LETTERS TO THE EDITOR

Chronic inflammatory demyelinating polyneuropathy during treatment with interferon-α

Interferon-α (IFN-α) is widely used for the treatment of chronic viral hepatitis. There have been some reports concerning the development of autoimmune diseases, particularly thyroid disease, in patients under treatment with IFN. Disorders including autoimmune haemolytic anaemia, pernicious anaemia, thrombocytopenic purpura, systemic lupus erythematosus, Raynaud’s disease, parotiditis, and epididymitis have been reported. Some neurological problems have also been described; although most such adverse events have involved the CNS, several cases of peripheral nervous system involvement have been reported—namely, axonal polyneuropathy, neuralgic amyotrophy, multiple mononeuropathies, and myasthenia gravis. On the other hand, some authors have reported that IFN-α may be an effective alternative therapy in patients with chronic inflammatory demyelinating polyneuropathy (CIDP) who are refractory to conventional treatments. Two trials using IFN-α and IFN-β on patients with CIDP are currently in progress. We describe one patient who developed CIDP during IFN-α treatment.

A 29-year-old man who had hepatitis C for 2 years, was started on IFN-α treatment. He had the usual related flu-like syndrome during the first month of treatment. Previously he had had some migraine headache attacks. The patient was given prednisone (60 mg/day) and progressively improved. One year later he had no symptoms and showed areflexia only on neurological examination. A further EMG showed appreciable improvement.

This is the first report of CIDP development during treatment with IFN-α. CIDP is an immune-mediated disorder that usually responds to plasma exchange, intravenous gammaglobulin, or corticosteroids, although occasionally the disease is refractory to these therapies. In the past, some authors have reported improvement in patients with CIDP who were receiving IFN-α. The mechanism by which IFN induced improvement in these patients is uncertain, although it may be related to complex immunomodulating effects, possibly by reduction of proinflammatory cytokine concentrations (tumour necrosis factor and IFN-γ) which may have a role in the development of autoimmune demyelination. The relation between IFN-α and CIDP in our patient is uncertain. Whether IFN-α was the cause of CIDP or whether their relation was only coincidental remains unknown. Nevertheless it seems clear that the treatment mentioned above did not prevent the development of this demyelinating disease with an immunological basis. IFN-α exerts complex immunomodulator effects, it can improve or worsen autoimmune diseases.

Although our findings could be coincidental, the data suggest caution, as IFN-α treatment might have undesirable effects involving autoimmune phenomena.

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Nerve conduction studies

The table below shows the results of nerve conduction studies on the patient during and after IFN-α treatment. The protein concentration in the serum was 208 mg/dl, there were no cells. Immunoelectrophoresis was normal, and antigastriloide antibodies (GMI, GD1a, GD1b, GT1b) were absent. Serum biochemical studies, including HIV antibody determination, were negative. We ruled out the presence of cryoglobulins. Although IFN-α was discontinued, the disease continued to worsen; the maximal neurological deficit was reached 5 months from onset. The patient was given prednisone (60 mg/day) and progressively improved. One year later he had no symptoms and showed areflexia only on neurological examination. A further EMG showed appreciable improvement.

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Posteroventral pallidotomy can ameliorate attacks of paroxysmal dystonia induced by exercise

Paroxysmal exercise induced dystonia is a rare disorder classified as one of the paroxysmal dyskinesias. In this condition patients develop dystonia, mostly involving their feet, after prolonged exercise, usually walking or swimming. Treatment response is poor to both antispasmodic drugs and to physiotherapy. In our patient was free for 3–4 years. Four years ago the attacks returned and got progressively worse, increasing in frequency and intensity. Over the past 2 years she could have an attack on walking even 10–15 steps. The attacks in the past few years not only made her right foot to toe in turn as before but caused her to fall as the right leg would rise up in the air and flex at the knee and hip and there would be some involvement of the trunk causing her spine to twist to the left. Recently the toes of the left foot were also noted to contract during attacks. She would never lose consciousness and the attacks would last 1–2 minutes and then subside. They never occurred in sleep. Internally the neurological examination was normal although postureing of the right foot could be induced by repeated prolonged passive flexion-extension movements of the right ankle. More recently she also began to have occasional spontaneous attacks. Investigations including repeated MRI of the head and spine were normal as were tests for Wilson’s disease and other causes of secondary dystonia. Examination of CSF gave normal results and disclosed no oligoclonal bands. The patient was negative for the common mitochondrial mutations. An EMG/nerve conduction study detected no evidence of a peripheral neuropathy and somatosensory evoked potentials were normal. Polygraphy confirmed cocontraction of agonists and antagonist muscle pairs in the right leg during an attack supporting an organic basis for the dystonia. Surface EEG during an attack showed slow spindles, without discharges. Different antispasmodic drugs were tried individually or in combination (1g sodium...