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Early IgA Nephropathy Complicated by Severe Anemia: A Rare Phenotype

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Letter to the Editor,

IgA nephropathy (IgAN), also termed Berger disease, is the prevalent form of primary glomerulonephritis worldwide and a major associated risk factor for chronic kidney disease (CKD), commonly among young adults.[1] Even though a substantial proportion of patients eventually progress to kidney failure, severe anemia is generally known to be a complication of advanced CKD. Here we report an unusual presentation of persistent IgAN with early glomerulosclerosis complicated by associated microcytic hypochromic anemia in a young female with early-stage CKD, highlighting the importance of anemia as a potential early marker of ongoing disease activity.[2,3]

A 19-year-old woman presented with the complaints of generalized weakness, exertional dyspnea, pruritus, and hematuria since 2 weeks, associated with a recent syncopal episode. The patient had a past history of biopsy-proven IgAN established in 2019 after presenting with painless gross hematuria and nephrotic-range proteinuria. The initial kidney biopsy showed mesangial proliferation with IgA deposition. The patient received long-term immunosuppressive therapy including corticosteroids, mycophenolate mofetil, and cyclophosphamide, with only partial clinical response, and had discontinued treatment a year ago. Written informed consent was obtained from the patient before the study.

On examination, the patient was pale in appearance but hemodynamically stable. Laboratory evaluation revealed severe anemia with hemoglobin of 4.9 g/dl, microcytosis, and hypochromia, along with mildly elevated serum creatinine of 2.0 mg/dL, consistent with early CKD. Given the severity of anemia, the patient was administered a packed red blood cell transfusion and iron supplementation. Anemia of this severity is rarely reported in patients with early-stage CKD, which signifies the need for further evaluation.

By taking the patient's disease history, a repeat kidney biopsy was done, which highlighted persistent mesangial hypercellularity with early glomerulosclerosis involving 2 of 6 glomeruli, without crescent formation. These findings suggest worsening active IgAN despite prior immunosuppressive therapy. The patient was initiated on angiotensin-converting enzyme inhibitor therapy for blood pressure and proteinuria control and reintroduced to mycophenolate mofetil, while corticosteroids were avoided due to previous toxicity as reported earlier.

The pathogenesis of anemia in this patient appears multifactorial. Chronic inflammation presented with persistent IgAN activity may cause iron-restricted erythropoiesis via hepcidin-mediated pathways, while ongoing microscopic hematuria can lead to iron loss.[4] Apart from this, CKD-related hypoxia and dysregulation of

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hypoxia-inducible factor pathways may impair erythropoietin response even in early kidney impairment. Previous exposure to immunosuppressive agents may have further influenced bone marrow reserve and iron metabolism.[5]

This case shows an uncommon and aggressive IgAN phenotype in which continuous histological disease activity coincided with the development of severe anemia early in the course of CKD. It emphasizes the significance of prompt repeat kidney biopsies, intensive clinical monitoring, and customized treatment plans for refractory IgAN. Crucially, anemia in these individuals may be an early sign of persistent inflammatory and structural kidney damage rather than being written off as a late consequence of kidney failure.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

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