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Global Aphasia Following Right Hemispheric Ischemic Stroke: A Rare Case Report

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ABSTRACT

Introduction: Global aphasia is a severe language disorder involving both perceptive and expressive abilities, commonly caused by a left hemispheric stroke due to its typical language lateralization. Rarely, it is associated with right hemispheric lesions, which can be explained by atypical language lateralization.

Case presentation: We report a rare case of a 53-year-old left-handed male presenting with global aphasia following an ischemic stroke in the right hemisphere. The patient experienced sudden left-sided hemiparesis and progressive speech impairment after an episode of chest pain. Neurological examination revealed left supranuclear facial nerve paresis, positive Babinski reflex on the left side, and severe language deficits, including inability to speak, comprehend, repeat, read, or write. Due to imaging limitations, thrombolysis was not performed. The patient was treated with oxygen, antiplatelet therapy, lipid-lowering medication, neuroprotective agent, and glucose control. Imaging at the referral hospital confirmed ischemia in the right frontotemporoparietal region, homologous to the left hemisphere's language-processing areas.

Discussion: This case illustrates that global aphasia can result from right hemispheric strokes due to atypical language lateralization. Factors contributing to this atypical lateralization include left-handedness, genetic predisposition, anatomical variations, and neuroplasticity. Management focused on addressing the ischemic stroke, treating comorbid conditions, and planning for speech therapy as a critical component of rehabilitation.

Conclusion: Recognizing atypical language lateralization is crucial for accurate diagnosis and effective management of global aphasia following right hemispheric strokes, especially in left-handed individuals. Increased clinical awareness can lead to timely interventions and improved patient outcomes.

Keywords: global aphasia, right hemisphere, ischemic stroke, atypical language lateralization

INTRODUCTION

Global aphasia is a condition characterized by impaired language function, which can be triggered by various causes, with stroke being the most common. The language functions affected in global aphasia include both perceptive and expressive abilities. Language processing dominance is known to reside in the left hemisphere for the vast majority of individuals, with studies showing that more than 95% of right-handed individuals and over 70% of left-handed individuals have language lateralization to the left hemisphere.[1] Additionally, left-handed individuals with atypical language lateralization—either to the right hemisphere or mixed—account for approximately 7–

24%.[2] Thus, global aphasia is more commonly associated with impaired language functions in the left hemisphere. Recognizing atypical language lateralization is crucial for accurate diagnosis and management, as it impacts the localization of brain lesions and subsequent rehabilitation strategies. We report a rare case of a 53-year-old left-handed male who developed global aphasia following an ischemic stroke in the right hemisphere.

CASE PRESENTATION

A 53-year-old left-handed man was brought to the Emergency Department on November 3, 2024, at 3 PM, complaining of sudden body weakness and chest pain that had started approximately 4 hours prior. The symptoms began after he completed leading a church service. Upon initial evaluation in the Emergency Department, the patient was alert and oriented, responding appropriately to the examiner's questions. He reported non-radiating chest pain without associated symptoms such as pain in the arm, jaw, or back. While he experienced nausea, he denied vomiting, headache, or fever.

On arrival, his vital signs were as follows: blood pressure 100/70 mmHg, heart rate 87 beats per minute, respiratory rate 20 breaths per minute, blood oxygen 94%, and body temperature 36.7°C. Physical examination revealed no immediate abnormalities apart from mild chest discomfort. Neurological findings at this stage were unremarkable, and the patient showed no language deficits.

Approximately one hour later, the patient experienced a sudden deterioration. He developed a left-sided hemiparesis and profound speech disturbances, which gradually worsened until he could no longer speak. A thorough neurological examination revealed flaccid hemiparesis on the left side with an Medical Research Council grade of 3/5. A positive Babinski sign was noted on the left side. Examination of cranial nerves revealed supranuclear paresis of the left facial nerve. Also, the patient was unable to produce or understand language, repeat phrases, identify objects, read, or write on the following day.

The patient had a history of head trauma from a motorcycle accident approximately five years ago, which resolved without any lasting sequelae and uncontrolled diabetes mellitus for the past year. He denied any history of hypertension, epilepsy, or previous strokes. He also reported no long-term medication use and was not an active smoker.

Laboratory investigations conducted on November 3, 2024, revealed a blood glucose level of 254 mg/dL, hemoglobin 13.6 g/dL, leukocyte count 12,500/mm³, and platelets 299,000/µL. The follow-up laboratory results on the following day showed a fasting blood glucose level of 187 mg/dL, and dyslipidemia (total cholesterol 265 mg/dL, triglycerides 165.1 mg/dL, LDL 131.2 mg/dL). Renal function and electrolytes were within normal limits. Further investigations included an electrocardiogram (Figure 1) and a chest X-ray (Figure 2) to support the diagnostic process.

