Emergent exploratory laparotomy for a patient with recent Guillain-Barré recurrence: A case report

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A case study is presented of a 20-year-old male with a recent exacerbation of Guillain-Barré syndrome who had an emergent exploratory laparotomy under general endotracheal anesthesia. His preoperative history and physical examination indicated complicating factors, including blunt abdominal trauma, ethanol ingestion, and a full stomach. The management of patients with Guillain-Barré syndrome and their special needs are discussed.

Key words: Emergent, Guillain-Barré syndrome, succinylcholine.

Introduction
Guillain-Barré syndrome (polyradiculoneuritis) is characterized by the acute or subacute onset of skeletal muscle weakness or progressive paralysis. Symptoms are generally first seen in the lower extremities, with subsequent progression toward the head. The two most serious symptoms of this syndrome are difficult swallowing and impaired ventilation due to intercostal muscle paralysis and bulbar involvement. Thirty percent of those with this syndrome will require ventilatory support. Guillain-Barré syndrome (GBS) is most common in young adults, and it may occur after viral illnesses and immunizations, as well as without an identifiable cause. Cytomegalovirus, para-influenza 2, herpes simplex, measles, and infectious mononucleosis have also been shown to precede GBS. Eighty-five percent of patients with GBS obtain a good to full recovery, while only 3-5% experience recurrent or chronic neuropathy.

Autonomic nervous system dysfunction may occur in patients with GBS. Such dysfunction can produce cardiovascular symptoms such as labile blood pressure, dysrhythmias, and even cardiac arrest. Physical stimulation, such as intubation or surgical incision, may precipitate hypertension, tachycardia, and dysrhythmias.

When anesthetizing such patients, the anesthesia provider must take into account this altered functioning of the autonomic nervous system. Because compensatory cardiovascular responses may be absent, the patient may manifest severe hypotension in response to perioperative events, such as blood loss, positive pressure ventilation, spinal anesthesia, or changes in position. Therefore, invasive intra-arterial blood pressure monitoring is indicated. Because adrenergic responses to sensory stimuli are unpredictable, it is prudent to have a complement of both alpha and beta agonists and antagonists (e.g., phenylephrine, epinephrine, ephedrine, esmolol, and labetalol) available.

It is unwise to administer succinylcholine to a patient with GBS. The possibility of transient, severe hyperkalemia in the presence of lower motor neuron disease is ever-present.

Cardiac arrest has been reported after succinylcholine administration in a pregnant patient, a 32-year-old woman who was seen for repeat emergency cesarean section and who had recovered from GBS. Her GBS had been diagnosed 8 weeks prior to admission, and her symptoms had resolved 3 weeks before surgery.
Within minutes after receiving succinylcholine, the patient experienced cardiac arrest and was successfully resuscitated. Her initial serum potassium following cardiac arrest was 9.2 mEq/L (normal, 3.5-4.6 mEq/L). Succinylcholine has also been reported to cause asystole and subsequent rhabdomyolytic renal failure. Therefore, the use of nondepolarizing muscle relaxants with minimal cardiovascular side effects is suggested.

Although they are not always ventilator-dependent preoperatively, patients with GBS may require prolonged ventilatory support postoperatively. Such support would be continued until the vital capacity is normalized, with the recognition that respiratory failure is the leading cause of death in patients with neuromuscular disease.

Case report

A 20-year-old, 75-kg, 167-cm male, ASA physical status II E, came to the surgical suite for an emergent repair of a ruptured urinary bladder after blunt abdominal trauma. Interestingly, the patient had a history of four GBS exacerbations. There was also a history of allergy to cephalaxin, which had caused a rash.

The patient had been anesthetized twice before, with the first time being uneventful. The second anesthetic, at age 13, resulted in an exacerbation of GBS. The patient’s initial onset of symptoms of GBS occurred at age 9, at which time his symptoms progressed to ventilatory paralysis, necessitating a tracheostomy and mechanical ventilation. The patient experienced less severe exacerbations of GBS at ages 11 and 13, during which ventilatory support was not required.

The most recent exacerbation, which occurred 5 months before this anesthetic, did not require endotracheal intubation. This episode was treated with plasmapheresis and prednisone early in its course. Plasmapheresis has been shown to blunt the severity of symptoms and shorten recovery time. The patient was taking no medications at the time of surgery.

