

Guillain-barré syndrome-clinical manifestations and management

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Abstract

Guillain-Barré Syndrome (GBS) is an acute immune-mediated disorder of the peripheral nervous system characterized by rapidly progressive muscle weakness and diminished or absent deep tendon reflexes. It is one of the most common causes of acute flaccid paralysis worldwide. The syndrome is often preceded by respiratory or gastrointestinal infections that trigger an abnormal immune response resulting in peripheral nerve damage. Early recognition and timely treatment are essential to prevent severe complications, including respiratory failure and long-term disability. This article discusses the etiology, clinical manifestations, diagnostic methods, and current management strategies of Guillain-Barré Syndrome.

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Keywords: Guillain-Barré syndrome, peripheral neuropathy, autoimmune disease, muscle weakness, paralysis, intravenous immunoglobulin, plasmapheresis.

Guillain-Barré Syndrome is a rare but serious neurological disorder in which the body's immune system mistakenly attacks peripheral nerves. The condition affects individuals of all ages and may develop rapidly over days to weeks. Although the exact cause remains unclear, GBS is frequently associated with preceding infections, particularly those caused by *Campylobacter jejuni*, cytomegalovirus, Epstein-Barr virus, influenza virus, and other pathogens. The incidence of GBS is

estimated at approximately 1–2 cases per 100,000 individuals annually. The pathogenesis of Guillain-Barré Syndrome involves an autoimmune response directed against components of peripheral nerves. Molecular mimicry between infectious agents and nerve tissue antigens leads to the production of antibodies that attack myelin sheaths or axonal structures. This immune-mediated injury disrupts nerve conduction and results in motor, sensory, and autonomic dysfunction. Several variants of GBS have been identified. The most common form is Acute Inflammatory Demyelinating Polyneuropathy (AIDP), characterized by demyelination of peripheral nerves. Other variants include Acute Motor Axonal Neuropathy (AMAN), Acute Motor and Sensory Axonal Neuropathy (AMSAN), and Miller Fisher Syndrome. The hallmark symptom of Guillain-Barré Syndrome is progressive symmetrical weakness that usually begins in the lower extremities and ascends to involve the upper limbs and trunk. Patients often experience tingling sensations, numbness, and pain before the onset of weakness. As the disease progresses, walking becomes difficult, and muscle strength declines significantly. Deep tendon reflexes are typically reduced or absent. Cranial nerve involvement may lead to facial paralysis, difficulty swallowing, impaired speech, and visual disturbances. In severe cases, respiratory muscles become affected, resulting in respiratory insufficiency that may require mechanical ventilation.

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Autonomic dysfunction is another important feature of Guillain-Barré Syndrome. Patients may develop blood pressure instability, cardiac arrhythmias, excessive sweating, urinary retention, and gastrointestinal dysfunction. These complications can be life-threatening and necessitate close monitoring in specialized healthcare settings. Diagnosis of Guillain-Barré Syndrome is based on clinical evaluation and supportive laboratory investigations. Neurological examination reveals progressive weakness and areflexia. Cerebrospinal fluid analysis commonly demonstrates

elevated protein levels with a normal white blood cell count, a finding known as albuminocytologic dissociation. Electromyography and nerve conduction studies are essential for confirming peripheral nerve involvement and differentiating between demyelinating and axonal variants. Additional investigations may be performed to exclude other neurological disorders and identify potential triggering infections. Early treatment is critical for improving outcomes. Intravenous immunoglobulin (IVIG) and plasma exchange are currently considered first-line therapies. Intravenous immunoglobulin works by modulating the immune response and reducing inflammatory nerve damage. Plasma exchange removes circulating antibodies and inflammatory mediators responsible for nerve injury. Both treatments have been shown to shorten recovery time and decrease disease severity when administered early in the course of illness. The prognosis of Guillain-Barré Syndrome is generally favorable, with most patients achieving substantial recovery within months to years. However, some individuals experience persistent weakness, fatigue, sensory disturbances, or chronic pain. Severe cases may result in long-term disability or death, particularly when respiratory complications occur. Early diagnosis and prompt treatment are associated with improved outcomes and reduced mortality.

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Conclusion

Guillain-Barré Syndrome is an acute autoimmune neuropathy that can cause rapid and potentially life-threatening neurological deterioration. Recognition of early clinical signs, accurate diagnosis, and timely initiation of immunotherapy are essential for successful management. Advances in supportive care and rehabilitation have significantly improved survival rates and functional recovery. Continued research into the immunological mechanisms of GBS may lead to more effective therapeutic strategies in the future.

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