

Persistent Hypotension After Neurolytic Coeliac Plexus Blockade in a Cachectic Cancer Patient: A Case Report

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Case Report

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Abstract

Introduction

Neurolytic coeliac plexus block is an effective intervention for refractory malignancy-related abdominal pain. Persistent hypotension is a rare but serious complication.

Case Presentation

We report a 68-year-old cachectic woman with metastatic gallbladder cancer who developed 12 days of refractory hypotension requiring dopamine infusion after bilateral coeliac plexus alcohol neurolysis. Besides postural dizziness and persistent hypotension, she did not have other symptoms. Laboratory tests were unremarkable. The hypotension resolved promptly with fludrocortisone and proactive nutritional support.

Conclusion

This case reports the rare occurrence of persistent hypotension after neurolytic coeliac plexus blockade. We discuss diagnostic challenges and the potential role of cancer-related autonomic dysfunction and cachexia in prolonging vasodilatory shock. We propose that early multimodal management (fludrocortisone plus nutritional optimisation) should be considered in high-risk palliative care patients undergoing neurolytic procedures.

INTRODUCTION

Neurolytic coeliac plexus block (NCPB) is a well-established intervention for managing severe malignancy-related abdominal pain in patients with advanced cancer [1, 2]. The coeliac plexus provides sympathetic innervation to the esophagus, liver, pancreas, diaphragm, spleen, stomach, small bowel, and the ascending and proximal portions of the colon [2, 3]. Neurolysis disrupts pain transmission pathways, providing long-term relief, reducing opioid consumption and related side effects, improving quality of life, and facilitating better pain management for patients with intractable abdominal pain [2, 4, 5].

Common complications include hypotension and diarrhoea related to sympathetic denervation, transient alcohol intoxication-like symptoms, and injection-site pain. Serious complications are uncommon, with a reported incidence of 1 in 683 [3]. These include vascular injury (dissection of the aorta, hepatic artery, and superior mesenteric artery), SMV thrombosis, retroperitoneal hematoma, intra-abdominal abscess, neurological injury (direct nerve puncture, inadvertent spread of neurolytic agent to nerve, spasm of Adamkiewicz or anterior spinal artery resulting in paraplegia), visceral injury, pneumothorax and chylothorax [2–4, 6–8].

Transient orthostatic hypotension occurs in 20–50% of cases, typically lasting up to 5 days [3, 6, 8, 9] and was responsive to fluid resuscitation [6, 10, 11]. Although rare, cases of prolonged hypotension and

treatment response with fludrocortisone were reported by Silva et al. [7] and Yousefshahi & Tahmasebi [6].

In palliative care settings, where patients with advanced malignancy often have cachexia and autonomic dysfunction, even well-established procedures such as neurolytic coeliac plexus block can lead to unexpected and prolonged complications that significantly affect quality of life. We present a case of severe refractory hypotension after NCPB in a cachectic patient with metastatic gallbladder cancer, requiring inotropic support and fludrocortisone. It highlights the importance of a comprehensive peri-procedural autonomic assessment, vigilant monitoring, and timely interdisciplinary management.

CASE PRESENTATION

A 68-year-old cachectic woman (body mass index 16.5 kg/m²) with metastatic gallbladder cancer involving the liver, peritoneum, and regional lymph nodes, was admitted with a 2-week history of severe epigastric pain. The pain improved only partially (from 10/10 to 7/10 on the numerical rating scale) with oral paracetamol, dihydrocodeine and pregabalin. Fluoroscopy-guided bilateral coeliac plexus alcohol neurolysis was performed at the L1 level using 10 mL 0.25% levobupivacaine and 10 mL anhydrous alcohol (resulting in an approximate final alcohol concentration of 50%). Transient hypotension during the procedure responded to intravenous phenylephrine boluses and 500 mL of crystalloid.

However, recurrent hypotension developed on post-procedure day 1 despite aggressive resuscitation with further 3 liters of colloid and crystalloid, requiring intravenous dopamine infusion up to 19 µg/kg/min. Other than orthostatic dizziness, she was fully conscious, neurologically intact with normal limb power and sensation, and was free of pain without abdominal distension.

