

# Neuropsychological Profile of a Patient with Acquired Brain Damage Following Vascular Lesion of the Left Anterior Cingulate Cortex

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## Abstract

Stroke is a physiological alteration associated with changes in blood flow that can result in sudden-onset cognitive impairment. It has a heterogeneous clinical presentation with varying degrees of severity correlated with specific central nervous system zones or areas, and its prognosis is uncertain. This case study describes a 62-year-old male patient with acquired brain damage of the anterior cingulate cortex as a result of an ischemic event in the territory of the left anterior cerebral artery. Cognitive function was assessed using the neuropsychological executive function and frontal lobe test battery (BANFE-2) as well as other neuropsychological tests. The results show a profile of higher mental functions characterized by the presence of dysexecutive syndrome with marked behavioral alteration and diencephalic amnesia.

## Keywords

Ischemic Stroke, Anterior Cingulate Cortex, Neuropsychology, Acquired Brain Damage

## 1. Introduction

Stroke is characterized by a change in blood flow caused either by obstruction or rupture of the blood vessels inside the brain, giving rise to metabolic, hemodynamic and functional changes in the central nervous system (CNS) [1] [2]. It is classified as hemorrhagic when there is rupture, occurring in the subdural, epi-

dural or subarachnoid spaces, or in an intracranial or ventricular brain area [3] [4].

On the other hand, an ischemic event may be caused by one of several pathophysiological mechanisms responsible for an interruption in cerebral blood flow (CBF), including embolism, venous thrombosis and systemic hypoperfusion. The physiological compensation occurring in this situation results in changes in neuronal metabolic dynamics due to systematic and progressive tissue oxygen deprivation, increasing the levels of lactic acid, intracellular water and free radicals as a result of anaerobic cell processes that produce necrosis and apoptosis [5] [6].

Spontaneous recovery of ischemic tissue is associated with two main factors: time elapsed between the onset of the event and the intervention, and collateral cerebral flow capacity. Therefore, different areas of ischemia can be identified: core, penumbra and benign oligemia, in which the first involves necrosis and inability to recover, while the other two have a high potential to improve as a function of time and treatment [4] [5] [6] [7].

Both ischemic as well as hemorrhagic strokes can occur in any brain vessel or artery and affect indeterminate segments, leading to focal neurological and neuropsychological deficits which correlate with the compromised areas of the brain. However, this study will only focus on the anterior cerebral artery (ACA) and the anterior communicating artery (AComm), specifically on the neurological and neurocognitive impairment resulting from damage to the anterior cingulate cortex (ACC).

The AComm is located at the base of the orbitofrontal region and is the segment that joins the bifurcation of the right and left anterior cerebral arteries. The ACAs supply the corresponding hemispheres, respectively, as they traverse along several segments of the corpus callosum, namely, rostrum, genu, trunk and the first portion of the splenium. They are divided into five segments designated as A1 to A5 and supply subcortical structures and zones such as the caudate nucleus and internal capsule; cortical areas such as the orbitofrontal, medial and anterior cortices in the superior ventral zones and parietal regions. In the cingulate cortex (CC), the course is the same, except that it traverses the entire corpus callosum, the CC being divided into two structures, namely, the posterior cingulate cortex (PCC), related to the posteromedial zone, and the anterior cingulate cortex (ACC) [7] [8] [9] [10].

Due to its connection with the frontal lobes, the limbic system and parietal areas, the CC plays a key role in intentional control functions, emotional and cognitive processing and participates in decision-making tasks, learning from experience, weighing behavioral costs and consequences of decision-making, social and motivated behavior, planning, search for solutions to emotional conflicts, and cognitive flexibility [8] [11] [12] [13] [14] [15]. Acquired brain damage of a vascular nature can present with neurological and cognitive symptoms with compromises that express various levels of severity. The clinical profile has the

potential to generate important imbalances in the basic functional capacity of the patient that are closely related to the extent of the brain damage acquired as result of cerebral hypoperfusion. Alterations in higher mental functions can be limited to failures in specific cognitive components or involve multiple cognitive domains that correspond with certain prefrontal syndromes to global executive dysfunction [16] [17].

