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Case Report

Anesthetic management in a patient with Guillain-Barré syndrome undergoing open reduction and internal fixation for subtrochanteric femur fracture: a case report

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ABSTRACT

Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy associated with peripheral nerve involvement, autonomic dysfunction, and heightened sensitivity to anesthetic agents, making anesthetic technique selection challenging. We report a patient with a prior diagnosis of GBS (axonal variant, acute motor-sensory axonal neuropathy) confirmed on nerve conduction velocity studies, who presented with a subtrochanteric femur fracture requiring open reduction and internal fixation. Pre-operatively the patient had recovered neurologically with no residual paralysis but showed sinus tachycardia, mild anemia, low-grade fever treated with antipyretics, a Mallampati grade III airway, and grade 2 left ventricular diastolic dysfunction. Focused autonomic assessment revealed no active autonomic dysfunction. General anesthesia was precluded by the predicted difficult airway and the risk of succinylcholine-induced hyperkalaemia in denervated muscle, while epidural anesthesia was impractical due to positional limitations from fracture pain. Spinal anesthesia was administered using 0.75% hyperbaric ropivacaine via a 25G Quincke needle. Post-spinally the patient developed hypotension and tachycardia, attributed to multi-factorial hemodynamic compromise including spinal sympathectomy, anemia, febrile illness and diastolic dysfunction, and was managed with colloid and mephentermine. The intra-operative and post-operative course were otherwise uneventful, with no neurological deterioration. Neuraxial anesthesia is feasible in neurologically recovered GBS patients undergoing urgent non-obstetric surgery when general anesthesia is contraindicated. Thorough pre-anesthetic assessment, anticipation of disproportionate hemodynamic instability, and close post-operative monitoring are essential.

Keywords: Guillain-Barré syndrome, Neuraxial anesthesia, Spinal anesthesia, Difficult airway, Succinylcholine, ORIF, Autonomic dysfunction, Hemodynamic instability, Axonal neuropathy

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute inflammatory polyneuropathy characterized by progressive ascending motor weakness, areflexia, and variable sensory and autonomic involvement. Autonomic dysfunction, present in up to 54-66% of hospitalized GBS patients, represents one of the most life-threatening aspects of the condition and can include cardiovascular instability, arrhythmias, labile blood pressure, and bradyarrhythmias.[1-3]

Patients with GBS requiring surgical intervention present a significant anesthetic challenge. General anesthesia carries the dual risk of difficult airway management: a recognized comorbidity in patients who may be debilitated from GBS and the well-documented risk of succinylcholine-induced fatal hyperkalaemia secondary to extra junctional acetylcholine receptor upregulation in denervated muscle.[4,5] Neuraxial techniques carry theoretical risks of exacerbating existing nerve injury or precipitating hemodynamic instability in autonomically

compromised patients, yet neuraxial anesthesia is not an absolute contraindication in neurological disease.[6]

The existing literature on neuraxial anesthesia in GBS consists almost entirely of case reports and series, predominantly in obstetric patients undergoing caesarean section or labour analgesia.[7,8] Evidence for neuraxial techniques in non-obstetric surgical settings, particularly urgent orthopedic surgery is sparse. Cases of GBS triggered after spinal anesthesia for orthopedic procedures have also been reported, further complicating decision-making.[9-11]

We present a case of successful spinal anesthesia in a patient with prior GBS (AMSAN variant, in neurological remission) undergoing ORIF for a subtrochanteric femur fracture, where the interplay of a predicted difficult airway, succinylcholine risk, and positional constraint mandated neuraxial anesthesia as the only viable technique.

METHODS

History and background

A patient presented to our institution with a subtrochanteric femur fracture requiring surgical fixation. Relevant past history included a diagnosis of GBS in 2024, presenting as sudden-onset progressive ascending flaccid quadriparesis without cranial nerve or bulbar involvement. Nerve conduction velocity (NCV) studies at diagnosis confirmed motor-sensory axonal neuropathy (AMSAN variant) affecting bilateral median, ulnar, tibial, and peroneal nerves. The patient was treated with intravenous immunoglobulin (IVIG) and achieved full neurological recovery with no residual paralysis, ambulating without support at presentation. Background history included mild chronic anemia. The patient was not on any regular medications.

In the days prior to surgery, the patient experienced a low-grade febrile episode (temperature 100°F), managed conservatively with antipyretic therapy. The fever resolved prior to surgery, though baseline tachycardia persisted. A reactive leucocytosis (total leukocyte count 14,870/mm³) was noted on pre-operative investigations, consistent with the recent febrile illness and trauma.

