

FOCAL FACIAL SPASMS ASSOCIATED WITH BENIGN PAROTID HYPERTROPHY

Paulo Eduardo Mestrinelli Carrilho¹, Carolina Ferraz de Paula³,
Emerson W.S. Soares², Orival Alves¹

Compression of the facial nerve by a cerebellopontine angle mass lesion, or by aberrant arteries in the posterior fossa can produce repetitive clonic and tonic contractions of one side of the face¹. Such condition is also observed after Bell's palsy as a tardive complication². In spite of being relatively uncommon, a specific VII nerve branch damage can eventually generate focal spasms³.

We report a patient with such manifestation after chronic compression of facial nerve in its parotid segment caused by a gland benign enlargement.

CASE

A Caucasian 47 years-old man, without previous diseases, suddenly developed clonic and, sometimes, tonic involuntary contractions of the right orbicular oris muscle. Even a smile could elicitate the spasms. Neurologic examination was unremarkable, but a right parotid enlargement was noticed. Magnetic resonance imaging (MRI) disclosed a parotid tumour which was

initially considered to be an adenoma. The macroscopic aspect of the VII nerve with all its branches was normal and the histopathological study disclosed only a benign parotid hypertrophy. After the surgery, the patient obtained a partial relief in his focal spasms. The Figure disclosed the local of surgery. The patient gave an informed consent for this publication.

DISCUSSION

Myoclonic jerking of the paraspinal muscles due to malignant tumours involving thoracic roots and focal myoclonus after lumbar laminectomy or lumbosacral radiculopathy, electric injury to the brachial plexus and other kind of peripheral nerve system damages are rare but well-known entities^{1,3,4}. A more common condition, hemifacial spasms can also be triggered by a VII nerve injury, usually by a mass lesion in the cerebellopontine angle but even in other sites related to the extra-cranial branches in the face¹⁻³.



Figure. Red arrow indicates the topography of surgery.

ESPASMOS FOCAIS FACIAIS ASSOCIADOS À HIPERTROFIA BENIGNA DA PARÓTIDA

State University of West Paraná (UNIOESTE), PR, Brazil: ¹Professor of Neurology and Neurosurgery; ²Professor of Propedeutic; ³Medicine Student.

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Dr. Paulo E.M. Carrilho – Rua Santa Catarina 925 - 85801-040 Cascavel PR - Brasil. E-mail: pemcarrilho@gmail.com

Multiple pathophysiological mechanisms are probably involved, including abnormal branching after aberrant axonal regeneration and enhanced facial motoneuronal excitability². Local irritation of facial nerve causing ectopic excitation and ephatic transmission associated with demyelination may also be associated with the genesis of facial spasms⁵. However, other evidences suggest the presence of facial nerve nucleus hyperactivity in this condition⁶. Both theories can be combined. The partial lesion of the VII nerve, promoting ectopic excitation and ephatic transmission, results in both orthodromic and antidromic nerve activity associated with deafferentation, which can induce a reorganization of the nuclear neurons connections and activity².

Interestingly, in the present case, the lesion is confined to a specific VII nerve branch, promoting spasms in a very restricted area of orbicular oris. There were no mac-

roscopic damage observed in the nerve or its branches, besides, there was an improvement after the surgery. We speculate that the ectopic and ephatic theory could be more prominent in the present case than the reorganization of VII nerve nucleus functions.

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