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TRANSCORTICAL SENSORY APHASIA AS A MANIFESTATION OF ISCHEMIC STROKE WITH INTRACARDIAC THROMBUS

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ABSTRACT Transcortical sensory aphasia is characterized by impaired auditory comprehension, with intact repetition and fluent speech. A 44-year-old right-handed patient with a history of hypertension on amlodipine and ischemic heart disease on aspirin was admitted to the neurology department for sudden onset of language impairment that started 2 days ago. The patient had features of transcortical sensory aphasia. Brain MRI showed an infarct in the territory of the left middle cerebral artery involving the tempo-parietal region. An apical thrombus was observed in the left ventricle on transthoracic echocardiography. This language impairment is thought to be caused by a disconnection between sensory language processes and semantic knowledge of objects. The prognosis is generally guarded and depends on the etiology and severity of the presentation.

KEYWORDS : Transcortical sensory aphasia, ischemic stroke, thrombus

INTRODUCTION:

Aphasia is an acquired disorder of language due to brain damage. Many specific aphasic syndromes have been reported such as Broca, Wernicke, conduction, transcortical motor, and transcortical sensory aphasias. In transcortical sensory aphasia (TCA), patients have a severe deficit in comprehension of speech, but they can produce fluent paraphasic speech [1]. Here, we report a patient with transcortical sensory aphasia secondary to an ischemic stroke.

OBSERVATION:

A 44-year-old right-handed patient with a history of hypertension on amlodipine and ischemic heart disease on aspirin was admitted to the neurology department for sudden onset of language impairment that started 2 days ago. The clinical examination on admission revealed a conscious, apyretic patient with a blood pressure of 150/90 mmHg and normal capillary blood sugar. The patient had difficulty of naming and understanding spoken speech with preserved repetition. He presented also an impairment of writing and reading that was contaminated with paraphasic errors. Those features were consistent with transcortical sensory aphasia. There were no motor or sensory deficits, and his cranial nerves were intact. Brain MRI showed an infarct in the territory of the left middle cerebral artery involving the tempo-parietal region (Fig 1A). On cardiovascular evaluation, the rhythm was normal on electrocardiogram but a floating thrombus was observed in the left ventricle on transthoracic echocardiography (TTE) with septal hypokinesia and an ejection fraction of 30% (N>60%) (Fig 1B). The patient was put on curative anticoagulation and speech therapy. The outcome at 14 days was marked by the persistence of language disorders and disappearance of thrombus on the control TTE.

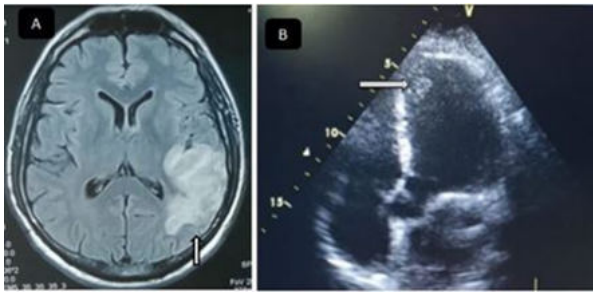

Figure 1A: Fluid-attenuated axial inversion recovery sequence showing hyperintensity in the left middle cerebral artery territory
Figure 1B: transthoracic echocardiography image showing an apical thrombus (14/16 millimeters) floating in the left ventricle

DISCUSSION

The name transcortical sensory aphasia was proposed by Lichtheim in 1885 [2]. TSA is characterized by fluent production with poor comprehension and preserved repetition that is often contaminated by paraphasia, including neological and semantic substitutions. Writing ability and reading are usually disturbed in a manner similar to that of patients with Wernicke aphasia [3]. This language impairment is thought to be caused by a disconnection between sensory language processes and semantic knowledge of objects [4]. Posterior focal neighborhood signs, such as Gerstmann's syndrome, visual field defect, agnosia, alexia, and ideational apraxia are reported in association with TSA [5]. Our patient had features of TSA with a normal neurological examination. Sometimes these findings can be misdiagnosed as psychogenic problems, such as schizophrenia, hence the importance of recognizing this syndrome and using neuroimaging to make the diagnosis [3].

TSA has been infrequently reported as a manifestation of ischemic stroke. The causative lesion of this syndrome is often localized in the parietal and temporal regions, posterior to the perisylvian region of the left hemisphere [3]. Other causes such as tumors, traumas and intracerebral hematomas involving the parieto-temporal-occipital junction area may also manifest as TSA [6]. Patients with degenerative disorders such as Alzheimer's disease are also described to exhibit signs of TSA [7].

In the context of ischemic stroke, TSA has been reported with lesions of the occipitotemporal and temporoparieto-occipital area related to occlusion of the left middle cerebral artery, the left posterior cerebral artery, or the area in common between these 2 arteries [5] [8]. The cerebral location involved in our observation was similar to what has been reported in the literature. In our case, the cause of the ischemic stroke was a left intraventricular thrombus, which is rarely involved in ischemic stroke. Hemorrhagic transformation of ischemic stroke is a feared complication in patients undergoing curative anticoagulation for left ventricular thrombus. Our patient did not develop complications during hospitalization, but the persistence of speech disorders explains the poor prognosis of this condition.

CONCLUSION:

Transcortical sensory aphasia is a rare subtype of aphasia that can occur when a lesion functionally isolates Wernicke's areas from the rest of the brain. The revelation of ischemic stroke by this type of aphasia is unusual, mainly in the context of left ventricular thrombus. The prognosis is generally guarded and depends on the etiology and severity of the presentation.

REFERENCES

[1] Boatman D, Gordon B, Hart J, et al. Transcortical sensory aphasia: revisited and revised. *Brain* 2000; 123: 1634–1642.
[2] Lichtheim L. *On Aphasia. Brain* 1885; 7: 433–85.
[3] Swanberg MM, Nasreddine ZS, Mendez MF, et al. Chapter 6 - Speech and Language. In: Goetz CG (ed) *Textbook of Clinical Neurology (Third Edition)*. Philadelphia: W.B. Saunders, pp. 79–98.
[4] Perry DC. Angular Gyrus Syndrome. In: Aminoff MJ, Daroff RB (eds) *Encyclopedia of the Neurological Sciences (Second Edition)*. Oxford: Academic Press, pp. 192–193.
[5] Saling MM. chapter 3 - Disorders Of Language. Neurology and Clinical Neuroscience 1996; 12: 31–42.
[6] Kertesz A, Sheppard A, MacKenzie R. Localization in Transcortical Sensory Aphasia. *Arch Neurol* 1982; 39: 475–478.
[7] Murdoch BE, Chenery HJ, Wilks V, et al. Language disorders in dementia of the Alzheimer type. *Brain Lang* 1987; 31: 122–137.
[8] Kwon M, Shim WH, Kim S-J, et al. Transcortical Sensory Aphasia after Left Frontal Lobe Infarction: Loss of Functional Connectivity. *Eur Neurol* 2017; 78: 15–21.