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Case Report

ISCHEMIC STROKE IN DISGUISE: AN ABNORMAL MOVEMENT ISCHEMIC STROKE CASE REPORT

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ABSTRACT

Introduction: This case report aims to heighten awareness that acute, sudden-onset abnormal movements can signal an underrecognized ischemic stroke, even without classic BEFAST (Balance, Eyes, Face, Arms, Speech) symptoms. Swift stroke diagnosis is vital, as time critically influences prognosis; yet, the typical presentation of hemiparesis often overshadows atypical features such as movement disorders, risking misdiagnosis.
Case Report: A 60-year-old woman presented to the emergency department with sudden abnormal jaw and left limb movements lasting one day, with no prior episodes or accompanying symptoms. Comprehensive physical exams, ancillary tests, and imaging revealed an ischemic stroke in the left corona radiata on MRI (Magnetic Resonance Imaging).
Discussion: These hyperkinetic movements stemmed from stroke infarction at corticostriatal fibers region, which normally transmit excitatory glutamatergic signals from the cortex to basal ganglia structures like the caudate nucleus and putamen. This input inhibits the globus pallidus internus, disinhibiting the thalamus to enable smooth movement initiation. Lesions in this pathway, as seen here, provoke abnormal hyperkinetic disorders.
Conclusion: Left corona radiata ischemia atypically manifested as isolated movement abnormalities due to corticostriatal pathway interruption. Clinicians must vigilantly consider ischemic stroke in patients with abrupt-onset movement disorders, regardless of the absence of traditional symptoms, to ensure timely intervention and better outcomes.

Keywords: abnormal movement; stroke; ischemic stroke

INTRODUCTION

Ischemic stroke typically manifests as sudden focal neurological deficits such as hemiparesis or facial droop. This clinical manifestation goes in accordance with the BE FAST acronym, which stands for Balance, Eyes, Face, Arms, Speech, and Time.[1] In contrast, movement disorders are rarely considered a primary presentation of acute cerebrovascular events.

In this case, an acute onset of abnormal movement could be easily overlooked in emergency settings. Its occurrence as the sole presenting sign of an acute infarct

remains underemphasized. The prevailing focus on hemiparesis and other BE FAST symptoms may inadvertently lead to cognitive biases, causing atypical presentations to be misattributed to other etiologies, posing a diagnostic challenge of stroke. Thereby delaying appropriate stroke workup and management. Given that time-sensitive treatments directly impact patient outcomes, recognizing these unusual stroke presentations is of cardinal clinical importance.

This case report aims to increase awareness that an acute, sudden onset of abnormal movements can signal an underrecognized ischemic stroke. It reinforces the critical principle that any sudden-onset neurological deficit—whether typical or atypical—warrants urgent stroke investigation to optimize patient outcomes through timely intervention.

CASE REPORT

A 59-year-old female patient presented to the Emergency Department (ED) of Kalideres General Hospital with complaints of abnormal movements of the limbs for 1 day before admission. The abnormal movements were felt in the lower jaw, left arm, and left leg. The movements were described as chewing movements, with the left arm and left leg moving involuntarily, rapidly, and without purpose, within a small range. The abnormal movements appeared intermittently and resolved spontaneously, with each episode lasting approximately 30 minutes. Since 15 hours before admission, the abnormal movements became continuous and did not stop. The patient denied any history of decreased consciousness, trauma, motor or sensory deficits, nausea, vomiting, diarrhea, or decreased appetite.

The patient has a history of breast malignancy for 18 months before admission. She also has a history of ischemic stroke for 2 months before admission. Her regular medications include anastrozole 1 mg PO (per oral)

OD (once daily), leparson-benserazide hydrochloride combination therapy 1 tablet PO BID (twice a day), acetylsalicylic acid 80 mg PO OD, clopidogrel 75 mg PO OD, citicoline 500 mg PO BID, and simvastatin 20 mg PO OD. She underwent a left mastectomy 7 months before admission. She was then confirmed for having mammary cancer stage IIIB by the biopsy result. Based on physical examination results, it was found that vital signs were within normal limits, with good nutritional status. General physical examination within normal limits. On the thorax sinistra, a post-operative scar measuring 0.5x10x20 cm with hyperpigmented excoriation around the scar. From neurological findings, the Babinski reflex was found to be positive, and motor function shows hemichorea involving the right mandible, left upper extremity, and left foot, and slight hemiplegia dextra.

Supporting examinations show laboratory tests were within normal limits. Non-contrast CT scan of the brain shows the impression of characteristic findings of Fahr syndrome. No other significant abnormal radiological findings were found. MRI of the brain shows the impression acute infarct in the left corona radiata; calcifications in the pons and cerebellar parenchyma suggestive of Fahr syndrome; cerebral atrophy (GCA-scale 1); Leukoaraiosis (Fazekas scale 2); no evidence of hemorrhagic lesions or space-occupying lesions (SOL).

