Correspondence

IgG monoclonal gammopathy in four patients with polymyalgia rheumatica

Sir. The aetiology and pathogenesis of giant cell arteritis (GCA) and polymyalgia rheumatica (PMR) are unknown. The cellular inflammation and immunoglobulin deposition in temporal artery biopsy specimens, and the dramatic clinical response to steroids suggest that an immunological abnormality might be associated with the pathogenesis of GCA or PMR. Two patients with serum monoclonal IgM and PMR have been previously reported. In the present study four patients are reported with benign monoclonal gammopathy (BMG) and PMR (with or without GCA); three of the patients had serum IgG which reacted with mouse femoral artery, raising the possibility that the IgG abnormalities may be associated with their clinical disease.

All four patients had bilateral shoulder and hip arthralgias, and two also had temporal headaches. All four patients had a Westergren erythrocyte sedimentation rate (ESR) greater than 50 mm/h. The four patients were treated with prednisone 15 mg daily for PMR and 60 mg daily followed by tapering for GCA, which dramatically reduced their arthralgias and ESR within one week. Sera from these four patients were obtained when they were asymptomatic 1, 6, 7, or 13 months after starting prednisone. Sera were studied from six controls: four with IgG BMG, one with GCA, and one with PMR; the last two controls were treated with prednisone and were asymptomatic.

Fresh specimens of mouse stomach and mouse femoral artery were embedded in Tissue Tek-2 (Miles) and quickly frozen in Cryokwik compound. Sections 4 μm thick were cut, incubated with sera from patients or controls for 30 minutes, washed, incubated with fluorescein isothiocyanate-labelled goat antihuman polyclonal or γ antisera (Tago), α or μ antisera (Hyland), or κ or λ (Meloy) antisera for 30 minutes, washed, and then examined with a Zeiss fluorescent microscope. Staining of mouse femoral artery was detected in three of the four patients’ sera but in none of the six controls’ sera (Table 1). In each case staining by the heavy or light chain corresponded to the heavy or light chain of the respective BMG.

Table 1 Reactivity of patients’ and controls’ sera with mouse femoral artery

<table>
<thead>
<tr>
<th>Age and sex</th>
<th>Four patients</th>
<th>Six controls</th>
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<tbody>
<tr>
<td>Biopsy of TA*</td>
<td>67, m</td>
<td>67, f</td>
</tr>
<tr>
<td>Rheumatic disease</td>
<td>PMR</td>
<td>GCA</td>
</tr>
<tr>
<td>Monoclonal Ig</td>
<td>IgG-λ</td>
<td>IgG-λ</td>
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<tr>
<td>Staining of mouse artery</td>
<td>γ 1:10</td>
<td>γ 1:10</td>
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</tbody>
</table>

Sections of mouse femoral artery were incubated with sera from four patients with IgG BMG and PMR (with or without GCA) and sera from four controls with IgG BMG, one control with GCA, and one control with PMR, washed, incubated with fluorescein isothiocyanate-labelled goat antihuman γ, α, μ, κ, or λ antisera, and examined by fluorescent microscopy.

References


IgG monoclonal gammopathy in four patients with polymyalgia rheumatica.
D Ilfeld, J Barzilay, D Vana, M Ben-Bassat, H Joshua and I Pick

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