Case Report

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Pseudoulnar palsy with concurrent wrist drop: case report

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Introduction and importance: Pseudoulnar palsy, characterized by weakness in the fourth and fifth digits, is a condition typically attributed to infarction of the medial aspect of the precentral gyrus's "hand knob." This anatomical site is located in the primary motor cortex of the brain, in the posterior lobe of the frontal cortex. This report presents a novel case of pseudoulnar nerve palsy in conjunction with wrist drop stemming from an infarction of the hand knob gyrus.

Case presentation: A 78-year-old female with hypertension and hyperlipidemia experienced sudden right wrist weakness and impaired mobility in her fourth and fifth digits. Clinical examinations, including neuroimaging, supported the diagnosis of an infarction in the medial precentral gyrus. Brain MRI confirmed the diagnosis of an acute infarction in the medial precentral gyrus. The patient was treated with enoxaparin, aspirin, and dexamethasone, and was discharged after symptom improvement.

Clinical discussion: Unlike the classical presentations, this case highlights the co-occurrence of ulnar and radial deficits following a unique infarction pattern. The distinct presentation of right pseudoulnar palsy with wrist drop was caused by an infarction at the level of the medial aspect of the hand knob.

Conclusion: This case underscores the importance of considering the central causes of peripheral-like deficits, especially in older individuals with vascular risk factors, emphasizing the significance of early intervention in mitigating potential long-term consequences. This report contributes to the evolving understanding of central neurological presentations, and serves as a reminder of the need for a comprehensive diagnostic approach.

Keywords: hand knob gyrus, precentral gyrus infarction, primary motor cortex, pseudoulnar palsy, wrist drop

Introduction

Isolated upper extremity weakness is predominantly attributed to an injury of the peripheral nervous system, with weakness resulting from damage of the radial, ulnar, or median nerve most commonly due to traumatic injury or compression neuropathy. Pseudoperipheral palsy, characterized by selective weakness in the radial, ulnar, or median nerve distributions without sensory deficits, rarely occurs due to central etiologies. Infarction of the precentral gyrus, often referred to as the "hand knob" area, has been established as a rare cause of pseudoperipheral palsy. Isolated weakness of the contralateral upper extremity mimics peripheral nerve damage and presents with a misleading symptomatology, raising the suspicion of peripheral neuropathy. In particular, pseudoulnar palsy is characterized by selective weakness of the fourth and fifth digits due to infarction of the medial aspect of the precentral gyrus. Classical presentations of this condition may manifest exclusively as deficits in ulnar-innervated phalanges. This case report presents a distinctive clinical scenario in which infarction in the medial precentral gyrus resulted in an unprecedented combination of pseudoulnar nerve palsy and concurrent wrist drop in the same patient.

HIGHLIGHTS

- Simultaneous symptoms of wrist drop and ulnar digit weakness in a patient with cardiovascular risk factors should be assessed with a computed tomography or MRI of the brain to evaluate for ischaemic changes.
- The site for voluntary hand muscle control is in the primary motor cortex within the precentral gyrus. The precentral gyrus is located on the lateral surface of the frontal lobe, just anterior to the central sulcus.
- The site of the ischaemia determines symptom presentation, with more medial involvement resulting in ulnar weakness and more lateral involvement resulting in radial digit weakness.

Case presentation

A 78-year-old right-handed female with a medical history of hypertension and hyperlipidemia presented with sudden onset weakness of her right wrist, along with the fourth and fifth digits. Before this episode, the patient had no history of extremity

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radiculopathy or other neurological symptoms. Upon presentation, she reported weakness and difficulty mobilizing the right wrist as well as the fourth and fifth digits. On physical examination, she was alert, without dysarthria, aphasia, apraxia, or other high cortical dysfunctions. Motor examination revealed normal ambulation, without ataxia. The range of motion and strength of the right shoulder and elbow were within normal limits. Her right wrist was weak, demonstrating limited range of motion and 4/5 strength in flexion and extension. Her fourth and fifth digits were held in slight flexion at the proximal interphalangeal joints and limited to 3/5 strength in flexion and extension. A neurological assessment revealed no cranial nerve deficits. Sensation was grossly intact in all body parts, as well as in the affected limbs. Differential diagnoses at this time included cerebrovascular accident, carpal tunnel syndrome, median/radial nerve palsy, cervical radiculopathy, and cerebral mass.

