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Unilateral Anterior Opercular Syndrome, Due to Cardio-Embolic Stroke: Case Report

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Abstract: ***This study describes a rare case of anterior opercular syndrome (AOS), a cortical form of pseudobulbar paralysis characterized by facio-labio-glosso-pharyngo-laryngeal dysfunction with autonomic-voluntary dissociation. A 74-year-old male presented with acute dysphagia, anarthria, and bilateral facial weakness. Comprehensive clinical, neuroimaging, electrophysiological, and cerebrospinal fluid assessments were conducted, followed by a three-year follow-up. Imaging revealed a unilateral opercular infarction associated with contralateral white matter changes, consistent with a cardio-embolic etiology due to atrial fibrillation. Management included anticoagulation therapy and enteral nutrition via percutaneous endoscopic gastrostomy. Gradual functional improvement was observed over time. This case highlights the diagnostic importance of autonomic-voluntary dissociation and neuroimaging in AOS and emphasizes the need for appropriate secondary stroke prevention strategies.***

Keywords: anterior opercular syndrome, cardio-embolic stroke, atrial fibrillation, stroke prophylaxis, differential diagnosis, Foix-Chavany-Marie syndrome, autonomic-voluntary dissociation

1. Background

Bilateral anterior opercular (Foix–Chavany–Marie) syndrome (AOS) is a rare condition, first described in 1926 (Nowak, 2010). It represents a cortical form of pseudobulbar paralysis (Cascio Rizzo, 2022) and is presented with facio-labio-glosso-pharyngo-laryngeal paralysis with autonomic-voluntary dissociation (Nowak, 2010; García-Grimshaw, 2021; Rivas, 2021; Cascio Rizzo, 2022). In most of the cases, it is vascular in origin and develops because of acute bilateral strokes at middle cerebral artery (MCA) or rarely because of acute unilateral stroke at dominant hemisphere and white matter changes at other (Nowak, 2010; García-Grimshaw, 2021; Rivas, 2021; Cascio Rizzo, 2022), although it can be caused by other factors such as viral encephalitis (Matsushima, 2016), traumatic brain injury (Digby, 2018), multiple sclerosis or other demyelinating central nervous diseases (Muñoz, 2015), neurosurgical complication (Salomi, 2025), etc.

We present a case of 74-year-old male, admitted to our clinic with 24 hours' history of difficulty swallowing, inability to speak, move his tongue and bilateral facial weakness. His previous medical history revealed surgical removal of colon carcinoma, followed by chemotherapy (10 years before) and arterial hypertension. 5 days before, he complained of transient dysarthria and right facial palsy. At the time of hospitalization, he was on good condition, with normal vital signs, fully orientated (he wrote his name, address and the exact date on a piece of paper). His neurological examination revealed bilateral facial palsy (R>L), anarthria, severe dysphagia and inability to move his tongue voluntary. Despite that, his ability for emotional expressions and his cough and autonomic swallowing reflex were preserved. He had no signs of limb palsy and no Babinski sign. He also was able to understand and execute commands and to write, and had normal sensory functions. However, the patient had some episodes of anxiety and emotional lability. His initial head magnetic resonance imaging (MRI, fig.1) showed multiple lesions at subcortical white matter and subacute stroke at left operculum. The patient had normal oto-rhino-laryngological examination, unremarkable thoracic and abdominal computer tomography, electromyography (incl. repetitory nerve stimulation, electroneurography and needle electromyography), blood examinations, Doppler sonography and electrocardiography. His cerebro-spinal fluid examination was also normal. The tests for herpesviruses (CSF), human immunodeficiency virus, neuroboreliosis, parasitic and mycotic infections were negative. We initially started secondary prophylaxis with Acetylsalicylic acid (300mg/d) and statin. 24 Holter-electrocardiography (Holter-ECG) revealed frequent attacks of atrial fibrillation (AF) which we suggested to be the cause of the ischemic stroke and leucoareosis and Apixaban (10mg/d) was initiated. Because of inability to drink, percutaneous endoscopic gastrostomy and enteral tube (PEG) was applied for nutrition and taking his medications. The patient was observed several times over the next 3 years. We found improvement in ability to voluntary move his facial muscles and tongue and to swallow and chew. The PEG was removed at the first year. His speech and emotional state also improved over time. The patient provided written informed consent for the publication of this case report and any accompanying images.

2. Discussion

AOS is due to bilateral damage of inferior parts of frontal and precentral gyrus and their connections with 5, 6, 9, 10, 11 and 12 cranial nerves (Cascio Rizzo, 2022), although modern techniques have revealed the significance of the bilateral injury of the white matter tract, such as the frontal aslant tract (Hayashi, 2019), which is strongly associated with speech and language processing (Zhong, 2022). Thus, the syndrome could be considered also as specific apraxia. The anterior part of the operculum plays role as pre-supplementary motor cortex for face and pharyngo-laryngeal movements and is supplied by multiple perforating arteries branching from the vessels of M2 segment of MCA (Ruan, 2018).

