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Bilateral wallerian degeneration of the middle cerebellar peduncles secondary to pontine infarction: A case series

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ABSTRACT

Objective: Wallerian degeneration (WD) of middle cerebellar peduncles (MCPs) secondary to pontine infarction is rarely reported in the literature. Our aim in this study is to characterize its clinical and neuroradiological features.

Methods: A retrospective review of 7 patients from a single institution was conducted. Only patients with pontine infarction and subsequent degeneration of the MCPs were included in the analysis. The features of clinical presentation and neuroimaging finding were summarized by our experienced neurologists.

Results: Seven patients (5 male, 2 female), ranging in age from 50 to 77 years, satisfied the inclusion criteria. All patients had cardiovascular risk factors and hypertension was the most common one. Almost all of the patients had hemiparesis and dysarthria, and could achieved good clinical outcome. On the initial scan, hyperintense on T2- and diffusion-weighted images suggested the acute pontine infarction. On the follow-up scan, however, hyperintensities of bilateral MCPs on T2-weight and FLAIR images were apparently demonstrated in all patients. The specific lesions in the MCPs were attributed to bilateral WD of the pontocerebellar fibres secondary to pontine infarction.

Conclusion: WD should be taken into account when patients are initially diagnosed with paramedian pontine infarction and follow-up MRI manifest as symmetrical hyperintense in the MCPs.

1. Introduction

Wallerian degeneration (WD) was first described in 1850 by Augustus Waller in the glossopharyngeal and hypoglossal nerves of frogs [1]. It is the process of progressive demyelination and disintegration of the distal axonal segment following the transection of the axon or damage to the neuron [2]. WD is most frequently observed involving the corticospinal tract, but can also be uncovered affecting other projecting systems such as corticopontocerebellar tract, dentate-rubro-olivary pathway, posterior column of the spinal cord, corpus callosum, limbic circuit, and optic pathway [3]. Infarction is the most common cause resulting in WD. Also neoplasms, hemorrhage, surgery, epilepsy, white matter diseases and multiply system atrophy are reported entities that may leading to degeneration. Histopathologically, WD develops through sequential stages. Initially, it is characterized by physical disintegration of the axons and myelin sheaths. Subsequently, the myelin sheaths break down structurally into smaller particles. This is followed by chemical decomposition of the protein components of myelin sheath. Finally, gliosis occupies the area of the degenerated axons and myelin sheaths [4].

To our knowledge, bilateral symmetrical hyperintense in the middle cerebellar peduncles (MCPs) on T2-weighted or fluid attenuation inversion recovery (FLAIR) images, which suggests WD of the cortico-pontocerebellar tract secondary to pontine infarction, has been rarely reported in the literature [17,18]. In this retrospective study, our aim is to characterize the clinical and neuroradiological features of WD involving bilateral MCPs. Both the clinician and radiologist must maintain a high level of suspicion for this entity in patients presenting with characteristic features.

2. Methods

This retrospective study was approved by the Institutional Review Board (IRB) of The Affiliated Hospital of Jiujiang University. Those

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patients with foregoing pontine infarction and subsequent hyperintense in the MCPs on brain magnetic resonance imaging (MRI) satisfied the inclusion criteria. A total of 7 patients were selected from our electronic case database between January 2014 and September 2017. As this was a retrospective study, ethics committee approval was not required. Clinical data, such as demographics (gender and onset age), cardiovascular risk factors, symptoms, radiological findings, National Institute of Health Stroke Scale (NIHSS) score, follow-up time and outcome, were independently collected by two of our authors (YS and WJ). The magnetic resonance (MR) studies, performed on a 1.5-T MR clinical system (Signa GE, Milwaukee, USA), were detailedly scanned by experienced radiologist. T1-weighted, T2-weighted, FLAIR and diffusion-weighted images were available in our cases at baseline and follow-up. Symptoms and the NIHSS scoring were documented at the time of the first admission. Functional outcome was measured by modified Rankin scale (mRS) on follow-up (an mRS score from 0 to 2 was considered as a good clinical outcome).

3. Results

3.1. Clinical presentation

The clinical summary of included cases was demonstrated in Table 1. This retrospective case series included seven patients (5 male, 2 female; age range: 50–77 years; mean age: 68 years). All patients had cardiovascular risk factors, including hypertension, diabetes mellitus, hyperlipidemia, smoking and drinking. Hypertension (n = 7) was the most common cardiovascular risk factor. On the first admission, almost all of the patients had sudden onset of hemiparesis (n = 6) and dysarthria (n = 7). Other symptoms included, vertigo (n = 3), gait ataxia (n = 2), headache (n = 2) and confusion (n = 1). The mean scores of NHISS on admission was 7.14 (range: 4–8 scores). At the time of follow-up (time range: 3–7 months), only one patient complained of gradually worsening dysarthria and ataxia of four limbs, while the other 6 patients achieved good clinical outcome. During the time between baseline and follow-up cranial MR examinations, however, all 7 patients did not report sudden-onset neurological symptoms.

