Introduction: Available data suggest an association between presence of secondary (AA) amyloidosis and MCP-1 (monocyte chemoatracttant protein-1) and MIP-1alpha (macrophage inflammatory protein-1 alpha) genes polymorphisms. Some studies have also shown an impact of polymorphisms in exon 3 of SAA 1 (serum amyloid A 1) gene on the incidence of AA amyloidosis in different populations. Methods: The incidence of single genotypes MCP-1, MIP-1alpha and SAA 1 genes was investigated. Serum levels of SAA, MCP-1 and MIP-1alpha were measured and potential relation between serum levels and genotypes were analyzed. All examinations were performed in patients with AA amyloidosis (43), rheumatoid arthritis (RA) without amyloidosis and healthy control group (100). Results: Significantly more frequent occurrence of 1.1/1.1 genotype in SAA 1 was recorded in AA amyloidosis group compared to RA group as well as in control group ($p<0,001$). No statistically significant differences in distribution of another genotypes were found. Distribution of neither 1.1/1.1 genotype nor another ones did not vary among RA group and control group. No significant difference in distribution of another examined genotypes was recorded among all three groups. Serum concentrations of SAA were statistically significantly higher in AA amyloidosis group and also in RA group compared to healthy controls ($p<0,001$). The difference between AA and RA group was not significant. Serum concentration of MCP-1 was statistically significantly higher in AA amyloidosis group compared to RA group ($p<0,05$). Concentrations of MIP were markedly (but not statistically significantly) higher in both groups of patients compared to healthy controls. Conclusions: Homozygosity of the 1.1 haplotype in SAA1 gene could be a risk factor for development of AA amyloidosis in Caucasian population. Our findings of significantly higher serum concentrations of MCP-1 in the AA amyloidosis group compared to RA group could advert to riskiness of another factors. This could have therapeutic consequence earlier and more assertive therapy of underlying diseases in patients with appropriate genotype in order to prevent or interfere with occurrence of AA amyloidosis. Further studies in larger populations of patients are warranted.