Acute Postoperative Baroreflex Failure following Radical Neck Dissection

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Abstract
Blood pressure lability as a result of altered baroreflex mechanism can occur in head and neck surgeries. We report a case in which hypertensive crisis occurred in the immediate postoperative period following unilateral radical neck dissection.

Introduction
Baroreflexes buffer arterial blood pressure (BP) against excessive rise or fall. Impairment of function of afferent baroreceptor nerves or their central connections results in baroreflex failure, with marked fluctuations of blood pressure and heart rate (HR). Literature concerning acute baroreflex failure in the perioperative setting is sparse. Clinicians are well aware of the life-threatening bradycardia and asystole that can occur during a radical neck dissection (RND) due to manipulation around the carotid sheath. Perhaps less well known is that hypertensive crisis, as an acute form of baroreflex failure, can occur in patients who have had surgery on the neck. We present a case of severe postoperative hypertension developing in a patient who underwent maxillectomy and neck dissection for squamous cell carcinoma of the left maxillary alveolar process.

Case Report
A 69 yr old, 58 kg, normotensive male (smoker for 30 years) presented with a growth in the left half of the hard palate and left buccal mucosa for 4 months. CECT face and neck revealed left maxillary alveolar process malignant growth with bone destruction and significant ipsilateral level II nodes. Clinical staging was T4N1M0. A biopsy was performed under local anaesthesia and histopathology confirmed the growth to be squamous cell carcinoma. Pre-anaesthetic evaluation was unremarkable. No airway difficulty was anticipated. He was scheduled for left maxillectomy and neck dissection.

The patient received oral alprazolam 0.5 mg and ranitidine 150 mg the night before and on the morning of surgery. In the operating room, standard monitoring (non-invasive BP, ECG, pulse oximetry, EtCO$_2$ and core body temperature) was instituted. Baseline BP was 110/80 mmHg and HR was 80 beats/min. Anaesthesia was induced with fentanyl 100 µg and thiopentone sodium 250 mg. Nasotracheal intubation was facilitated with rocuronium. Anaesthesia was maintained with 66% N$_2$O in O$_2$ and isoflurane (0.6%-1%) with controlled ventilation. Intraoperative analgesia was provided with morphine. Left partial maxillectomy with removal of the left buccal mucosa with selective (supra-omohyoid) neck dissection with pectoralis major myofascial flap reconstruction was performed. The HR and BP remained stable at induction of anaesthesia (84-96 beats/min and 120/76 to 146/90 mmHg, respectively). The intraoperative course was uneventful, with EtCO$_2$ ranging between 28 to 37 mmHg, SpO$_2$ > 98%, BP between 110/70 mmHg and 130/84 mmHg, and HR in sinus rhythm at 72-86 beats/min. Blood loss was approximately 1200 ml. The patient received crystalloid 4 L, colloid 0.5 L and 3 units whole blood. CVP was maintained between 8 and 12 cm H$_2$O. Urine output was greater than 1 ml/kg/h. The total duration of surgery was 7 hours.

At the end of surgery neuromuscular block was not antagonized. Additional doses of morphine 3 mg and midazolam 1 mg were administered intravenously. The plan was to transfer the patient to the Intensive Care Unit (ICU) (with the trachea intubated) on controlled ventilation followed by weaning and possible extubation after 24 hrs when the patient would be fully awake, edema has subsided and a patent airway is guaranteed. At the time of transfer to the ICU the patient’s condition was stable with HR 80 beats/min, BP 118/80 mmHg, temperature 36.4°C, respiration controlled, CVP 11 cm H$_2$O, SpO$_2$ 100% and EtCO$_2$ 35 mmHg.

In the ICU, approximately 30 min after cessation of anaesthetic, the patient’s BP started to increase from 150/90 mmHg to 228/128 mmHg within 5 min. HR was 74 - 82/ min and the rhythm was sinus. The patient had bleeding from the surgical site and flap elevation by hematoma. Sedation and analgesia was reinforced with midazolam 2 mg iv and fentanyl 100 µg iv. Amlodipine 5 mg was administered through the nasogastric tube and flushed with saline. Labelotol 20 mg iv was administered. BP decreased to 180/93 mmHg. There was no further decrease in BP for the next 10 min. Nitroglycerine infusion was started at 0.5 µg/kg/min and slowly increased to 5 µg/kg/min over 10 min. Labelotol 20 mg was repeated after 20 min of the first dose. BP was maintained between 140/80 mmHg to 150/90 mmHg and HR between 70-80/min. The patient received 2 units of blood and 2 units of fresh frozen plasma.

