

## Subcortical Aphasia

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### Abstract

Aphasia, a language deficit caused by an acquired cerebral lesion was initially believed to be triggered exclusively by cortical damage. However, recent findings have confirmed that aphasia can also arise from subcortical lesions. This paper presents a case of a 53-year-old man with acquired language impairment associated with a lesion in the left lenticular nucleus caused by a hemorrhagic cerebrovascular event. The patient exhibited clinical and imaging characteristics consistent with subcortical aphasia of the thalamic and striato-capsular type. Subcortical structures are increasingly recognized for their involvement in language processing, alongside the cerebral cortex. Despite current acceptance of subcortical aphasia, there are limited studies on this type of aphasia and its interlinguistic variations. Consequently, extensive research is warranted to gain a better understanding of it as a clinical entity.

### Introduction

Aphasia is defined as a language deficit caused by an acquired cerebral lesion [1]. Despite the cerebral cortex being considered the main anatomical structure implicated in language for a long time, nowadays there is growing evidence that this function is not exclusively limited to the cortex, but also involves several subcortical structures [2].

Wernicke introduced the term “subcortical aphasia” in the 19th century in his classical classification. However, other authors refuted his claim, asserting that damage to these structures would result in dysarthria rather than aphasia [3]. It was not until the 20th century, with the introduction of the computed tomography, that it was confirmed that aphasia resulting from subcortical lesions is possible [4]

The substantia nigra, globus pallidus, lenticular nucleus, and subthalamic nuclei have classically been described with important motor functions. Nevertheless, more recently, they have also been recognized with significant non motor functions that are closely linked to speech and language [2].

In this paper, we present the case of a 53-year-old man with acquired language impairment, whose onset was associated with a lesion in the left lenticular nucleus caused by a hemorrhagic cerebrovascular event.

### Case Report

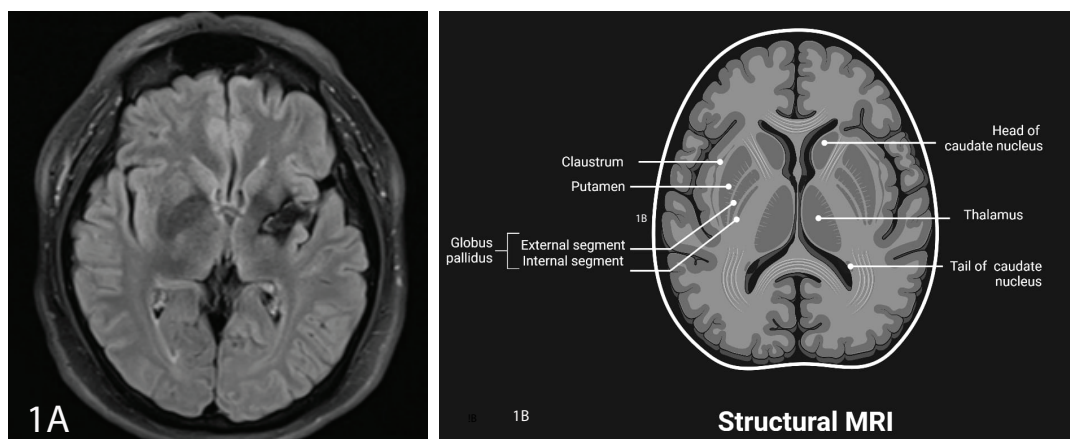
A 53-year-old man arrives, complaining of a language problem characterized by anomia and intermittent articulation deficit, especially in stressful situations and during rapid speech. The man is currently retired, previously worked as an accountant and has university education. He has a history with systemic arterial hypertension and experienced a hypertension-related hemorrhagic cerebrovascular event 3 years ago, which affected the left basal ganglia and debuted with right hemiparesis, gait instability, dysarthria and amnesia. He is currently being treated with nifedipine, telmisartan, pravastatin, and acetylsalicylic acid.

