



Case Report

Perioperative Management of Idiopathic Sensory Ganglionopathy During Hemiarthroplasty: A Case Report

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ABSTRACT

Autonomic dysfunction is associated with impaired cardiovascular regulation, resulting in labile blood pressure and unpredictable responses to anaesthetic agents. We report a case of a 58-year-old female with idiopathic immune-mediated sensory ganglionopathy undergoing hemiarthroplasty under spinal anaesthesia. Despite stable baseline haemodynamics, the patient developed severe hypotension following neuraxial blockade, requiring repeated vasopressor administration and fluid resuscitation. With timely intervention and vigilant monitoring, haemodynamic stability was achieved and the postoperative course was uneventful. This case highlights the importance of anticipating haemodynamic instability and optimising perioperative care in patients with autonomic dysfunction

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INTRODUCTION

Autonomic dysfunction represents a spectrum of disorders characterised by impaired regulation of involuntary physiological processes, particularly cardiovascular homeostasis. One of the defining clinical features is orthostatic hypotension, typically described as a sustained reduction in systolic blood pressure of ≥ 20 mmHg or diastolic pressure of ≥ 10 mmHg within three minutes of standing.¹ This condition reflects failure of autonomic reflex pathways, especially baroreceptor-mediated responses that normally maintain vascular tone and cardiac output during postural changes.

The pathophysiology of autonomic failure involves degeneration or dysfunction of sympathetic and parasympathetic pathways, resulting in impaired vasoconstriction, reduced heart rate variability and an inability to compensate for haemodynamic stress.² Consequently, patients may exhibit marked blood pressure lability, including episodes of hypotension and supine hypertension.

From an anaesthetic standpoint, these physiological alterations have important implications. Neuraxial anaesthesia can precipitate significant hypotension due to sympathetic blockade, while general anaesthesia may further impair cardiovascular stability and increase the risk of aspiration due to gastrointestinal dysmotility.^{3,4} In addition, denervation hypersensitivity may lead to exaggerated or unpredictable responses to vasoactive drugs, complicating intraoperative management.⁵

Idiopathic immune-mediated sensory ganglionopathy is a rare neurological condition characterised by selective involvement of dorsal root ganglia, leading to sensory ataxia and proprioceptive deficits. In some patients, autonomic fibres may also be affected, resulting in dysautonomia.⁶

Given the rarity of this condition and the paucity of evidence-based guidelines, perioperative management relies largely on an understanding of underlying physiology and careful clinical judgement. This case highlights the anaesthetic challenges and perioperative considerations in a patient with sensory ganglionopathy and suspected autonomic dysfunction undergoing hemiarthroplasty.

CASE REPORT

A 58-year-old female presented with pain in the right hip following a fall at her residence and was diagnosed with an intracapsular fracture of the right hip. She was scheduled for hemiarthroplasty.

She had a history of idiopathic immune-mediated sensory ganglionopathy, hypertension and type 2 diabetes mellitus. She reported progressive tingling sensations in both feet since 2014, followed by gait instability and slippage of footwear since 2016. She also experienced tingling in the right hand and right side of the face. Over time, she developed sensory ataxia, described as swaying while walking, which worsened in darkness and in confined spaces. She also reported a “rubbery” sensation in both feet.

She had no history of addictions, and family history was non-contributory. The patient had previously received intravenous immunoglobulin therapy and was on maintenance treatment with azathioprine.

Clinical Examination

The patient was conscious, alert and oriented. She was moderately built and nourished, with no pallor, icterus, cyanosis, clubbing or oedema.

Vital signs:

- Supine BP: 130/90 mmHg
- Pulse: 78/min
- Standing BP (3 minutes): 130/90 mmHg
- Pulse: 82/min

Neurological examination showed normal cranial nerve function and normal motor strength. Sensory examination revealed reduced pinprick and fine touch sensation in both lower limbs, more pronounced on the left side, with significant loss over plantar surfaces.

