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MINOR HEAD TRAUMA AND ISOLATED UNILATERAL INTERNUCLEAR OPHTHALMOPLEGIA

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Cerovski Traumatic internuclear ophthalmoplegia

SUMMARY

Aim. Internuclear ophthalmoplegia is a syndrome which develops due to a lesion of the medial longitudinal fasciculus. This lesion is mostly caused by multiple sclerosis (usually bilaterally), and only rarely by head injury. A case is presented of unilateral internuclear ophthalmoplegia as an isolated sequel of minor head trauma, which resolved in five months.

Methods. Case report. **Results.** A 40-year-old woman with isolated internuclear ophthalmoplegia secondary to closed head trauma with anatomical lesions of the mesencephalon in the region of medial longitudinal fasciculus is described. A minor contusion was detected by magnetic resonance imaging. Diplopia resolved in five months.

Conclusion. Internuclear ophthalmoplegia should be taken in consideration on differential diagnosis in patients with recent head injuries showing adduction impairment. The connection between the clinical picture and anatomical lesions is visualized by magnetic resonance imaging.

Key words: internuclear ophthalmoplegia, magnetic resonance imaging, minor head trauma

Introduction

Oculomotor disturbances following head trauma are by no means rare. Direct injury of the eyeball or oculomotor nerves and orbital hematoma with or without orbit fracture may cause post-traumatic adduction impairment. Ocular adduction impairment secondary to head injury may be caused by internuclear ophthalmoplegia (INO) due to a lesion of the medial longitudinal fasciculus (MLF). When the lesion is unilateral, the INO is characterized by weakness of adduction ipsilateral to the side of the lesion. This weakness may vary from a complete loss of adduction beyond the midline to a mild decrease in the velocity or acceleration of adduction without any limitation in range of motion. The fibers subserving horizontal gaze in the MLF each carry commands for all types of conjugate eye movements. When testing the range of ocular movement, the examiner should ask the patient to follow a target through the full range of movement, including the cardinal positions of gaze. The eyes are tested individually with one eye covered and together with both eyes open. The normal range of movements is fairly stable throughout life for all directions except upgaze. Normal abduction is usually 50°; adduction 50°; and depression 45°. Upward gaze decreases

somewhat with advancing age from 40° to only 16°. When the range of motion is limited, several tests may be used, like Hess and Lancaster tests and cover tests. The interested reader should consult von Noorden and Perenin et al (1,2). INO is most commonly associated with demyelinating or cerebrovascular diseases, then with tumors and infection, or other brain stem symptoms (3-6). Isolated unilateral (as well as bilateral) INO after minor blunt head trauma has been less frequently reported (7-10).

Since most lesions were not visible on routine CT obtained at the time of the injury, this case report points to the importance of clinical diagnosis with magnetic resonance topographic identification of the injury. Magnetic resonance imaging (MRI) has the ability to correlate clinical findings with anatomical lesions (11).

Case Report

A 40-year-old woman sustained a minor head trauma when a car struck against the right side of her car while driving slowly. The patient was on the back seat, behind the driver, and struck the window with her head. Immediately after the accident, she felt nausea, vomiting on several occasions. On presentation, she complained of headache and diplopia. Diplopia was horizontal and not present on looking straight forward, only on looking sideways. There was no monocular diplopia. Neurologic status was normal, except for oculomotor impairment. Ophthalmologic examination revealed normal visual acuity and anterior segment finding. Ophthalmoscopy also produced normal finding. However, left eye adduction beyond the medial line was not possible, when testing the range of ocular movement. Adduction was present on convergence and there was no convergence impairment. Monocular nystagmus occurred on the eye in adduction on conjugated movements. MRI performed five days of the injury revealed an enhanced signal in the left half of the pons, suggesting axonal traumatic lesion of the white matter in the presumed area of the medial longitudinal fasciculus (Fig. 1). No other abnormalities were observed on MRI, neurologic, audiologic or vestibular examinations. The clinical symptoms resolved within five months.

Discussion

Internuclear ophthalmoplegia (INO) is a syndrome characterized by unilateral or bilateral adduction paresis with concurrence of dissociated nystagmus on the eye in adduction. The syndrome is caused by lesion to the medial longitudinal fasciculus (MLF). The term internuclear denotes lesion localization between the optic nerve III and VI nuclei. Clinically, when testing the range of ocular movement, the syndrome presents with diplopia on looking sideways, which is not present on looking straight forward, and nystagmus of the eye in adduction on conjugated movements. INO is usually associated with multiple sclerosis, i.e. with vascular disorders, and less commonly with tumors, encephalitis, Arnold Chiari syndrome with hydrocephalus, and degenerative diseases. INO is a rare complication of head injury. The neuroanatomy of INO is complex and the mechanism of trauma is controversial. A blow to the head produces a pressure gradient through the skull, resulting in shear force. This force exerts greatest effect in the ventricular area and at the site of white to gray substance junction. Traumatic MLF lesions may occur in two ways. Shear forces can push the posterior part of the brain stem downwards. This leads to extension of MLF nerve fibers and their possible damage. Shear force has also been postulated to cause circulatory impairment, thus leading to localized MLF infarction. On trauma infliction, shear forces may lead to extension and damage to blood vessels supplying the area of brain stem deep nuclei. This results in ischemic or hemorrhagic MLF infarction. Accordingly, an injury of the mesencephalon may cause contusion and extension of the fibers adjacent to the ventricular system (11-14).

As multiple causes can be involved in INO (11,14), this case report points to the need of the clinical examination be supplemented by neuroradiologic examination in order to connect clinical symptoms with the anatomical lesion. Also, other causes of the lesion should be excluded, because a mild trauma is frequently declared as the cause of INO while the real cause being only subsequently detected. In young individuals, bilateral INO quite commonly develops in association with multiple sclerosis due to MLF demyelination. INO may also be associated with intracranial tumor, pernicious anemia, hepatic encephalopathy, and various malformations (11,14). In the present case, MRI revealed a small pontomesencephalic paramedial lesion following head trauma, which was responsible for isolated INO. As mild trauma is extremely rarely the cause of INO, this case points to the importance of MRI in ruling out other possible etiologic causes of the condition.

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