Foix–Chavany–Marie Syndrome Triggered by Acute Unilateral Opercular Infarction in a Patient with a Previous Contralateral Stroke

Sibel Şahin Demirtaş, Caner Baydar*

Department of Neurology, Van Yüzüncü Yıl University Faculty of Medicine, Türkiye

***Correspondence:** Caner Baydar, MD, Department of Neurology, Van Yüzüncü Yıl University Faculty of Medicine, 65090 Van, Türkiye

canerbaydar@yyu.edu.tr

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AUTHORS' CONTRİBUTİONS

Sibel Şahin Demirtaş: Patient evaluation, clinical data collection, literature review, manuscript drafting, and final approval of the manuscript.

Caner Baydar: Study conception and design, supervision, imaging interpretation, manuscript revision, critical intellectual content, and final approval of the manuscript.

All authors have read and approved the final version of the manuscript.

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DİSCLOSURES

The authors declare that they have no financial or non-financial conflicts of interest related to this work.

CONSENT

Yes.

Written informed consent was obtained from the patient for publication of this case report and the accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

HUMAN AND ANİMAL RİGHTS

This study was conducted in accordance with the ethical standards of the institutional and national research committee and with the principles of the Declaration of Helsinki (1975), as revised in 2000 and its subsequent amendments. No experimental intervention involving humans or animals was performed

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ABSTRACT

Foix–Chavany–Marie syndrome is a rare neurological disorder characterized by impaired voluntary movements of the face, tongue, pharynx, and masticatory muscles with preservation of spontaneous and emotional facial movements. We report a 52-year-old woman who presented with sudden anarthria and dysphagia. Brain imaging demonstrated an acute infarction involving the left opercular region together with a chronic infarction in the contralateral frontoparietal region. The diagnosis was established through clinicoradiological correlation. The patient received standard stroke treatment and swallowing rehabilitation. Swallowing function partially improved, whereas severe speech impairment persisted. This case highlights that unilateral opercular infarction may produce the full clinical syndrome in patients with previous contralateral cerebral injury, emphasizing the importance of early recognition and appropriate imaging evaluation.

CASE REPORT

BACKGROUND

Foix–Chavany–Marie syndrome, also known as anterior opercular syndrome, is an uncommon cortical disorder caused by disruption of the corticobulbar pathways. It classically results from bilateral opercular lesions, most frequently secondary to ischemic stroke, and is characterized by automatic–voluntary dissociation, in which voluntary movements of the face, tongue, pharynx, and masticatory muscles are lost while spontaneous and emotional movements remain intact. Unilateral opercular infarction is an unusual cause of the syndrome and is typically associated with a pre-existing contralateral cerebral lesion. Because of its rarity and distinctive clinicoradiological presentation, recognition of this entity is essential for accurate diagnosis, appropriate management, and differentiation from brainstem and neuromuscular disorders.

CASE REPORT

Clinical Presentation

A 52-year-old right-handed woman presented to our stroke unit approximately 48 hours after the sudden onset of anarthria and dysphagia. Her medical history included hypertension, diabetes mellitus, active cigarette smoking, uterine myoma, and two previous ischemic strokes. Before the current event, she was functionally independent with a premorbid modified Rankin Scale score of 1.

On admission, she was alert, fully oriented, and able to understand spoken language and follow verbal commands. Her National Institutes of Health Stroke Scale score was 3.

Neurological examination demonstrated complete loss of voluntary lower facial, lingual, and pharyngeal movements. She was unable to open her mouth on command, protrude her tongue, purse her lips, or voluntarily close her eyes. In contrast, spontaneous and emotional facial movements, including smiling, yawning, and blinking, were preserved, demonstrating the characteristic automatic–voluntary dissociation. Corneal reflexes remained intact, whereas gag and swallowing reflexes were markedly diminished. Mild facial asymmetry was present without limb weakness, sensory deficits, cerebellar dysfunction, or aphasia.

