Clinical and Radiological Features of Wallerian Degeneration of the Middle Cerebellar Peduncles Secondary to Pontine Infarction

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Abstract

Background: Wallerian degeneration (WD) of bilateral middle cerebellar peduncles (MCPs) can occur following pontine infarction, but its characteristics have not yet been clarified because of the low incidence. Thus, the present study discussed the clinical and radiological features to improve the awareness of this disease.

Methods: Clinical and radiological information from consecutive individuals diagnosed with WD of bilateral MCPs following pontine infarction in three hospitals over the past 4 years between October 2012 and October 2016 were retrospectively investigated and compared with a control group (patients with pontine infarction had no secondary WD).

Results: This study involved 30 patients with WD of MCPs, with a detection rate of only 4.9%. The primary infarctions ($\chi^2 = 24.791$, $P = 0.001$, vs. control group) were located in the paramedian pons in 21 cases (70.0%), and ventrolateral pons in nine cases (30.0%). WD of the MCPs was detected 8–24 weeks after pons infarction using conventional magnetic resonance imaging (MRI); all secondary WDs were asymptomatic and detected incidentally. All WD lesions exhibited bilateral, symmetrical, and boundary blurring on MRI. The signal features were hypointense on T1-weighted imaging, hyperintense on T2-weighted imaging and fluid-attenuated inversion recovery, and slightly hyperintense or isointense on diffusion-weighted imaging and apparent diffusion coefficient maps. Secondary brainstem atrophy was found in six (20.0%) cases. A Modified Rankin Scale score 0–2 was found in 10 (33.3%) cases and score >2 in 20 (66.7%) cases at 90 days after discharge, and the short-term prognosis was worse than that in control group ($\chi^2 = 12.814$, $P = 0.001$).

Conclusions: Despite the rarity of bilateral and symmetrical lesions of MCPs, secondary WD should be highly suspected if these lesions occur within 6 months after pontine infarction, particularly paramedian pons. Conventional MRI appears to be a relatively sensitive method for detecting WD of MCPs, which might affect the short-term prognosis.

Key words: Magnetic Resonance Imaging; Middle Cerebellar Peduncles; Neurological Prognosis; Pontine Infarction; Wallerian Degeneration

INTRODUCTION

Wallerian degeneration (WD) refers to an anterograde degeneration of nerve fiber (s) distal to the injured neuronal cell body or proximal axon. WD can be secondary to some diseases, especially stroke.[1] It has been reported that WD may occur in the 1st week after stroke (at the earliest), and that corticospinal tracts are commonly involved.[1,2] The nerve fibers in the pons are concentrated and complex, particularly in the basis pontis. Infarction at this special location may lead to bilateral and symmetrical WD of the middle cerebellar peduncles (MCPs).[3-5] However, because

bilateral lesions of the MCPs may also occur in ischemic, demyelinating, metabolic, other neurodegenerative diseases and intoxication,[6,7] misdiagnosis in clinical practice may

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occur; consequently, clinicians need to become more familiar with secondary lesions. In addition, the existing literatures pertaining to WD of MCPs following pontine infarction mainly contain case reports or small-scale retrospective studies because of its low incidence. Moreover, the relationship between the prognosis of pontine stroke and WD of MCPs remains unclear. Therefore, in this retrospective study, we analyzed and summarized the clinical and radiological features of WD following pontine infarction in patients from three medical centers, and compared short-term prognosis with a control group to heighten awareness of WD of MCPs after pontine stroke.

METHODS

Ethical approval

The study was conducted in accordance with the *Declaration of Helsinki* and was approved by the local ethics committee of the institute. Given the retrospective nature of the study and the use of anonymized patient data, requirements for informed consent were waived.

Patients

Data from patients with isolated, unilateral, and incipient pontine infarction admitted to China-Japan Friendship Hospital, Xi'an Central Hospital, and Beijing Chao-Yang Hospital between October 2012 and October 2016 were retrieved from the medical image database in the three hospitals. Cases involving WD of bilateral MCPs revealed on follow-up magnetic resonance imaging (MRI) were included. Patients with WD of bilateral MCPs caused by other known reasons, or patients with no available cranial computed tomography angiography (CTA) or magnetic resonance angiography (MRA) data during hospitalization for the pons infarction, or low compliance during the follow-up, were excluded. Ultimately, 30 consecutive patients were included as the case group. To comparatively analyze the radiological feature and prognosis, a control group of patients with the above-mentioned pons infarction, in whom WD bilateral MCPs were not detected on MRI during the follow-up period (>1 year), was formed (because the previous literature indicated that WD always occurred within 1 year after infarction and WD would be considered absent if it was not detected after 1 year). Finally, a total of 580 patients were enrolled as the control group. Selection of patients for inclusion and exclusion in this study is shown in Figure 1. Given the retrospective nature of the study and the use of anonymized patient data, requirements for informed consent were waived.

