Case Report

Unilateral trigeminal motor nerve neuropathy✩,✩✩

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Abstract

Pure trigeminal motor neuropathy is represented by trigeminal motor weakness without signs of trigeminal sensory or other cranial nerve involvement. We present a case of 45-year-old male complaining of difficulty in chewing with facial asymmetry on right side. He had no sensory disturbances. History, neurological examination, CT and MRI led to the diagnosis of unilateral trigeminal motor nerve neuropathy. The cause may have been an autoimmune reaction to the viral infection.

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Introduction

Pure trigeminal motor neuropathy is commonly seen in the distribution of fifth cranial nerve [1]. This was first described by Chia in 1988, [2]. Unilateral trigeminal motor neuropathy is characterized by muscle weakness in the territory of one or more divisions of trigeminal nerve. Combined sensory and motor lesions are with tumors and viral infection [3–5]. However, pure trigeminal motor neuropathy is one of the rarely reported [6].

Here we present a patient with unilateral trigeminal motor nerve neuropathy on right side associated with motor

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weakness and muscle atrophy and not accompanied by trigeminal sensory involvement.

Case Report

A 45-year-old male was referred for CT investigation for facial asymmetry. The patient presented with shrunken right cheek and sense of weakness on same side. He also complained of difficulty in chewing on right side.

On general examination there was mild flattening of right side of cheek. On clinical examination pain and touch perception was present and equal on both sides of face in ophthalmic, maxillary, and mandibular division. No evidence of herpetic rash. No obvious tenderness on sinuses.

Corneal reflex was normal. Tongue sensation and taste was unaffected.

On asking clinching of teeth to patient, the normal tone and bulk of right masseter was absent. There was significantly reduced right lateral movement of the jaw.

CT imaging showed volume loss and fatty degeneration of right sided masticatory muscles (Fig. 1). Considering the rare nature of the pathology we decided to obtain MRI images on academic basis and also screened the intracranial and bask skull course of trigeminal nerve for any lesion.

Fatty degeneration was confirmed on MRI that revealed right sided masticatory muscles showing uniformly hyperintense signal on T1 and T2 wt. images (Figs. 3 and 4). Fat suppressed image (STIR) shows loss of normal outline of right sided masticatory muscles with signal drop (Fig. 2).

Screenings of brain and skull base do not show any abnormal mass lesion involving trigeminal nerve or mandibular division. Central pons showed old ischemic changes. The course of nerve within preptontine cistern was normal (Fig. 5).

In view of clinical history and imaging findings, final diagnosis of unilateral pure trigeminal motor nerve neuropathy was considered.

Discussion

Pure trigeminal motor nerve neuropathy is characterized by mandibular branch motor weakness without any signs of trigeminal sensory or other cranial nerve involvement [1,2]. It is seldom reported and histologic evidence of such lesion has not yet been reported. Even in idiopathic trigeminal neuropathy, weakness of muscles innervated by trigeminal motor nerve is rare, [1,7]. The etiology of this disease remains obscure. Suggested causes include neurofibromatosis, viral infection, multiple sclerosis, trauma or unknown factors [8,9].

The clinical manifestations vary with the chronicity of the process like facial asymmetry, weakness on chewing, and [3]. Our patient had similar symptoms. In some patients there may be initial symptoms like mastication problems and feeling of weakness in jaw muscle and most of the patients re-
Similar complaints suggest fatty degeneration of right-sided masticatory muscles.

Fig. 3 – Figure is axial T1 weighted; Image 4 is axial T2 weighed image. Both these images show homogeneously hyperintense signal of right-sided masticatory muscles as denoted by red arrows. Green arrows in all images denote normal appearance of left sided masticatory muscles. All these findings suggest fatty degeneration of right-sided masticatory muscles.

Fig. 4 – Figure is axial T1 weighted; Image 4 is axial T2 weighed image. Both these images show homogeneously hyperintense signal of right-sided masticatory muscles as denoted by red arrows. Green arrows in all images denote normal appearance of left sided masticatory muscles. All these findings suggest fatty degeneration of right-sided masticatory muscles.

Fig. 5 – Figure is T2 axial image at the levels of pons. It shows old ischemic changes in the central pons (denoted by yellow arrow). However no obvious lesion along the cisternal course of right trigeminal nerve (denoted by red arrow).

anatomical relationship, they turn sharply around the bone crest and finally run a narrow course between bone and their own target muscles, these nerves may suffer from entrapment, [7]. The cause of neuropathy remains obscure but we presume that it is very likely to be an autoimmune response to the past viral infection and the patient may not have noticed it.

The lesions that are difficult to detect by an imaging study such as entrapment of the nerve may be present, [7]. This may be true in our case also. We thoroughly evaluated patient through history, clinical, and radiological examination, it was finally diagnosed as damaged motor branch of trigeminal nerve. Facial asymmetry was due to right sided muscle atrophy due to pure motor trigeminal neuropathy. There was no sensory involvement.

There is no effective treatment for this condition. Where no etiology is confirmed, the treatment for this condition includes reassurance and physiotherapy, [3]. Our patient was referred to physiotherapy department for further management.

Conclusion

Diagnosis of pure motor trigeminal neuropathy involves clinical, neurological and radiological evaluation. However, high resolution cranial MRI is recommended to rule out any treatable cause.

Patient consent information

The patient had been explained that, details of case file and photographs will be used for academic publication. All per-
sonal information will be kept confidential and at no time will identity be revealed. Copies of investigation reports will also be published as per need. This was explained the in patient's native language. Patient had no objection to any of the above and gave permission for the same. Informed consent for publication has been obtained.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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