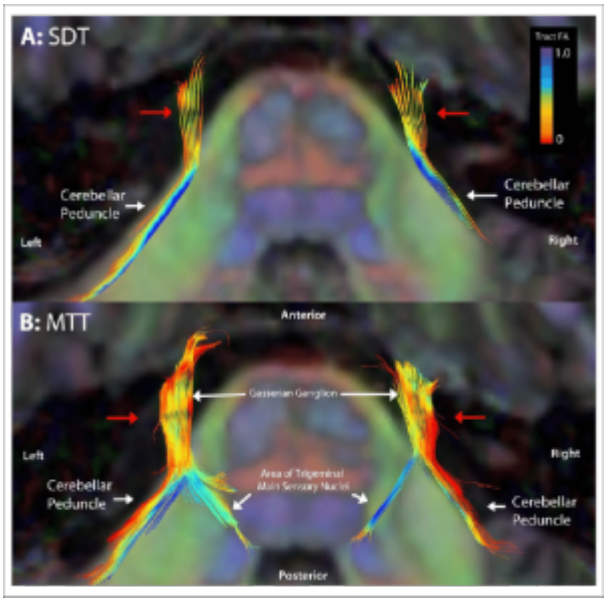
Diffusion tensor imaging for trigeminal neuralgia



A total of 22 patients with classic trigeminal neuralgia and 22 healthy controls (HC) with matching age, gender, and education were selected. All subjects underwent 3.0 T magnetic resonance diffusion tensor imaging and high resolution T1-weighted imaging. The corpus callosum (CC) was reconstructed by DTI technology, which was divided into three substructure regions: genu, body, and splenium. Group differences in multiple diffusion metrics, including fractional anisotropy (FA), mean diffusivity (MD), axial diffusivity (AD) and radial diffusivity (RD), were compared between CTN patients and HC, and correlations between the white matter change and disease duration and VAS in CTN patients were assessed.

Compared with HC group, CTN patients had extensive damage to the CC white matter. The FA of the genu (P = 0.04) and body (P = 001) parts decreased, while RD (P = 0.003; P = 0.02) and MD (P = 0.002; P = 0.04) increased. In addition, the authors observed that the disease duration and VAS of CTN patients were negatively correlated with FA.

The corpus callosum substructure region has extensive damage in chronic pain, and the selective microstructural integrity damage was particularly manifested by changes in axons and myelin sheath in the genu and body of corpus callosum [1].

Diffusion tensor imaging (DTI) has revealed microstructural changes in the symptomatic trigeminal root and root entry zone of patients with unilateral TN.

Noninvasive DTI analysis of patients with TN may lead to improved trigeminal neuralgia diagnosis of TN subtypes (e.g., TN1 and TN2) and improve patient selection for surgical intervention. DTI measurements may also provide insights into prognosis after intervention, as TN1 patients are known to have better surgical outcomes than TN2 patients [2].

As diffusion tensor imaging (DTI) is able to assess tissue integrity, Leal et al., used diffusion to detect abnormalities in trigeminal nerves (TGN) in patients with trigeminal neuralgia (TN) caused by neurovascular compression (NVC) who had undergone microvascular decompression (MVD).

Using DTI sequencing on a 3-T MRI scanner, we measured the fraction of anisotropy (FA) and apparent diffusion coefficient (ADC) of the TGN in 10 patients who had undergone MVD for TN and in 6 normal subjects. We compared data between affected and unaffected nerves in patients and both nerves in normal subjects (controls). We then correlated these data with CSA and V. Data from the affected side and the unaffected side before and 4 years after MVD were compared.

RESULTS: Before MVD, the FA of the affected side (0.37 ± 0.03) was significantly lower ($p < 0.05$) compared to the unaffected side in patients (0.48 ± 0.03) and controls (0.52 ± 0.02), and the ADC in the affected side (5.6 ± 0.34 mm2/s) was significantly higher ($p < 0.05$) compared to the unaffected side in patients (4.26 ± 0.25 mm2/s) and controls (3.84 ± 0.18 mm2/s). Affected nerves had smaller V and CSA compared to unaffected nerves and controls ($p < 0.05$). After MVD, the FA in the affected side (0.41 ± 0.02) remained significantly lower ($p < 0.05$) compared to the unaffected side (0.51 ± 0.02), but the ADC in the affected side (4.24 ± 0.34 mm2/s) had become similar ($p > 0.05$) to the unaffected side (4.01 ± 0.33 mm2/s).

CONCLUSIONS: DTI revealed a loss of anisotropy and an increase in diffusivity in affected nerves before surgery. Diffusion alterations correlated with atrophic changes in patients with TN caused by NVC. After removal of the compression, the loss of FA remained, but ADC normalized in the affected nerves, suggesting improvement in the diffusion of the trigeminal root [3)].

Herveh et al. studied the trigeminal nerve in seven healthy volunteers and six patients with trigeminal neuralgia using the diffusion tensor imaging derived parameter fractional anisotropy (FA). While controls did not show a difference between both sides, there was a reduction of FA in the affected nerve in three of six patients with accompanying nerve-vessel conflict and atrophy. Reversibility of abnormally low FA values was demonstrated in one patient successfully treated with microvascular decompression [4)].

