

Chronic PICA Territory Infarction Presenting as Isolated Inferior Cerebellar Peduncle Atrophy: A Case Report and Quantitative Radiological Analysis

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Case Report

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Abstract

Unilateral inferior cerebellar peduncle (ICP) atrophy is an uncommon, subtle neuroradiological finding that can easily be overlooked on routine brain MRI [2]. We present the case of a 55-year-old female who presented with a 1-year history of persistent vertigo, imbalance, and intermittent headache [8, 12]. Magnetic resonance imaging demonstrated marked asymmetric atrophy of the left ICP, measuring 5.29 mm at its midpoint compared with 8.82 mm on the preserved right side (a ~ 40% reduction in transverse diameter), alongside complete loss of its distal tract and compensatory *ex vacuo* CSF space expansion in the left cerebellopontine angle cistern [8, 10, 16]. These findings reflect chronic ischemic insult in the posterior inferior cerebellar artery (PICA) territory complicated by secondary Wallerian degeneration [4, 11, 14]. This case emphasizes the clinical necessity of systematically scrutinizing the cerebellar peduncles and utilizing quantitative metrics when evaluating chronic vestibular symptoms [12, 16].

INTRODUCTION

The inferior cerebellar peduncle (restiform body) serves as a vital neural conduit within the vestibulocerebellar pathways, transmitting critical afferent proprioceptive and vestibular input from the spinal cord and medulla oblongata to the cerebellum [8, 10, 15]. Vascular insult involving the posterior inferior cerebellar artery (PICA) traditionally manifests in the acute setting with cerebellar or lateral medullary syndromes (Wallenberg syndrome) [1, 3, 5, 7]. However, chronic sequelae presenting primarily as focal, isolated peduncular degeneration remain under-reported in radiological literature [16].

Recognizing subtle structural volume loss within the cerebellar peduncles is crucial, as standard cortical imaging may appear largely unremarkable despite substantial underlying neural degeneration [2, 6, 9, 14]. Here, we describe a case of chronic PICA territory infarction presenting as isolated left ICP atrophy with complete distal fiber effacement, detailed through quantitative MRI measurements [10, 16].

CASE PRESENTATION

A 55-year-old female presented for neurological evaluation with a 12-month history of persistent, non-positional vertigo and intermittent occipital headaches [8, 12]. She characterized the vertigo as a continuous "internal swaying" sensation accompanied by mild gait instability [8, 12]. She denied acute focal motor weakness, dysarthria, diplopia, or sensory disturbances [1, 3]. Her past medical history was notable only for primary hypertension managed with oral antihypertensives. There was no history of prior head or neck trauma, neurosurgical procedures, or documented acute cerebrovascular events [13, 15].

Neurological examination revealed a conscious, oriented patient with intact cranial nerve function [1, 3]. Cerebellar testing demonstrated no upper limb dysmetria or intention tremor; however, subtle tandem gait imbalance was elicited [7, 8]. Otoneurological evaluation ruled out peripheral vestibular etiologies [12].

Magnetic resonance imaging (MRI) of the brain was performed to investigate the etiology of her persistent vestibular symptoms:

Coronal T1-Weighted Post-Contrast MRI

Demonstrated striking asymmetric volume loss of the left ICP [2, 10, 16]. Comparative caliper measurement at the mid-medullary level revealed a right ICP width of **8.82 mm** (normal caliber) compared with **5.29 mm** on the left, corresponding to a ~40% reduction in transverse diameter and complete distal loss of the peduncular fiber bundle entering the cerebellum [10, 16]. No pathological tissue contrast enhancement or mass effect was observed [2] Fig. 1.

Axial T2-Weighted MRI

Confirmed focal structural volume loss at the left dorsolateral medulla and inferior peduncle level [1, 8, 16]. Prominent focal T2 hyperintensity was noted within the left cerebellopontine angle cistern, representing compensatory *ex vacuo* expansion of the subarachnoid CSF space rather than a primary cyst or mass [8, 9]. No acute diffusion restriction or active T2/FLAIR parenchymal signal changes were present [2, 5] Fig. 2.