The results of routine and laboratory investigations were unremarkable. Electrocardiography revealed sinus tachycardia with a rate of 107 beats/min. Transthoracic echocardiography revealed mild calcification of the aortic valve leaflets, moderate aortic valve thickening, and normal ejection fraction. Brain computed tomography (CT) without contrast was unremarkable. CT of the cervical spine revealed moderate bilateral neural foraminal narrowing of C6–C7. CT tomography angiography (CTA) of the circle of Willis and the neck was unremarkable. MR of the cervical spine revealed disc bulges between C5–C6 and C6–C7 resulting in mild to moderate neural foramen stenosis. Brain MRI revealed diffusion restriction within the left frontal lobe, compatible with acute infarction in the medial precentral gyrus (Fig. 1). The patient was treated with Enoxaparin (40 mg, SubCutaneous, every 24 h for 3 days), Aspirin (81 mg, Oral, Daily), and Dexamethasone (10 mg, in dextrose 5% 50 ml, Intravenous, Once), and showed a gradual improvement in strength. She remained in the hospital for monitoring of her symptoms and was discharged 3 days after completion of medical therapy. The patient did not experience any adverse events and recovered well; she was given instructions to follow-up with her neurologist on an outpatient basis.

Discussion

The planned voluntary motor function of the hand originates from the primary motor cortex within the precentral gyrus. The precentral gyrus is a fissure in the lateral aspect of the frontal lobe, anterior to the central sulcus. Here lies the origin of the corticospinal tract, corticobulbar tract, and cortico-rubro-spinal tract, which are all associated with movement control. The gyrus illustrates the "knee" of the central sulcus and is referred to as a "hand knob" because of its curved shape forming an omega or epsilon sign[1,2]. As this anatomical landmark is responsible for controlling movement, an injury or infarct of this area can result in isolated paralysis of the 4th and 5th digits, also referred to as pseudoulnar palsy[2]. In particular, lesions within the hand knob predominantly involving the ulnar phalangeal distribution are located more medially[3]. Whereas, lesions involving the radial phalangeal distribution are located more laterally[3]. Additionally, the medial aspect of the hand knob corresponds to a known border-zone area associated with hemodynamic mechanisms such as severe stenosis or occlusion of the carotid artery[4]. Contrastingly, the lateral aspect of the hand knob is supplied by distal branches of the middle cerebral artery (MCA), which are subject to artery-to-artery or cardiogenic embolisms[4]. A study by Timsit *et al.*[5] reported six patients who presented with pseudoulnar palsy of the hand with infarctions localized to the junction of the anterior cerebral artery (ACA), MCA, and posterior cerebral artery (PCA) within the inferior parietal lobule. These infarctions corresponded to the border-zone area associated with the medial aspect of the hand knob.

Some cases of contemporary pseudoradial, pseudoulnar, and pseudomedian involvement associated with infarction of the hand knob have previously been described. One case report denoted an instance of pseudoperipheral palsy, in which an infarction secondary to cerebral angiitis located in the right posterior part of the precentral hand knob area led to sudden severe motor deficits of the left hand. This was characterized by weakness with extension of the three ulnar fingers and wrist, muscle atrophy in the first dorsal interosseous muscle, and claw hand deformity without sensory deficits[6]. Another case report illustrated pseudoperipheral palsy due to cerebral metastasis involving the right precentral gyrus corresponding to the hand knob area. The patient demonstrated isolated left-sided upper extremity weakness without sensory disturbance characterized by wrist drop, decreased grip strength, and positive "pinch-grip" test, suggesting a constellation of deficits involving the radial, ulnar, and median nerve distributions[7].

