E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/15.01.121

Case Report

Marin-Amat Syndrome: A Rare Entity

Dr. Kotthinti Anil Kumar[1*], Dr. Uma M.A[2] , Dr.Vennapusa Girish Kumar[3], Dr. Allareddy sathvika[4], Dr. Pillarsetty Pavan Kumar[5], Dr. Nukala s prasad[6]

[1]Post Graduate, Department of General Medicine, PES Institute of Medical Science & Research, Kuppam

[2]Professor and HOD of Medicine, Department of General Medicine, PES Institute of Medical Science & Research, Kuppam.

[3]Post graduate, Department of General Medicine, PES Institute of Medical Science & Research, Kuppam

[4]Post Graduate, Department of General Medicine, PES Institute of Medical Science & Research, Kuppam

[5]Post Graduate, Department of General Medicine, PES Institute of Medical Science & Research, Kuppam

c

***Corresponding Author**: Dr. Kotthinti Anil Kumar, Email: anilkotthinti@gmail.com

Received: 02.03.25, Revised: 22.04.25, Accepted: 19.05.25

ABSTRACT

Marin Amat syndrome is a rare type of synkinesis that occurs when the jaw is fully opened or the involuntary closure of the eyelids during lateral movement of the jaw. It has been suggested that aberrant regeneration of 7 th cranial nerve and incorrect innervation between cranial nerves 5 and 7 may be responsible for this syndrome. In Case report A 60 year old male with no chronic diseases presented with a droopy right eyelid that he developed 1 year after sustaining right-sided Bell's palsy. Since then, the patient has made a reasonable recovery.

INTRODUCTION

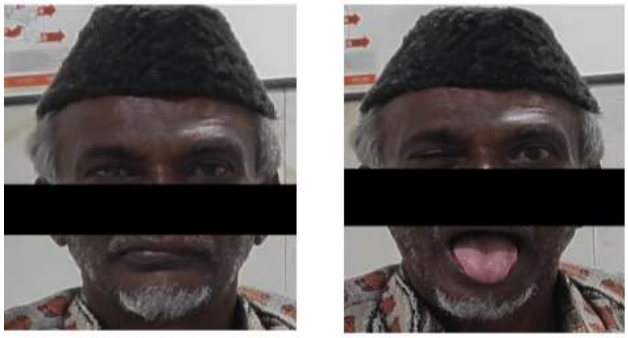
It was first reported by Marin–Amat, a Spanish ophthalmologist, in 1918. It is postulated that the disorder is due to the contraction of the orbicularis oculi muscle (OOM), with muscle stretch acting as a trigger.[1] This was synonymously used with the inverse Marcus Gunn phenomenon, which differs widely in presentation and aetiology. The latter is a congenital phenomenon with misfiring between the third and fifth nerve, involving the levator palpebrae superioris. Inverse Marcus Gunn will have drooping of the lids, whereas Marin–Amat syndrome will have a blepharospasm-like eyelid closure.[2] This shows beyond doubt that understanding the etiopathogenesis precisely is the crux of providing appropriate treatment for these patients. Postparalytic facial synkinesis brings anguish and social awkwardness to patients. Apart from suffering from a motor disease, they also suffer from psychological issues.[3] The patient's well-being and quality of life are dramatically affected, requiring timely management. Botulinum toxin injections are used in the dosage of 2.5 IU per 0.1 ml. In this case, there is closure of lids on opening of the mouth. The main muscle involved in the synkinesis is the orbicularis occuli. Hence, injecting Botox in orbicularis occuli will help in reducing the narrowing of the palpebral aperture.[4]

Case presentation

A 60 year old male with no chronic diseases presented with a droopy right eyelid that he developed 1 year after sustaining right-sided Bell's palsy. Since then, the patient has made a reasonable recovery. However, he noticed an increase of the right eyelid drooping with the jaw movement.

Physical Examination:

The patient showed an asymmetrical face. Right brow ptosis, Bell's phenomenon was positive, and a lid crease was formed. The other cranial nerves were normal after proper examination. When the patient asked to open his mouth, we observed the right eyelid closing involuntary. The observation was similar with the lateral movement of the jaw.



Diagnosis was based on history and clinical examination.

