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Case Report

Bilateral anterior opercular syndrome

Mohd Mubarik Naqash[1]*, Feroze Ahmad[2], Arif Nabi Bhat[1], Umar Jalalie[1]

[1]Department of Internal Medicine, SKIMS Medical College, Bemina, Srinagar, Jammu and Kashmir, India
[2]Consultant Medicine, Directorate of Health Services Kashmir, Jammu and Kashmir, India

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***Correspondence:**
Dr. Mohd Mubarik Naqash,
E-mail: mubarikn@rediffmail.com

ABSTRACT

Opercular syndrome, also known as Foix-Chavany-Marie syndrome is a rare disorder due to bilateral lesions of opercular cortex surrounding the insula. It is characterized by paralysis of facial, lingual, pharyngeal, and masticatory voluntary muscles with preservation of autonomic, involuntary, and reflexive functions. In the present case, a 71-years old female presented with acute onset of anarthria with difficulties in chewing, speaking, and swallowing that was diagnosed with opercular syndrome.

Keywords: Bilateral labioglossopharyngeal paresis, Glossopharyngeal diplegia, Opercular syndrome

INTRODUCTION

Bilateral anterior opercular syndrome (AOS) first described by Magnus and is also known as Foix-Chavany-Marie syndrome (FCMS), facio-labio-glosso-pharyngolaryngo- brachial paralysis or cortical type of pseudobulbar paralysis.[1-3] It consists of lower facial and glossopharyngeal diplegia secondary to dysfunction of the rolandic opercula.

The lesions are usually located at the anterior part of the operculum, so it is also called anterior opercular syndrome.[4] It is usually seen in adults. It can result from tumour or infection but is mainly due to successive strokes involving both opercular regions. We report the case of an adult patient in whom AOS developed following bilateral strokes.

CASE REPORT

A 71-years female patient with past history of ischemic stroke 2 years back, manifesting right hemiplegia started suddenly with left faciobrachial paresis secondary to atrial fibrillation of hypertensive origin. Approximately 24 hours after the patient presents with anarthria, facial hypomimia without asymmetries, dysphagia for liquids and bilateral lingual paresis.

Understanding of spoken and written language was maintained and there were no apraxia or agnosia. On neurological examination, her mouth was always open and she drooled continuously. She had bilateral lip, tongue, and pharyngeal weakness with dissociation of automatic and voluntary movements of the lower face (voluntary movements impaired and automatic movements preserved).

Eye closure and extra ocular movements were normal. Corneal reflex was normal. Affect was normal. She had a normal jaw jerk. She was not able to speak, but her comprehension was normal. She had a minimal left upper limb paresis and generalized hyperreflexia, more pronounced on the left. Computed tomography and MRI showed atrophic lesions involving both rolandic opercula (Figure 1). The association of both lesions, mirrored, justified all the symptomatology.

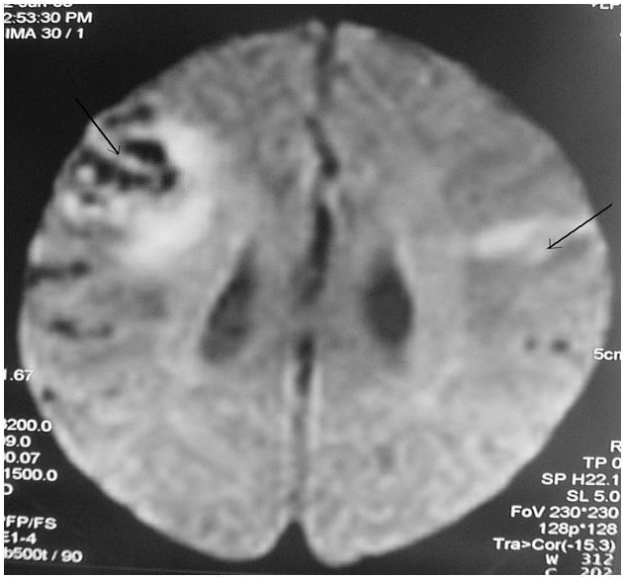

Figure 1: MRI of atrophied lesions in both rolandic opercula.

DISCUSSION

Bilateral facio-pharyngo-laryngo-glosso-masticatory palsy with automatic-voluntary dissociation is known as opercular syndrome. It is usually due to bilateral cortical lesions involving both anterior opercula.[5]

The clinical signs in this patient were consistent with AOS. This condition, may be congenital ,acquired, persistent or intermittent, and includes severe anarthria, loss of voluntary muscular functions of the face and tongue, and impaired mastication and swallowing, with preservation of reflex and autonomic functions.[6] The etiology of AOS is vascular insults in adulthood, such as bilateral subsequent strokes; infections of the CNS, such as herpes simplex encephalitis or acute disseminated encephalomyelitis.[7] It can be congenital, owing to bilateral dysgenesis of the perisylvian region.[8]

Voluntary control of facial muscles is dependent on the normal function of the motor cortex and of the corresponding pyramidal tract. Subcortical structures, particularly basal ganglia, thalamus, and sub thalamic nuclei, are involved in automatic or emotional movement. A bilateral lesion involving the anterior opercular region is manifested by facial diplegia with dissociation of voluntary and involuntary movements.[9] Symmetric lesions of the posterior limbs of both internal capsules may produce a similar picture because the projections from the anterior opercular cortex are represented there. The differential diagnosis of AOS, therefore, is based not only on bulbar dysfunction but also on other clinical features of pseudobulbar palsy.[10] The absence of automatic voluntary dissociation and affective lability point to the presence of AOS.[9] Bilateral stroke is the main etiology of AOS and the two events may be separated in time. A unilateral lesion may lead to AOS in a patient who already has contralateral opercular dysfunction on the other side.

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