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Wallerian Degeneration of the Bilateral Middle Cerebellar Peduncles Secondary to Pontine Infarct

Case Report

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Abstract

Bilateral middle cerebellar peduncle hyperintensities in MRI can be described in several diseases. Previous studies have indicated that neurodegenerative diseases like OPCA, SDSare most likely to affect bilateral MCP. Recent studies have proven that there are many causes of bilateral MCP involvement of which most common being ACI with arterial occlusion. WD, MSA, NMO, heroin-induced leukoencephalopathy, and PCNSL are other causes. Specific neuroimaging findings& clinical features help in differentiating such lesions.

We present a case of 40year old male presented with recurrent pontine stroke having bilateral middle cerebellar peduncle T2 and FLAIR hyperintensities in repeat MRI due to Wallerian degeneration secondary to pontine infarction. It is important to know how to differentiate WD from Acute stroke which is highlighted in this study.

Keywords: OPCA: olivopontocerebellar atrophy; SDS: schwamon diamond syndrome; WD: Wallerian degeneration; MCP: middle cerebellar peduncle; ACI: acute cerebral infarction; MSA: multisystem atrophy; NMO: neuromyelitis optica

Introduction

Wallerian degeneration is the process of demyelination and disintegration of distal axonal segment following the interpretation of axonal integrity or damage to the proximal neuron. Wallerian degeneration of bilateral MCP's can be seen after a pontine stroke. This can be seen as T2,FLAIR hyperintensities in MRI. Although it looks similar to an infarct in MRI, stroke causing bilateral symmetrical lesions in MCP's is very rare. However, it is important to differentiate it from other causes of bilateral MCP's hyperintensities in MRI.

We report a case presented with recurrent CVA pontine infarct showing Bilateral symmetrical T2 flair hyperintensities in middle cerebellar peduncle 2months after the onset of symptoms.

Case Discussion

A 49-year-old male hypertensive and diabetic presented with chief complaints of acute onset weakness of left upper limb and lower limb for 1 week with complaints of swaying to left while walking. History of similar complaints 3 months back from which he was partially recovered with MRS 2/6.On examination: patient was awake, alert, oriented to time, place and person with normal vitals. Higher mental functionand Cranial nerves were normal. Motor examination – bulk & tone of all 4limbs– normal, Power – left upper limb and lower limb -4/5, right side 5/5.DTR 2+ bilateral, plantar extensor on left sidewith

normal sensory examination. FNT, FFNT normal bilateral. No dysdiadochokinesia, broad-based gait and impaired tandem walking present.

Routine investigations like complete blood picture, renal and liver functions tests, lipid profile were normal, with normal ECG,ECHO and bilateral CV doppler. Serum copper, serum calcium, fundus were normal.

Due to financial issues MRI brain was done 20 days after the first stroke which was showing Gliotic foci in central mid-pons in T2W(Figure1B), MRA normal (Figure1C).

MRI brain which was repeated after the onset of symptoms second time showing T2 (Figure 2A) / flair (Figure 2B) hyperintensity noted in anterior aspect of bilateral pons with diffusion restriction (Figure 2C), with hypodensity in ADC (Figure 2D)T2 (Figure 2A) / FLAIR(Figure2B) hyperintensityis noted in Bilateral middle cerebellar peduncle with subtle diffusion restriction (Figure 2C), with no ADC changes(Figure2D) likely to be acute infarct in pons with Wallerian degeneration in Bilateral middle cerebellar peduncle.

Discussion

Wallerian degeneration refers to progressive anterograde disintegration with demyelination of the distal axons following injury to the proximal axon or soma [1]. The histologic and metabolic characteristics of the different stages of WD are correlated with specific findings on conventional MRI [2]. The first stage (within 20 days after injury) is characterized by disintegration of the axons and myelin sheaths without abnormal signals on conventional MRI. However, several studies have revealed that DWI can depict transient signal abnormalities at this stage, especially within the first 14 days of the stroke (pre-WD) [3,4]. The second stage (from 20 days to 60-120 days after injury) is characterized by rapid myelin protein breakdown, during which the tissue becomes more hydrophobic, resulting in hypointensities on proton-density and T2-weighted imaging. Myelin and lipid breakdown, gliosis, and increase in hydrophilic tissue during the third stage (98 days after injury) result in the appearance of hyperintensities on T2-weighted and FLAIR imaging and hypointensities on T1-weighted imaging. The last stage (after several years) is characterized by volume loss due to atrophy. Therefore, WD entails the degeneration of axonal structures and demyelination, and finally fibrosis and atrophy of the affected fibertracts [2].

