Case Report

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Guillain-Barre Syndrome after General Anesthesia for Endoscopic Retrograde Cholangiopancreatography: A Case Report

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Abstract

Guillain-Barre syndrome (GBS) is a rare acute immune-mediated neuropathy. Predisposing factors being respiratory or gastrointestinal infection occurring 1-4 weeks before the symptoms. The knowledge about GBS occurring after surgery or anesthesia is limited by the number of cases reported. Anesthesia, especially the general anesthesia is known to cause delayed recovery, muscular weakness in susceptible individuals. Regional anesthesia leads to sensory and motor block to the area given. The symptoms of GBS, which is muscular weakness including respiratory muscles, tend to confuse the diagnosis after anesthesia. The etiology is not known. Here, we report a case of GBS occurring few hours after the general anesthesia given for the procedure of endoscopic retrograde cholangiopancreatography in the patient with 1 week history of gastrointestinal infection.

Key words: Demyelinating, Endoscopic, General anesthesia, Guillain-Barre syndrome, Respiratory insufficiency

INTRODUCTION

Guillain-Barre syndrome (GBS) is an acute immune-mediated demyelinating polyradiculoneuropathy disorder. If respiratory muscles are affected or the autonomic nervous system is involved, then life-threatening complications can occur. About 5% of the patients die, and more than 5% live with a disabling motor deficit.[1] Epidemiology suggests an annual incidence of 1.55/100,000 with incidence rate being more in patients older than 75 years than in patients lesser than 35 years. Male:female ratio being 1.5:1.[2] There are no incidence studies of GBS in the Indian population, but some case based studies are reported.[3,4] It is associated mostly following respiratory tract infection (40%), gastrointestinal infection (20%).[5] Death is documented in 4-15% of GBS patients[6] and 12-20% of patients with GBS may require ventilator support.[7]

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Surgery, though is a rare cause of GBS, certain mechanical factors such as compression, stretch, or contusion are attributable. There are case reports of GBS occurring on days 4, 7, 9, 11 after surgery.[8-10] Furthermore, there are some case reports of GBS following epidural anesthesia.[11-13] GBS occurring after general anesthesia for hepatobiliary surgery is also reported.[14]

At this moment, reporting a case of GBS occurring in a patient who suffered from acute gastroenteritis (GE) for about a week and manifesting 5 h after the general anesthesia given for endoscopic retrograde cholangiopancreatography (ERCP) procedure.

CASE REPORT

A diabetic, hypertensive female patient aged 50 years came to our hospital referred from some other hospital for the procedure of ERCP. She was in the previous hospital for the complaints of diarrhea and abdominal pain for evaluation for the past 5 days. She was diagnosed to be having acute GE and common bile duct stone. Due to lack of ERCP facility in that hospital, she was referred here. She was on antibiotics and other supportive treatment for acute GE.

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Pre-anesthetic evaluation of the patient was done. She was conscious, oriented, moving all four limbs and comfortable weighing 82 kg. Her blood pressure was 150/80 mm of Hg on tablet telmisartan 40 mg, pulse rate 84/ min, respiratory rate 14/min and oxygen saturation 98% on room air. She was on tablet metformin 500 mg BD. Auscultation of chest revealed no abnormality. Investigations hemoglobin (Hb) 10 g/ dl, total white blood cell count 12,000 cells/cu mm (N84 L12 E03 M01 B00), random blood sugar 136 mg/dl, HbA1C 7, urea 35 mg/dl, creatinine 1.1, electrocardiography, thyroid function tests, and chest X-ray were normal. Liver function test slightly deranged due the pathology. Nil by mouth and medication protocols followed.

18G cannula was in place in the upper limb and fluid on flow. The patient was made to lie in a prone position, head turned toward the gastroenterologist. Head and eyes well protected from pressure points. Mouth gag inserted, oxygen given through nasal cannula. Injection glycopyrrolate 0.2 mg, midazolam 1 mg, fentanyl 50 μg given. Injection propofol was given in titrated doses to induce sleep. The procedure started which lasted for 30 min. Anesthesia maintained with boluses of 10 mg of propofol and one more dose of 25 μg of fentanyl. Total propofol dose given was 90 mg and fentanyl 75 μg. 750 ml of crystalloids given intravenous (IV) during the procedure. The patient turned supine. Blood pressure was 130/80 mm of Hg, pulse rate 80/min, respiratory rate 12/min and oxygen saturation 100% on room air.

