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Short-latency afferent inhibition remains intact without cortical somatosensory input: Evidence from a patient with isolated thalamic infarct

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To the Editor

Short-latency afferent inhibition (SAI) is a transcranial magnetic stimulation (TMS) paradigm elicited by pairing a peripheral nerve electrical stimulation with TMS of the motor cortex. Therefore, SAI is suggested as a neurophysiological marker of sensorimotor integrity and excitability [1]. Even though it is widely used in neurophysiological studies, the exact physiological mechanism that produces SAI still remains unclear.

We report a case of thalamic infarction with an absent N20 component of somatosensory evoked potential (SEP). We aimed to evaluate neural circuits involved in SAI together with inadequate sensory input.

Case report

A 72-year-old right-handed female patient was admitted to the emergency department of our hospital with sensory loss at the left side of her body, including her face. Neurological examination confirmed sensory deficit with light touch, pinprick and vibration across the left side of the body compared to the contralateral side. Cranial nerve testing was intact, along with normal motor and cerebellar examination findings. Babinski sign was absent. The past medical history revealed hypertension, and the patient was taking anti-hypertensive medications. Diffusion-weighted magnetic resonance imaging (DW-MRI) showed acute ischemic infarction in the right posterolateral thalamus (Figure- 1A). Short-term dual antiplatelet (acetylsalicylic acid and clopidogrel) therapy was initiated, and the patient was further investigated for stroke etiology in the stroke unit. The left hemisensory loss gradually improved in four weeks.

The neurophysiological evaluation was held on the seventh day of the stroke. Median SEP was obtained by stimulating the median nerve at the wrist. Peripheral (N9), spinal (N13) and cortical (N20) potentials were recorded bilaterally. N20 potential was absent in the right hemisphere while obtained in the left hemisphere (N20 = 19.7 ms) (Figure- 1B).

Motor evoked potentials (MEP) were acquired from the abductor pollicis brevis (APB) muscle by transcranial magnetic stimulation of the opposite primary motor cortex (M1). Minimum TMS intensity required to evoke a MEP of 1 mV amplitude (motor threshold for 1 mV MEP-MT) was measured in both hemispheres. Conditioning electrical stimulation intensity was adjusted to produce 1 mV compound muscle action potential (CMAP) from APB muscle applied to the median nerve at the wrist. Data acquisition and stimuli output were controlled in QTRAC software (Hugh Bostock, Institute of Neurology, Queen Square, London, United Kingdom). SAI was evaluated at 18, 19, 20, 21, 22, 23, 24, 25 ms interstimulus intervals (ISIs). MEPs evoked by conditioned stimulation were expressed as the percentage of MEPs evoked by TMS alone for each ISI. MT of the right hemisphere was considerably higher (84%) than the left hemisphere (54%). Mean (±SD) MEP amplitudes evoked by test stimuli were 1,01 ± 0,54mV in the affected (right) hemisphere and 1 ± 0,23 mV in the unaffected (left) hemisphere without any significant difference in the Mann-Whitney-U test (Z = −0,148; p = 0,924). Nevertheless, SAI could be obtained from both hemispheres, and inhibition values were similar in most ISIs for each side (Figure- 1C).

Discussion

Although the SAI mechanism is thought to have a direct cortical inhibitory connection between somatosensory (S1) and motor cortices (M1), the situation is more complex. Indeed, considering the relation of cerebral cholinergic activity and SAI, it seems that alternative and complex pathways may have a role [2].

Oliviero et al. reported a hypersomnic patient with paramedian thalamic infarction along with loss of SAI while SEP is intact [3]. The authors proposed that blockade of excitatory paramedian thalamocortical projections (which are modulated by pontomesencephalic cholinergic neurons) leads to altered mental status and insufficient response of cortical neurons to sensory input. Additionally, they suggested that the main generator of SAI is located in the

Abbreviations: SAI, Short Latency Afferent Inhibition; TMS, Transcranial Magnetic Stimulation; SEP, Somatosensory Evoked Potential; DW-MRI, Diffusion-weighted Magnetic Resonance Imaging; MEP, Motor Evoked Potential; APB, Abductor Pollicis Brevis; M1, Primary Motor Cortex; CMAP, Compound Muscle Action Potential; MT, Motor Threshold; ISI, Interstimulus Interval; S1, Somatosensory Cortex.

