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Distinction between Guillain-Barre Syndrome and Chronic Inflammatory Demyelinating Polyneuropathy

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Most of the cases are straightforward and are easily distinguishable as Chronic Inflammatory Demyelinating Polyneuropathy [CIDP] or Acute Inflammatory Demyelinating Polyneuropathy [AIDP]. AIDP is the most common form of Guillain-Barre syndrome [GBS]. AIDP has a more rapid course. Symptoms typically reach a nadir in 4 weeks or less. Back pain and autonomic symptoms are common in AIDP, along with bulbar involvement or respiratory compromise. CIDP has a more indolent course. As per well-established criteria, it takes more than 8 weeks to develop the greatest weakness in CIDP. Patients typically present with weakness in both proximal and distal muscle groups, sensory loss, and paresthesias which could be slowly progressive or they present with a more relapsing/remitting course. Unlike AIDP, autonomic symptoms and back pain are less common in CIDP. Also, respiratory compromise or bulbar involvement is rare in CIDP patients [1].

Although, as per established criteria, greater than 8 weeks is a time threshold considered to separate CIDP from AIDP, 16% of CIDP patients present with temporal evolution of symptoms in less than 8 weeks. Therefore, these patients simulate GBS due to more acute presentation. On average 8-16% of GBS patients suffer worsening symptoms after starting treatment, which is referred to as Treatment-Related-Fluctuations [TRFs]. The occurrence of 3 or greater TRFs should raise suspicion of CIDP. Worsening of symptoms 8 weeks after onset of disease should raise concerns of CIDP rather than GBS [2-6]. Moreover, GBS-TRF patients nadir in symptoms sooner than CIDP [less than 4 weeks in comparison to 4-8 weeks]. Although prolonged but nadir is less severe in CIDP.

Both GBS and CIDP are immune-mediated disorders. The exact pathogenesis of these immune-mediated illnesses is not clear to date. Two-thirds of GBS patients have preceding infection [7]. It is considered that the occurrence of infection creates the immune response, which later leads to a cross-reaction towards the normal body tissues. Antibodies are produced directed to epitopes of peripheral nerves and myelin sheath leading to an autoimmune process. Molecular mimicry between nerve cell proteins and microbial proteins is a widely considered mechanism behind the pathogenesis of demyelinating autoimmune diseases. There are numerous antibodies such as GM1, GD1a, GD1b, GQ1b associated with GBS.

CIDP is typically an idiopathic disease. Although both humoral and cell-mediated immune mechanisms are postulated, T-cell mediated infiltration of nerve cells is the main immune mechanism in CIDP. It is rare to have an association of preceding infection before the onset of symptoms in CIDP [8]. Usually, there are no autoantibodies identified in CIDP patients. Rarely, that too in more severe disease phenotypes of CIDP, antibodies directed against myelin or Ranvier node proteins are described [such as contactin-1, gliomedin, and neurofascin] [8].

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