Chapter 8

A NOVEL IMMUNOMODULATION TECHNIQUE FOR ACUTE MOTOR AXONAL NEUROPATHY TREATMENT: ZIPPER METHOD

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INTRODUCTION

Guillain-Barré syndrome (GBS) has become the most common cause of acute flaccid paralysis. It occurs with a rate of 0.5 to 2 cases per 100,000 persons per year. The disorder is a polyradiculoneuropathy involving mainly motor but sometimes also sensory and autonomic nerves with an acute, usually symmetric, typically ascending, paralyzing clinical pattern. GBS usually follows a preceding infection or rarely follows vaccination. GBS can be classified into two major subtypes, acute inflammatory demyelinating polyneuropathy (AIDP) and acute motor axonal neuropathy (AMAN) depending on whether the myelin or the axonal components of the peripheral nerves are affected (El-Bayoumi et al., 2011).

Accumulated evidence support the likelihood that GBS is an autoimmune disorder, self-antibodies being targeted to attack various components of peripheral nerve myelin, and sometimes the axon. Axonal involvement on EMG studies and this more severe presentation of GBS has a higher incidence of preceding *Campylobacter jejuni* gastroenteritis (Rees et al., 1995). Patients who develop AMAN following *C. jejuni* enteritis have IgG antibodies against GM1 (Yuki et al., 1990). But, patients who have *C. jejuni* enteritis with no neurological involvement do not have similar autoantibodies. Autopsy studies show axolemmal deposition of IgG on the spinal anterior root in AMAN patients (Hafer-Macko et al., 1996), indicating that IgG, which binds effectively with complement, is an important factor in the development of disease.

These findings suggest that GM1 is an autoantigen for IgG antibodies in patients with AMAN related to *C. jejuni* enteritis. IgG anti-GD1a antibodies are

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ma, controls hypercytokinemia, decreases leukocyte degranulation, inhibits macrophage activation and phagocytosis. Also IVIG infusion started at the end of plasma exchange blocks Fc receptors and prevents formation of membrane attack complex by inhibiting complement activation. Since the plasma is cleared from the autoantibodies after the plasma exchange, there is an autoantibody transition from the tissue to the plasma (Kaplan, 1999). Meanwhile, the IVIG given at the same time neutralizes these autoantibodies. Antibody movement from tissue to plasma is complete in 24 hours (Kaplan, 1999). This is the reason why the next plasma change after IVIG treatment is initiated preferably after 24 hours in zipper method.

Our patients were separated from the mechanical ventilator on average 7 days and discharged from the hospital on average 30 days. In previous studies, IVIG applied patients were separated from the mechanical ventilator on average by 26 days, plasma exchange applied patients on average 29 days, and combined treatment patients 18 days. The zipper method is more effective than both treatments and also the combined treatment. In the same studies, patients given IVIG were discharged from the hospital on an average of 53 days, patients who were treated with plasma exchange on average 63 days and patients treated with combined therapy on 51 days. In our study, the patients were discharged from the hospital in 18 days. The most important cause of death in previous studies was hospital infections due to prolonged intensive care hospitalizations. None of our patients developed a hospital infection and no mortality was observed.

Although the cost of using two treatments at the same time seems to increase the cost twice, the cost is decreasing cumulatively because of decreased mortality, decreased intensive care and hospital duration and decreased sequelae rate.

CONCLUSION

The zipper method as a novel treatment modality reduces mortality, speeds up weaning from mechanical ventilation and shortens hospital stay in AMAN patients who require severe intensive care.

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