Teaching NeuroImages: Morphology of lumbosacral dorsal root ganglia and plexus in hereditary transthyretin amyloidosis

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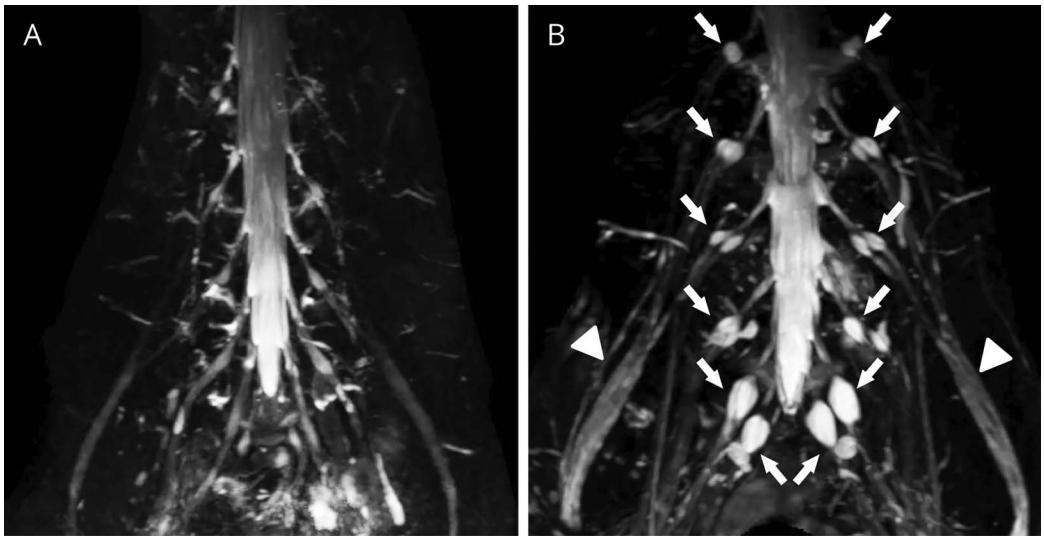
Figure 3D magnetic resonance neurography (3D-MRN) study of lumbosacral nerves



3D-MRN with reconstruction of fat-suppressed T2-weighted images. (A) Healthy volunteer. (B) Patient with the V30M mutation of ATTRm. Dorsal root ganglia: arrows. Lumbosacral plexus: arrowheads.

A 66-year-old man presented with a 5-year history of progressive distal dominant sensorimotor disturbance, which suggested length-dependent polyneuropathy. 3D magnetic resonance neurography (3D-MRN) showed enlargement of the dorsal root ganglia and the lumbosacral plexus (figure). The diagnosis of hereditary transthyretin (ATTRm) amyloidosis was based on genetic and histopathologic findings.

ATTRm amyloidosis commonly causes length-dependent neuropathy with enlargement of the sciatic nerve during the early disease stage.[1] Histopathologically, amyloid deposits occur mainly in the dorsal root ganglia and proximal region of the sciatic nerve.[2] 3D-MRN may detect nerve injury associated with amyloid deposits, which leads to early diagnosis of the disease.

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Author contributions

Dr. Masuda: study concept, study design, acquisition of data, data analysis, drafting of the manuscript. Dr. Ueda: study concept, study design, acquisition of data, data analysis, critical revision of the manuscript for intellectual content, study supervision. Dr. Misumi, Dr. Yamashita, Dr. Obayashi: critical revision of the manuscript for intellectual content. Dr. Morita, Dr. Kitajima, Dr. Yamashita: MRI acquisition, critical revision of the manuscript for intellectual content. Dr. Ando: study concept, study design, critical revision of the manuscript for intellectual content, study supervision.

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References

1. Kollmer J, Hund E, Hornung B, et al. In vivo detection of nerve injury in familial amyloid polyneuropathy by magnetic resonance neurography. Brain 2015;3:549–562.
2. Misu K, Hattori N, Nagamatsu M, et al. Late-onset familial amyloid polyneuropathy type I (transthyretin Met30-associated familial amyloid polyneuropathy) unrelated to endemic focus in Japan: clinicopathological and genetic features. Brain 1999; 122:1951–1962.