Clinical data collection and analysis

Clinical data during hospitalization were collected from the hospital information system and included general information (age and sex); risk factors for cerebrovascular diseases (hypertension, diabetes, hyperlipidemia, hyperhomocysteinemia, previous stroke, coronary heart diseases, atrial fibrillation, smoking, alcoholism, and a family history of atherosclerotic cardiovascular diseases); and initial clinical manifestations of pontine infarction. After discharge,
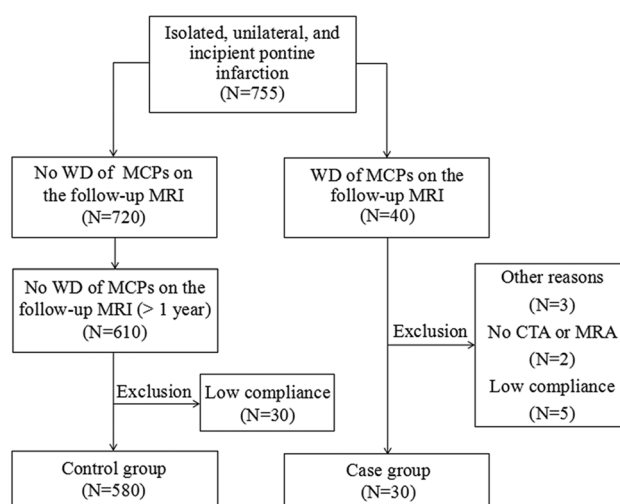

Figure 1: Flowchart of selection of patients for inclusion and exclusion in this study. WD: Wallerian degeneration; MCPs: Middle cerebellar peduncles; MRI: Magnetic resonance imaging; CTA: Computed tomography angiography; MRA: Magnetic resonance angiography.

all enrolled patients were followed up mainly through clinical visits with telephone counseling. The Modified Rankin Scale (mRS) score at 90 days after discharge was evaluated and recorded. Based on the prognostic outcomes, all patients were divided into favorable (mRS 0–2) and unfavorable (mRS >2) outcome to compare the short-term prognosis between the two groups.

Imaging information collection and analysis

Conventional MRI and MRA scans were performed using a 1.5 Tesla (GE signal 1.5 T Excite HD, USA) or 3.0 Tesla MR scanner (Philips Ingenia 3.0 T, The Netherlands). MRI sequences included T1-weighted imaging (T1WI), T2WI, fluid-attenuated inversion recovery (FLAIR), diffusion-WI (DWI), and apparent diffusion coefficient (ADC) maps. CTA scans were performed using a 256-slice (Philips Brilliance iCT or Philips MX-8000, Netherlands) or 320-slice CT scanner (Toshiba Aquilion ONE, Japan). These images were reviewed and reanalyzed by two experienced neuroradiologists who were blinded to the clinical data. Recorded information included the location of the primary pons infarction divided by different arterial supply regions (paramedian, lateral [ventrolateral and dorsolateral], tegmental, or mixed region); plaque formation or stenosis at the basilar artery (BA); the timing of WD of the MCPs detected by MRI; and the signal features of WD on different MRI sequences and other secondary changes in the brainstem. Finally, the location of infarction and the degree of atherosclerosis of BA between two groups were analyzed comparatively.

Statistical analysis

SPSS Version 17.0 (SPSS Inc., Chicago, IL, USA) was used for all analyses. Measurement data are presented as mean ± standard deviation; count data are presented as ratio. Intergroup comparison of count data was performed using the Chi-squared test; A value of $P < 0.05$ was considered statistically significant.

