Peripheral Nerve Disorders: Chapter 37. Fabry disease (Handbook of Clinical Neurology)

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Fabry disease results from deficient activity of the enzyme α-galactosidase A and progressive lysosomal deposition of globotriaosylceramide (GL-3) in cells throughout the body. The main neurological presentations of Fabry disease patients are painful neuropathy, hypohidrosis, and stroke. Fabry neuropathy is characterized as a length-dependent peripheral neuropathy affecting mainly the small myelinated (Aδ) fibers and unmyelinated (C) fibers. Enzyme replacement therapy (ERT) has been shown to have some positive effects on the reduction of neuropathic pain, the improvement of detection threshold for thermal sensation, and sweat function. On the contrary, the effect of ERT on the central nervous system has not been established. Early initiation of ERT before irreversible organ failure is extremely important, and alternative therapeutic approaches are currently being explored. Heterozygotes suffer from peripheral neuropathy at a higher rate than previously shown, significant multisystemic disease, and severely decreased quality of life. As well as being carriers, heterozygotes also display symptoms of Fabry disease, and should be carefully monitored and given adequate therapy.

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