Session X: Disease Related Morbidities in

Waldenström's Macroglobulinemia

Abstract 153

Presenter: T. Levine

Treatment of Peripheral Neuropathies Associated with IgM Monoclonal Gammopathies. Todd D. Levine, Phoenix Neurological Associates, Phoenix, AZ, USA.

Peripheral neuropathy is frequently associated with an IgM monoclonal gammopathy. However gammopathies and peripheral neuropathies increase in frequency as people age, therefore in many cases this may be a comorbid association and not a causal relationship. This is further complicated by the fact that there are several mechanisms by which monoclonal gammopathies may damage nerves. IgM monoclonal proteins may react directly against specific neural antigens and many of these antigens are now commercially detectable by ELISA. In some cases these autoantibodies define a specific clinical and electrophysiologic syndrome while other autoantibodies are associated with a more varied c;inical picture. If there are no specific autoantibodies detectable, then one must decide whether there is a causal relationship. The specific electrophysiologic and clinical characteristics of the neuropathy can often suggest whether the MGUS is causal in the development of the neuropathy. Demyelinating features on nerve conduction studies such as slowed conduction velocities or prolonged distal latenceis, are associated with immune mediated neuropathies and suggest that the monoclonal protein is responsible for the neuropathy. Further testing such as nerve biopsiescan reveal that the proteinsmay aggregate and deposit within neural or vascular tissues. Once the clinical decision has been reached that the IgM monoclonal gammopathy is possibly responsible for the peripheral neuropathy, then once must choose from a number of available treatment options.If a very high level of IgM is detected suggesting an underlying malignancy such as Waldenström's, then treatment should be directed at the most appropriate chemotherapy for the malignancy and the hope is that the neuropathy will improve coincidentally with the underlying disease. If there are features of demyelination on nerve conduction studies or nerve biopsy then Intravenous Gammoglobulin can be used safely and easily. Finally treatments designed to lower the specific IgM monoclonal protein can be entertained such as Rituximab or even plasmaphresis in combination with chemotherapy.