

Loss of Vision and Renal Function in a Patient with Miliary Tuberculosis

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Abstract

Introduction: Visual loss is a rare complication of tuberculosis; it can be related to anti-tuberculous drugs or to the infection itself. For the treatment of visual loss, differential diagnosis is important between infection and adverse effect of anti-tuberculous drugs.

Case: A 48-year-old male patient with a history of tuberculosis and visual loss during anti-tuberculous drug therapy was admitted to our hospital. Anti-tuberculous drugs had been stopped on the 2nd day of therapy due to development of optic neuritis secondary to ethambutol administration at another hospital. He had miliary tuberculosis, renal failure requiring dialysis and visual disturbances. Anti-tuberculous drugs, including ethambutol, were initiated at our clinic because the period between the ethambutol therapy and visual loss was too short and the dose of ethambutol was not very high. Computed brain tomography was normal. Fundoscopic examination revealed only hypertensive retinopathy. Our diagnosis was tuberculosis-related visual loss, which could be due to neuroretinitis, intraocular tuberculosis or chiasmal tuberculoma. In addition, ethambutol rarely causes visual loss during the early period or when given at lower doses. In our case no complications developed from the treatment and the patient's visual loss and renal function improved. At his last visit, 12 months later, his vision had improved and his serum creatinine was lower, at 2.2 mg/dL.

Conclusion: With anti-tuberculous treatment, renal functions and visual disturbances were improved in a patient with miliary tuberculosis. During the anti-tuberculous therapy, visual loss can be related to ethambutol toxicity or the tuberculosis infection itself. Differential diagnosis is very important and anti-tuberculous drugs must be continued if the diagnosis is tuberculosis.

Key Words: Anti-tuberculosis drug toxicity, visual loss, tuberculosis, renal failure.

THE FORM OF DISSEMINATED TUBERCULOSIS that is associated with primary infection or reactivation of tuberculosis is often seen in children and immunocompromised individuals, and it causes multi-organ failure if it cannot be controlled. Sudden blindness is a rare complication during active tuberculosis (1). We report a case of disseminated tuberculosis associated with acute renal failure and loss of vision.

Case

A 48-year-old man had previously been admitted to another hospital because of productive cough and dyspnea. The patient had a history of abdominal pain, nausea, vomiting, fever, night

sweat and weight loss of 15 kg over 5 months. On physical examination, bilateral supraclavicular and axillary lymph nodes were found; pathological diagnosis indicated hyperplastic lymph nodes. This patient had acute renal failure and required hemodialysis 8 times. Anti-tuberculous treatment included ethambutol (15 mg/kg/day), isoniazid (300 mg/day), rifampicin (600 mg/day) and pyrazinamide (15 mg/kg/day). Two days later, sudden blindness occurred. Due to the possibility of optic neuritis from ethambutol, this drug was stopped. Prednisolone (100 mg/day) and pyridoxine (20 mg/day) were started. His vision improved one day later and he was transferred to our nephrology clinic. On admission, his temperature was 37.8°C, pulse 92 beats/min, and blood pressure 110/80 mm Hg. Hypertensive retinopathy was found on fundoscopic examination. Axillary and supraclavicular lymph nodes of 1×1 cm and 1×1.5 cm, respectively, were detected. Laboratory investigation showed a blood leukocyte count of 9,200/mm³, hematocrit 31.8%, hemoglobin 11.02 g/dL, platelets 390,000/mm³, erythrocyte sedimentation

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rate 64 mm/hour, serum glucose 78 mg/dL, blood urea nitrogen (BUN) 140 mg/dL, creatinine 10.8 mg/dL, aspartate transaminase (AST) 20 IU/L, alanine transaminase (ALT) 28 IU/L, total protein 6.5 g/dL, serum albumin 3.4 g/dL, calcium 8.9 mg/dL, and phosphate 5.3 mg/dL. Proteinuria was 1 g per day. Antinuclear antibody and anti-DNA were negative. Computed brain tomography was normal (Figs. 1 and 2). Computed tomography (CT) of thorax and abdomen revealed a cavitary infiltration at right upper lobe and miliary reticulo-nodular opacity in both lungs, thickness of terminal ileum, and enlarged lymph nodes on mediastinum, mesenterium, retroperitoneum and axillae. Colonoscopy was performed. The histopathologic diagnosis of ileocecal lesions was chronic ulcerative inflammation. Presence of acid-fast bacilli was determined by aspiration of lymph node. The patient was treated with isoniazid (300 mg/day), rifampicin (600 mg/day), ethambutol (1 g/day), pyrazinamide (1.5 g/day) and pyridoxine (20 mg/day). He responded to anti-tuberculosis treatment and his vision returned to normal. In addition, his renal function improved and lymph nodes disappeared. At the last visit, 12 months later, his serum creatinine and BUN levels were 2.2 mg/dL and 34 mg/dL, respectively. Erythrocyte sedimentation rate had declined to 35 mm/hour.