Amlodipine 5 mg bid was administered via nasogastric tube. NTG infusion was tapered and then discontinued on the third postoperative day. The patients’ respiration was assisted and lungs were ventilated and weaned to a t-piece over 48 hrs. Trachea was extubated on postoperative day 2. There was a transient elevation of serum bilirubin to 3.7 mg% (0.9 direct and 2.8 mg indirect). Other biochemical tests were normal. Patient...
was transferred to the ward on postoperative day 3 with BP controlled with amlodipine 5 mg bid.

In the ward, the patient complained of continuous headache which was treated with oral NSAIDs. Partial necrosis of flap cover occurred. The wound was allowed to heal by secondary intention. The patient received antibiotics, anti-inflammatory medication, amlodipine and alprazolam.

A retrospective analysis of BP readings obtained from the pre-surgery follow-up medical records of the patient showed normal BP readings. The patient was investigated for renal artery stenosis and pheochromocytoma. Abdominal ultrasound and renal Doppler study were normal. Urinary catecholamine levels were in the normal range. There was no diaphoresis, postural lightheadedness or emotional instability. There was no evidence of orthostatic hypotension as seen by BP readings recorded in the supine and the standing position. Deep breaths test and hand grip test were negative for autonomic dysfunction. Oral amlodipine was decreased to 5 mg od after 2 weeks. BP was maintained at 130/80 mmHg. When the antihypertensive drug dosage was reduced further to 2.5 mg od, BP rose. The patient was discharged home after 6 weeks on amlodipine 5 mg od.

Discussion

Postoperative hypertension has been reported following head and neck surgery, notably carotid endarterectomy and radical neck dissection. It typically occurs after vasodilatation caused by general anaesthesia has subsided.\(^1\)\(^,\)\(^2\) Carotid sinus denervation has been implicated in the occurrence of such hypertensive episodes.\(^1\)\(^,\)\(^2\)

The carotid sinus baroreceptors, located at the bifurcation of each common carotid artery, play a central role in blood pressure homeostasis. Changes in stretch and transmural pressure in the carotid sinus are detected and the signals are transmitted by the carotid sinus nerve, a branch of the glossopharyngeal nerve, to the vasomotor center in the medulla (nuclei tractus solitarius). The principal efferent pathway of the reflex arc is the vagus nerve. An increase in intrasinus tension leads to inhibition of the vasomotor center resulting in a decrease in systemic BP, bradycardia, and slowing of respiration; the opposite effects follow a decrease in tension within the sinus. In the absence of afferent inhibitory impulses, vasomotor center stimulation leads to a sympathetically mediated increase in heart rate, myocardial contractility, and peripheral vasoconstriction leading to an increase in BP. Injury to the carotid sinus nerve during head and neck surgery results in interruption of signals from the carotid baroreceptors that causes stimulation of the vasomotor center, resulting in hypertension and tachycardia.\(^3\)

McGuirt et al\(^1\) found a 9.6% incidence of hypertension following radical neck dissection in 94 patients (postoperative BP readings of 200/100 mmHg or higher or of more than 40 mmHg systolic and more than 20 mmHg diastolic above preoperative levels). These BP elevations occurred in the first two postoperative hours and lasted approximately nine hours. Interventional therapy (iv nitroglycerine or sodium nitroprusside) was required in 6.4% patients. There were no cardiac or central nervous system sequelae, but two patients had postoperative haemorrhage attributed to hypertension.

Postoperative hypertension after radical neck dissection was detected in 20.2% of 109 patients.\(^2\) Hypertension appeared after the vasodilatation generated by anaesthesia had ended. The authors observed that if postoperative hypertension is encountered after the first RND, the risk of such hypertension after surgery on the contralateral side increased significantly. Contrary to this, Koc et al\(^5\) reported that sparing of the carotid sinus nerve during RND had no influence on the incidence of postoperative hypertension.

The reported incidence of hypertension after carotid endarterectomy is 19% to 38%.\(^3\) Carotid sinus area infiltration with bupivacaine after carotid endarterectomy does not reduce the incidence of postoperative hypotension but significantly increases the incidence of postoperative hypertension and results in more frequent requirement of vasodilators.\(^6\) Venkatesan et al\(^6\) reported a case in which hypertensive crisis was associated with laryngectomy under general anaesthesia. They suggest baroreflex failure due to manipulation around the carotid sheath as the possible cause.

