Treatment of Guillain-Barré Syndrome by Plasma Exchange*

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Summary. Plasma exchange has been used for therapy in eight patients with the Guillain-Barré syndrome. All patients were severely ill. They became tetraplegic and showed cranial nerve involvement. Five patients received assisted respiration, but the others were also at risk of ventilatory insufficiency. Recovery was abrupt in all cases after the first plasma exchanges. Improvement was more marked when plasmapheresis was done on three successive days with plasma exchanges of 2.0–3.0 l each in the initial progressive stage of the disease. A considerable advantage of this therapy is the avoidance of continued artificial respiration and nutrition, which both carry the risk of further complications.

Key words: Plasma exchange – Guillain-Barré syndrome – Artificial respiration – Recovery


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Introduction

The Guillain-Barré syndrome (GBS) is an acute polyradiculoneuropathy of unknown etiology [16]. The occurrence of GBS after injection of rabies vaccine containing nervous tissue has supported the view that the disorder might be mediated through a common immunologic mechanism [25]. Experimental allergic neuritis has shown striking similarity with the disease in humans [5, 9]. The immune pathogenesis of GBS was further supported by the finding of complement fixing antibodies [24], of precipitating antibodies against trypsinized white matter extracts [29] and of myelinotoxic serum antibodies of the IgM class in patients with GBS [10]. Convincing evidence is still lacking that serum antibodies play the primary role in GBS [4, 31]. Cellular hypersensitization to peripheral nervous antigens presented by circulating immunoblasts and lymphocytes supported the role of cellular mechanisms in pathogenesis [2, 6, 21].

The role of treatment with prednisone or ACTH remains controversial [14, 15, 18, 19, 32]. There is also no striking success in therapy by the use of azathioprine [12]. In view of this lack of effective treatment and of the pathogenetic role of myelinotoxic antibodies Brettle et al. [7] tried plasma exchange in one patient with GBS. The good recovery of this patient suggested the use of plasma exchange for treatment of GBS in other patients.

Methods

Plasma exchanges were done using a Haemonetics 30 cell separator. The cells were reinfused into the patient. One third to one half of the plasma was replaced by prewarmed deep frozen fresh plasma, the rest was substituted for 5% human albumin solution with proper electrolyte adjustment. Up to 15 plasma exchanges varying from 0.5 to 3.01 each in varying one day to one week intervals were undertaken. Cardiovascular problems, but also problems in coagulation and allergic reactions made it necessary to interrupt plasma exchange and therefore influenced the amount of exchanged plasma.

Case Reports

Case 1. A 66-year-old male patient with no history of previous illnesses became ill with weakness of both his legs 10 days after a mild afebrile upper respiratory tract infection and four days after influenza vaccination. The weakness progressed rapidly to involve the arms. On admission, on the second day of disease, the patient was already tetraplegic including mild bilateral facial nerve palsy with complete abolition of the tendon reflexes. The pupils and ocular movements were normal. Sensory examination revealed no abnormality. Two days later, the patient had to be tracheotomized and needed assisted ventilation for the next three weeks. After this period spontaneous respiration was regained. At this time the first 1.51 plasma exchange was tried. One day later, the patient was able to swallow. A second 1.51 plasma exchange was performed in the following week. A further slight improvement appeared with return of some finger movements. One week later, the patient started to move both his arms and legs. The patient underwent a total of ten 1.51 plasma exchanges. Four months later, the patient was able to walk with support. Eight months later, there only was slight weakness of the extensors of the left toes.

The CSF on admission contained 36 mg/dl of protein and 1/mm³ lymphocytes. A repeat lumbar puncture revealed 1/mm³ lymphocytes and a total protein of 112 mg/dl two months later.