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Guillain-Barre Syndrome and *Campylobacter* Species

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Abstract: The polio has been eradicated in most parts of the world, Guillain-Barre syndrome (GBS) has become the most common cause of acute flaccid paralysis. GBS is an autoimmune disorder of the peripheral nervous system characterized by weakness, usually symmetrical, evolving over a period of several days or more. *Campylobacter* species was isolated from stool specimens. *Campylobacter* infection is known as the single most identifiable antecedent infection associated with the development of GBS. *Campylobacter* causes this autoimmune disease through a mechanism called molecular mimicry, whereby *Campylobacter* contains ganglioside-like epitopes in the lipopolysaccharide moiety that elicit auto-antibodies reacting with peripheral nerve targets. *Campylobacte*r is associated with several pathologic forms of GBS, including the demyelinating (acute inflammatory demyelinating polyneuropathy) and axonal (acute motor axonal neuropathy) forms. Different strains of *Campylobacter* as well as host factors likely play an important role in determining who develops GBS as well as the nerve targets for the host immune attack of peripheral nerves.

Keywords: Guillain-Barre, *C. jejuni*, autoimmune, *Campylobacter.*

Introduction

Guillain-Barre Syndrome

Guillain Barre syndrome (GBS) is an acute inflammatory neuropathy, traditionally considered to affect large-diameter of myelinated nerves. It can also be defined as an autoimmune disorder of the peripheral nervous system (PNS) characterized by weakness, usually symmetrical, evolving over a period of several days or more.[1] Affected persons rapidly develop weakness of the limbs, weakness of the respiratory muscles, and areflexia. Patients with GBS have sensory symptoms and signs including neuropathic pain, allodynia and reduced sensitivity to thermal or nociceptive stimuli. The disease is self-limited, followed by partial or complete recovery taking place over weeks to months. Up to 20% of patients may require mechanical ventilation.[2-4] Although most people have an uneventful recovery, some GBS patients are left with severe neurologic deficits. [5-10] Mortality rates of GBS have been reduced to 2 to 3% in the developed countries but remain higher in much of the developing countries. [4,11] The two beneficial treatments are plasmapheresis and intravenous human immunoglobulin (IVIG) administration, that have lowered the patient fatality rate of GBS, [8,12, 13,14] then also the GBS remains a major health problem.

GBS is defined as a single homogeneous clinical entity and it can be divided into several electrophysiological and pathologic patterns.[15-18] As observed by various clinical, electrophysiological, and pathologic techniques, these different patterns suggest that there

are different immune targets of an autoimmune response in the PNS. GBS is an acute inflammatory demyelinating polyneuropathy (AIDP) [5,19,20] characterized by an immune-mediated attack on myelin and varying degrees of lymphocytic infiltration. In severe cases along with the, axonal degeneration accompany the demyelination.

The various diagnostic criteria's for Guillain-Barre Syndrome are:

Features required for diagnosis
- Progressive weakness in both arms and both legs
- Areflexia

Features strongly supporting the diagnosis
- Progression of symptoms over days to four weeks
- Relative symmetry of symptoms
- Mild sensory symptoms or signs
- Cranial nerve involvement, especially bilateral weakness of facial muscles
- Recovery beginning two to four weeks after progression ceases
- Autonomic dysfunction
- Absence of fever at the onset
- Elevated concentration of protein in cerebrospinal fluid, with fewer than 10 cells per cubic millimeter
- Typical electrophysiological features

Features making the diagnosis doubtful
- Sensory level
- Marked persistent asymmetry of symptoms or signs
- Severe and persistent bladder or bowel dysfunction
- More than 50 cells per cubic millimeter in cerebrospinal fluid

Two patterns of predominantly axonal involvement can be distinguished. The first pattern, originally called axonal GBS[21] and more recently termed acute motor-sensory axonal neuropathy (AMSAN) is usually severe, involving both motor and sensory fibers. [15,16,21] The second form is limited to nearly pure motor involvement and is termed as acute motor axonal neuropathy (AMAN).[16, 22] AMAN is more-benign with more-severe cases producing the AMSAN pattern. Miller-Fisher syndrome is a related disorder, characterized by acute onset of unsteadiness of gait (ataxia), areflexia, and an inability to move the eyes, usually associated with nonreactive pupils (ophthalmoplegia) [23].

