



Hemifacial Spasm Revealing Contralateral Peripheral Facial Palsy

Periferik Fasiyal Sinir Paralizi Zemininde Kontralateral Hemifasiyal Spazm

İpek Güngör Doğan¹, Hande Alibaş², Kayıhan Uluç², Elif Kocasoy Orhan³

¹Darica Farabi State Hospital, Clinic of Neurology, Kocaeli, Turkey

²Marmara University Pendik Training and Research Hospital, Clinic of Neurology, Istanbul, Turkey

³Istanbul University Istanbul Faculty of Medicine, Department of Neurology, Istanbul, Turkey

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Dear Editor,

Peripheral facial nerve palsy (PFP) is a benign and self-limiting disease. Its etiology is usually unknown and it is known as idiopathic facial palsy or Bell palsy. However, it can cause transient or long lasting severe motor dysfunction and involuntary movement disorders (1).

A 58-year-old female was admitted to our neurology polyclinic with a slip in her mouth, dysfunction of tasting, and difficulty in closing her left eye. A facial nerve examination revealed asymmetry of the frontal muscles and oral commissures and weakness in closing her left eye. Findings suggested left PFP with a grade 4 House- Brackmann score. Otolaryngologic examination of the same side was normal. In biochemical tests, blood glucose level was 145 mg/dL, which was higher than normal. The patient had not been previously diagnosed as having diabetes mellitus. Diet and regular measurement of her blood glucose levels were advised. Oral methylprednisolone with a dosage of 64 mg/day was initiated. While oral steroid treatment was going on, physiotherapy was initiated at the second week of the palsy and lasted for 4 weeks. During follow-up, her glycated hemoglobin 'HbA1c' was found as 8% (normal: <5.7%) and oral anti-diabetic treatment was initiated. The patient was admitted to the neurology polyclinic with involuntary movements involving the right side of her face. Neurologic examination revealed partial improvement of left PFP. She could close her left eye and better pull the left commissure of her mouth (grade 2 House-Brackmann score).

On the other hand, involuntary movements showing clonic and sometimes tonic patterns involving the periocular muscles, cheek and lips of the right side were observed suggesting hemifacial spasm (HS). Contrast-enhanced magnetic resonance imaging of cranium showed normal parenchymal and vascular structures. The patient was considered as having 'contralateral HS on PFP'. Electromyography (EMG) showed lower compound muscle action potential (CMAP) amplitude on the left facial nerve compared with the right side (facial nerve CMAP amplitude recording on musculus nasalis was 1.8 mV on the right and 0.7 mV on the left; facial nerve CMAP amplitude recording on musculus orbicularis oculi was 1.1 mV on the right and 0.2 mV on the left). Needle EMG revealed motor unit potentials showing chronic denervation and reinnervation findings in the muscles innervated by left facial nerve. The findings were suggestive of chronic partial axonal damage in the regeneration period in the left facial nerve. Surface electrode recordings on the contralateral musculus levator anguli oris and musculus orbicularis oculi showed findings suggestive of HS.

Movement disorders occurring contralateral to peripheral nerve palsies have been reported rarely in the literature including an increase in spontaneous blink frequency and muscle activity similar to blepharospasm (1). HS, as a such movement disorder, has been reported very infrequently in the literature. Bonnet et al. (2) reported a patient with a prior HS who developed contralateral idiopathic PFP. Ekmekçi et al. (3) discussed HS-PFP as a crossed

Address for Correspondence/Yazışma Adresi: İpek Güngör Doğan MD, Darica Farabi State Hospital, Clinic of Neurology, Kocaeli, Turkey

Phone: +90 535 510 65 77 E-mail: dripegngnr@gmail.com ORCID ID: orcid.org/0000-0002-8667-9119

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involvement in a patient who had multicranial neuropathy secondary to trauma.

Chuke et al. (4) first described that insufficient response of the eye lid to corneal irritation in the paralytic side activates compensatory and adaptive mechanisms and afferent inputs originating from the cornea of the paralytic side, which has compensatory and adaptive changes that activate the contralateral facial nerve. Indeed, electrophysiologic recordings performed in subsequent years showed that inputs carried by the afferent fibers of the trigeminal nerve ophthalmic branch in the paralytic side cause increased excitability of the blink reflex in the non-paralytic side and proved quantitatively that the same increase cannot be provided by the inputs carried by the afferent fibers in the non-paralytic side (5). In light of the studies, pathophysiologic mechanisms are based on the sensitization in polysynaptic pathways of the blink reflex and increased excitability in facial motor neurons (1).

On the other hand, if axonal damage occurs, regeneration activity starting in the following weeks with abnormal axonal branching and inappropriate target innervation presents in the paralytic side and in the non-paralytic side. The mechanism as to how this process, which causes postparalytic facial syndromes by clinical-subclinical hyperactivity also affects the non-paralytic side, is not known well. It is suggested that the chemical changes occurring in the paralytic side can act like remote motoneuronal reorganizations created by local botulinum toxin injections. In addition, muscle fibers showing denervation in the musculus orbicularis oris, which is a circular muscle, can act as an additional stimulus for the contralateral facial nerve to show axonal regenerative changes (1).

As a result, retrospectively, we could not find evidence indicating that our patient had blepharospasm-like muscle activity

in the non-paralytic side in the early period. We found it worthy to present our patient with contralateral HS that occurred in the improvement period of the paralytic side, as an example of crossed involvement in the spectrum of postfacial syndrome.

Ethics

Informed Consent: Consent form was filled out by all participants.

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Authorship Contributions

Surgical and Medical Practices: İ.G.D., Concept: İ.G.D., E.K.O., Design: İ.G.D., E.K.O., Data Collection or Processing: İ.G.D., H.A., K.U., Analysis or Interpretation: İ.G.D., H.A., K.U., Literature Search: İ.G.D., Writing: İ.G.D., E.K.O.

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