Laboratory investigations demonstrated severe iron-deficiency anemia (hemoglobin 6.8 g/dL, serum iron 15 μg/dL, total iron-binding capacity 409 μg/dL). Routine biochemical investigations, thyroid function tests, electrocardiography, transthoracic echocardiography, and 24-hour Holter monitoring showed no alternative etiology for the acute neurological presentation.

Imaging Findings

Non-contrast cranial computed tomography demonstrated chronic encephalomalacia involving the right frontoparietal region (Figure 1A). Fluid-attenuated inversion recovery magnetic resonance imaging demonstrated hyperintense signal involving the left opercular cortex with extension into the adjacent insular cortex, superior temporal gyrus, and perisylvian region (Figure 1B,1C). Diffusion-weighted magnetic resonance imaging confirmed an acute ischemic infarction involving the same anatomical regions by demonstrating marked diffusion restriction (Figure 1D).

Computed tomography angiography demonstrated non-obstructive atherosclerotic plaques within the left internal carotid artery without hemodynamically significant stenosis

Management

Because the patient presented beyond the recommended therapeutic window, reperfusion therapy, including intravenous thrombolysis and mechanical thrombectomy, was not indicated. Standard medical management consisted of antiplatelet therapy with aspirin, low-molecular-weight heparin, and comprehensive stroke care. Owing to severe dysphagia, enteral nutrition was initiated through a nasogastric tube, and multidisciplinary speech and swallowing rehabilitation was started during hospitalization.

Follow-up

At hospital discharge, the patient remained anarthric with persistent dysphagia (modified Rankin Scale score = 4). At the one-month follow-up visit, swallowing function had moderately improved; however, severe speech impairment persisted, and the modified Rankin Scale score improved to 3

DİSCUSSİON

Etiology & Demographics

Foix–Chavany–Marie syndrome (FCMS), also known as anterior opercular syndrome, is an uncommon cortical form of pseudobulbar palsy resulting from disruption of the bilateral corticobulbar pathways controlling voluntary movements of the facial, lingual, pharyngeal, and masticatory muscles [1-3]. Most reported cases occur after bilateral opercular infarctions, whereas unilateral opercular lesions account for only a small minority of published cases. In addition to ischemic stroke, less common etiologies include multiple sclerosis, frontotemporal lobar degeneration, infections, tumors, trauma, and congenital bilateral perisylvian syndromes [1-6]. Because of its rarity, most available evidence originates from isolated case reports and small case series, with ischemic stroke representing the most frequent etiology [1-4,7-12]. Recent reports have further demonstrated that unilateral opercular infarction may produce the complete syndrome in the presence of a previous contralateral cerebral lesion, supporting the concept that functional disruption of the bilateral corticobulbar network, rather than anatomically bilateral opercular destruction alone, is sufficient to produce FCMS [9-12,15,16].

Clinical & Imaging Findings

Automatic–voluntary dissociation remains the hallmark clinical feature of FCMS and represents the most reliable bedside clue for diagnosis [1-3]. Voluntary corticobulbar motor control is impaired, whereas spontaneous and emotional facial movements remain preserved through alternative extrapyramidal pathways involving limbic and brainstem circuits [3]. This characteristic dissociation helps distinguish FCMS from brainstem stroke, bulbar palsy, and neuromuscular disorders, in which both voluntary and involuntary movements are typically affected [3,10-12].

Neuroimaging plays a pivotal role in confirming the diagnosis and excluding alternative structural lesions. Diffusion-weighted magnetic resonance imaging is particularly valuable for detecting acute opercular infarction, whereas computed tomography may demonstrate chronic contralateral lesions that

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explain the bilateral functional disruption responsible for the syndrome [9-13]. In our patient, diffusion-weighted magnetic resonance imaging demonstrated an acute left opercular infarction, whereas computed tomography documented a chronic contralateral infarction, illustrating the bilateral functional network disruption emphasized in recent reports [9-12,15,16]. These imaging findings closely correlated with the patient's characteristic neurological examination, allowing a confident clinicoradiological diagnosis of FCMS.

