ISSN: 2754-4516

Journal of Medicine and Healthcare

SCIENTIFIC Research and Community

Opinion Article

Open Access

Chronic Inflammatory Demyelinating Polyneuropathy [CIDP]

Simranjit Singh[1], Fnu Sanna[1] and Jashan J S Dhaliwal[2]

[1] Department of Clinical Medicine, Indiana University School of Medicine, Indianapolis, IN, USA

[2]Manipal College of Medical Sciences, Pokhara, Nepal

***Corresponding author**

Simranjit Singh, Department of Clinical Medicine, Indiana University School of Medicine, Indianapolis, IN, USA. E-mail: dr.simsingh@outlook.com

Received: May 28, 2022; **Accepted:** June 02, 2022; **Published:** June 09, 2022

Chronic Inflammatory Demyelinating Polyneuropathy [CIDP] is the most common autoimmune polyneuropathy in adults. It is characterized by weakness in both proximal and distal muscle groups, areflexia, slower onset [greater than 8 weeks], electrodiagnostic features of conduction block with asymmetric conduction velocity slowing, and cytoalbuminologic dissociation [elevated CSF protein without pleocytosis] [1]. Electrodiagnostic studies in acquired demyelination are consistent with conduction block which shows a decreasing amplitude of the compound muscle action potential [CMAP] at more proximal stimulation sites. It also shows temporal dispersion which is prolonged CMAP duration after proximal stimulation compared to distal stimulation. CIDP is distinguished from other neuropathies by F-wave latency. Nerve biopsy findings in CIDP are consistent with segmental demyelination, edema, onion bulb formation, and perineural inflammation [1].

CIDP presents in multiple variants such as Multifocal Acquired Demyelinating Sensory and Motor Neuropathy [MADSAM or Lewis-Sumner syndrome], Sensory Predominant CIDP, and Distal Acquired Demyelinating Symmetric [DADS] CIDP [2-4].

The exact etiology of CIDP is still unknown, however, inflammation or autoimmunity is the key mechanism behind its pathogenesis. Humoral immune factors are involved as patients respond to corticosteroids, intravenous immunoglobulins [IVIG], or plasma exchange treatments. Cellular immune mechanisms are also considered a key feature in CIDP pathogenesis. The presence of macrophages and T cells in the nerves as perivascular inflammation and infiltrates are suggestive of cell-mediated mechanisms of nerve damage resulting in demyelination. Moreover, cerebrospinal fluid analysis of CIDP patients has shown elevated T helper cells [5].

CIDP diagnosis is largely dependent on characteristic history and typical clinical features. Electrophysiological studies and CSF examination plays an important role. Rarely, nerve biopsy is required, more so to exclude other disorders than to confirm the diagnosis of CIDP. In the presence of typical clinical features of CIDP, the mere absence of typical electrophysiological or pathological features should not exclude the diagnosis. On the same hand, the presence of demyelinating features on electrophysiological studies or nerve biopsy in a patient otherwise lacking clinical features of CIDP does not confirm CIDP diagnosis.

Guillain-Barre syndrome [GBS] and CIDP distinction remain important and challenging task in clinical practice. CIDP has a slower course in comparison to GBS. As per established criteria, CIDP takes more than 8 weeks to develop the greatest weakness whereas GBS reaches a nadir in 4 weeks or less. Unlike GBS, autonomic symptoms and back pain are less common in CIDP. In comparison to GBS, CIDP has a lesser incidence of bulbar involvement or respiratory compromise. Typically, CIDP patients present with a slowly progressive or relapsing/remitting course of paresthesias, sensory loss, and weakness in both proximal and distal muscle groups.

Similar to multiple sclerosis, CIDP has a variable and heterogeneous prognosis. 20-65% of the CIDP patients follow a relapsing-remitting course whereas others follow a more progressive course [6, 7]. Patients with CIDP without associated conditions, especially when cerebrospinal fluid protein is elevated, generally have a good response to the treatment. The mainstay of treatment is a combination of corticosteroids, plasma exchange, and/or IVIG. Monoclonal protein presence in patients portends a poor prognosis and poor response to immunomodulatory treatment.

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