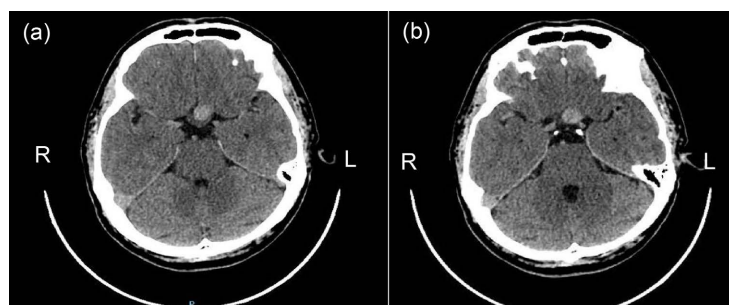
## 2. Method

This study was developed under a single case study approach, in which a descriptive design based on the analytical empirical approach is implemented, which aims to specify the properties, characteristics and important profiles of people who undergo an analysis. It was executed in three stages. In the first, informed consent was obtained from the patient and family. Likewise, authorization was requested from the research ethics committee of the Samaritana University Hospital to carry out the study. In the second stage, neuropsychological evaluation instruments are selected based on the patient's clinical characteristics, emphasizing the analysis of the integrity of the functioning of the frontal lobe. Finally, in the third stage, the analysis of results obtained through the different clinical evaluation tools was carried out [18].

## 3. Case Presentation

A 62-year-old male patient with right laterality, three years of schooling, who does construction work. He has a medical history of hypertension, with no relevant functional, motor or cognitive alterations. He came to the emergency service with a clinical picture lasting five days, characterized by unrelenting headache which did not improve with analgesics (acetaminophen). Over the next few days, the patient lost bilateral visual acuity consistent with left amaurosis. Plain brain computed tomography (CT) imaging showed a thrombosed saccular aneurysm of the anterior communicating complex with dome pointing to the left, causing severe compression of both optic nerves (see **Figure 1**).

After craniotomy for clip placement, a repeat plain CT showed multiple poorly defined, hypodense cortical and subcortical areas in relation to the zones



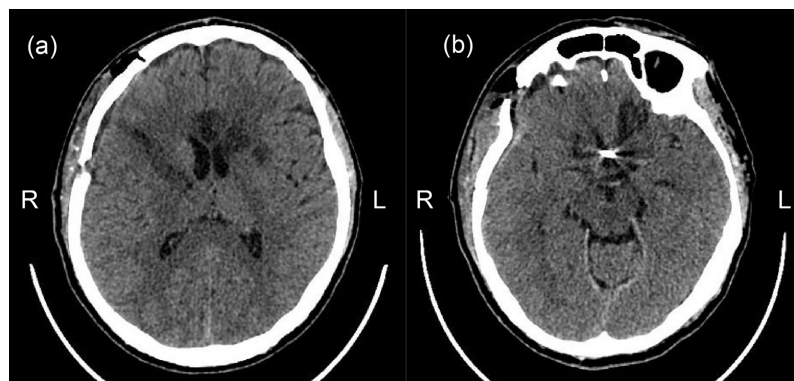
**Figure 1.** (a)-(b): Plain brain CT showing hyperdense areas suggestive of communicating artery aneurysm, with no early signs of bleeding or active hydrocephalus.

of ischemia, involving the genu of the corpus callosum anteriorly and the frontal aspect of the lateral ventricles, as well as the left frontal lobe paramedial area, partially corresponding to the anterior cerebral artery territory. Postoperative changes with wide right frontotemporal craniotomy area and mid frontal lobe hyperdensity area corresponding to clipping during the surgical intervention of the aneurysm (see **Figure 2**).

