Binocular vision in patients undergoing haemodialysis

Sir,

Many patients on chronic haemodialysis (HD) suffer from chronic eye diseases, and the interaction of these diseases with the dialysis procedure can lead to worsening of vision [1]. Different studies have emphasized the effect of HD on visual acuity (VA), ocular surface disorders and tear function changes, fundus changes, VEP parameters, visual field findings and contrast sensitivity [2–5].

The purpose of this study was to investigate the relation between binocular vision disorders in patients with chronic renal failure undergoing HD. In this prospective study, 51 patients (32 males and 19 females, mean age 55.73 ± 11.22) with chronic renal failure underwent orthoptic evaluation, just before and half an hour after the treatment.

Orthoptic assessment included Jaeger VA, cover test (CT), ocular motility, convergence, binocular correspondence (Maddox wing and Worth test) and stereo tests (Lang I and Titmus). χ² test did not confirm any significant difference in CT and motility before and after the HD (P > 0.05). Student’s t-test confirmed a significant difference in near VA and convergence, measured before and after treatment (P < 0.001).

Maddox wing, Worth and TNO tests showed significantly worse results after HD compared with results gained before HD (Wilkinson test, P < 0.001). In Lang I test, there was no significant difference in recognizing before and after the treatment (P > 0.05).

According to our knowledge, there is no previous report on changes in binocular vision induced by HD in chronic renal patients. Rapid shifts of body fluid and disturbances of glycaemic control could affect changes of monocular and binocular VA. The pathophysiology of visual system disarrangement could be metabolic in origin, such as uremia, hyponatraemia or hypocalcaemia in so far as they influence the neurological status.

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References


Chlamydia pneumoniae infection and MPO-ANCA-associated glomerulonephritis

Sir,

The pathogenesis of myeloperoxidase antineutrophil cytoplasmic autoantibody-associated glomerulonephritis (MPO-ANCA-associated GN) is still unknown. Various exogenous factors, such as silica exposure or propylthiouracil, have been suggested to be associated with MPO-ANCA-associated vasculitis [1,2], but little is known about the role of microbial agents. Chlamydia pneumoniae, a cause of respiratory tract infection [3], was recognized as the third Chlamydia species in 1986 [4]. In addition to respiratory diseases, the organism has been linked with atherosclerosis and related clinical manifestations such as coronary heart disease, carotid artery stenosis, aortic aneurysm, claudication and stroke [5,6]. Recent evidence suggests C. pneumoniae organisms can survive and multiply within, not only macrophage, but also polymorphonuclear neutrophils (PMN) [7]. There is a possibility that persistent infection of C. pneumoniae within PMN may lead to autoimmunity, and plays a role in enhancement of the process of ANCA production. In addition, C. pneumoniae infected macrophages adhere to the endothelium and migrate to the subendothelium in atherosclerotic lesion. These processes result in the release of cytokines and growth factor synthesis, which up-regulate endothelial cell adhesion molecules, leading to increased leucocyte adhesion [8]. These components, including macrophage infiltration, cytokine release, up-regulation of adhesion molecules and leucocyte adhesion, are indispensable to the pathogenesis of MPO-ANCA-associated GN [9]. Furthermore, C. pneumoniae produces chlamydial heat shock protein (CHSP) in infected cell macrophages and elicits a hypersensitivity reaction of the host, resulting in severe endothelial injury [10]. It is likely that the focal inflammatory reaction would be strongly enhanced resulting in necrotizing vasculitis in the presence of circulating ANCA, the production of which may be the result of interactions between T cells and B cells activated by microbial superantigens. Thus, we were interested in whether there is a high prevalence of active C. pneumoniae infection in patients with MPO-ANCA-associated GN.

We examined the level of anti-C. pneumoniae IgG-, IgA- and IgM-antibodies (abs), as a marker of an inactive, chronic persistence of active, and active infection, respectively, using ELISA in 15 patients with active idiopathic MPO-ANCA-associated GN (ANCA (active)) (mean ± SD age, 64.80 ± 12.64 years) and 50 controls (65.80 ± 13.45 years). We also studied paired sera from 10 patients in ANCA (active) who were in the remission phase of the disease [ANCA (remission)] (71.30 ± 9.18 years). There was no significant difference in the three groups with respect to seropositivity to anti-C. pneumoniae IgG and IgA abs; whereas seropositivity to anti-C. pneumoniae IgM ab, which was measured in 60% of the patients in ANCA (active), 30% of the patients in ANCA (remission) and 26% of controls, was associated with the risk of active MPO-ANCA-associated GN (P = 0.01) (Table 1). To comprehensively


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

<table>
<thead>
<tr>
<th>Antibody to C. pneumoniae</th>
<th>Patients</th>
<th>ANCA (active)</th>
<th>ANCA (remission)</th>
<th>Control subjects</th>
<th>ANCA (active) vs ANCA (remission) OR (95% CI)</th>
<th>P</th>
<th>ANCA (active) vs ANCA (remission) OR (95% CI)</th>
<th>P</th>
<th>ANCA (remission) vs control subjects OR (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgG seropositivity</td>
<td>6 (40.0)</td>
<td>28 (56.0)</td>
<td></td>
<td></td>
<td>0.52 (0.16-1.69)</td>
<td>0.28</td>
<td>1.56 (0.28-8.53)</td>
<td>0.69</td>
<td>0.34 (0.08-1.45)</td>
<td>0.17</td>
</tr>
<tr>
<td>IgA seropositivity</td>
<td>11 (73.3)</td>
<td>23 (46.0)</td>
<td></td>
<td></td>
<td>3.23 (0.90-11.52)</td>
<td>0.08</td>
<td>1.83 (0.33-10.10)</td>
<td>0.67</td>
<td>1.76 (0.44-7.01)</td>
<td>0.50</td>
</tr>
<tr>
<td>IgM seropositivity</td>
<td>9 (60.0)</td>
<td>13 (26.0)</td>
<td></td>
<td></td>
<td>4.27 (1.27-14.33)</td>
<td>0.01</td>
<td>3.50 (0.64-19.20)</td>
<td>0.22</td>
<td>1.22 (0.27-5.43)</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Data are expressed as n (%).

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.

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.