The patient shifted to the recovery room where her vitals remained stable. After 3 h of observation in recovery and being stable, she was shifted to the ward. There, after 2 h, she became tachypneic and then had difficulty in breathing. Oxygen saturation went down to 85% and was not responding to oral commands. She was immediately intubated with 7.5 internal diameters endotracheal tube, bilateral air entry confirmed and shifted to ICU with Ambu ventilation with 4 L of oxygen. In the ICU, she was connected to mechanical ventilator (synchronized intermittent mandatory ventilation mode - tidal volume 450 ml, rate 12/min, positive end-expiratory pressure 5, FiO_2 40%). She tolerated the ventilator mode well with minimal sedation responding to oral commands. Blood pressure was 120/80 mm of Hg and pulse rate 76/min. Arterial blood gas sample taken on the ventilator with FiO_2 40% was normal except for slight high PCO_2 of 50 which normalized after 2 h of ventilation. She also complained of inability to move her lower limbs and weakness in both upper limbs.

Neurological examination revealed her to be conscious, responding to oral commands and bilateral symmetrical ascending flaccid quadriparesis. Tone and power of the both lower and upper limbs were diminished. Power in both upper and lower limbs was 2/5. All deep tendon reflexes were absent. Bilateral plantar responses nonreactive. Cranial nerve examination was normal. Pupils were bilaterally symmetrical and reacted to light. Sensory functions were normal. There was no abnormality in spine and cranium.

Routine blood investigations showed mild infection. Hb – 10 g%, total leukocyte count - 12000/cu mm (N84 L12 E03 M01 B00) platelet - 1, 65,000/cu mm. However, Blood, stool and urine culture showed no growth of the organism on day 5 of admission into ICU probably due to the use of antibiotics for acute GE. Serum electrolytes were within normal limits.

Cerebral spinal fluid (CSF) study revealed – protein – 150 mg/dl, mononuclear cells - 5/cu mm, glucose - 75 mg/dl with plasma glucose - 95 mg/dl. CSF study showed increased number of proteins with few mononuclear cells (albuminocytological disassociation). Hence, a diagnosis of GBS was done. Nerve conduction studies not done due to non-availability.

Immediately, treatment with IV immunoglobulin was initiated and given as a total dose of 400 mg/kg body weight/day for 5 days. Simultaneously respiratory therapy, physiotherapy, nutritional care, postural support, deep vein thrombosis prophylaxis were given. Measures to prevent bed sores undertaken.

Gradually with treatment, power and tone of both upper limb and lower limb improved. Weaning trials from the ventilator carried out, and she was out of it after 2 weeks of support. The patient shifted to the ward from ICU and then discharged.

After 30 days, she came for follow-up while her recovery was satisfactory and was advised to escalate her exercising capabilities.

DISCUSSION

GBS is the one of the common causes of acute polyradiculoneuropathy in adults. It has slight predominance in males and in older individuals. The weakness starts in the lower extremities, and over the course of hours or days, it ascends to the arm, the respiratory and the facial muscles. Most of the patients have a history of upper respiratory tract or gastrointestinal system infections in the 1-4 weeks before the symptoms.[15] GBS patients have preceding bacterial enteritis caused by *Campylobacter jejuni*.[16] Our patient was suffering from acute GE for 1 week being treated with antibiotics. Stool cultures done in the 2nd week were, however, negative for any organisms.

The occurrence of GBS after surgical operations and anesthesia are increasingly debated in recent times.[8-14] Our patient who was otherwise normal except for the history of gastrointestinal infection manifested symptoms of GBS 5 h after the general anesthesia given to her for the procedure of ERCP.

Anesthetic agents are often known to cause prolonged muscle weakness, but here general anesthesia was given in the forms of propofol, fentanyl, and midazolam and none of these are reported for such weakness. Muscle relaxants were not at all used.

Electrolyte imbalance to cause muscle weakness cannot be considered as the patient's higher functions and serum electrolytes level were normal. History of prolonged use of steroid was negative, which may cause the same clinical picture.[17]

One of the variants of GBS is AIDP (acute inflammatory demyelinating Polyradiculopathy) involving the cranial nerves especially 3rd and 7th leading to ptosis, facial nerve palsy and pupillary dysfunction. The patient did not have any cranial nerve involvement as per the examination.

The pathophysiology of post-operative GBS is not clear yet. The stress associated with the surgery or anesthesia triggers the immune response leading to inflammatory macrophage infiltration into the nerve.[18] Without clear evidence yet, it is hypothesized that inflammation is induced by ischemia, general trauma after surgery which causes humoral and cytokines response by the immune system.[19] The calculated Relative risk of GBS within 6 weeks of surgery is 13.1 times greater than the incidence in study population.[20]

In our patient, gastrointestinal infection receiving treatment added with surgery and anesthesia had aggravated and hastened the onset of GBS symptoms. Fortunately, our patient was saved with timely diagnosis and treatment.

CONCLUSION

The symptom of GBS, which is muscular weakness starting from lower limbs, ascending to upper limbs and respiratory muscles leading to respiratory insufficiency, confuse the scenario, especially after anesthesia. Timely diagnosis and intervention is important in improving the patient's condition. Although the incidence of GBS is rare, it has to be kept in mind in the presence of relevant predisposing factors and symptoms.

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