Treatment & Prognosis

There is no disease-specific treatment for Foix–Chavany–Marie syndrome (FCMS), and management is primarily directed toward the underlying etiology together with supportive multidisciplinary rehabilitation [13,14]. In patients with ischemic stroke, treatment should follow current stroke guidelines, including reperfusion therapy when indicated and secondary prevention strategies thereafter [13]. Early recognition of dysphagia, prompt nutritional support, and intensive speech and swallowing rehabilitation remain the cornerstones of management because aspiration pneumonia is a major contributor to post-stroke morbidity and mortality [13,14]. Consistent with previously published cases, our patient demonstrated partial recovery of swallowing despite persistent severe anarthria, highlighting that swallowing function may improve with rehabilitation, whereas speech impairment often remains the most disabling long-term deficit [9-14].

Differential Diagnoses

The principal differential diagnoses of FCMS include bulbar palsy secondary to brainstem infarction, pseudobulbar palsy caused by diffuse bilateral cerebral lesions, motor neuron disease, myasthenia gravis, and other neuromuscular disorders presenting with dysarthria and dysphagia [3,10-12]. Careful neurological examination remains fundamental for distinguishing these conditions. The presence of automatic–voluntary dissociation, preserved emotional facial movements, intact language comprehension, and the absence of significant limb weakness strongly favor a cortical opercular lesion rather than brainstem or neuromuscular pathology [3,10-12]. Diffusion-weighted magnetic resonance imaging plays a pivotal role in confirming the diagnosis by demonstrating acute opercular infarction while excluding alternative structural lesions [9-12,16].

The present case further supports the concept that unilateral opercular infarction may produce the complete clinical syndrome in the presence of a previous contralateral cerebral lesion. Early recognition of automatic–voluntary dissociation together with appropriate neuroimaging facilitates accurate diagnosis, prevents misdiagnosis as brainstem stroke or neuromuscular disease, and allows timely initiation of supportive multidisciplinary management (Tables 1,2).

TEACHİNG POİNT

Foix–Chavany–Marie syndrome should be suspected in patients with acute anarthria and dysphagia despite preserved spontaneous facial movements. Diffusion-weighted magnetic resonance imaging demonstrating opercular infarction, together with recognition of automatic–voluntary dissociation, is essential for accurate diagnosis and differentiation from brainstem and neuromuscular disorders.

QUESTIONS

Question 1: Which of the following clinical findings is the hallmark feature of Foix–Chavany–Marie syndrome?

1. Bilateral facial paralysis
2. Limb ataxia
3. Automatic–voluntary dissociation (applies)
4. Visual field defect
5. Hemiplegia

Explanation

1. Bilateral facial paralysis is not the defining feature of Foix–Chavany–Marie syndrome.
2. Limb ataxia is not a characteristic manifestation of the syndrome.
3. Automatic–voluntary dissociation is the hallmark clinical feature, characterized by impaired voluntary facial, lingual, pharyngeal, and masticatory movements despite preserved spontaneous and emotional facial movements. [Automatic–voluntary dissociation remains the hallmark clinical feature of FCMS and represents the most reliable bedside clue for diagnosis.]
4. Visual field defects are unrelated to the syndrome.
5. Hemiplegia may accompany stroke but is not the defining feature of Foix–Chavany–Marie syndrome.

Question 2: Which imaging modality is most useful for confirming acute opercular infarction in Foix–Chavany–Marie syndrome?

1. Skull radiography
2. Carotid ultrasonography
3. Diffusion-weighted magnetic resonance imaging (applies)
4. Positron emission tomography
5. Electroencephalography

Explanation

1. Skull radiography has no role in diagnosing opercular infarction.
2. Carotid ultrasonography evaluates extracranial vessels but cannot demonstrate cortical infarction.
3. Diffusion-weighted magnetic resonance imaging is the most sensitive imaging modality for detecting acute opercular infarction. [Diffusion-weighted magnetic resonance imaging is particularly valuable for detecting acute opercular infarction.]
4. Positron emission tomography is not routinely used for diagnosing acute ischemic stroke.
5. Electroencephalography evaluates cortical electrical activity rather than ischemic lesions.

