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Case Report

Cheiro-Oral Syndrome as a Clinical Clue to Thalamic Lacunar Infarction: A Case Report and Review of Diagnostic Challenges

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Abstract

Lacunar infarcts represent a specific category of ischemic strokes that impact deep penetrating arteries. Cheiro-oral syndrome (COS) is a rare neurological manifestation of lacunar infarcts, marked by sensory disturbances in the perioral area and the ipsilateral hand. We report a 56-year-old male presented with recurrent right-sided paresthesias affecting the face, tongue, and upper limb. Ultimately leading to a diagnosis of a lacunar infarct in the left lateral thalamus. Initial CT brain was unremarkable; however, MRI brain indicated an acute lacunar infarct in the left thalamus. This case highlights the clinical significance of identifying COS as a manifestation of thalamic stroke and stresses the necessity for heightened clinical suspicion and prompt MRI in atypical presentations to facilitate timely secondary prevention.

Introduction

Cheiro-oral syndrome represents a rare variant within the spectrum of thalamic stroke syndromes which characterized by sensory impairment in the fingers and perioral regions. Approximately 20% of all strokes, and around 25% of ischemic strokes, are classified as lacunar strokes [1]. Lacunar infarcts, affecting deep brain areas, often do not produce cortical manifestations such as neglect, aphasia, visual field deficits, or alterations in behavior. Symptoms are dependent upon the precise geographical location of the blocked artery [2]. Cases of this syndrome have been documented in the literature as a result of both ischemic and hemorrhagic infarctions, predominantly affecting the thalamo-cortical projections, thalamus, or brainstem [3]. COS is categorized as a pure sensory lacunar syndrome characterized by infarction in the ventral posterior medial (VPM) and ventral posterior lateral (VPL) nuclei when thalamic involvement is present. Deficits frequently manifest as incomplete involvement of the face or extremities, which can lead to the incorrect interpretation of this diagnosis in acute situations. We reported a 56-year-old male exhibited recurrent right-sided paresthesias involving the face, tongue, and upper limb. The MRI of the brain revealed an acute lacunar infarct in the left thalamus, following an unremarkable initial CT scan of the brain. This paper explores the literature on cheiro-oral syndrome and underscores the necessity for emergency physicians to establish a low threshold for diagnosing this subtle yet clinically significant condition. Prompt recognition enables the early identification and management of ischemic strokes. Neglecting this may postpone stroke-specific interventions and elevate the patient's risk of recurrent events, long-term disability, and mortality..

Case Report

A 56-year-old male patient, who has a documented history of hypertension and dyslipidemia, arrived at the Emergency Department (ED) reporting a 4-day history of repeated episodes of paresthesias affecting the right side of his face, tongue, and upper limb. Each episode had a duration of about 10–15 minutes, occurring spontaneously during periods of rest without identifiable triggering factors, and generally resolved on its own without the need for intervention. On the day of the presentation, the symptoms continued for two hours, leading the patient to seek medical care. During the onset, he was at his workstation when the numbness appeared abruptly and persisted longer than in previous episodes.

Upon further assessment, the patient reported a clear pattern of numbness affecting the right side of the face and tongue, which extended into the right upper limb below the elbow. The sensory disturbances impacted both the anterior and posterior regions of the

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***Figure 1**. No acute infarction nor hemorrhage detected*

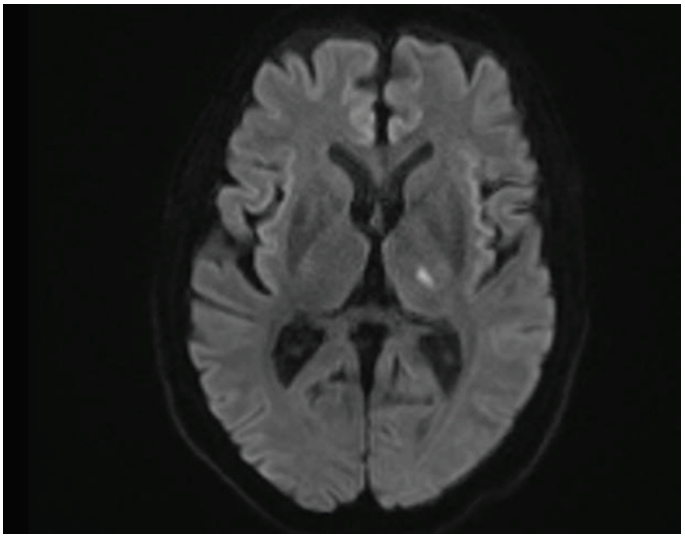

***Figure 2**. There is an acute lacunar infarction characterized by 6 mm linear diffusion restriction in the lateral left thalamus bordering the posterior limb of left internal capsule.*

arm and hand, encompassing the fingers, and did not align with a particular dermatomal pattern.