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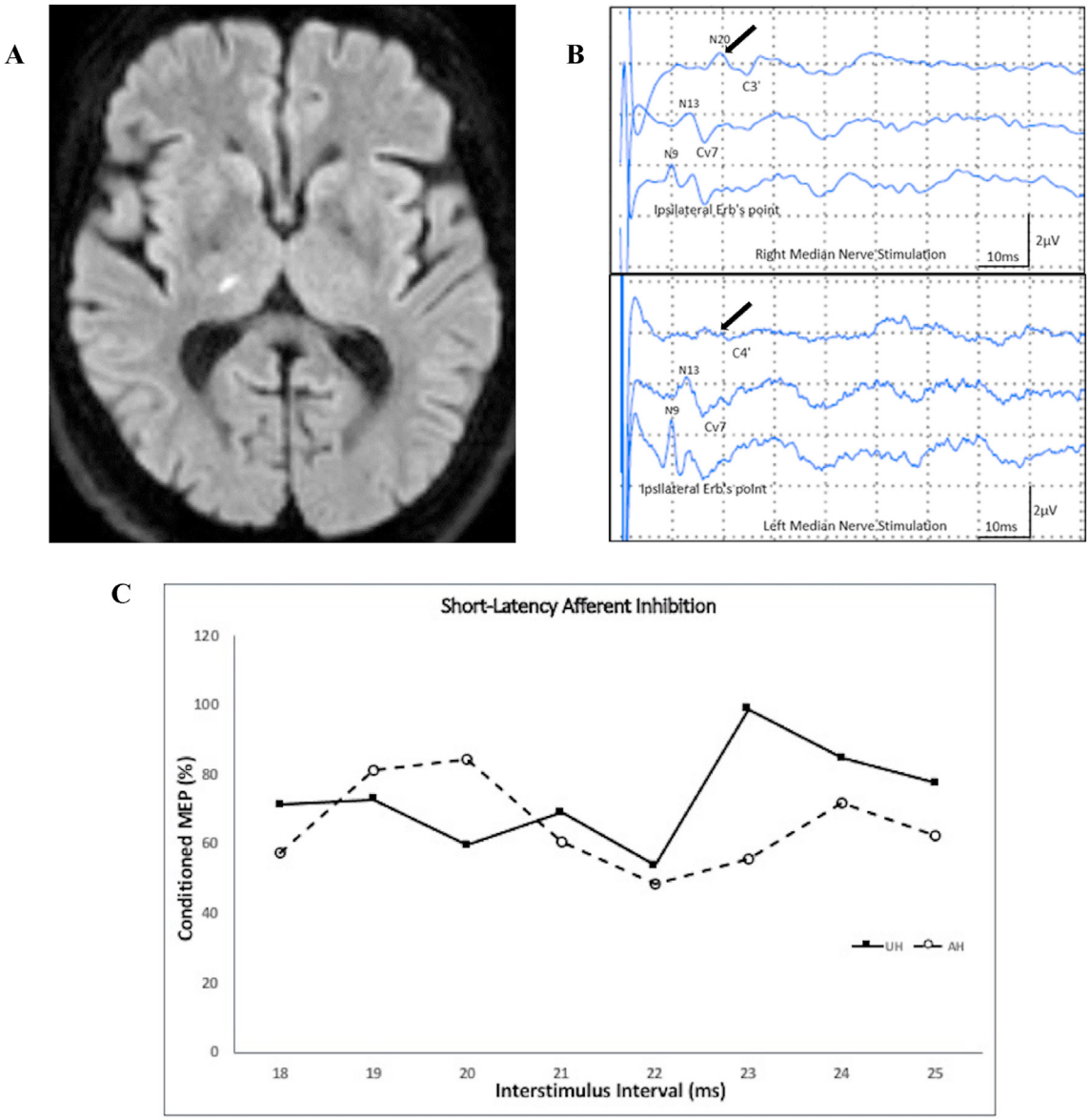


Fig. 1. A) DW-MRI of the patient showed acute ischemic infarct in the right posterolateral nuclei of the thalamus. B) Median nerve SEPs were obtained by averaging 2000 potentials from each side and recorded from the cortex, C7 spinal vertebrae and Erb's point, respectively. Arrows indicate the expected N20 peak latencies in both hemispheres C) Short-latency afferent inhibition at ISIs of 18–25 ms. Notably, both hemispheres have maximum inhibition at 22 ms ISI, which corresponds to N20+2 ms of the unaffected hemisphere (UH: Unaffected hemisphere; AH: Affected hemisphere).

paramedian thalamus. The findings of our patient, normal SAI with posterolateral thalamus infarct, supports this hypothesis. Besides, our findings suggested that SAI may not originate via relays from S1 to M1 solely. The motor threshold for 1 mV MEP was markedly increased in the affected hemisphere than the unaffected hemisphere in our patient. This finding is consistent with a study that reported increased cortical MEP latency and unable to obtain MEP in some patients with thalamic hemorrhage [4]. Moreover, two weeks after the evacuation of the thalamic hematoma, the cortical MEP of the upper and lower extremities was recorded in all of the patients, which demonstrated decreased MEP latency and increased amplitude compared with values that were obtained on admission. Taken the findings together, we conclude that posterolateral thalamic infarct disturbs projections from the thalamus to the sensory cortex, as well as decreases motor cortical excitability in the affected hemisphere. These findings favor the thalamus as the primary generator of SAI. However, the somatosensory cortex may have a modulatory role as reported previously [1,5]. Further studies evaluating afferent inhibition in subjects with S1, M1 and thalamic lesions may provide detailed information about the roles of thalamic nuclei and thalamocortical projections.

This case made us think that SAI is a complimentary neurophysiological test with SEP in patients with sensory symptoms/signs and that performing both tests together in cases may offer the clinician to be able better localization of the sensory involvement.

In summary, we believe that this case is instructive since it shows that SAI can be preserved even in the case of cortical sensory input defect.

Declaration of competing interest

None.

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