Question 3: Which statement regarding the etiology of Foix–Chavany–Marie syndrome is correct?

1. It is exclusively caused by bilateral opercular infarction.
2. Ischemic stroke is the most common cause, although several other etiologies have been reported. (applies)

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3. It occurs only in congenital brain malformations.
4. It is caused exclusively by multiple sclerosis.
5. It is always associated with brainstem infarction.

Explanation

1. Bilateral opercular infarction is the classic cause but is not the only etiology.
2. Ischemic stroke is the most frequent cause; however, multiple sclerosis, frontotemporal lobar degeneration, tumors, trauma, infections, and congenital disorders have also been reported. [Because of its rarity, most available evidence originates from isolated case reports and small case series, with ischemic stroke representing the most frequent etiology.]
3. Congenital bilateral perisylvian syndrome is only one of several reported causes.
4. Multiple sclerosis is an uncommon etiology.
5. Brainstem infarction is a differential diagnosis rather than a cause of Foix–Chavany–Marie syndrome.

Question 4: Which management strategy is most important for reducing complications in patients with Foix–Chavany–Marie syndrome and severe dysphagia?

1. Immediate corticosteroid therapy
2. Deep brain stimulation
3. Speech and swallowing rehabilitation together with nutritional support (applies)
4. Whole-brain radiotherapy
5. Intravenous immunoglobulin

Explanation

1. Corticosteroids are not standard treatment for ischemic Foix–Chavany–Marie syndrome.
2. Deep brain stimulation has no established role.
3. Early nutritional support and multidisciplinary speech and swallowing rehabilitation are essential to reduce aspiration-related complications. [Early recognition of dysphagia, prompt nutritional support, and intensive speech and swallowing rehabilitation remain the cornerstones of management because aspiration pneumonia is a major contributor to post-stroke morbidity and mortality.]
4. Whole-brain radiotherapy has no role.
5. Intravenous immunoglobulin is not indicated in ischemic Foix–Chavany–Marie syndrome.

Question 5: Which combination of clinical and imaging findings most strongly supports the diagnosis of Foix–Chavany–Marie syndrome?

1. Brainstem infarction with complete facial paralysis
2. Automatic–voluntary dissociation with diffusion-weighted magnetic resonance imaging demonstrating opercular infarction (applies)
3. Normal magnetic resonance imaging with dysarthria
4. Peripheral facial nerve palsy with temporal bone fracture
5. Bilateral occipital infarctions with visual loss

Explanation

1. Brainstem infarction is an important differential diagnosis but does not produce the characteristic clinicoradiological findings of Foix–Chavany–Marie syndrome.
2. The diagnosis is established by correlating automatic–voluntary dissociation with diffusion-weighted magnetic resonance imaging demonstrating opercular infarction. [Early recognition of automatic–voluntary dissociation together with appropriate neuroimaging facilitates accurate diagnosis, prevents misdiagnosis as brainstem stroke or neuromuscular disease, and allows timely initiation of supportive multidisciplinary management.]
3. Normal magnetic resonance imaging would not support the diagnosis of acute opercular infarction.
4. Peripheral facial nerve palsy lacks the characteristic automatic–voluntary dissociation.
5. Occipital infarction primarily causes visual deficits rather than corticobulbar dysfunction.

REFERENCES

[1] Gordon N. The opercular syndrome, or Foix-Chavany-Marie syndrome. *J R Coll Physicians Edinb.* 2007; 37(2): 103-106.

[2] Bakar M, Kirshner HS, Niaz F. The opercular-subopercular syndrome: four cases with review of the literature. *Behav Neurol.* 1998; 11(2): 97-103. PMID: 11568407.