In the emergency department, he was found to have a ruptured urinary bladder that required surgical intervention. Laboratory data were normal except for urinalysis, which demonstrated turbid, bloody urine with a specific gravity of 1.020, a pH of 7.0, an albumin of 1,200 mg/dL, and a sugar of 1+.

Soon after arrival, the patient was seen in the emergency department. He was awake and oriented. A tracheostomy scar was noted on his anterior neck, and decreased muscle mass was observed in his lower extremities. Capillary refill in all his extremities was less than 2 seconds. There had been a history of alcohol ingestion 2 hours before admission.

After a thorough evaluation, an anesthesia care plan was formulated and discussed with the patient and his parents. Once consent was obtained, the patient was intravenously given cimetidine 300 mg, metoclopramide 10 gm, glycopyrrolate 0.2 mg, and hydrocortisone 100 mg. Four units of blood were typed and crossmatched as a precautionary measure.

Approximately 1 hour following administration of the premedications, the patient was transferred to the anesthesia holding area, where a continuous left radial intra-arterial catheter was placed.

The patient was then transferred to the surgery suite and positioned on an operating table that was equipped with a heating blanket. Initial monitors were applied, and preinduction vital signs were obtained.

Preinduction vital signs included arterial blood pressure (ABP) 130/66 mmHg, heart rate 74 beats per minute (BPM), respiratory rate 12 per minute, and SatO2 100%. The patient was preoxygenated with 100% oxygen and pretreated with vecuronium 1 mg. He was sedated with midazolam 2 mg and fentanyl 100 µg. A modified rapid sequence induction with cricoid pressure was completed after administration of lidocaine 100 mg, propofol 150 mg, and vecuronium 7 mg.

The patient was smoothly intubated, a nasogastric tube was inserted, and the stomach was suctioned without return. An esophageal stethoscope was placed without difficulty. Isoflurane 1% and nitrous oxide 50% were administered, and the patient was given additional fentanyl 150 µg. Immediate postlaryngoscopy vital signs were arterial blood pressure (APB) 135/62 mmHg and heart rate 75 BPM. Ventilator settings were Vt 750, respiratory rate 10, peak inspiratory pressure 14 cm H2O, and ETCO2 33. The electrocardiographic monitor demonstrated sinus rhythm.

Surgery began 15 minutes after induction. Postincision vital signs were ABP 108/55 mmHg, heart rate 62 BPM, and temperature 36.6°C. The intraoperative course was uneventful. During the 100-minute procedure, the range of the patient’s vital signs was APB 108/55 mmHg to 146/88 mmHg, heart rate 55 to 100 BPM, and temperature 36.3°C to 37.1°C. The patient required an additional dose of vecuronium 2 mg after 40 minutes of operative time.

When subcutaneous skin closure began, the patient was given neostigmine 2.5 mg, edrophonium 10 mg, and glycopyrrolate 0.4 mg. Isoflur-
ane and nitrous oxide were then discontinued, and the patient was removed from the ventilator and hand ventilated. Four minutes later, the patient resumed spontaneous breathing. The peripheral nerve stimulator demonstrated sustained tetanus and four equal twitches during train-of-four. Five minutes after discontinuation of anesthesia, the patient was able to sustain a head lift, his hand grips were strong, and his tidal volume was 600 cc. The oropharynx was suctioned, and the patient was exubated with positive pressure.

The patient was transferred to the postanesthesia care unit (PACU) where he received oxygen by nasal cannula. Upon admission to the PACU, the patient was communicative, his respirations were regular and nonlabored, and his skin was pink and warm. His admission vital signs were ABP 140/62 mmHg, heart rate 90 BPM, and respiratory rate 12 per minute. After 1 hour, the patient was transferred to the surgical intensive care unit for observation. His total intraoperative fluid intake was 1,000 mL of lactated Ringer’s solution, 1,500 mL of 0.9% saline solution, and 1,000 mL of hetastarch. His blood loss was estimated to be 300 mL.

**Discussion**

Patients presenting for emergent surgery following acute injury often have special problems, such as hypovolemia, airway complications, intoxication, a full stomach, and there is often little time for evaluation before surgery begins.

In this case, the patient had a full stomach, had ingested alcohol, and had been assessed as being hypovolemic in the prehospital phase. Fortunately, his condition allowed time for adequate examination and preparation for surgery.

Manchikanti has suggested that anticholinergics may reduce the severity of aspiration pneumonitis; therefore, in this case, glycopyrrolate was administered. Glycopyrrolate has been shown to be capable of decreasing the volume and acidity of gastric contents. However, Stoelting stated that the use of low-dose glycopyrrolate (3 μg/kg) did not alter the gastric volume or acidity.

