Case Report

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Marin-Amat Syndrome: A Case Report of a Rare Facial Synkinesis Following Traumatic Facial Nerve Injury

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ABSTRACT

Marin-Amat syndrome is a rare form of facial synkinesis resulting from aberrant connections between the trigeminal and facial nerves. This condition manifests as involuntary eyelid closure upon voluntary jaw movement. It is distinct from the more common Marcus Gunn jaw-winking syndrome (MGJWS), which involves upper eyelid elevation during mastication. Here, we reported a case of a 64-year-old woman who developed this syndrome following combat-related maxillofacial trauma to the lateral mandible, which resulted in a left facial nerve injury. After her mandibular fractures were stabilized using 2 mm plates, her medical history was notable only for hypothyroidism. Six months post-injury, and following an attempted free fibula flap procedure, the patient began experiencing involuntary facial movements. These symptoms caused significant social discomfort and difficulty with eating. Treatment options, including botulinum toxin and surgery, were discussed; however, the patient opted for a conservative management approach.

Keywords: Facial nerve, Marin-Amat syndrome, Marcus Gunn phenomenon, Mandibular defect.

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Introduction

Facial nerve paralysis is a significant clinical condition with considerable physical and psychological implications for patients. The loss of facial animations, asymmetry, impaired eye closure, and drooling substantially diminishes the quality of life of the affected patients. While idiopathic Bell's palsy accounts for up to 70% of lower-motor neuron facial palsy cases, traumatic events are responsible for 10-23% of all cases [1]. Such injury often involves a temporal bone fracture, frequently accompanied by facial or spinal trauma [2]. Extratemporal injuries can result from sharp trauma, gunshot wounds, animal bites, and falls [3].

Facial synkinesis refers to involuntary mimetic muscle movements that accompany voluntary facial expressions. The most clinically significant form is oculo-oral synkinesis, whose proposed pathomechanism involves misguided growth of collateral axonal sprouts [4]. Several specific syndromes have been characterized. In 1883, Marcus Gunn jaw-winking syndrome (MGJWS) was identified as a synkinetic movement of the upper

eyelid during jaw movements, caused by miswiring between the motor branches of the medial pterygoid muscle and the levator palpebrae superioris [5]. Later, in 1918, the Marin-Amat syndrome was described, featuring synkinetic eyelid closure upon jaw movement or opening, resulting from orbicularis oculi muscle stimulation triggered by jaw movements [6]. As congenital cases are extremely rare, most reported cases are acquired and linked to abnormal connections between cranial nerves V and VII [7]. Proposed processes for its development include axonal misdirection, demyelination, multiple axonal sprouting, and abnormal branching [8, 9]. The extent of axonal injury can influence synkinesis development, which may emerge anywhere from four months to 40 months after the initial insult [10].

Diagnosis begins with a clinical examination at rest and during facial exercises, documented with photos and videos. The severity of facial asymmetry is evaluated using grading systems such as the Sunnybrook Facial Grading System (SFGS) and the automated machine-learning tool (auto-eFACE). The Synkinesis Assessment Questionnaire (SAQ) is used for patient-reported outcomes. Electromyography (EMG) can also serve as a diagnostic tool.

While various treatment methods have been explored, none have proven to be ideal. Non-surgical options include physiotherapy, cognitive therapy, neuromuscular retraining, and EMG feedback exercises. Chemodenervation with botulinum toxin is also widely used [11]. For patients unresponsive to symptomatic treatment, surgical options, such as levator aponeurosis advancement, orbicularis oculi myectomy, and nerve branch neuroectomy could be considered, typically after synkinesis has stabilized within 18 months post-injury.

Case Presentation

A 64-year-old woman from the Gaza Strip was transferred to Egypt for the reconstruction of a lateral mandibular defect. Upon admission in February 2025, she presented with left facial palsy, left sensorineural hearing loss, and a history of hypothyroidism. Physical examination revealed facial asymmetry with scarring on the lower third of the face, laterognathia upon mouth opening, and limited swallowing to soft foods. The patient was anemic, with a hemoglobin level of 10.4 g/dL, and had an uncontrolled thyroid profile: TSH 180 µIU/mL, free T3 0.2 ng/mL, and free T4 0.76 µg/dL. An echocardiogram showed an ejection fraction (EF) of 61% with grade I diastolic dysfunction. Liver and kidney function tests were within normal ranges. An ophthalmological examination revealed left ptosis, dry eyes, a right pterygium, and a normal fundus.

Six months prior, the patient sustained a blast injury to the left side of her face, resulting in a substantial loss of the left lateral mandible. Initial management at a healthcare center in Gaza involved stabilization of the bony fragments using 2 mm fixation plates (Figures 1 and 2). The patient had not taken her L-thyroxine medication for several weeks before admission due to a shortage, rendering her unsuitable for surgery until her hypothyroidism was managed.

After six weeks of treatment, the microsurgery team deemed her ready for a free fibula flap to reconstruct her mandibular defects. Although pre-operative computed tomographic angiography (CTA) showed a patent external carotid artery and its branches, the procedure was aborted due to significant atherosclerotic and fibrosis. The plates and screws were removed, and a left commisuroplasty was performed.