Furthermore, a case series of selective hand knob lesions described two cases of pseudoperipheral palsy. The first case denoted a patient with weakness of the right wrist and finger extensor muscles in the distribution of pseudoradial palsy. MRI revealed an acute left precentral gyrus infarction with restricted diffusion on diffusion weighted images along with watershed left MCA-PCA left parietal area infarction, likely embolic in nature[8]. The second case presented a patient with pure motor paresis of the muscles of the hand innervated by the involvement of the median nerve with an "ape thumb" deformity and positive "pen test." Ulnar and radial innervated muscles were unremarkable. MRI displayed a ring-enhancing lesion over the grey-white junction of the right frontal lobe involving the right precentral hand knob area. A metastatic aetiology was suspected after CT-guided biopsy confirmed adenocarcinoma of the lung[8].

Based on the aforementioned cases, pseudoperipheral palsy can manifest with deficits in the pseudoradial, pseudoulnar, and pseudomedian nerve distributions. Instances where combined manifestations have also been described, speaking to the variability in presentation of a hand knob area infarction. Clinicians must take note of the potential manifestations of this infarction, as well as the individual risk factors and lack of sensory involvement commonly displayed.

Radiographic imaging can be used in conjunction with symptom presentation to narrow the differential diagnoses. A CT scan or MRI can be used for diagnosis, and in a case series of 25 patients with ischaemic strokes at the level of the hand knob, infarction signs were visualized in the precentral gyrus[9]. In the majority of patients in this study, cerebral small-vessel disease was seen along with internal carotid artery stenosis without ulcerated plaques[9]. A subsequent case series evaluated the imaging findings of hand knob stroke patients and found that in the majority of ischaemic stroke events, there was variation in the brain lesions, with lesions being present either in the territory of the hand knob or distributed along the path of the MCA. Thirty-

