

Pseudotumoral Posterior Fossa Infarction

Clinical Image

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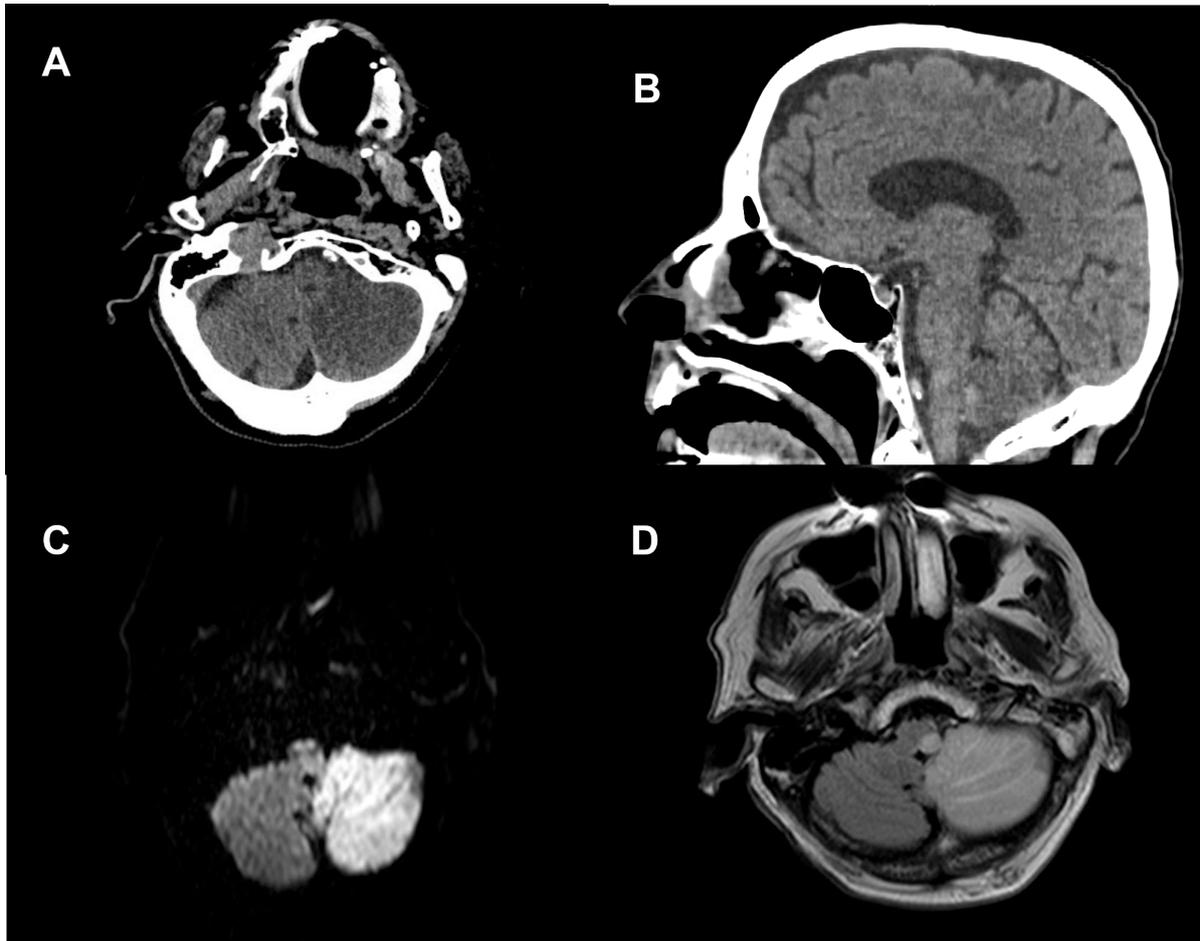


Figure 1: Panel 1,2, 3, and 4. 1-A and 1-B non-enhanced CT scan showing a posterior fossa tumefactive hypodense lesion in the left cerebellar hemisphere and cortical and subcortical atrophy. 1-C Diffusion-weighted imaging (DWI) revealed restriction in the left cerebellar hemisphere. 1-D FLAIR sequence with a hyperintense lesion involving the left cerebellar hemisphere and dorsolateral medulla.

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A 67-year-old man with a history of hypertension, diabetes, and prostate cancer presented with an episode of acute vertigo and unstable gait. Neurologic examination showed left dysmetria, dysdiadochokinesia, and left ataxic gait. The patient was alert, with no fluctuations in consciousness (NIHSS score of 2 points). CT scan revealed a hypodense lesion in the left cerebellar hemisphere (Figura 1, Panel A,B), while MRI revealed a complete posterior inferior cerebellar artery (PICA) infarction with medullary involvement (Figura 1, Panel C,D).

Pseudotumoral infarction comprises a life-threatening condition characterized by increased intracranial pressure with space-occupying edema. We believe the patient's generalized cortical and subcortical atrophy compensated in a way that surgical intervention was unnecessary. After extensive workup, we classified the underlying mechanism of infarction as an embolic stroke of unknown source (ESUS). Cerebellar infarcts are uncommon, accounting for 1.5% to 3% of all ischemic strokes with a mean age of 65.¹ Of all cerebellar infarctions, posterior inferior cerebellar artery ischemic (PICA) strokes are the most common and produce extensive infarcts, about 40% of all cases.^{2,3}

The clinical manifestations of cerebellar infarcts are diverse, unspecific, and often mistaken for other conditions. Space-occupying cerebellar infarction may be misdiagnosed as cerebellar glioma (pseudotumoral).^{1,3} Pseudotumoral cerebellar infarction may lead to complications in the posterior fossa, such as brainstem compression and obstructive hydrocephalus, intracranial hypertension, and malignant edema. Medical management comprises elevating the head of the bed, blood pressure control, and the use of neuroprotective measures (normothermia, normocapnia). Older patients are less likely to suffer the consequences of cerebral edema due to increased intracranial compliance obtained from aging-related cortical atrophy.⁴ In case of mass effect

resulting in hydrocephalus, tonsillar herniation, and brainstem compression, insertion of an external ventricular drain with suboccipital decompressive craniectomy may be an option, although decompression is the gold standard.⁴ Altered consciousness establishes the need for surgical protocol.

KEY POINT:

Pseudotumoral posterior fossa stroke comprises a potentially life-threatening condition due to increased intracranial pressure. Cortical atrophy may help in this setting allowing shifting pressures to avoid complications and decrease the need for surgical intervention.

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