Diffusion abnormalities may occur in degenerating fibers and are time-related, irrespective of the ADC sequence, especially during the first and third stages. This occurrence might be related to cell swelling, demyelination with axonal degeneration, phagocytotic activity, and water uptake and indicate that diffusion abnormalities in degenerating fibers are not specific and may provide evidence to differentiate WD from ACI[2].

Wallerian degeneration is observed most frequently in the corticospinal tract following injury to the motor cortex or internal capsule and presents as ipsilateral T2 hyperintensity or atrophy of the cerebral peduncle[5].

The middle cerebellar peduncle (MCP) consists of the transversely coursing pontocerebellar fibers that arch across the midline and gather on each side [6]. The pontine nuclei are intermediary gray matter scattered in the basis pons and part of the cortico-ponto-cerebellar pathway (closed loop communication between the cerebellum and pre-central /prefrontal cortex that control not only the action of motor tasks but also planning and initiation of movements)[7].

Bilateral MCP lesions were found in many diseases. ACI (acute cerebral infarction), WD,MSA(multiple system atrophy),NMO(neuromyelitis optica), heroin induced leukoence-phalopathy and PCNSL(primary central nervous system lymphoma). Most of these are associated with other cerebral lesions[2].

Patients with ACI exhibited bilateral MCP-restricted diffusion hyperintensities on diffusion-weighted imaging, hypointensity on ADC and corresponding stenosis or occlusion of the vertebrobasilar system. However bilateral MCP infarctions are rare.In initial MRI of patients with WD depicted pontine infarctions, while symmetrical

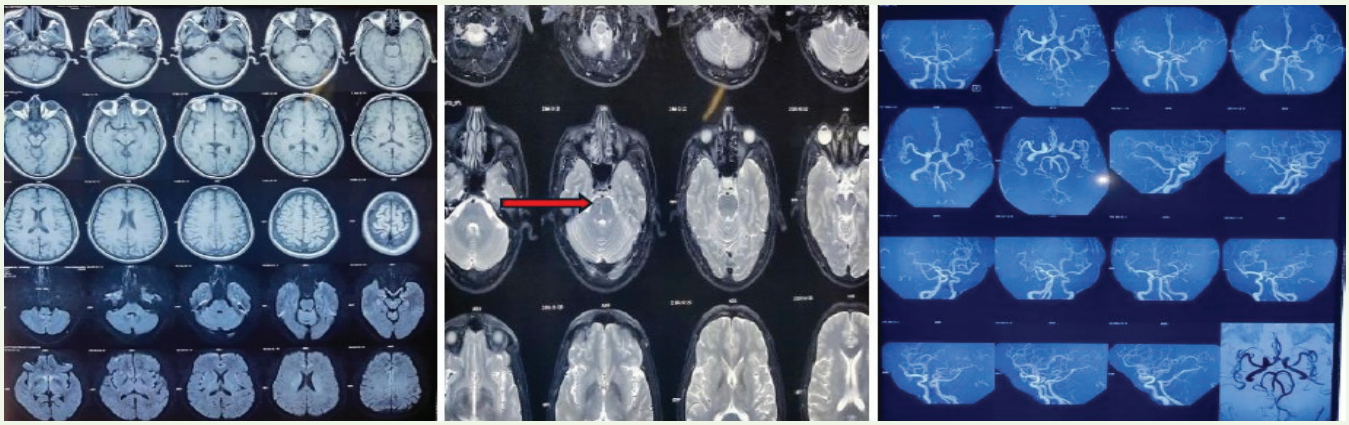

Figure 1: A: DWI and FLAIR images with no hyperintensities or diffusion restriction. B) T2W image showing gliotic focus in midpons. C) MRA and MRV images with no abnormality.

