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Case Report

A case report on IgA nephropathy

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Abstract

IgA nephropathy (IgAN) is one of the most common glomerulonephrites in the Western world. Its diagnosis is based on clinical history, laboratory data, and histopathological reports. The clinical picture and spectrum of severity upon presentation vary between cases. In this case report, we present a previously healthy 23-year-old Indian male with rapidly progressing IgA nephropathy leading to severe acute renal impairment. This case report highlights histopathological and clinical characteristics of a case of severe IgA nephropathy.

Keywords: IgA, Mesangial Deposits, Nephropathy.

Case report

A 23-years-old male was admitted to the casualty of a tertiary care center with chief complaints of vomiting and loss of appetite for 20 days. He had a history of vomiting which was aggravated with food intake and the vomitus was mixed with food particles, but not bile/blood stained. Otherwise, there was no history of reduced urine output/fever/cough/abdominal pain/loose stools.

He was provisionally diagnosed with glomerulonephritis in 2013 in another hospital but was on irregular treatment. He is a newly diagnosed hypertensive. No history of diabetes/asthma/heart disease. No history of surgical illness. He is a non-smoker and non-alcoholic. There was no family history of renal disease/hypertension/diabetes in the family.

On examination, he was conscious, oriented and had pallor. Otherwise, no signs of icterus/cyanosis/clubbing/pedal edema/lymphadenopathy were observed. His vitals were stable. His cardiovascular, respiratory, neurological system examinations were normal. His per abdominal inspection was normal and palpation was soft with no evidence of organomegaly or mass.

His investigations revealed an elevated Blood Urea Nitrogen of 200 mg/dl and serum creatinine of 41.63 mg/dl on admission. He had dyselectrolytemia with elevated serum calcium (15.8 mg/dl), serum phosphorus (16.1 mg/dl) and serum uric acid levels (14.1 mg/dl). He had reduced levels of serum sodium (122 mmol/l), serum potassium (2.5 mmol/l), serum chloride (75 mmol/l) and serum bicarbonate levels (10 mmol/l).His complete hemogram picture was also deranged with low levels of blood hemoglobin (4.4 g/dl) and packed cell volume (PCV=13%). He had low RBC count (1.48 million/cu.mm) and platelets (77,000/cu.mm) with an elevated total WBC count (17,500 cells/cu.mm with 81.4% of neutrophils). Other baseline investigations were also done. Ultrasound abdomen revealed bilateral inward renal cortical echoes with the cortico-medullary disease. A renal biopsy was taken and it showed normal renal cortex and medulla.

But immunohistochemistry showed small mesangial IgA deposits in the glomeruli. Thus he was diagnosed with IgA nephropathy.

He was treated with all supportive medications to correct his derangements. He was given packed blood cells transfusion and hemodialysis. His repeat blood samples were reported with values of BUN as 143 mg/dl and serum creatinine as 32.24 mg/dl. As per nephrologist advice, he was continued with intensive care and was stabilized. The patient was advised on regular treatment, follow-up and necessary dietary advice.

IgA nephropathy is one of the most common glomerulonephritis worldwide and is an important cause of renal failure. Abnormally high levels of circulating poorly O-galactosylated IgA1 and O-glycan antibodies forms of IgA1 immune complex molecules and its subsequent mesangial deposits cause inflammation and glomerular injury. Among both the subclasses of IgA namely IgA1 and IgA2, the former is only observed in mesangial deposits.

Various genetic and environmental factors are also expected to play a role in the pathogenesis of the disease. But the entire mechanisms behind the disease are yet to be resolved. It is a slowly progressive disease. Renal biopsy of IgA1 deposits remains the confirmative diagnostic method. Other mesangial deposits like C4D and IgA subepithelial deposits have also been observed rarely in early nephropathy.

The mainstay of treatment is mainly immunosuppressants and supportive measures to control blood pressure. Newer research on the therapy is still ongoing due to heterogeneity and the complex pathogenesis of the disease.

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