Discussion

Sudden blindness may occur in patients with tuberculosis and tuberculosis meningitis. The most frequent causes of visual loss secondary to tuber-

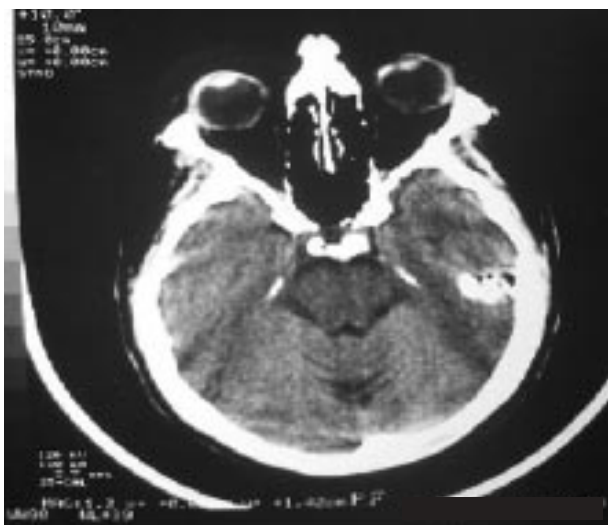


Fig. 1. Normal appearance of optic chiasma in computed brain tomography.

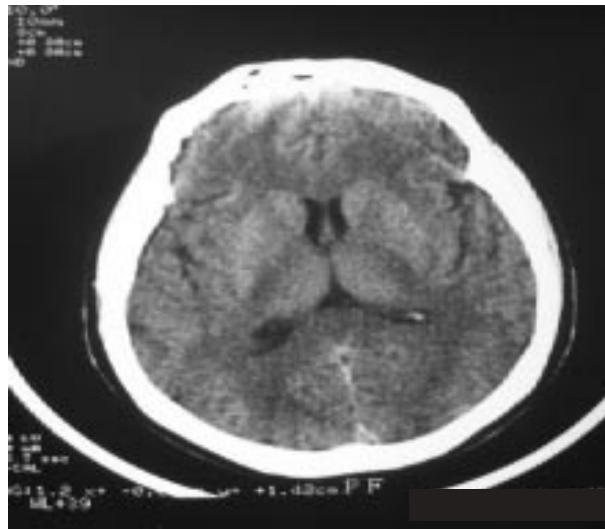


Fig. 2. Normal appearance of occipital lobe in computed brain tomography.

culosis infection are neuroretinitis, intraocular tuberculosis, perichiasmal tuberculoma and optic disc tuberculoma (2–5). Neuroretinitis, intraocular tuberculosis and chiasmal tuberculoma have been reported as first manifestations of tuberculosis (2, 4, 5). Besides direct ocular manifestation of tuberculosis, significant reversible and irreversible loss of vision can occur during ethambutol therapy (6). The risk of toxic optic neuropathy is related to dosage and duration of therapy. The safe daily dosage of this agent is 15 mg/kg. Reaction commonly occurs at higher doses (25 mg/kg) and several months after initiation of treatment (7). But during the early period, lower doses of ethambutol can cause toxic optic neuropathy. Isoniazid can also cause toxic optic neuritis, but less frequently. Common risk factors of optic neuritis are diabetes mellitus, chronic renal failure, alcoholism and advanced age (8).

Our patient received multiple anti-tuberculous agents; the daily dose of ethambutol was 15 mg/kg. After 2 days of treatment visual loss occurred. After using intravenous corticosteroid prednisolone (100 mg), his vision improved. We suggest that his sudden blindness might have been related to tuberculoma, even though brain tomography was normal, because this test is less sensitive than magnetic resonance imaging (MRI). Anti-tuberculous treatment with a steroid is one proposed treatment for tuberculoma. The need for surgical treatment is rare (3). We did not perform orbital CT because his clinical manifestation and blindness diminished with anti-tuberculous treatment. Hepatic and optic complications were not seen attributed to the therapy. He

showed improvement of renal function, with a stable creatinine clearance of 35–40 mL/min. Although ethambutol-related reversible blindness has been reported soon after treatment, loss of vision at the beginning of tuberculosis treatment may also be associated with the disease itself. Anti-tuberculous treatment with the supportive effect of steroid can prevent tuberculoma and neuroretinitis-related vision loss.

Conclusion

This case involved sudden blindness in a patient with disseminated tuberculosis. During the clinical course of disseminated tuberculosis, sudden loss of vision can be related not only to drug toxicity, but also to the disease itself (tuberculoma, etc.), and differential diagnosis is very important. Anti-tuberculous treatment should not be interrupted until the main cause of vision loss is determined.

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