Case Report

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Ipsilateral Ankle Clonus in Cerebral Infarction: An Uncommon Clinical Phenomenon

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ABSTRACT

Clonus is defined as involuntary, rhythmic muscular contractions caused by persistent damage to descending upper motor neurons. Clonus can occur in the ankle, patella, triceps surae, wrist, jaw and biceps brachii. It is a marker of hyperreflexia and indicates disruption of upper motor neurons. These signs, especially when observed together in the right context, signal a central nervous system insult, which may result from various causes such as vascular issues (stroke), infections (encephalopathy), congenital conditions (cerebral palsy), hereditary spastic paraplegia, autoimmune disorders (multiple sclerosis), traumatic injuries (spinal cord insult, chronic myelopathy), severe hepatic failure and serotonin syndrome. In cases of cerebral infarcts, ankle clonus is typically demonstrated on the contralateral side. However, we have observed a clinical sign of ipsilateral ankle clonus following acute ischemic stroke. We believe the origin to be compression of the left crus cerebri as part of the Kernohan-Woltman notch phenomenon. We consider that ipsilateral ankle clonus in acute cerebral infarction is an early indicator of poor neurological outcome before the sensorium drops and its disappearance indicates a promising prognosis for stroke recovery.

Keywords: Clonus, Hyperreflexia, Upper motor neuron syndrome, Ipsilateral cerebral infarction

1. Introduction

Ipsilateral ankle clonus in cerebral insult is very rare entity and its presence indicate early clue for raised cerebral oedema before the sensorium worsens, hence examination of ipsilateral limb for ankle clonus and other UMN signs is important to decide regarding early antioedema intervention.

2. Case Presentation

A 56-year-old man with a history of diabetes and hypertension who had been off medication for one week complained of acute onset of left upper and lower limb weakness, facial deviation to the right and slurring of speech. Within 2 hours of symptom onset, he was transported to the emergency for a comprehensive neurological assessment. On examination, his computed NIHSS score was 18. GCS (Glasgow coma scale) was E2 V3 M 4 (9/15). He was found to have left hemiparesis, hyperreflexia and expressive aphasia. To our astonishment, the patient had a bilateral plantar extensor and a well-sustained right ankle clonus which is ipsilateral to the side of the infarction. However, the patient neither had a prior history of a stroke in the left cerebral hemisphere nor had he previously experienced cervical myelopathy symptoms. The initial CT scan of the brain showed no acute bleed. The patient received thrombolysis with 0.25 mg/kg of body weight of tenecteplase. Later, an MRI brain scan revealed evidence of a right Middle Cerebral Artery (MCA) territory infarct involving the cortex and subcortical region (Figure1 and 2).

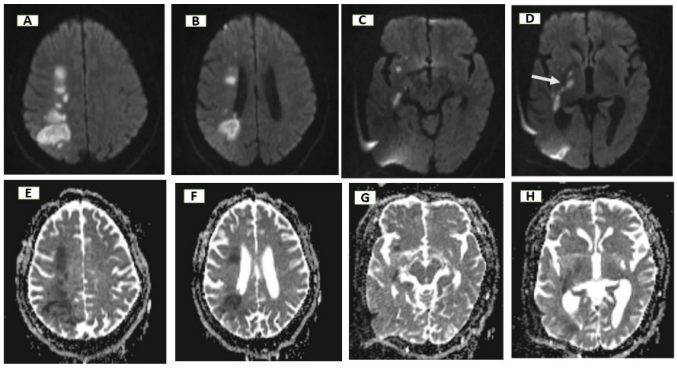

Figure 1: Showing restricted diffusion in right posterior limb of internal capsule, capsuloganglionic region, frontal and parietal cortical and subcortical region (A, B, C, D) with hypo intensity in ADC in corresponding regions (E, F, G, H) suggestive of acute infarct in right MCA territory infarct.

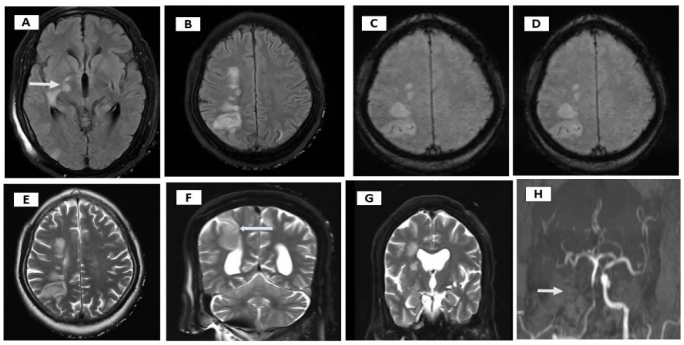

Figure 2: Showing T2 flair hyperintensity in right capsuloganglionic compressing over right crus cerebri (A), right frontoparietal cortex and subcortical region hyperintensity (B,C,D), T2 hyperintensity in right frontoparietal cortex (E), large right frontal hyperintensity in coronal section (marked with arrow) compressing ipsilateral ventricle (F), T2 hyperintensity in right frontal, right capsuloganglionic region (G) and non-visualization of right internal carotid artery (H). Features consistent with right MCA territory large infarct with mild mass effect.

