T-cells, and a susceptible gene (HLA-DR1).[2] Interestingly, different from the previously reported case, the patient in our case was on azathioprine only during the course of therapy when IgAN was diagnosed, instead of being treated with biological agents or 5-aminosalicylates.[3] Moreover, our patient had good control of the primary disease. Consequently, we have confidence to conclude that the patient's renal insufficiency in our case was associated with CD.

Our case suggests that when a patient with CD experiences renal insufficiency, the suspicion of IgAN should be considered.

FUNDING STATEMENT

This work was supported by the Medical and Health Guiding Project of Xiamen (3502Z20214ZD1028).

ETHICAL STATEMENT

Written informed consent was obtained from the patient for publication of this "Online Images in the Medical Sciences".

DECLARATION OF COMPETING INTEREST

The authors have no conflicts of interest to declare.

REFERENCES

1. **Shield DE, Lytton B, Weiss RM, et al.** Urologic complications of inflammatory bowel disease. *J Urol*. 1976;115(6):701–706.
2. **Terasaka T, Uchida HA, Umebayashi R, et al.** The possible involvement of intestine-derived IgA1: a case of IgA nephropathy associated with Crohn's disease. *BMC Nephrol*. 2016;17(1):122.
3. **Oikonomou K, Kapsoritakis A, Eleftheriadis T, et al.** Renal manifestations and complications of inflammatory bowel disease. *Inflamm Bowel Dis*. 2011;17(4):1034–1045.