During the postoperative recovery process, the patient developed significant behavioral alteration and impaired cognition characterized by erratic behavior, disinhibition, fluctuating arousal levels, disorientation in time and space, and memory problems. The patient was assessed by the neuropsychology service using the Mini-Mental State Examination (MMSE), the INECO Frontal Screening (IFS), the Neuropsychologic Executive and Frontal Lobe Function Battery (BANFE-2), the Trail Making Test (TMT A & B), the revised Hopkins Verbal Learning test (HVLN-R), the Token Test (RTT), and the short form of the Boston Naming Test (BNT) [19] [20] [21].

#### 4. Results

The neurocognitive profile derived from the neuropsychological assessment showed fluctuations in arousal levels, altered allopsychic and time and space orientation ability, deficits in attention, semantic, prospective episodic, recent and remote memory, the latter two characterized by lacunar phenomena, and inability to retain and recall accurate information about new events, associated with impaired executive functions represented by problems in inhibitory control, monitoring, planning, organization, social cognition, aggressiveness episodes, anhedonia and dysthymia (see **Table 1**). In terms of language ability, the patient had problems repeating and understanding long sentences, low verbal and semantic fluency, with preserved writing, reading and number recognition abilities.



**Figure 2.** (a): Plain CT scan showing multiple hypodense areas of recent ischemia in the anterior aspect of the corpus callosum, around the frontal horn of the left lateral ventricle and brain parenchyma, in the paramedial region of the frontal lobe. (b): Hyperdense area in the mid frontal lobe corresponding to clipping in the surgical intervention of the aneurysm.

**Table 1.** Results of the neuropsychological assessment.

<b>Subacute phase following craniotomy for stroke</b>		
<b>Mini-Mental State Examination (MMSE)</b>		
	Score	Expected score
Natural score	13	30
<b>INCO Frontal Screening</b>		
Natural score	2	30
Trail Making Test A (TMT-A)		
Natural score	0	25
Percentile	20	45-75
<b>Trail Making Test B (TMT-B)</b>		
Natural score	7	25
Percentile	15	40-70
<b>Hopkins Verbal Learning Test (HVLt-R)</b>		
Initial volume	1	5
Maximum volume	3	9
Recall	1	8
<b>Neuropsychological Executive Function and Frontal Lobe Battery-BANFE</b>		
Orbitomedial		
Coded score	94	211
Anterior prefrontal		
Coded score	1	24
Dorsolateral		
Coded score	40	217
Total Dysexecutive		
Coded score	135	441
Token Test - RTT		
Natural score	131	163
<b>Short form of the Boston Naming test (BNT)</b>		
Natural score	9	15

## 5. Discussion

Higher mental functions are supported by the physiological activity of the various neurons located throughout the cerebral cortex, forming circuits and levels of organization that provide cognitive processes with their characteristics and degrees of interdependence. Consequently, damage in a specific zone could compromise diverse mental functions with different levels of severity depending on the extent and severity of the lesion. This characteristic of brain activity is currently known as functional and structural connectivity and reflects the coordina-

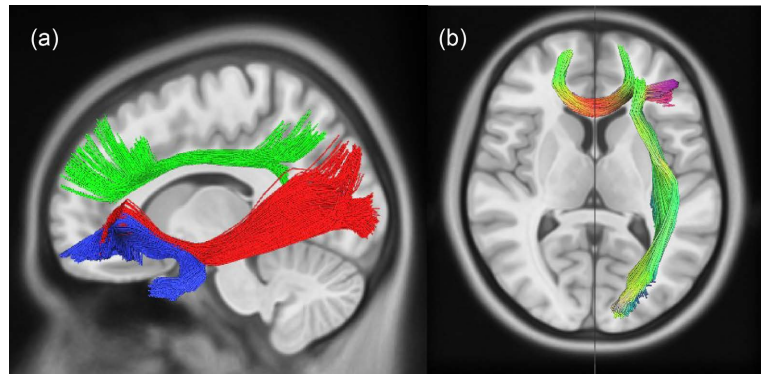
tion of brain areas for the execution of mental activity based on specific neurophysiological patterns and relationships mediated by neuronal axon tracts.

