TERUMO BLOOD AND CELL TECHNOLOGIES

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ARTICLE SUMMARY:

Intensive Management and Treatment of Severe Guillain-Barré Syndrome

This document summarizes the following article:

Hund EF, Borel CO, Cornblath DR, Hanley DF, McKhann GM. Intensive management and treatment of severe Guillain-Barre syndrome. *Crit Care Med*. 1993;21(3):443-446. doi: 10.1097/00003246-199303000-00023

The article reviewed approaches to treatment of patients with severe acute Guillain-Barré Syndrome (GBS). The summary focuses on the safety and efficacy of plasmapheresis in the treatment of GBS.

This article summary was developed by Terumo Blood and Cell Technologies.

Guillain-Barré syndrome

- Guillain-Barré syndrome (GBS) is an acutely evolving, immune-mediated, inflammatory disorder of the peripheral nervous system, leading to demyelination and axonal loss.
- Clinical indications of GBS include symmetric flaccid muscle paresis and areflexia in the presence of an increased cerebrospinal fluid (CSF) protein content, and electrophysiological study results demonstrating evolving demyelination.
- Available data indicate that the incidence of GBS is 0.6 to 1.9 cases per 100,000 people in the population per year and that this occurrence rate is roughly the same throughout the world.
- The disease occurs at all ages, with a minor peak frequency in young adults and a second, larger one in the fifth through eighth decades of life. The occurrence rate is slightly higher for men than for women, and slightly higher among whites than in blacks.
- Case-control studies have shown that recent nonspecific respiratory and gastrointestinal infections or recent cytomegalovirus infection are important risk factors for the development of GBS. Two-thirds of patients have preceding infections.

Diagnostic criteria

- Diagnostic criteria for GBS are summarized in **Table 1**.

Table 1. Diagnostic Criteria for GBS

Features required for diagnosis
▪ Progressive motor weakness of more than one limb ▪ Areflexia (loss of tendon jerks)
Features strongly supportive of the diagnosis
Clinical features (ranked in order of importance)
▪ Symptoms of motor weakness develop rapidly but cease to progress by 4 weeks into the illness. ▪ Symmetry is seldom absolute, but if one limb is affected, the other is likely to be affected. ▪ Mild sensory symptoms or signs. ▪ Facial weakness is common and frequently bilateral. Other cranial nerves may be involved, particularly those nerves innervating the tongue, muscles of deglutition, and extraocular motor nerves. ▪ Recovery usually begins 2 to 4 weeks after progression stops. ▪ Sinus tachycardia, other arrhythmias, and labile blood pressure support the diagnosis. ▪ Absence of fever at the onset of neurologic symptoms.
CSF features
▪ After the first week of symptoms, cerebrospinal fluid (CSF) protein is increased or increases on serial examinations. ▪ Counts of ≤ 10 mononuclear leukocytes/mm^3 in the CSF.
Electrodiagnostic features
▪ Approximately 80% of patients have evidence of nerve conduction slowing or blockage at some point during the illness.

Adapted from Asbry et al.[2]

Role of plasmapheresis in the treatment of GBS

- Plasmapheresis presumably removes or dilutes circulating factors implicated in the pathogenesis of the GBS.

- Three clinical studies demonstrated beneficial effects of plasmapheresis in patients with severe GBS at the time when this review was written (1993). All demonstrated that this intervention shortens time to recovery (none were sham-controlled):
  - In two of these studies, plasmapheresis shortened patient time on a respirator.
  - Multivariate analysis of the data provided in one study identified factors associated with a poor outcome. These included:
    - Mean of the summed compound muscle action potential amplitudes from distal stimulation of < 20% of normal
    - Older age
    - Time from onset of disease to randomization of < 7 days
    - Need for ventilatory support
  - There is a risk that plasmapheresis may worsen the disease by inducing a rebound antibody production. This consideration prompted combining plasmapheresis with administration of steroids. However, this combination treatment has failed to provide any benefit and should be abandoned.
  - Plasmapheresis was compared with the administration of intravenous immunoglobulins in one trial and the efficacy of the two treatments was shown to be comparable. However, it was noted that results for the plasma exchange group in this trial were inferior to those reported in other similar studies. Thus, the suggestion that immunoglobulin treatment produced results equivalent to plasmapheresis must be viewed with caution.

References

1. Hund EF, Borel CO, Cornblath DR, Hanley DF, McKhann GM. Intensive management and treatment of severe Guillain-Barré syndrome. *Critical Care Medicine*.1993;21(3):443-446.
2. Asbury AK, Amason BG, Karp HR, et al. Criteria for diagnosis of Guillain-Barré syndrome. *Ann Neurol.* 1978;3:565-566.

Therapeutic apheresis is not indicated as a treatment for Guillain-Barré syndrome in the United States.

TERUMO BLOOD AND CELL TECHNOLOGIES

Terumo BCT, Inc.
10811 West Collins Ave.
Lakewood, Colorado 80215-4440
USA
USA Phone: 1.877.339.4228
Phone: +1.303.231.4357
Fax: +1.303.542.5215

Terumo BCT Europe N.V.
Europe, Middle East and Africa
Ikaroslaan 41
1930 Zaventem
Belgium
Phone: +32.2.715.0590
Fax: +32.2.721.0770

Terumo BCT Asia Pte. Ltd.
89 Science Park Drive
#04-25 (Lobby B)
The Rutherford
Singapore 118261
Phone: +65.6715.3778
Fax: +65.6774.1419

Terumo BCT Latin America S.A
La Pampa 1517–12th Floor
C1428DZE
Buenos Aires
Argentina
Phone: +54.11.5530.5200
Fax: +54.11.5530.5201

Terumo BCT Japan, Inc.
Tokyo Opera City Tower 49F,
3-20-2, Nishi-Shinjuku,
Shinjuku-ku, Tokyo 163-1450,
Japan
Phone: +81.3.6743.7890
Fax: +81.3.6743.9800

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