Reverse Fisher's Syndrome : A Case Report

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A sixty one-year-old man who was perhaps the first case of reverse Fisher's syndrome due to posterior cerebral artery occlusion is presented. The patient experienced conjugate gaze disorders in which that the preserved eye movement was adduction in the contralateral eye, as opposed to the abduction described in the Fisher's original report. There was total paralysis in the ipsilateral eye. In this case, abduction in the contralateral eye was due to lesion of the left fronto-pontine pathway from frontal eye field. An infarct due to left posterior cerebral artery occlusion may produce a reverse Fisher's syndrome in which that the preserved eye movement is adduction. [Journal of Turgut Özal Medical Center 1997;4(1):93-95]

Key Words: *Fisher's syndrome, conjugate gaze disorders, posterior cerebral artery occlusion*

Ters Fisher sendromu : olgu sunumu

Altmışbir yaşında bir erkek, muhtemelen posterior serebral arter tıkanmasına bağlı ilk ters Fisher sendromu olarak sunulmaktadır. Hastada, Fisher'in orijinal raporundakinin aksine, karşı gözdeki hareket dışa bakma şeklinde idi. Karşı gözde total paralizi mevcuttu. Bu hastadaki kontralateral abdüksiyon sol fronto-pontin traktüsteki bir lezyona bağlıydı. Sol posterior serebral arter tıkanmasına bağlı bir infarkt, etkilenmeyen gözde addüksiyona (ters Fisher sendromu) yol açabilir. [Turgut Özal Tıp Merkezi Dergisi 1997;4(1):93-95]

Anahtar Kelimeler: *Fisher sendromu, konjuge bakış bozuklukları, posterior serebral arter okluzyonu*

In 1967, Fisher (1) first described two cases with clinical disorder of extraocular movements characterized by a lateral gaze palsy in one direction with an internuclear ophtalmoplegia (INO) in the other direction (Fisher's syndrome). Since then, 116 similar cases have been reported in the literature and confirmed the specifity of this syndrome (2-6).

In 1994, John et al. (7) described a patient with a similar syndrome of a single preserved horizontal movement in one eye, but their patient's syndrome differed only in that adduction, rather than abduction, was preserved. Their patient had mucormycosis of the sphenoid sinus involving the cavernous sinus and produced occlusion of the intracavernous internal carotid artery. Herein, we describe reverse Fisher's syndrome in a 61-year-old man in whom the preserved eye movement was adduction.

REPORT OF CASE

A 61-year-old man was admitted to the hospital because of gradual progression of marked weakness and hypoesthesia of the right side. The patient was well until two days earlier, when he experienced the onset of weakness and hypoesthesia of the right side that caused him to give up in walking. Computed tomography (CT) of the cranium, performed elsewhere, was reported as normal. Two days after

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the scan, he became right hemiplegic. His family observed that his speech was slightly slow and dysarthric. He complained of diplopia. There was no history of use of tobacco, excess alcohol ingestion, or illicit drugs. There was a 6-year history of hypertension, which was managed with enalapril maleat (10 mg/d).

On physical examination, blood pressure was 180/120 mmHg, heart rate was 84/min, and his body temperature was 36.5°C. The patient appeared tanned and well. General physical examination was normal except for right hemiplegia and ptosis of the left eyelid.

On neurologic examination, the patient was alert and fully oriented. His speech was slow and dysarthric. There was 3 mm of ptosis of the left eyelid. His pupils were anisocoric (right: 2.5 mm, left: 4 mm) and pupillary reflex was absent on the left side. Horizontal gaze to the right showed a total conjugate gaze paresis. On left gaze, there was paresis of abduction of the left eye, although the right eye did adduct with gaze-evoked horizontal jerk nystagmus. Vertical movements were absent at both eyes (Figure 1). Right central seventh nerve palsy and right hemiplegia were present. Corneal and gag reflexes on the right side decreased. On sensory examination, there was a marked decrease in all sensorial modalities at the right side. The plantar response was extensor at the right.

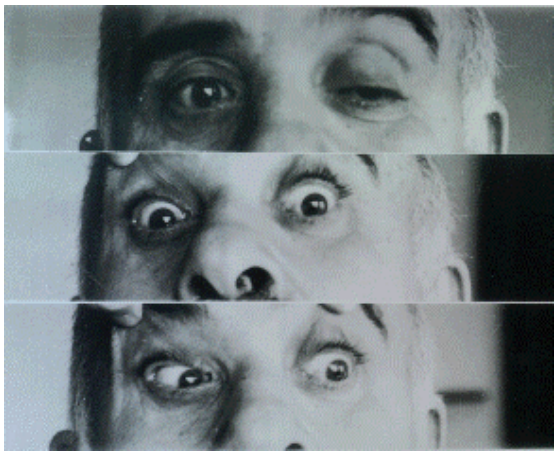

Figure 1. **Top**, Note marked ptosis of the left eye in the primary position of gaze. **Center**, Horizontal conjugate gaze palsy looking right. **Bottom**, On left gaze, there was paresis of abduction of the left eye, although the right eye did adduct.