The patient was examined daily for ankle clonus. It was present on the first and second days of assessment, which coincided with the patient's worsening neurological condition [high blood pressure and worsening sensorium]. Antiedema measures and strict control of hypertension were done during subsequent days. However, on day three of the stroke, the ankle clonus became ill sustained and the patient's neurological state improved. After the patient's clonus disappeared, he was discharged in a stable condition. An MRI of the cervical spine was performed to rule out cervical myelopathy. Upon receiving informed consent from the patient's attendants, a video of the ankle clonus was captured at regular intervals over the first five days following the stroke onset [Video]. Clonus is described as involuntary, rhythmic muscle contractions resulting from persistent damage in descending motor neurons[1]. Clonus can occur in various muscles, including the ankle, patella, triceps surae, wrist, jaw and biceps brachii. It can happen in any muscle at a frequency of 5-8 Hz and the average duration of oscillations for ankle clonus is 160-200 ms. Plantar Flexion (PF) accounts for 45% of the time, while Dorsiflexion (DF) accounts for 55%. The first beat is always longer, then the time shortens in subsequent beats before becoming stable in the fourth or fifth period. The specific mechanism of clonus remains unknown. Two alternative explanations have been proposed for the development of clonus. The most accepted explanation is that the hyperactive stretch reflexes in clonus are caused by self-excitation. Another alternative explanation for clonus is central generator activity, which arises as a consequence of appropriate peripheral events and produces rhythmic stimulation of the lower motor neurons.

The presence of clonus during a physical examination indicates hyperreflexia, which is a sign of upper motor neuron syndrome[2]. This condition is usually accompanied by other upper motor neuron signs such as spasticity and weakness[3]. When these signs are observed together in the right context, they can point to a central nervous system insult. Ankle clonus can occur in various conditions including stroke, infective encephalopathy, cerebral palsy, multiple sclerosis, traumatic myelopathy, hereditary spastic paraplegia, severe hepatic failure and serotonin syndrome[4]. Ankle clonus occurs contralateral to a cerebral lesion, while it occurs ipsilateral in spinal cord pathology due to the crossing of the corticospinal tract at the lower medulla. Ipsilateral ankle clonus in cerebral lesions is rarely reported. The Kernohan-Woltman notch phenomenon may explain the mechanism behind this occurrence[5]. Compression of the contralateral cerebral peduncle against the tentorial edge is caused by the displacement of the brain tissue at the ipsilateral side of the paradoxically supratentorial localized lesion, resulting in ipsilateral ankle clonus.

The case highlights that routine neurological examinations should include an assessment for ankle clonus, even on the non-paralyzed side, as it can be a sign of poor neurological outcome in the early stages. It indicates the need for anti-oedema measures and strict control of hypertension, which can reverse the clonus, as was the case in our situation. Therefore, the presence of ipsilateral ankle clonus is an early marker of raised intracranial pressure and a poor neurological condition and its disappearance suggests a promising prognosis for stroke recovery. Further large-scale studies are needed to confirm our findings.

3. References

1. Boyraz I, Uysal H, Koc B, et al. Clonus: definition, mechanism, treatment. Med Glas (Zenica). 2015;12(1): 19-26.
2. Mummidisetty CK, Bohorquez J, Thomas CK. Automatic analysis of EMG during clonus. J Neurosci Methods. 2012;204(1): 35-43.
3. Ivanhoe CB, Reistetter TA. Spasticity: the misunderstood part of the upper motor neuron syndrome. Am J Phys Med Rehabil. 2004.
4. Kheder A. Nair KPS. Spasticity: pathophysiology, evaluation and management. Pract. Neurol. 2012;12: 289-298.
5. Beucler N, Cungi PJ, Baucher G, et al. The Kernohan-Woltman Notch Phenomenon: A Systematic Review of Clinical and Radiologic Presentation, Surgical Management and Functional Prognosis. J Korean Neurosurg Soc. 2022;65(5): 652-664.