Brief Note on Guillain Barre Syndrome

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About the Study

Guillain Barre Syndrome is a rare, but potentially fatal, immune-mediated disease of the peripheral nerves and nerve roots that is typically caused by infections. Guillain Barre Syndrome comprises of at least 4 subtypes of acute peripheral neuropathy. Major advances have been made in considerate the mechanisms of some of the subtypes. The histological arrival of the Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) subtype resembles experimental autoimmune neuritis, which is primarily triggered by T cells directed against peptides from the myelin proteins P0, P2, and PMP22. The role of T cell mediated immunity in AIDP remains unclear and there is confirmation for the involvement of antibodies and complement. Durable confirmation now exists that axonal subtypes of guillain barre syndrome, Acute Motor Axonal Neuropathy (AMAN), and Acute Motor And Sensory Axonal Neuropathy (AMSAN), are triggered by antibodies to gangliosides on the axolemma that target macrophages to occupy the axon at the node of Ranvier. About a quarter of patients with Guillain Barre Syndrome have had a current Campylobacter jejuni infection, and axonal forms of the ailment are especially common in these people. The lipo oligosaccharide from the C jejuni bacterial wall comprises ganglioside like structures and its injection into rabbits persuades a neuropathy that resembles acute motor axonal neuropathy. Antibodies to GM1, GM1b, GD1a, and GalNac-GD1a are in specific implicated in acute motor axonal neuropathy and, with the exclusion of GalNacGD1a, in acute motor and sensory axonal neuropathy. The Fisher's syndrome subtype is particularly accompanying with antibodies to GQ1b, and

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