Cimetidine also inhibits the secretion of gastric acid, but it has no effect on the acid that is already sequestered in the stomach. Cimetidine was given preoperatively to help diminish further secretion of gastric acid. Metoclopramide causes opening of the pylorus, stimulates gastric motility, and facilitates emptying of the stomach into the duodenum. However, the timing of the dose is important, so it is suggested that at least 30 minutes are required for metoclopramide to exert its full effect.

The patient’s preoperative history indicated that he had taken a course of prednisone only 4 months prior to the time of injury. Patients who have a history of current or previous administration of corticosteroids may be at risk for cardiovascular collapse, due to inhibition of endogenous cortisol production during surgical stresses. Therefore, the patient received hydrocortisone 100 mg immediately before induction.

Placement of an intra-arterial catheter in a young, healthy patient who is not hemodynamically unstable may be arguable. The published data regarding GBS that was reviewed for this case indicates that these patients are most susceptible to hemodynamic instability during the period when symptoms are evident. However, because this patient reported a recent relapse and also was seen with evidence of muscle wasting, it was decided that aggressive intraoperative monitoring and corticosteroid administration was the more prudent course of treatment.

Several authors have suggested that it is unwise to administer succinylcholine to a patient with GBS. However, it is important to note that although a patient’s symptoms may have resolved, hyperkalemia following succinylcholine administration can still occur. Until such time that a reliable time interval or diagnostic procedure is introduced to measure the status of the lower motor neurons, the use of succinylcholine in patients with GBS or a history of it is ill-advised. For this reason, vecuronium was used, since mivacurium was not available. Mivacurium may well have been the drug of choice.

The induction, maintenance, and emergence from anesthesia were uncomplicated in this patient. This can be attributed to early assessment in the preoperative phase, adequate monitoring, appropriate anesthesia drugs, and adequate intraoperative hydration. The importance of the cooperation of the anesthesia care team with the surgical, nursing, and medical staff cannot be overstated.

**Anesthesia management recommendations**

The anesthesia provider must consider the pathophysiology of GBS when planning anesthesia for a patient with symptoms of it. It is also essential to remember that patients who have recovered from GBS may also manifest an adverse reaction to anesthesia and surgical insult.

A thorough preoperative assessment is essential. The presence of sweating, hypertension, flattened or inverted T waves on the electrocardiogram, bradycardia, tachycardia, or lack of R-R wave variability may signal the presence of autonomic instability or dysfunction.
The patient’s respiratory function should be evaluated. Difficult swallowing and general or specific muscle weakness may indicate the need for prolonged mechanical ventilation. A history of prolonged ventilation or tracheostomy may signal the presence of fibrotic changes or tracheal narrowing.

If the anesthetic requirement necessitates the use of a muscle relaxant, succinylcholine is contraindicated in patients with a history of paralysis, neuromuscular degeneration, or lower motor neuron lesions. Excessive potassium release and cardiac arrest are possible.

Small changes in intravascular fluid volume can produce profound hypertension or hypotension in a patient with a dysfunctional autonomic nervous system. Therefore, it is essential to ensure optimal fluid volume status with blood or blood volume expanders as the situation dictates.

Sudden changes in body position can also precipitate exaggerated blood pressure fluctuations. Therefore, patients should be moved slowly and their blood pressure checked frequently during any change in position. A full complement of alpha and beta agonists and antagonists should be readily available.

Patients who are to receive spinal anesthesia should be screened carefully. An elderly patient with GBS experienced cardiac arrest after L4-5 spinal anesthesia. This cardiac arrest was attributed to acute changes in position, profound sympathetic blockade far above the L1 sensory blockade, and subsequent orthostatic instability.

The patient’s psychological needs are an important consideration in anesthesia management. Careful explanation of each procedure and reassurance will help the patient overcome the fears and frustrations inherent in a debilitating disease.

Summary

Patients with GBS or a history of it who come to the surgical suite present the anesthesia provider with a wide range of clinical challenges. The anesthetist must have a complete understanding of GBS and its effect on major body systems. The more obvious clinical symptoms—paralysis and weakness—may obscure more subtle sympathetic and parasympathetic nervous system dysfunction. Therefore, careful preoperative evaluation and preparation can help prevent many anesthesia complications.

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