LETTERS TO THE EDITOR

From wheels to feet: a dramatic response of severe chronic psoriatic arthritis to etanercept

A 47 year old man presented with seven years of severe psoriatic polyarticular arthritis of progressive, symmetrical, and additive course, affecting the shoulders, elbows, wrists, metacarpophalangeal joints, proximal interphalangeal joints, distal interphalangeal joints, hips, knees, and feet. Also, his skin and nails were completely affected with severe erythroderma. The patient was treated with methotrexate, chrysoterythra, antimalarial drugs, salazopyrine, azathioprine, cyclosporin, minocycline, and intra-articular and systemic corticosteroids. All these treatments consecutively and in combinations failed or were stopped owing to side effects. During 1999 his condition deteriorated further with morning stiffness of more than six hours, active synovitis of more than 20 joints, and systemic symptoms, such as extreme fatigue and weight loss of more than 10 kg. The patient became confined to a wheelchair. His blood tests disclosed increased erythrocyte sedimentation rate (ESR) and C reactive protein (CRP) levels, and normocytic normochromic anaemia (table 1).

In July 1999 etanercept (25 mg) subcutaneously weekly was started. A week later the patient reported a dramatic improvement in his general wellbeing, morning stiffness, and joint pain accompanied by a significant decrease in the number of swollen and tender joints and the erythematous component of the skin lesions. His clinical improvement continued during the following six weeks. He regained 6 kg weight; the ESR improvement continued during the following six weeks. He regained 6 kg weight; the ESR decreased to 11 mm/1st h and the haemoglobin concentration increased to 120 g/l (table 1).

Etanercept, a recombinant human tumour necrosis factor (TNF) receptor Fc fusion protein (Enbrel, Immunex Corp, Seattle, Washington) is a dimer consisting of the Fc portion of human IgG1 fused to the Fc portion of human IgG1. 1

Previous authors have suggested that TNFα plays an important part in the pathogenesis of psoriatic skin lesions and psoriatic arthritis. 2

Table 1 Clinical and laboratory variables immediately before (week 0) and after (weeks 1–8) etanercept treatment

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Week 0</th>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 4</th>
<th>Week 6</th>
<th>Week 8</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of swollen joints</td>
<td>22</td>
<td>11</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>No of tender joints</td>
<td>26</td>
<td>16</td>
<td>9</td>
<td>6</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Grip strength (mm Hg)</td>
<td>43</td>
<td>80</td>
<td>97</td>
<td>88</td>
<td>95</td>
<td>110</td>
</tr>
<tr>
<td>Right</td>
<td>60</td>
<td>86</td>
<td>78</td>
<td>106</td>
<td>110</td>
<td></td>
</tr>
<tr>
<td>ESR (mm/1st h)</td>
<td>85</td>
<td>45</td>
<td>25</td>
<td>15</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>CRP (g/l)</td>
<td>0.18</td>
<td>ND*</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>0.01</td>
</tr>
<tr>
<td>Haemoglobin (g/l)</td>
<td>101</td>
<td>121</td>
<td>112</td>
<td>131</td>
<td>131</td>
<td>134</td>
</tr>
</tbody>
</table>

*ESR = erythrocyte sedimentation rate; CRP = C reactive protein (normal range 0–0.01 g/l); ND = not determined.

The dramatic effect of etanercept in the present resistant and extremely severe case of psoriatic arthritis suggests a possible role for this drug also in the treatment of severe psoriatic arthritis.

ORI ELKAYAM
MICHAEL YARON
DAN CARPI
Department of Rheumatology,
Tel Aviv “Sourasky” Medical Centre and “Sackler” Faculty of Medicine,
University of Tel Aviv,
Israel

Correspondence to: Dr Ori Elkayam, Department of Rheumatology, Tel Aviv Medical Centre, 6 Weizman street, Tel Aviv 64239, Israel
Email: unitel87@netvision.net.il

follow up the patient remains free of clinical symptoms taking prednisone 9 mg/day. In December 1999 liver tests were normal. PM/DM are systemic inflammatory disorders affecting the skeletal muscles and other organs, notably the gastrointestinal tract. The prevalence of oesophageal impairment in patients with PM/DM has therefore been reported to be as high as 60%.2 Involvement of the liver is less recognised, and is considered to be rare in PM/DM where it is usually due to immunosuppressive treatment. To date, only a few investigators have described liver manifestations in patients with PM/DM; liver impairment in these patients was related to associated conditions—that is, primary biliary cirrhosis3 and HCV infection.4–6

We report, to our knowledge, the first case of a patient with PM associated with autoimmune hepatitis and HCV infection. In this instance, the diagnosis of autoimmune hepatitis could be made, because the patient fulfilled all the criteria for autoimmune hepatitis—namely, (a) increased ALT and AST, with alkaline phosphatase less than threefold the normal value; (b) total gammaglobulin or IgG levels >1.5 times the upper limit of normal; (c) ANA titres >1/80; and (d) characteristic histological liver damage. Our findings raise the question of an association between PM and autoimmune hepatitis. PM/DM and may be due to immunity dysfunction. From a practical point of view, these data indicate that the diagnosis of autoimmune hepatitis may be suspected in patients with PM/DM presenting with cytolytic hepatitis. However, autoimmune hepatitis might also be related to HCV infection in our patient. As previous authors have recently shown that the prevalence of HCV infection was similar in patients with autoimmune hepatitis and the general population, they concluded that HCV could be excluded as a cause of autoimmune hepatitis.7 Further prospective trials are required to evaluate the prevalence of autoimmune hepatitis in patients with PM/DM, with and without HCV infection. Finally, our unusual case is reminiscent of one reported by Ferri et al1, who described a patient with cranial mononeuritis associated with PM and HCV infection. In our experience the diagnosis of PM related left V nerve palsy could be made as both neurological and muscle manifestations completely resolved concomitantly with steroid treatment.

I MARIE
H LEVESQUE
H COURTOIS
Department of Internal Medicine,
Centre Hospitalier Universitaire de
Rouen-Boisguillaume,
7631 Rouen Cedex, France
A FRANCOIS
Department of Pathology and Cytology
G RIACHI
Department of Gastroenterology

Correspondence to: Dr Isabelle Marie


www.annrheumdis.com

Figure 1  Course of the patient’s polymyositis clinical and biochemical manifestations and virological status with steroids.