EARLY ONSET OF AA AMYLOIDOSIS ASSOCIATED WITH JUVENILE IDIOPATHIC ARTHRITIS

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Key words. Juvenile idiopathic arthritis, Chronic inflammation, AA amyloidosis, Renal involvement

Introduction. Juvenile idiopathic arthritis (JIA) is an autoimmune disease of unknown origin and one of the more common chronic illnesses of childhood [*Kliegman et al., 2013*]. Systemic AA amyloidosis is the result of chronic inflammatory disease with frequent manifestation of renal functional impairment and with massive urinary protein excretion. Amyloidosis-associated kidney disease usually progress to end-stage-renal-disease (ESRD) and is major source of morbidity [*December, 2006*].

Case report description. A 26-year-old man with anamnesis of JIA since the age of 8 was admitted for the Infliximab infusion. The patient has follow-up by rheumatologist, and the use of medications due to diagnosis is regular: methotrexate once a week, folic acid, methylprednisolone, calcium and D3 vitamin every day, the course of Infliximab is received in a ward. The main complaint is diffuse morning stiffness in the backbone. Physical examination findings are unremarkable, except the Cushing syndrome due to steroid use and presentation of diminished vision in both eyes because of chronic uveitis. Proteinuria of 2.35g/day is revealed therefore the kidney biopsy is indicated. Laboratory results displays hypochromic microcytic anemia, leukocytosis. C-reactive protein (CRP) is 123.8mg/l with the previous value of 203.1mg/l one months ago. Glomerular filtration rate (GFR) is 357.7ml/min (creatinine=36μmol/l) but the previous result accounted 224ml/min (creatinine=58μmol/l), serum albumin- 27g/l.

The kidney biopsy sample contains 22 glomeruli, none of them were globally sclerotic. Congo Red stain is positive with apple green birefrigence under polarized light for amyloid accumulation in the 5% of the glomerular

area, arteries and arterioles. Immunohystochemical reaction for AA protein is positive in amyloid depositions. The pathological diagnosis of a very early AA amyloidosis is established. 2 months follow-up laboratory results: hypochromic microcytic anemia, leukocytosis with neutrophylia, ESR is 65mm/h, CRP of 84.7mg/l, proteinuria of 0.75g/l and GFR 186 ml/min (creatinine=70μmol/l).

Conclusions. AA amyloidosis is the result of continuous, long term inadequate control over chronic inflammation. The early stage of renal involvement is based on absence of nephrotic syndrome, hypertension, edema and ESRD. Early diagnosis of renal AA amyloidosis and management of JIA is paramount to prevent progression of chronic kidney disease.

Summary. The case of 26-year-old man with anamnesis of JIA for 18 years is presented. During the regular follow-up and medication course due to JIA the proteinuria is observed in urine analysis. The suspicion of AA amyloidosis is proved by kidney biopsy. The early AA amyloidosis is ascertained by only 5% morphological changes and absence of the nephrotic syndrome and hypertension. Therapy to suppress the inflammatory disease is used whenever possible, and laboratory parameters during follow-up after the kidney biopsy are slightly better than before, however, the significant improvement is not observed which raises the suspicion of still present activity of JIA and ongoing chronic inflammation despite aggressive therapy and consequently AA amyloidosis.