Dear Sir,

The association of hepatitis C infection and peripheral neuropathy has been described with and without cryoglobulinemia [1]. Autoimmune hepatitis (AIH) is a chronic autoimmune disease of the liver with unexplained etiology, potentially progressing to hepatic failure [2]. So far, AIH associated with neuropathy has been described in a few individual patients [3]. Here we describe a patient suffering from AIH who developed a motor-axonal polyneuropathy.

In 2004, a 61-year-old female Caucasian patient presented with an 8-kg weight loss during the preceding 3 months, fatigue, abdominal distension, yellow sclerae, jaundice, pale stool, and dark urine. Physical examination revealed an enlarged liver and laboratory values were total bilirubin 10.6 mg/dl (normal 0.2–1.3), alkaline phosphatase 172 U/l (35–104), glutamic-oxaloacetic transaminase (GOT) 1,134 U/l (<35), glutamic-pyruvic transaminase (GPT) 915 U/l (<35), γ-glutamyl transpeptidase 340 U/l (<39), smooth muscle antibodies 1:1,280 and hypergammaglobulinemia IgG 3,332 mg/dl (700–1,600). Other AIH-associated antibodies including anti-liver-kidney microsomal antibody, antibodies against soluble liver antigen, antinuclear antibody, antimitochondrial antibody and rheumatoid factor were not found. Biochemical screening for viral hepatitis did not reveal antibodies against any type of viral hepatitis. Immunofixation was normal and cryoglobulins were not detected. Histological evaluation of liver biopsy demonstrated macronodular cirrhosis, portal tract fibrosis and proliferating biliary tracts with infiltrates of mainly T lymphocytes and few plasma cells. As an associated autoimmune disease, Hashimoto’s thyroiditis with slightly elevated thyroid-stimulating hormone 3.9 mU/l (0.2–3.8) and thyroid autoantibodies 8 U/l (0–5) were found and a therapy with 50 μg levothyroxine was initiated.

In our patient the diagnosis of AIH was made according to suggested parameters [4]. AIH is known to respond to steroid therapy with clinical and biochemical improvement to normal liver parameters. After initiation of a corticosteroid therapy the patient recovered and all liver laboratory parameters remained within normal limits. A 6-mg budesonide therapy was suggested but incompletely continued. However, for 2 years the controls showed GOT and GPT elevated up to 96 U/l (<35) and 100 U/l (<35), respectively, but all other laboratory parameters remained within normal limits.

Pathogenesis of neuropathy in AIH is not known but may stem from deposits of immune complexes in vasa nervorum which then cause vasculitis and ischemia of the nervous fibers as was shown with cryoglobulins [5]. In this patient we confirm that disease activity of AIH is not associated with activity of peripheral neu-
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A prednisolone therapy with 1 mg/kg/day was initiated and improved symptoms. When the dosage was gradually tapered, symptoms of polyneuropathy reappeared. In addition to 5 mg/day prednisolone, a therapy with cyclophosphamide 100 mg/day was initiated and the patient was free of symptoms within 1 week.

Further studies including descriptions of individual patients are required to evaluate the prevalence of AIH with polyneuropathy and to increase knowledge on possible and successful therapies.

References