RESULTS

Clinical characteristics

In this study, 20 men (66.7%) and 10 women (33.3%) were included in the case group. The mean age was 63.0 ± 9.2 years (range, 44–76 years). Risk factors for cerebrovascular disease included hypertension ($n = 24$ [80.0%]), hyperlipidemia ($n = 20$ [66.7%]), diabetes ($n = 15$ [50.0%]), smoking ($n = 15$ [50.0%]), alcoholism ($n = 15$ [50.0%]), coronary artery disease ($n = 12$ [40.0%]), family history of atherosclerotic cardiovascular diseases ($n = 10$ [33.3%]), and hyperhomocysteinemia ($n = 8$ [26.7%]). Every patient experienced hemiplegia and other initial clinical symptoms of pontine infarction, including central facial and/or glossal palsy ($n = 20$ [66.7%]), dizziness ($n = 18$ [60.0%]), diplopia ($n = 16$ [53.3%]), hemianesthesia ($n = 10$ [33.3%]), vertigo ($n = 7$ [23.3%]), bulbar paralysis ($n = 5$ [16.7%]), and typical Millard-Gubler syndrome ($n = 5$ [16.7%]). All secondary WDs were asymptomatic and detected incidentally by conventional MRI during the follow-up period.

Radiological features

The detection rate of WD of bilateral MCPs following pons infarction on conventional MRI was only 4.9% (37/755). In the case group, MRI revealed that all of the primary infarctions were located above MCPs and extended to the basis pontis, with left in 12 cases (40.0%), right in 18 (60.0%), paramedian pons in 21 (70.0%), and ventrolateral pons in nine (30.0%). There was a statistical difference of the site of pons infarction between two groups ($\chi^2 = 24.791$, $P = 0.001$, Table 1). CTA or MRA presented normal BA in seven cases (23.3%), plaque formation of BA in 13 (43.3%), and BA stenosis (>50.0%) in 10 (33.3%). There was no statistical difference of the structure of BA between two groups ($P = 0.579$, Table 1). The follow-up control MRI revealed the WD of MCPs occurred at 8–24 weeks (mean, 16.1 ± 4.3 weeks) after pontine infarction, which revealed bilateral, symmetrical, and boundary blurring on MRI. The signal features were hypointense T1WI signals, hyperintense T2WI signals, hyperintense FLAIR signals, and slightly hyperintense or isointense signals on DWI and ADC maps. Secondary shrinkage of the brainstem was found in six cases (20.0%) on MRI after 1 year. Representative images are shown in Figures 2–4.

Short-term prognosis

According to the follow-up evaluation, the mRS score 0–2 was in 10 (33.3%) cases and >2 in 20 (66.7%) at 90 days after discharge, owing to pons infarction in the case group. Further analysis revealed that the short-term prognosis of patients in the case group was worse than that in the control group ($\chi^2 = 12.814$, $P = 0.001$, Table 1).

DISCUSSION

WD is the process of progressive demyelination and disintegration of the distal axonal segment following transection of the axon or damage to the neuron.[5] It was first discovered and named by Waller in experiments involving frogs in 1850.[2] Compared with other fiber systems, it is typically observed to affect the corticospinal tracts after an injury to the motor cortex or the internal capsule.[8] Although poststroke WD is also a secondary neurodegenerative disorder, WD of the bilateral MCPs following pons infarction is a relatively rare phenomenon. To our knowledge, there have been fewer than 50 reported cases of WD successfully detected using conventional MRI. This study reviewed data from 3 large medical centers over a period of 4 years, and its detection rate was only 4.9%, which also supports its low incidence. Nevertheless, the sample size analyzed in the present study might currently the largest to be reported in the English literature.

Neuroanatomically, MCPs, also known as the pontibrachium, are the largest peduncles connecting the brainstem to the cerebellum. Cerebral cortical neurons send impulses via the corticopontine tracts to the pontine basal nuclei in the ventral pons. The pontine basal nuclei are oriented toward the contralateral cerebellum via the transverse pontine fibers and MCPs, which constitute the pontocerebellar tract. Finally, the pontocerebellar fibers terminate in the cerebellar hemispheres, vermis, and paraflocculus.[3,9] Based on the special anatomical structures of decussation of fibers in the pons, an infarct in the ventral pons might affect the ipsilateral nuclei, and the tracts that pass to the contralateral MCPs and those passing across from the contralateral nuclei [Figure 5]. Consequently, WD

Table 1: Comparison of the radiological and prognostic data between two groups, *n* (%)

