



Prediction of Outcome in Patients with Guillain Barre Syndrome

Thesis

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قَالَ

سَبِّحْكَ لَا إِلَهَ إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ
الْعَلِيمُ الْعَظِيمُ

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List of Abbreviations

Abb.	Full term
<i>A-CIDP</i>	<i>Acute onset chronic inflammatory demyelinating polyradiculoneuropathy</i>
<i>AIDP</i>	<i>Acute inflammatory demyelinating polyradiculoneuropathy</i>
<i>AMAN</i>	<i>Acute motor axonal neuropathy</i>
<i>AMSAN</i>	<i>Acute motor and sensory axonal neuropathy</i>
<i>APCs</i>	<i>Antigen presenting cells</i>
<i>BUN</i>	<i>Blood urea nitrogen</i>
<i>CIDP</i>	<i>Chronic inflammatory demyelinating polyradiculoneuropathy</i>
<i>C. Jejuni</i>	<i>Cambylobacter jejuni</i>
<i>CMAP</i>	<i>Compound muscle action potential</i>
<i>CMV</i>	<i>Cytomegalovirus</i>
<i>CSF</i>	<i>cerebrospinal fluid</i>
<i>CV</i>	<i>Conduction velocity</i>
<i>DM</i>	<i>Diabetes mellitus</i>
<i>DVT</i>	<i>Deep venous thrombosis</i>
<i>EGRIS</i>	<i>Erasmus GBS Respiratory Insufficiency Score</i>
<i>EMG</i>	<i>Electromyography</i>
<i>H.F- score</i>	<i>Hughes Functional Score</i>
<i>GBS</i>	<i>Guillain Barre Syndrom</i>
<i>GE</i>	<i>Gastroenteritis</i>
<i>HIV</i>	<i>Human immunodeficiency virus</i>

List of Abbreviations *cont...*

Abb.	Full term
<i>ICU</i>	<i>Intensive care unite</i>
<i>IQR</i>	<i>Inter quartile range</i>
<i>IVIG</i>	<i>Intravenous immunoglobulin</i>
<i>LLN</i>	<i>Lower limit of normal</i>
<i>LOS</i>	<i>Lipooligosaccharide</i>
<i>LPS</i>	<i>Lipopolysaccharide</i>
<i>MAC</i>	<i>Membrane attack complex</i>
<i>MFS</i>	<i>Miller Fisher Syndrome</i>
<i>MRC</i>	<i>Mediac Research Council</i>
<i>MRI</i>	<i>Magnetic resonance imaging</i>
<i>NCS</i>	<i>Nerve conduction study</i>
<i>PE</i>	<i>Plasma exchang</i>
<i>RCTs</i>	<i>Randomized controlled trials</i>
<i>RTI</i>	<i>Respiratory tract infection</i>
<i>SIMV</i>	<i>Synchronized intermitten mandatoryl ventilation</i>
<i>TRF</i>	<i>Treatment related fluctuation</i>

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INTRODUCTION

Guillain-Barre syndrome (GBS) is an acute-onset, monophasic immune-mediated disorder of the peripheral nervous system that often follows an infection. The reported incidence rates for GBS are 1 to 2 in 100 000. GBS is equally common in men and women and can occur at any age (*Willison et al., 2005*).

Pathologically, GBS is characterized by demyelination, lymphocytic infiltration and macrophage-mediated clearance of myelin. Two thirds of GBS cases occur weeks after an infection. These infectious agents have epitopes on their surface that are similar to epitopes on the surface of peripheral nerves, resulting in peripheral nerve acting as a “molecular mimic” of the infectious agent. The complement fixing IgG antibodies that arise to attack the infection also bind to peripheral nerve gangliosides, inducing autoimmune injury (*Chiba et al., 1997*).

The diagnosis of GBS should depend on the history and neurological examination. Guillain-Barre syndrome usually begins abruptly with relatively symmetrical onset of paresthesia and sometimes pain. Sensory disturbances are accompanied by progressive weakness. Neuropathic pain is a prominent feature in more than half of patients (*Ruts et al., 2010a*).

Patients showed distal and often proximal, relatively symmetrical, weakness. Sensory examination is usually normal

in the early phase of disease. Areflexia or hyporeflexia is the cardinal feature. About fifty percent of GBS patients develop cranial nerve weakness, usually as facial or pharyngeal weakness. Diaphragmatic weakness due to phrenic nerve involvement is also common (*Hughes et al., 2005*).

Also, autonomic disturbance is seen in more than 50%. The autonomic disturbance usually manifests as tachycardia and systolic hypertension, but more serious disturbances including life-threatening arrhythmias and hypotension may occur (*Burns et al., 2001*).

Nerve conduction study is performed to support the diagnosis of GBS. Nerve conduction study testing may demonstrate features of acquired demyelination; temporal dispersion and prolonged distal and F-wave latencies. Slow motor conduction velocities and conduction block may also occur but less common (*Cleland et al., 2006*).

Commonly variants of GBS include those with severe axonal loss, variants in which one particular fiber type (sensory, motor or autonomic) is predominantly affected and variants categorized by distribution of involvement such as Miller Fisher Syndrome. There are also differences in abruptness of onset and time to complete course of the disease (*Susuki et al., 2009*).

As GBS is an autoimmune disease is active, the goal of immunotherapy is to limit the damage to the nerves and myelin, therapy enhancing the ability of the peripheral nerves to survive and regenerate. Plasma exchange (PE) and intravenous immunoglobulin (IVIg) are effective immunotherapy for adult and pediatric patients with GBS if given during the first few weeks of disease (*Kuitwaard et al., 2009a and Caress et al., 2009*).

The outcome and prognosis of GBS depend on many factors such as the etiology, clinical features, electro physiology and biochemistry (*Leone and Giordana, 2003*). It is believed that extremes of age, rapid disease progression, preceding diarrhea, positive C.jejuni serology and electro physiological evidence of axonal GBS are all factors that are associated with poor prognosis (*Van Koningsveld et al., 2007*).

Consideration of prognosis is important for inpatient management (need for mechanical ventilation), patient and family counseling (*Kuitwaard et al., 2009a*).

AIM OF THE WORK

This study aims to assess the factors (clinical, investigatory tools, and therapies) that may affect the outcome of patients with Guillain Barre Syndrome.