Major Surgery with Guillain–Barré Syndrome: a Case Report*

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Guillain–Barré syndrome (GBS) is an acute demyelinating polyneuropathy characterized by progressive muscle weakness and areflexia. The pathogenesis of GBS is unknown, but it is generally believed to result from aberrant humoral and cellular immune responses against components of the peripheral nervous system. The overall prognosis of GBS is quite good with approximately 85% of survivors making a good functional recovery. When a diagnosis of GBS has been made, appropriate treatment should be started as early as possible. This may include supportive care in intensive care units, ventilatory assistance, monitoring of blood pressure, fluid status, cardiac rhythm, nutritional supports and medical therapy. Our patient reached maximum deficiency 3 weeks after the onset of GBS. Full recovery took 8 months. The occurrence of GBS after major surgery is rare. We believe that major surgical stress may be the potential triggering factor for the occurrence of GBS in this case report.

KEY WORDS: GUILLAIN–BARRÉ SYNDROME; NEUROPATHY; SURGERY

Introduction

Guillain–Barré syndrome (GBS) is an acute demyelinating disorder of the peripheral nervous system. Cumulative evidence supports the view that the peripheral nerve injury in GBS is immune-mediated, although the precise mechanism is unknown.1 Two-thirds of patients have preceding bacterial or viral infections. Significant antecedent events include Campylobacter jejuni (4 – 66%), cytomegalovirus (5 – 15%), Epstein–Barr virus (2 – 10%) and Mycoplasma pneumoniae (1 – 5%) infections.2 The annual incidence of GBS is fairly uniform, with between one and four cases per 100 000 throughout the world. The disease affects more males than females.2 The clinical picture of GBS is usually a generalized polyradiculo-neuropathy affecting the limbs proximally and distally, commonly spreading to involve the facial and other cranial peripheral nerves. Mortality rates in developed countries have fallen by 50% in recent decades, mainly because of improvements in nursing and critical care measures, as well as the early use of plasma exchange or high-dose intravenous immunoglobulin.3 This syndrome has been associated with a variety of conditions, such as infections, pregnancy, and malignancies including Hodgkin’s and non-Hodgkin’s lymphoma, and carcinomas. It has also been reported to occur following vaccination and during the bone marrow transplantation procedure.4,5 Post-surgical GBS may occur in 1.5 – 2.0% of all cases. In this report we comment on the clinical and neurophysiological features of GBS associated with major surgery, and we

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review the evidence for the pathogenetic role of major surgery.

Case report

A 68-year-old man developed neurological symptoms 4 days following right hemicolectomy for adenocarcinoma. The patient had a febrile attack 48 h before the neurological symptoms occurred. The initial symptoms were fine paraesthesias in the toes and fingertips, followed by lower extremity weakness that ascended over several days to involve the arms, and the muscles of respiration. The following day he developed dysphagia, depressed gag reflex and respiratory weakness. He was intubated endotracheally and mechanically ventilated. Serum urea electrolytes, complete blood count, protein electrophoresis, and chest X-ray were all normal. Cerebrospinal fluid analysis revealed proteins of 76 mg/dl, glucose 3.8 mmol/l and lymphocyte 1/mm³. Nerve conduction studies done on the eighth day of onset of neurological symptoms were suggestive of demyelinating polyneuropathy with conduction blocks. Distal latencies for the right median motor nerve were 4.6 m/s, and 6.9 m/s for the posterior tibial nerves; conduction velocities were 33 m/s and 30 m/s, respectively. Treatment included supportive care in the intensive care unit, ventilatory assistance, monitoring of blood pressure, fluid status and cardiac rhythm. He received five doses of intravenous immunoglobulin (400 mg/kg per day). One week later he was discharged from the intensive care unit, and 14 days post-operation he was discharged from the hospital. He was assessed 1 year later and found to be completely asymptomatic.

Discussion

The pathogenesis of GBS is unknown, but it is generally believed to result from aberrant humoral and cellular immune response directed against components of the peripheral nervous system. This acute, inflammatory, demyelinating polyneuropathy occurs most frequently after infections and immunizations, but it has also been described in patients with malignancies.6

We describe one patient who developed GBS following colorectal cancer surgery. The relationship of GBS to major surgery is unclear. Our patient had a febrile attack before developing GBS, but no aetiologial agent was identified, although an immunological cause could not be ruled out. The immune system is believed to have a role in initiating and perpetuating the ongoing neural damage in GBS. Infectious agents may initiate the immune attack, given that as many as 66% of patients with GBS have a respiratory-tract infection or gastrointestinal illness 1 – 3 weeks before the onset of symptoms.

