Anatomy and pathology of the pontocerebellar fibers in patients with paramedian pontine infarct demonstrated with MRI and DTI

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Learning objectives

Discuss the anatomy of the pontocerebellar fibers (PCF) and post-ischemic wallerian degeneration (WD) of PCF utilizing diffusion tensor imaging (DTI)
Background

The PCF originate from the precentral and postcentral gyri and are part of the cortico-ponto-cerebellar pathway. The fibers arise in the premotor, supplementary motor, posterior parietal, prefrontal, and temporal cortices and then course through the anterior limb of the internal capsule, extending through the cerebral peduncle and terminate in the paramedian pontine nuclei. The second order neurons arise from such pontine nuclei, decussate (Figure 1) and extend to the contralateral cerebellar hemisphere via the middle cerebellar peduncles to give rise to the pontocerebellar pathways. The neural signals are hence transmitted from the cerebral cortex to the intermediate and lateral cerebellar zones which are involved in initiation, planning, and timing of volitional motor activity. These fibers also play an important role in maintaining posture, balance, and coordination. The afferent fibers connect the contralateral paramedian pontine nuclei to the cerebellum. Therefore, this conduit links the cerebral cortex to the contralateral cerebellar hemisphere, affecting motor coordination and lesions along this pathway result in ipsilateral limb and gait ataxia.\(^4\)

A paramedian pontine infarct (PPI) results in damage to the ipsilateral pontine nucleus, the ipsilateral PCF and the proximal course of the contralateral PCF. A PPI may result in WD affecting bilateral PCF, a rare phenomenon that has been previously reported in the literature, based on demonstration of WD using conventional MR imaging. However, the effect of a PPI on the PCF can also be demonstrated employing diffusion tensor imaging (DTI). DTI demonstrates the degree of anisotropy of the fibers, as well as the overall fiber direction. DTI can be utilized to determine fractional anisotropy changes within the PCF. To our knowledge, DTI has not been previously used to quantify WD affecting bilateral PCF with PPI.\(^1,3,4\)
**Fig. 1**: Axial DTI illustrates the middle cerebral peduncles (MCPs), indicated by the yellow arrows. The MCPs connect the contralateral pontine nuclei to the cerebellum. The green color of the MCPs indicates the anteroposterior orientation of the fibers to the cerebellum. The pontine fibers have a left-right direction as illustrated by the red color.

Findings and procedure details

Cases collected in our institution illustrate WD of PCF due to PPI. Patients diagnosed with PPI infarcts had follow up MRI’s demonstrating evolution of symmetric abnormal signal in bilateral MCPs in the course of the PCF and reduction of fractional anisotropy in the PCF on DTI.

DTI is helpful in quantifying the degree of anisotropy and fiber directions. Typically, there are 6 or greater gradient encoding directions with an optional number of encoding directions, typically 20-30. Fractional anisotropy is derived from the standard deviation of 3 eigenvalues and ranges from 0 (isotrophy) to 1 (maximum anisotropy). Orientation of maximal diffusivity is as follows: left to right (red), anterior-posterior (green), and superior-inferior (blue). Regions of decreased fractional anisotropy imply decreased directional coherence and may be a biomarker for microstructural changes in the corresponding white matter.4

At our institution, the MRI with DTI was performed on a 1.5 T Siemens Aera, Erlangen, Germany. The DTI sequence parameters were as follows: Diffusion weightings: 2; B value 1: 0, B value 2: 1000, TR: 5000, TE: 111, ETL: 48, Flip angle: 90, Slices: 30, Slice Thickness: 4, EPI Factor: 128, Noise level: 40, and Number of Differential Directions: 30. The scan was completed in 7 minutes and 39 seconds. Quantification of the DTI images was completed on Invivo DynaSuite Neuro Product workstation, Philips Healthcare, Best, Holland.

