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Case Report

Wallerian degeneration of the ipsilateral middle cerebellar peduncle after lower pontine paramedian infarct diagnosed by magnetic resonance imaging^{*}

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ABSTRACT

We reported a case of Wallerian degeneration of the unilateral middle cerebellar peduncle (MCP) that developed after ipsilateral paramedian lower pontine infarction. The patient was a 70-year-old woman with right hemiparesis and dysarthria. Using a 3-Tesla scanner, cranial magnetic resonance imaging was performed, and an infarct was found at the left paramedian lower pons. Seven months later, an abnormal signal was found at the central portion of the left MCP, indicative of Wallerian degeneration of the pontocerebellar tract (PCT). There was no abnormality at the contralateral MCP. Usually, Wallerian degeneration of the bilateral MCPs may develop after unilateral paramedian pontine infarction, because bilateral PCTs cross each other at the midline of the basis pontis. In the present case, however, Wallerian degeneration was found at only the ipsilateral MCP. The contralateral PCT was not affected because the PCT runs in the craniocaudal direction, and our patient had a lower pontine infarct. The location of the pontine infarct (affected PCT) and the Wallerian degeneration of the side of the MCP were well correlated.

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Introduction

There are 2 types of secondary degeneration of the brain after focal brain damage, Wallerian degeneration and transneuronal degeneration [1]. The most frequently observed Wallerian degeneration occurs in the corticospinal (pyramidal) tract. After pontine infarction or hemorrhage, Wallerian degeneration of the pontocerebellar tract (PCT) may develop at the central portion of the middle cerebellar peduncle (MCP). In the case of unilateral paramedian pontine infarction, Wallerian degeneration of the bilateral PCTs is usually seen, because bilateral PCTs cross each other at the midline of the basis pontis [2–6].

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Fig. 1 – Diffusion-weighted images obtained 9 days after the onset show a subacute infarct at the left paramedian lower pons (arrows). No lesion is seen at the upper pons.

We herein present a case of Wallerian degeneration of the ipsilateral MCP after unilateral paramedian lower pontine infarction.

Case report

A 70-year-old woman with atherosclerotic risk factors, such as diabetes mellitus and hyperlipidemia, experienced suddenonset right hemiparesis and dysarthria, underwent cranial magnetic resonance (MR) imaging and MR angiography at 9 days after the onset. Used machine was a 3-Tesla scanner (Signa Architect; GE Healthcare, Milwaukee, WI). On diffusionweighted images (DWIs), subacute infarction of the left paramedian lower pons was found (Fig. 1). MR angiography showed no significant abnormality in the vertebrobasilar system.

The clinical course was uneventful. Seven months later, follow-up MR imaging was performed using another 3-Tesla scanner (Magnetom Skyra; Siemens Healthineers, Erlangen, Germany). On fluid-attenuated inversion recovery (FLAIR) sequences, an abnormal signal was found at the central portion of the left MCP, indicative of Wallerian degeneration of the PCT. There was no abnormality at the contralateral right MCP (Fig. 2).

Discussion

The MCP is the largest peduncle connecting the brainstem and cerebellum, forming the PCT. The corticopontine tracts have a synapse with the ipsilateral pontine nuclei, which are mainly located at the ventromedial upper pons. The axons arising from the pontine nuclei cross the midline of the pons craniocaudally and pass through the contralateral MCP to reach the cerebellar cortex.

Paramedian pontine infarcts are regarded as atherosclerotic branch disease [7]. Our patient had atherosclerotic risk factors, including diabetes mellitus and hyperlipidemia. In the case of unilateral paramedian pontine infarct, bilateral proximal PCTs are affected, resulting in Wallerian degeneration of the distal PCTs, bilaterally and symmetrically (Fig. 3A) [2-6]. Okamoto et al. [8] reported many kinds of pathological conditions presenting bilateral and symmetrical MCP lesions. The present case had paramedian lower pontine infarction, and Wallerian degeneration of the distal PCT appeared ipsilaterally, because contralateral proximal PCTs, which were located in the upper pons, were not affected (Fig. 3B). It was reported that Wallerian degeneration of the ipsilateral and unilateral PCT developed in a case with a laterally-located pontine infarct [2], similar to our patient. Because contralateral proximal PCTs were not located in the lateral pons. In contrast, in the case of localized paramedian upper pontine infarct, Wallerian degeneration of the contralateral and unilateral PCT may develop.

It is well known that Wallerian degeneration of the corticospinal (pyramidal) tract is observed along the total length of the tract [9]. In contrast, Wallerian degeneration of the PCT is detectable in a limited area. It is not known why Wallerian degeneration is limited to the central portion of the MCP. We speculated that because the central portion of the MCP has the highest concentration of myelinated fibers, signal changes are most apparent.

The differential diagnosis of unilateral MCP lesions includes cerebral infarction of the anterior inferior cerebellar artery territory, brain tumors (e.g., glioma), demyelinating lesions, inflammatory lesions and multiple system atrophy [10]. The present case had atherosclerotic risk factors and a history of paramedian lower pontine infarction. Although the clini-



Fig. 2 – Fluid-attenuated inversion recovery sequences obtained 7 months later show a hyperintense lesion at the left middle cerebellar peduncle (*arrow*), indicative of Wallerian degeneration of the left pontocerebellar tract (PCT). No lesion is seen on the right side.





cal course was uneventful, a new lesion appeared at the MCP. Therefore, Wallerian degeneration of the PCT at the MCP can be regarded as the most reliable diagnosis.

Conclusions

We presented a case of ipsilateral Wallerian degeneration of the MCP that developed after paramedian lower pontine infarction. The location of the pontine infarct (affected PCT) and Wallerian degeneration on the side of the MCP were well correlated.

Patient consent

Informed consent was obtained from the patient for publication of the Case Report.

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