Upward Gaze Paralysis as the Initial Manifestation of HIV-Infected Patient : A Case Report

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Abstract

Upward gaze palsy developed in a 23-year-old man without any other abnormal findings except pupillary light-near dissociation in ocular examination. The neuroradiologic examination was consistent with cerebral toxoplasmosis - the most frequent opportunistic infection of the brain in AIDS patients. The serologic work-up revealed a positive HIV test. Symptoms and neuroradiologic abnormality improved after treatment for cerebral toxoplasmosis. HIV infection should be considered in patients who have upward gaze paralysis and neuroimaging compatible with cerebral toxoplasmosis. Empirical treatment may alleviate the upward gaze palsy.

Key word : Upward Gaze Paralysis, Initial Manifestation, HIV, AIDS

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The initial manifestation of HIV infection is highly variable. Various ocular presentations have been reported in patients who have never been diagnosed with HIV infection. Blepharitis[1] and lid Kaposi's sarcoma[2] are potentially related to AIDS so ophthalmologists should consider HIV infection in all patients with eyelid lesions. Early recognition and identification of AIDS ocular manifestations would allow for timely palliative care. Toxoplasmic retinochoroiditis[3,4] has been reported as an initial manifestation in HIV-infected patients. Sison et al[5] estimated that approximately 1.8 per cent of patients with AIDS had cytomegalovirus retinopathy as the first manifestation and that fewer than 1 per cent of HIV-infected persons would develop cytomegalovirus retinopathy as an initial manifestation of AIDS

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in the first seven years after infection with HIV. Upward gaze paralysis has never been reported as the sole manifestation of cerebral toxoplasmosis.

CASE REPORT

A 23-year-old man presented with upward gaze palsy. He had never had any visual symptoms or underlying diseases before. He had no history of trauma or ocular surgery. On physical examination, he had normal appearance and vital signs. His visual acuity was 20/20 bilaterally. The intraocular pressure by applanation tonometer was 18,18 mmHg. The pupil diameter was 5 mm in both eyes, with a sluggish reaction to light, and 3 mm when the patient fixed on a near stimulus. Slit-lamp biomicroscopy and ophthalmoscopic examinations were normal. The ocular motility examination revealed paralysis of the upward saccades and the smooth-pursuit eye movements in both the duction and version tests of both eyes. The patient was able to move his eyes normally in the downward and horizontal directions (Fig. 1). The vertical oculocephalic (Doll's eye) test and Bell's phenomenon were preserved. No gross nystagmus was detected. The forced duction test revealed neither restriction nor entrapment of the inferior recti and superior obliques.

Magnetic resonance imaging (MRI) of the brain revealed scattered T2W hyperintense lesions that showed enhancement on postcontrast T1W images with surrounding edema at the posterior commissure. His blood tests were positive for HIV infection on both the ELISA and Western Blot tests. A loading dose of 150 mg of pyrimethamine followed by 25 mg daily was used[6], with a loading dose of 4 g of sulfadiazine followed by 1 g four times a day for 6 weeks[6]. Five days after starting treatment, his ocular signs began to improve. The upward gaze paralysis recovered completely after 2 weeks of medical treatment (Fig. 2).

DISCUSSION

This patient had limited saccades and smooth-pursuit eye movements in the upward direction but normal downward and horizontal eye movements.

