

Postoperative hypertension following radical neck dissection

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Abstract

Baroreflex failure results in wide excursions of blood pressure and heart rate. We report two cases that developed severe postoperative hypertension after radical neck dissection. Carotid sinus denervation during neck dissection may be the cause of the reflex hypertension once general anesthesia-induced vasodilatation has ended.

Key words: Blood pressure, baroreceptors, complication, carotid sinus, denervation, hypertension, neck dissection

Introduction

Excessive variations in blood pressure (BP) are guarded against by baroreflexes. Impaired baroreflex mechanisms in the perioperative period can result in hemodynamic instability. Anesthetists are aware of life-threatening bradycardia and asystole that can occur because of the manipulation around the carotid sheath during a radical neck dissection (RND). Perhaps less well known is that an acute form of baroreflex failure can manifest as a hypertensive crisis in patients who have had surgery on the neck.^[1, 2] We present two cases of severe postoperative hypertension developing in patients who underwent neck dissection.

Case Reports

Case 1

An 80-year-old, 48-kg, hypertensive man was scheduled for wide local excision of carcinoma lip and supra-omohyoid neck dissection. He was a smoker for 25 years and a known hypertensive (controlled with amlodipine and atenolol). Patient was premedicated with alprazolam and antihypertensive

medication. Baseline heart rate (HR) was 50/min and BP was 129/83 mmHg. Anesthesia was induced with fentanyl 100 mcg and propofol 80 mg IV. Nasotracheal intubation was facilitated with vecuronium 5 mg. Lignocaine 75 mg IV was administered to attenuate sympatho-adrenergic response to laryngoscopy and tracheal intubation. Anesthesia was maintained with morphine, desflurane 3% in nitrous oxide 66% and oxygen. Hemodynamic parameters were stable intraoperatively. Wide local excision of carcinoma lower lip and supra-omohyoid neck dissection was performed. The patient received 2700 ml of Ringers' lactate solution. Surgery lasted 3.5 h and blood loss was about 350 ml while urine output was >0.5 ml/kg/h.

At the end of surgery, neuromuscular block was antagonized with neostigmine and glycopyrrolate. Lignocaine 75 mg IV was administered to blunt the hemodynamic response to tracheal extubation. After tracheal extubation the patient was conscious and following verbal commands, HR was 62/min, BP 156/94 mmHg, and SpO₂ 99% on 100% oxygen. Ten minutes following tracheal extubation there was a progressive increase in BP up to 202/115 mmHg, with HR 65/min. Midazolam 0.5 mg and fentanyl 40 mcg IV were administered for sedation and analgesia. Chest auscultation revealed fine basal crepitations. The saturation dropped to 82%. The patient was propped up and 100% oxygen by mask was administered. Morphine 1.5 mg and frusemide 5 mg were administered IV. As there was an inadequate decrease in BP (196/118 mmHg with HR 58/min), nitroglycerine (NTG) infusion was started at 0.5 mcg/kg/min. Within 10 min, BP stabilized between 140/100 and 130/80 mmHg and HR 56 and 60/min. SpO₂ increased to 92% with FiO₂ 1.0. Ninety minutes later NTG infusion was tapered and stopped. Chest was clear. Arterial blood gas

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(ABG) analysis at a FiO_2 of 1.0 revealed PO_2 65 mmHg, PCO_2 46 mmHg and pH 7.42.

The patient was transferred to the intensive care unit (ICU) for further management. The vital signs remained stable with HR 68/min, BP 130/86 mmHg, $\text{SpO}_2 > 95\%$ at FiO_2 0.4 and urine output > 0.5 ml/kg over 4 h. ABG analysis (at FiO_2 0.4) revealed PO_2 84 mmHg, PCO_2 44 mmHg, and pH 7.43. The patient was observed overnight and transferred to the ward the following day and discharged home on the seventh postoperative day.

