Case Report

Hand Knob Infarct Mimicking Carpal Tunnel Syndrome: A Rare case report from Sikandrabad, Uttar Pradesh

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Abstract

Background: The infarction of the hand knob is a subtype of cortical stroke, often presenting with isolated hand weakness that closely resembles peripheral neuropathy. Its quality of presentation and lack of sensory symptoms can be ignored because of its insidious nature, leading to a decrease in the time of proper stroke treatment. Early detection is necessary to prevent recurrence and reduce disability. **Case Presentation:** This patient with hypertension had a history of transient ischemic attack, and 10 hours after starting his speech became slurred; he continued to demonstrate left-hand grip weakness. Hand weakness is isolated, and the NIHSS score is 1, with no sensory or proximal motor weakness. Vascular examination, ECG, and echocardiography were normal, and lipid profile showed a very high LDL cholesterol level. The MRI brain revealed a tiny acute infarction in the right precentral gyrus, including the hand knob region. He received a loading dosage of aspirin and clopidogrel, was put on dual antiplatelet therapy, high-dose atorvastatin, optimization of blood pressure, and lifestyle change. **Conclusion:** The case shows that it is crucial to remember cortical hand knob infarction in patients who present with isolated hand weakness. MRI is essential in the diagnosis process, and early secondary prevention is important in alleviating the risk of future strokes.

Keywords: Hand Knob infarct; Isolated single hand weakness; Cortical stroke; MRI; Dual antiplatelet therapy; Minor stroke; Precentral gyrus.

Received: 06 November 2025 **Revised: 22 November 2025** **Accepted: 15 December 2025** **Published: 29 December 2025**

Introduction

Isolated weakness of the hand is a rare but clinically significant feature of stroke that causes diagnostic error. Selective fine motor control of a contralateral hand and fingers occurs in the hand knob area, a unique omega/epsilon area on the precentral gyrus. This area has a low infarct frequency of 0.7-1.5 percent of all ischemic strokes and is often similar to lesions of peripheral nerves, radial neuropathy, median nerve palsy, or cervical radiculopathy.[1,2] Due to this confusion, it is easy to overlook these cortical strokes, hence failing to administer timely antiplatelet therapy, insufficient risk factor control, and high recurrence risks.

Identification is particularly important amongst older patients with proven vascular predisposing conditions. Significant risk factors of small cortical infarcts are hypertension, dyslipidemia, diabetes, atrial fibrillation, and prior TIA, despite the possibility that such deficits may be mild or of a transient nature. It has been demonstrated that minor stroke syndromes, even NIHSS 02, can represent clinically important cortical ischemia, especially in motor eloquent areas like the hand knob.[3] What is more, even minor deficits, such as a selective weakness of the fingers or grip, might resolve or change, leading clinicians toward a benign diagnosis.

Neuroimaging is critical for verifying cortical strokes in hand areas. CT scans can be normal, particularly when the lesions are small or in the high convexity. However, MRI with diffusion-weighted imaging (DWI) is certain to detect acute infarcts in the precentral gyrus and accurately outline the hand knob area permanently, facilitating early diagnosis and direct treatment.[4] Anatomical mapping research has demonstrated that the hand knob position is highly consistent; therefore, MRI is an invaluable method for distinguishing cortical injury from peripheral neuropathies, motor neuron disease, or functional disorders.[1]

Diagnosis: The necessary distinction between hand knob infarct and peripheral lesions is made clinically. In contrast to peripheral neuropathies, where dermatomal or nerve distribution patterns are generally followed and often involve sensory deficits, cortical hand knob strokes result in motor weakness (without sensory impairment), fasciculations, and changes in reflexes (Lee, 2004). The index of suspicion thus demands a high level, especially in cases where the onset is acute, the deficits are central, and there are vascular risk factors.

In this case, the diagnostic and treatment significance of an isolated hand knob infarct when a patient presents with partial weakness of grip is observed. With its specific clinical observations, radiological associations, and customized secondary prevention interventions, the report demonstrates the

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DOI:
10.21276/amit.2025.v12.i3.276

How to cite this article: Kaushik R, Kaalkhanday A, Saini V. Hand Knob Infarct Mimicking Carpal Tunnel Syndrome: A Rare case report from Sikandrabad, Uttar Pradesh. Acta Med Int. 2025;12(3):1305-1309.

necessity of timely assessment of even subtle motor impairments and the important role of MRI in detecting subtle cortical strokes that could otherwise go unnoticed.

Case Presentation

Patient Information: A 65-year-old male patient who had a history of hypertension and a history of transient ischemic attack presented to the emergency department 10 hours after the onset of the symptoms. He had no complaints of sensory loss, visual acuity, gait, or consciousness.

