MEASUREMENT OF AXON DIAMETER AND AXON DENSITY OF THE CORTICOSPINAL TRACT IN IDIOPATHIC NORMAL PRESSURE HYDROCEPHALUS BY Q-SPACE IMAGING

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Target audience: neurologist, neurosurgeon, and those interested in q-space imaging

Purpose: Measurement of axon diameter by diffusion MRI is becoming a topic for investigation of brain microstructural changes in neurological disorders, although it usually requires quite a long scan. Recently proposed two-component low-q fit method for q-space imaging (QSI) enabled analysis of fibers running perpendicular to the image section with a reasonable scanning time.[1] This study aimed to investigate microstructural changes in the cortico-spinal tract (CST) in patients with idiopathic normal pressure hydrocephalus (iNPH).

Methods: 11 patients with iNPH and 10 age-matched controls were recruited. QSI data were obtained with a 3-T unit (Achieva, Philips Healthcare, Best, The Netherlands) using single-shot, echo planar imaging (EPI) sequence with diffusion gradient applied parallel to the y-axis. The scan parameters were: TR/TE = 4500/99 ms, FOV = 240x240 mm^2, matrix size = 96x96, slice thickness = 5 mm, NEX = 2, 16 b-values (0, 1000, 2000, … 15000 s/mm^2), δ/Δ = 39.3/48.7 ms, and acquisition time = 828 sec. Root mean square displacement (RMSD) of intra-axonal space (= axon diameter) and intra-axonal volume fraction (= axon density) of the CST at the levels of internal capsule and body of lateral ventricle were calculated by using two-component low-q fit (1).

Results: Excellent fitting was obtained in all ROIs (R^2 > 0.95). Wilcoxon's rank-sum test revealed significant increase in axon density of CST at the level of ventricular body in the patients (p = .001), whereas no significant difference was observed in the axon diameter. At the level of internal capsule, neither axon diameter or axon density differed significantly between the two groups.

$$E(q) = (1-f_I) \exp(-2\pi^2 q^2 Z_E^2) + f_I \exp(-2\pi^2 q^2 Z_I^2) \quad … (1)$$

f$_I$: the relaxation-weighted intra-axonal space

Z$_E$ and Z$_I$: RMSDs in the extra- and intra-axonal space

Discussion: Our study suggested that CST axon density was increased in areas adjacent to the lateral ventricle in iNPH, whereas axon diameter was not altered. The present results support the idea that patients with iNPH do not suffer from irreversible axonal damage of CST, but have compression and/or stretching of the fiber tract.[2] These findings give us insights into microstructural alterationss of CST and may account for treatable gait disturbance in iNPH.

Conclusion: Two-component low-q fit method of QSI analysis suggested increased CST axon density in patients with iNPH. The clinical relevance, such as monitoring the effect of treatment or prediction of response to surgery, needs further investigation.

Fig 1. Examples of axon diameter map (above) and axon density map (below)

Fig 2. Comparison between the two groups

References

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