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High prevalence of *Chlamydophila pneumoniae* infection in patients with myeloperoxidase antineutrophil cytoplasmic autoantibody (MPO-ANCA)-associated glomerulonephritis

Sir,

Exposure to *Chlamydophila pneumoniae* (*Chlamydia pneumoniae*, CP) is very common in the general population [1]. CP infection has been proposed as a risk factor of atherosclerosis as a chronic vascular inflammation [2], though current clinical data do not warrant the use of antibiotics for prevention or treatment of cardiovascular diseases [3]. Interestingly, it was recently reported that CP infection might be associated with myeloperoxidase antineutrophil cytoplasmic autoantibody-associated glomerulonephritis (MPO-ANCA-associated GN) [4], which is attributable to systemic vasculitis and subsequently carries an increased risk for end-stage renal disease (ESRD) and death. The pathogenesis of MPO-ANCA-associated GN is still unclear, and the association with CP infection has not been closely investigated in clinical settings yet. In this study, we examined cross sectionally a prevalence of CP infection in patients with MPO-ANCA-associated GN, compared with those with immunoglobulin A nephropathy (IgAN).

Thirty-three case patients with MPO-ANCA-associated GN (mean age 70.2 ± 12.0 years, mean MPO-ANCA 321 ± 240 U/ml) and 40 control patients with IgAN, who were of similar age to the MPO-ANCA-associated GN group (mean age 69.5 ± 4.9 years), were investigated. The levels of anti-CP IgM-, IgA-, IgG-antibodies were measured as markers of active, chronic persistent active and past inactive CP infection, respectively. Multivariable logistic regression models were used to assess the association of CP infection with MPO-ANCA-associated GN, adjusting for age, sex and estimated glomerular filtration rate (eGFR).

As a result, MPO-ANCA-associated GN patients had a higher prevalence of CP infection in each phase than IgAN patients, though the difference was statistically significant only for active CP infection (Table 1). This result was consistent with the previous report [4]. Multivariable analyses suggested that active CP infection was significantly associated with MPO-ANCA-associated GN (OR = 9.79, $P = 0.001$), while insignificant were chronic persistent active infection (OR = 3.10, $P = 0.11$), and past inactive CP infection (OR = 2.93, $P = 0.11$).

Table 1. Prevalence of *Clamydophila pneumoniae*

	MPO-ANCA-associated GN ($n = 33$)	IgA nephropathy ($n = 40$)	p-value
Age (years)	70.2 ± 12.0	69.5 ± 4.9	0.76
Sex (male, %)	66.7	52.5	0.22
eGFR (ml/min)	19.4 ± 14.7	46.7 ± 24.0	<0.001*
Seropositivity			
CP IgM-antibody (%)	39.4	7.5	0.003*
CP IgA-antibody (%)	72.7	50.0	0.06
CP IgG-antibody (%)	66.7	45.0	0.07

*$P < 0.05$.

In a separate analysis, the difference in renal prognosis (progression to ESRD) between MPO-ANCA-associated GN patients with and without CP infection was also examined, but MPO-ANCA-associated GN patients with CP infection in each infection phase were not statistically different in the renal outcome from those patients without CP infection.

This study confirmed the high prevalence of active CP infection in patients with MPO-ANCA-associated GN, and active CP infection could potentially enhance the risk of MPO-ANCA-associated GN. However, the difference in renal prognosis between MPO-ANCA-associated GN patients with and without CP infection was not identified in the present study maybe due to the small sample size. Further analyses are required to examine closely whether CP infection is involved in the pathogenesis of MPO-ANCA-associated GN, and the evidence linking CP infection might lead to the benefit from antibiotic treatment to prevent or cure MPO-ANCA-associated GN by eradicating CP as a causative agent.

Conflict of interest statement. None declared.

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