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### Case Report

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# Bilateral wallerian degeneration of middle cerebellar peduncles following unilateral pontine infarction

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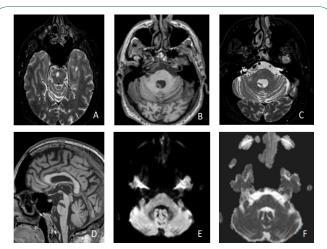
#### Introduction

Bilateral lesions of medial cerebellar peduncles are rare and are most frequently observed in acute cerebral infarction due to large-artery atherosclerosis, wallerian degeneration, neurodegenerative diseases (i.e. multiple system atrophy), inflammatory diseases (i.e. neuromyelitis optica, acute demyelinating encephalomyelitis), toxic encephalopathies (i.e. heroin abuse) and primary central nervous system lymphomas [3]. Wallerian degeneration regards progressive anterograde demyelination of the distal axons following injury to the proximal axon. As a result, fibrosis and atrophy of the affected fiber tracts occurs [2]. We present a case report of a patient with the rare finding of bilateral middle cerebral peduncle lesions, due to wallerian degeneration following pontine infarction. We further discuss differential diagnosis upon imaging findings and their clinical utility.

#### **Case presentation**

A 67- year-old man was hospitalized because of temporary encephalopathy which was attributed to possible mushroom intoxication. The patient had a medical history of ischemic stroke at the right pons eleven months ago with residual dysarthria and left arm weakness, arterial hypertension, diabetes mellitus, and treated brucellosis fifteen years ago. A Magnetic Resonance Imaging (MRI) scan of the brain (Figure 1) revealed: Right large paramedian pontine chronic infarction with hyperintense T2WI/hypointense T1WI signals (Figure 1A,1D) and symmetrical, bilateral lesions of medial cerebellar peduncles with **Citation:** Apostolakopoulou L, Tountopoulou A, Pantou E. Bilateral wallerian degeneration of middle cerebellar peduncles following unilateral pontine infarction. J Clin Images Med Case Rep. 2022; 3(4): 1777.

hyperintense T2WI/hypointense T1WI signals (Figure 1B,1C), slightly hyperintense signals on diffusion-weighted image (Figure 1E) and isointense signals on apparent diffusion coefficient image maps (Figure 1F). The lesions of the medial cerebellar peduncles were attributed to bilateral wallerian degeneration of the crossing pontocerebellar fibres. Also atrophy of midbrain/ cerebral peduncles was noted (Figure 1D). Magnetic resonance



**Figure 1:** (A) Axial T2-weighted image, (B) Axial T1-weighted image, (C) Axial T2- weighted image, (D) Saggital T1- weighted image, (E) Axial Diffusion-weighted image, (F) Axial apparent diffusion coefficient image.

angiography showed short length flow deficit at the right dorsolateral part of the basilar artery compatible with mural thrombus, corresponding to the area of the pontine infarct with no other significant stenosis or anatomical variation. Imaging findings were characteristic of third stage wallerian degeneration of bilateral medial cerebellar peduncles following right pontine infarction due to atherosclerosis [2,3]. Conservative management was decided. Electroencephalographic, cerebrospinal fluid and serum laboratory findings were normal when the encephalopathy subsided.

#### Discussion

Reported cases of wallerian degeneration of the bilateral medial cerebellar peduncles secondary to pontine infarction, result from involvement of the pontocerebellar tract [4]. A unilateral paramedian pontine infarction results in symmetrical wallerian degeneration in bilateral medial cerebellar peduncles. A unilateral pontine basal infarction damages the ipsilateral pontine nucleus and any surrounding axons, along with the crossed axons that originate from the contralateral pontine nucleus. Anatomically, pontocerebellar tract fibers arise from the pontine nuclei, they cross horizontally the midline at the basis of the pons, and pass through the medial cerebellar peduncles to reach the cerebellar cortex. An insult in paramedian pons would damage the ipsilateral pontine nuclei and axons of pontocerebellar tract. The axons originating from contralateral pontine nuclei that have crossed the midline course through the pons to reach medial cerebellar peduncles. Therefore, damage in one side of the pons, affects the homolateral pontine nuclei as well as the contralateral pontocerebellar tracts. The specific neuroimaging finding of symmetrical hyperintensities in the medial cerebellar peduncles can be interpreted as wallerian degeneration of pontocerebellar tracts following pontine infarct [5]. Wallerian de-

