investigate the relative association between *C. pneumoniae* infection and the development of MPO-ANCA-associated GN, we compared the titres of *C. pneumoniae* abs in the active and remission phase of the disease in the same patients. The titres of anti-*C. pneumoniae* IgM and IgA, but not IgG abs in ANCA (remission) group significantly decreased compared with that in ANCA (active) (IgM ab: 1.22±0.47 vs 1.05±0.49 U, *P*<0.05; IgA ab: 1.70±1.20 vs 1.29±0.88 U, *P*<0.05, ANCA (active) vs ANCA (remission)). Our study revealed that the presence of positive IgM ab against *C. pneumoniae* was closely associated with the development of MPO-ANCA-associated GN, while the presence of positive IgG- and IgA ab against *C. pneumoniae* as a risk factor did not reach statistical significance. In addition, our study confirmed that the increased titres of IgM- and IgA ab against *C. pneumoniae* in the ANCA (active) group was significantly reduced in the ANCA (active) group. The serological pattern of increased IgM and IgA titres has been suggested to indicate active infection and chronic persistence of active infection, respectively. IgG titres in the absence of IgM or IgA titres may be a serological marker of an older, inactive infection. Thus, active or chronically active rather than inactive *C. pneumoniae* infection may increase the risk of MPO-ANCA-associated GN. Based on the results, we propose a possibility that *C. pneumoniae* influences the pathogenesis of MPO-ANCA-associated GN.

Our experiment indicates for the first time, a significantly higher prevalence of active *C. pneumoniae* infection in patients with MPO-ANCA-associated GN. The hypothesis that *C. pneumoniae* is aetiologically involved in MPO-ANCA-associated GN is of particular therapeutic relevance, because this is a potentially eradicable infectious agent. However, the number of patients in our study was small, a large-scale prospective confirmation of these findings is thus required.

**Table 1. Results of Chlamydial analysis in patients and control subjects**

| Antibody to C. pneumoniae | Patients | ANCA (active) vs ANCA (remission) | ANCA (active) vs control subjects | OR (95% CI) | *P* | OR (95% CI) | *P* | OR (95% CI) | *P*
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<tr>
<td></td>
<td>[n=15 (%)]</td>
<td>[n=10 (%)]</td>
<td>[n=50 (%)]</td>
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<tr>
<td>IgG seropositivity</td>
<td>6 (40.0)</td>
<td>3 (30.0)</td>
<td>28 (56.0)</td>
<td>0.52</td>
<td>0.28</td>
<td>1.56</td>
<td>0.69</td>
<td>3.50</td>
<td>0.22</td>
</tr>
<tr>
<td>IgA seropositivity</td>
<td>11 (73.3)</td>
<td>6 (60.0)</td>
<td>23 (46.0)</td>
<td>3.23</td>
<td>0.08</td>
<td>1.83</td>
<td>0.67</td>
<td>1.76</td>
<td>0.50</td>
</tr>
<tr>
<td>IgM seropositivity</td>
<td>9 (60.0)</td>
<td>3 (30.0)</td>
<td>13 (26.0)</td>
<td>4.27</td>
<td>0.01</td>
<td>3.00</td>
<td>0.17</td>
<td>1.22</td>
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Data are expressed as *n (%)*. 

Conflict of interest statement. None declared.

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**Primary biliary cirrhosis associated with minimal change disease**

Sir,

Primary biliary cirrhosis (PBC), which is probably of autoimmune origin, is a chronic inflammatory disease of the intrahepatic biliary system. However, its pathogenesis is still unclear. PBC has been reported to be associated with certain autoimmune diseases, such as systemic lupus erythematosus, rheumatoid arthritis and Sjögren’s syndrome [1]. The association of glomerular diseases with PBC has been rare. Rai *et al.* [2] first noted the association of PBC with diffuse membranous glomerulonephritis. There have been six PBC cases reported with nephrotic syndrome, in which the patients had membranous glomerulonephritis, with or without vasculitis [3]. We present a case of minimal change disease (MCD) in a PBC patient, in which the renal pathology diagnosis has not been previously reported.

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A 36-year-old woman was admitted to our hospital for evaluation of nephrotic syndrome. The patient had a right intrahepatic stone and had had abnormal liver function for 3 years. Alkaline phosphatase and \( \gamma \)-glutamyltransferase (\( \gamma \)-GT) levels were elevated significantly for 1 year prior to this admission. The titre of antimitochondrial antibodies was positive at 20:1. PBC was suspected. One month before admission she noticed progressive leg swelling, along with weight gain and foamy urine. On admission, the patient's blood pressure was 120/86 mmHg and pulse rate 92 beats/min. Physical examination was unremarkable, except for marked pitting oedema of both legs. Laboratory data showed heavy proteinuria (24 h urinary protein 6.23 g), hypoalbuminaemia (2.0 g/dl), and hypercholesterolaemia (579 mg/dl). The haemogram showed a haematocrit value of 36.9% and her renal function tests were within the normal range. The serum level of IgG was decreased (502 mg/dl) and IgM was elevated (581 mg/dl) with a normal serum IgA (230 mg/dl) and complement levels. The serum levels of both alkaline phosphatase (601 U/l) and \( \gamma \)-GT (488 U/l) were elevated with negative hepatitis serology tests. The anti-mitochondrial antibody was positive. An ultrasound scan also showed normal-sized kidneys.

A liver biopsy was consistent with PBC in an early stage. Kidney biopsy showed no increase in mesangial cellularity and matrix in 11 glomeruli without any tubular atrophy or interstitial fibrosis. Periodic acid-Schiff (PAS) and chromotrope-2R silver methenamine (CSM) stains revealed no positive staining. Immunofluorescent study also disclosed no deposition of immunoglobulins. Electron microscopy revealed focal effacement of podocyte, compatible with minimal change disease (Figure 1).

The patient was treated with prednisolone 60 mg/day for 1 month, and her nephrotic syndrome markedly remitted. However, 5 months later, proteinuria relapsed after the steroid was tapered to prednisolone 10 mg/day and then subsided again in response to a larger dose steroid administration (Figure 2). Sixteen and 35 months later, similar episodes of proteinuria relapse occurred while the steroids were discontinued. Renal and liver functions remained stable and the patient remained without symptoms.

MCD is a clinical and pathological entity defined by selective proteinuria and hypoalbuminaemia that occurs in the absence of cellular glomerular infiltrates or immunoglobulin deposits. It is assumed that MCD is caused by an aberrant immune response [4]. PBC is considered to be of autoimmune origin, but the pathogenesis remains unclear. The classical histopathological lesion of PBC is accompanied by a T-cell-rich mononuclear cell infiltrate and up-regulation of T-cell surface markers, which suggests T-cell activation and cytokine release [5]. The coexistence of PBC and MCD, as in our case, is probably due to the same cause—induction of cell-mediated immunity. Although more case reports are still needed to explore the exact mechanism, our case supports the hypothesis that a cell-mediated immunity disorder might play a role in the association between PBC and nephropathy.

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