

# Facial Aberrant Reinnervation Syndrome (FARS) Causing Synkinetic Dystonia of Platysma

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Facial Aberrant Reinnervation Syndrome (FARS) is a sequela of facial nerve injury. The clinical presentations are varied in form of facial synkinesis, facial muscle hypertonicity, and facial muscle spasm/twitching, which occur following facial nerve injury and recovery. Facial synkinesis is a symptom that manifests as pathologic contraction and baseline hypertonicity of muscles innervated by the facial nerve because of FARS.

A 72-year-old female presented to us with history of abnormal movements involving the right lower part of her face and neck region with associated tightness since 20 years. She was apparently alright 20 years back when while working in the kitchen the stove blasted and she sustained burn injury to right neck and face. The burn injury was reported as deep partial to full thickness involving approximately 8-10% Total Body Surface Area (TBSA), predominantly affecting the right lower face and upper neck region. The injury likely involved the marginal mandibular and cervical branches of the facial nerve based on anatomical location. She subsequently underwent reconstructive plastic surgery, which included debridement and split-thickness skin grafting over the affected neck and lower facial region. One month after surgery she developed the abnormal movements. It predominantly involved her right lower face and neck. Typically, it aggravated when she used to speak. Otherwise it was mild and intermittent in nature and occurred in bursts lasting for seconds to one or two minutes. It did not occur during sleep. The abnormal movements progressed gradually over weeks and remained non-progressive over the subsequent years. The patient did not seek specialised neurological evaluation earlier as the symptoms were intermittent and not functionally disabling. No prior botulinum toxin therapy, physiotherapy, or electrophysiological evaluation had been attempted. Her general examination was normal. Other systemic examinations were normal.

On neurologic examination, higher function was normal. Cranial nerves were intact.

Facial nerve function was graded as House-Brackmann Grade I Facial Grading System and composite score was 92/100, with points deducted for synkinesis component. Facial nerve function was graded as House-Brackmann Grade I at rest and during voluntary movements, indicating normal facial symmetry and strength without weakness. However, House-Brackmann grading does not account for synkinetic or aberrant muscle activation. In this patient, despite preserved voluntary facial strength, synkinetic activation of the right platysma was observed during speech and lower facial movements, consistent with aberrant reinnervation rather than facial palsy. Differential diagnoses such as cervical dystonia, hemifacial spasm, focal myokymia, and scar-related mechanical contracture were considered. Cervical dystonia was unlikely as there was no sustained abnormal neck posture. Hemifacial spasm was excluded due to absence of orbicularis oculi involvement and lack of spontaneous spasms. There were no continuous rippling movements suggestive of myokymia. The movement was strictly task-induced and reproducible with

speech, favouring synkinetic activation due to aberrant facial nerve regeneration.

The patient appeared comfortable at rest, though there was a baseline hypertonic band of the right side of the platysma with no visible neck muscle activity. But when she spoke there was a distinct tightening of the platysma on right side of the neck. As shown in the video; the muscle contracts in short bursts, producing a narrow vertical band along the lower jaw line and upper neck. This contraction happens each time she moves her mouth for talking, but it disappears when she stops talking [Video-1]. Her facial strength was otherwise normal, with good symmetry of mouth, eye and forehead movements. There are no abnormal movements elsewhere in the face or neck. The pattern suggests that the platysma is being activated unintentionally during lower facial movements. Given the history of previous cosmetic surgery, this abnormality is most consistent with synkinesis caused by misdirected/aberrant re-growth of the marginal, mandibular, or cervical branch of the facial nerve into platysma muscle. Essentially, FARS causes platysma hypertonicity and synkinetic dystonia. Functionally, the patient reported social discomfort during conversation due to visible neck tightening but denied pain, dysphagia, or speech impairment. Activities of daily living were not significantly affected.

The FARS can cause various types of clinical abnormalities. These are synkinesis that is involuntary contraction of one muscle during voluntary movement of another, muscle hypertonicity caused by increased resting muscle tension, and spasmodic synkinetic dystonias or twitching following facial nerve injury and regeneration like in this case [1]. It signifies the abnormal, aberrant regeneration of branches of facial nerve after injury [2].

The FARS is by far an underrated clinical scenario not only because it does not cause overt difficulty for the patient but also due to the limited research into its epidemiology and risk factors. The basic pathophysiology is misguided nerve regeneration, polyneuronal innervation, and cortical neuroplastic changes that contributes to these manifestations.

Treatment options include physiotherapy, targeted botulinum toxin injection into the right platysma muscle and facial physiotherapy with biofeedback techniques. Botulinum toxin therapy was recommended as first-line symptomatic management [3,4]. The patient was counseled regarding expected benefits and the need for repeat injections. At the time of reporting, she was considering treatment options.

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