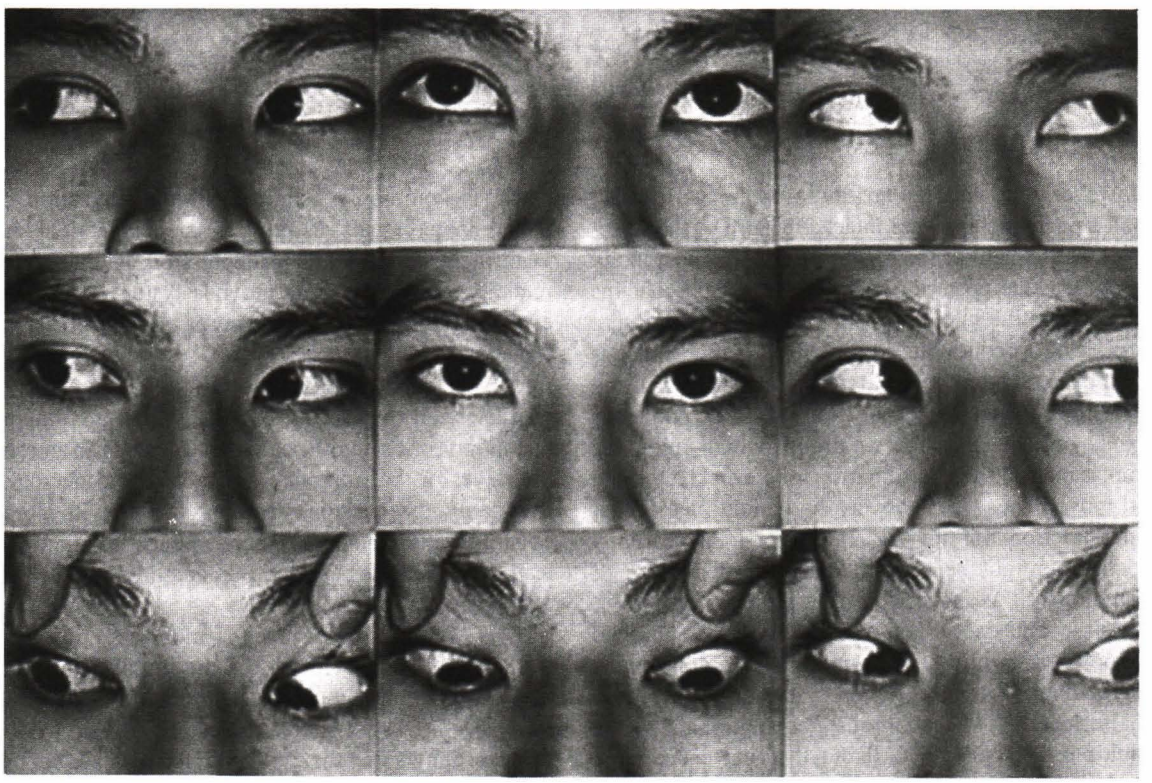

Fig. 1. Shows the upward gaze paralysis with normal downward gaze and horizontal gaze.

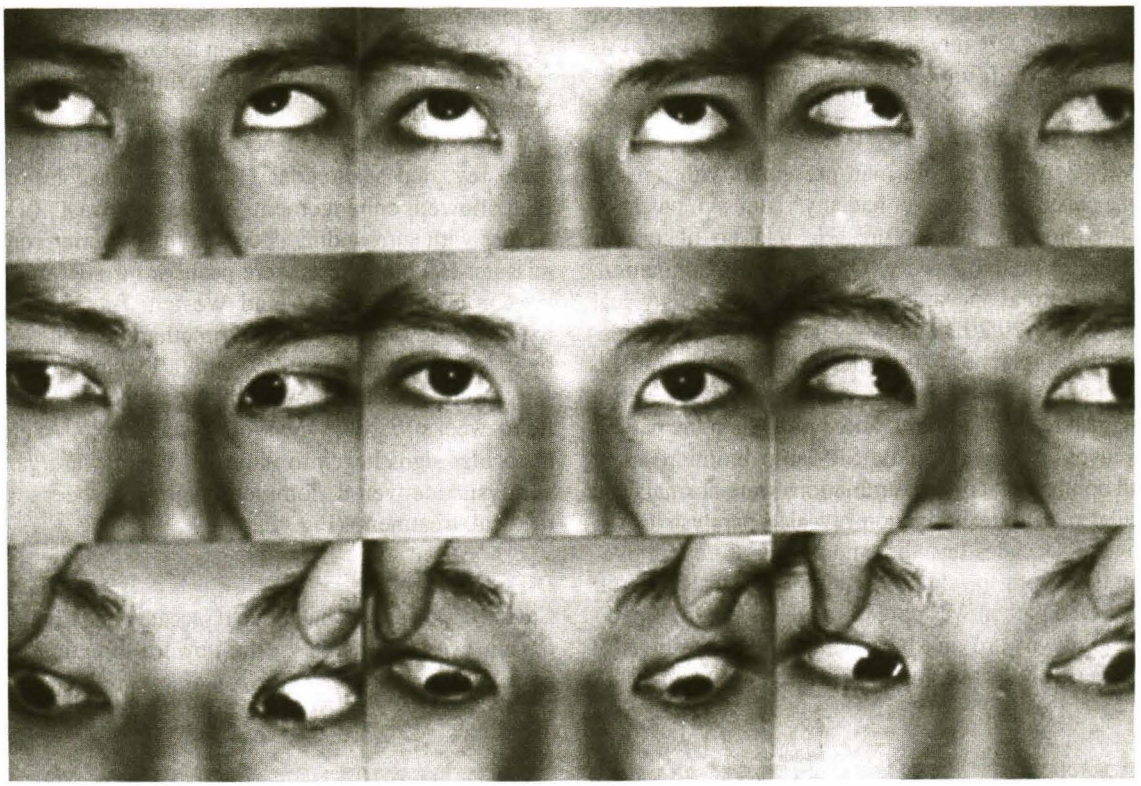

Fig. 2. Shows the improvement of upward gaze paralysis after medical treatments.

