A Rare Complication of Benign Intracranial Hypertension: Bilateral Facial Nerve Palsy

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Benign intracranial hypertension may occasionally be associated with cranial nerve palsies. Abducens nerve palsies occur in 10 % to 20 % patients with benign intracranial hypertension, other cranial nerve palsies occur much less frequently. We reported a 25-year-old woman benign intracranial hypertension with bilateral seventh nerve palsy. Complete improvement of her cranial nerve palsy was achieved with lumboperitoneal shunting within 14 days.

Key words: Benign intracranial hypertension, facial nerve palsy


Benign Intrakranial Hipertansiyonun Seyrek Görülen Bir Komplikasyonu: Bilateral Fasial Sinir Felci


Anahtar kelimeler: Benign intrakranial hipertansiyon, fasial sinir felci


Benign intracranial hypertension (BIH) is encountered most frequently in young, overweight women between the ages of 20 and 45 and BIH was first described in 1937 (1). Benign intracranial hypertension is a heterogeneous group of conditions characterized by increased intracranial pressure with no evidence of intracranial mass, hydrocephalus, infection or hypertensive encephalopathy (1). The pathogenesis is not fully understood. BIH is a syndrome that is defined by five criteria: increased intracranial pressure, normal or small-sized ventricles by neuroimaging, no evidence of an intracranial mass, normal cerebrospinal fluid composition, and papilledema. The most common presenting symptom in patients with BIH is headache, which occurs more than 90 % of cases (1). Dizziness, nausea, and vomiting may also be encountered, but typically there are no alterations of consciousness or higher cognitive function. Many conditions and factors have been proposed as causative agents of BIH, including excessive dosages of some exogenously administered medications (e.g., vitamin A, tetracycline, minocycline, nalidixic acid, corticosteroids), endocrinologic abnormalities, anemia, blood dyscrasias, and chronic respiratory insufficiency.

We presented a case with headache and bilateral facial nerve palsy as a result of BIH related to using oral contraceptive.
CASE REPORT

A 25-year-old woman was complaining bifrontal headaches and decreased peripheral vision. She has suffered from bilateral 7th cranial nerve palsies. An ophthalmologic examination revealed papilledema. Cranial magnetic resonance imaging revealed slit ventricles. Cranial computed tomography was not showed any bone deformity. We performed lumbar puncture and opening pressure was 650 mmHg (N: 150-180 mmHg). The fluid was acellular with normal protein and glucose levels. The cerebrospinal fluid (CSF) gram stain, cultures, VDRL, and cytology were negative. Our diagnosis was benign intracranial hypertension with bilateral facial nerve palsy. We thought reason of BIH is usage of birth control pills. Patient was given prednisolone 80 mg and acetazolamide 250 mg three times daily and lumbar puncture was performed daily. Her headache and facial palsies were not resolved. We treated our patient with a lumboperitoneal shunt. Her headache was resolved postoperatively and her facial palsies resolved in 14 days.

DISCUSSION

Cranial nerve palsies have been reported in BIH by authors with abducens nerve palsy in 10 % to 20 % of cases. Involvement of other cranial nerves has been rarely reported such as oculomotor, trochlear, trigeminal, facial nerve palsies (5). Authors hypothesized that small brainstem shifts from elevated intracranial pressure (ICP) exert additional traction on the extra-axial facial nerves (6). Increased intracranial pressure in the posterior fossa may displace the facial nerve in such a way that it is stretched throughout its entire length in the facial canal (4). Brackmann et al (3) reported unilateral facial paralysis with an enlarged fallopian canal as result of BIH. Imaging findings suggest that CSF is filling the enlarged proximal fallopian canal (3). During embryologic development, the facial canal frequently does not completely close at its cranial surface (facial hiatus) so that the perineural tissue of the geniculate ganglion maintains direct contact with the dura (3). Temporal bones contained a subarachnoid space that is limited to the petrosal fallopian canal, whereas 12 % displayed extension of the subarachnoid space laterally into the fallopian canal (3). However, we could not found any bone defect in our case. Our opinion same as other authors; facial nerve is stretching throughout its entire length in the facial canal.

In the literature many of BIH cases with facial nerve palsy were treated with serial lumbar punctures and dexamethasone, acetazolamide. Few of them need lumboperitoneal shunting. Several cases’ symptoms were permanent (5,6). First we tried to treat our case with lumbar punctures and medical treatment but we could not decrease the ICP. ICP was controlled with a lumboperitoneal shunting. The pathogenesis of BIH is unclear, but several theories have been proposed. The basis of the prevailing theory is that a relative obstruction of cerebral venous outflow exists, causing resistance to CSF absorption (2-4). Chutorian et al (2) first described BIH and Bell’s palsy in 1977. Abducens palsy occurs in 10-20 %, and diplopia is present in up to 38 % of cases. Anosmia occurs in 12 % (3). Cases involving trochlear, trigeminal and lower cranial neuropathies have been reported (3,4).

Even in case of benign increased intracranial pressure we should immediately diagnose and ICP should be decreased to normal range. ICP maintenance within normal range will improve patient’s neurological status and prevent permanent neurological defects such as facial nerve palsy in this report.

REFERENCES