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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 parallel cross-reactivity with ganglioside structures in the wall of C jejuni has been discovered. Anti GQ1b antibodies have been shown to harm the motor nerve terminal in vitro by a complement-mediated mechanism. Results of international randomised trials have shown equal efficacy of both plasma exchange and intravenous immunoglobulin, but not corticosteroids, in hastening retrieval from Guillain Barre Syndrome.

The Guillain Barre Syndrome is an idiosyncratic neuropathy characterized pathologically by the presence of inflammatory lesions which occur distributed throughout the peripheral nervous system. The lesions consist of bounded areas in which myelin is lost in the presence of lymphocytes and macrophages. Myelin damage is affected largely by macrophages, which infiltrate the basement membrane around nerve fibers and strip what appears to be normal myelin away from the body of the Schwann cell and off the axon. While there is confirmation that this activity is immune mediated, the specific mechanism that leads macrophages to seek out and amputate a specialized region of the Schwann cell plasma membrane remains unexplained.

Diagnosis and management of GBS can be difficult as its clinical presentation and ailment course are heterogeneous, and no international clinical guidelines are presently available. To support clinicians, especially in the context of an outbreak, the ten steps then cover early recognition and diagnosis of GBS, admission to the intensive care unit, treatment indication and selection, monitoring and treatment of disease development, forecast of clinical course and outcome, and management of complications and sequelae.

Guillain Barre Syndrome is an identifiable entity for which the basis for diagnosis is descriptive in our present state of knowledge. Diagnosis rests upon pattern recognition of the clinical picture plus other features including elevated cerebrospinal fluid protein level, electrophysiological variations of marked slowing of conduction velocities, prolonged distal latencies, dispersion of the rise responses, and frequent evidence of conduction block, together with pathological changes, when known, of low-grade inflammation and demyelination remyelination in peripheral nerve. The precise diagnostic limits of GBS remain indeterminate.