On further review, she had a preadmission 2-month history of diarrhoea, persistent poor oral intake, and 9.2% weight loss (from 39 kg to 35.2 kg). Despite an unrestricted diet since admission, she tolerated only 2 spoonfuls of food and 150 mL of water daily. Multiple loose stools and concentrated urine were noted. Thorough workup excluded acute complications of bleeding and infection. Laboratory tests showed hypokalemia (2.8 mmol/L), hemoglobin 12 g/dL, white blood cell count 4.8×10^9 /L, urea 3 mmol/L, creatinine 40 µmol/L, normal coagulation profile, normal thyroid-stimulating hormone and morning cortisol levels, and negative stool microbiology. The provisional diagnosis was severe persistent vasodilatory shock secondary to sympathetic neurolysis, exacerbated by pre-existing negative fluid balance and cachexia.

Multimodal management was subsequently commenced:

1. Pharmacological: 1-week course of oral fludrocortisone (0.1 mg daily) was given, based on efficacy in similar case reports [6,7] and proposed pharmacological effect on volume expansion.
2. Nutritional and supportive: A formal dietetic consultation was obtained to tailor energy-dense oral nutrition. Intravenous maintenance fluid was later tapered to oral hydration. Intravenous dopamine

infusion was tapered gradually and stopped on post-procedure day 12. Diarrhoea spontaneously resolved.

3. Rehabilitative: physiotherapist was invited for functional training, and she was discharged to a convalescent unit on post-procedure day 21.

DISCUSSION

This case illustrates a protracted hypotensive episode following NCPB, contrary to the expected typical transient presentation. Despite physiological plausibility, it is important to scrutinise other possible causes and manage accordingly. The common causes of shock are listed in Table 1. In addition, procedure-specific complications should be carefully considered, as the needle trajectory during NCPB passes in close proximity to many vital structures. Table 2 provides a list of potential complications of NCPB that may cause shock, although reported severe complication rate is as low as 1 in 683 [3].

Table 1
Common causes of shock

Type of Shock	Differential Diagnosis
Hypovolemic	Gastrointestinal and urinary loss, poor oral intake, gastrointestinal bleeding, trauma, burns
Cardiogenic	Acute myocardial ischaemia, severe arrhythmias, severe valvular dysfunction, myocarditis
Obstructive	Cardiac tamponade, pulmonary embolism, tension pneumothorax
Vasodilatory	Septic shock, systemic inflammatory response syndrome, anaphylaxis
Endocrine	Adrenal insufficiency, severe hypothyroidism, thyroid storm

Table 2
Potential complications of neurolytic coeliac plexus block that may cause shock

Category	Complication	Nature of Hypotension	Key Presentation	Expected Outcome
Sympathectomy-related	Hypotension	Vasodilatory	Orthostatic hypotension	Usually self-limiting; complete resolution within 5 days
Sympathectomy-related	Diarrhoea	Hypovolemic	Associated with opioid cessation after pain relief	Usually transient and self-limiting
Procedure-related	Vascular injury (aorta, coeliac artery, superior mesenteric artery, etc.)	Hypovolemic	Retroperitoneal or intra-abdominal haematoma, abdominal distension	Depends on severity; may require vascular repair
Procedure-related	Spinal cord injury (intrathecal injection)	Neurogenic	Lower limb weakness, numbness, sphincter dysfunction	Risk of long-term neurological damage
Procedure-related	Spinal cord ischaemia (anterior spinal artery spasm)	Neurogenic	Lower limb weakness, numbness, sphincter dysfunction	Depends on severity and duration; may range from full recovery to permanent paralysis
Procedure-related	Epidural or spinal infection	Neurogenic + Vasodilatory (septic)	Fever, back pain, neurological deficits, sphincter dysfunction	Early diagnosis and treatment are critical; risk of permanent paralysis or death
Procedure-related	Tension pneumothorax	Obstructive	Shortness of breath, chest pain, tracheal deviation, hyperresonance	Life-threatening; requires urgent thoracocentesis
Procedure-related	Pulmonary embolism	Obstructive	Sinus tachycardia, pleuritic chest pain, ECG changes (S1Q3T3)	Life-threatening in severe cases
Medication-related	Acute alcohol toxicity	Vasodilatory	Tachycardia, flushing, mild hypotension	Usually self-limiting
Medication-related	Local anaesthetic systemic toxicity	Cardiogenic + Neurogenic	Early signs: perioral numbness,	Life-threatening; treat with intralipid and supportive care

Category	Complication	Nature of Hypotension	Key Presentation	Expected Outcome
			metallic taste, tinnitus; Late signs: confusion, seizure, cardiac arrest	
Medication-related	Anaphylaxis	Vasodilatory	Hypotension, tachycardia, bronchospasm, angioedema, urticaria	Life-threatening; treat with adrenaline and supportive care

Diagnostic Consideration

We postulate that the persistent hypotension resulted from transient pharmacological sympathectomy and was sustained by cancer-related autonomic dysfunction and depleted physiological reserve from cancer-related cachexia, which created a "perfect storm" for refractory shock [12, 13].

