Optic Neuritis following Treatment with Isoniazid in a Hemodialyzed Patient

M.A.	Miguel A.	González-Gay
A.	Amalia	Sánchez-Andrade
J.J.	Juan J.	Agüero
M.D.	Maria D.	Alonso
E.	Elena	Rodríguez
J.R.	José R.	Criado

Department of Internal Medicine (Sections of Rheumatology and Nephrology), and Department of Ophthalmology, Hospital Xeral Lugo, Lugo (Spain)

Miguel A. González-Gay, Section of Rheumatology, Hospital Xeral Lugo, Dr. Ochoa S/N, E-27004 Lugo (Spain)

Dear Sir,

Tuberculous skeletal disease is a relatively common entity in our country. Optic neuritis due to its treatment with isoniazid (INH) is a very unusual condition [1]. We report a case of optic neuritis following treatment with INH in a hemodialyzed patient with tuberculous arthritis. A 56-year-old man maintained on intermittent hemodialysis since 1983 because of chronic renal failure was admitted to our unit after suffering monoarthritis of chronic and insidious onset in his left knee for 3 months. Physical examination revealed swelling and tenderness in the left knee. Laboratory tests were consistent with the diagnosis of chronic renal failure. A tuberculin skin test (5 TU PPD) was negative, and chest x-ray studies were normal. A knee x-ray demonstrated soft tissue swelling, juxtaarticular osteoporosis and marginal bone erosions in tibia, without a significant joint space loss. Arthrocentesis and synovial biopsy were performed, showing an inflammatory synovial fluid with a very low glucose level. Acid-fast stains and culture of synovial fluid were positive for *Mycobacterium tuberculosis*, and examination of the synovium revealed caseating granulomata. Considering that the treatment of tuberculosis in patients on hemodialysis is similar to those having normal glomerular filtration rate [2], we started oral treatment with INH 300 mg once daily, rifampicin 600 mg once daily, and ethambutol 1,200 mg once daily (patient's weight: 75 kg). Prior to onset of therapy, a complete ophthalmologic exam was carried out and repeated again after 3 months when ethambutol was ceased. During the treatment, the patient received supplements of pyridoxine, 50 mg/day. After 8 months of treatment he noticed slow and progressive loss of vision. Ophthalmologic examination revealed a very low visual acuity (right eye: 0.100; left eye: 0.200). Both optic disks showed mild pallor with blurred borders. The rest of the retinal parenchyma was normal. Visual field examination disclosed centrocaecal scotomata. A diagnosis of optic neuritis was considered and treatment with INH was discontinued. We also increased the dose of pyridoxine to 360 mg/day. Four weeks later, the patient had almost recovered his normal vision level. A new ophthalmologic

examination showed normal visual fields and disappearance of the bilateral centrocecal scotomata, as well as the other ophthalmologic anomalies. Visual acuity was 0.800 in the right eye and 1.000 in the left.

Optic neuritis is a common entity, and optic neuropathies compose a wide range of conditions, specially related with inflammatory diseases. Optic neuropathy may also occur as a toxic effect of treatment with several drugs, specially in patients receiving ethambutol for tuberculosis [3].

We think that in our case there was a close relationship between treatment with INH and the optic neuropathy.

We believe that, due to the negative effects of hemodialysis over water-soluble vitamins, the possibility of an optic neuropathy following treatment with INH on dialyzed patients should be taken into account because of the potential reversibility of this complication if the drug is rapidly withdrawn after symptoms appear.

References

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