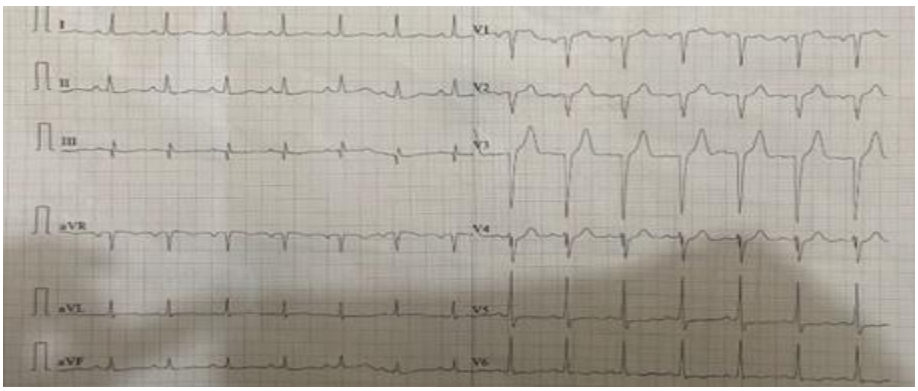

Figure 1. Electrocardiogram findings suggested the possibility of an anteroseptal myocardial infarction

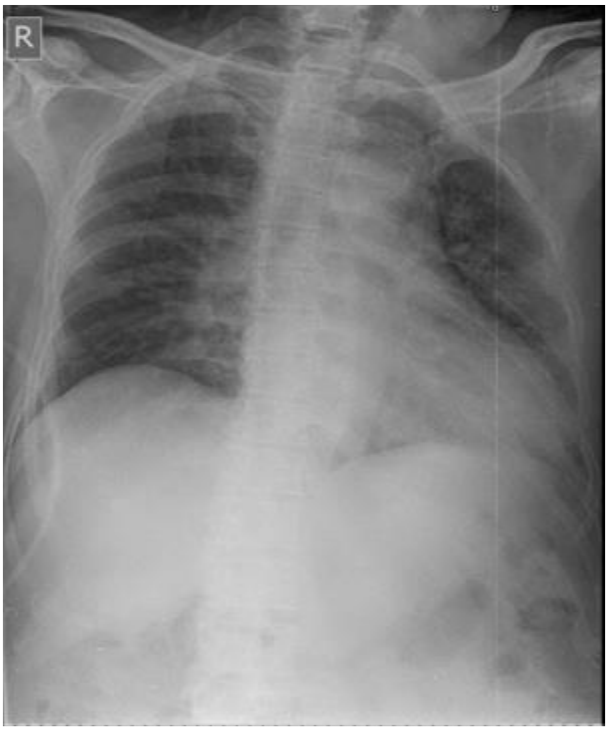

Figure 2. Chest X-ray (AP view) showed signs of cardiomegaly with aortic sclerosis, an aortic knob, and pneumonia.

The patient was diagnosed with a right hemispheric ischemic stroke, presenting with global aphasia, type 2 diabetes mellitus, anteroseptal myocardial infarction, and dyslipidemia.

The patient was managed with a series of interventions aimed at stabilizing his condition. He was positioned with his head elevated at 30 degrees and was placed on bed rest. Oxygen was administered via nasal cannula at 2 liters per minute, which resulted in an increase in blood oxygen from 94% to 97%. To address the ischemic stroke, a loading dose of 320 mg of aspirin was given, followed by 80 mg orally once a day. Citicoline was administered intravenously at a dose of 500 mg twice daily. The patient was also prescribed atorvastatin 20 mg once daily. Isosorbide dinitrate (ISDN) 5 mg was administered orally three times a day. Insulin Drip was started with a dose of 50 IU in 50 cc of normal saline at a rate of 1 cc per hour, while blood glucose levels were monitored hourly. Due to the unavailability of advanced imaging facilities at our hospital, the patient was referred to a higher-level hospital equipped with CT scan capabilities for comprehensive evaluation and continued management. To ensure a safer transfer, we provided one day of treatment at our hospital to optimize the patient's condition before referral. Given that the transfer to the referral hospital takes approximately 4-5 hours and involves geographical challenges that could increase the risk of deterioration during transit, stabilizing the patient beforehand was prioritized to minimize potential complications. Upon arrival at the referral hospital on November 4, 2024, a non-contrast head CT scan was performed, the results of which are presented in this case (figure 3).

