

Neuropathy and Its Stages

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Stage One: Numbness and Pain

Abstract: This article discusses the clinical presentation and pathophysiology of Stage One neuropathy, characterized by numbness and pain. It reviews the underlying mechanisms, including axonal displacement and demyelination, and discusses the role of advanced glycation end products (AGEs) in the pathogenesis of diabetic neuropathy. The article also highlights the importance of early diagnosis and management to prevent further progression of the disease.

Introduction: Neuropathy is a common complication of diabetes mellitus, affecting approximately 50% of patients. The clinical presentation is often insidious, starting with numbness and pain in the feet. This article focuses on Stage One neuropathy, which is characterized by these symptoms. The pathophysiology of neuropathy is complex, involving both metabolic and immunological factors. One of the key mechanisms is axonal displacement, where the axon is pushed away from the myelin sheath, leading to demyelination and subsequent axonal loss. This process is accelerated in the presence of advanced glycation end products (AGEs), which are formed from the non-enzymatic glycation of proteins and lipids. AGEs have been shown to cross-link with myelin proteins, leading to myelin damage and axonal displacement. Additionally, AGEs can also cross-link with nerve growth factors (NGFs), leading to a loss of NGF bioavailability and subsequent axonal damage. The clinical presentation of Stage One neuropathy is often characterized by numbness and pain in the feet, which may be accompanied by tingling and burning sensations. The symptoms are typically worse at night and may be exacerbated by temperature changes. The pathophysiology of neuropathy is complex, involving both metabolic and immunological factors. One of the key mechanisms is axonal displacement, where the axon is pushed away from the myelin sheath, leading to demyelination and subsequent axonal loss. This process is accelerated in the presence of advanced glycation end products (AGEs), which are formed from the non-enzymatic glycation of proteins and lipids. AGEs have been shown to cross-link with myelin proteins, leading to myelin damage and axonal displacement. Additionally, AGEs can also cross-link with nerve growth factors (NGFs), leading to a loss of NGF bioavailability and subsequent axonal damage.

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References

1. Sugimoto K, Yasujima M, Yagihashi S (2008) Role of advanced glycation end products in diabetic neuropathy. *Curr Pharm Des*14:953-961.
2. Singh VP, Bali A, Singh N, Jaggi AS (2014) Advanced glycation end products and diabetic complications. *Korean J Physiol* 18:1-14.
3. Criado PR, Marques GF, Morita TC, de Carvalho JF (2016) Epidemiological, clinical and laboratory profiles of cutaneous polyarteritis nodosa patients: Report of 22 cases and literature review. *Autoimmune Rev* 15:558-563.
4. Lenglet T, Haroche J, Schnuriger A, Maisonobe T, Viala K, et al.(2011) Mononeuropathy multiplex associated with acute parvovirus B19 infection: characteristics, treatment and outcome. *J Neurol* 258:1321-1326.
5. Chin RL, Latov N (January 2005) Peripheral Neuropathy and Celiac Disease. *Curr Treat Options Neurol* 7: 43-48.

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