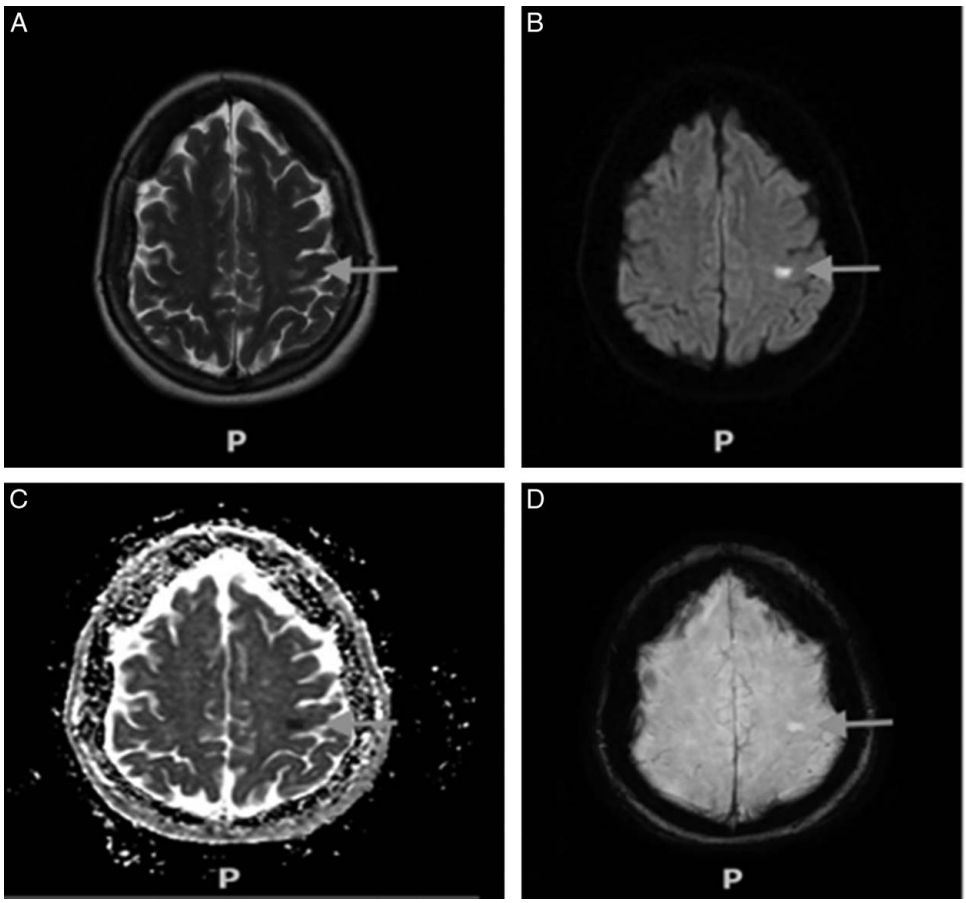

Figure 1. T2 axial view (A), diffusion-weighted imaging (B), apparent diffusion coefficient (C), and three-dimensional susceptibility-weighted angiography (D) MRI of the brain show an infarct in the left medial precentral gyrus (arrows).

one percent of these cases involved medial lesions that resulted in ulnar finger weakness[10].

Isolated wrist drops have been previously described to develop from either a central or a peripheral cause. However, most cases develop due to peripheral, neurovascular, neuromuscular, or musculoskeletal causes[11]. Takashi *et al.*[12] reported isolated radial type hand palsy in five patients with an infarction localized to the middle-to-lower aspect of the anterior wall of the central sulcus. One study reported two cases of isolated wrist drop following posterior border-zone infarctions in the MCA and PCA territory[13]. The existing literature demonstrates variability in the aetiology of central wrist drop, which physicians should be aware of.

Risk factors known to contribute to hand knob ischaemia include atherosclerosis of the supra-aortic arteries, hypertension, hyperlipidemia, smoking, and diabetes[9]. It is imperative that patients with these risk factors and the aforementioned symptom presentation be evaluated with a CT scan or MRI of the brain to workup for hand knob ischaemia.

The lesion in our patient was located in the left precentral gyrus at the medial aspect of the hand knob. This resulted in a unique presentation of right pseudoulnar palsy with a concurrent right wrist drop. To our knowledge, this is the only documented case of motor deficit related to this area of infarction. The patient was unfortunately lost to follow-up and thus there is no evidence of follow-up imaging, laboratory testing, or symptom continuity. Due to our patient's risk factors, such as old age, hypertension, and hyperlipidemia, along with a lack of evidence for acute cerebral haemorrhage, we believe that the infarction was embolic in nature. Pseudoulnar palsy and wrist drop may be caused by various etiologies; thus, it is essential to have a broad differential diagnosis and distinguish between central and peripheral causes to avoid misdiagnosis. Acute stroke must be considered in cases of apparent peripheral nerve palsies in the appropriate patient population, as early aggressive treatment may prevent permanent deficits.

Ethical approval

Approval by the ethics committee was not obtained as this report is a consolidation and interpretation of the care of one patient. During the production of this manuscript, there was no intervention that differed from standard of care for this to be considered a research study. The authors of the manuscript aim to illustrate the patient's care in an objective and informative manner. In the production of the manuscript, the patient's verbal and written consent was obtained for the writing and publication of the work.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editor-in-chief of this journal on request.
This is to certify that no personal or recognizable information was included in this manuscript.

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Author contribution

B.H.: contributed drafting of the manuscript, revising the manuscript, approving the final version to be published, and agreed to be accountable for all aspects of the work. S.W.: contributed data collection, drafting the manuscript, approving the final version to be published, and agreed to be accountable for all aspects of the work. A.E.: contributed revising the manuscript, approving the final version to be published, preparing and submitting the final version of the manuscript, and agreed to be accountable for all aspects of the work. J.H.D.: contributed revising the manuscript, approving the final version to be published, and agreed to be accountable for all aspects of the work.

Conflicts of interest disclosure

None.

Research registration unique identifying number (UIN)

Not applicable as this is a case report.

Guarantor

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Data availability statement

There were no datasets generated or analyzed during the production of the manuscript.

Provenance and peer review

This paper was not invited.

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