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generation should be considered when the primary infarct site is close to the secondary site, but not in the same region of the vascular innervations [6]. The exact mechanism underlying the pathogenesis of bilateral medial cerebellar peduncles' lesions is unknown. Most probably the degeneration of transverse pontocerebellar fibers occurs as a part of diffuse white matter lesions or the continuous spread of closely anatomically related pontic lesions. Certain imaging characteristics can assist clinicians with the differential diagnosis. Wallerian degeneration can be distinguished from inflammation and lymphoma because of the lack of contrast enhancement. Lesions elsewhere are usually found in vascular, demyelinating, inflammatory and toxic etiologies.

Neurodegenerative diseases are the only ones found to have signal hyperintensities strictly limited in the areas aforementioned, accompanied by atrophy [1]. Histologic and metabolic characteristics of different stages of wallerian degeneration are correlated with specific findings on conventional magnetic resonance imaging. First stage (within 4 weeks) may show diffusion abnormalities. Second stage (4–14 weeks) exhibits imaging hypointensities on T2-weighted images. Third stage (14 weeks to several years) is characterized by hyperintensities on T2weighted imaging and hypointensities on T1-weighted imaging. Fourth stage occurs after several years and is characterized by shrinkage of the white matter fiber tracts with volume loss [6]. Interestingly, wallerian degeneration can also present with restricted diffusion on diffusion-weighted imaging especially during the first and third stages and should be differentiated from a new infarction [1]. Diffusion abnormalities which may occur in degenerating fibers are time-related, nonspecific and irrespective of the apparent diffusion coefficient sequence. Wallerian degeneration of the pontocerebellar tracts is considered to be easier to diagnose in the third stage because new neurological deficits, though rare, can appear in this period, but mostly because conventional magnetic resonance imaging can detect abnormal signals [1]. Clinical presentation can be new onset ataxia, dysarthria, vertigo, hearing impairment and dyskinesias [6]. However there is no absolute association between the time of symptom onset and the stage of wallerian degeneration from magnetic resonance imaging findings. The pathophysiological process from initial pontine infarction to secondary wallerian degeneration of the medial cerebellar peduncles is consecutive. Symptoms could be present from the infarction and could be compensated later on by daily activities modification and/or rehabilitation.

Differential diagnosis of bilateral medial cerebellar peduncles' lesions following unilateral pontine infarction can be tricky, with wallerian degeneration and new infarction being the hardest to differentiate. Clinicians should keep in mind the imaging and clinical pitfalls. A helpful imaging tip in the presence of two closely related infarct-like lesions of different chronicity, could be to observe the vascular distribution of the lesions. In cases of different vascular supply, suspicion for wallerian degeneration should be raised. The diagnosis of wallerian degeneration should be considered when MRI findings are obscure. We suggest no change regarding patient management as long as second line prophylaxis for stroke is already given. In complex cases, new stroke work up is encouraged. Clinical reevaluation and new magnetic resonance imaging after a month, could enlighten such cases of diagnostic dilemma.

#### Conclusion

In conclusion, although bilateral medial cerebellar peduncles' lesions are relatively rare with a wide range of differential diagnosis, they should strongly raise the suspicion of wallerian degeneration in patients with past pontine infarction.

#### Declarations

**Conflicts of interest/competing interests:** All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this manuscript.

**Consent to publish:** Patient consent was given for this publication.

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