

A rare complication of interferon treatment: optic neuropathy

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Alpha-interferon (alpha-IFN) is widely used in the treatment of chronic viral hepatitis, either alone or combined with other antiviral therapies such as ribavirin [1]. It can induce various side effects, particularly systemic effects such as fever, influenza-like symptoms, thrombocytopenia and neutropenia [2]. Ocular toxicity, including retinopathy, optic neuropathy and ocular vision loss, has been infrequently (<1%) reported as a potentially serious adverse event associated with alpha-IFN. Anterior ischemic optic neuropathy (AION) is presumed to be a small infarct within the optic nerve head. Many patients have arteriosclerotic risk factors [3]. We report a patient in whom bilateral AION occurred shortly after the onset of treatment with alpha-IFN for chronic hepatitis C (CHC).

A 28-year-old man has been followed up with chronic renal failure and CHC infection probably as a result of hemodialysis treatment. Pegylated-IFN- α 135 μ g/week was started. Two months after treatment, sudden onset, painless, left visual field defect happened occurred. Visual acuity was 20/30 in the right eye and 20/80 in the left eye. Fundus examination of the red eye revealed pallid edema of the optic nerve head with cotton wool spots. In the left eye disc, there was optic disc edema, more elevated than in the right, with splinter hemorrhages on the nasal side, and a few cotton wool spots at the edge of the edema. Visual field examination demonstrated defects suggestive of inferior nerve fiber bundle defects in both eyes, worse in the OS. Neurologic assessment was otherwise normal. Standard blood study results were normal, including erythrocyte sedimentation rate, white blood cell count, hemoglobin, prothrombin time, partial thromboplastin time, protein S, protein C, antithrombin III, antiphospholipid antibodies, antinuclear antibody, venereal disease research laboratory titer. Thyroid-stimulating hormone was on the lower edge of normal values, other thyroid function test results were normal. Because our patient was did not have

systemic symptoms of vasculitis and cryoglobulin antibodies were negative, we ruled out essential mixed cryoglobulinemia. Cranial and orbital computed tomography findings were normal. Pegylated-IFN- α was discontinued. The patient was treated with orally with methylprednisolone 32 mg/d for 14 days, with tapering off the next two months. After this treatment visual disturbances resolved and the parameters of neurophysiologic testing returned to normal.

In summary, we describe a patient undergoing treatment with pegylated-IFN- α who developed optic neuropathy two months from the onset of this medication. The patient had established risk factors for conventional AION, including hypertension and chronic renal failure and so it is doubtful whether the HCV infection precipitated the disease. We conclude that clinically evident optic tract neuropathy may complicate pegylated-IFN- α administration. Candidates for pegylated-IFN- α treatment may need routine examination of optic fields and visual evoked potentials, before and during the administration of the drug to avoid possibly permanent visual sequelae.

References

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