

Oculomotor nerve palsy due to compression by posterior cerebral artery: case report

Posterior serebral arter basısına bađlı okulomotor sinir parezisi: olgu sunumu

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Abstract

Isolated oculomotor palsy due to vascular compression at the root exit zone is an extremely rare entity. A 59-year-old male patient was presented with complaints of diplopia and mild ptosis. A thin-sliced MRI demonstrated that, the right posterior cerebral artery was impinging on the right oculomotor nerve at the root exit zone. Carbamazepine therapy was initiated, and after a month of medication, both diplopia and ptosis had completely resolved. Although it is extremely rare, vascular compression of the oculomotor nerve at the root exit zone should also be considered in the differential diagnosis of isolated oculomotor palsy.

Key Words: **Oculomotor nerve disease; Paralysis; Posterior cerebellar artery.**

Özet

Kök çıkış bölgesindeki damar basısına bađlı izole okulomotor sinir parezisi son derece nadir görülen bir olgudur. Elli dokuz yaşında erkek hasta kliniđimize çift görme ve göz kapađında düşüklük şikayetleriyle başvurdu. Yapılan nörolojik muayenesinde sađ okulomotor sinir parezisi tespit edildi. Hastanın kranial manyetik rezonans incelemesinde sađ okulomotor sinirin kök çıkış bölgesine, sađ posterior serebral arterin bası yaptıđı görüldü. Karbamazepin başlanan hastanın tüm şikayetleri, birinci ayın sonunda tamamen düzelmişti. Nadir görülse de, damar basısı, izole okulomotor sinir parezilerinin ayırıcı tanısında mutlaka akılda bulundurulmalıdır.

Anahtar Kelimeler: **Okulomotor sinir hastalıđı; Paralizi; Posteriyör serebellar arter.**

Introduction

Isolated oculomotor palsy can be encountered in various situations (1). However, oculomotor palsy due to vascular compression at the root exit zone is an extremely rare entity, and there is only one case reported in the literature (2). In the present case report, a new case of oculomotor palsy due to the compression of the oculomotor nerve at the root exit zone by the posterior cerebral artery is presented.

Case Report

A 59-year-old male patient was presented with complaints of diplopia and mild ptosis. Diplopia was first noticed 3 months previously, and had not progressed since then. The patient was initially presented to an ophthalmologist, and then referred to a neurologist. A cranial magnetic resonance imaging (MRI) and digital subtraction angiography were performed, and both were negative. His past medical history was eventless. His blood glucose, triglyceride, cholesterol and remaining chemistry were within normal ranges. His clinical examination was completely normal. Tensilon test was performed in order to rule out myasthenia gravis. At the end of this evaluation, no diagnosis was established and acetylsalicylic acid was administered as a general precaution.

The patient was admitted to the Neurosurgery out-patient unit due to continuing diplopia. On examination, mild ptosis and minimal deviation of the right eye, laterally and inferiorly was detected. Deviation was more profound during left lateral gaze. Light reaction was intact, although sluggish on the right side, and anisocoria, which can only be noticed in bright light, was diagnosed. The clinical picture was isolated right peripheral oculomotor palsy, but the etiology was obscure.

A thin-sliced (2mm) MRI was performed, in order to evaluate the right oculomotor nerve. MRI demonstrated that, the right posterior cerebral artery, just as it originates from the basilar artery, was intending on the right oculomotor nerve at the root exit zone (Picture 1).

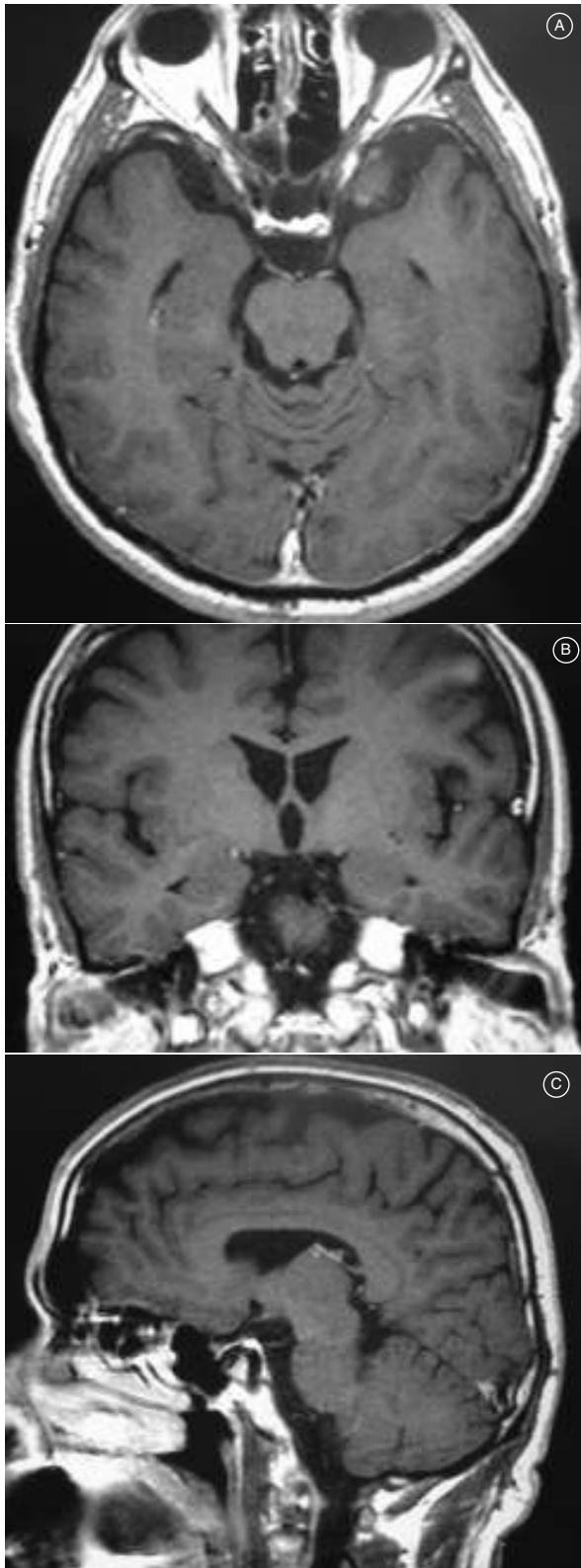
After the diagnosis had been established, microvascular decompression was advised, but refused by the patient. Therefore, carbamazepine therapy was initiated at a dose of 200mg, 3 times a day. After a month of medication, both diplopia and ptosis had completely resolved. After one year of follow-up, the patient is still symptom-free.