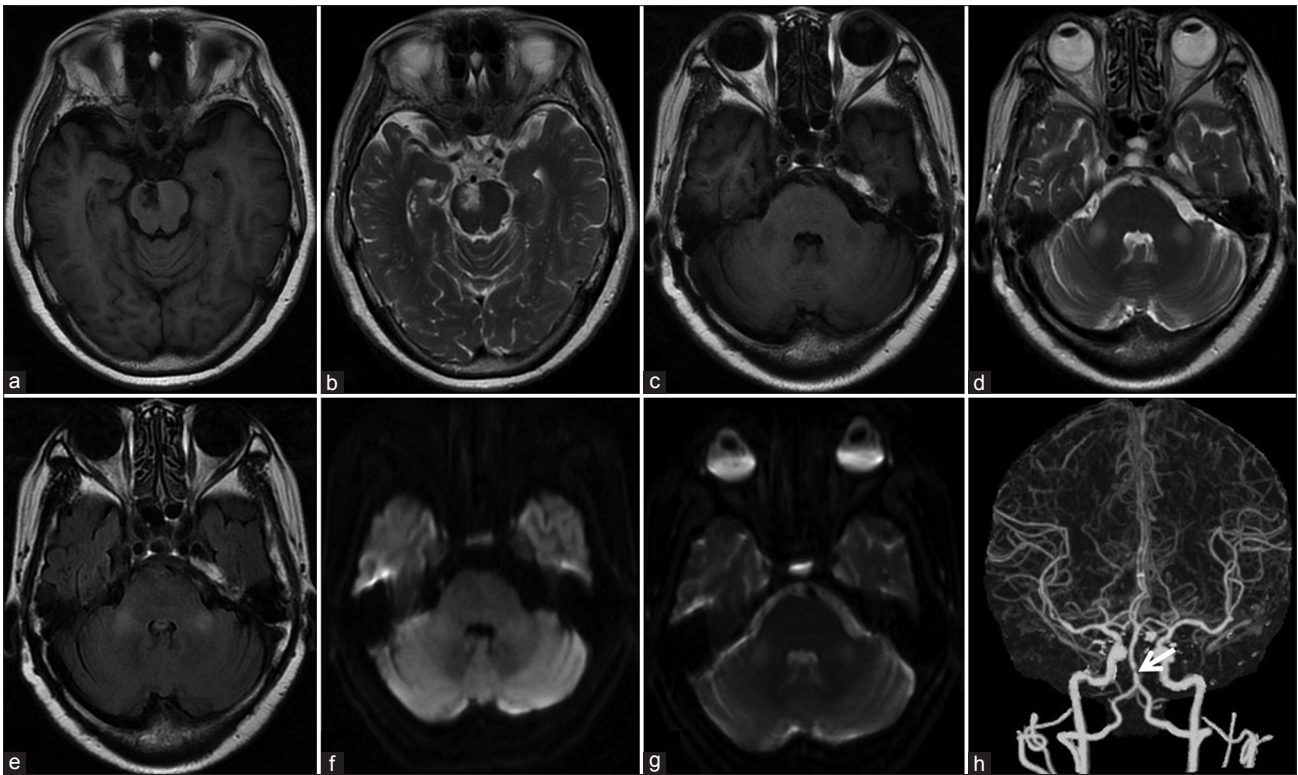| Items	Case group ($n = 30$)	Control group ($n = 580$)	χ^2	P
Pons infarction				
Paramedian	21 (70.0)	185 (31.9)	24.791	0.001
Lateral	9 (30.0)	155 (26.7)		
Tegmental	0	30 (5.2)		
Mixed	0	210 (36.2)		
Basilar artery				
Normal	7 (23.3)	127 (21.9)	1.092	0.579
Plaque	13 (43.3)	206 (35.5)		
Stenosis (>50.0%)	10 (33.4)	247 (42.6)		
Short-term prognosis				
Unfavorable	20 (66.7)	200 (34.5)	12.814	0.001
Favorable	10 (33.3)	380 (65.5)		

Figure 2: MRI findings from a 74-year-old woman of 12 weeks after initial pons infarction. MRI findings revealed right large paramedian pontine chronic infarction with hypointense T1WI signals (a), hyperintense T2WI signals (b), and symmetrical, bilateral, and quasi-circular lesions of MCPs with hypointense T1WI signals (c), hyperintense T2WI (d) and FLAIR signals (e), and slightly hyperintense signals on DWI (f) and ADC maps (g). MRA revealed severe stenosis of the basilar artery (h) (white arrow). MRI: Magnetic resonance imaging; FLAIR: Fluid-attenuated inversion recovery; T1WI: T1-weighted imaging; MCPs: Middle cerebellar peduncles; DWI: Diffusion-weighted imaging; ADC: Apparent diffusion coefficient; MRA: Magnetic resonance angiography.