[3] Weller M. Anterior opercular cortex lesions cause dissociated lower cranial nerve palsies and anarthria but no aphasia: Foix-Chavany-Marie syndrome and automatic-voluntary dissociation revisited. *J Neurol.* 1993; 240(4): 199-208. PMID: 7684439.

[4] Posteraro L, Pezzoni F, Varalda E, Bottacchi E, Beccaria M. A case of unilateral opercular syndrome (Foix-Chavany-Marie syndrome). *J Neurol.* 1991; 238(6): 337-339.

[5] Pender MP, Ferguson SM. Dysarthria and dysphagia due to the opercular syndrome in multiple sclerosis. *Mult Scler.* 2007; 13(6): 817-819. PMID: 17613613.

[6] Otsuki M, Nakagawa Y, Mori F, et al. Progressive anterior opercular syndrome due to FTLD-TDP: a clinicopathological investigation. *J Neurol*. 2010; 257(7): 1148-1153. PMID: 20177696.

[7] Kutluay E, Colakoglu Z, Dirik A, Kumral K. Brain SPECT in anterior opercular syndrome due to a unilateral lesion. *J Neurol.* 1996; 243(5): 427-429. PMID: 8741087.

[8] Baydar C, Güneş HN, Şener U, Yoldaş TK. Opercular syndrome due to a unilateral opercular infarction. *Arch Neuropsychiatry.* 2012; 49: 74-76.

[9] Sá F, Menezes Cordeiro I, Mestre S, Nzwalo H. Unilateral opercular infarction presenting with Foix-Chavany-Marie syndrome. *BMJ Case Rep.* 2014; 2014: bcr2014206439. PMID: 25427932.

[10] Yoshii F, Sugiyama H, Kodama K, Irino T. Foix-Chavany-Marie syndrome due to unilateral anterior opercular damage with contralateral infarction of the corona radiata. *Case Rep Neurol.* 2019; 11(3): 319-324. PMID: 31824287.

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[11] Toledo-Trevino R, Manrique-Otero D, Castellanos-Pedroza E, et al. Foix-Chavany-Marie syndrome as a manifestation of unilateral opercular stroke. *Eur J Case Rep Intern Med.* 2021; 8(4): 002462. PMID: 33987126.

[12] Yadav PS, Shah L, Rayamajhi A, et al. Foix-Chavany-Marie syndrome due to unilateral opercular infarction: a case report. *Clin Case Rep.* 2024; 12(1): e8392. PMID: 38173897.

[13] Powers WJ, Rabinstein AA, Ackerson T, et al. 2019 Guidelines for the Early Management of Patients With Acute Ischemic Stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke.* 2019; 50(12): e344-e418. PMID: 31662037.

[14] Martino R, Foley N, Bhogal S, Diamant N, Speechley M, Teasell R. Dysphagia after stroke: incidence, diagnosis, and pulmonary complications. *Stroke.* 2005; 36(12): 2756-2763. PMID: 16269630.

[15] Rivas K, Pan J, Julayanont P. Foix-Chavany-Marie syndrome caused by unilateral frontal opercular infarction. *Neurology.* 2021; 96(15 Suppl): 2628.

[16] Panchal A, Rohatgi A, Verma P, et al. Opercular syndrome without involvement of opercular area: an uncommon presentation of stroke. *J Assoc Physicians India.* 2025; 73(1): 85-86. PMID: 39893537.

