

CLINICAL SPECIFICATIONS

ASIALOGLANGLIOSIDE

Function:

Gangliosides are glycosphingolipids. Asialo GM1 has important physiological properties and impacts neuronal plasticity and repair mechanisms, as well as the release of neurotrophins in the brain. Asialo GM1 acts as the site of binding for both Cholera toxin and *E. coli*.

Antibodies Appear:

Chronic Inflammatory Demyelinating Polyneuropathy²
Cerebrovascular Accidents¹
Cranial Trauma¹
Guillain Barré Syndrome³
Miller Fisher Syndrome³
Motor Neuron Disease^{1, 2}
MMNBC¹
Multifocal Motor Neuropathy²
Multiple Sclerosis¹
Myasthenia Gravis¹
PANDAS / ANDAS / OCD⁵
Rheumatoid Arthritis¹
Sensorimotor Neuropathy²
Systemic Lupus Erythematosus^{1, 2}

Known Cross-Reactions: *Campylobacter jejuni*,³ Streptococcal proteins,⁴ gliadin⁶

Clinical Significance:

GM1 is exposed at the surface of spinal motor neurons, and in the peripheral nerves, it is limited to the node and paranodal region. Low levels of antibodies can be found in normal individuals and in patients with certain autoimmune disorders, however, high titers may be helpful in the diagnosis of multifocal motor neuropathy with conduction block (MMNCB),² and paraproteinemia including motor neuron disease and multifocal motor neuropathy.¹ Autoantibodies to ganglioside GM1 are thought to be involved in the pathophysiology of some motor neuropathies due to relatively high level detection prior to therapeutic intervention, and a subsequent decrease of antibodies concurrent with clinical improvement.¹ High titers of IgM Antiganglioside GM1 may be etiologically important in certain types of motor neuropathy, while on the other hand, low levels of antibodies may represent B-cell dysfunctional regulation.² The origin of anti-ganglioside antibodies is unknown, however, it is speculated that they can be formed against certain infectious microorganisms. *Campylobacter jejuni* has been identified as a frequent cause of Guillain Barré syndrome and Miller Fisher syndrome; during infection of *C. jejuni* anti-GM1 antibodies bind with whole cells of different *C. jejuni* isolates.⁴ Likewise, this occurs with Streptococcal infection, which is a trigger for PANDAS, the adult version, ANDAS and OCD.^{4, 5}

References:

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3. Jacobs, et al. Cross-reactive antibodies against gangliosides and *Campylobacter jejuni* lipopolysaccharides in patients with Guillain-Barré or Miller Fisher syndrome. J Infect Disease, 1997; 175:729-733.
4. Mertens, et al. Molecular analysis of cross-reactive anti-myosin/anti-streptococcal mouse monoclonal antibodies. Molecular Immunol, 2000; 37(15):901-913.
5. Vojdani. Obsessive compulsive disorder and differentiation between non-autoimmune OCD and the autoimmune version of the disease called PANDAS. Latitudes, 6(2):1-6
6. Vojdani and Tarash. Cross-reaction between gliadin and different food and tissue antigens, Food Nutri Sci, 2013; 4:20-32.