The aim of this case study is to describe the neuropsychological profile of a 62-year-old male patient with acquired brain damage following a vascular event involving the left anterior cingulate cortex. Given that, like others, this clinical condition can potentially derange the functional and structural connectivity dynamics previously described, it can result in the presence of multiple neuropsychological syndromes.

The neuropsychological assessment showed deficits in cognitive processes, mainly in executive functions, due to impaired information processing associated with dorsolateral, anterior and orbital prefrontal regions. The clinical manifestations of damage in these areas relate to frontal-subcortical and prefrontal circuitry supported by the control system of hierarchical executive functions of the prefrontal cortex [22] [23].

Different studies show that compromise of the dorsolateral prefrontal region results in impaired ability to plan and elaborate strategies to solve a problem through the evaluation of a phenomenon and hypothesis generation, considering that this requires high levels of mental flexibility and adequate monitoring or control of the mentalizing process that matches behavior to the dynamic situation of reality [24] [25]. At the same time, it results in a significant reduction of abstraction ability which hinders information storage and handling, giving rise to mental concretism, low processing speed, poor learning ability, impaired retrieval of semantic information, as well as impaired verbal fluency. Compared to language ability, although the literature only describes a decrease in verbal fluency, in the patient, in addition to the slowing of information processing, slight failures in understanding complex instructions as a result of dysexecutive syndrome and difficulty in naming by semantic component also predominated. In this way, with respect to language, altered components are identified that are scarcely described as characteristic of stroke in frontal areas supplied by the anterior cerebral artery [26].

On the other hand, recent research shows that damage to the ventromedial prefrontal cortex with its afferent and efferent connections with the limbic system, alters emotional and behavioral regulation, affecting the ability to make decisions mediated by reward or punishment feedback mechanisms [24]. It also affects processing and storage of episodic and semantic information related to emotional aspects such as sensory-affective integration processes, affecting reactivity and behavior triggered by external stimuli [27] [28] [29]. In general terms, it results in personality changes characterized by exacerbated apathetic, quiet, anhedonic and dysthymic behaviors [16] [24] [30]. When damage is predominantly orbital, underlying alterations are characterized by impulsiveness and disinhibition, giving rise to problems with behavioral and emotional regulation. Characteristics found during clinical observation of the patient and reported by family members and nursing workers during hospitalization [31] [32] [33].



**Figure 3.** (a): Left sagittal section on diffusion tensor imaging (DTI). Red: inferior fronto-occipital fasciculus; Blue: uncinate fasciculus; Green: cingulum bundle. (b): Axial section in DTI directional segmentation of forceps minor and inferior longitudinal fasciculus. The image shows the projection of fasciculi involved in structural connectivity of the prefrontal regions.

In view of the above, the neuropsychological assessment identified symptoms that are characteristic of the three prefrontal areas and which reflect impaired functional and structural connectivity of these regions (see **Figure 3**). The manifestations included marked impairment of both remote as well as distant episodic memory, with altered learning and information consolidation and recall, associated with a pattern of aggressive behavior, apathy and event mutism, reflecting impaired emotional and behavioral regulation. Additionally, during the subacute and acute periods following the neurosurgical intervention, the patient exhibited mood fluctuations with psychomotor agitation, apathy, verbosity, irritability, concrete and illogical thinking, hypersexuality and anxious background mood, episodes of drowsiness and hyperactivity at different times during hospitalization. All these characteristics are consistent with a dysexecutive syndrome and diencephalic amnesia.

The patient's neurocognitive disorders coincide with those widely reported in the literature and correlate with damage to the anterior zone of the cingulate cortex which is closely and directly connected through axonal bundles with the ventromedial, dorsolateral and orbital prefrontal regions, participating in higher mental functions that comprise specific emotional, behavioral and motor components [6] [8] [11] [12] [34] [35].

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### Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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