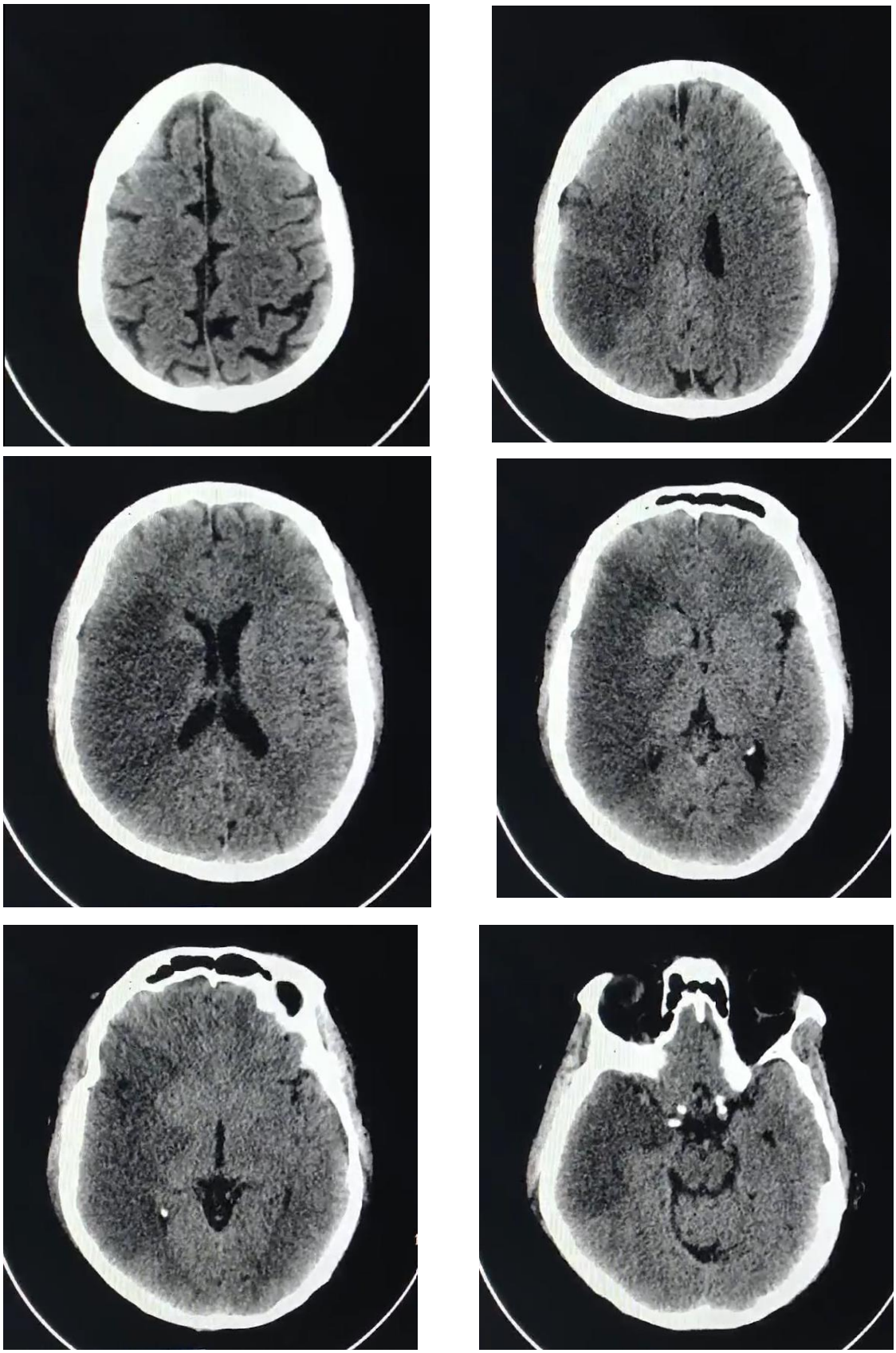

Figure 3. Non-contrast head CT scan on November 4, 2024, showed an infarction in the right frontotemporoparietal region

DISCUSSION

Evaluation of Ischemic Stroke and Possible Thromboembolism

The patient presented with acute neurological deficits characterized by the sudden onset of left-sided hemiparesis and speech disturbances, which rapidly progressed to global aphasia. Neurological examination revealed flaccid hemiparesis on the left side with a Medical Research Council (MRC) grade of 3/5, a positive Babinski sign on the left, and left-sided supranuclear facial nerve paresis. A non-contrast computed tomography (CT) scan of the brain performed at the referral hospital demonstrated hypodensity in the right frontotemporoparietal region, indicating an infarction in the territory supplied by the right middle cerebral artery (MCA).