Guillain–Barré syndrome has been reported rarely in immunocompromised patients. Lisak et al.7 reported three patients with Hodgkin’s disease who developed GBS. In all three, suppression of cellular immunity was demonstrated by lack of lymphocyte proliferative response to standard antigens with in vitro testing. Drachman et al.8 reported GBS in a pharmacologically immunosuppressed patient following renal transplantation. There was substantial depression in lymphocyte-mediated immunity, demonstrated by failure to elicit a delayed hypersensitivity response to intradermally injected antigens.

Baldwin et al.9 observed GBS in two cardiac transplant patients within 3 months of transplantation. Both patients were taking cyclosporine, azathioprine and steroids. Qureshi et al.10 reported two patients with AIDS who developed GBS. El-Sabrout et al.4 reported five GBS cases following solid organ transplantation. The high rate of infections in
patients with a suppressed immune system may increase their likelihood of developing aberrant immunological responses to the infections, resulting in GBS.

Diagnosis is based on physical examination showing loss of motor strength in more than one limb and loss of deep tendon reflexes. Cerebrospinal fluid abnormalities are characterized by albumino-cytological dissociation.11 The typical illness is a predominantly motor disorder with weakness of all four limbs in an ascending pattern. Proximal muscle groups are usually affected before distal ones. Post-surgical GBS has been described rarely. The simplest explanation for the association between major surgery and GBS would be a non-specific mechanism, whereby major surgery triggers an immune reaction targeted to peripheral nerves as part of a systemic response to the surgical stress.

In GBS, 3 – 8% of patients die from complications, such as sepsis, adult respiratory distress syndrome, pulmonary emboli or, in rare cases, unexplained cardiac arrest perhaps related to dysautonomia.12 Only 15% of patients have no residual deficit.13

Optimal management and treatment of GBS is critically important. An important aspect of treatment is to provide optimum supportive care during the acute stages.

In early stages, patients must be observed frequently for the possibility of incipient respiratory failure. Vital capacity should be monitored on a frequent basis, often every 2 h, and assisted respiration must be undertaken if the vital capacity falls to 15 ml/kg body weight, before the patient becomes exhausted or hypoxic. Tracheal intubation is the preferred initial measure, but if mechanical ventilatory assistance is required for more than a few days, a tracheotomy is preferable.

Nutritional issues must be considered early, since nutritional depletion and tissue changes will begin after even a few days of intravenous fluids. Repletion of nutrients should be undertaken, either via a feeding tube, gastrostomy or parenterally, within 5 days for patients who cannot swallow. It is important to perform chest physiotherapy regularly and to pay close attention to pulmonary hygiene. Bronchopneumonia is a common complication of GBS, and every effort should be expended to avoid pulmonary infection.

Deep vein thrombosis and pulmonary embolism are the two common complications seen in bedridden patients. Prophylactic subcutaneous heparin is recommended, along with intermittent positive-pressure leg boots.3

Three large, multicentre, controlled trials have demonstrated clear benefit from plasma exchange when it is used within the first 2 weeks of disease onset.1 Plasma exchange was ineffective when started later than 2 weeks from the onset of symptoms. Plasma exchange is reasonably safe, but not totally free of risks, particularly in haemodynamically unstable GBS patients.1 Intravenous immunoglobulin G therapy is promising in various disorders with a presumed autoimmune basis, and has the advantage of low risk and ease of application. This therapy is therefore introduced as an alternative to plasma exchange.

In summary, the overall prognosis of GBS is quite good, with approximately 85% of survivors making a good functional recovery. Once a diagnosis of GBS has been made, appropriate treatment should be started as early as possible. This may include discontinuation of any suspected medications, initiation of supportive measures, nutritional supports and medical therapy. The occurrence of GBS after major surgery is rare. We believe that major surgical stress may be the potential triggering factor for the occurrence of GBS in this case report.
References

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