3T MR diffusion weighted, T1, T2 and FLAIR sequences were acquired for Multiple sclerosis related trigeminal neuralgia MS-TN, TN, and controls. Multi-tensor tractography was used to delineate CN V across cisternal, root entry zone (REZ), pontine and peri-lesional segments. Diffusion metrics including fractional anisotropy (FA), and radial (RD), axial (AD), and mean diffusivities (MD) were measured from each segment.

CN V segments showed distinctive diffusivity patterns. The TN group showed higher FA in the cisternal segment ipsilateral to the side of pain, and lower FA in the ipsilateral REZ segment. The MS-TN group showed lower FA in the ipsilateral peri-lesional segments, suggesting differential microstructural changes along CN V in these conditions.

The study demonstrates objective differences in CN V microstrucuture in TN and MS-TN using non-invasive neuroimaging. This represents a significant improvement in the methods currently available

to study pain in MS [5].

The aim of a study was to evaluate the microstructural tissue abnormalities in the trigeminal nerve in symptomatic trigeminal neuralgia not related to neurovascular compression using diffusion tensor imaging. Mean values of the quantitative diffusion parameters of trigeminal nerve, fractional anisotropy and apparent diffusion coefficient, were measured in a group of four symptomatic trigeminal neuralgia patients without neurovascular compression who showed focal non-enhancing T2-hyperintense lesions in the pontine trigeminal pathway. These diffusion parameters were compared between the affected and unaffected sides in the same patient and with four age-matched healthy controls. Cranial magnetic resonance imaging revealed hyperintense lesions in the dorsolateral part of the pons along the central trigeminal pathway on T2-fluid-attenuated inversion recovery sequences. The mean fractional anisotropy value on the affected side was significantly decreased ($P = 0.001$) compared to the unaffected side and healthy controls. Similarly, the mean apparent diffusion coefficient value was significantly higher ($P = 0.001$) on the affected side compared to the unaffected side and healthy controls. The cause of trigeminal neuralgia in our patients was abnormal pontine lesions affecting the central trigeminal pathway. The diffusion tensor imaging results suggest that microstructural tissue abnormalities of the trigeminal nerve also exist even in non-neurovascular compression-related trigeminal neuralgia [6].

DTI analysis allows the quantification of structural alterations, even in those patients without any discernible neurovascular contact on MRI. Moreover, our findings support the hypothesis that both the arteries and veins can cause structural alterations that lead to TN. These aspects can be useful for making treatment decisions [7].

The mean diameter of compression arteries (DCA) in NVC patients with TN (1.58 ± 0.34 mm) was larger than that without TN (0.89 ± 0.29 mm). Compared with NVC without TN and HC, the mean values of RD at the site of NVC with TN were significantly increased; however, no significant changes of AD were found between the groups. Correlation analysis showed that DCA positively correlated with radial diffusivity (RD) in NVC patients with and without TN ($r = 0.830$, $p = 0.000$). No significant correlation was found between DCA and axial diffusivity (AD) ($r = 0.178$, $p = 0.077$).

Larger-diameter compression arteries may increase the chances of TN, and may be a possible facilitating factor for TN [8].

Fractional anisotropy (FA) value quantitatively showed the alteration of trigeminal nerve (TGN) caused by Neurovascular compression (NVC). It provided direct evidence about the effect of NVC which facilitated the diagnosis and surgical decision of Type 2 trigeminal neuralgia (TN) . Besides, significant reduction of FA value may predict an optimistic outcome of microvascular decompression (MVD) [9].

Sophisticated structural MRI techniques including diffusion tensor imaging provide new opportunities to assess the trigeminal nerves and CNS to provide insight into TN etiology and pathogenesis.

Specifically, studies have used high-resolution structural MRI methods to visualize patterns of trigeminal nerve-vessel relationships and to detect subtle pathological features at the trigeminal REZ. Structural MRI has also identified CNS abnormalities in cortical and subcortical gray matter and white matter and demonstrated that effective neurosurgical treatment for TN is associated with a reversal of specific nerve and brain abnormalities [10].

Forty-three patients with trigeminal neuralgia were recruited, and diffusion tensor imaging was performed before radiofrequency rhizotomy. By selecting the cisternal segment of the trigeminal nerve manually, they measured the volume of trigeminal nerve, fractional anisotropy, apparent diffusion coefficient, axial diffusivity, and radial diffusivity. The apparent diffusion coefficient and mean value of fractional anisotropy, axial diffusivity, and radial diffusivity were compared between the affected and normal side in the same patient, and were correlated with pre-rhizotomy and post-rhizotomy visual analogue scale pain scores. The results showed the affected side had significantly decreased fractional anisotropy, increased apparent diffusion coefficient and radial diffusivity, and no significant change of axial diffusivity. The volume of the trigeminal nerve on affected side was also significantly smaller. There was a trend of fractional anisotropy reduction and visual analogue scale pain score reduction (P = 0.072). The results suggest that demyelination without axonal injury, and decreased size of the trigeminal nerve, are the microstructural abnormalities of the trigeminal nerve in patients with trigeminal neuralgia caused by neurovascular compression. The application of diffusion tensor imaging in understanding the pathophysiology of trigeminal neuralgia, and predicting the treatment effect has potential and warrants further study [11].

References

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