Pre-operative assessment

General condition was fair; the patient was conscious and coherent. Vital signs: blood pressure 130/70 mmHg, pulse rate 124 bpm, SpO2 96% on room air, temperature 100°F. Heart rate fluctuated between 102 and 124 bpm across recordings taken at different times pre-operatively, with a resting electrocardiogram showing sinus tachycardia at 102 bpm. Pallor was present. Minimal bilateral pitting edema was noted. Airway assessment: adequate mouth opening, Mallampati grade III (predicted difficult airway), short thyromental distance and adequate sternomental distances, class 3 upper lip bite test, no restricted neck mobility, no loose dentition.[12]

Systemic examination: respiratory: bilateral air entry, no added sounds; cardiovascular: S1S2 heard, no murmurs; abdomen : soft, non-tender; CNS : cranial nerves intact, motor power 4/5 bilateral upper limbs and 4/5 bilateral lower limbs (limited by fracture pain; dorsiflexion intact contralaterally); sensory : no focal deficit; deep tendon reflexes: present; plantar : flexor response.

Autonomic assessment

A focused autonomic evaluation was performed. No recordable heart rate variability abnormality was identified beyond the baseline tachycardia. Formal orthostatic hypotension assessment was not feasible due to the patient's inability to bear weight secondary to the femur fracture; no episodic blood pressure fluctuations were observed at rest. No other symptoms of autonomic dysfunction were present. Cardiovascular autonomic dysfunction, which occurs in up to 30-54% of GBS patients, was therefore considered unlikely to be a primary contributing factor in this patient, with the tachycardia attributed to pain, anemia, and the recent febrile episode.[13,14]

Table 1: Pre-operative investigations.

Investigation	Result
Hemoglobin (Hb)	8.8 g/dL - B-negative; 2-unit PCV arranged
Total leukocyte count (TLC)	14,870 /mm³ (Reactive leucocytosis)
Platelet count	2.98 lakh/cumm
Blood glucose (random)	91.9 mg/dl
Serum creatinine	0.47 mg/dl
Serum sodium (Na+)	138 mEq/l
Serum potassium (K+)	4.26 mEq/l
Serum chloride (Cl-)	103 mEq/l
ECG	Sinus tachycardia, HR 102 bpm
Chest X-ray (CXR)	Within normal limits
Viral markers	Non-reactive
2D echocardiography	LVEF 60% Grade 2 LV Diastolic Dysfunction
ASA physical Status	ASA-PS II

Anesthetic management

The pre-anesthetic risk stratification identified three principal concerns governing technique selection: a predicted difficult airway (Mallampati III) precluding safe general anesthesia; the well-established risk of succinylcholine-induced hyperkalemia in post-GBS denervated muscle, which is independent of clinical recovery status; and the axonal neuropathy variant, warranting avoidance of further pharmacological insult to

peripheral nerves. General anesthesia was therefore considered high-risk and was avoided.[4,5,15]

Among neuraxial options, epidural anesthesia : generally preferred over spinal in GBS due to its titratable onset and lesser hemodynamic perturbation: was not feasible because the patient could not maintain the sustained spinal flexion required for catheter placement, owing to fracture pain and positional restriction.[6,16] Spinal anesthesia in the sitting position was selected as the only safe and practical technique (Figure 1). A similar clinical reasoning, spinal chosen over general anesthesia due to a predicted difficult airway when alternative regional techniques were impractical has been documented in patients with severe hemodynamic instability.[17]

