INTRODUCTION

Acute poststreptococcal glomerulonephritis (APSGN) is the most common and most studied post-infectious renal disease in humans and frequently associated with autoimmune phenomena ***(Rodriguez-Iturbe et al., 2007).***

Auto antibodies against the first component of the classical pathway of complement (anti-C1q) can be found in a number of autoimmune, renal and infectious diseases. The prevalence of anti-C1q is highest in patients with hypocomplementemic urticarial vasculitis where anti-C1q can be used as a diagnostic marker ***(Wisnieski et al. 1995), (Prohaszka et al., 1999)***.

Anti-C1q has mostly been investigated in patients with systemic lupus erythromatosus (SLE). In adult patients with SLE, the prevalence of anti-C1q varied between 20% and 100% depending on the population of SLE patients studied. The highest prevalence of anti-C1q was found in those having active lupus nephritis (LN) ***(Marto et al., 2005).***

Increasing titers of anti-C1q seemed to precede renal flares by 2–6 months . In addition, after the successful treatment of a renal flare, anti-C1q mostly decreases or becomes undetectable, also Pathogenic role for anti-C1q in APSGN is similar to that proposed for adult SLE patients ***(Haseley et al.,1997)***, ***(Trendelenburg et al., 1999).***

Anti-C1q has a common secondary pathogenic role in SLE and APSGN. Both SLE nephritis, as well as APSGN, share several histopathological characteristics, such as glomerular sub endothelial deposition of IgG-containing immune complexes, deposition of complement compon-

ents of the classical pathway, mesangial proliferation, and local influx of neutrophils and monocytes/macrophages Furthermore, even an overlap syndrome between APSGN and SLE has been described ***(Itos et al., 2003).***

Hypocomplementemia, including the activation of the classical pathway, is a frequent finding in APSGN. Anti-C1q accounted for the long-lasting effects of classical pathway activation that had been observed in some patients ***(Wayat et al., 1988).***

In a mouse model of immune-complex glomerulonephritis, the injection of anti-C1q exacerbated a preexisting sub clinical disease whereas the injection of anti-C1q alone did not lead to significant glomerular inflammation ***(Trouw et al., 2004)***.