Case 2

A 68-year-old, 58 kg, normotensive male, a case of cervical lymph node metastasis with unknown primary, was scheduled for right sided RND. Preanesthetic evaluation was unremarkable. Patient was premedicated with alprazolam. Baseline HR was 93/min and BP was recorded as 116/85 mmHg. Anesthesia was induced with fentanyl and propofol, and orotracheal intubation facilitated with vecuronium. Anesthesia was maintained with pentazocine, isoflurane 0.6% in nitrous oxide 66% and oxygen. Right sided RND was performed. Intraoperative BP ranged from 115/64 to 142/82 mmHg and HR 65 to 74/min. Patient received 3000 ml Ringers' lactate solution. Surgery lasted 3.5 h and blood loss was about 250 ml with urine output > 0.5 ml/kg/h. At the end of surgery, HR was 64/min and BP was 145/77 mmHg. Neuromuscular block was antagonized with neostigmine and glycopyrrolate. Prior to tracheal extubation, BP rose to 210/110 mmHg with HR 102/min. Lignocaine 100 mg IV was administered. The trachea was extubated as respiratory efforts were adequate and as the patient was awake and not tolerating the tracheal tube. Fentanyl 50 mcg and midazolam 1 mg IV were given, but the BP increased to 246/114 mmHg with HR 96/min. Labetalol 10 mg IV was administered and repeated after 20 min. BP decreased to 178/116 mmHg with HR 79/min. A rising trend in BP was observed and NTG infusion was started at 0.5 mcg/kg/min and gradually increased to 1.5 mcg/kg/min. BP stabilized between 140/89 and 115/58 mmHg and HR between 75 and 82/min. Upper airway obstruction was observed, which was relieved by jaw thrust. As the obstruction persisted, the trachea was reintubated after administering propofol 80 mg and succinylcholine 100 mg IV. Tracheal intubation was not associated with a hypertensive response and surprisingly BP dropped to 90/54 mmHg with HR 69/min following propofol administration. NTG infusion was discontinued. The patient remained hemodynamically stable and was transferred to the ICU for further management with elective ventilation.

On arrival in the ICU, HR was 70/min, BP 140/70 mmHg, and SpO_2 was 100% at FiO_2 0.4 on controlled ventilation.

BP started to rise and at 4 h postoperative was 160/100 mmHg. Morphine 3 mg and midazolam 1 mg IV were administered. BP increased to 196/118 mmHg with HR 62/min. NTG infusion was restarted at 1 mcg/kg/min and increased to 2 mcg/kg/min. At 10 h postoperatively, BP had stabilized at 110/70 mmHg, HR 82/min, and SpO_2 100% at FiO_2 0.4, with NTG infusion at 0.5 mcg/kg/min. Amlodipine 5 mg orally was given via a Ryle's tube. By 20 h postoperatively, the NTG infusion was tapered off. Mechanical ventilation was gradually weaned to a T-piece and the trachea extubated 22 h postoperatively. The patient remained stable and was transferred to the ward on the third postoperative day and was discharged from the hospital on day 12, on amlodipine 5 mg once daily therapy.

Discussion

Postoperative hypertension has been reported following carotid endarterectomy, RND, and laryngectomy.^[1-5] The reported incidence of hypertension after carotid endarterectomy is 19-38%^[4, 5] and after RND it is 9.6-20.2%.^[1, 2] It characteristically occurs after vasodilatation caused by anesthetic agents has subsided.^[1,2] Carotid sinus denervation caused by mobilization of the carotid bifurcation during the operation has been implicated in its etiology.^[1,2]

The carotid sinus reflex plays a central role in BP homeostasis [Figure 1]. Changes in stretch and transmural pressure are detected by baroreceptors in the heart, carotid sinus, aortic arch, and other large vessels. Afferent impulses are transmitted by the carotid sinus, glossopharyngeal, and vagus nerves to the nuclei tractus solitarius and the para-median nucleus in the brain stem. Efferent limbs are carried through sympathetic and vagus nerves to the heart and blood vessels, controlling HR