of the MCPs following pons infarction is usually bilateral in nature.[10] In this study, bilateral MCPs lesions appeared after the unilateral and isolated infarction involving the ventral pons without additional abnormalities, which is consistent with the diagnosis of WD of MCPs, owing to damage of the above-mentioned anatomical structures. In addition, it has been reported that the primary infarction often involves the basis pontis, and that paramedian pontine infarction is the most common location,[3,7,11-14] which is consistent with observations in this study. Moreover, the BA in previously reported cases was normal,[11] or exhibited atherosclerosis plaque formation,[12] severe stenosis, or occlusion.[13] In this study, BA lesions were also nonspecific; thus, we suspect that the etiology of pons infarction might have been small vascular disease, atherosclerotic stenotic disease of the BA, or branch artery disease.

As early as 1989, Kuhn *et al*.[8] divided WD in the corticospinal tracts into 4 stages according to dynamic signal intensity changes on MRI: Stage 1 (within 4 weeks) exhibited no signal intensity abnormality; Stage 2 (4–14 weeks) exhibited hypointense signals on T2WI; Stage 3 (14 weeks to several years) presented hyperintense signals on T2WI; and Stage 4 (several years later) exhibited shrinkage of the white matter fiber tracts with volume loss. This staging scheme is reflected only in T2WI signal intensity changes of WD in the corticospinal tracts in different periods, which

is not comprehensive. With advances in MRI, however, some studies have reported signal intensity changes in DWI or ADC maps, and not only limited to the corticospinal tracts.[13,15] De Simone *et al*.[5] described two cases with WD of the pontocerebellar tracts, with abnormal hyperintense signals on T2WI and DWI demonstrated for 5 and 7 months after infarction. Fitzek *et al*.[3] reported three cases with large paramedian pons infarctions, no abnormal signal of MCPs was detected on T2WI and DWI within 2 weeks after infarction, while bright hyperintensities on T2WI and moderately increased signal intensities on DWI were found after 4 months. The aforementioned case reports suggest that conventional MRI can only reveal WD of MCPs in the subacute or chronic phases;[3,5] however, others do not support this viewpoint.[12,13] Musson *et al*.[12] described an individual who experienced progressive stroke in the pons and underwent repeated MRI at days 4, 9, and 23 after onset due to neurological deterioration. The acute WD of bilateral MCPs was detected on the final MRI, with high-intensity signals on T2WI and DWI, and low-intensity signals on ADC maps. Gala *et al*.[13] described a patient with WD of MCPs due to pontine infarction caused by BA thrombosis. The acute WD was detected on MRI 21 days after onset with signal features similar to those reported by Musson *et al*.[12] However, control MRI performed 12 weeks later revealed persistent hyperintense signals on T2WI, mild hyperintense signals on
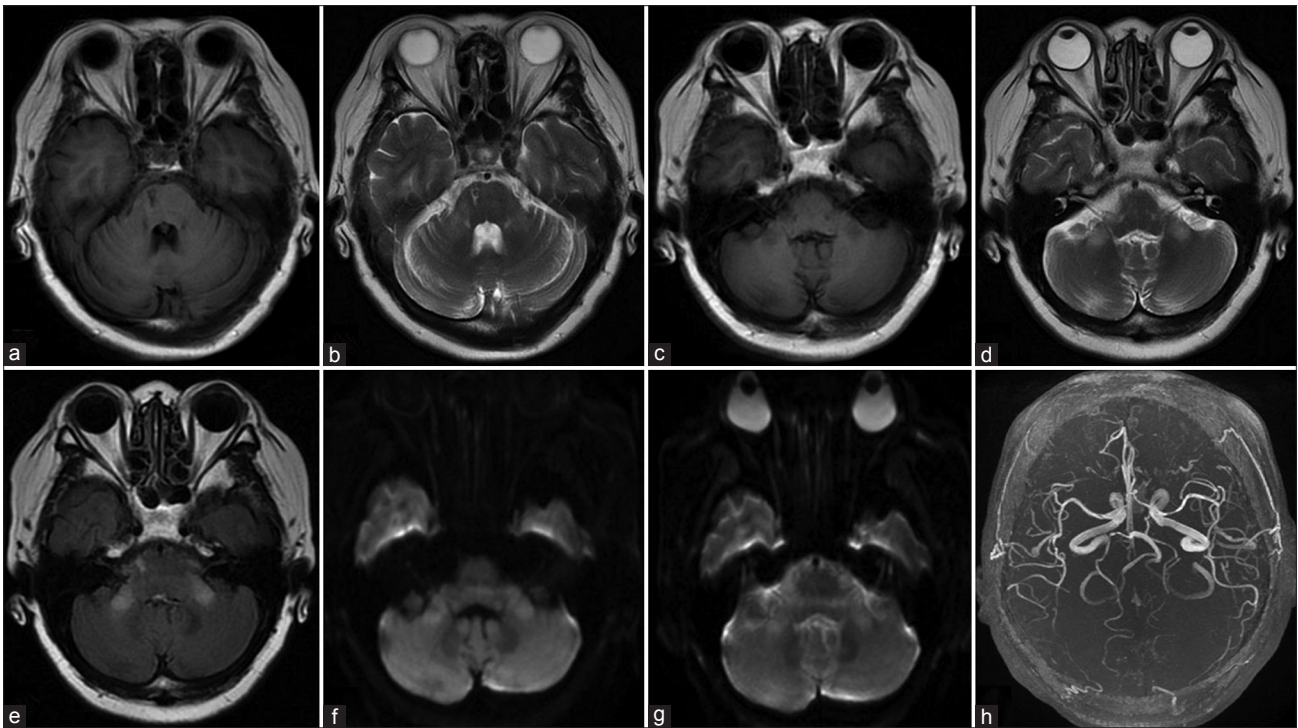
Figure 3: MRI findings from a 61-year-old man of 16 weeks after initial pons infarction. MRI findings revealed right ventrolateral pontine chronic infarction (a and b), and symmetrical, bilateral and boundary blurring lesions of middle cerebellar peduncles with hypointense T1WI (c), hyperintense T2WI (d), and FLAIR signals (e), and slightly hyperintense signals on DWI (f) and ADC maps (g). MRA revealed no local stenosis, only a tortuous vertebrobasilar artery (h). MRI: Magnetic resonance imaging; FLAIR: Fluid-attenuated inversion recovery; T1WI: T1-weighted imaging; DWI: Diffusion-weighted imaging; ADC: Apparent diffusion coefficient; MRA: Magnetic resonance angiography.