The physical exam reveals semi-fluent spontaneous language, with appropriate cohesion, coherence and meaning. Further examination unfolds occasional anomia, perseveration and semantic paraphasia. A NEUROPSI test was performed, with the following results:

**Orientation:** Oriented in person and space. Oriented in day and in month but disoriented regarding the year.

**Attention and concentration:** Can perform the 4-element regressive digit test and achieved 13/16 correct answers in the visual detection tests. However, can't complete a three-by-three regressive digit cut out from 20 to 0, responding only 2 numbers correctly.

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**Figure 1A)** Axial MRI of the skull in T2-dark fluid sequence. Area of gliosis and encephalomalacia towards the deep left frontal white matter; involving the left thalamus and lentiform nucleus. **1B)** Representative anatomical image of the basal ganglia in an axial cranial MRI section. Image credits: Created with Biorender.com.

**Codification:** Spontaneous verbal memory with a rising learning curve, being able to retain 5/6 elements in the second and third attempt. Can also perform visuo-spatial processes in an organized manner, as evident from the semi-complex figure copying task, maintain balanced proportions and achieving a score of 9.5/12.

**Language:** Adequate denomination and repetition for words, phrases and sentences. Appropriate comprehension for simple orders but difficulty with complex and logical-grammatical orders. Achieves 22 elements in semantic verbal fluency task. However, only 6 elements in the fluency task for phoneme /f/ and displays 5 perseverations.

**Reading:** Reads polysyllabic words, with adequate diction. Satisfactory reading comprehension.

**Writing:** Can copy and write from dictation.

**Executive function:** Appropriate handling of similarities. Capable of simple arithmetic calculations but not complex ones. Additionally, cannot perform complex sequencing tasks. Regarding motor functions, the individual is unable to correctly perform standardized hand movement tasks. Alternating hand movements are non-automatic and performs opposite reactions with errors.

**Evocative function:** Completes the visuo-spacial memory task with a 7/12 score. Recalls 5/6 elements in the spontaneous verbal memory task, 4/6 elements in the verbal memory task with hints, and 5/6 elements in the verbal memory task by recognition.

The NEUROPSI results, corrected by education level, indicate that the patient has a mild deficit for neuropsychological performance.

Additionally, Western aphasia tests were conducted with the following results:

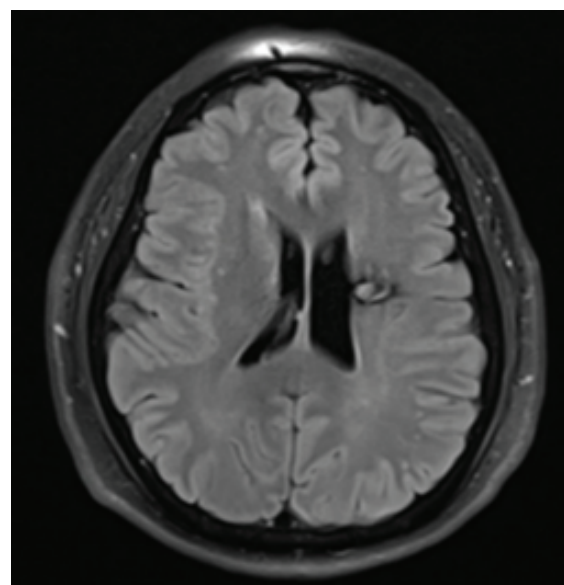
**Spontaneous language:** Language fluency is compromised, with occasional grammar mistakes, articulation disturbances and undesired pauses despite regular use of complex sentences. Has adequate prosody and can answer all of the items correctly. When describing an image, he uses complex and coherent sentences, but sporadic anomias and semantic paraphasias are present. These findings align with level 4 in the Goodglass and Kaplan severity scale, indicating an obvious alteration of speech fluency and comprehension, without significant limitation in expressing ideas.

