Original Research

TRENDELENBURG POSITION–ASSOCIATED INTRA-OPERATIVE HYPOTENSION IN A PATIENT WITH DESCENDING COLON CARCINOMA AND ACUTE INTESTINAL OBSTRUCTION: A CASE REPORT

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ABSTRACT

Background and Aims: Intraoperative hypotension (IOH), particularly when mean arterial pressure falls below 65 mmHg, has been linked to adverse perioperative outcomes. Although the Trendelenburg position is frequently employed to augment venous return and improve surgical exposure, it may produce unexpected cardiovascular compromise in selected clinical settings. We describe a case of severe positional hypotension during emergency laparotomy for large bowel obstruction.

Case Description: A 58-year-old woman without known comorbidities underwent emergency exploratory laparotomy for descending colon carcinoma causing acute intestinal obstruction. Shortly after induction of general anesthesia and placement in moderate Trendelenburg position, she developed marked hypotension (70/40 mmHg) that was poorly responsive to fluid resuscitation and vasopressors. Hemodynamic stability was rapidly restored after returning the patient to the neutral supine position.

Conclusion: In patients with raised intra-abdominal pressure secondary to bowel obstruction, Trendelenburg positioning may impair venous return rather than enhance it. Awareness of this phenomenon and prompt positional correction are essential to prevent sustained hypotension and end-organ compromise.

Keywords: Trendelenburg Position, Intraoperative Hypotension, Bowel Obstruction, Colon Carcinoma, Anesthesia.

INTRODUCTION

Intraoperative hypotension is a common yet clinically significant event during major surgery. Sustained reductions in mean arterial pressure below 65 mmHg have been independently associated with myocardial injury, acute kidney injury, and increased postoperative mortality [1–3]. The causes of IOH are multifactorial and may include anesthetic-induced vasodilation, myocardial depression, hypovolemia, and physiological alterations due to surgical positioning.

The Trendelenburg position, characterized by head-down tilt, is traditionally believed to improve venous return by promoting gravitational redistribution of blood toward the thorax [4]. However, contemporary physiological studies suggest that the cardiovascular effects of this position are complex and dependent on intrathoracic and intra-abdominal pressures [5].

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In conditions associated with abdominal distension, such as intestinal obstruction, elevated intra-abdominal pressure may reduce venous return by compressing the inferior vena cava and impairing cardiac preload [8].

We present a case in which moderate Trendelenburg positioning precipitated significant intraoperative hypotension in a patient undergoing emergency laparotomy for obstructing descending colon carcinoma.

CASE REPORT

A 58-year-old woman presented with a one-day history of worsening abdominal pain, repeated vomiting, progressive abdominal distension, and failure to pass stool or flatus. She had no documented history of hypertension, diabetes mellitus, ischemic heart disease, or chronic respiratory illness. On admission, she was hemodynamically stable with a blood pressure of 130/80 mmHg and heart rate of 96 beats per minute. Oxygen saturation was 97% on room air. Physical examination revealed a distended abdomen with exaggerated bowel sounds. Laboratory

investigations were unremarkable except for mild leukocytosis. Contrast-enhanced computed tomography of the abdomen demonstrated a mass lesion in the descending colon with significant proximal bowel dilatation consistent with acute large bowel obstruction.

Emergency exploratory laparotomy was planned, and the patient was categorized as American Society of Anesthesiologists (ASA) physical status II (E). Preoperative preparation included intravenous crystalloid resuscitation and nasogastric decompression. Standard monitoring electrocardiography, non-invasive blood pressure, pulse oximetry, and capnography was instituted in the operating room.

Given the risk of aspiration, rapid sequence induction was performed after adequate preoxygenation. Intravenous fentanyl (2 µg/kg) and propofol (2 mg/kg) were administered, followed by rocuronium (1 mg/kg) to facilitate tracheal intubation. Cricoid pressure was applied. The airway was secured uneventfully on the first attempt, and correct placement was confirmed with capnography and bilateral chest auscultation. Anesthesia was maintained using isoflurane in an oxygen–air mixture, supplemented with intermittent fentanyl and neuromuscular blockade. Ventilation was adjusted to maintain end-tidal carbon dioxide between 35 and 40 mmHg.

Following induction and stabilization, the patient was positioned in approximately 15–20° Trendelenburg to facilitate surgical exposure. Within three to four minutes of head-down positioning, her blood pressure declined abruptly from 118/72 mmHg to 70/40 mmHg, accompanied by sinus tachycardia (110 beats per minute). No significant blood loss had occurred, and airway pressures as well as end-tidal carbon dioxide remained within acceptable limits.

Immediate corrective measures included administration of a rapid 500 mL crystalloid bolus, reduction of volatile anesthetic concentration, and incremental intravenous boluses of ephedrine and phenylephrine. Despite these interventions, hypotension persisted. The patient was promptly returned to the neutral supine position. Within approximately two minutes, blood pressure improved to 110/70 mmHg without additional vasopressor support.

Surgery was subsequently continued with minimal head-down tilt only when strictly necessary. Hemodynamic parameters remained stable throughout the remainder of the procedure. Estimated blood loss was less than 200 mL, and urine output was adequate. Neuromuscular blockade was reversed at the end of surgery, and the patient was extubated after meeting standard criteria. The postoperative period was uneventful, with no recurrence of hypotension.

DISCUSSION

Evidence indicates that even brief periods of intraoperative hypotension may adversely affect postoperative outcomes [1–3]. General anesthetic agents such as propofol and volatile anesthetics are known to cause dose-dependent vasodilation and myocardial depression, predisposing patients to hemodynamic instability [7].

Although Trendelenburg positioning has historically been used to augment venous return, its actual effect depends on the interaction between venous capacitance, intrathoracic pressure, and abdominal compliance [4,5]. In patients with increased intra-abdominal pressure, venous return may be compromised due to mechanical compression of the inferior vena cava [8]. In such circumstances, head-down tilt may further elevate intrathoracic pressure and reduce effective cardiac output rather than improve it.

In the present case, the close temporal relationship between Trendelenburg positioning and hypotension, along with rapid resolution upon repositioning, strongly supports a positional mechanism. The lack of response to fluids and vasopressors further emphasizes that mechanical factors affecting preload were likely predominant.

This case highlights the importance of cautious positional adjustments in patients with bowel obstruction and tense abdominal distension. Early recognition of positional hypotension and prompt correction can prevent prolonged episodes of reduced organ perfusion.

CONCLUSION

Trendelenburg positioning may precipitate significant intraoperative hypotension in patients with elevated intra-abdominal pressure secondary to intestinal obstruction. Vigilant hemodynamic monitoring and immediate positional correction are crucial to maintaining cardiovascular stability and preventing perioperative complications.

Declaration of Patient Consent: Appropriate patient consent was obtained for publication of anonymized clinical information.

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