DWI and ADC maps, and gliosis in the midbrain and ventral pons. Thus, it is clear that the detection of WD on MRI is different and, moreover, that it is difficult to uniformly stage MRI. In addition, the previous case reports mainly focused on signal intensity changes in WD on T2WI, DWI or ADC maps, rather than all sequences. WD in our 30 cases was detected 8–24 weeks after pons infarction on conventional MRI, with hypointense signals on T1WI, hyperintense signals on T2WI and FLAIR, and slightly hyperintense or isointense signals on DWI and ADC maps; furthermore, six cases also involved brainstem atrophy. Although these cases were partly in accord with previously reported cases, we acknowledge that MRI was not a real-time modality reflecting sequential evolution of WD in this study, and that the exact appearance of WD may be earlier than the time suggested by MRI results. In addition, previous study has commented that diffusion tensor imaging (DTI) could detect early changes in the integrity and direction of fiber bundles after ischemia and, therefore, could detect WD earlier than conventional MRI.[16] However, it is not a routine technique, and the application value is limited to a certain extent.

WD secondary to cerebral infarction is always asymptomatic and detected incidentally,[5] and these characteristics were reflected in our patients. WD of MCPs can also occur after pontine hemorrhage and central pontine myelinolysis, not only pons infarction.[6] Moreover, in clinical practice, WD may mimic ischemic cerebrovascular disease due to similar signals on MRI,[6] especially in patients with multiple vascular risk

factors. Because WD follows pons infarction, the identified key points on MRI are that WD of MCPs generally exhibit regular, bilateral, and symmetrical lesions, which are located below the infarcted pons and along the course of the nerve fibers.[13,14] Our diagnoses in this study were primarily based on these radiological characteristics. Furthermore, symmetric signal abnormalities of bilateral MCPs might also be apparent in other clinical conditions including Wilson disease, hepatic encephalopathy, extrapontine myelinolysis, acute disseminated encephalomyelitis, leukodystrophy, olivopontocerebellar atrophy, spinocerebellar degeneration, toluene abuse, adrenoleukodystrophy, alcoholic liver disease, hypoglycemic coma, and progressive multifocal leukoencephalopathy.[6,7,9,17] Although these lesions are rare, clinicians still need to carefully identify them using clinical and laboratory investigations combined with imaging findings to prevent misdiagnosis.