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FIGURES

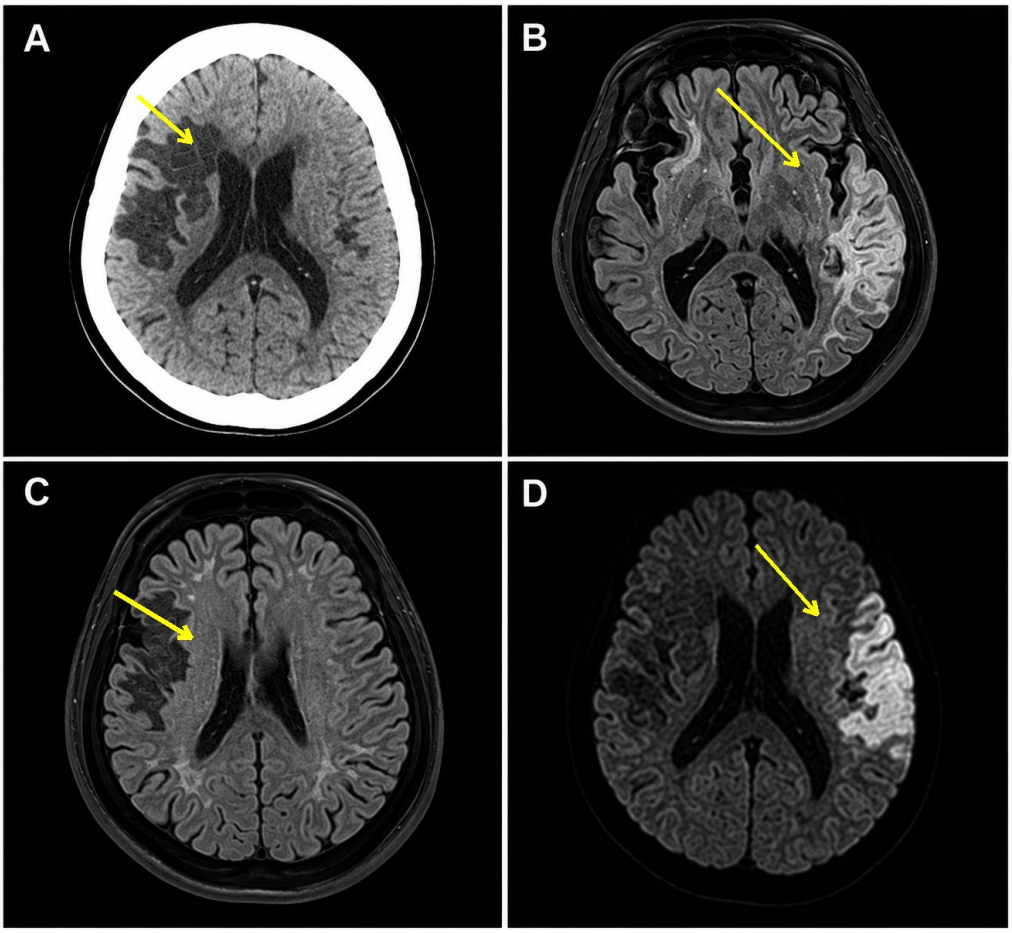

Figure 1: Clinicoradiological imaging findings of Foix–Chavany–Marie syndrome secondary to acute unilateral opercular infarction.

Age, Gender, Diagnosis

52-year-old woman with Foix–Chavany–Marie syndrome secondary to an acute left opercular infarction in the presence of a chronic contralateral right opercular infarction.

Findings

(A) Axial non-contrast brain computed tomography (CT) demonstrates chronic encephalomalacia involving the right frontal opercular cortex (arrow), consistent with a previous ischemic infarction.

(B) Axial fluid-attenuated inversion recovery (FLAIR) magnetic resonance (MR) image demonstrates cortical and subcortical hyperintensity involving the left frontal opercular cortex with extension into the adjacent insular cortex (arrow), compatible with an acute ischemic infarction.

(C) Axial FLAIR MR image obtained at a slightly superior level again demonstrates chronic encephalomalacia involving the right frontal opercular cortex (arrow), confirming previous contralateral cerebral injury.

(D) Axial diffusion-weighted magnetic resonance (DWI) image demonstrates marked diffusion restriction involving the left frontal opercular cortex and adjacent insular cortex (arrow), confirming an acute ischemic infarction. The combination of chronic right opercular encephalomalacia and acute left opercular infarction explains the bilateral functional corticobulbar disruption responsible for Foix–Chavany–Marie syndrome and correlates closely with the characteristic automatic–voluntary dissociation observed on neurological examination.