However, the vertical vestibulo-ocular reflex and Bell's phenomenon were intact. Because of the selective involvement of the upward gaze, the most likely lesion was the posterior commissure, which contains axons from the nucleus of the posterior commissure projecting to contralateral rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) and interstitial nucleus of Cajal (inC)[7]. It also contains axons projecting to the M group, which might be important for the coordination of vertical eye and lid movements[7]. However, the patient had neither lid retraction nor lid lag. Pupillary light-near dissociation might be explained by the disruption of ganglion cell axons entering the pretectal region[8]. Possible causes of lesions in this case included toxoplasmosis in the dorsal midbrain region, pineal region tumor[9], pineal region cyst[10], arteriovenous malformation[11], cavernous hemangioma[12] and multiple sclerosis[13]. All but the first cause were excluded by the MRI findings.

Toxoplasma gondii, which is commonly associated with infection in immunocompromised hosts, is commonly acquired through ingestion of contaminated meats resulting in latent infection[14]. With the onset of immunosuppression, it may preferentially infect the central nervous system (CNS) and eyes, resulting in a wide range of clinical presentations[6,14].

Ocular involvement in toxoplasmosis is characterized by several features, which may manifest singly or as multifocal retinal lesions in one or both eyes or with massive areas of retinal necrosis. Invariably, these lesions are unassociated with a preexisting retinochoroidal scars suggesting that the lesions are manifestations of an acquired rather than a congenital disease[14].

CNS toxoplasmosis is more common than ocular involvement. It may be the initial manifestation of AIDS and associated with single or multiple lesions[15]. Despite increasing attention paid to primary prophylaxis, the potential for this to be the presenting illness in previously unidentified HIV-infected individuals is likely to make toxoplasmosis an important source of neurologic morbidity in AIDS

(16). The majority of these patients present with focal neurological abnormalities in the presence of the characteristic ring-shaped enhancement after contrast injection during CT scan and MRI(17). In addition, Parkinsonian symptoms(18) and central diabetes insipidus(19,20) may also be the first sign of cerebral toxoplasmosis.

Neuro-ophthalmic findings have been found as an initial manifestation of AIDS. Precipitous visual loss, which subsequently led to the diagnosis of toxoplasmic papillitis and cerebral involvement, has been reported as an initial manifestation of AIDS(21,22). Parinaud's syndrome was reported as the sole manifestation of brainstem toxoplasmosis in an HIV-1 positive patient but it represents a rare manifestation(23,24).

MRI is more sensitive than CT in the diagnosis of cerebral toxoplasmosis(25). Non-contrast CT typically shows several hypodense lesions with solid or rim enhancement on postcontrast images. MRI typically shows several T2W hyperintense lesions that show enhancement on postcontrast T1W images. The lesions are usually associated with edema and mass effect(26). Bleeding may be present in some lesions. Lesions are multifocal in about 70 per cent of cases. Most lesions are supratentorial, and located at the gray-white matter interface or within the basal ganglia. The posterior fossa structures and cerebral cortex can also be involved(25).

SUMMARY

Toxoplasmosis is the most common opportunistic infection of the brain in patients with AIDS. Occasionally, patients who have had no history of HIV infection may present with CNS toxoplasmosis as the initial manifestation, therefore, HIV infection should be considered in any patient who has upward gaze paralysis and neuroimaging compatible with cerebral toxoplasmosis. Empirical treatment for toxoplasmosis may alleviate the upward gaze palsy.

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ภาวะอัมพาตของการกลอกตาขึ้นบนเป็นอาการแสดงแรกในผู้ป่วยติดเชื้อไวรัสเอ็ชไอวี : รายงานผู้ป่วย

*กิตติศักดิ์ กิจทวีสิน, พ.บ.**

รายงานภาวะอัมพาตของการกลอกตาขึ้นบนในผู้ป่วยชายอายุ 23 ปี โดยตรวจไม่พบอาการแสดงผิดปกติอื่นยกเว้นการตอบสนองของรูม่านตาต่อแสงและวัตถุระยะใกล้ MRI เข้าได้กับ Toxoplasmosis ซึ่งเป็นโรคติดเชื้อฉวยโอกาสในสมองที่พบบ่อยที่สุดในผู้ป่วยโรคภูมิคุ้มกันบกพร่อง ผลการตรวจพบเชื้อไวรัสเอ็ชไอวีโดยวิธี ELISA และ Western Blot ภายหลังการรักษา อาการและ MRI ดีขึ้น ดังนั้นควรคิดถึงการติดเชื้อไวรัสเอ็ชไอวีในผู้ป่วยที่มีภาวะอัมพาตของการกลอกตาขึ้นบนและ MRI เข้าได้กับ Toxoplasmosis การรักษาด้วยยาต้าน toxoplasma อาจทำให้ภาวะอัมพาตของการกลอกตาขึ้นบนดังกล่าวดีขึ้น

คำสำคัญ : ภาวะอัมพาตของการกลอกตาขึ้นบน, อาการแสดงแรก, ไวรัสเอ็ชไอวี, กลุ่มอาการภูมิคุ้มกันเสื่อม

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