DISCUSSION

The ICP carries pivotal afferent inputs, including the dorsal spinocerebellar tract, olivocerebellar fibers, and vestibulocerebellar tracts [8, 10, 15]. Perfusion to the ICP and adjacent dorsolateral medullary/tonsillohemispheric structures relies on the branches of the PICA [10, 11, 15]. While acute occlusion yields prominent cytotoxic edema and acute neurological deficits [1, 5, 6], chronic vascular insufficiency or an unrecognized subclinical thromboembolic event can lead to localized, progressive neural collapse via **Wallerian degeneration (WD)** [4, 14, 16].

WD involves progressive anterograde disintegration of axons and their myelin sheaths following proximal cellular or axonal injury [4, 14]. In our patient, the complete effacement of the distal ICP fibers on coronal T1-weighted imaging illustrates the end-stage structural outcome of this process [10, 14, 16]. The expanded CSF space in the adjacent left cerebellopontine angle cistern further illustrates *ex vacuo* dilatation secondary to parenchymal retraction [8, 9].

Differential Diagnosis

Accurate interpretation of focal cerebellar peduncle atrophy requires differentiation from other posterior fossa pathologies:

1. **Multiple Sclerosis (MS):** Demyelination frequently affects the cerebellar peduncles; however, MS typically presents with multifocal T2/FLAIR hyperintense plaques and hyperintense demyelinating lesions rather than pure focal volume loss without signal abnormality [2].
2. **Multiple System Atrophy (MSA-C):** MSA-C causes olivopontocerebellar atrophy, but it characteristically exhibits symmetric bilateral involvement of the middle cerebellar peduncles, pontine atrophy, and the classic "hot cross bun" sign [2].
3. **Infectious / Post-Inflammatory Cerebellitis:** Typically demonstrates persistent glial scarring or irregular structural distortion rather than isolated, clear-cut tract degeneration along a known arterial distribution [2, 10, 11].

The strict unilaterality, absence of inflammatory signal changes or enhancement, and exact anatomical alignment with the PICA territory strongly favor a chronic vascular etiology with secondary WD in this patient [2, 4, 10, 11, 16].

CONCLUSION

Isolated inferior cerebellar peduncle atrophy is an uncommon but clinically pivotal radiological entity [10, 16]. In patients presenting with non-specific, persistent vestibular symptoms or chronic gait imbalance, radiologists should perform targeted quantitative and qualitative comparisons of the cerebellar peduncles [10, 12, and 16]. Identifying localized peduncular volume loss provides a definitive structural correlate for otherwise cryptogenic chronic vestibular complaints [10, 12, 16].

Declarations

Ethics Approval and Consent to Participate

Ethical approval for single case reports is waived by our institutional review board in accordance with local institutional policies. This study was conducted in full accordance with the ethical principles for medical research involving human subjects set forth in the Declaration of Helsinki.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying anonymized radiological images.

Competing Interests

The authors declare that they have no competing interests, financial or non-financial, related to the content of this manuscript.

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Author Contribution

I.A.: Conceptualization, manuscript drafting and design, quantitative neuroimaging analysis and electronic caliper measurements, writing – original draft preparation, submission management. Yahaya Mohammed Y.M.: Clinical evaluation and management of the patient, clinical case history compilation, neurological differential diagnosis, writing – review & editing. A.Y.M.: Literature review, reference curation, initial manuscript structuring, figure preparation and layout. N.B.A.: Diagnostic radiologic supervision, image verification and annotation review, critical revision of manuscript for intellectual content. A.S.: Image processing and high-resolution formatting, data validation, secondary literature review, final proofreading and approval.

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Availability of Data and Materials

All relevant diagnostic data and neuroimaging studies supporting the conclusions of this article are included within the submitted manuscript and figure files.