WD following stroke may be related to neurological prognosis, especially with regard to motor function.[18] The application of DTI can detect WD secondary to infarction early and quantitatively to evaluate the impact of infarction on neurological function, which is of great value in preventing further damage to neurological function and improving functional recovery.[18] Using DTI, Puig et al.[19] reported that WD in the corticospinal tract was associated with motor dysfunction at 30 days following middle cerebral artery ischemic stroke. The investigators reported that it is useful to tailor individualized rehabilitation plans for patients with motor dysfunction to investigate the evolution of DTI
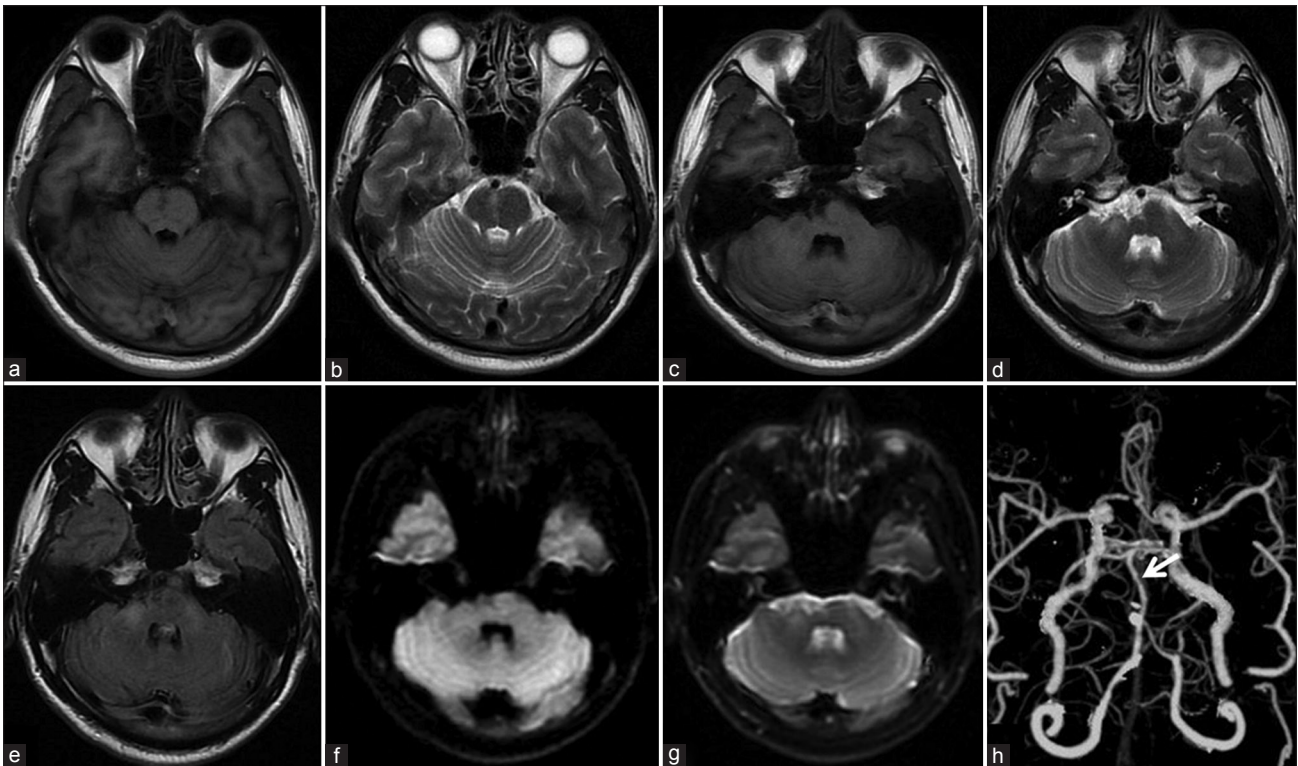
Figure 4: MRI findings from a 45-year-old man of 1 year after primary pons infarction. MRI findings revealed right paramedian pontine chronic infarction, extending from the surface to tegmentum (a and b), bilateral lesions of middle cerebellar peduncles with hypointense T1WI (c), hyperintense T2WI (d) and FLAIR signals (e), and isointense signals on DWI (f), slightly hyperintense signals on ADC maps (g), and the secondary shrinkage of the ipsilateral pontine. MRA detected mild stenosis of the basilar artery (h) (white arrow). MRI: Magnetic resonance imaging; FLAIR: Fluid-attenuated inversion recovery; T1WI: T1-weighted imaging; DWI: Diffusion-weighted imaging; ADC: Apparent diffusion coefficient; MRA: Magnetic resonance angiography.
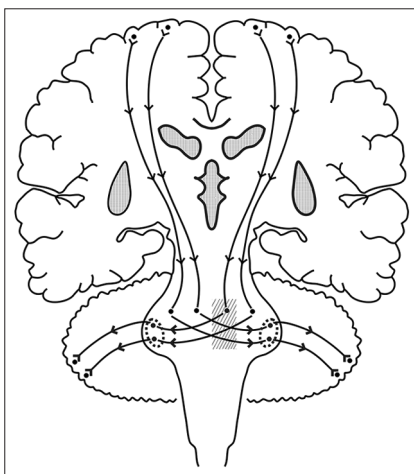

