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Febrile Inaugural Ileitis Revealing Lupus Extra Membranous Glomerulonephritis: A Case Report

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Abstract | **Case Report**

Lupus extra membranous glomerulonephritis [EMG], corresponding to class V lupus nephritis, is usually revealed by nephrotic syndrome. Gastrointestinal manifestations of Systemic Lupus Erythematosus [SLE] are rare but are now better characterized in recent guidelines, and are exceptionally inaugural, potentially delaying diagnosis. We report the case of a 27-year-old woman initially hospitalized for a febrile digestive syndrome characterized by diarrhea, abdominal pain, and general deterioration. Abdominal computed tomography scan showed inflammatory ileitis, initially suggestive of an infectious etiology, but an extensive microbiological workup proved negative. The clinical course was marked by the subsequent development of generalized edema revealing severe nephrotic syndrome, leading to transfer to our nephrology department. Laboratory investigations demonstrated profound hypoalbuminemia, massive proteinuria, and microscopic hematuria. Immunological testing revealed severe hypocomplementemia, positive antinuclear antibodies, weakly positive anti-dsDNA antibodies, and positive anti-SSA and anti-RNP antibodies, suggesting active systemic lupus disease. The patient also presented with moderate pericardial effusion and inflammatory myositis on thigh MRI. Kidney biopsy confirmed lupus membranous nephropathy with IgG1, IgG2, and C1q deposits, allowing differentiation from primary membranous nephropathy. Treatment combining corticosteroids, mycophenolate mofetil, and hydroxychloroquine led to a favorable outcome, with regression of digestive manifestations and significant reduction in proteinuria.

Keywords: Lupus ileitis; nephrotic syndrome; systemic lupus erythematosus; class V lupus membranous nephropathy.

Clinical Presentation and Management

The patient was a 27-year-old woman with no significant past medical history, was initially admitted to the internal medicine department for febrile diarrhea evolving over several weeks, associated with diffuse abdominal pain, anorexia, weight loss, and marked deterioration of general condition, with fever reaching 40°C.

The initial thoraco-abdomino-pelvic computed Tomography scan revealed mild mural thickening of dependent ileal loops suggestive of inflammatory ileitis. Bilateral pulmonary micronodules were also noted. Assuming an infectious origin, empiric antibiotic therapy with a third-generation cephalosporin and metronidazole, later supplemented with spiramycin, was initiated without clinical improvement. Extensive infectious investigations, including blood cultures, stool cultures, stool parasitology, testing for Clostridium difficile, and investigations for tuberculosis, remained negative; the Quantiferon test was non-contributory.

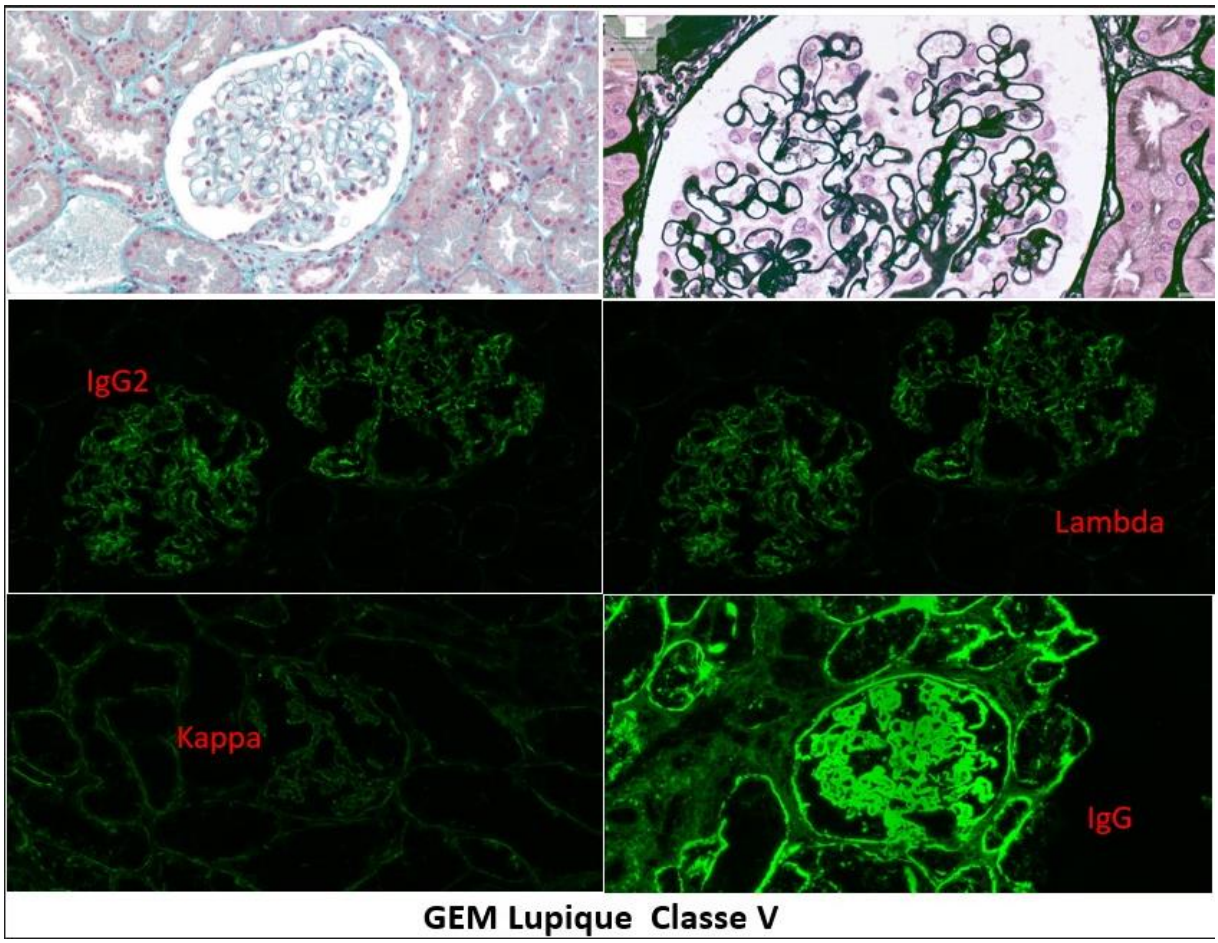
The clinical course was marked by the secondary appearance of generalized edema syndrome with ascites, prompting transfer to the nephrology department on day 7 of hospitalization. On admission to nephrology, the patient was afebrile but had diffuse edema. There was no skin rash, photosensitivity, arthritis, or palpable lymphadenopathy. She complained of thigh pain, predominantly on the left side, associated with sensorimotor deficit of the left lower limb and absence of the patellar reflex.