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Figures

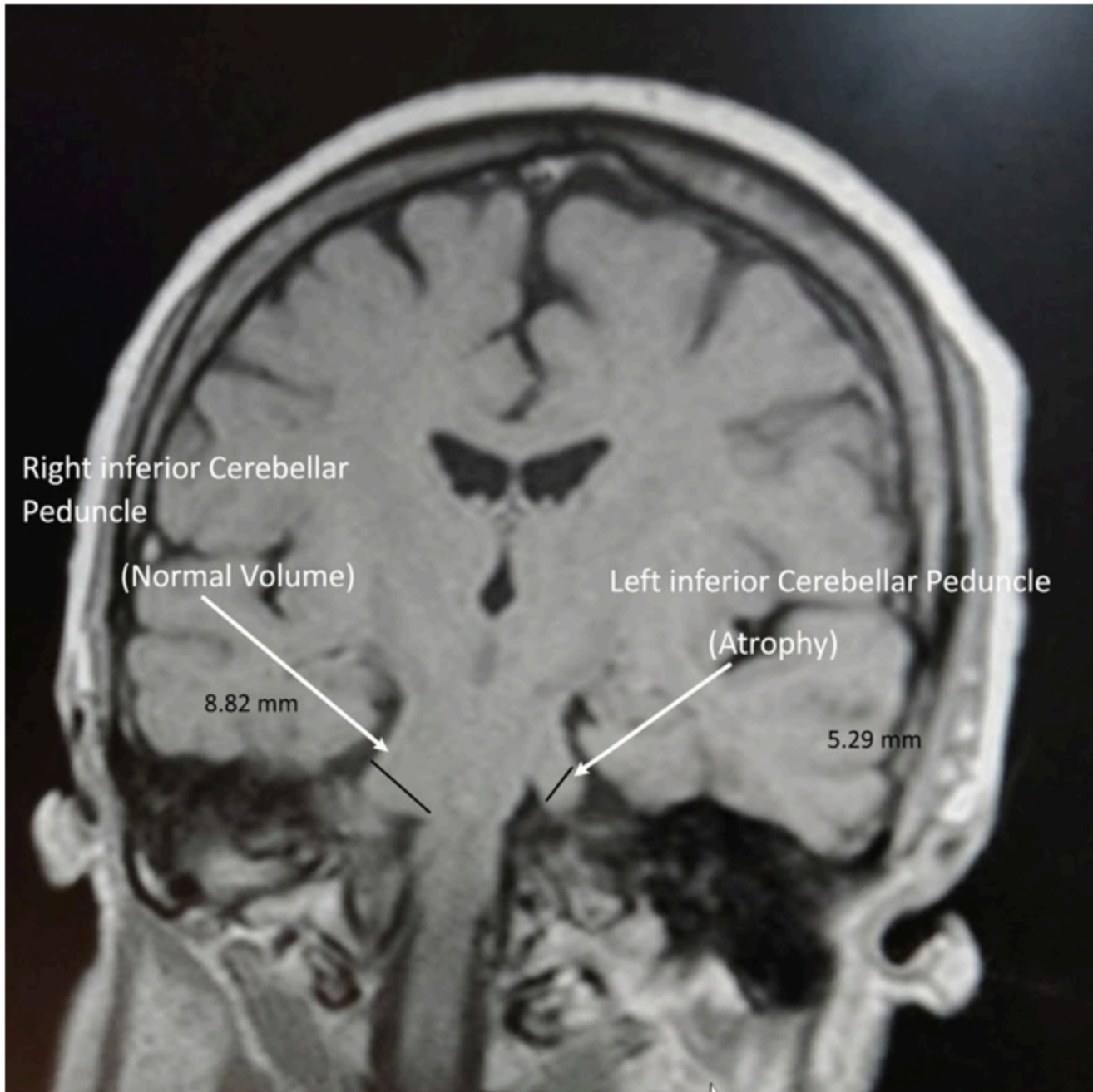


Figure 1

(Coronal T1-Weighted MRI): Quantitative comparison of the inferior cerebellar peduncles (ICP). The right ICP exhibits a normal transverse caliber of **8.82 mm**. The left ICP demonstrates severe focal atrophy measuring **5.29 mm** at its midpoint (~40% reduction), with complete distal loss of ascending peduncular fibers entering the cerebellum. No pathological contrast enhancement or mass effect is noted

