

Waldenstrom's and Neurologic Complications IgM Neuropathy and the Bing-Neel Syndrome

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Although Waldenstrom's can produce neurologic difficulties of vision and thought as a result of hyperviscosity associated with deposition of IgM within blood vessels; more common are the non-hyperviscosity nerve and vessel effects of IgM.

In this presentation we will review the effects on nerve and brain of IgM in the blood that deposits in each location. For the nerve, IgM deposition leads to unwrapping of the insulating myelin sheaths surrounding peripheral nerve. The *demyelinated* nerve and the disappearance of axons produce numbness and tingling and sensation loss. Diagnosis of this condition rests on patient awareness of *neuropathic* symptoms, the demonstration of elevated levels of IgM in blood and the demonstration of IgM proteins directed against myelin (*MAG: myelin associated glycoprotein*). Prior to therapy with Rituxan electrical studies confirm the axonal and demyelinative neuropathy, increasingly used are small samples of skin to demonstrate the loss of nerve fibers and the demonstration of IgM deposits. Therapy consists of reduction of IgM as well as the use of both prescription and non-prescription drugs to reduce tingling and numbness.

It must be assumed that IgM deposition also occurs in the brain to produce headaches and changes of coordination and thought. The term *Bing-Neel syndrome* refers to the consequences of either this deposition or the passage of Waldenstrom's cells into the brain substance and covering of the brain. We will discuss the experience with Bing-Neel syndrome to facilitate a study (of the International Waldenstrom's Macroglobulinemia Foundation) by prospective identification of these Bing-Neel cases. In this fashion a consensus definition of both peripheral and central WM-related IgM diseases can be reached and we can develop markers for the development of these complications.