



Clinical Reasoning: A 58-year-old man with right facial numbness

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SECTION 1

A 58-year-old man with a history of uncontrolled hypertension presented to the emergency department for evaluation of right facial numbness. He went to bed feeling normal and woke up with numbness and tingling in the right face limited to the right perioral area. He denied any associated neurologic symptoms or having similar experiences in the past. His medical history was significant for uncontrolled hypertension; surgical history was negative. Social history was pertinent for occasional alcohol use, but no smoking or drug use. Family history was significant for hypertension in both parents and essential tremor

in his sister. His only home medication was oral lisinopril (10 mg daily).

On the review of systems, he reported bilateral symmetric, kinetic, and postural upper extremity tremors, which had been progressively worsening for the past 20 years and interfering with daily activities such as eating, drinking, and writing. The tremor improved with alcohol and worsened with action and stress.

Questions for consideration:

1. What is the differential diagnosis for the right facial numbness?
2. What is the likely diagnosis for his tremor?

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SECTION 2

The differential diagnosis for acute/subacute right perioral facial numbness includes stroke (especially given the acute/subacute onset), demyelinating disease such as multiple sclerosis, tumor, infection, or psychiatric disease. This clinical syndrome can localize to the contralateral thalamus (stroke, demyelinating disease, tumor, or infection) or the ipsilateral mandibular or maxillary nerve (V2/V3), since the distribution of sensory loss is in the nerve root distribution of V2/V3.

The most likely etiology of the patient's tremor is essential tremor, given the description of the tremor, time course and progression, exacerbating/relieving factors, and the positive family history. Essential tremor manifests as a postural and kinetic tremor predominantly affecting the upper limbs.^{1,2} It tends to be progressive, leading to difficulties with activities of daily living such as writing, eating, and driving, and family history is usually positive.³

On arrival to the emergency department, the patient's blood pressure was 182/97 mm Hg, with otherwise normal vital signs and general physical examination. Cranial nerve examination revealed decreased sensation to light touch and temperature

on the right side of his face in V2 and V3 distribution compared to left, but no difference with vibration. On sensory examination, he had decreased sensation to light touch, temperature, pinprick, and vibration in his right hand and forearm. Left upper extremity and bilateral lower extremity sensation was intact to all modalities. When reaching with his left index finger toward the examiner's finger, he was noted to have an upper extremity tremor with increasing amplitude upon sustained posture. When the same examination was done with his right upper extremity, he also displayed a tremor, but the amplitude was significantly reduced compared to the contralateral side. During the examination, the patient noted that relative to the prior day, his right upper extremity tremor had improved, but his left upper extremity tremor had remained unchanged. The remainder of the cranial nerves, mental status, motor, reflexes, coordination, and gait were all within normal limits.

Questions for consideration:

1. Given the examination, where does the lesion localize?
2. What is the most likely etiology?

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SECTION 3

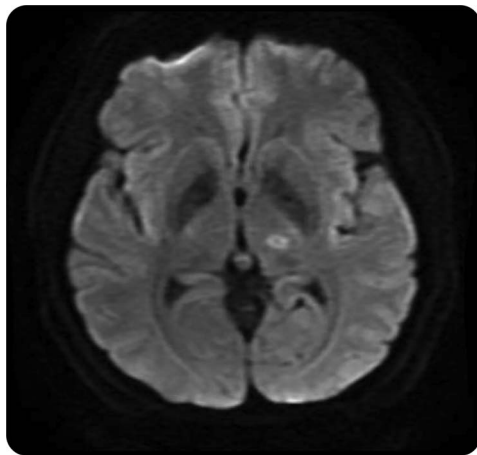
Given the patient's constellation of symptoms, we suspected a left thalamic lesion affecting the ventral posterior-lateral (VPL), ventral posterior-medial (VPM), and ventral intermediate (VIM) nuclei, which was later confirmed on MRI (figure). Clinical symptoms of decreased sensation around the patient's mouth and distal ipsilateral upper extremity suggest a cheiro-oral syndrome, which has been reported with thalamic lesions.⁴ Given the acute presentation, the etiology of this syndrome was thought to be a small left thalamic infarct. The thalamus contains the VPL nucleus that carries sensory input from contralateral limbs and the VPM nucleus that carries sensory input from the contralateral face. The classic combination

of isolated sensory loss around the corner of the mouth and in the ipsilateral arm is thought to result from a small lesion affecting the VPL and VPM nuclei in the thalamus, given their close proximity.

Our patient's essential tremor likely improved due to the contralateral infarct in the VIM nucleus, which is in close proximity to the VPL and VPM. The pathophysiology of essential tremor is not well-understood but some prevailing hypotheses include neurodegeneration, abnormal GABA functioning, and oscillatory activity in the cerebellothalamocortical circuit.⁵ It is presumed that pharmacologic agents as well as deep brain stimulation (DBS) and surgical lesions of the VIM nucleus and nearby structures interfere with this oscillatory activity and have a dramatic impact on tremor.² Although a long-term study of thalamic DBS showed clinical benefit of over 3 years in a cohort of patients with essential tremor,⁶ no long-term double-blind placebo-controlled DBS studies exist.² Stereotactic surgery or external radiation to create noninvasive focal lesions of the VIM nucleus has been shown to be an effective option for disabling essential tremor that is not responsive to drug therapy.^{7,8}

Our patient had a noncontrast head CT that showed no evidence of hemorrhage, mass effect, or large infarct. MRI diffusion-weighted sequence showed a restricted diffusion lesion involving the VPL, VPM, and VIM nuclei of the left thalamus, consistent with an acute infarct (figure).

Figure MRI diffusion-weighted imaging sequence of left thalamic infarct



Question for consideration:

1. What is the diagnostic evaluation and treatment?

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SECTION 4

The patient underwent an inpatient stroke evaluation including magnetic resonance angiography (MRA) of the intracranial and extracranial vessels that showed no significant intracranial or extracranial stenosis. In addition, he received an ECG, echocardiogram, and telemetry, which showed no evidence of a cardioembolic source. The appearance of the infarct on MRI suggested small vessel disease as the stroke mechanism, especially given his history of uncontrolled hypertension; this was further supported by his normal intracranial and extracranial vessel MRA and negative cardiac evaluation, obviating the need for further diagnostic testing.⁹ In patients with stroke secondary to small vessel disease, stroke prevention strategies include antiplatelet agents, blood pressure reduction, and lipid-lowering agents.¹⁰ The patient was therefore started on aspirin and a statin, and his antihypertensive regimen was modified for better blood pressure control. The patient's symptoms of numbness and tingling had significantly improved upon discharge the following day.

At the 2-month follow-up, the patient reported no new symptoms, the facial numbness had resolved, the right upper extremity tremor continued to improve, and there was a significant recovery of his ability to carry out daily activities that were substantially impaired prior to the stroke, such as eating and writing.

DISCUSSION Contralateral essential tremor can improve in patients who have a thalamic stroke affecting the VIM nucleus. The improvement in the tremor in our patient confirmed localization of the patient's symptoms to the thalamus.

AUTHOR CONTRIBUTIONS

Kelsey A. Egan: manuscript preparation. Jennifer L. Sevush: manuscript editing and data collection. Elvedin Lukovic: manuscript editing and data collection. Ayesha Z. Sherzai: manuscript editing. Shadi Yaghi: manuscript preparation and editing.

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