Severity of Diabetes in Patients with Allodynia and Hyperalgesia as Major Symptoms

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Description

Diabetic Neuropathy and Diabetic Neuropathic Pain (DNP) are common complications of long-term diabetes. DNP severs an intractable and characterized by spontaneous painful sensations (e.g., burning or sharp pain) cutaneous allodynia and hyperalgesia, impacting patients' quality of life and causing mood disturbances. There is no treatment for diabetic neuropathy and disturbingly long history of therapeutic approaches showing promise in preclinical studies in which failing to translate the clinic [1]. Hyperglycemia-induced nerve damage has been considered as a pivotal role in the pathophysiology of DNP. Damaged myelination in afferent nerve fibers which may induce dysfunction in nociceptive transduction, resulting in hyperalgesia and allodynia. Myelin abnormalities have been observed in patients with diabetes and animal models of DNP.

Do the myelin abnormalities represent the severity of diabetes in patients with allodynia and hyperalgesia as major symptoms I which molecules may regulate the myelin alteration? The findings presented in the research recently published in Aging and Diseases support the notion that axonal demyelination plays a key role in the development of DNP and may represent the severity of diabetic painful symptoms manifested as allodynia and hyperalgesia [2]. The myelin damage may be used as markers for diagnosis diabetic neuropathy. The underlying

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