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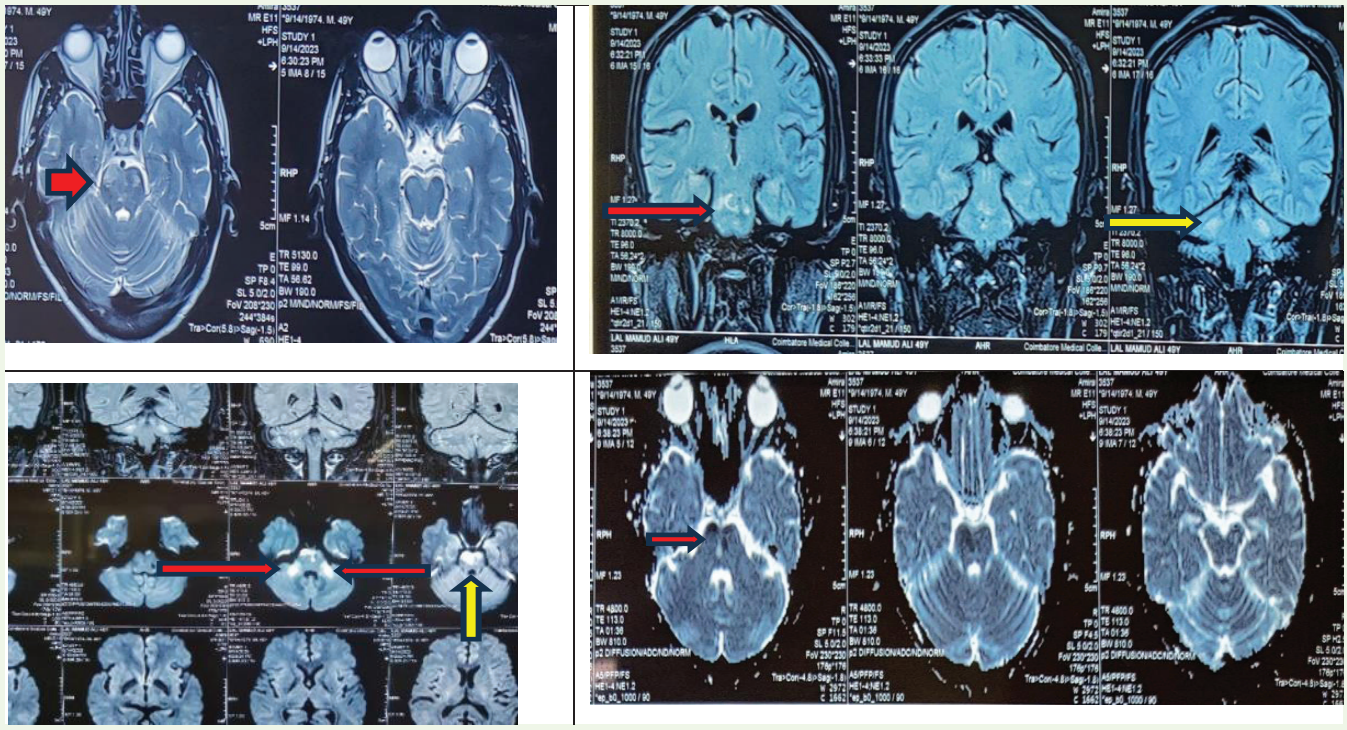

Figure 2: A: T2W image with hyperintensities in bilateral pons and bilateral middle cerebellar peduncles. B) FLAIR image showing bilateral pons (red arrow) and middle cerebellar peduncles(yellow) hyperintensities C) DWI images with diffusion restriction in bilateral pons (yellow arrow) and middle cerebellar peduncles (red arrow). D) ADC images with hypointensities in bilateral pons.

MCP lesionsas above mentioned, with chronic pontine lesions and gliosis were observed on follow-up MRI. Symmetrical MCP lesions, cruciform hyperintensity, and marked atrophy in the posterior fossa were characteristic manifestations of MSA-C[8,9]. Longitudinally extensive myelitis affecting more than three vertebral segments on cervical MRI and positive serum AQP4-IgG may be indicative of NMOSD[10]. Heroin-induced leukoencephalopathy often results in extensive, symmetrical lesions of the cerebral and cerebellar white matter, posterior limb of internal capsule & splenium of corpus callosum[11]. PCNSL was indicated by a significant and characteristic "fist" sign on contrast enhanced MRI[12].

ACI	Hyperintensity on DWI and Hypointensity in ADC occlusion of Vertebrobasilar System
WD	Hyperintensity in DWI [Time Related] Variable in ADC Old Pontine Infarctions With Gliosis and Atrophy

Conclusion

In bilateral symmetrical MCP hyperintensities in MRI following pontine infarction Wallerian degeneration should also be considered before labelling it as a new infarction and subsequent changes should be identified. Neurologists should be familiar with WD of the bilateral MCPs to avoid misdiagnosis as an additional infarction.

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