The diagnosis of ischemic stroke was primarily established through clinical evaluation, supported by the Siriraj Stroke Score (SSS). The SSS was utilized as a practical tool to differentiate ischemic stroke from hemorrhagic stroke in the absence of advanced imaging.[3] The SSS was calculated to be -11, strongly indicative of an ischemic rather than hemorrhagic stroke. Key clinical features contributing to this score included the absence of altered consciousness, vomiting, or headache, combined with the presence of risk factors such as uncontrolled diabetes mellitus and angina pectoris.

The etiology of the ischemic stroke in this patient is highly suggestive of a thromboembolic mechanism. Thromboembolic strokes commonly result from emboli originating from the heart or large arteries, which occlude cerebral vessels. The patient had significant cardiovascular comorbidities, including an anteroseptal myocardial infarction identified on electrocardiography, dyslipidemia, and uncontrolled diabetes mellitus. These conditions are established risk factors for thrombus formation and subsequent embolization.[4,5] Additionally, chest radiography revealed signs of cardiomegaly and aortic sclerosis, further supporting the likelihood of a cardiac or large-vessel source of emboli.[6]

Echocardiography, particularly transesophageal echocardiography, is instrumental in detecting cardiac sources of emboli, such as left ventricular thrombi or valvular abnormalities.[7] Unfortunately, these advanced diagnostic tools were unavailable in this case, limiting the ability to confirm the thromboembolic etiology definitively.

Global Aphasia and Atypical Language Lateralization

Aphasia is a language disorder resulting from damage to brain regions responsible for language processing, leading to impairments in comprehension, production, or both. In severe cases, such as this patient, it manifests as global aphasia, characterized by a profound loss of all language functions, including speaking, understanding, reading, writing, and repetition.[8] Key areas involved in language function include Broca's area, Wernicke's area, the angular gyrus, and Heschl's gyrus, which are interconnected by the arcuate fasciculus, facilitating coordination between language comprehension and production.[9]

In most individuals, language functions are predominantly processed in the left hemisphere, a phenomenon known as typical language lateralization. The right hemisphere is often associated with the "emotional" aspects of language, such as intonation, melody, phrasing, and prosody. However, atypical language lateralization occurs when language functions are processed in the right hemisphere or shared between both hemispheres.[10] This atypical pattern is more commonly observed in left-handed individuals.

In this patient, evidence suggests that language lateralization is in the right hemisphere. The ischemic stroke affecting the right frontotemporoparietal region resulted in global aphasia, indicating that the damaged areas were dominant for language processing. The patient's inability to speak, comprehend language, repeat words, name objects, read, or write supports this conclusion. Neurological deficits on the left

side of the body further confirm the involvement of the right hemisphere.

Several factors contribute to atypical language lateralization, including handedness, genetic influences, anatomical structures, and neuroplasticity. While approximately 70% of left-handed individuals still exhibit left-hemisphere language dominance, the remaining 30% demonstrate right-hemisphere or mixed lateralization. This patient's left-handedness may have predisposed him to right-hemisphere language dominance.[11] Genetic factors also play a significant role, as studies on monozygotic twins have shown strong correlations in language lateralization patterns.[12] Anatomical asymmetries, such as variations in the size of the planum temporale and pars triangularis, are associated with language dominance but could not be assessed in this case due to lack of advanced imaging facilities.[13,14]

Neuroplasticity, the brain's ability to adapt and reorganize functions, is another important factor. In individuals who experience brain injuries, particularly at a young age, the contralateral hemisphere may compensate for damaged language areas.[15] Although the patient had a history of head trauma five years prior without lasting sequelae, it is unlikely that this contributed significantly to a shift in language lateralization. Nonetheless, neuroplastic mechanisms cannot be entirely ruled out.

Management

The management of acute ischemic stroke focuses on rapid restoration of cerebral blood flow, prevention of further neuronal damage, and management of underlying risk factors. Thrombolytic therapy with recombinant tissue plasminogen activator (rtPA) is most effective when administered within 4.5 hours of symptom onset.[16] However, in this case, rtPA was not administered due to limited imaging facilities to rule out hemorrhagic stroke and the delay beyond the therapeutic window.