Based on the history, physical examination, and supporting investigations, the diagnosis was established as hemichorea secondary to ischemic stroke.

The patient was admitted for inpatient care, and she was given NaCl 0,9% 500cc q12h (every twelve hours). The administered drugs were pantoprazole 40mg IV (intravenous) BID, ondansetron 8mg IV TID (three times a day), acetylsalicylic acid 80 mg PO OD, clopidogrel

75mg PO OD, atorvastatin 40mg PO OD, valproic acid 500mg PO BID, mecobalamin 500mcg PO BID, trihexyphenidyl 2mg PO TID, levodopa-benserazide hydrochloride combination therapy 1 tablet PO BID.

DISCUSSION

Epidemiology of Stroke

Stroke remains a major global health problem, as well as an important cause of death and disability worldwide. In the United States, 795,000 strokes occur annually which 185,000 are recurrent strokes. Approximately only 10-20% of stroke patients experience hemorrhagic stroke, and about 88% experience ischemic stroke. About 8-12% of ischemic strokes result in death within 30 days.[2] The epidemiology of ischemic stroke symptoms reflected in the BE FAST framework demonstrates clear differences between anterior and posterior circulation events, while also highlighting the presence of atypical presentations. Classical BE FAST features such as facial weakness, arm weakness, and speech disturbance are more frequently observed in anterior circulation strokes rather than the posterior circulation, occurring in 48.3% versus 23.4% for facial weakness, 71.2% versus 44.7% for arm weakness, and 55.9% versus 29.8% for speech impairment. Conversely, symptoms included in the balance and eye components of BE FAST are more commonly associated with posterior circulation involvement, with balance problems or lower limb weakness reported in 72.3% of posterior cases compared with 61.0% of anterior cases, and eye signs or visual impairment occurring in 23.4% versus only 0.8%, respectively.[3] In addition to these typical manifestations, atypical presentations also occur. Within the definite ischemic stroke patients, 43.1% presented with isolated symptoms, of which 9.6% were atypical, and 33.6% were typical.[4]

Risk Factors of Stroke

Age is the strongest risk factor for stroke's incidence, increasing

as the individual grows older. The risk of stroke doubles with each decade after age 55, and mostly occurs in individuals older than 75. Sex differences also influence stroke risk and outcomes. Men have higher stroke incidence between the ages of 45 and 75. After the age of 75, women have higher stroke incidence rates. Women also tend to have higher case fatality rates compared with men.[2] Ethnicity also becomes one of the influences in both stroke subtypes and outcomes. Asian populations show higher rates of hemorrhagic stroke compared with Caucasians. Japan accounts for a larger proportion of hemorrhagic stroke, and India predominates in ischemic stroke.[2]

In modifiable risk factors, arterial hypertension leads as it affects at least one quarter of the adult population. Elevated blood pressur promotes atherosclerosis and accelerates cardiovascular disease, increasing stroke risk by three- to fourfold. Effective blood pressure control significantly reduces stroke incidence and recurrence, and even modest reductions in blood pressure are associated with substantial decreases in stroke risk.[2,5]

Metabolic disorders such as diabetes mellitus and dyslipidemia are also major contributors to stroke risk. Diabetes mellitus increases the risk of ischemic stroke by two- to fourfold and is associated with higher morbidity and mortality after stroke. The combination of diabetes and hypertension further increases the likelihood of cardiovascular and cerebrovascular events. Dyslipidemia, particularly elevated total cholesterol and low-density lipoprotein cholesterol,promotes atherosclerosis and increases the risk of ischemic stroke.[2,5]

Cardiac conditions are important sources of embolic stroke. Atrial fibrillation is the most common sustained cardiac arrhythmia and increases stroke risk approximately five- to sixfold.

Structural heart abnormalities, including left atrial enlargement and left ventricular hypertrophy, are also associated with increased stroke risk.[2,5]

Cancer or having a history of it could also increase stroke risk. Cancer survivors have shown a one-point-five to one-point-seven fold higher stroke risk than cancer-free individuals. It increases the risk for ischemic stroke rather than hemorrhagic stroke.[2,5,6]

Lifestyle factors significantly influence stroke risk. Cigarette smoking is a major modifiable risk factor and increases stroke risk two- to threefold through mechanisms including accelerated atherosclerosis, increased blood coagulability, reduced oxygen delivery, and vascular spasm. Alcohol consumption demonstrates a dose-dependent relationship with stroke risk, with light to moderate intake associated with a lower risk of ischemic stroke. In contrast, heavy consumption increases the risk of both ischemic and hemorrhagic stroke. Obesity and physical inactivity indirectly contribute to stroke risk through their effects on blood pressure, lipid metabolism, and insulin resistance. Obstructive sleep apnea is strongly associated with stroke and is more common among stroke patients than in the general population. Habitual snoring and severe sleep apnea are linked to increased stroke risk and poorer outcomes.[2,5]