3.2. Radiological findings

All 7 patients underwent cranial MR examinations at baseline and follow-up. On the initial scan, hyperintense in paramedian pontine on T2-weighted and diffusion-weighted images was recognized in all patients. The acute onset of neurological deficits together with the accordance of the lesion on diffusion-weighted image (DWI) indicated the acute pontine infarction. Among these patients, the lesions were located in different parts of the pons, including left sided (n = 3), right sided (n = 3) and bilateral (n = 1) (Table 1). At that time, however, the MCPs were normal and did not display any signal changes. On the follow-up MRI, no alterated diffusion could be seen in the pons. However, bilateral and symmetrical hyperintensities along the MCPs on T2-weighted and FLAIR images were apparently showed in all patients (Fig. 1)). Furthermore, subtle hyperintense in the MCPs on DWI could also be showed in two patients. The lesions of the MCPs were attributed to bilateral WD of the crossing pontocerebellar fibres.

4. Discussion

WD refers to the progressive anterograde disintegration of axons and accompanying demyelination, which occurs after injury to the proximal axon or cell body. It is most commonly seen following cerebral infarction but can also be secondary to a variety of disease processes, such as neoplasms, hemorrhage, surgery, epilepsy, and white matter diseases [3]. Presently, few case reports have depicted the MR findings of WD in the MCPs following pontine infarction [15–18]. Conventional MR sequences have been utilized to describe changes of WD in different phases. Moreover, histologic and metabolic features on different stages of WD are correlated to specific findings on MR imaging. The first stage is characterized by disintegration of the axons and myelin sheaths within 20 days after injury. But no signal intensity abnormalities are usually recognizable. The second stage is characterized by the rapid destruction of the myelin sheath from 20 days to 2–4 months after stroke. As the tissue becomes more hydrophobic accompanying by myelin-protein breakdown, the high lipid-protein ratio results in hypointense on T2-weighted image. In the third stage, with gliosis and changes in water content and structure, the hydrophilic tissue shows hyperintense on T2-weighted and FLAIR images and hypointense on T1-weighted image. The terminal stage is characterized by volume loss of degenerated tissue and signal intensity abnormalities may be persistent existence for several years after infarct [2].

Anatomically, the MCPs are mainly composed of pontocerebellar tracts that connect the basal portion of the pons with the cerebellum. The middle cerebellar peduncle (MCP) is vulnerable to WD because it is the largest and the the main path for pontocerebellar tracts [13]. Pontocerebellar tracts arise from the controlateral pontine nuclei which receive cortico-pontine tracts. They cross the midline at an upper pontine level and pass through the MCP to reach the cerebellar cortex. When damage (such as ischemic insult) occurs in one side of the pons, homolateral pontine nuclei as well as the contralateral pontocerebellar tracts are simultaneously affected (Fig. 2. Therefore, the specific neuroimaging finding of symmetrical hyperintese in the MCPs can be interpreted as WD of pontocerebellar tracts following pontine infarct.

In this study, we present a case series to highlight the clinical presentation and radiological finding of WD in both MCPs secondary to pontine infarction. Patients with pontine infarction may present with a variety of symptoms. Hemiparesis and dysarthria are the most common symptoms among the 7 patients. When WD of the MCPs was revealed on follow-up MR examination, MRS score was not increased in all but

Table 1
Summary of the clinical presentation and radiological finding results of 7 cases.

Case No.	Age (yrs), Sex	Cardiovascular risk factor	Symptom on admission	Infarction location in the pons	NIHSS score	Follow-up time (months)	Outcome mRS
1	73, M	Hypertension, diabetes mellitus	Hemiparesis, confusion, vertigo, headache, dysarthria	Bilateral	12	5	4
2	77, M	Hypertension, hyperlipidemia	Hemiparesis, vertigo, dysarthria	Left	7	4	2
3	77, M	Hypertension, smoking, drinking	Hemiparesis, gait ataxia, dysarthria	Left	8	3	2
4	68, F	Hypertension	Gait ataxia, dysarthria	Right	4	7	1
5	60, M	Hypertension, diabetes mellitus	Hemiparesis, dysarthria	Right	6	5	2
6	72, F	Hypertension, hyperlipidemia	Hemiparesis, vertigo, dysarthria	Right	7	4	2
7	50, M	Hypertension, Hyperlipidemia, smoking	Hemiparesis, headache, dysarthria	Left	6	6	2

Abbreviations: F female, M male, yrs years, NIHSS National Institute of Health Stroke Scale, mRS modified Rankin Scale.