Vascular AOS is divided into classical bilateral AOS (caused by bilateral MCA infarctions) and unilateral AOS, due to opercular stroke at dominant hemisphere plus white matter

changes at subdominant one (Nowak, 2010; Hayashi, 2019; García-Grimshaw, 2021; Rivas, 2021; Cascio Rizzo, 2022). Unlike bilateral ischemic AOS, unilateral one tends to spontaneous improvement over time, probably because of functional remodelation of brain networking (García-Grimshaw, 2021; Rivas, 2021; Cascio Rizzo, 2022). However, such process is relatively slow (about a year for our patient) and such patients require placement of PEG for adequate nutrition.

AOS presents a differential-diagnostic problem, particularly in cases with emergency. It has to be distinguished from specific forms of acute Guillain-Barre syndrome, crises of myasthenia gravis, paraneoplastic syndrome, classical bulbar or pseudobulbar palsy, brainstem encephalitis, brainstem lesion (stroke, tumor, etc.), local throat problem or even allergic reaction with local edema. The keystone for the right syndrome diagnosis is automatic-voluntary dissociation (Hayashi, 2019; Rivas, 2021), but it could be missed during the routine neurological examination. Moreover, AOS could be non-vascular in origin, so other specific etiologies also should be excluded via blood and CSF examinations.

Vascular AOS requires searching for specific vascular risk factors for adequate secondary prophylaxis. In our case the stroke is cardio-embolic, possibly due to newly diagnosed AF so we decided to start oral anticoagulant prophylaxis. However, the treatment of such patients is also a medical challenge (Cho, 2016). Although they have preserved involuntary movements, they have voluntary dysphagia and increased risk for aspiration and malnutrition (Hayashi, 2019) and need PEG enteral tube placement. The problem in such cases is which new oral anticoagulant medication, in particular, should be chosen. Because of good pharmacokinetic profile and ability to be given via enteral tube, we decided to use Apixaban (Peterson, 2016).

3. Conclusions

This case illustrates a rare presentation of unilateral anterior opercular syndrome associated with cardioembolic stroke due to atrial fibrillation. The presence of autonomic-voluntary dissociation and characteristic neuroimaging findings are essential for accurate diagnosis. Early recognition and appropriate management, including anticoagulation and nutritional support, are critical for improving outcomes. Gradual functional recovery may occur over time, highlighting the importance of long-term follow-up and rehabilitation strategies.

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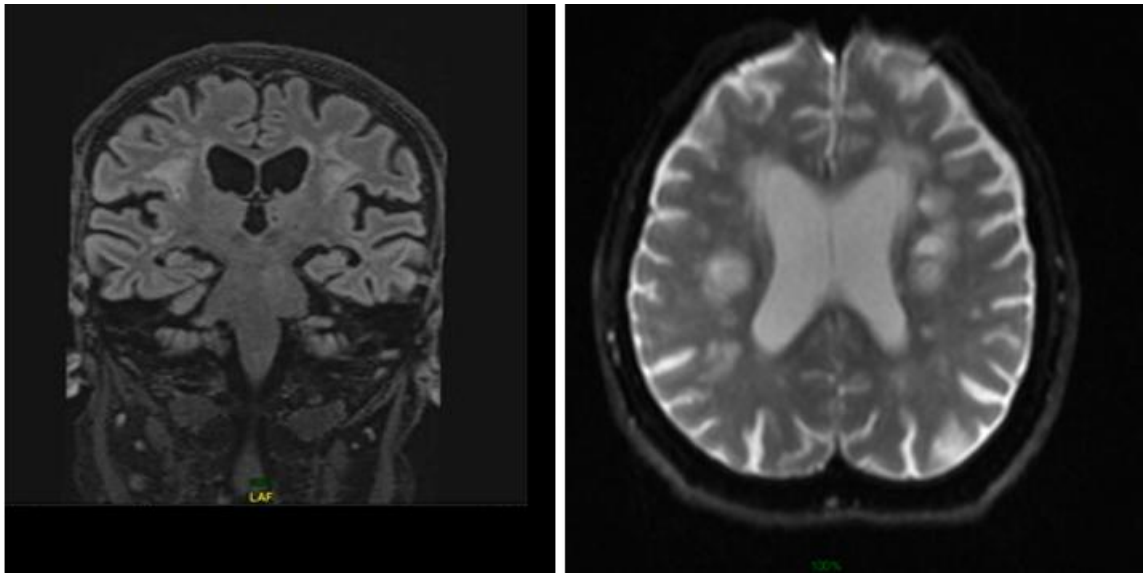

Figure 1: Head MRI of our patient

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