Case Reports & Case Series (CRP)

Neuroanatomical considerations of isolated hearing loss in thalamic hemorrhage

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ARTICLE INFO

Article history:
Received 14 July 2016
Revised 31 July 2016
Accepted 31 July 2016
Available online xxxx

Keywords:
Thalamus
Thalamic syndrome
Hearing loss

ABSTRACT

Background and importance: Thalamic lesions are associated with a wide variety of clinical syndromes. Due to the close anatomical proximity of the nuclei, many of these syndromes have considerable overlap in clinical sequelae and, as such, a lesion affecting only one modality is exceedingly rare. Clinical presentation: In this case, a 55 year-old right handed man with a past medical history significant for hypertension, polysubstance abuse, and a 25 year history of seizure disorder following clipping of a middle cerebral artery aneurysm presented with isolated bilateral hearing loss. Conclusion: Presumably, this neurological deficit was caused by a hypertensive hemorrhage in the posterior right thalamus. The following case and discussion will review the potential neuroanatomical pathways that we suggest could make isolated hearing loss be part of a “thalamic syndrome.”

1. Introduction

Thalamic nuclei are composed of 5 major functional classes: sensory nuclei that relay inputs from all sensory modalities, effector nuclei concerned with motor function and aspects of language, reticular nuclei that subserve arousal and nociception, associative nuclei that participate in high-level cognitive functions, and limbic nuclei concerned with mood and motivation. Damage to thalamic nuclei has consistently been found to induce a variety of motor, sensory and neuropsychological impairments with varying degrees of overlap between the functionally distinct modalities. To date, a consistent clinical picture of a “thalamic syndrome” has not emerged. Due to the close anatomical proximity and overlapping vascular supply, a lesion which affects only one modality is exceedingly rare [1–3]. In this case, a patient presented with isolated bilateral hearing loss from a presumed hypertensive hemorrhage in the posterior right thalamus.

2. Case report

The patient is a 55 year-old right handed man with a past medical history significant for hypertension, polysubstance abuse and a 25 year history of seizure disorder following clipping of a middle cerebral artery aneurysm. The patient presented to the Emergency Department after suffering three generalized tonic-clonic seizures over the 24 h prior to admission. The patient reported that over the past 25 years he has had multiple seizures (approximately one per month). His antiepileptic medication had been switched to keppra recently and his seizure frequency had subsequently significantly decreased (no seizure episodes in the prior 2 years). On the date of admission the patient suffered three generalized tonic-clonic seizures. After the third seizure that reportedly lasted greater than 10 min the patient developed profound hearing loss and tinnitus. The seizures were described as generalized tonic-clonic, the last 2 seizures prior to presentation reportedly lasted 10 min each separated by a 30-minute interval. The patient reported loss of consciousness with all seizure episodes and postictal confusion. The patient’s neurologic exam on presentation to the emergency department was awake, alert, and oriented to person place and time, comprehension was intact, with normal writing and fluent speech. Cranial nerves II–XII were intact and bilaterally symmetric except bilateral hearing loss. Detailed hearing examination demonstrated that Weber test was midline, while Rinne test was significant for bilateral sensorineural hearing loss. The patient’s motor exam was normal and symmetric in all extremities and sensation to light touch, pin prick and joint position sense were intact. A head CT scan revealed an isolated 1.5 by 1.0 by 0.5 cm hemorrhage centered in the posterior aspect of the right thalamus, along with multiple clips in the region of the left middle cerebral artery (Fig. 1A). An MRI was unable to be performed for safety concerns due to the possibility of incompatible aneurysm clips. A CT angiogram was done which did not show any vascular anomalies. However, audiometric testing on admission revealed severe bilateral sensorineural hearing loss. On admission, the patient was started on a second seizure medication. He had no further seizures during his hospital stay and showed no signs of further neurologic deterioration. The Otolaryngology service was consulted and determined that

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http://dx.doi.org/10.1016/j.inat.2016.07.003
2214-7519/© 2016 Published by Elsevier B.V.
there was no evidence of cochlear involvement and that the patient’s
deficits were likely isolated sensorineural hearing loss. Audiometric
testing done on hospital day 3 showed no improvement over the initial
test. Repeat head CT showed the clot to be stable in size without mass
effect or signs of hydrocephalus, and so the patient was discharged
home in stable condition on hospital day 3 (Fig. 1B). Following dis-
charge the patient failed to follow up with any of the clinical services in-
volved in his care.

3. Discussion

Thalamic lesions are associated with a wide variety of clinical syn-
dromes. Due to the close anatomic proximity of the nuclei, many of
these syndromes have considerable overlap in clinical sequelae and, as
such, a lesion affecting only one modality is exceedingly rare. In this
case, a patient presented with isolated bilateral hearing loss from a pre-
sumed hypertensive hemorrhage in the posterior right thalamus. This
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