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Leukemic Infiltrative Plexopathy: Diagnosis and Follow-Up with Diffusion Tensor Imaging

A 72-year-old woman with stage A B-cell chronic lymphocytic leukemia developed motor deficit and hypoesthesia of the left lower limb. Neurophysiologic examination showed severe axonal changes with denervation abnormalities in the left proximal and distal muscles. Left peroneal nerve biopsy revealed severe acute axonopathy in association with epi- and endoneurial lymphocytic infiltration with CD5+CD20+CD23+ cells and CD3+ T-cells at immunohistochemical phenotyping. Polymerase chain reaction assessment of clonal immunoglobulin gene rearrangements confirmed the clonal identity between the lymphocytes found on the nerve biopsy and the circulating clonal B-cells. Following the diagnosis of leukemic infiltrative plexopathy, the patient was treated with 6 courses of rituximab, fludarabine, and cyclophosphamide associated with monthly intrathecal injections of methotrexate, cytarabine, and hydrocorti-

sone. After treatment, the patient had no pain; both motor deficit and neurophysiologic examination findings were improved.

The patient was examined on a 3T MR imaging scanner (Achieva 3T; Philips Healthcare, Best, the Netherlands) by using a single-shot spin-echo echo-planar imaging diffusion sequence in 15 directions with 2 b-values of 0 and 1000. Trace-weighted images were displayed by using maximum intensity projection (MIP) views. Fiber tracking was performed by positioning multiple regions of interest within the L5 and S1 ventral rami. Mean fractional anisotropy (FA) and apparent diffusion coefficient (ADC) were measured for the tracts. Before chemotherapy, left L5 and S1 ventral rami appeared enlarged and hyperintense compared with the contralateral side on MIP trace-weighted images (Fig 1A). Mean ADC values were lower within the left ventral ramus (L5, $0.856 \times 10^{-3} \text{ mm}^2/\text{s}$; S1, $0.826 \times 10^{-3} \text{ mm}^2/\text{s}$) compared with the right (L5, $1.169 \times 10^{-3} \text{ mm}^2/\text{s}$; S1, $1.210 \times 10^{-3} \text{ mm}^2/\text{s}$) (Fig 1C). Mean FA values were similar between the left (L5

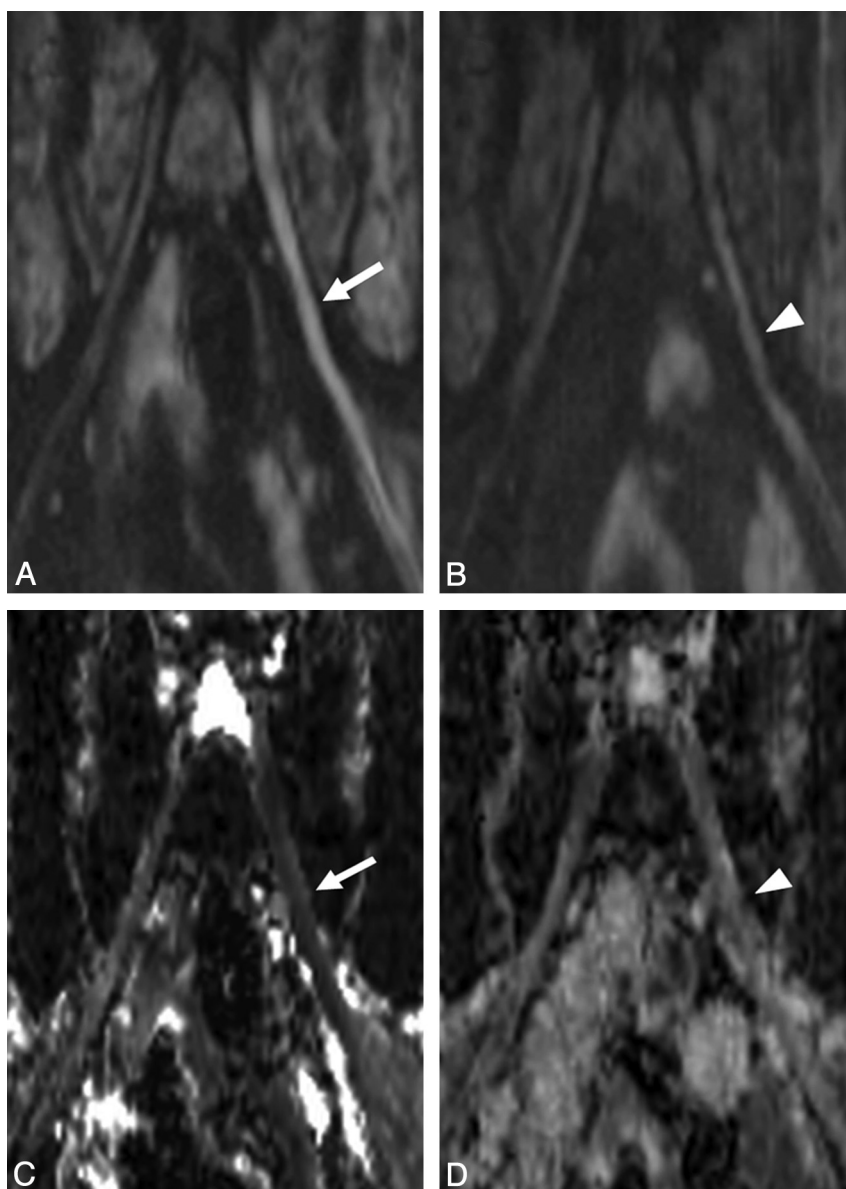


Fig 1. Diffusion tensor imaging of the lumbosacral plexus before (A and C) and after chemotherapy (B and D). A, MIP reformatted view of the trace-weighted images before chemotherapy shows enlargement and hypersignal (arrow) of the left S1 ventral ramus. B, MIP reformatted view of the trace-weighted images after chemotherapy shows that both the enlargement and the hypersignal have decreased (arrowhead). C, ADC map of the S1 ventral rami before chemotherapy shows restricted ADC values within the left S1 ventral ramus (arrow), suggesting an increased cellularity. D, ADC map of the S1 ventral rami after chemotherapy shows a visual increase in ADC values within the left S1 ventral ramus (arrowhead).

and S1, 0.50) and right ventral rami (L5, 0.51; S1, 0.50). After chemotherapy, both the abnormal thickening and hypersignal decreased (Fig 1B). Within the left ventral ramus, mean ADC values increased (L5, $1.369 \times 10^{-3} \text{ mm}^2/\text{s}$; S1, $1.334 \times 10^{-3} \text{ mm}^2/\text{s}$) (Fig 1C), whereas mean FA values decreased (L5, 0.41; S1, 0.44). There were no significant changes in ADC and FA values within the right ventral ramus.

The value of diffusion-weighted imaging has been reported for the investigation of peripheral nerve diseases.¹ In patients with lymphoma, decreased ADC values are commonly observed due to an increased cellularity.² Our results showed a similar feature in leukemic infiltrative plexopathy. The increase in ADC values after chemotherapy also suggested that the ADC changes were related to the amount of infiltrative cells within the pathologic plexus. The mechanism of FA decrease observed after treatment remains unclear; one may speculate an increase in extracellular space secondary to fiber tract destruction.³ Further studies may investigate the value of ADC and FA measurements to distinguish demyelination or remyelination changes from tumoral involvement.

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