

GBS OVERVIEW

Everything You Need To Know

Guillain-Barré (Ghee-yan Bah-ray) Syndrome is an inflammatory disorder of the peripheral nerves. It does not affect the brain and spinal cord.

The incidence of Guillain Barré syndrome (GBS) is 1 to 2 people per 100,000 population per year in North America and Europe. There are however higher incidences in areas of Bangladesh and India as well as in other countries through-out the world reflecting local conditions.

What causes GBS?

Fifty to 80% of cases are preceded by either an upper respiratory infection or diarrheal illness. Substantive evidence suggests that antibodies in the blood that are stimulated by molecules on the surface of infectious viruses and organisms cross react with molecules on the peripheral nerve. These antibodies facilitate activation of molecules called complement and white blood cells called macrophages that mediate damage to peripheral nerve resulting in weakness and sensory loss.

What causes GBS?

Fifty to 80% of cases are preceded by either an upper respiratory infection or diarrheal illness. Substantive evidence suggests that antibodies in the blood that are stimulated by molecules on the surface of infectious viruses and organisms cross react with molecules on the peripheral nerve. These antibodies facilitate activation of molecules called complement and white blood cells called macrophages that mediate damage to peripheral nerve resulting in weakness and sensory loss.

How is GBS diagnosed?

To diagnosis GBS as the cause of your weakness or sensory loss, three tests may be performed:

- A neurological examination
- Electrical tests of nerve and muscle function called nerve conduction and electromyography
- A spinal fluid analysis

How is GBS treated?

GBS in its early stages is unpredictable, so except in very mild cases, most newly

diagnosed patients are hospitalized for observation. At onset, a patient with GBS can be admitted to ICU (Intensive Care) or step down unit to monitor breathing and other body functions until the disease is stabilized. Plasma exchange (a blood "cleansing" procedure) or high dose intravenous immune globulins are often given to shorten the course of GBS. The acute progressive phase of GBS typically varies in length from a few days to 4 weeks, with over 90% of patients moving onto the rehabilitative phase within four-six weeks. Longer periods of hospitalization may be required if a patient develops an intercurrent infection of the blood, lung or kidney. Patient care involves the coordinated efforts of a team such as a neurologist, physiatrist (rehabilitation physician), internist, family physician, physical therapist, occupational therapist, social worker, nurse, and psychologist or psychiatrist. Some patients require speech therapy if speech muscles have been affected.

Variants

There are variants of GBS, but they all share the characteristic of being 'rapid onset':

- *Acute Inflammatory Demyelinating Polyneuropathy (AIDP)* 75% – 80% of cases in the north America and Europe fall into this 'classic' category that affects motor, sensory and autonomic nerves in a symmetrical fashion
- *Acute Motor Axonal Neuropathy (AMAN)* Similar to AIDP, but without sensory symptoms, affects the motor axons of the nerves
- *Acute Motor Sensory Axonal Neuropathy (AMSAN)* Severe variant of GBS more prevalent in Asia, Central America, and South America causing severe, rapid destruction to nerves throughout the body
- *Miller Fisher Syndrome* is characterized by double vision, loss of balance, and loss of deep tendon reflexes

Living with GBS

Recovery may occur over six months to two years or longer. A particularly frustrating consequence of GBS is long-term recurrences of fatigue and/or exhaustion as well as abnormal sensations including pain and muscle aches. These can be aggravated by 'normal' activity and can be alleviated by pacing activity and rest.