Presenting Complaints: The patient stated that his speech had become largely unintelligible, but he still had intermittent weakness of the grip in his left hand, with no gradual onset or improvement. He also mentioned having problems with holding objects and fine motor actions, which led him to seek medical care.

Image 1. Clinical image on the left of the patient's left hand showing a zero grip strength and not able to flex the fingers as an isolated distal hand weakness, which is a result of a cortical infarct in the right precentral gyrus (hand knob area). The posture demonstrates motor deficit selectivity, with loss of sensation, indicating a central etiology rather than a peripheral one.

Current Clinical Examination.

Neurological assessment showed isolated, fused weakness of the left hand; grip strength was initially recorded at 3/5. The patient had no weakness of the proximal upper limb, no lower limb involvement, and no sensory deficiency. Asymmetry of the face, mildly present at the time of arrival, was barely noticeable and had subsided when examined by the stroke team. It is important to note that his grip in his left hand deteriorated to 1/5 during serial examination, and this has led to a high degree of functional impairment. His NIHSS was 1, indicating a solitary, slight motor deficit.

Vital Signs

His blood pressure was 165/95 mmHg on presentation, and his heart rate, respiratory rate, and oxygen saturation were within normal physiological limits.

Investigations

Vascular and Cardiac Examination

A carotid Doppler showed normal intimal flow with no stenosis or plaque. ECG was normal with no atrial fibrillation. The 2D echocardiogram demonstrated normal cardiac chamber size, intact wall motion, and no structural lesions.

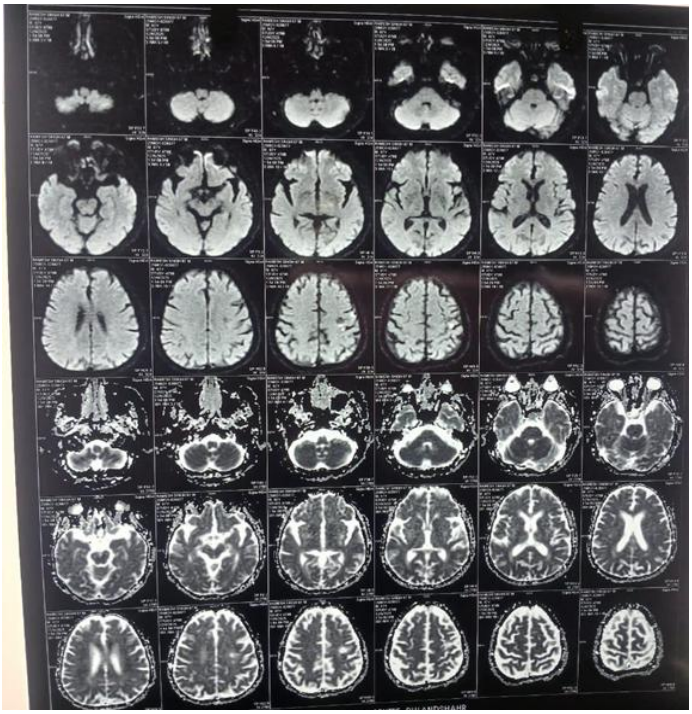
Laboratory Findings

His lipid profile showed significantly elevated cholesterol: 266 mg/dL total and 194 mg/dL LDL cholesterol; both are major modifiable vascular risk factors. Thyroid function tests and other metabolic parameters, including renal and liver function tests, were normal.

Neuroimaging

Diffusion-weighted imaging of the brain showed a small acute infarct of the right precentral gyrus, the area in the brain that is involved in the hand knob, and this area is exactly what is associated with his isolated hand weakness. This affirmed a cortical ischemic.

Image 2: Diffusion-weighted (DWI) and apparent diffusion coefficient (ADC) MRI images, which show an acute infarct in the right precentral gyrus, including the hand knob area, which is associated with isolated left-hand weakness in the patient.



Management

Aspirin 300mg and clopidogrel 300mg loading doses were administered to the patient, and the patient subsequently underwent dual antiplatelet therapy. Atorvastatin 80mg was prescribed because of the considerably high levels of LDL and secondary stroke prevention. Amlodipine 10 mg was added to his antihypertensive medication to enhance the long-term management of blood pressure. Both extensive lifestyle counselling was provided, focusing on salt restriction, physical activity, lipid control, and medication adherence.

Final Diagnosis

Cortical, right precentral gyrus (hand knob area) infarct, which has left-hand grip weakness only.

Differential Diagnosis

The infarction of the cortical hand knob (Most Likely)

The acute onset of isolated hand weakness, a pure motor deficit, and the absence of a nerve distribution pattern strongly indicated cortical pathology. MRI supported this diagnosis, showing an infarct in the right precentral gyrus.

Radial, Median, or Ulnar Neuropathy (Peripheral Nerve Palsy).