Demographic Characteristics

GBS is slightly more common in males than in females. [24,25] GBS is slightly more common in whites than in blacks [25]. The incidence of GBS increases with age, but some studies have shown an early peak among 15 to 30 year old [26], suggesting a possible bimodal distribution of cases by age. The AIDP form of GBS appears to affect an older population, whereas the AMAN form tends to affect primarily children and young adults. [22]

Relation between *C. jejuni* infection and GB

A variety of preceding infectious illnesses (mostly viral and upper respiratory) have been described in association with GBS. [27,28,29] Gastrointestinal illnesses occurring in up to 20% of GBS patients was recognized many decades ago. *Campylobacter* infection was first reported as a potential cause of GBS. *C. jejuni*-associated cases of GBS involve axonal injury.[30-33] Humoral immunopathogenic mechanisms are operative because the time course from the onset of enteritis to the onset of neurologic symptoms is 1 to 3 weeks.

Pathogenesis

Peripheral nerves are composed of numerous motor and sensory fibers. The motor fibers originate from motor neurons in the ventral horns of the spinal cord and carry nerve impulses to the muscles. The sensory fibers carry nerve impulses from the specialized sensory receptors in the periphery to the spinal cord. Their cell body resides in the dorsal root ganglia next to the spinal cord. In order to speed the conduction of these nerve impulses, some of these fibers are wrapped by insulating layers of myelin formed by Schwann cells. Between two adjacent myelin sheaths is a gap called the node of Ranvier, where the sodium channels are concentrated. This specialized structure allows nerve impulses to regenerate. The myelin sheaths prevent impulses from leaking away and allow impulses to jump from one node to the next. The nerve impulses can be efficiently conducted at up to 75 m/s by the myelinated nerves.

Access to the PNS by the immune system requires that the blood-nerve barrier be altered. Specialized endothelial cells line the blood vessels inside the endoneurium (the connective tissue enveloping individual nerve fibers within a peripheral nerve). Part of the blood-nerve barrier is due to the presence of negatively charged sialic acid containing glycoconjugates in the lumen that repel negatively charged molecules [34,35]. Tight junctions between endothelial cells contribute to this barrier. Entry of molecules around the nerve is also limited by the perineurium (the connective tissue sheath surrounding a fascicle of nerve fibers in a peripheral nerve). This structure consists of layers of specialized fibroblasts, each layer of which is bounded by a basal lamina with tight junctions between adjacent perineural cells. However, the blood-nerve barrier is not as tight as the blood-brain barrier, so that small amounts of circulating proteins such as albumin, IgG, and exogenously administered horseradish peroxidase (none of which can enter the central nervous system [CNS]) [36] can gain entrance to the endoneurial space [37]. This relative leakiness may render the PNS more

vulnerable than the CNS to antibody-mediated disorders. The blood-nerve barrier is particularly leaky within the dorsal root ganglia and is altogether absent at nerve terminals in the periphery (for example, at the neuromuscular junction), making these areas especially vulnerable to immune-mediated attacks

Mechanisms of Immune Injury to Nerve Fibers in GBS

Acute Inflammatory Demyelinating Polyneuropathy (AIDP):

On examination, patients with AIDP present with flaccid paralysis, areflexia, and usually some sensory loss. Electrophysiological testing typically reveals findings suggestive of demyelination in both motor and sensory nerves.[38,39] Pathologically, macrophage-mediated demyelination and lymphocytic infiltrates are evident.[5,17] AIDP has long been presumed to be a T-cell-mediated disorder based on the lymphocytic inflammation found in many cases and on the analogy to experimental allergic neuritis (EAN). Many markers of T-cell activation can be detected in the serum of AIDP patients, including soluble interleukin-2 receptor and gamma interferon.[40] The importance of antibody-mediated nerve fiber damage in AIDP, including the response to plasmapheresis,[8,41] the presence of antimyelin antibodies as detected in complement activation assays,[42,43] the frequent presence of antiglycoconjugate antibodies, and the demonstration of demyelinating immunoglobulins in sera by either injecting the sera intraneurally [44] or incubating the sera with nerve or Schwann cells *in vitro*. [42,45,46,47]

An antibody directed against antigens on the outermost surface of the Schwann cell (the abaxonal Schwann cell plasmalemma) binds to the complement, which results in sublytic complement activation and the development of transmembrane pores formed by complement. The subsequent entry of calcium might be sufficient to activate calcium-sensitive enzymes, potentially including phospholipase A2 and proteases capable of degrading myelin proteins. Macrophages then participate in removal of damaged myelin. [20] The chief role of T cells may be to open the blood-nerve barrier.

