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Acute onset of trigeminal neuralgia, facial paresis and dysphagia after mild head injury

Nikolaos Gkekas, Panagiotis Primikiris & Nikolaos Georgakoulia

Department of Neurosurgery, Athens General Hospital, G. Gennimatas, Athens, Greece

Abstract
The authors report the rare and first documented case of concomitant microvascular decompression of trigeminal, facial and glossopharyngeal nerves for the management of intractable to medical therapy acute onset of trigeminal neuralgia, facial paresis and dysphagia after mild head injury.

Keywords: cranial nerve; dysphagia; facial nerve; head trauma; microvascular decompression; trigeminal neuralgia

Introduction
Although there are extensive descriptions in the literature of cranial nerve lesions following head injury, triggering of trigeminal neuralgia, facial paresis and dysphagia after minor head trauma in the setting of multiple vascular compression has never been described before. To the best of the authors’ knowledge, this report describes the first case in the literature of concomitant microvascular decompression of trigeminal, facial and glossopharyngeal nerve for the management of trigeminal neuralgia, facial paresis and dysphagia after mild head injury. Head trauma as a possible triggering factor of this condition is further discussed, while the importance of careful and accurate pre-operative imaging control is emphasized.

Case report
History and examination
This 78-year-old woman was referred to our department with a 3-month history of acute onset of trigeminal neuralgia of left V1 and V2 distribution, with simultaneous left facial nerve paralysis (House-Brackmann Grade 3 progressively deteriorating to Grade 5) and swallowing difficulty, immediately after mild head injury. The cause of head trauma was a fall and the patient presented to the emergency department with peritraumatic amnesia and Glasgow Coma Scale 15. The initial computerized tomography (CT) brain scan was unremarkable with no evidence of skull fracture and her symptoms were intractable to medical therapy, while gradually deteriorating during the last 2 weeks. The patient’s hearing function was unaffected pre- and post-trauma.

Neuromaging
A magnetic resonance imaging (MRI) brain scan revealed a venous structure crossing the left trigeminal nerve and the anterior inferior cerebellar artery (AICA) entering the left internal acoustic meatus in proximity with the VII and VIII nerves (Fig. 1).

Operation
The patient underwent a left retromastoid craniotomy and through a retrosigmoid approach she was subjected to microvascular decompression of the left trigeminal, facial and glossopharyngeal nerves. Specifically, the glossopharyngeal nerve was decompressed from a loop of the left posterior inferior cerebellar artery, the trigeminal nerve was decompressed from the lateral group of superior petrosal veins, while, to our surprise, the postmeatal segment of the AICA was found embedded throughout the length of the vestibulocochlear and facial nerve complex, a possible anatomic variation that has not been described before in the setting of facial nerve compression. After drilling of the internal acoustic meatus and with meticulous microsurgical technique the postmeatal segment of the AICA was released passing through VII and VIII nerves. A fascia sleeve anchored to the tentorium with aneurysm clips held the AICA and the preserved superior petrosal veins away from facial and trigeminal nerves, respectively (Fig. 2).

Post-operative course
The patient had an uneventful post-operative course and was discharged free of pain and was capable of swallowing without difficulty and closing her left eye with effort (House-Brackmann Grade 3). The patient’s hearing function was left intact and she remained clinically improved with no need for medication at 6 and 12 months post-operatively.
Discussion

Mild head trauma (Glasgow Coma Scale 14 and 15) can be the cause of cranial nerve injury with a relatively high incidence. Trigeminal and glossopharyngeal nerves are affected rarely. A negative initial CT scan indicates usually a good prognosis with more chances for recovery. The acute onset of trigeminal neuralgia, facial nerve paralysis and dysphagia immediately after mild head injury with a negative brain CT scan led us to consider trauma as the cause of this condition. Given the fact that our patient was progressively deteriorating despite medical treatment and with an unremarkable brain CT scan, a MRI scan was conducted. Thorough analysis and correlation of the aforementioned imaging findings and symptoms led us to postulate trauma as a triggering factor for at least trigeminal neuralgia and probably facial nerve paralysis in the setting of vascular compression. Intraoperative findings of trigeminal, facial and also glossopharyngeal nerve vascular compression...
made the presumption of trauma as a triggering factor even more powerful. For reasons that have not been elucidated yet, trauma could act as a provocative factor for the manifestation of cranial nerve vascular compression symptoms that would otherwise be expressed or not, later in the life of the patient. Either direct mechanical irritation or indirect neuroendocrine pathways elicited by trauma could affect an already compressed cranial nerve and trigger pain or loss of nerve function. Research should shed light on the pathophysiology of this condition, helping further in elucidating the aetiology of trigeminal neuralgia or cranial nerve palsies induced by post-injury changes in neuronal function.\(^3\) Furthermore, any research effort should take account of the fact that both mild head injury and asymptomatic vascular compression of cranial nerves are common, yet symptomatic vascular compression of cranial nerves after head trauma is an extremely rare situation.

In the case of cranial nerve dysfunction after mild head injury with negative CT scan, vascular compression should be ruled out with further imaging control, especially when symptoms are intractable to medical therapy. In the setting of multiple vascular compression and after cautious interpretation of imaging findings, microvascular decompression can be a feasible and effective surgical method.

**Declaration of interest:** The authors report no declarations of interest. The authors alone are responsible for the content and writing of the paper.

**References**

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