Case 11796

Wallerian degeneration of the pontocerebellar fibres
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Section: Neuroradiology
Area of Interest: Neuroradiology brain
Procedure: Imaging sequences
Imaging Technique: MR
Special Focus: Ischaemia / Infarction Case Type: Clinical Cases
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Patient: 75 years, male

Clinical History:
A 75-year-old man, with a history of high blood pressure (HBP), NIDDM and a previous left pontine infarction. Four months later, he presents with worsening of right limb weakness and an MRI examination was performed.

Imaging Findings:
The MRI was performed and showed symmetrical and bilateral hyperintense lesions in both middle cerebellar peduncles (MCPs) on T2WI and FLAIR sequences, compatible with acute-subacute Wallerian degeneration of the pontocerebellar fibres, secondary to chronic ischaemic infarction of the left pons. On T1WI after intravenous contrast no anomalous enhancement was present.

Discussion:
Wallerian degeneration (WD) also known as anterograde or orthograde degeneration, is the process after progressive demyelination of the distal neuronal axons due to proximal damage (transection of the axon or damage to the neuron) [1, 2]. The most common cause of WD is cerebral infarction and is mainly described as affecting the corticospinal tract (CST).

WD can also result from a variety of conditions including haemorrhage, trauma, necrosis, and focal demyelination. WD of MCPs is rarely described [1].

A detailed knowledge of the white matter pathways is essential. Neuroanatomically, MCPs are the largest and the main package of fibres that connect the pons with the cerebellum. The pontine nuclei are mainly located at the ventromedial upper pons, they receive input from the cerebral cortex via the corticopontine tract and cross almost exclusively to the contralateral cerebellum via the transverse pontine fibres and MCPs, which constitute the pontocerebellar tract [2, 3]. The pontocerebellar fibres cross at an upper pontine level, hence, when damage occurs in one side of the pons, homolateral nuclei (MCP) can be involved as well as the contralateral MCP after its decussation.

Four stages of WD in the cerebrum have been described and they can be observed after 4 weeks (involves a sufficiently large bundle of fibres) [4]. The increased signal on T2WI is due to increased accumulation of water with atrophy along the neural pathways as a final result after 3 months of the onset of symptoms and should not be
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References:


Description: (A,B) T2WI. Hyperintense lesions in both MCPs (red-arrows) and hyperintense lesion in the left aspect of the pons (yellow-arrows).
(C) 3D T1WI. Non enhancement.
(D) DWI. Signal restriction in both MCPs. Origin: Department of Radiology. "Virgen de la Arrixaca" University Hospital, Murcia, Spain.