Peripheral palsies were originally suggested but were ruled out by the fact that there was no loss of sensation, no wrist drop or weakness of fingers in flexion that characterized nerve damage, and the onset was sudden, which was incompatible with compressive neuropathy.

Cervical Radiculopathy (C7–T1)

Though there may be cases of hand weakness induced by radiculopathy, the patient did not experience any neck pain, no dermatomal sensory changes, and retained reflexes. MRI brain results showed cortical pathology, eliminating radicular pathology.

Lacunar Infarct (Internal Capsule/ Thalamus)

Lacunar strokes may cause pure motor deficits, but lesions are typically wider, involving the face, arm, or leg. The presence of isolated hand involvement is unlikely, and thus the diagnosis is less likely.

Motor Neuron Disease

Motor neuron disease was also found unlikely because of the acute onset, lack of fasciculations, and the absence of progressive upper motor neuron signs.

Neurological Disorder, functional.

The objective neurological deficit and the obvious vascular risk factors in the patient, and MRI findings of an infarct ruled out the possibility of a functional cause.

Final Clinical Diagnosis: Isolated cortical stroke in the hand knob, constituted by a combination of neurological results and MRI.

Investigations

1. Neuroimaging

MRI Brain (DWI) showed a small acute infarct in the right precentral gyrus, which includes the area of the hand knob, confirming that it was a cortical ischemic stroke with isolated hand weakness. In the CT brain, an early CT would have been normal, probably because the lesion is small and located in the cortex, which, compared to MRI, is highly sensitive.

2. Vascular Studies

A normal flow pattern with no stenosis or plaque was observed on the carotid Doppler ultrasound, allowing the conclusion of no embolic etiology and suggesting a small-vessel cortical infarct.

3. Cardiac Evaluation

ECG was normal, no atrial fibrillation. The cardiac structure was in optimal condition, with no thrombus and intact septal integrity, as demonstrated by a 2D echocardiogram, minimizing the risk of cardioembolic stroke.

4. Laboratory Investigations

His lipid assessment identified a significant, modifiable risk factor, with considerable increases in total cholesterol and LDL cholesterol. Blood glucose levels, renal levels, and liver levels were normal. Tests of thyroid functioning were normal. The level of coagulation, in case of evaluation, would have been normal, considering that the ischemic episode was minor.

5. Stroke Severity Assessment

The patient's NIHSS score indicated a single mild motor impairment restricted to the left hand.

Treatment

The patient was put on acute antiplatelet therapy once it was confirmed that he had an ischemic stroke. He was loaded with aspirin 300 mg and clopidogrel 300 mg, and the dual antiplatelet treatment (DAPT) was continued in case of secondary stroke prevention because of the minor stroke (NIHSS 1), and cortical involvement. The maximum dose of atorvastatin (80mg) was started because of elevated LDL cholesterol (194 mg/dL) and the established efficacy of intensive lipid lowering, as shown by a significantly lower incidence of recurrent vascular events.

Amlodipine 10 mg was used to optimise blood pressure and aim at progressive normalisation, as suggested in the treatment of acute stroke ischaemia without thrombolysis. Counselling on lifestyle was provided, focusing on salt restriction, dietary changes, physical exercise for the patient, and strict adherence to antihypertensive and antiplatelet medicines.

Rehabilitation focused on physiotherapy, including hand-strengthening and fine-motor coordination exercises to improve grip strength. The patient was sent home with the follow-up appointment of a neurological evaluation, lipids, and management of risk factors.

Table 1: Summary of Case Details

Parameter	Details
Age / Sex	65-year-old male
Handedness	Right-handed
Vascular History	Past history of hypertension; past transient ischemic attack (TIA)
Time to Presentation	10 hours after symptom onset
Current Symptoms	Relapse of slurred speech; left-hand grip weakness
Neurological Findings	Isolated left-hand weakness (initially 3/5 → 1/5); no functional loss; cranial symptoms recovered
NIHSS Score	1 (isolated minor motor deficit)
Vital Signs	Blood pressure: 165/95 mmHg; Heart rate, respiratory rate, and SpO_2 within normal limits
Carotid Doppler	Normal; no evidence of stenosis
ECG	Normal sinus rhythm
2D Echocardiography	No structural abnormalities
Laboratory Results	Total cholesterol: 266 mg/dL; LDL: 194 mg/dL; TSH and metabolic profile within normal limits
MRI Brain	Acute infarct in the right precentral gyrus (hand knob area)
Final Diagnosis	Cortical hand knob infarction with isolated left-hand weakness
Management	Aspirin 300 mg + Clopidogrel 300 mg (DAPT); Atorvastatin 80 mg; Amlodipine 10 mg; lifestyle counselling
Rehabilitation	Hand-focused physiotherapy advised
Clinical Significance	Cortical stroke can mimic peripheral neuropathy; MRI is essential for accurate diagnosis