This patient only experienced postural dizziness and 12 days of hypotension, while abdominal distension, neurological deficit, cardiac and respiratory symptoms were absent. Laboratory findings did not suggest infective or endocrinological causes.

Serious procedure-related complications were reasonably excluded on clinical grounds. Vascular injury (aortic or other major vessel dissection, retroperitoneal hematoma), neurological complications (spasm of anterior spinal artery, inadvertent spread of neurolytic agent to radicular nerves, accidental intrathecal injection, spinal infection), pneumothorax and pulmonary embolism were unlikely. Acute alcohol intoxication-like symptoms are typically transient and resolve within hours [1, 2].

Cases of prolonged hypotension after NCPB and successful treatment with fludrocortisone were reported [6, 7]. We propose a physiologically plausible explanation for this phenomenon.

Fludrocortisone in Vasodilatory Shock

Transient hypotension related to vasodilatory response after NCPB is preventable by intravenous fluid [10, 11]. However, persistent refractory hypotension is uncommonly documented. Treatment success with fludrocortisone was reported [6, 7] and it was a logical therapeutic choice.

Fludrocortisone exerts mineralocorticoid action at the distal renal tubule to promote renal sodium and water retention, thus expanding intravascular volume [14, 15] and counteracting relative hypovolemia from sympathectomy vasodilation. Furthermore, corticosteroids decrease capillary leak and enhance vascular responsiveness to catecholamines and angiotensin II [7, 15]. Emerging evidence from meta-analyses showed fludrocortisone plus hydrocortisone benefits in sepsis-induced vasodilatory shock, reduces the duration of vasopressor use [16], improves all-cause mortality [16–18], without elevated risk

of secondary infection. Likewise, persistent vasodilatory shock beyond 48–72 hours after NCPB, refractory to fluid resuscitation should trigger consideration for fludrocortisone therapy, instead of escalation of high-dose inotropic support.

From a palliative care perspective, this case illustrates the importance of recognizing that patients with advanced cancer and cachexia are at higher risk of prolonged complications after neurolytic procedures. Autonomic dysfunction and poor nutritional reserve can transform a typically self-limiting side effect into a major clinical challenge that threatens quality of life and prolongs hospitalization. Early involvement of the palliative care team for nutritional optimisation and symptom control, together with prompt use of fludrocortisone, can shorten vasopressor dependence and facilitate earlier return to community care.

Autonomic Dysfunction and Malnutrition in Cancer Patients

Autonomic dysfunction is prevalent, affecting up to 70–80% of patients with advanced cancer [19, 20]. Anticancer therapy, tumour cytokine, paraneoplastic processes, stresses, physical inactivity, and malnutrition can all contribute to this dysfunction [13, 19, 20]. Multi-system manifestations of autonomic dysfunction include cardiovascular (orthostatic hypotension, resting tachycardia, reduced exercise tolerance, coat-hanger headache), gastrointestinal (nausea, vomiting, gastroparesis, early satiety, nocturnal diarrhoea) and genitourinary (bladder emptying problem, male erectile dysfunction) symptoms [19, 20]. Ewing classification is a systematic approach used to evaluate cardiovascular sympathetic and parasympathetic responses, however frail patients may be unable to complete it [19, 20]. Systematic assessment could be beneficial to assess the risk of exacerbated hemodynamic collapse after NCPB.

Cancer-related malnutrition, cachexia, and physical inactivity often render the patient vulnerable with severe loss of skeletal muscle mass and progressive functional impairment. It is often not fully reversible by conventional nutritional support [12, 21]. In this patient, poor oral intake of solids and fluids perpetuated her negative fluid and caloric balance, hence exacerbated the persistent hypotension. Nutritional intervention is crucial but difficult due to anorexia and early satiety. Energy-dense supplements may be better tolerated [21]. Prolonged bedrest due to orthostatic dizziness may result in further functional deterioration. To mitigate the risks of a patient with advanced cancer, timely multimodal management is essential, integrating pharmacological, nutritional, and physical exercise to mitigate symptoms and improve overall patient outcomes [12].