**Verbal auditory comprehension:** Performs simple, complex and some logical-grammatical commands. Normal verbal auditory recognition for real objects, drawn objects, shapes, letters, numbers, furniture, body parts, and fingers. Can differentiate right from left. Exhibits fluid performance for simple sequential commands but experiences difficulty in executing complex commands.

**Repetition:** Repeats words and short phrases, but is unable to repeat long sentences.

**Naming:** Appropriate. Demonstrates normal semantic fluency, completes sentences, and answers questions.

**Praxis:** Praxis for upper limb, face movements, instrument use, and complex movements is preserved.



**Figure 2.** Axial MRI of the skull in T2-dark fluid sequence. Multiple hyperintense nodular images towards the subcortical white matter associated with hemosiderin deposits. No findings to report in the cranial nerve sequence. Additionally, chronic microangiopathic damage was observed.

Due to the aforementioned data, a simple and contrast-enhanced magnetic resonance imaging (MRI) of the skull (Figures 1 and 2) was requested, revealing suggestive findings of the late-stage intracranial parenchymal hemorrhage in the area of the left basal ganglia.

Findings were consistent with diagnosis of subcortical striatocapsular/thalamic aphasia. The patient was sent to the speech and language pathology department for rehabilitation.

**Discussion**

In recent years, modern neuroscience has emphasized the role of functional brain networks rather than solely focusing on localized areas. These networks consist of numerous interconnected functional regions, with strong connections between the basal ganglia (Figure 3) and cortical areas through five basal ganglia-thalamo-cortical circuits. Consequently, disruptions in this intercommunication can lead to disturbances in higher cortical functions such as language [2].

Subcortical aphasia has been classified into three types by some authors based on lesions observed in imaging studies: striatocapsular aphasia, periventricular white matter-related aphasia and thalamic aphasia.

A study on subcortical aphasia conducted by Lahiri et al. concluded that the most frequently affected location was the putamen (53.23%), followed by the striatocapsular region

(33.87%). On the other hand, aphasia due to periventricular white matter lesions (6.45%) and thalamic lesions (6.45%) were less common. In general, the clinical presentation of subcortical aphasia consists of preserved repetition. However, all three subgroups exhibit characteristics of some degree of language impairment [3].

The researchers also observed a strong predominance towards non-fluent forms of aphasia in subcortical infarctions, with global aphasia being predominant in initial evaluation, while Broca's aphasia was frequently among the follow-up cases [3].

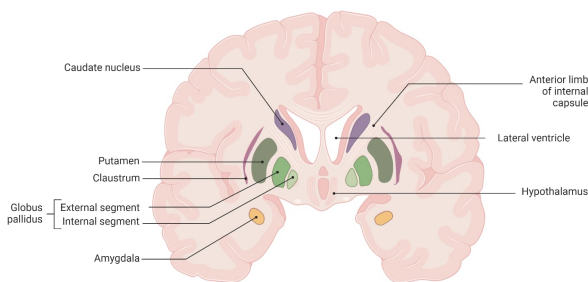
This same author mentions that striatocapsular aphasia and aphasia related to periventricular white matter lesions are mainly characterized by a loss of language fluency, appearance of literal paraphasias with preservation of comprehension and naming. Recently, Bouvier et al. conducted a literature review describing language impairments after subcortical cerebrovascular accident, without thalamic involvement, affecting the basal ganglia and surrounding white matter. Based on a selection of 22 articles involving 114 participants, the results suggested a predominance of deficit in more demanding linguistic levels (language alterations at the discourse and syntactic level) and in language production (in contrast to comprehension, which is more frequently affected in thalamic conditions). The prognosis indicated rapid recovery, especially for lexical-semantic and receptive impairments [3].