A few weeks later, the patient began experiencing involuntary closure of her left eye upon opening her mouth to swallow food (Figure 3). She also reported social discomfort due to these movements. Based on clinical examination and medical history, she was diagnosed with Marin-Amat syndrome.

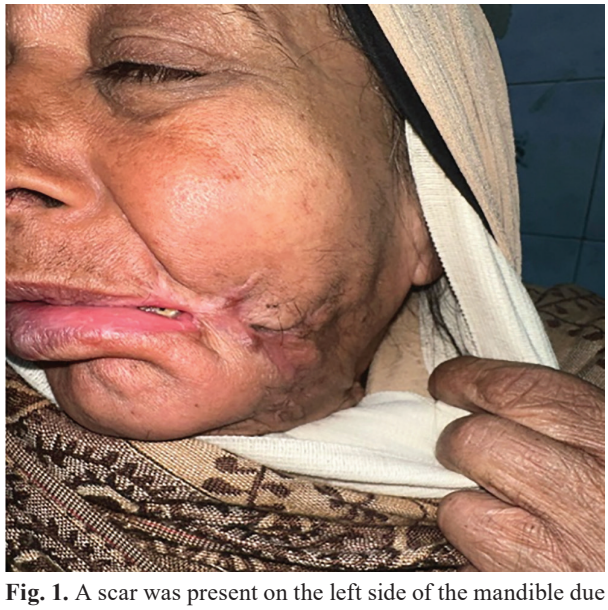

Fig. 1. A scar was present on the left side of the mandible due to the blast injury.

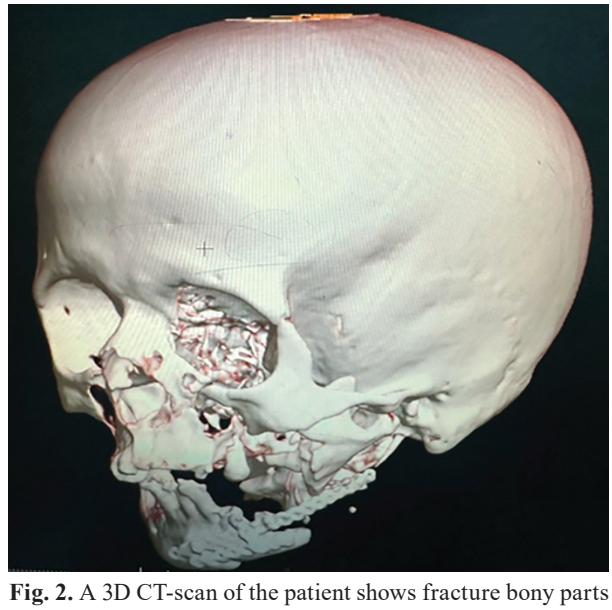

Fig. 2. A 3D CT-scan of the patient shows fracture bony parts fixed by 2 mm plates and screws.

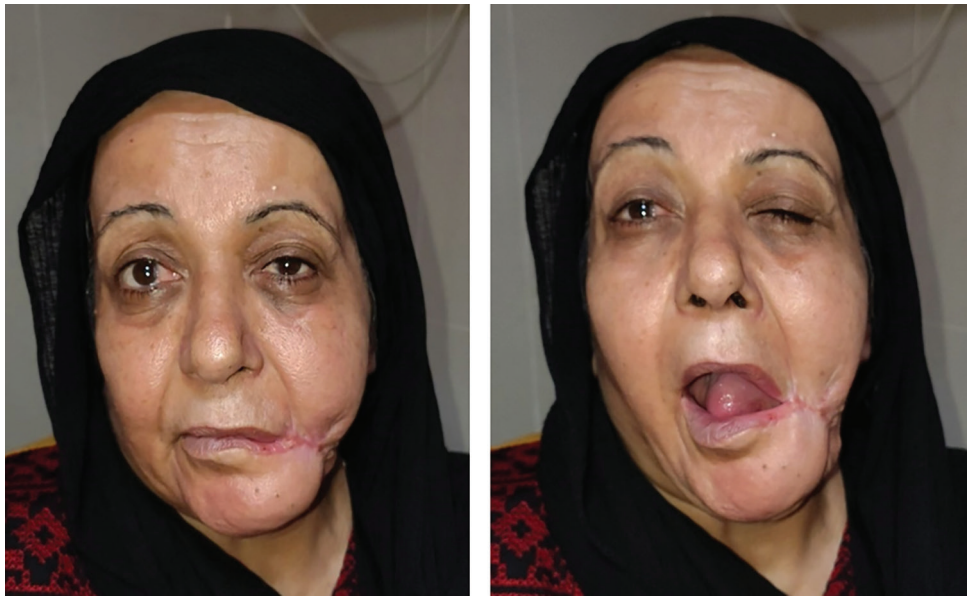

Fig. 3. Left eyelid closure accompanies voluntary movements of the jaw.

