

# IgA nephropathy in an Ankylosing Spondylitis patient during infliximab therapy: chicken, egg or mother and child reunion?

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ACTA REUMATOL PORT. 2013;38:310

To the editor,

A 35-year-old man was seen during a control visit for ankylosing spondylitis (AS). After a 7-8 year history of low back and hip pain, he had been diagnosed with AS 3 years ago and he has been under infliximab treatment for the last 2 years. The medical history was otherwise noncontributory. His current physical examination revealed mild limitations in bilateral hip joints and cervical/lumbar motions. Complete blood count, liver function tests and acute phase reactants were all normal. However, he had increased creatinine (1.42 mg/dL, N: 0.7-1.2) and blood urea nitrogen (22.93 mg/dL, N: 6-20) levels and proteinuria (100 mg/dL). Upon consultation to the nephrology department, he was diagnosed to have immunoglobulin A (IgA) nephropathy (with 40-50% of tubular atrophy and interstitial fibrosis in renal biopsy). He was switched onto adalimumab and followed in accordance with recommendations of his nephrologist (antihypertensive treatment and diet).

Ankylosing spondylitis (AS) can present with a wide variety of extra-articular manifestations one of which is renal involvement. The clinical scenario may comprise amyloid nephropathy, IgA disease, hematuria, microalbuminuria and decreased renal functions<sup>1</sup>. Although the coexistence of AS and IgA nephropathy has been reported a few times in the previous literature, the exact underlying mechanism is yet unclear<sup>2,3</sup>. Further, because they have common etiopathogenetic features, 'whether they occur as two distinct disorders' becomes disputed when they coexist<sup>2, 4, 5</sup>. Increased IgA levels are mainly due to overproduction and/or IgA catabolism defect<sup>4</sup>. IgA deposits and immune complexes (mainly composed of IgA1) which occur as a result of the glycosylation defect, activate mesangial cell and extra-cellular proliferation, and induce pro-inflammato-

ry mediators/growth factors. In relevant patients, uric acid and IgA levels can be used for the prediction of renal involvement; however a renal biopsy is crucial for prompt diagnosis and treatment<sup>3,6</sup>.

Concerning the treatment of IgA nephropathy, previous data show conflicting and insufficient results. While anti-TNF $\alpha$  agents improve the rheumatological disease, they cannot control IgA nephropathy. Further, IgA nephropathy can even develop during anti-TNF $\alpha$  treatment<sup>2,3</sup>. On the other hand, angiotensin converting enzyme inhibitors and TNF $\alpha$  blockers can be used to diminish the proteinuria<sup>2</sup>. Initial serum creatinine level might be a predictor for the treatment response as well<sup>3</sup>. As a result, herein presenting the unusual case of ours, we would like to highlight once again the importance of early/correct diagnosis of renal involvement in AS. Whether or not AS shows a controlled disease activity, clinicians should perform a close follow-up to prevent mortality and morbidity due to renal involvement.

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