Guidance from the literature: Guillain-Barré

Disclaimer: This document is not intended to provide definitive guidance on diagnosis and treatment of patients with Guillain-Barré. It provides clinicians with general information on certain disease processes that may assist in clinical decision making. Specifically, Empi/VitalStim is not aware of any published empirical data on the use of NMES for dysphagia in this patient population and has not requested nor received specific clearance from the US FDA for such labeling. Clinicians are advised to consult the professional literature for information specific to that condition and use best practice guidelines in determining treatment intervention.

Description

Guillain-Barré (GB) syndrome is an autoimmune disorder in which the body's immune system attacks the myelin sheaths of the peripheral nervous system.[1]

Pathophysiology and Presentation

GB is due to an immune response to foreign antigens (such as infectious agents or vaccines) which are mis-targeted to host nerve tissues. The result is an autoimmune attack on the peripheral nerves which leads to inflammation of myelin and conduction block. This results in muscle paralysis that may be accompanied by sensory or autonomic disturbances. Muscle function resolves as the nerves regenerate, but residual weakness may be present.[1]

The disease is characterized by weakness which affects the lower limbs first and rapidly progresses over periods of hours or days. Frequently the lower cranial nerves may be affected and lead to bulbar weakness resulting in dysphagia and/or respiratory difficulties. Although ascending paralysis is most common, other variants also exist. In severe cases, loss of autonomic function is common which results in wide fluctuations in blood pressure, orthostatic hypotension, and cardiac arrhythmias. [1]

Most of the time recovery starts after the 4th week from the onset of the disease. Approximately 70% of patients have a complete recovery within a few months to three years. [1]

Typical dysphagia dysfunction

Patients with GB may have dysphagia characterized by general weakness of the swallowing system which includes decreased hyolaryngeal excursion and weakness in constriction. The muscles which open the upper esophageal sphincter (UES) may not have enough output to pull the UES open.

Management

There is no known cure for GB, but therapies can lessen the severity of the illness and accelerate the recovery. Following the acute phase, the patient can participate in rehabilitation to regain lost functions.

Management of dysphagia:

1. Modification of food and fluid consistency as needed

2. Exercise aimed at strengthening the weak muscle groups when the peripheral nerves to those muscles are recovered

Role of NMES: During the acute stages of the disease when the peripheral nerves are impacted, NMES is incapable of evoking a contraction of the denervated muscle and is not indicated. However, as the peripheral nerves recover, treatment using NMES may be used to improve strength and functioning.

Since different parts of the body will recover at different rates, monitoring to determine when a patient is a candidate for NMES may be indicated. It is reasonable to assume that if the muscle is observed to contract, even though this contraction may be weak, then the nerve has resumed communication with the muscle. At this point in the recovery process, NMES may be used on the muscles that appear to have resumed innervation from the peripheral nerves.

References

1. NINDS Guillain-Barré Syndrome Information Page. National Institute of Neurological Disorders and Stroke. July 30, 2008. www.ninds.nih.gov/disorders/gbs/gbs.htm

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