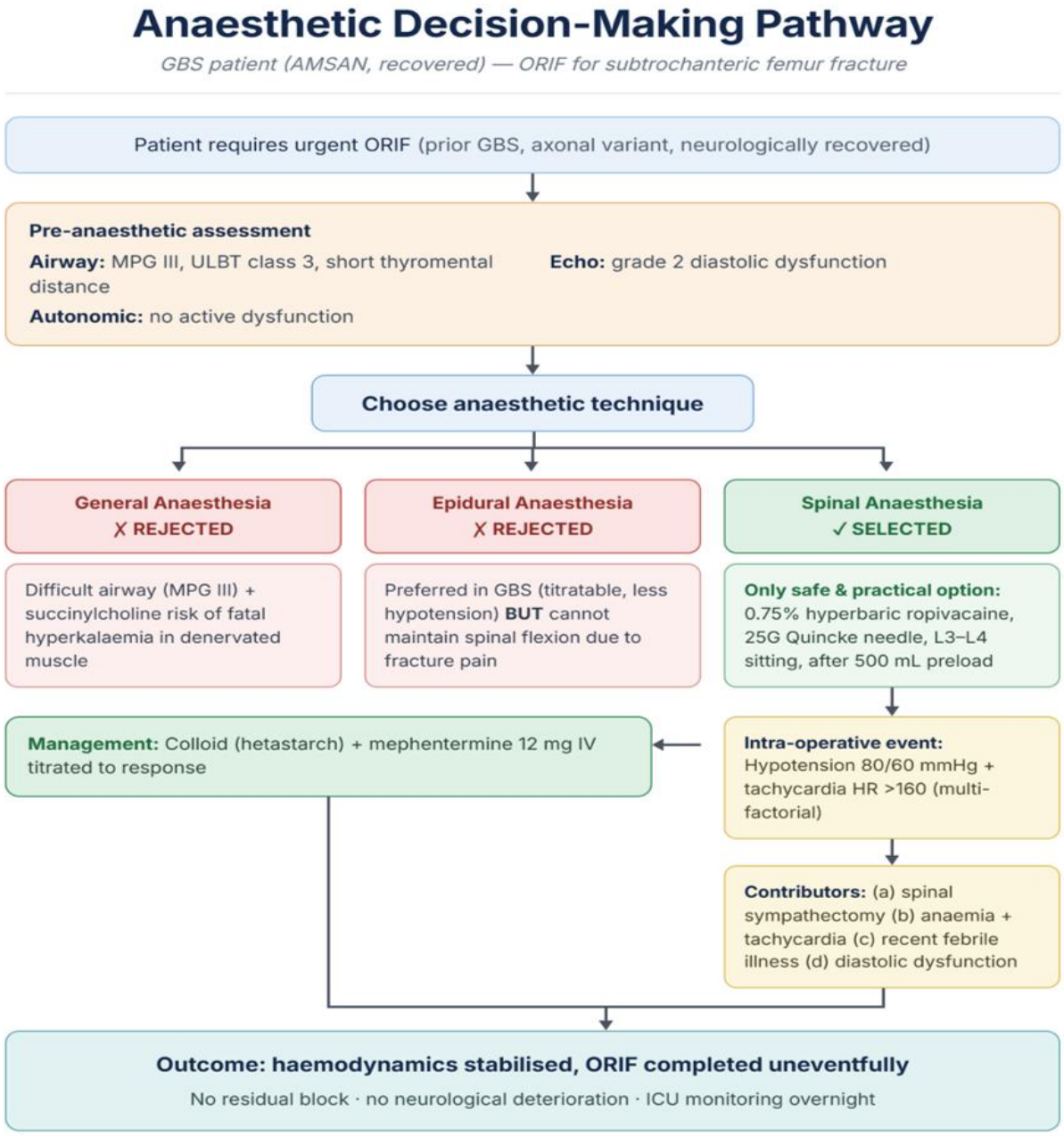

Figure 1: Anaesthetic decision-making pathway for the patient with prior Guillain-Barré syndrome (axonal variant) undergoing ORIF for subtrochanteric femur fracture. General anaesthesia was rejected due to a predicted difficult airway and the risk of succinylcholine-induced hyperkalaemia; epidural anaesthesia was impractical owing to fracture-related positional limitation; spinal anaesthesia was therefore selected. The figure also summarises the multi-factorial intra-operative hypotension and its management.

Informed consent was obtained after detailed discussion of the uncertain but documented risk of neurological deterioration with neuraxial techniques in GBS patients, as well as the risks of alternative techniques.

Standard ASA monitoring was established (non-invasive BP, HR, SpO2, ECG). Wide-bore intravenous access (20G cannula) was secured and the patient was preloaded with 500 ml crystalloid. Spinal anesthesia was administered at the L3-L4 interspace, sitting position, using a 25G Quincke needle. Hyperbaric ropivacaine 0.75% was injected intrathecally after confirming free CSF flow. The 2022 ASA guidelines on difficult airway management were followed with appropriate backup airway equipment prepared.[18]

Intra-operative course

Post-spinal, the patient developed hypotension (BP 80/60 mmHg) and tachycardia (HR >160 bpm). This hemodynamic event is best understood as multi-factorial: sympathetic blockade-induced vasodilation from spinal anesthesia, documented to cause hypotension in approximately 17-35% of patients; pre-existing reduced cardiovascular reserve from anemia (Hb 8.8 g/dl) and baseline tachycardia (HR 124 bpm); relative hypovolemia and vasodilation from the recent febrile illness; and grade 2 left ventricular diastolic dysfunction on pre-operative echocardiography (LVEF 60%), which renders cardiac output preload-dependent and therefore particularly vulnerable to the venodilation and reduced venous return produced by spinal sympathectomy. The combination of these factors likely resulted in a hemodynamic response disproportionate to that expected from spinal sympathectomy alone.[19,20]

Management: rapid colloid infusion (hetastarch) and mephentermine 12 mg intravenously, titrated to response. Hemodynamic stabilized and the remainder of the intra-operative period was uneventful. The ORIF was completed without further complications.