Limitations

We acknowledge the speculative nature of our proposed explanation. Considering the patient's symptomatology, the palliative intent of care and the unlikely surgical candidacy, contrast CT and MRI were not arranged to exclude internal hemorrhage and spinal infection. Formal autonomic function testing (for example Ewing battery) was not performed due to hemodynamic instability, which renders our proposition speculative. We view this gap as an opportunity for further research to elucidate this unproven plausibility. We suggest that pre-procedural screening and early optimised management may be beneficial to patients undergoing NCPB.

CONCLUSION

This case highlights a prolonged occurrence of persistent hypotension after neurolytic coeliac plexus block in a physiologically compromised patient with advanced cancer and cachexia. We propose a physiologically plausible explanation for this phenomenon and demonstrate successful treatment with fludrocortisone. We suggest that comprehensive pre-procedural assessment of autonomic function and nutritional status, together with timely use of fludrocortisone and nutritional support, may improve safety and outcomes in high-risk patients undergoing this procedure.

Declarations

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Conflicts of Interest / Competing Interests

The authors declare they have no competing interest

Ethics Approval

The study was approved by the Central Institutional Review Board of Hospital Authority Hong Kong (CIRB-2025-208-4).

Consent to Participate

Informed consent was waived and all obtained data were anonymised.

Consent for Publication

Informed consent was waived and all obtained data were anonymised.

Availability of Data and Materials

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Code Availability

Not applicable.

Authors Contribution

Lo wrote the manuscript text, tables and figure. Lim was the clinical advisor and revised the manuscript text. All authors reviewed the manuscript.

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Figures

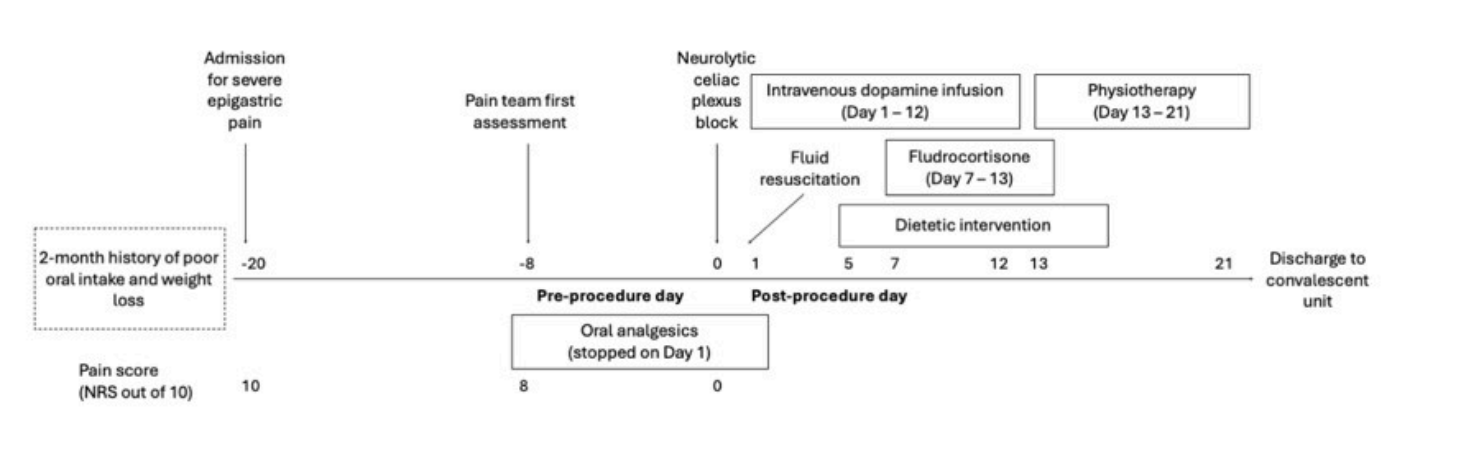


Figure 1

Timeline of clinical course relative to neurolytic coeliac plexus block (Day 0). Pain scores are shown on the numerical rating scale (NRS 0–10). Key interventions, including dopamine infusion (Day 1–12) and fludrocortisone (Day 7–13), are indicated.