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Spontaneous intracranial hypotension (SIH) is caused by a sudden reduction in cerebrospinal fluid (CSF) pressure and can manifest as sudden, persistent, orthostatic headache, which improves with lying down. Imaging generally reveals diffuse meningeal enhancement, subdural fluid collection, rounding of the superior sagittal sinus, or fullness of the pituitary from brain sagging, among others.¹ Although patients most commonly present with posterior neck pain/stiffness, nausea, vomiting, photophobia, or phonophobia, a much less common presenting symptom is cranial nerve palsies.¹ Spontaneous intracranial hypotension is often treated with epidural blood patches with resolution of headache,¹ followed by slower resolution of cranial nerve palsies over weeks. This case presents an unusual symptom of facial nerve palsy in association with SIH.

A 37-year-old male presented with new-onset positional headache (worse with standing, better while lying down) 1 day after performing vigorous abdominal “sit-up” exercise. The patient denied any recent or prior history of trauma. The initial brain magnetic resonance imaging (MRI) revealed subdural fluid collection, superior sagittal sinus rounding, and enlargement of the pituitary (Figure 1). The patient was thus admitted and placed on bed rest. The following morning, the patient developed new-onset left-sided facial paralysis. On examination, he had grade 6/6 left facial nerve palsy. There was no evidence of hearing impairment or CSF rhinorrhea or middle ear effusion. Magnetic resonance imaging myelogram did not reveal obvious CSF leaks. The patient was started on oral prednisone. Given the patient’s facial nerve paralysis as well as a remaining orthostatic headache possibly from an undetected CSF leak, 30 mL of autologous blood was epidurally injected in the thoracolumbar junction. The patient experienced significant improvement in his headache the following day. His facial nerve paralysis resolved over the following 3 weeks. The patient was asymptomatic and headache-free with completely intact facial expressions on his 6-month follow-up.

Facial paralysis is an uncommon sign in SIH with very few reported cases.^{2,3} Most commonly, patients with SIH with affected cranial nerves present with ophthalmoplegia, with an incidence of 30% to 35%.⁴ The sixth cranial nerve is primarily

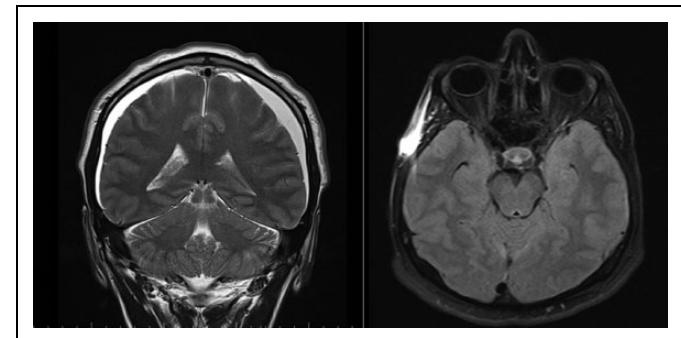


Figure 1. Magnetic resonance imaging (MRI) showing subdural fluid collection, superior sagittal sinus rounding, and enlargement of the pituitary.

involved among these cases of ophthalmoplegia secondary to SIH. The increased frequency of sixth nerve palsy when compared to other cranial nerves has been hypothesized to be due to the long course of the abducens nerve through the brain. Less commonly, patients present with diplopia secondary to third and fourth cranial nerve palsies.⁵ However, the case of facial nerve paralysis secondary to SIH has rarely been reported. Maus et al reported a case of isolated facial palsy secondary to SIH from CSF shunt overdrainage,⁶ and there have been sporadic reports of facial nerve paralysis secondary to intracranial hypotension status post epidural analgesia in the obstetrics

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literature, at times in conjunction with other cranial nerve palsies.^{3,7} In the cases related to epidural analgesia, cranial nerve palsy was preceded by a postdural puncture headache. Spontaneous intracranial hypotension-related cranial neuropathies tend to resolve within a few weeks to months after treatment as occurred in this patient.^{1,3,5}

Spontaneous intracranial hypotension is largely attributed to spontaneous unidentified spinal CSF leaks. The causes of such leaks are generally unknown, but in about one-third of patients, a history of “trivial” trauma is present.¹ This is consistent with our patient’s history of vigorous “sit-up” exercise prior to onset of symptoms. Other similar cases of SIH have been reported in patients after doing heavy lifting,⁸ racket sports,⁹ and horseback riding.¹⁰ Pachymeningeal enhancement and subdural fluid collection among others are seen on MRI and evidence of a CSF leak may or may not be seen on myelography or cisternography. Proposed mechanisms for these cranial nerve palsies have included traction on nerves due to downward displacement of intracranial contents, mechanical irritation/compression, and vascular congestion of the nerve,^{1,4,6} but the actual pathophysiology remains unclear. Workup includes computed tomography (CT) of the sinuses and temporal bones if fluid is seen in those within structures as well as CT or MRI myelography to look for the source of CSF leak. Treatment involves treating the source of the CSF leak, which usually entails an epidural blood patch procedure even in the absence of a defect on myelography.

Authors’ Note

Autefeh Sajjadi, Irene Chang, and Hamid R. Djalilian contributed equally to this manuscript.

Declaration of Conflicting Interests

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