Technique

(A) Axial non-contrast brain computed tomography was performed as part of the routine emergency evaluation for acute ischemic stroke.

(B, C) Axial brain magnetic resonance imaging was performed on a 1.5-T GE magnetic resonance scanner using a routine fluid-attenuated inversion recovery (FLAIR) sequence.

(D) Axial diffusion-weighted magnetic resonance imaging (DWI) was acquired on the same 1.5-T GE magnetic resonance scanner using a standard acute stroke imaging protocol.

The imaging protocol included axial diffusion-weighted imaging and fluid-attenuated inversion recovery sequences for the assessment of acute cerebral ischemia.

All imaging examinations were obtained as part of the routine diagnostic evaluation of acute ischemic stroke. Imaging findings were interpreted in conjunction with the neurological examination to establish the clinicoradiological diagnosis of Foix–Chavany–Marie syndrome.

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Table 1: Summary Table
Summary of the epidemiology, clinical features, imaging findings, treatment, and prognosis of Foix–Chavany–Marie syndrome based on the current literature.

Feature	Findings
Disease	Foix–Chavany–Marie syndrome (Anterior opercular syndrome)
Etiology	Usually bilateral opercular infarction; less commonly unilateral opercular infarction with previous contralateral cerebral injury, multiple sclerosis, FTLD, tumors, infections, trauma, congenital bilateral perisylvian syndrome.
Incidence	Extremely rare; exact incidence unknown.
Gender ratio	No definite sex predilection.
Age predilection	Middle-aged/elderly adults (vascular); childhood in congenital forms.
Risk factors	Hypertension, diabetes, smoking, atherosclerosis, previous stroke.
Pathophysiology	Functional bilateral corticobulbar pathway disruption causing automatic–voluntary dissociation.
Clinical findings	Anarthria/dysarthria, dysphagia, impaired voluntary facial-lingual-pharyngeal movements with preserved emotional facial movements.
CT	Chronic opercular encephalomalacia; acute changes may be subtle.
MRI T1	Hypointense opercular lesion.
MRI T2/FLAIR	Hyperintense opercular lesion; chronic contralateral encephalomalacia.
MRI DWI	Restricted diffusion in acute infarction.
Contrast enhancement	Usually absent.
Treatment	Stroke management, secondary prevention, nutritional support, speech/swallow rehabilitation.
Prognosis	Swallowing often improves; speech deficit may persist.

Table 2: Differential Diagnosis of Foix–Chavany–Marie Syndrome
Differential diagnosis of Foix–Chavany–Marie syndrome with key imaging findings.

Disease	CT	MRI T2/FLAIR	MRI DWI	Enhancement	Key distinguishing feature
FCMS	Opercular infarct/ encephalomalacia	Opercular hyperintensity	Restricted diffusion if acute	None	Automatic–voluntary dissociation
Brainstem infarction	Brainstem hypodensity (late)	Brainstem lesion	Restricted diffusion	None	Brainstem signs
Pseudobulbar palsy	Diffuse bilateral lesions	White matter/cortical lesions	Usually absent	Variable	No automatic–voluntary dissociation
Motor neuron disease	Usually normal	Possible corticospinal tract signal	Absent	None	Progressive LMN/ UMN signs
Myasthenia gravis	Normal	Normal	Normal	None	Fatigable weakness; normal brain MRI

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KEYWORDS

Foix–Chavany–Marie syndrome; Anterior opercular syndrome; Opercular infarction; Automatic–voluntary dissociation; Magnetic resonance imaging

ABBREVIATIONS

ADC = Apparent Diffusion Coefficient
CT = Computed Tomography
DWI = Diffusion-Weighted Imaging
FCMS = Foix Chavany Marie Syndrome
FLAIR = Fluid-Attenuated Inversion Recovery
MRI = Magnetic Resonance Imaging
NIHSS = National Institutes of Health Stroke Scale
MRS = Modified Rankin Scale

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