

PROGNOSTIC FACTORS FOR RENAL AMYLOIDOSIS DEVELOPMENT IN RHEUMATOID ARTHRITIS PATIENTS

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Background: Secondary amyloidosis (SA) is common renal damage in RA. Reported 5-year survival rate in patients with SA was 30%[1]. Renal amyloidosis is considered to develop in 10-15% RA patients [2]. Male sex, early RA onset, positive RF, severe course, extraarticular manifestations, poor activity control, high persistent serum CRP level were published as risk factors for SA [3]. We assumed that persistent *Chlamydia trachomatis* infection may also contribute to SA development in RA.

Objectives: To estimate the prognostic factors for renal amyloidosis development in RA Byelorussian patients.

Methods: We examined 104 RA patients - 45 with histologically confirmed renal amyloidosis and 59 without it for *SAA1* gene allele polymorphism and *C. trachomatis* infection. Other possible risk factors (sex, age, RA activity by SDAI, X-ray stage, RA onset, duration, extraarticular manifestations, CRP and RF level, therapy required for disease control) were also assessed. Segment of *SAA1* gene from blood leucocyte native DNA including -13 T/C, 2995 C/T and 3010 C/T sites was genotyped by PCR (MyCyclerTM Termal cycler, BIORAD) with subsequent restriction enzyme digest analysis. -13 T/C locus polymorphism was detected by AcII endonuclease ("Fermentas", Vilnius) and electrophoretic separation in 8% polyacrilamide gel. 2995 C/T and 3010 C/T polymorphic sites were detected by BanI (2995 C/T) and BclI (3010 C/T) endonucleases correspondingly. All restriction reactions were conducted according to MBI Fermentas instruction. Each polymorphic site was detected separately. Fragments were separated by electrophoresis in 2% agarose gel with UV detection (Vilber Lourmat transilluminator, France), results were documented by Nikon 2100 digital camera. *C. trachomatis* infection was detected by PCR and/or by cultural method in urethral or cervical scrapes.

Results: We revealed association between 2995C/T and 3010C/T (*SAA1*) genotype variants and renal amyloidosis ($R=0.93$; $p<0.0001$). The most notable differences were observed in α/α genotype ($\chi^2=31.1$; $p<0.001$). 43 of 45 AA-positive RA patients had α/α genotype vs 32.2% AA-negative RA patients (OR for α/α genotype was 45.3; 95%CI:9.9-206.8). -13T allele of *SAA1* gene presented in 10.2% AA-positive vs 11.1% AA-negative RA patients. 84.4% AA-positive RA patients had concomitant *C. trachomatis* infection during the course of RA vs 16.9% AA-negative RA patients. *C. trachomatis* infection in RA patients associated with the higher incidence of renal amyloidosis ($R=0.93$; $p<0.0001$). OR for *C. trachomatis* infection was 26.6 (95%CI:9.36-76.4). To assess the predictive value of revealed factors for renal amyloidosis development in RA logit-regression analysis was performed. Probability of true positive and true negative prognosis for renal amyloidosis development using the drawn curve was 80% and 93.2% correspondingly. OR for two factors was 55.0 (95%CI:15.8-192.1).

Conclusions: *SAA1* gene α/α genotype variant and *C. trachomatis* infection are important prognostic factors for renal amyloidosis development in Belarusian RA patients.

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