Investigations

- Nerve biopsy (left sural nerve): inflammatory demyelinating neuropathy
- Nerve conduction studies and electromyography: sensory axonal neuropathy
- ANA profile: negative
- MRI brain: normal
- Whole spine imaging: diffuse disc bulge with mild cervical canal narrowing

Autonomic Function Tests

Parasympathetic tests:

- Heart rate response to deep breathing: normal
- Valsalva ratio: normal
- 30:15 ratio: normal

Sympathetic tests:

- Blood pressure response to sustained handgrip: normal
- Blood pressure response to standing: no significant fall

Despite normal test results, clinical findings suggested subclinical autonomic dysfunction.

PERIOPERATIVE MANAGEMENT

Preoperative Phase

The patient was kept nil per oral for 6 hours. Intravenous fluid preloading was administered to optimise intravascular volume. Blood glucose levels were maintained within normal limits. Given the possibility of autonomic dysfunction, preparedness for haemodynamic instability was ensured.

Intraoperative Phase

Spinal anaesthesia was administered at the L3–L4 level using 0.5% bupivacaine, achieving a sensory level up to T6. Immediately following the block, the patient developed severe hypotension (60/50 mmHg), likely due to sympathetic blockade in the presence of impaired autonomic compensation.

Management included:

- Mephentermine 6 mg IV bolus
- Repeated boluses (total 18 mg)
- Continuous infusion (12 mg in 300 mL normal saline)
- Intravenous fluid resuscitation

Blood pressure gradually stabilised to 120/80 mmHg and remained stable thereafter. No intraoperative complications were observed.

Postoperative Phase

The patient remained haemodynamically stable with blood pressure around 130/90 mmHg. Analgesia was provided with minimal opioid use. She was mobilised gradually, and recovery was uneventful.

DISCUSSION:

Autonomic dysfunction significantly alters cardiovascular physiology by impairing reflex mechanisms responsible for maintaining haemodynamic stability. The inability to generate an appropriate vasoconstrictor response, combined with reduced heart rate variability, predisposes these patients to hypotension during anaesthesia.²

In the present case, profound hypotension occurred immediately following spinal anaesthesia. This is consistent with the effects of sympathetic blockade, which leads to vasodilation and decreased venous return. In patients with intact autonomic function, compensatory mechanisms such as tachycardia and peripheral vasoconstriction help maintain blood pressure; however, these responses are blunted or absent in autonomic failure.³

Another important factor is denervation hypersensitivity, which results in altered responsiveness to vasoactive drugs.⁵ The requirement for repeated vasopressor administration in this case reflects this variability. Additionally, these patients are highly dependent on intravascular volume, and even minor reductions in preload can lead to significant haemodynamic instability.⁷

The choice of anaesthetic technique in patients with autonomic dysfunction remains controversial. While regional anaesthesia avoids airway manipulation and reduces aspiration risk, it is associated with a higher incidence of hypotension. General anaesthesia, on the other hand, may exacerbate cardiovascular instability and is associated with risks related to delayed gastric emptying.⁴

Postoperative care is equally important, as patients may develop delayed hypotension due to ongoing autonomic dysfunction. Careful monitoring, gradual mobilisation and judicious use of analgesics are essential.

Overall, this case demonstrates that even in the presence of apparently normal baseline haemodynamics, patients with suspected autonomic dysfunction can develop severe intraoperative instability, highlighting the need for careful planning and vigilant monitoring.

ANAESTHETIC CONSIDERATIONS

In patients with autonomic dysfunction, perioperative management should address blood pressure variability, dependence on intravascular volume, altered drug responsiveness, impaired heart rate compensation and sensitivity to positional and ventilatory changes. In the present case, these factors were reflected by severe hypotension following spinal anaesthesia and variable response to vasopressors.

CONCLUSION:

Autonomic dysfunction presents significant perioperative challenges, with a high risk of haemodynamic instability even in patients with apparently normal baseline parameters. This case emphasises the importance of anticipating autonomic involvement, optimising intravascular volume and ensuring vigilant intraoperative monitoring to achieve favourable outcomes.

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