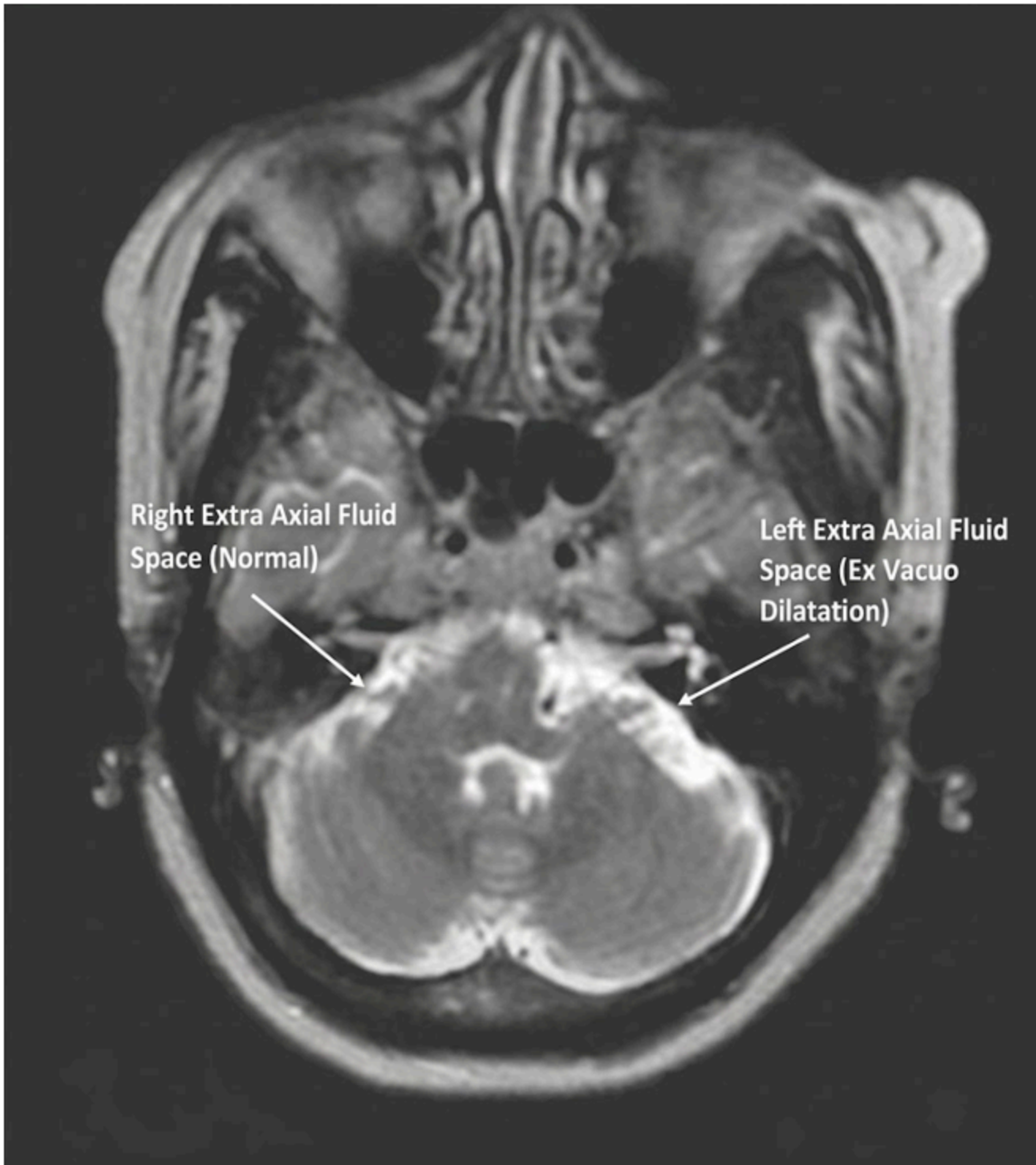


Figure 2

(Axial T2-Weighted MRI): T2-weighted section at the level of the upper medulla / lower pons displaying focal T2 hyperintensity in the left cerebellopontine angle cistern. This represents compensatory ex vacuoexpansion of the subarachnoid space secondary to tissue atrophy of the left ICP and adjacent cerebellar hemisphere