Discussion

Main oculomotor nucleus is situated in the anterior part of the gray matter that surrounds the aqueduct, at the level of the superior colliculus (3). Lesions involving the main nucleus lead to a pupil-sparing oculomotor palsy, because accessory parasympathetic nucleus of the oculomotor nerve, also known as Edinger-Westphal nucleus, lies posterior to the main nucleus. The most common pathological processes involving this region are infarction and hemorrhage (4). There are also neurological midbrain syndromes, such as Benedikt syndrome or Weber syndrome, involving the oculomotor nucleus, but these syndromes present with associated signs, such as flapping hand tremor or hemiparesis (1). Other pathologies involving the region are neoplasms, demyelinating diseases and infections. Myasthenia gravis may mimic the clinical picture, and therefore should be included in the differential diagnosis (5).

After passing the red nucleus, the oculomotor nerve emerges from the midbrain. Before entering the interpeduncular cistern, it has a root exit zone of 1.88 mm covered with pia (6). The nerve lies in close proximity to the posterior cerebral artery in this segment. In 13.7% of the cases, fiber bundles of the oculomotor nerve are pierced by arteria laminea tecti, which is a small branch of the posterior cerebral artery, generally arising from the P1 segment (7). However, vascular compression of the oculomotor nerve at the root exit zone is an extremely rare clinical situation, and to date there is only one case reported in the literature (2). This patient presented in the current case report will be the second such case in the literature.

The average length of the intracisternal segment is 18.82mm (6). It lies in close proximity to the posterior communicating artery in this segment. The most common pathology of this segment is the aneurysmal compression of the nerve. The most common aneurysm leading to oculomotor palsy is the posterior communicating artery aneurysm, followed by basilar tip aneurysm (8). There is a case report of atherosclerotic tortuous basilar artery compressing the oculomotor nerve in the cistern and leading to oculomotor paresis (9). Sphincter pupillae and ciliary muscles are also involved in these compression syndromes, because of the fact that parasympathetic fibers travel superficially within the nerve. Infectious, inflammatory and infiltrative pathologies may also be



responsible for oculomotor palsy in this region, but they generally present with multiple cranial nerve involvement. Although it is rare, schwannoma of the oculomotor nerve can also be encountered (10).

Cavernous segment of the nerve is 6-8mm long. Any mass lesion involving the cavernous sinus, such as tumors, aneurysms, and carotid-cavernous fistulas, can lead to oculomotor paresis (5). Isolated oculomotor palsy can be seen, especially in case of lateral extension of pituitary adenomas and intrasellar tumors, due to the fact that the oculomotor nerve becomes trapped between the interclinoid ligament above and petroclinoid ligament below, within the sinus. In addition, pathological processes involving the cavernous sinus such as thrombosis or Tolosa-Hunt syndrome, can also involve the oculomotor nerve along with the other cavernous sinus pathologies (1).

The oculomotor nerve leaves the skull and enters the orbit through the superior orbital fissure. It divides into superior and inferior branches within the posterior orbit (6). The nerve can be involved by inflammatory, endocrine or neoplastic conditions within the orbit. These pathologies are generally associated with proptosis.

As a conclusion, although extremely rare, vascular compression of the oculomotor nerve at the root exit zone should also be considered in the differential diagnosis of isolated oculomotor palsy.

Picture 1: Enhanced T1-weighted axial (A), coronal (B), and sagittal (C) magnetic resonance image of the patient.

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