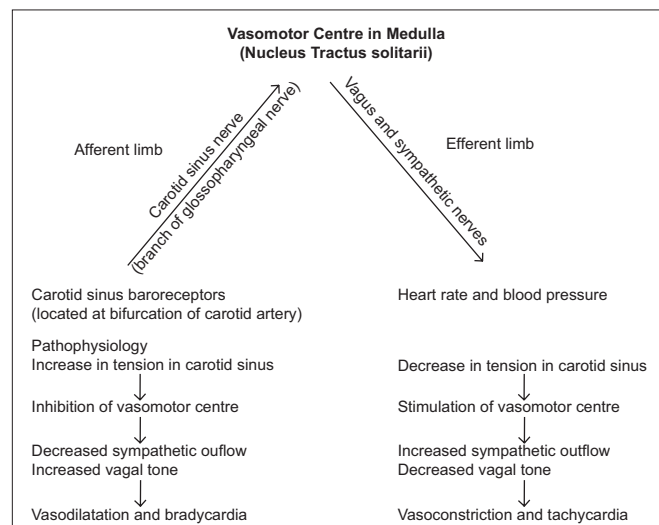


Figure 1: Carotid sinus baroreceptor reflex pathway and physiology

and vasomotor tone. Carotid sinus nerve injury during neck surgery leads to an interruption of signals from the carotid baroreceptors that causes stimulation of the vasomotor centre, resulting in hypertension and tachycardia.^[6]

McGuirt and May^[1] found the incidence of hypertension following RND to be 9.6% and that it usually occurred in the first two postoperative hours and lasted approximately 9 hours. Six patients required interventional therapy (IV NTG or sodium nitroprusside). We have earlier reported a case of severe postoperative hypertension following maxillectomy and RND for squamous cell carcinoma of the maxilla.^[7] Venkatesan *et al.*^[5] reported a hypertensive crisis following laryngectomy under general anesthesia attributed to baroreflex failure due to manipulation around the carotid sheath.

Acute baroreflex failure following bilateral carotid sinus denervation may produce severe labile hypertension, headache, diaphoresis and emotional instability.^[8] Unilateral denervation of the carotid sinus results in temporary elevation of the BP and pulse, and no change in the postural vascular reflexes.^[8,9] Long-term consequences of bilateral carotid sinus denervation on arterial BP are unclear. Reports vary from normalization of BP,^[9] to increased BP variability^[8] and sustained hypertension in individual patients.^[10]

Advanced age, hypertension, and preoperative radiotherapy have been considered important in the etiology of such hypertensive episodes.^[3] Both patients described in the present report were elderly, Case 1 had preoperative hypertension, but neither had received preoperative radiotherapy. An exaggerated hemodynamic response at laryngoscopy and intubation was not observed. Intraoperative BP was not labile. BP increase in both the cases occurred after cessation of anesthetic agents (20 and 10 min in Case 1 and Case 2, respectively), when anesthesia-induced vasodilatation had abated. The response to sedation, narcotic analgesic, and labetalol administration was limited. High BP responded to NTG-induced vasodilatation. There was a drastic reduction in BP observed in Case 2 following postoperative propofol administration in preparation for re-intubation. Venkatesan *et al.*^[3] also found propofol to be effective in treating the hypertensive response when their patient did not respond to high doses of NTG. However, the pharmacological treatment of choice for BP surges in procedures in which baroreceptor dysfunction is anticipated is clonidine.^[11] Clonidine acts centrally and peripherally to attenuate sympathetic activation and limit the extent to which pressor surges can occur.^[11] Availability of alpha adrenergic agonists such as clonidine or dexmedetomidine in the operation theatre should be ensured during surgical procedures that can cause baroreflex failure in the postoperative period.

Inadequate analgesia and preoperative hypertension can both lead to a hypertensive response in the post operative period. Analgesia was adequate in both cases as is evident from the stable intraoperative hemodynamic profile and lack of complaints of pain on awakening. Additional sedation and analgesia were administered as a first measure in both patients to rule out the possibility of inadequate analgesia with no beneficial response. Case 1 was an elderly hypertensive, well controlled on amlodipine and atenolol, had no episodes of intraoperative hypertension, and vital signs were within normal limits immediately following tracheal extubation. Development of severe postoperative hypertension following RND in both patients suggests baroreflex failure as the possible etiology.

In conclusion, baroreflex failure is an alarming and underestimated complication following RND. It should be suspected in elderly patients with otherwise unexplained labile hypertension in the postoperative period.

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