Unilateral Thalamic Infarct Presenting as a Convulsive Seizure

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ABSTRACT
Lesions of the thalamus and those extending into midbrain can cause various types of movement disorders such as dystonia, asterixis, and ballism-chorea. Seizures are rare manifestation of thalamic disorder. Occurrence of seizures in bilateral thalamic infarct has been reported; but seizures in unilateral thalamic infarct have been reported very rarely. Literature review showed only single case of perinatal unilateral thalamic infarct presenting with seizures. We are reporting a unique case of convulsive seizure at the onset of unilateral thalamic infarct in an adult male, which has never been reported to the best of our knowledge.

Key Words: Thalamic infarct. Movement disorder. Seizures.

INTRODUCTION
Early-onset seizures in ischemic stroke occur most commonly in cardio embolic stroke involving large cortical area or those involving multiple lobes of the brain.¹ Thalamic lesions usually cause various types of movement disorders like dystonia, asterixis, and ballism-chorea.² Convulsive seizures have been reported with bilateral paramedian thalamic infarctions,³ but they are rarely reported with unilateral thalamic infarct. Literature review showed only a single case of perinatal unilateral thalamic infarct presenting with seizures.⁴ Here, we are reporting a unique case of convulsive seizures as initial manifestation of unilateral thalamic infarct in an adult male which has never been reported to the best of our knowledge.

CASE REPORT
A 50-year man, known case of hypertension and diabetes mellitus, presented to our emergency department with three to four episodes of generalized tonic clonic (GTC) fits associated with uprolling of eyes, jaw tightness, frothing from mouth, followed by loss of consciousness. The duration of episodes ranged about three to five minutes and then patient remained unconscious at home. Two days later, family brought him in emergency department. On admission in emergency, his blood pressure was 140/110 mmHg, pulse was 84 beats/minute, and the neurological examination revealed that the patient was comatose with Glasgow coma scale (GCS) of 10/15; pupils were bilaterally equal and reactive with upgoing planters. Neck was supple. His blood sugar and metabolic profile was normal. Urgent MRI brain was performed and showed unilateral high signal intensity in the left thalamic and the restriction of water diffusion was confirmed on the apparent diffusion coefficient (ADC) maps. There was also a dilation of all the ventricles representing communicating hydrocephalus. Magnetic resonance angiography (MRA) showed segmental narrowing with markedly reduced peripheral cortical branches in both anterior and posterior circulations (Figure 1).

His baseline tests including ECG, Chest X-ray, complete blood count, urea, creatinine, electrolytes, liver function tests and urine analysis were normal. The lumber

Figure 1: DWI brain shows hyper-intense signals and ADC shows restriction in left thalamic area showing infarction and high signal on T2WI and FLAIR.
Unilateral thalamic infarct presenting as a convulsive seizure

puncture was done showing normal CSF analysis and negativity for herpes simplex virus (HSV) PCR. He was admitted in neurology ward and started on aspirin, 75 mg/day, atorvastatin, 10 mg/day per nasogastric tube and injectable levetiracetam 500mg, BID. One day after admission, he started regaining consciousness but remained sleepy throughout the day. He did not take initiative to speak but could answer simple questions. He was able to move all his limbs. Neurological examination remained normal. Electroencephalograph (EEG) showed diffuse cerebral dysfunction. There was no fit during the hospital stay. He was able to recognize family members and follow simple commands, but remained drowsy on and off till discharge.

**DISCUSSION**

The association between seizures and thalamic lesion is understandable as reticular thalamic nucleus is regarded as a pacemaker; and it can transform rhythmic cortical oscillations into generalized slow wave activity and generalized epilepsy.\(^5\) The thalamus is central structure with compact nucleus. It has critical role in global brain function. Its major blood supply comes from posterior circulation. Bithalamic infarctions are infrequently reported and represent 0.6% of ischemic stroke.\(^6\) Overall early onset post-ischemic seizure rate is 2-4%. Seizures are more common in the distribution of anterior circulation and are most likely due to acidosis, brain edema, altered electrolyte imbalance and neurotransmitter activity.\(^7,8\)

In literature, seizures have been reported to occur with bilateral paramedian thalamic infarcts with occlusion of artery of Percheron.\(^3\) However, evidence for seizures due to unilateral infarct is scare. In one case series of three children, two patients had unilateral perinatal lesion, and the third one had tumor. It was noted that their EEG record exhibited bilateral spike wave discharges.\(^5\) Our case is unique in that it occurred in an adult who presented with 3 to 4 episodes of GTCs. On workup, his MRI brain showed acute unilateral thalamic infarct. No other abnormality could be detected to account for seizures.

In summary, seizures could be the initial symptom of unilateral thalamic infarct and should be recognized early because seizure control along with acute ischemic stroke management is important for better outcome of the patient.

**REFERENCES**