Although a thromboembolic mechanism was suspected, anticoagulant therapy was not initiated in the acute phase. Current clinical guidelines do not recommend routine anticoagulation in the acute management of ischemic stroke without specific indications and diagnostic confirmation. The decision to withhold anticoagulants was based on the increased risk of intracerebral hemorrhage without definitive imaging to exclude hemorrhagic stroke and confirm a cardioembolic source. Therefore, antiplatelet therapy with aspirin was chosen as a safer initial treatment.

Antiplatelet therapy was initiated with a loading dose of aspirin 320 mg, followed by 80 mg daily, to reduce the risk of stroke progression and prevent recurrent events. Although there is a slight increase in bleeding risk, the benefits in reducing thrombotic complications outweigh the risks.[17] Mechanical thrombectomy, which can be effective up to 24 hours after symptom onset for large-vessel occlusions, was not feasible due to the unavailability of specialized facilities.

Management of comorbid conditions was also essential. The patient's hyperglycemia was addressed with an insulin drip, allowing for precise control of blood glucose levels and rapid dose adjustments based on frequent blood glucose monitoring, typically performed hourly.[18] Frequent monitoring of electrolytes, particularly potassium, was necessary due to the risk of hypokalemia associated with insulin therapy.[18] To support neuronal survival, citicoline was administered intravenously at a dose of 500 mg twice daily. Citicoline therapy aimed to protect the ischemic penumbra and enhance recovery potential during the acute phase. Dyslipidemia was managed with atorvastatin 20 mg daily to reduce low-density lipoprotein (LDL) cholesterol levels and provide plaque-stabilizing effects.[19]

Cardiovascular support included oxygen supplementation via nasal cannula at 2 liters per minute, increasing oxygen saturation from 94% to 97%. Isosorbide dinitrate (ISDN) 5 mg was administered orally three times a day to alleviate chest discomfort associated with the myocardial infarction. Due to facility limitations, definitive cardiac

interventions such as percutaneous coronary intervention (PCI) or thrombolysis were not possible.

Management of global aphasia was not initiated at our facility due to prioritization of medical stabilization and resource limitations. Early speech and language therapy is crucial for patients with global aphasia to regain language functions. However, the patient's critical condition, along with the need to manage acute comorbidities such as myocardial infarction and uncontrolled diabetes mellitus, necessitated focusing on life-saving interventions first. Additionally, our hospital lacked specialized rehabilitation services required for aphasia management. As a result, the initiation of specialized rehabilitation was delayed.

Limitations

A significant limitation in this case was the limited access to advanced imaging facilities, which delayed definitive diagnosis and intervention. This delay prevented the timely administration of thrombolytic therapy and mechanical thrombectomy, both of which are most effective within specific time windows after stroke onset. The necessity to stabilize the patient before a prolonged transfer further extended the time before advanced diagnostics could be performed, potentially affecting the overall prognosis.

Prognosis

The prognosis for global aphasia after an ischemic stroke depends on the extent of brain damage, timing of interventions, and initiation of rehabilitation. In this case, extensive infarction in the right frontotemporoparietal region—dominant for language due to atypical lateralization—makes recovery more challenging. Early speech and language therapy is vital for regaining language functions, but its initiation was delayed because of medical stabilization priorities and lack of specialized services. This delay may limit the effectiveness of rehabilitation efforts. The patient's comorbid conditions, such as uncontrolled diabetes mellitus and cardiovascular disease, further complicate the prognosis by increasing the risk of recurrent strokes and affecting overall recovery potential.

Given these factors, the prognosis is guarded. While improvement is possible with comprehensive rehabilitation and careful management of comorbidities, the extent of functional recovery may be limited.

CONCLUSION

This case highlights the rare occurrence of global aphasia resulting from a right hemispheric ischemic stroke in a left-handed individual, underscoring the significance of atypical language lateralization. Recognizing atypical language lateralization is essential for accurate localization of brain lesions and effective management of global aphasia, particularly in left-handed patients with right hemispheric strokes. The patient's left-handedness likely contributed to right hemisphere dominance for language, leading to profound language deficits typically associated with left hemispheric strokes. Management in this case focused on addressing the ischemic stroke, controlling comorbidities, and planning speech therapy as a vital component of rehabilitation. Delayed access to imaging and specialized rehabilitation services posed challenges to timely intervention, potentially impacting the patient's prognosis.

Declaration by Authors

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