Figure 5: Schematic drawing demonstrating the orientation of the corticopontine tracts, the transverse pontine fibers, and the pontocerebellar tracts with their synapses. The left paramedian pontine lesion (rectangular dotted area) damages the ipsilateral pontine neurons and the tracts passing to the contralateral MCPs (oval dotted areas), and also the contralateral fibers originating from the right pontine nuclei to the ipsilateral MCPs (oval dotted areas). MCPs: Middle cerebellar peduncles.

indices. Previous prognostic studies investigating WD were primarily focused on the pyramidal tracts and, due to the limitation of rare occurrence, there have been only a few studies investigating the prognosis of WD of MCPs. Liang

et al.[20] found that WD of the bilateral MCPs following pontine infarction might hinder the process of recovery of neurological function. Although we did not use DTI technology in the study, we found that the majority (66.7%) of patients with pontine infarction in the case group had a mRS score >2 at 90 days after discharge, and their prognosis was worse compared with the control group, which hints at the short-term neurological dysfunction associated with WD following pons infarction.

The limitations of this study include its retrospective design, the absence of sequential MRI to monitor the evolution of WD, and the effect of medication and/or physical therapy to MRI signals, which may have introduced bias, and the latter will be our future research.

In conclusion, the results demonstrate that WD of the bilateral MCPs following pons stroke is a secondary neurodegenerative disorder, which is always asymptomatic and detected incidentally. It can be detected using conventional MRI 8–24 weeks after pontine basal infarction - particularly paramedian infarction - and exhibits relatively regular, bilateral, and symmetric lesions of the MCPs with signal features of hypointensity on T1WI, hyperintensity on T2WI and FLAIR, and slightly hyperintense or isointense signals on DWI and ADC maps. Furthermore, WD of MCPs might affect the short-term neurological prognosis of patients

with pontine infarction. Because its signal characteristics are similar to those of some other diseases, combination of clinical and radiological features is required to make the correct clinical or differential diagnosis.

Conflicts of interest

There are no conflicts of interest.

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继发于脑桥梗塞的双侧小脑中脚华勒氏变性的临床及影像学特征

摘要

目的： 探讨脑桥梗塞后继发性双侧小脑中脚（middle cerebellar peduncles，MCPs）华勒氏变性（Wallerian degeneration，WD）的临床及影像学特征。

方法： 回顾性联合分析国内三家医院神经科过去4年间脑桥梗塞后双侧MCPs继发性WD患者的临床及影像学资料，并与对照组（同期脑桥梗塞无继发性WD者）对比分析。

结果： 本研究共纳入30例WD患者，仅占脑桥梗塞的4.9%，其中原发脑桥梗塞位于旁正中21例（70.0%）、腹外侧9例（30.0%），病变部位构成比与对照组有显著差异（$\chi^2=24.791, P=0.001$）。常规MRI发现WD距原发脑桥梗塞的时间不等（8-24周），但均无临床症状。其在MRI上表现为双侧对称性类圆形病变，边界模糊，信号特点为T1低信号、T2及Flair高信号、DWI及ADC轻度高信号或等信号，另外6例（20.0%）患者伴同侧下位脑干萎缩。随访发现病例组出院90天时mRS评分（0-2分）者占10例（33.3%），2分以上者占20例（66.7%），其短期不良预后显著高于对照组（$\chi^2=12.814, P=0.001$）。

结论： 尽管双侧MCPs对称性病变罕见，但如出现在脑桥梗塞后6个月之内，临床应高度怀疑继发性WD。常规MRI检查是诊断该病较为敏感的方法。另外，该变性可能与脑桥卒中后短期预后不良相关。