Thalamic aphasia, on the other hand, is characterized by preserved fluency but exhibits alteration in comprehension and naming, with a predominance of verbal paraphasias [3]. According to a study conducted by Fritsch et al. thalamo-cortical language networks, specifically encompassing the nuclei in the left anterior thalamus, play a crucial role in integrating information from the left cortex. Disconnection in these networks can lead to aphasic symptoms [5]. The initial language abnormality is mutism, which typically progresses to verbose and paraphasic output, albeit with hypophonic speech. Anomia is frequently observed and often severe, accompanied by significant impairment of comprehension. Thalamic aphasia commonly observed in cases where there is a pathology affecting the left pulvinar nucleus [3].

Aphasiology has been a field of study focused on the examination of language impairment resulting from acquired brain injuries. Formal tests with approved psychometric studies have been developed, such as the Boston Diagnostic Aphasia Examination, the Western Aphasia Battery, the Battery for Assessment of Aphasic Disorders (BETA), as well as the Barcelona Test. Although the latter ones have been designed according to a more recent linguistic processing model, their assessment domains still primarily focus on linguistic structure. In this case, we decided to conduct the patient's assessment using the Western Aphasia Battery test [6].

The Neuropsi test is a brief neuropsychological assessment developed in Mexico that is objective and reliable. It allows us to assess cognitive processes in neurological and psychiatric patients. This test comprises a series of assessments aimed at understanding overall cognitive functioning. It includes tests for orientation, attention, memory, language, visuospatial skills, and visuo cognitive abilities in a wide range of age groups [7].

Our patient exhibited a partially fluent language with verbal stereotypes, perseverations, and intermittent articulatory alterations, mainly affecting compound syllables. These symptoms intensified during spontaneous speech, particularly when asked about the situation that led to his visit and while



**Figure 3.** Basal ganglia in a coronal section. Image credits: Created with Biorender.com.

**Table 1.** Main characteristics of different types of subcortical aphasia

<b>Subcortical aphasia</b>	<b>Striato-capsular / Periventricular white matter</b>
	Non-fluent language Preserved comprehension Preserved naming Altered morphosyntax
	<b>Thalamic</b>
	Fluent language Verbal paraphasias Altered comprehension Altered naming

describing an image while using complex sentences. Such alterations were expected due to the lesion's location observed in the MRI. However, during the continuation of the Western Aphasia Battery test, unexpected language impairments emerged, including difficulties in comprehending complex and logical-grammatical commands, as well as occasional anomias and semantic paraphasias.

Radanovic et al. propose that lesions in the basal ganglia result from occlusion of the initial segment of the dominant middle cerebral artery, or occasionally the internal carotid artery. Consequently, aphasia occurs due to ischemic neuronal death in the white matter pathways connecting the thalamus to the fronto-subcortical-temporal language network, leading to additional aphasic symptoms as a consequence of thalamic disconnection. Several authors argue that a clinically uniform and persistent condition defined as "subcortical aphasia" can only be consistently observed in cases of striato-capsular lesions [3].

Xu et al., on the other hand, propose that the basal ganglia play a significant role in the comprehension of complex syntax and the processing of syntactic rules through the fronto-striatal circuit. Therefore, the observed damage in subcortical regions, exemplified by hyperintense nodular lesion in our patient (Figure 2), may account for these unexpected findings in a patient diagnosed with striato-capsular aphasia. 2

While it is generally believed that the cerebral representation of language remains similar across all languages, variations have been observed among them. Contemporary literature suggests the presence of interlinguistic variation, leading to the hypothesis that variation in language influences their respective brain organization. Consequently, conducting more studies involving Spanish-speaking patients would be valuable [3].

## Conclusions

Subcortical aphasia is a relatively new concept. Our case exhibits clinical and imaging features consistent with subcortical aphasia of the striato-capsular type. In routine practice, these lesions are often not isolated, as seen in our patient who also presented subcortical white matter and thalamic lesions, potentially explaining the additional findings.

Given the extensive role of subcortical structures in language and the possibility that their function may vary depending on the spoken language, further research is crucial in the Spanish-speaking population. Therefore, extensive research is needed to gain a better understanding of this clinical entity and its variation accounting for spoken language.

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