Case Report

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Trigeminal Neuralgia as a Manifestation of Increased Intracranial Pressure: A Case Report

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ABSTRACT

The present case report describes a patient who had presented with resistant trigeminal neuralgia and was later discovered to suffer from idiopathic increased intracranial pressure. The related symptoms were resolved by decreasing the intracranial pressure. As in our case, symptom relief can predict a good prognosis in these cases, resulting in an improved quality of life. Although it is not frequently reported, this potential association can be significant as resolving intracranial hypertension can easily improve the symptoms in those suffering from this condition.
To our knowledge, no relationship between increased intracranial pressure and trigeminal neuralgia had yet been reported in the literature.

Background

Trigeminal Neuralgia is defined by frequent paroxysms of stabbing pain in the territory of trigeminal nerve. Vascular compression of the nerve is believed to be the most common underlying etiology in classical type. Moreover, it has been shown that TN can be secondary to some identifiable neurological problems, including Multiple Sclerosis (MS), cerebellopontine angle tumors such as schwannoma, meningioma, epidermoid cysts and basilar artery aneurysm.

Idiopathic intracranial hypertension is defined by specific clinical manifestations of intracranial hypertension when other causes of intracranial hypertension have been ruled out. The common manifestations of this condition include headache, transient visual obscuration, visual field defects, pulsatile tinnitus, diplopia, papilledema and abducent nerve palsy, while other cranial nerve disturbances are less common in this condition.

The present case report describes a patient who had presented with resistant TN and was later discovered to suffer from idiopathic increased intracranial pressure.

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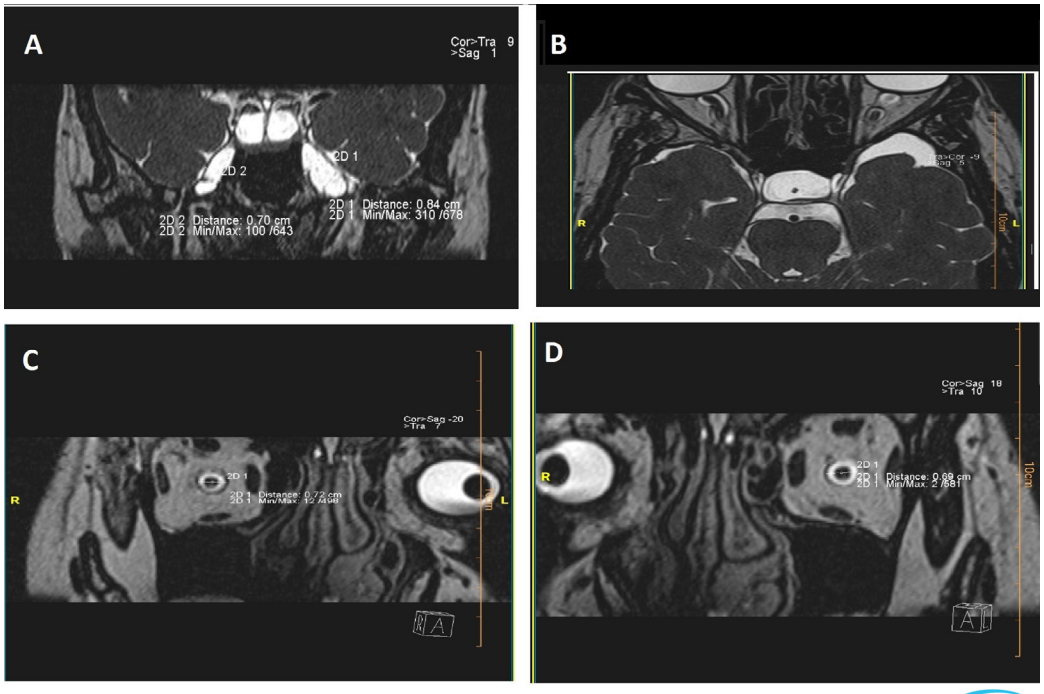

Fig. 1. Enlarged Meckel's cave and thickened optic nerve sheath

Case report

A 36-year-old woman with a history of neuralgiform pain in V2 territory for two years ago was presented to our center. Her chief complaint was frequent episodes of electric-shock-like pain radiating from maxillary molars to the upper jaw, which was triggered by applying pressure on maxillary molars. The patient had been diagnosed with TN and was primarily treated with carbamazepine. In the following, baclofen and pregabalin had been added as well. However, she was unresponsive to the medical treatment and underwent a nerve block, which caused a symptom relief for about 3 months. Then, there was a recurrence of symptoms, and the patient had to undergo a nerve block procedure for the second time. With another recurrence of symptoms, she presented to our center.

The Body Mass Index (BMI) of the patient was 27. Moreover, she had no other medical history, such as thyroid disorders or diabetes mellitus.

We obtained a cerebral MRI with FIESTA view, indicating no contact between the nerve and vascular loop. However, there was an enlarged Meckel's cave and thickened optic nerve sheath, suggesting an increased intracranial pressure (Fig. 1).

The patient underwent Lumbar Puncture (LP), showing an opening CSF pressure of 220 mmH_2O. We observed that the patient's symptoms were relieved entirely after LP and acetazolamide prescription.

She stated that the tinnitus she was suffering for years was relieved after LP. However, she had not reported this symptom previously. The patient was put on acetazolamide and is still symptom-free in follow-up visits after several months.

Discussion

The present case is reported as an unusual cause of TN. To our knowledge, no relationship between increased intracranial pressure and TN had yet been reported in the literature. However, there are reports of the involvements of the trigeminal nerve due to increased intracranial pressure, leading to manifestations such as facial numbness, absent corneal reflex, and trigeminal neuropathy [1-4]. The emergence of TN symptoms does not always follow a consistent pattern and different associated symptoms have been attributed to TN, suggesting the need for further investigation of the course and presentations of this condition, as well as the underlying pathophysiology leading to these various presentations.

The underlying mechanism of the observed association in our patient is unknown. However, potential mechanisms might be similar to those of other cranial nerve disturbances observed in increased intracranial hypertension, such as the underlying mechanism of VI cranial nerve palsy that is believed to be due to nerve stretching by dural tethering [5].

The dramatic therapeutic response of the present patient, who was known to be suffering from a medically refractory TN, may suggest that evaluating TN patients for other underlying causes by meticulously interpreted paraclinical tests might be beneficial. As in our case, symptom relief can predict a good prognosis in these cases, resulting in an improved quality of life. Although it is not frequently reported, this potential association can be significant as resolving intracranial hypertension not only improves the symptoms of trigeminal neuralgia but also prevents further complications of intracranial hypertension.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this article.

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Conflict of Interests

The authors have no conflict of interest to declare.

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