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: unit87@netvision.net.il


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Autoimmune disorders are common in hepatitis C virus (HCV) infection, and may be due to dysfunction of both cell and humoral immunity.1 HCV infection has therefore been associated with essential cryoglobulinaemia, polyarteritis nodosa, leucocytoclastic vasculitis, glomerulonephritis, and idiopathic pulmonary fibrosis.2,3 Polymyositis (PM) and dermatomyositis (DM) have more rarely been reported during the course of HCV infection.4,5 Previous authors have suggested that HCV infection might be responsible for the formation of autoantibodies and circulating autoantibodies, leading to autoimmune complexes, resulting in PM/DM manifestations.6,7 We observed a new case, which is of particular interest, as the patient with PM and cranial neuropathy developed chronic active autoimmune hepatitis and HCV infection. A 48 year old woman presented in March 1997 with a five month history of generalised muscle weakness and myalgia associated with dysphagia. She had no previous medical history. Examination showed muscle weakness affecting both arms and legs. Muscle power was gauged for eight proximal muscles (that is, neck flexors, trapezius, deltoid, biceps, psoas, maximus and medius gluteus, and quadriceps) by a modification of the British Medical Research Council grading system, resulting in scores ranging from 0 to 11 (theoretical maximum score 88 points). Muscle power of the patient was 65 points. Physical examination also showed dysaesthesias in the area of the left V nerve.

The following laboratory findings were abnormal: erythrocyte sedimentation rate 60 mm/1st h, C reactive protein 70 mg/l (normal <5), creatinine 600 μM/l (normal 5–130), and aldolase 11.3 U/l (normal 0.5–3.1). Liver tests showed alanine aminotransferase (ALT) 145 U/l (normal 3–21), aspartate aminotransferase (AST) 98 U/l (normal 3–26), γ-glutamyltransferase 210 U/l (normal 5–25), alkaline phosphatase 96 U/l (normal 73–207), and total bilirubin 6 U/l (normal 2–18). Blood electrophoresis yielded total protein 70 g/l, albumin 37 g/l, polyclonal gammopathy of increased IgG at 19 g/l (normal 6–12). An electromyogram showed both low amplitude and polyphasic motor unit potentials affecting muscles of the arms and legs.

Muscle biopsy of the left rectus femoris confirmed the diagnosis of PM; microscopic analysis showed infiltratory inflammatory associates associated with disruption of the muscle architecture and muscle fibre necrosis. Facial electromyogram showed sensorimotor impairment affecting both first and second branches of the left trigeminal nerve. Autoantibody screening was positive for antinuclear antibodies (ANA) >1000; other tests—that is, anti-Jo1 antibody, rheumatoid factors, antineutrophil cytoplasmic antibodies, and cryoglobulin were negative.

Blood cultures, bacterial (Mycoplasma pneumoniae, Ochlolela) and parasitic (Toxoplasma gondii) serologies were negative. Viral serologies (cytomegalovirus, Epstein-Barr virus, human immunodeficiency virus, coxsackie, parvovirus B19, hepatitis B) were negative, whereas hepatitis C serology was positive. Oesophageal manometry showed a low pressure in the upper oesophageal sphincter and decreased peristalsis within the upper third of the oesophageal body. Pulmonary function tests showed obstruction of the monoxide transfer factor (TLco) 63% of predicted. Other investigations, including thoracic computed tomography, echocardiography, and abdominal ultrasound, were within normal limits. Because of increased aminotransferases (AST and ALT) and positive HCV serology, polymerase chain reaction amplification of HCV RNA was performed on the patient’s serum, which was positive. Liver biopsy specimens showed characteristic damage of chronic active autoimmune hepatitis—that is, interface hepatitis. Autoantibody screening was negative for antimitochondrial, anti-smooth muscle, and anti-LKM1 antibodies.

Steroid treatment, at an initial dose of 1 mg/kg/day, was started. Because of both PM and chronic active autoimmune hepatitis, treatment of hepatitis C with interferon α was not instituted. Both muscular and neurologiccal (that is, dysfunction of the left V nerve) manifestations related to PM improved progressively and disappeared completely in June 1997 (fig 1). Abnormalities of liver tests improved concomitantly (fig 1). At two year...
HCV RNA + + + + + +
Muscular manifestation + – – – – –
Oesophageal manifestation + – – – – –
Cranial neuropathy + – – – – –

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

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 as part of a continuum, suggesting that autoimmune hepatitis should be included within the spectrum of liver manifestations of 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. 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 al., 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-Beauvechain,
76031 Rouen Cedex, France
A FRANCOIS
Department of Pathology and Cytology
G RIACHI
Department of Gastroenterology

Correspondence to: Dr Isabelle Marie