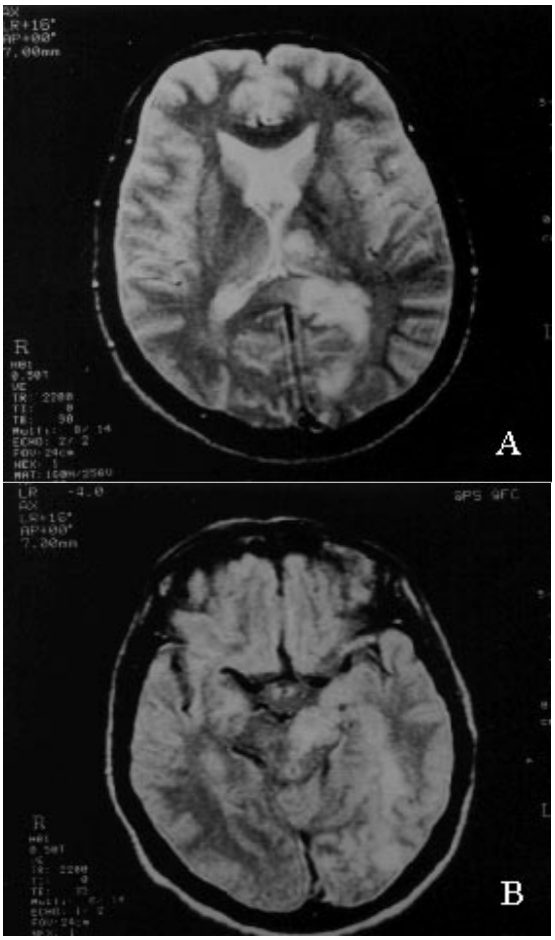

Figure 2. A) Axial T2 weighted (TR/TE: 2200/90) and B) proton density (TR/TE: 2200/35) MR images showing a large hyperintense lesion from pons to thalamus, and parieto-occipital white matter on the left..

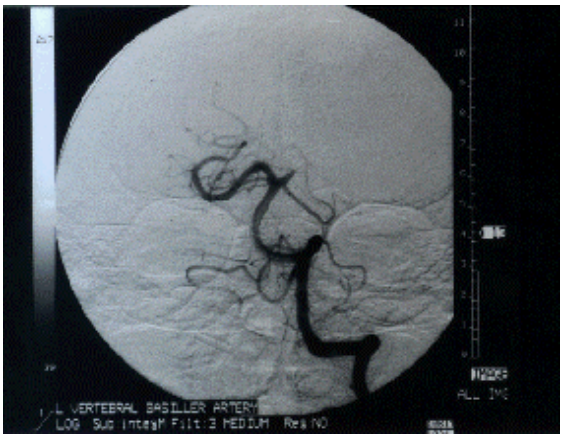

Figure 3. Digital subtraction angiography showing proximal left posterior cerebral artery occlusion.

Routine laboratory tests were normal. A cranial magnetic resonance imaging (MRI) scan that was obtained 3 days later revealed a large hyperintense lesion from pons to thalamus, and parieto-occipital white matter on the left (Figure 2 A and B). A digital subtraction angiography (DSA) scan that was obtained 5 days after the MRI showed proximal left posterior cerebral artery occlusion (Figure 3). The patient was started on antiplatelet and antihypertansive therapy. Within weeks, the patient improved minimally. Control cranial MRI that was taken 19 days later revealed minimal decrease in lesion size.

COMMENT

This is perhaps the second reported case of conjugate gaze paresis in which that the preserved eye movement was adduction (Reverse Fisher's syndrome, as opposed to the abduction described in the original report). To our knowledge, only one similar case was reported, in 1994 by John et al. (7), but they attributed the conjugate gaze paresis to mucormycosis infection of the sphenoid sinus that involved the cavernous sinus and resulted in occlusion of the intracavernous internal carotid artery. In contrast, we attributed the conjugate gaze paresis to posterior cerebral artery occlusion and confirmed by angiographically and our patient had not any infection.

We explained the mechanism of conjugate gaze disorder in our case as follows. Abduction paralysis in the contralateral eye is attributable to a left fronto-pontine pathway lesion from frontal eye field (8). Total ophtalmoplegia, dilated pupil, and ptosis of the eyelid in the ipsilateral eye are attributable to an ipsilateral third and sixth-nerve palsies due to midbrain and pontine lesions. Thus, only adduction of the contralateral eye was preserved. The additional findings of hemiplegia, central facial palsy, decreased corneal and gag reflexes, dysarthria, and hemihypoesthesia of contralateral side are attributable to left corticospinal, corticobulbar and right spinothalamic tracts lesion in the ipsilateral cerebral pedincule and thalamus (9). The sensory symptoms in our patient's face suggested that the trigeminothalamic tract was becoming involved.

In the future, with increasing recognition of the ocular movement disorders at the bedside and a growing opportunity for precise localization of brainstem lesions by improved MRI, we can look forward to better clinical definition of this distinctive syndrome. This is important to the neurologist because of the differences in the investigation, management, and prognosis of patients with hemorrhage, demyelination, infarction, or malignancy.

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