She was counseled on her condition and advised to pursue conservative management, including mirror exercises, stretching, and passive massage. At the six-month follow-up, her condition improved, and she reported being able to cope with the symptoms.

Discussion

This case report detailed a rare type of oculo-oral synkinesis. Although typically associated with idiopathic facial nerve palsy, there is limited documentation of this condition following traumatic facial nerve injury. The pathophysiology of synkinesis is most commonly attributed to aberrant axonal sprouting and miswiring; other proposed mechanisms include maladaptive cortical plasticity, ephaptic transmission, and nuclear hyperexcitability.

Since Marcus Gunn jaw-winking syndrome is a common facial synkinesis, the diagnosis of Marin-Amat syndrome, also known as inverse Marcus Gunn syndrome, can often be overlooked. Management options for this rare synkinesis include low-dose botulinum toxin injections into the orbicularis oculi muscle [12]. For patients who are not suitable candidates for botulinum toxin, surgical interventions such as levator aponeurosis advancement and partial resection of the orbicularis oculi muscle have shown promising results [7]. Additionally, biofeedback rehabilitation can improve the patients' quality of life.

While most reported cases of Marin-Amat syndrome occur after Bell's palsy, the present case highlighted its rare development following traumatic facial nerve injury [13]. The potential influence of comorbid medical conditions on the abnormal axonal sprouting that leads to this syndrome is not yet fully understood. As diagnosis currently relies primarily on clinical assessment, future studies should focus on developing objective diagnostic tools to aid clinicians in managing patients with facial nerve palsy.

Declaration

Ethical approval and participation consent: The study protocol received approval from the Ethics Committee of Mansoura Faculty of Medicine (code: R.25.01.3011). Written informed consent was obtained from the patient.

Consent for publication: Written informed consent was obtained from the patient and could be provided upon request.

Conflict of Interests: The authors declare that they have no conflicts of interest.

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References

1. Singh A, Deshmukh P. Bell's Palsy: A Review. *Cureus*. 2022;**14**(10):e30186.
2. Baugh RF, Basura GJ, Ishii LE, Schwartz SR, Drumheller CM, Burkholder R, et al. Clinical practice guideline: Bell's palsy. *Otolaryngol Head Neck Surg*. 2013;**149**(3 Suppl):S1–27.
3. Chávez-Serna E, Fukumoto-Inukai KA, Romero-Caballero R, Telich-Tarriba JE, Haro-Álvarez EN, Palafox D, et al. Acute traumatic extratemporal facial nerve injury: A 5-year review. *Chinese Journal of Plastic and Reconstructive Surgery*. 2024;**6**(4):161–5.
4. Guntinas-Lichius O, Prengel J, Cohen O, Mäkitie AA, Vander Poorten V, Ronen O, et al. Pathogenesis, diagnosis and therapy of facial synkinesis: A systematic review and clinical practice recommendations by the international head and neck scientific group. *Front Neurol*. 2022;**13**:1019554.
5. Salema HJ, Joshi S, Pawar SR, Nair VS, Koranne V, Limbdiwala ZZ. Marcus Gunn Syndrome - A Rare Neural Misdirection Phenomenon Case Report. *Ann Maxillofac Surg*. 2024;**14**(1):124–6.
6. Varsha V, Ramesh SV, Ramesh PV, Ray P, Devadas AK, Ramesh MK, et al. A Deceptive Wink/Marin–Amat Syndrome, A Clever Lookalike of Inverse Marcus Gunn Syndrome. *tnoa Journal of Ophthalmic Science and Research*. 2023;**61**(2):261-2.
7. Noguchi K, Osawa I, Kurihara S, Yokoyama S, Tanabe Y. Levator aponeurosis advancement with partial orbicularis oculi muscle resection for the treatment of Marin Amat syndrome with aponeurotic ptosis: two Case reports. *Case Reports Plast Surg Hand Surg*. 2025;**12**(1):2451633.
8. Choi D, Raisman G. Disorganization of the facial nucleus after nerve lesioning and regeneration in the rat: effects of transplanting candidate reparative cells to the site of injury. *Neurosurgery*. 2005;**56**(5):1093–100; discussion –100.
9. Crumley RL. Mechanisms of synkinesis. *Laryngoscope*. 1979;**89**(11):1847–54.
10. Celik M, Forta H, Vural C. The development of synkinesis after facial nerve paralysis. *Eur Neurol*. 2000;**43**(3):147–51.
11. Saeedi A, Varelas AN, von Sneidern M, Lee JW. The use of botulinum toxin for oral-ocular synkinesis in facial paralysis. *Operative Techniques in Otolaryngology-Head and Neck Surgery*. 2024;**35**(3):231–6.
12. Martin AJ. Marin-Amat Syndrome: More Than Meets the Eye. *Neurology*. 2023;**100**(3):143.
13. Pavone P, Garozzo R, Trifiletti RR, Parano E. Marin-Amat syndrome: case report and review of the literature. *J Child Neurol*. 1999;**14**(4):266–8.