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Review Article

A CRITICAL REVIEW ON GUILLAIN BARRE SYNDROME (ACUTE INFLAMMATORY DEMYELINATING POLYNEUROPATHY- AIDP) AND THEIR MANAGEMENT

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ABSTRACT

Guillain-Barre Syndrome is serious health problem, that occurs when the body`s defence system mistakenly attacks parts of the nervous system. In about 50% of people with these syndrome symptoms begin about 5 days to 3 weeks after a mild infection symptoms include weakness and pin and needle sensation or loss of sensation. It is form of polyneuropathy that cause one episode of increasing muscle weakness. Weakness is more prominent than abnormal sensation reflex. Usually weakness begins both legs and moves up the body. In GBS, the body`s immune system attacks the myelin sheath which surround the axon of many nerves and enable nerve impulses to travel quickly. It may lead to life threatening complication in particular if respiratory muscles are affected or if the autonomic nervous system is involved. Worldwide, the annual incidence is about 0.6- 4.0 occurrences per 1,00,000 people. Electromyography and nerve conduction studies can help confirm the diagnosis. Plasmapheresis or immunoglobulin given intravenously may speed recovery. GBS as such is not correlated with any disease which is described in *Ayurveda* but pathology of GBS is *Vata* dominating disorder along with association of *Pitta* and *Kapha dosha*. As a precipitating factor produced functions of GI biofire plays an important role by producing *Ama* like reactive species in genesis of this disease. So at the management of GBS through Ayurveda anti-*Vata* and anti-*Ama* measures kept in mind while prescribing the drug.

KEYWORDS: GBS, Autoimmune reaction, Nervous system, *Ayurvedic* management.

INTRODUCTION

GBS syndrome is the commonest acute polyneuropathy which is presumed to autoimmune in nature.[1] The syndrome is named after the French physician Georges Guillian and Jean Alexandre Barre, who described it in 1916.[2] It is heterogeneous group of immune mediate condition with an incidence of 1-2/100000/year.[3] Possibility of any person acquiring GBS in lifetime is 1: 1000. The term GBS is often considered to be synonymous of AIPD but with increasing recognition over the past few decades of variants the number of disease that fall under the rubric GBS has grown to include axonal variants and more restricted variants, such as miller- fisher syndrome. In India peak incidence is between June-July and September – October. In India it is more common in younger age. Generally there is no sex preponderance and can occur at any age.[4] In Western countries adults are frequently affected then children. [5] GBS occurs when the body's defence system mistakenly attack parts of nervous system. An autoimmune reaction may damage the myelin sheath around nerve. This lead to nerve inflammation results in conduction block, severe form show secondary axonal degeneration and cause muscle weakness or paralysis and other symptoms.[6] The hallmark is an acute paralysis evolving over days or weeks with loss of tendon reflexes.[3] Ascending paralysis weakness at beginning in the feet hands and migrating towards the trunk is most typical symptoms. While some subtypes causes changes in sensation or pain as well dysfunction of the autonomic nervous system. The disease is usually triggered by an infection. It is most common cause of non trauma related paralysis. It may lead to life threatening complication, in particular if the respiratory muscles are affected or if the autonomic nervous system is involved.[2] In *Ayurveda* GBS is not correlated with any diseases. But seeing the pathophysiology of GBS we can say that it is *Vata* dominating disorder along with association of *Pitta* and *Kapha dosha*. As precipitating factor reduced functions of GI biofire play an important role by producing *Ama* like reactive species in genesis of the disease. *Ama* itself have immense role along with it also vitiate the other *Doshas* too. Therefore at the time of management of GBS through *Ayurveda* anti

Vata and anti *Ama* measures kept mind while prescribing the drugs.[7]

Etiology[8]

Many infections can trigger GBS. Commonly implicated infections are Campylobacter jejuni infection, cytomegalovirus, Epstein-Barr virus, and human immunodeficiency virus (HIV). Other triggering events are immunization (influenza, meningococcal, etc), Systemic lupus erythemetosus, Hodgkin disease, Mycoplasma pneumonia, surgery, trauma and bone marrow transplantation.