During the physical examination, the patient seemed comfortable yet exhibited mild sweating. Initial vital signs are as follows: blood pressure measured at 138/86 mmHg, heart rate at 111 beats per minute, respiratory rate at 20 breaths per minute, oxygen saturation at 99% while on room air, and temperature at 36.7°C. The measured blood glucose level was 7.2 mmol/L. The neurological examination indicated a reduction in both crude and fine touch sensation on the right side of the face, while the function of the other cranial nerves remained intact (taste sensation was not evaluated). Motor strength remained intact at 5/5 across all four extremities. The sensory examination revealed a reduction in both fine and crude touch sensation in the right upper extremity. The coordination tests, such as the finger-nose and heel-shin assessments, yielded normal results, and the patient showed no abnormalities in gait.

Preliminary laboratory assessments revealed a venous blood gas analysis indicating a pH of 7.406, potassium level at 2.75 mmol/L, sodium at 137.9 mmol/L, bicarbonate at 25.3 mmol/L, and a carboxyhemoglobin concentration of 7%. The analysis of serum electrolytes indicated a urea level of 3.42 mmol/L, creatinine at 67 µmol/L, and potassium measured at 3.0 mmol/L. A non-contrast CT scan of the brain revealed typical age-related cerebral involutional changes alongside mild small-vessel disease (subcortical arteriosclerotic encephalopathy), with no signs of acute ischemia present.

Considering the patient's unusual sensory distribution and the positional characteristics of the symptoms, it was determined that a peripheral or systemic cause was highly unlikely. Consequently, the patient was moved to the closest stroke center for additional assessment. Follow-up magnetic resonance imaging (MRI) of the brain without contrast demonstrated an acute lacunar infarct affecting the left lateral thalamus at the junction of the posterior limb of the internal capsule.

Discussion

Cheiro-Oral Syndrome is documented in the literature as exhibiting several variations. including lower extremity sensory impairment, ataxic hemiparesis, and oculomotor dysfunction [4]. Since the thalamus serves as a crucial relay center in the brain, almost all pathways that project to the cortex go through it. The ventral posterior medial (VPM) and ventral posterior lateral (VPL) thalamic nuclei, among a total of seven relay nuclei, are particularly significant in the pathogenesis of COS. The VPM processes sensory input from the trigeminal nerve, while the VPL nuclei process sensory input from the upper limbs. Structural lesions, such as infarcts, can lead to sensory deficits that are characteristic of the syndrome [5]. Lesions associated with COS are generally found at the border zone between the two nuclei. The proximity of sensory representations for the hand and face in the thalamus clarified the characteristic presentation of the syndrome. In addition, tongue involvement arises as the general sensation of the anterior two-thirds of the tongue is transmitted by the lingual nerve, a branch of CN V3, which subsequently projects to the VPM [6]. In general, lacunar lesions correlate with abnormal deep perforating arterioles; however, the reduction in size of both the brain and vascular lesions complicates the pathological examination of lacunar lesions [7]. The pathophysiology of lacunar infarction is characterized by lipohyalinosis of small penetrating arteries; thus, managing risk factors is crucial for preventing future infarctions.

COS is categorized into four different types according to the pattern of sensory involvement. Type I affects the perioral region and one side of the fingers or hand. Type II impacts the perioral region as well as the bilateral fingers or hands. Type III is distinguished by the involvement of the perioral region and fingers or hands in a mixed pattern, with one side exhibiting bilateral effects while the other side is afflicted unilaterally. Type IV manifests as sensory abnormalities in the perioral region and in the fingers or hand on the contralateral side [8]. The main risk factors consist of hypertension, diabetes mellitus, and smoking. While these conditions elevate the prevalence of lacunar infarcts [9].