Bilateral carotid sinus denervation in dogs and baboons increases BP and BP variability with severe hypertensive surges during agitation, with return of BP to preoperative levels within 14 days postoperatively.\(^7\) In humans, bilateral carotid sinus denervation may result in acute baroreflex failure, producing severe labile hypertension, headache, diaphoresis and emotional instability.\(^8\) Unilateral denervation of the carotid sinus in man resulted in only temporary elevation of the BP and pulse, and no change in the postural vascular reflexes.\(^8\),\(^9\) Data on the long term effects of bilateral carotid sinus denervation on arterial BP are limited and controversial. Reports vary from normalization of BP, to increased BP variability and sustained hypertension in individual patients.\(^10\)

Factors other than carotid sinus denervation have been implicated in the occurrence of such hypertensive episodes.\(^5\) Preoperative hypertension has been considered important in the etiology of hypertension during carotid endarterectomy. There is deterioration of baroreceptor function with age. Radiation therapy for head and neck tumors leads to attenuated baroreflex sensitivity with doses in excess of 70 Gy resulting in baroreceptor failure.

Our patient was not hypertensive. Previous medical records documented normal BP values. There was no previous exposure to radiotherapy. An exaggerated haemodynamic response at laryngoscopy and intubation was not observed. Intraoperative BP was not labile. BP increase occurred approximately 30 min after cessation of inhalational anaesthetic agents. The response to sedation, narcotic analgesic and labetalol administration was limited. BP responded to nitroglycerine-induced vasodilatation. The severe vasoconstriction resulting in hypertensive crisis also took its toll on the myofascial flap that suffered partial necrosis. Labile BP, malignant hypertypia, pheochromocytoma, and renovascular hypertension were other considerations. These possibilities were excluded by all previous BP readings and temperature in the normal range, normal abdominal ultrasound examination and renal Doppler study, and normal urinary catecholamine levels.

The baroreflex failure syndrome is a severe and underestimated complication during and after head and neck surgery. It should be considered in patients with otherwise unexplained labile hypertension.

References


### Announcement

**API Orations / Lectureships**

Recommendations are invited from members for the following assignments so as to reach, Hon. General Secretary – API, Dr. Sandhya Kamath by 31st July 2009.

**Category No. (i) (General Medicine)**

- Netaji Oration – 2010 & 2011
- Dr. G.S. Sainani Oration – 2010 & 2011
- Dr. PJ Mehta Oration – 2010 & 2011

The selected candidate has to deliver his/her lecture at the Annual Conference of API 2010 / 2011. The above orators will get the Award money of Rs. 10,000/- and TA for Orator by economy class airfare from API, complimentary registration and complimentary one night stay in the designated Conference hotel by the APICON Organising Committee.

**Category No. (ii)**

- Searle Oration - 2010 & 2011 (General Medicine)
- Prof. Rathinavelu Subramania Endowment Oration -2010 & 2011 (General Medicine)
- Ranbaxy Oration -2010 & 2011 (for Infectious diseases)

The selected candidate has to deliver his/her lecture at the Annual Conference of API 2010 / 2011. The above orators will get the award money of Rs. 5,000/- and TA by economy class airfare from API, complimentary registration and complimentary stay in the designated conference hotel by the APICON Organising Committee.

**Category No. (iii) : All lectureships viz**

1. Dr. Coelho Memorial Lectureship in Experimental Medicine – 2010 & 2011,
2. Sinofi Aventis Lectureship in Diabetes – 2010 & 2011
3. Dr. Yodh Memorial and Gwalior Conference Training Fellowship - 2009 / 2010;

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1. Dr. Yodh Memorial and Gwalior Conference Training Fellowship - 2009 / 2010;

For the above all lectureships and awards are open to eminent persons from the discipline of medicine and allied subjects such as Pharmacology, Biochemistry, Pathology and Physiology.

The selected candidate has to deliver his/her lecture at the Institution of his/her choice in the year 2009. The candidate has to get a notification in writing from the Institution that he/she has delivered the lecture.

Persons are selected from the recommendations received from members of the API. The orator in the discipline of medicine should preferably be a member of API. The recommendations for the above assignments must be accompanied with reasons for recommending a particular person showing the value of his/her research and eight copies each of three of his/her best publications. All relevant papers in connection with the suggestions, such as the bio-data, list of publications etc., should be submitted in 8 sets by the proposer. The recipient of the above oration should deliver a lecture pertaining to his/her work at the Annual Conference in January, 2010.

Those who have received Oration / Lectureship in a given category are eligible for application for the other two categories.

The members of the Governing Body of API and the Members of the Faculty Council of ICP are not eligible to receive any Oration, Lectureship or Award.

The prescribed nomination form for the above orations / Lectureship are on the API website “apindia.org”

The completed application forms for the above Lectureship should reach to Dr. Sandhya Kamath, Hon. General Secretary of API, Unit No. 6 & 7, Turf Estate, Opp. Shakti Mill Compound, Off. Dr. E. Moses Road, Near Mahalaxmi Station West, Mumbai – 400 011 not later than 31st July 2009.

**Dr. Sandhya Kamath**, Hon. General Secretary

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