Pathogenesis

Guillain-Barre syndrome results from an immune response to a preceding infection that cross-reacts with peripheral nerve components because of molecular mimicry. The immune response can be directed towards the myelin or the axon of peripheral nerve, resulting in demyelinating and axonal forms of GBS. Nerve damage is due to activated T cells and circulating antibodies such as antimyelin antibodies.[8] GBS damages parts of nerves that cause tingling, muscle weakness and paralysis. Damage to other parts of the nerve, can cause, the nerves to stop working altogether.[9]

Pathophysiology[10]

In the demyelinating forms of GBS, the basis for flaccid paralysis and sensory disturbance is conduction block. This finding, demonstrable electro physiologically, implies that the axonal connection remain intact. Hence, recovery can take place rapidly as remyelination occurs. In severe cases of demyelinating GBS, secondary axonal degeneration usually occurs; its extent can be estimated electro physiologically. More secondary axonal degeneration correlates with a slower rate of recovery and a greater degree of residual disability. When a primary axonal pattern is encountered electro physiologically, the implication is that axons have degenerated and become disconnected from their targets, specifically the neuromuscular junction, and must therefore regenerate for recovery to take place. In motor axonal cases in which recovery is rapid, the lesion is thought to be localized to preterminal motor branches, allowing regeneration and reinnervation to take place quickly.

Subtypes of Guillain- Barre syndrome[11]

Acute inflammatory demyelinating polyneuropathy (AIDP): It is the most common form of GBS, and the term is often used synonymously with GBS. It is caused by an autoimmune response directed against Schwann cell membranes.

Miller fisher syndrome: Demyelinating type. Accounting for about 5% of GBS cases, it manifests as a descending paralysis, proceeding in the reverse order of the more common form of GBS. It usually affects the eye muscles first and presents with the triad of opthalmoplegia, ataxia, and areflexia. The ataxia predominantly affects the gait and trunk with the limbs relatively spared. Anti-GQ1b antibodies are present in 90% of cases.

Acute motor axonal neuropathy (AMAN): It also known as Chinese paralytic syndrome. It is probably due to an auto- immune response directed against the axoplasm of peripheral nerves. The disease may be seasonal and recovery can be rapid. Anti –GD1 A antibodies are present.

Acute motor sensory axonal neuropathy AMSAN): It is similar to AMAN, but also affects sensory nerves with severe axonal damage. Like AMAN, It is probably due to an autoimmune response directed against the axoplasm of peripheral nerves. Recovery is slow and often incomplete.

Acute panautonomic neuropathy: it is the rarest variants of GBS, sometimes accompanied by encephalopathy. It is associated with a high mortality rate, owing to cardiovascular involvement, and associated with dysrrhythmias. Frequently occurring symptoms include impaired sweating, lack of tear formation, photophobia, Dryness of nasal & oral mucosa, itching & peeling of skin, nausea, dysphagia, and constipation unrelieved by laxatives or alternating with diarrhoea.

**Bickerstaff`s brainstem encephalitis (BBE):** It is further variant of GB syndrome, which is characterized by acute onset of opthalmoplegia, Atexia, disturbance in consciousness, hyperreflexia or babinsbki`s sign. The course of the disease can be monophasic or remitting-relapsing.