Laboratory investigations revealed serum creatinine of 99 µmol/L and an impure nephrotic syndrome with proteinuria estimated at 4 g/L, severe hypoalbuminemia at 7.2 g/L, and microscopic hematuria. Hepatic cholestasis, rhabdomyolysis, and non-regenerative anemia with folate deficiency and a positive direct antiglobulin test without evidence of hemolysis were also noted. CRP was moderately elevated at 24 mg/L and LDH slightly increased.

Immunological workup revealed antinuclear antibodies at a titer of 1:320, weakly positive anti-dsDNA antibodies, positive anti-SSA and anti-RNP antibodies, associated with profound hypocomplementemia [C3 at 0.47 g/L, C4 at 0.09 g/L, decreased CH50], supporting the diagnosis of active systemic lupus erythematosus.

Echocardiography showed moderate pericardial effusion without signs of tamponade. Brain and spinal MRI were normal. Thigh MRI demonstrated inflammatory myositis predominantly affecting the anterior compartment with early posterior involvement, and electroneuromyography suggested associated neurogenic involvement.

Renal biopsy confirmed secondary lupus membranous nephropathy, characterized by subepithelial deposits of IgG1, IgG2, and C1q, allowing distinction from primary membranous nephropathy, a key element for therapeutic strategy, as showed on the figure below:



GEM Lupique Classe V

The patient received transfusion of two units of packed red blood cells and was treated with methylprednisolone pulses at a dose of 500 mg/day for three days, followed by oral corticosteroids at 1 mg/kg/day, combined with mycophenolate mofetil and hydroxychloroquine. The outcome was favorable, with progressive regression of edema, improvement of digestive and muscular symptoms, stabilization of renal function [serum creatinine 63 µmol/L], and significant reduction of proteinuria to 0.8 g/L.

DISCUSSION

Gastrointestinal manifestations of Systemic Lupus Erythematosus are rare and exceptionally inaugural, often making diagnosis difficult [4, 8]. Lupus ileitis generally occurs within the context of lupus enteritis, an entity related to mesenteric small-vessel vasculitis secondary to immune complex deposition [4, 9].

Clinically, it frequently mimics gastrointestinal infection or inflammatory bowel disease, exposing patients to diagnostic delay [4, 9]. Abdominal computed tomography scan is the key diagnostic examination, demonstrating intestinal wall thickening, "target sign" mucosal enhancement, and mesenteric congestion, with a predilection for the small intestine, particularly the ileum [4, 9].

Several series have shown that lupus enteritis is often associated with high systemic SLE activity, marked by profound hypocomplementemia, and may precede or accompany active lupus nephritis [2, 4, 8]. A Korean study of 17 patients with lupus enteritis reported a good response to immunosuppressive treatment [9]. A 2013 review of published cases found that, unlike in our case, lupus ileitis is very rarely the inaugural manifestation of the disease [10].

Lupus membranous nephropathy accounts for approximately 10–20% of lupus nephritis cases [2, 3]. The presence of IgG and C1q deposits reflects activation of the classical complement pathway and helps distinguish this entity from primary membranous nephropathy, thereby guiding therapeutic strategy [5, 6]. The association observed with inflammatory myositis and pericardial effusion, in the absence of cutaneous or articular manifestations, illustrates the marked clinical heterogeneity of SLE.

Treatment based on corticosteroids combined with mycophenolate mofetil and hydroxychloroquine generally results in favorable outcomes.

CONCLUSION

Febrile ileitis may constitute a rare inaugural manifestation of Systemic Lupus Erythematosus and may mask underlying lupus renal involvement. In any case of unexplained ileitis associated with hypocomplementemia and immunological abnormalities, the diagnosis of SLE should be considered. Early kidney biopsy and appropriate immunosuppressive management can improve prognosis and avoid therapeutic delay [7, 12].

Conflict of interest

The authors declare no conflict of interest.

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