Lastly, hormonal and reproductive factors influence stroke risk in women. Stroke is uncommon in women of childbearing age. The consumption of hormonal agents increases the risk of ischemic stroke, especially in the presence of other risk factors such as smoking and hypertension. Hormone replacement therapy in postmenopausal women has not been shown to reduce stroke risk and may increase the likelihood of thrombotic events. Pregnancy and the postpartum period are also associated with an increased risk of stroke, particularly during the weeks following delivery.[2,5]

Clinical Manifestations of Stroke

BE FAST reflects the most frequent and typical symptoms of acute stroke. It usually presents with a sudden disturbance of focal neurological function, including balance, vision, facial movement, limb strength, or speech. This approach improves stroke recognition, as those symptoms typically occur during acute interruption of cerebral blood flow.[1]

The clinical manifestations of stroke can also vary depending on its vascular etiology. In an embolic stroke, neurological deficits appear suddenly and reach maximal severity within seconds or minutes. It can also involve the posterior circulation, resulting in transient symptoms such as dizziness, vertigo, diplopia, or dysarthria. Thrombotic stroke often evolves gradually and may be preceded by transient signs. The neurological deficits commonly progress in a stepwise manner over minutes or hours and occasionally over several days. Partial deficits may improve temporarily before progressing into a complete stroke. Rapid deterioration may also occur if a thrombus occludes a major vessel.[2]

Meanwhile, lacunar stroke produces characteristic clinical manifestations because it involves small penetrating arteries in deep brain structures. Symptoms develop relatively quickly but less abruptly than the embolic stroke. A key feature is the absence of cortical dysfunction. Common lacunar syndromes include pure motor hemiparesis, pure sensory stroke, dysarthria–clumsy hand syndrome, and ataxic hemiparesis. These deficits often involve contralateral motor or sensory impairment and may partially recover over time.[2,5]

Clinical manifestations can also vary according to the involved vascular territory. Infarction in the middle cerebral artery territory commonly produces contralateral hemiparesis and hemisensory loss predominant in the face and upper extremity,

and can be accompanied by visual field disturbance. Dominant hemisphere involvement may cause aphasia, while nondominant hemisphere involvement may cause visuospatial disturbance. Anterior cerebral artery infarction typically manifests in contralateral weakness predominantly affecting the lower extremity, along with changes in cognitive and behavioural function. Posterior cerebral artery infarction frequently results in visual disturbance. Brainstem and cerebellar infarctions produce distinctive neurological manifestations. Thus includes vertigo, ataxia, diplopia, dysarthria, cranial nerve deficits, and altered consciousness. Occlusion of cerebellar arteries could manifest in dizziness, gait instability, and coordination deficits. Basilar artery occlusion may lead to severe neurological impairment, including quadriplegia or locked-in syndrome. On the other hand, watershed infarctions occurs between vascular territories may produce bilateral neurological deficits depending on the affected zone.[5]

Corona Radiata Anatomy and Physiology

The corona radiata is a collection of projection nerve fibers that connect the cerebral cortex with deeper structures of the brain, including the brainstem and spinal cord. These fibers consist of both afferent pathways, which transmit sensory information to the cerebral cortex, and efferent pathways, which carry motor commands from the cortex to lower centers. As nerve fibers travel between the cerebral cortex and the brainstem, they pass through dense masses of gray matter within the cerebral hemispheres. At the level of the upper brainstem, these fibers converge into a compact structure known as the internal capsule, located between the caudate nucleus and thalamus medially and the lentiform nucleus laterally. It is supplied by the perforating artery, which are small branches of the medial cerebral artery.[7,8]

DISCUSSION

This case describes a 59 year old woman presenting with an acute onset of involuntary movements involving the mandible and left extremities. Based on clinical findings and neuroimaging, the patient was diagnosed with abnormal movement secondary to acute stroke infarction in the left corona radiata. The majority of strokes are stroke infarcts, and this also goes with what the patient experienced. The patient had several risk factors for ischemic stroke, such as older age, previous history of stroke, and history of mammary cancer. Increasing age is the strongest non-modifiable risk factor for stroke, with incidence rising significantly after the age of 55. In addition, a prior stroke is an important predictor of recurrent stroke. Cancer-associated hypercoagulability is an important mechanism contributing to ischemic stroke in patients with malignancy. Tumor cells produce several procoagulant substances that promote thrombosis, including tissue factor, podoplanin, plasminogen activator inhibitor, and protein disulfide isomerase. Among these, tissue factor plays a key role in arterial thrombosis because malignant cells can release macrovesicles rich in tissue factor that interact with macrophages within atherosclerotic plaques, further enhancing thrombus formation. In addition, cellular mechanisms involving activated platelets and neutrophil extracellular traps contribute significantly to the prothrombotic state. In patients with malignancy, neutrophil extracellular trap formation occurs chronically and accelerates arterial thrombosis.[9,10]

Clinical manifestations in this patient were considered atypical as it does not fit the BE FAST criteria or the classical neurological deficits as often met. Stroke lesions presenting in the deep-brain structure could produce non-classical symptoms. In this case, the patient had no motor weakness or sensory deficit, which is consistent with the involvement

of subcortical structure rather than cortical region.