Discussion

Hand knob infarction is a type of rare cortical stroke with isolated weakness of the opposite hand, which clinically can cause a patient to appear to have nerve pathology of the peripheral nerves. This is a diagnostic embarrassment because the neuropathy can resemble radial, median, or ulnar neuropathy, as it presents with a pattern of weakness. Nonetheless, except for neuropathies, cortical hand knob stroke is usually unaccompanied by sensory impairment, dermatomal injury, and reflex asymmetry. The importance of early recognition is that, when a diagnosis is not made, secondary prevention is missed, exposing the individual to future events of cerebrovascular disease.[5,6]

A study of the population points to a steep rise in vascular risk factors that underlie such cortical strokes. India is still reporting a high incidence of hypertension, dyslipidemia, and metabolic disease as the main cause of small-vessel and cortical ischemia, as emphasized by national surveys and burden-of-disease investigations.[7-10] Mental and neurological disorders, such as stroke, continue to be one of the leading causes of disability-adjusted life years (DALYs), and it is difficult to overestimate the importance of identifying and vigorously treating even the mild stroke syndromes as early as possible.[5] Besides that, the National Mental Health Survey points out that neurological disorders such as cerebrovascular difficulties are not diagnosed early enough, particularly where the symptoms are subtle or uncharacteristic.[8]

Markedly high levels of cholesterol in the patient are not only compatible with the long-established links between dyslipidemia and the risks of ischemic stroke. The national epidemiological statistics indicate that a significant proportion of lipid disorders in older adults are under-controlled, which increases the frequency of the occurrence of cerebrovascular events in people with a history of hypertension.[7] The importance of risk-factor modification in the early stages is consistent with national strategies aimed at preventing noncommunicable diseases and stroke among older adults.[7,10]

Though TIA history is an established prerequisite for further ischemic stroke, late onset is frequent, as observed in this situation. National stroke registries have reported that a significant number of individuals with less severe or short-lived symptoms do not report in time to receive reperfusion therapies and are instead dependent on second-line prevention measures.[6] His clinical syndrome, characterized by rapid improvement in speech symptoms and isolated persistence of hand weakness, is the typical clinical presentation of a small cortical infarction in the precentral gyrus.

MRI is still essential in ruling out information on hand knob infarction since CT does not readily show small lesions affecting the cortex in high-convexity areas. Identification of the omega- or epsilon-shaped hand knob enables accurate localization in neuroimaging. It prevents misdiagnosis as a peripheral motor deficit, a diagnostic issue that has been brought to the forefront of the neurological literature.

Overall, the case indicates the significance of suspicion against cortical stroke in patients with isolated hand weakness, especially those whose vascular risk factors have been reported in nationwide health records. Early imaging, the establishment of dual antiplatelet therapy, intensive statin treatment, and the design of an educational rehabilitation program are all needed to maximize outcomes and prevent recurrence.

Conclusion

The present case demonstrates the significance of the identification of hand knob infarction as either an infrequent but clinically important type of stroke presenting with focal weakness in the hand only, and potentially reported as mimicking the peripheral nerve pathology. Clinicians should suspect a cortical etiology in the absence of sensory symptoms, with an acute onset of the deficit, or with vascular risk factors. The most important diagnostic test is MRI, which can detect small infarcts in the precentral gyrus that cannot be identified on CT. Early diagnosis led to the initiation of dual antiplatelet therapy, high-intensity statin treatment, excellent blood pressure management, and systematic rehabilitation, all of which are necessary to reduce the disability and avoid recurrent ischemic events. The case highlights the importance of increased clinical caution and methodical assessment of focal hand weakness in patients to prevent misdiagnosis and to enable effective stroke management.

Learning Points

Isolated hand weakness can always be an indication of a cortical stroke, especially when the person has a risk factor like high blood pressure or dyslipidemia. The area of the precentral gyrus that can be identified as having a hand knob is a prominent anatomical landmark, and infarction in the region normally produces pure motor deficiency and no associated sensory deficit. This implies that the absence of dermatomal distribution and loss of sensation, along with the absence of peripheral neuropathy, should point a clinician toward a central etiology. Diffusion-weighted MRI is necessary to make an accurate diagnosis because small cortical lesions cannot be easily detected on CT. Intensive risk-factor management, early introduction of dual antiplatelet therapy and high-intensity statins, and the effect of early intervention on minor cortical stroke are highly beneficial. Also, hand-specific physiotherapy is significant in the recovery of functional grip strength. All in all, the high level of suspicion is essential to prevent readmission due to recurrent cerebral incidents and to promptly establish an initial diagnosis.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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