Clinical Features: AIDP usually presents with numbness and tingling in the feet that gradually progresses up the legs and then into the arms. Numbness and paresthesia can also involve the face. Severe, aching, prickly, or burning neuritic pain sensations in the back and limbs are present in at least half of patients. Large fiber modalities (touch, vibration, and position sense) are more severely affected than small fiber functions (pain and temperature perception)

Axonal forms of GBS.

Acute Motor-Sensory Axonal Neuropathy (AMSAN)

GBS is due to primary axonal degeneration without preceding demyelination and the target antigen might lie on the axon.[21] The patients typically have fulminant and widespread paralysis with slow and usually incomplete recovery. [48] It is suggested that the axon rather than the Schwann cell or myelin is the primary target. In some cases where patient died after developing the weakness confirmed that axons may be the primary target of immune attacks in some cases of GBS.[16,17]

Acute Motor Axonal Neuropathy (AMAN)

The another pattern of GBS, is purely motor by clinical and electrodiagnostic findings and is termed as AMAN, has been identified,[16,17,22] and this pattern of GBS can usually be distinguished from other forms of GBS. [39] AMAN is characterized by weakness or paralysis without sensory loss. Electrodiagnostic data suggest that motor fibers can be lost selectively, while sensory nerve fibers are preserved and features of demyelination are absent.[22,39] The AMAN pattern is closely associated with antecedent *Campylobacter* infection. [49]

Pathology of AMAN

In these axonal patterns lymphocytic infiltration is usually absent or scanty.[21,22] The earliest identifiable change is found in the nodes of Ranvier of motor fibers. The nodal gap lengthens at a time when the fibers appear normal. Immunopathologically, this change is due to the binding of IgG and the activation of complement, due to the presence of complement activation marker C3d on the nodes of Ranvier. The macrophages are recruited to the nodes of Ranvier, perhaps as a result of the elaboration of C5a and other complement-derived chemoattractants. These macrophages insert processes into the nodal gap, penetrate the overlying basal lamina of the Schwann cell, and then encircle the node and frequently dissect beneath the myelin sheath attachment sites of the paranode to enter the periaxonal space of the internode. Many fibers express complement activation markers in the periaxonal space, that is, the 11-nm space between the axolemma and the Schwann cell. This periaxonal space is normally extremely regular in its spacing, and it is sealed from both ions and macromolecules of the endoneurial fluid by junctional complexes between the myelin terminal loops and the axolemma. The intrusion of the macrophage probably opens the periaxonal space to endoneurial constituents and allows antibody and complement to enter the internodal region. Immunocytochemical studies have demonstrated that the antigen to which IgG binds is on

the axolemma (as it is in the node of Ranvier). As the macrophages invade the periaxonal space, the axon collapses away from the Schwann cell, resulting in a marked dilatation of the periaxonal space.[50,51] However, most of the axon evidently survives for some time, even though surrounded by macrophages. The end stage of this occurs when motor axons interrupt and degenerate, extending as far up as the ventral root exit zone. [22]

Animal Models of Disease

EAN is a T-cell-mediated disease in Lewis rats and is considered to be the *in vivo* model for GBS. [52] Injection of Lewis rats with proteins or peptides derived from myelin of the PNS induces a primarily T-cell-mediated disease with pathologic features of GBS (demyelination). Numerous chickens infected with *C. jejuni* isolated from an AMAN patient developed paralysis.[53] These preliminary studies suggested that an animal model of AMAN can be developed by using the specific strain of *C. jejuni*.

Diagnostic Considerations

The isolation of *Campylobacter* organisms from patients with GBS greatly depends upon the methods used and upon whether the patient has been given antimicrobial therapy for previous illness.[54] Antimicrobial agents, including the fluoroquinolones and macrolides, commonly used for treating diarrheal disease have excellent activity against *Campylobacter* species. Such agents quickly clear *Campylobacter* organisms from the gastrointestinal tract, making the isolation of the organisms nearly impossible. Other antimicrobial agents used to treat seemingly unrelated illnesses may also affect the recovery of *Campylobacter* species.

For optimal isolation of *Campylobacter* from patients with GBS, multiple stool samples increase the sensitivity.[55] Both direct plating and enrichment methods should be used.

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