Clinical features

The cardinal features of GBS are progressive symmetric muscle weakness and absent or depressed deep tendon reflexes.[8] GBS manifests as rapidly evolving areflexic motor paralysis with or without sensory disturbance. The usual pattern is an ascending paralysis that may be first noticed as rubbery legs. Weakness typically evolves over hours to a few days and is frequently accompanied by tingling dysesthesias in the extremities[5]. Weakness can vary from mild weakness of legs to complete paralysis of all extremity, facial, respiratory, and bulbar muscles. Weakness usually starts in the lower limbs, and then ascends up to involve trunk and upper limbs (ascending paralysis). However, in some Patients weakness can begin in the arm or facial muscles and then descend down to involve trunk and lower limbs (descending paralysis).[8] The legs are usually more affected than the arms, and xs is present in 50% of affected individuals. The lower cranial nerves are also frequently involved, causing bulbar

weakness and difficulty with handling secretions and maintaining an airway.[5] Because the facial and swallowing muscle become weak, a few people need to be fed intravenously or through a tube placed directory through the abdominal wall into the stomach.[12] Severe respiratory muscle weakness may lead to respiratory failure and requires ventilator support.[8] Most patients require hospitalization, and almost 30% require ventilator assistance at some time during the illness. Fever and constitutional symptoms are absent at the onset, and, if present, cast doubt on the diagnosis. Deep tendon reflexes usually disappear within the first few days of onset. Cutaneous sensory deficits, e.g., loss of pain and temperature sensation, are usually relatively mild, but functions sub served by large sensory fibers such as deep tendon reflexes and proprioception, are more severely affected. Bladder dysfunction may occur in severe cases but usually transient. If bladder dysfunction is a prominent feature and comes early in the course, possibilities other than GBS should be considered, particularly spinal cord disease. In severe cases of GBS requiring critical care management, autonomic involvement is common. Usual features are loss of vasomotor control with wide fluctuation in blood pressure, postural hypotension, and cardiac dysrhythmias.

Pain is another common feature of GBS; several types are encountered. Most common is deep aching pain in weakened muscles, which patients liken to having over exercised the previous day. Other pains in GBS include back pain involving the entire spine and sometimes dysthetic pain in the extremities as a manifestation of sensory nerve fiber involvement. These pains are self limited and should be treated with standard analgesics. GBS usually progress over a period of about two weeks and recovery starts after about a month.[5]

Course: Muscle weakness gradually progresses over 1-3 weeks and then plateaus over next several days to weeks before gradual recovery. On recovery, nearly 15% patients do not have any deficit while minor deficits and major deficits occur in nearly 70% and 5-10% cases, respectively.[1]

On examination:[13]

Onset: Acute or subacute with fever, backache and pain in the limbs.

Sensory: Pain and paraesthesia over the affected limbs with or without sensory loss. Muscle tenderness may be present.

Motor: weakness of all the four limbs simultaneously or first in the lower limbs and then spreading to the upper limbs. Proximal weakness more than distal.

Cranial nerves: Unilateral or bilateral facial palsy, dysphagia (from pharyngeal palsy) and external ophthalmoplegia.

CSF: Xanthochromia with cytoalbuminic dissociation.

Investigation

GBS is difficult to diagnose at first. This is because the symptoms are very similar to those of other neurological disorders or conduction that affects the nervous system such as botulism, Heavy metal poisoning, or meningitis. A careful medical history along with the following tests is useful to confirm the diagnosis.

Nerve conduction studies (NCS) and electromyography (EMG) are used to confirm the diagnosis and also to know the type of GBS. Abnormalities in NCS that are consistent with demyelination are delayed distal latencies, slowed nerve conduction velocities, conduction block, etc. In case of axonal damage, needle EMG will show decreased recruitment and rapid firing motor units in weak muscles.

CSF analysis: CSF may be normal in the first 10 days. Protein is elevated with normal WBC count. This is known as albuminocytologic dissociation, and is present in most patients one week after the onset of symptoms. However, cell count may be increased in patients with HIV infection.

Antibodies: Against nerve component can be detected in the blood of GBS patients. However, antibody testing is not routinely used. Antibodies to the ganglioside GM1 are found in about 25%, usually the motor axonal form.[8]

Reflexes, especially the knee jerk are usually diminished.[12]

Other causes of an acute neuromuscular paralysis should be excluded (e.g. poliomyelitis, botulism, diphtheria, spinal cord syndrome or myasthenia), via the history and examination rather than investigation.[3]

Treatment

Plasmaphresis remove the circulating antibodies and help in fast recovery. 4 sitting of plasmaphresis are recommended.