Case 1:

38-year-old African female with a remote history of prior stroke, uncontrolled hypertension, uncontrolled hyperlipidemia and morbid obesity presented to the emergency room with progressive speech difficulties. The patient reported occasional word finding difficulty. Subsequent MRI of the brain without contrast demonstrated a 1.7 cm acute/subacute infarct located in the right paramedian pons.

The patient returned to the emergency room 80 days after the initial presentation complaining of weakness for the past 2-3 days and difficulties with ambulation. A brain MRI with DTI was performed. The conventional MRI demonstrated evolution to chronicity of the previously noted PPI and interval development of signal abnormality in the bilateral brachia pontis, consistent with WD affecting bilateral PCF from the recent PPI. (Figure 2). DTI imaging demonstrated asymmetric reduction of fractional anisotropy. (Figure 3). Quantification of the PCF from the DTI demonstrated a decreased fractional anisotropy
of 0.51 (Figure 4). Such reduction was substantial, as compared to a normal patient of similar age in whom fractional anisotropy of the pontocerebellar fibers was 0.68 (Figure 5).

**Case 2:**

55-year-old man with history of uncontrolled diabetes mellitus, presented to the emergency room for intermittent right face and arm numbness/tingling, right facial droop, and moderate dysarthria/stuttering. A stroke code was initiated and the patient was treated with tPA, administered 2 hours and 10 minutes since onset of patient’s symptoms. A brain MRI demonstrated a 12 mm subacute left PPI without hemorrhage (Figure 6).

Eight months following the acute PPI, the patient returned to the emergency room on with blurriness of vision on the right side for several weeks. On physical examination, he was found to have papilledema and a visual acuity of 20/50 in the right eye. A brain MRI was preformed with demonstrated evolution to chronicity of the left PPI and interval development of signal abnormality in bilateral brachia pontis. (Figure 6)

Both clinical cases demonstrate development of abnormal hyperintense FLAIR signal in bilateral brachia as a result of a preceding PPI infarct; while the PPI was unilaterally located in both cases, the evolution of the infarct led to abnormal signal in the ipsilateral and contralateral middle cerebellar peduncles. These findings represent Wallerian degeneration occurring in bilateral PCF secondary to the PPI affecting the location where the PCF decussate, thus accounting for the apparent bilateral involvement. As expected with evolving WD, disruption of the white matter tracts in the PCF will lead to reduction of fractional anisotropy, as shown by DTI evaluation performed in Case 1.
Fig. 2: MRI without contrast. DWI/ADC map for acute presentation (left) with acute infarct in the right paramedian pons. FLAIR sequence on MRI performed 2 months after initial presentation (right) demonstrating abnormal hyperintense signal in the bilateral brachia pontis, consistent with WD in bilateral pontocerebellar fibers secondary to the initial right paramedian pons infarct.

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Fig. 3: Axial DTI imaging at the level of the descruating pontine fibers demonstrating increased fractional anisotropy in the brachia pontis bilaterally from the right paramedian pons infarct.

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Fig. 4: Axial DTI demonstrating quantification of the pontocerebellar fibers with decreased fractional anisotropy.

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Fig. 5: Axial DTI demonstrating quantification of the PCF in a normal control patient, revealing higher fractional anisotropy values as compared to the patient with PPI and WD of the PCF, shown in Figure 4.

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Fig. 6: MRI without contrast. DWI/ADC map for acute presentation (left) with acute infarct in the left paramedian pons. FLAIR sequence (right) demonstrating abnormal hyperintense signal in the bilateral brachia pontis demonstrating the bilateral pontocerebellar fibers being affected by the left paramedian pons infarct.

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Conclusion

A PPI affects the ipsilateral pontine nucleus and bilateral PCF, which can lead to WD in bilateral MCP. Previous case reports in the literature have described this evolution with conventional imaging. We demonstrate the utility of DTI in patients with PPI, revealing fractional anisotropy changes in PCF.
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References