The clinical suspicion of lacunar syndrome, based on the history and physical examination, led to a comprehensive work-up and a definitive diagnosis for the patient. Outpatient

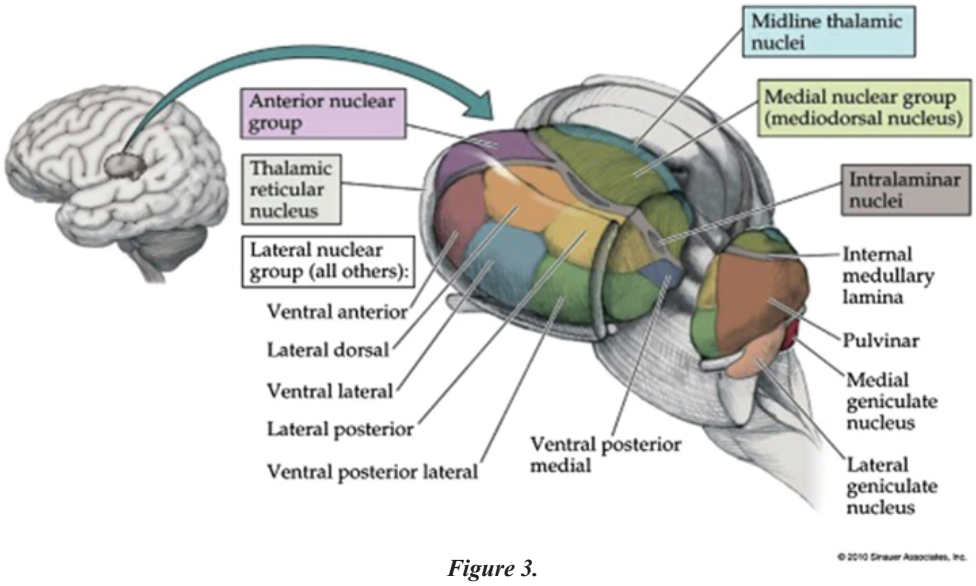

Figure 3.
https://www.sciencedirect.com/topics/neuroscience/thalamus

physicians, emergency department admitting officers, and neurologists should consider that seemingly unrelated sensory deficits may suggest the presence of a small lacunar infarction. In recent years, advanced neuroimaging has become more importance for the selection of acute stroke patients for revascularization treatment. Both Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) protocols have been validated for this purpose and applied as per guidelines; however, multimodal CT is increasingly advocated as the imaging assessment for acute stroke patients [10]. The clinical relevance of this instance exceeds its rarity. The initial standard CT scan reveals a significant restriction in the assessment of acute stroke, particularly for tiny deep infarcts. This emphasizes the necessity of sustaining a heightened index of suspicion and seeking advanced imaging, such as MRI, when symptoms are persistent or unusual. The patient's vascular risk factors, such as hypertension and dyslipidemia, reinforced the probability of a small vessel ischemic event.

This case illustrates the typical presentation of isolated sensory deficits localized to the mouth and the ipsilateral upper extremity, attributed to the proximity of these sensory homunculi regions within the thalamus. Diagnosing a stroke early may enhance the patient's adherence to long-term stroke prevention therapies, including lifestyle modifications, and underscores the significance of risk factor management to his healthcare providers. Consequently, a close outpatient follow-up was established to effectively minimize his future stroke risk.

It underscores the need of emergency department physicians identifying subtle symptoms of stroke. The prompt recognition of COS facilitated quick referral, neuroimaging, diagnosis, and the commencement of secondary preventive efforts, essential for lowering future stroke risk.

Conclusion

This case displays the common manifestation of isolated sensory deficits in the perioral region and the ipsilateral upper extremity, resulting from a lacunar infarction in the thalamus, referred to as COS. This presentation's clinical significance is rooted in its potential for misdiagnosis, attributed to its subtlety and rarity. The limitations of non-contrast CT in identifying small deep infarcts underscore the need for advanced imaging modalities, such as MRI, in specific instances. Timely identification of COS enhances early stroke diagnosis, resulting in suitable management and secondary prevention strategies. Clinicians in emergency and outpatient settings should be attentive to localized sensory symptoms, as these may indicate a lacunar stroke. This case underscores the importance of thorough clinical examination and risk factor evaluation in patients presenting with focal neurological symptoms.

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