DISCUSSION

Facial synkinesia is defined as involuntary movement in the other part of the face accompanied by voluntary contractions in one part of the face that occurs following atypical reinnervation of the facial nerve. Facial paralysis or facial nerve grafting may cause facial nerve synkinesis.[5] In this case, facial synkinesia occurred approximately 1 year after facial paralysis. However, the development of synkinesia in patients with severe facial paralysis may be earlier. MarcusGunn, Inverse MarcusGunn phenomena and Marin-Amat syndrome are forms of facial synkinesis that can be confused with each other and whose pathophysiological mechanisms are not fully understood.[6] Marcus Gunn phenomenon; is a common synkinesis in patients with congenital ptosis. With the opening of the jaw, the upper eyelid opens and ptosis improves.

It has been shown that there are abnormal connections between the motor branches of the external pterygoid muscle innervating the trigeminal nerve and the superior branch of the occulomotor nerve that innervates the superior of the levator palpebra (false innervation between the 3rd and 5th cranial nerves). [7-9]

In reverse Marcus Gunn syndrome, the upper eyelid closes by opening the jaw. Improper firing between the third and fifth cranial nerves is thought to be responsible for this syndrome. Although it is not clear, it has been suggested that there are abnormal neural connections in the innervation of the superior muscle of the levator palpebra and that the levator palpebral superior is not related to the 3rd nucleus and that the 5th nucleus is innervated by the external pterygoid branches.[5,9] Valve closure mechanism is caused by inhibition of levator palpebra superior rather than orbicularis oculi contraction as seen in Marin-Amat syndrome. The LPS may be linked not only to the third core, but also to the outer pterygoid portion of the fifth core. The exact level of this abnormal connection is controversial.

In Marin-Amat syndrome, blepharospasm occurs in the affected eyelid by opening the jaw or lateral movement. It is usually acquired and develops after peripheral facial paralysis. Marin Amat syndrome has been suggested to result from acquired anastomosis of the trigeminal (CN V) and facial (CN VII) nerve innervating multiple muscle groups.[6] Whether the reverse Marcus Gunn phenomenon and Marin-Amat syndrome is different is controversial. While some authors think that both express the same disorder [3, 4], there are views suggesting that the two syndromes are different.[9] The most common treatment modalities for patients with Marin-Amat syndrome today are: Botulinum toxin type A injection, facial neuromuscular training, biofeedback method, selective neurolysis or myectomy.[10] Botulismus toxin treatment may be beneficial on blepharospasm. [10,11] In our case, it was found that the closure of the left eyelid concomitantly decreased with laughter after low-dose botulinum toxin type A injection. In addition, resection of preseptal orbicularis ocular muscle has been reported to be successful. It is important that Marin-Amat syndrome, a rare synkinesis, be considered in patients with involuntary closure of the eyelid with jaw opening or laughter developing after peripheral paralysis.

REFERENCES

1. Pai PK, Sphoorti PP. Marin Amat syndrome: A clinical case report of a rare syndrome. APIK J Intern Med 2016;4:30.
2. Jethani J. Marin-Amat syndrome: A rare facial synkinesis. Indian J Ophthalmol 2007;55:402-3.
3. Volk GF, Hesse S, Geißler K, Kuttenreich AM, Thielker J, Dobel C, et

al. Role of body dysmorphic disorder in patients with postparalytic facial synkinesis. Laryngoscope 2021;131:E2518-24.
4. Cabin JA, Massry GG, Azizzadeh B. Botulinum toxin in the management of facial paralysis. Curr Opin Otolaryngol Head Neck Surg 2015;23:272-80.
5. Rana PV, Wadia RS. The Marin-Amat syndrome: an unusual facial synkinesia. J Neurol Neurosurg Psychiatry 1985;48:939-941. https://doi.org/10.1136/ jnnp.48.9.939
6. Jethani J. Marin-Amat syndrome: a rare facial synkinesis. Indian J Ophthalmol 2007;55:402-403. https://doi.org/10.4103/0301-4738.33842
7. Pavone P, Garozzo R, Trifiletti RR, Parano E. Marin Amat syndrome: case report and review of the literature. J Child Neurol 1999;14:266-268.
8. Lubkin V. The inverse Marcus Gunn phenomenon. An electromyographie Neurol 1978;35:249. contribution. Arch https://doi.org/10.1001/ archneur.1978.00500280067015
9. Lai CS, Lu SR, Yang SF, et al. Surgical treatment of the synkinetic eyelid closure in Marin-Amat syndrome. Ann Plast Surg 2011;67:498-501. https://doi.org/10.1097/ SAP.0b013e318201fdc3
10. Davis G, Chen C, Selva D. Marcus Gunn syndrome. Eye (Lond) 2004;18:88-90. https://doi.org/10.1038/ sj.eye.6700509