Abnormal movement, being the clinical manifestation, is usually associated with lesions involving the basal ganglia. However, lesions affecting the pathways, such as the corona radiata, may also produce movement disorders by disrupting the connections between the cortex and basal ganglia. Corona radiata contains projection fibers that transmit motor signals between cortical motor areas and subcortical nuclei. Damage to these pathways may disturb the balance between excitatory and inhibitory motor circuits, resulting in abnormal movements.

CONCLUSION

Ischemic lesions involving the corona radiata may present with atypical clinical manifestations. Interruption of these motor regulatory circuits can produce abnormal movements rather than the more typical findings fitting the BE FAST acronym, such as motor or sensory deficits. In this case, ischemia of the left corona radiata likely interfered with descending motor fibers and modulatory pathways to the basal ganglia, resulting in disinhibition of motor activity and the development of hemichorea. Such presentations may obscure the diagnosis because classical stroke symptoms may be minimal or absent. Therefore, clinicians should maintain a high index of suspicion for ischemic stroke in patients presenting with sudden-onset movement disorders, even when traditional focal neurological deficits are not evident, since early recognition and treatment are essential to prevent progression of brain injury and to improve functional outcomes.

Further research is needed to better understand the relationship between ischemic stroke and movement disorders, particularly from lesions outside the basal ganglia, such as the corona radiata. Although basal ganglia involvement is classically associated with choreiform movements, evidence suggests that disruption of its pathways

may also produce similar clinical manifestations. Larger clinical studies are required to clarify the mechanisms by which subcortical white matter infarctions lead to abnormal movements and to determine the frequency of such atypical presentations.

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REFERENCES

1. Aroor S, Singh R, Goldstein LB. BE-FAST (Balance, Eyes, Face, Arm, Speech, Time): reducing the proportion of strokes missed using the FAST mnemonic. Stroke. 2017;48(2):479–481. doi:10.1161/STROKEAHA.116.015169.
2. Ropper AH, et al. Cerebrovascular diseases. In: Ropper AH, Samuels MA, Klein JP, Prasad S, editors. Principles of Neurology. 12th ed. New York: McGraw Hill; 2023. p. 772–878.
3. Lavallée PC, Sissani L, Labreuche J, Meseguer E, Cabrejo L, Guidoux C, et al. Clinical significance of isolated atypical transient symptoms in a cohort with transient ischemic attack. Stroke. 2017;48(6):1495-1500. doi:10.1161/STROKEAHA.117.016743.
4. Tanglay O, Cappelen-Smith C, Parsons MW, Cordato DJ. Enhancing stroke recognition: a comparative analysis of balance and eyes–face, arms, speech, time (BE-FAST) and face, arms, speech, time (FAST) in identifying posterior circulation strokes. J Clin Med. 2024;13(19):5912. doi:10.3390/jcm13195912.
5. Biller J, Schneck MJ, Ruland S. Ischemic cerebrovascular disease. In: Daroff RB, et al, editors. Neurology in Clinical Practice. 8th ed. Philadelphia: Elsevier; 2022. p. 964–1013.
6. Turner M, et al. Is stroke incidence increased in survivors of adult cancers? A systematic review and meta-analysis. J Cancer Surviv. 2022;16(6):1414–1448. doi:10.1007/s11764-021-01122-7.
7. Snell RS. Cerebral hemispheres and internal structure. In: Snell RS, editor. Clinical Neuroanatomy. 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2010. p. 251–283.
8. Vogels V, Dammers R, van Bilsen M, Volovici V. Deep cerebral perforators: anatomical distribution and clinical symptoms: an overview. *Stroke*. 2021 Oct;52(10):e660-e674. doi:10.1161/STROKEAHA.120.034096.
9. Cheong M.A., Leader M. Cancer and arterial thromboembolism: therapeutic options. Res Pract Thromb Haemost. 2024;153:103181. doi:10.1016/j.rpth.2024.102393.
10. Bang OY, et al; OASIS-Cancer Study Investigators. Cancer-related stroke: an emerging subtype of ischemic stroke with unique pathomechanisms. J Stroke. 2020;22(1):1–10. doi:10.5853/jos.2019.02278.