Post-operative course

Post-operatively, the patient was transferred to the Post-Anesthesia Care Unit (PACU) for initial recovery, followed by Intensive Care Unit (ICU) admission for overnight monitoring given the background of GBS. The post-operative period was uneventful. No residual motor or sensory blockade was observed. No new neurological deficits were detected on examination. The patient was discharged from the ICU the following morning and continued recovery on the ward without complications.

DISCUSSION

GBS involves peripheral nerve and often autonomic dysfunction, making any anesthetic technique carry inherent risks. The available data on neuraxial anesthesia in GBS come almost entirely from case reports, predominantly in obstetric patients.[7,8] Evidence in non-obstetric surgical contexts particularly urgent orthopedic procedures are sparse, and this case addresses a clinically important gap.

The axonal variant of GBS (AMSAN), as seen in our patient, involves primary axonal injury to motor and sensory fibers and was confirmed by NCV studies. AMSAN is less commonly reported in the neuraxial anesthesia literature than AIDP. A subacute onset of AMSAN following epidural anesthesia has been previously described, emphasizing the need to include axonal GBS variants in the differential diagnosis when neurological symptoms follow neuraxial techniques. Whether the axonal variant confers different vulnerability to local anesthetic neurotoxicity remains unstudied.[21]

The decision-making in our case was governed by a three-way constraint: first, the difficult airway (Mallampati III) rendered general anesthesia hazardous. Second, succinylcholine, routinely used for rapid sequence intubation in difficult airway scenarios: is contraindicated in GBS due to the risk of life-threatening hyperkalaemia from extra junctional acetylcholine receptor upregulation in denervated muscle.[4,5,6,15,16] This risk persists for months after clinical recovery, as upregulation of nicotinic acetylcholine receptors occurs independently of the patient's functional neurological status. Third, epidural anesthesia the preferred neuraxial technique in GBS for its titratable sympathectomy was impractical due to positional limitations from the fracture.

Postoperative GBS is a recognized but uncommon complication of surgery, with spinal procedures carrying particular risk.[10,22,23] In our patient, however, surgery was performed for a traumatic fracture and could not be deferred. The absence of clinically demonstrable autonomic dysfunction, supported by a focused pre-operative assessment including heart rate variability evaluation and clinical observation for episodic haemodynamic fluctuations at rest, was a critical factor supporting the decision to proceed with spinal anesthesia. Autonomic dysfunction has been documented in 30-54% of hospitalized GBS patients and its presence would have substantially increased perioperative risk.[13,14]

The intra-operative hemodynamic event warrants specific discussion. Spinal anesthesia causes hypotension in approximately 17-35% of patients through sympathetic blockade-mediated vasodilation.[19,20] In our patient, three compounding factors amplified this response: pre-existing anemia (Hb 8.8 g/dL) with compensatory tachycardia (HR 124 bpm), relative hypovolemia and vasodilation from the preceding febrile illness, grade 2 left ventricular diastolic dysfunction with preserved ejection fraction (LVEF 60%) making cardiac output critically preload-dependent, and potentially subclinical residual autonomic impairment not fully detectable by clinical assessment. The presence of diastolic dysfunction is an under-recognised contributor to spinal-induced hypotension and reinforces the value of pre-operative echocardiography in this population. Mephentermine, as an indirect sympathomimetic with both alpha and beta-adrenergic activity, was an appropriate vasopressor choice addressing both heart rate and vascular tone components simultaneously.

The uneventful neurological recovery with no new deficits and no residual blockade is consistent with published reports of successful neuraxial anesthesia in recovered GBS patients.[7,8,17,24] Close post-operative monitoring in the ICU, as recommended by multiple authors, facilitated prompt detection of any early neurological change.

This case makes a unique contribution to the literature in several respects: it describes a non-obstetric orthopedic surgical context; documents the axonal (AMSAN) GBS variant specifically; provides a detailed account of layered clinical constraints that forced deviation from guideline-preferred technique (epidural over spinal); and offers a structured multi-factorial analysis of an intra-operative hemodynamic event that goes beyond simple spinal hypotension.

CONCLUSION

Neuraxial anesthesia is feasible in a neurologically recovered GBS patient (axonal variant) undergoing urgent non-obstetric surgery when general anesthesia is contraindicated by both a predicted difficult airway and succinylcholine risk. When the preferred neuraxial technique (epidural) is itself impractical due to positional constraints, spinal anesthesia with careful preparation remains a viable option. Clinicians must anticipate disproportionate hemodynamic instability, particularly in patients with compounding factors such as anemia, baseline tachycardia, and recent febrile illness. Vasopressor readiness, adequate volume preloading, and ICU-level post-operative monitoring are essential. Multidisciplinary involvement of neurology and experienced anesthesia teams, along with transparent informed consent regarding the uncertain neurological risks, should be the standard of care. Further case reports and prospective registries are needed to guide evidence-based technique selection in GBS patients requiring surgical intervention.

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