Intravenous immune globulin (IVIG) probably acts by neutralizing circulating antibodies and immunomodulation. IVIG is given in a dose of 0.4 g/kg daily for 6 days.

Both plasmapheresis and IV immunoglobins have equal efficacy and combining both of them is not better than anyone given alone.

Steroids: IV methyl prednesolone (1 gm IV infusion daily for 5 days) used to be popular earlier, but studies have shown that it does not provide any benefit in GBS.

Supportive therapy: bowel and bladder care, adequate nutrition, monitoring for respiratory failure and providing ventilator support if required, cardiac monitoring, and physiotherapy are all important.[14]

Physical therapy helps to keep joints and muscles healthy.

Blood thinners may be used to prevent blood clots.

Proper body positioning or a feeding tube may be used to prevent choking during feeding if the muscles used for swallowing are weak.[7]

In *Ayurveda* aspect

Guillain Barre Syndrome commonly occurs post viral or any other infections. Seeing the pathophysiology of GBS we can say that it is *Vata* dominating disorder with association of *Pitta* and *Kapha dosha*. Due to *Kapha Avaranum,* Vitiated *Vata* and *Pitta* accumulated in the *Majja Dhatu* and cause *Dhatu Kshaya* and *Oja Kshya*. Here is *Mandagni* play an important role as a precipitating factor.[15]

Line of treatment:[16]

Principle applicable as for *Jvara Chikitsa*

Avarana treatment and *Dhatu Paka* are needed.

Sannipata Jvara Chikitsa, Kapha Jvara Chikitsa

***Ayurvedic* management**[17]

Medicines to treat nerve damage: Drugs like *Yograj Guggulu, Kaisore Guggulu, Trayodasanga Guggulu, Panchtiktagrita Guggulu, Mahavatavidhvansani Rasa, Vata Gajankusha Rasa, Lashunadi Vati, Chitrakadi Vati, Hinguvadi Vati, Kupiluhinguvadi Vati, Kapikacchu Churna, Ashvagandha Churna* and *Dashamularista* are usually treat nerve damage.

Herbal medicine: *Yastimadhu, Manjistha, Mandukaparni, Nirgundi,* and *Dashamula* are also very useful in this condition.

Medicines which act on the *Majja dhatu* of the body are also very effective in treating this condition such as *Punarnava mandura, Tapyadi lauha, Guduchi, Amalaki* and *Mustaka.*

In addition localized *Panchakarma* therapy can also be used for the management of GBS. In acute phase, lasting for the first 3 to 6 weeks, only medicated steam fomentation is advised. After this period, medicated oils are used to massage the body, followed by medicated steam fomentation. Medicines used for these procedures are *Mahanarayana* oil, *Mahamasa* oil, *Mahasaindhava* oil, *Dashamula Kvath* and *Nirgundi Kvath*.

Ashvagandha, Yastimadhu, Tulasi, and *Bhringaraja* are used to correct immune dysfunction in the body. Assurance therapy is also advised to counteract the mental stressors.

Yogasana: *Pranayama* and *Suryanamaskara* are useful.

CONCLUSION

GBS is an acute, frequently severe, and fulminant polyradiculoneuropathy that is autoimmune in nature.[5] An autoimmune reaction may damage the myelin sheath around the nerve. This damage is called demyelination. Damage parts of nerves cause tingling, muscle weakness, and paralysis. It causes nerve signals to move slowly. Damage to other parts of the nerve, can cause, the nerves to stop working altogether. In Ayurvedic aspect GBS occurs due to *Kapha avarana,* vitiated *Vata* and *Pitta* accumulated in the *Majja dhatu* and cause *Dhatu kshya, Oja kshya*. So line of treatment of GBS is *Jvara chikitsa, Avarana treatment,* & *Dhatu paka*. Thus medicines that work on the nerve damage, immune dysfunction, and *Majja dhatu* are useful in the treatment of GBS.

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