



# **Peripheral Nerve Disorders: Chapter 39. Hereditary gelsolin amyloidosis (Handbook of Clinical Neurology)**

*Sari Kiuru-Enari, Matti Haltia*

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Hereditary gelsolin amyloidosis (HGA) is an autosomally dominantly inherited form of systemic amyloidosis, characterized mainly by cranial and sensory peripheral neuropathy, corneal lattice dystrophy, and cutis laxa. HGA, originally reported from Finland and now increasingly from other countries in Europe, North and South America, and Asia, may still be underdiagnosed worldwide. It is the first and so-far only known disorder caused by a gelsolin gene defect, namely a G654A or G654T mutation. Gelsolin is a principal actin-modulating protein, implicated in multiple biological processes, also in the nervous system, e.g. axonal transport, myelination, neurite outgrowth, and neuroprotection. The gelsolin gene defect causes expression of variant gelsolin, followed by systemic deposition of gelsolin amyloid (AGel) in HGA patients and even other consequences on the metabolism and function of gelsolin. In HGA, specific therapy is not yet available but correct diagnosis enables adequate symptomatic treatment which decisively improves the quality of life in these patients. A transgenic murine model of HGA expressing AGel is available, in anticipation of new treatment options targeted toward this slowly progressive but devastating amyloidosis. Present and future lessons learned from HGA may be applicable even in diagnosis and treatment of other hereditary and sporadic amyloidoses.

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