



GLOMERULONEPHRITIS AND RENAL FAILURE IN RHEUMATOID ARTHRITIS: A DIAGNOSTIC AND THERAPEUTIC CHALLENGE.

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BACKGROUND

Renal manifestations in rheumatoid arthritis (RA) can be divided into sequelae of RA (secondary amyloidosis, glomerulonephritis (GN), increased cardiovascular risk factors) and medication nephrotoxicity (nonsteroidal anti-inflammatory drugs, gold, penicillamine, and cyclosporine). Other nephropathies, such as minimal change disease, focal segmental glomerulosclerosis (FSGE), lupus GN after TNF inhibitors and cases of antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis have been rarely reported. The level of proteinuria, active urinary sediment, and clinical symptoms do not accurately predict the renal histology in RA. We will report a case in which renal biopsy was necessary to guide therapeutics.

CASE REPORT

A 44 year old white female with RA for 11 years, positive rheumatoid factor and ACPA, presented for a routine follow-up visit. She had a past history of resistant hypertension, chronic kidney disease stage IIIb, and obesity. Her RA medication consisted of methotrexate 15 mg/week and prednisone 15 mg/day. Upon consultation she had a CDAI score of 10, DAS28-CRP score of 3,32, and laboratory results showed acute-over-chronic kidney injury (baseline creatinine was 1.7 mg/dL, consultation day creatinine was 2.26 mg/dL), proteinuria and hematuria. She had presented hematuria several times before, occasionally with dysmorphic red cells, which prompted an investigation of glomerulonephritis. An ANCA essay was positive with a c-ANCA pattern, titrated at 1/80. She was admitted to hospital for a renal biopsy. During her days as an inpatient there was marked increase of creatinine levels (max creatinine 3.17 mg/dL) with no need for dialysis. Her 24 hour proteinuria was 330 mg/dL. Anti-myeloperoxidase and anti-proteinase 3 antibodies were negative. Histopathological analysis of renal biopsy showed FSGE, discrete mesangial hypercellularity, acute tubular necrosis and hyaline arteriosclerosis. Immunofluorescence staining showed mesangial deposits of IgA, IgM, C3 and lambda. The biopsy was consistent with chronic hypertensive and IgA-associated nephropathy.

CONCLUSION

The major histopathological finding in kidney biopsies in RA is mesangial glomerulonephritis predominantly with IgM deposits, followed by IgA. IgA is usually deposited with C3, and its intensity correlates with serum IgA levels, higher RF titers, duration and intensity of RA. In more recent years, renal failure in RA appears to be predominantly associated with cardiovascular metabolic risk factors (hypertension and insulin resistance), rather than the severity of RA. Our patient presented the two most common renal patterns of RA, and her treatment should consist of rigorous control of disease activity and cardiovascular metabolic risk factors.