

A Case of Cardiac Arrest During Parotidectomy Surgery

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Abstract

Introduction: Sudden brady-arrhythmias during anaesthesia can be life threatening. Bradycardia and asystole have been reported during Maxillofacial surgeries due to tri-gemino-cardiac reflex. We report a case of a rare complication of asystole following dissection and handling of deep lobe of parotid gland during total parotidectomy under general anaesthesia.

Case Presentation: A 70-year-old, 82 kg, normotensive female scheduled for total left parotidectomy for tumor excision with nerve monitoring under general anaesthesia developed severe bradyarrhythmia soon progressing to asystole; 85 min. into surgery during dissection of deep lobe of parotid possibly due to tri-gemino-cardiac reflex.

Conclusion: We present this case of asystole during parotidectomy to demonstrate the significance and possible severity of the reflex bradycardic response during surgical handling of deep lobe of parotid gland.

Keywords: Cardiac arrest, Parotidectomy, Resuscitation, General anaesthesia, Nerve monitoring.

Introduction

Parotidectomy, a standard reliable treatment for tumours may damage essential nerves and tissues in head and neck. More often it's an elective surgery and it isn't considered a high-risk procedure. Parotidectomy whether subtotal or total is associated with certain possible complications namely facial nerve injury, haemorrhage, infection, salivary fistula, seroma, keloid formation, gustatory sweating and recurrent tumour [1]. Trigemino-cardiac reflex (TCR) is a rare complication during total parotidectomy surgery. TCR causing severe dysrhythmias have been reported during maxillofacial surgeries [2-4] but the literature is sparse in reporting severe brady-arrhythmias during parotid surgery [5]. We report a case of asystole during dissection of the deep lobe of parotid during left parotidectomy. We are reporting this case to propose a hypothesis of etiology and to signify the possible existence of TCR causing severe brady-arrhythmias that may occur during dissection of deep lobe during parotid surgeries.

Case Report

A 70-year-old, 82 kg, normotensive female was scheduled for total left parotidectomy for tumor excision. Patient was a known asthmatic for thirty years, on asthalin inhaler on as need basis. She was also on once-a-day Tab Eltroxin 125 mcg for her hypothyroidism for last fifteen years. She had undergone appendicectomy and hysterectomy, both uneventfully many years ago. Physical examination was

unremarkable. Routine preoperative investigations, including serum electrolytes, ECG and 2D echo were within normal limits.

In the operating room a 20 G intravenous access was achieved, preoxygenation started and standard monitoring was instituted. Baseline heart rate (HR) was 90 beats/min and blood pressure (B.P.) 180/76 mmHg. Anaesthesia was induced with Propofol (130 mg) and Fentanyl (100 mcg). Orotracheal intubation with cuffed 7.0 no. endotracheal tube was facilitated with Cisatracurium (10 mg). Anaesthesia was titrated with Sevoflurane 1.2-1.5% in air and oxygen (50%) to maintain bi-spectral (BIS) index around 40-45 on controlled ventilation on PCV-VG mode. There were no haemodynamic disturbances at induction. The surgeon proceeded with left parotidectomy with facial nerve monitoring. Patient was maintained on 1% Propofol infusion (10ml/hr) and Dexmedetomidine infusion (0.3 mic/kg/hr) without any further doses of muscle relaxant to facilitate nerve monitoring. As per the hospital protocol intravenous Glycopyrrolate 200 mcg was given on starting the Dexmedetomidine infusion. The surgery proceeded well with patient's all vitals stable with sinus rhythm, 60-70 beats/min, BP around 100/60 -120/70 mm of Hg, end tidal CO₂ (ETCO₂) ranging between 30-35 and SpO₂ >99%.

After approximately 85 min into surgery, while the deep lobe of the gland was being dissected; patient developed sudden bradycardia with HR reduced to 45. Surgeon was immediately asked to halt and IV glycopyrrolate 0.2 mg administered. But very rapidly the HR further

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plunged down to 34; IV atropine 0.6 mg were administered immediately. Heart rate remained unresponsive and further plunged down to 26 proceeding very rapidly to asystole. Cardiopulmonary resuscitation (CPR) was initiated with chest compressions. FiO₂ was made 100% and all anaesthetics were stopped. Adrenaline 1mg IV was administered and CPR continued. Eight minutes later i.e after 4 cycles of CPR of 2 min. each there was initially some pulseless electrical activity after a total of 4mg intravenous adrenaline; followed by return of spontaneous circulation (ROSC) with sinus tachycardia with HR 165 beats/min. Soon this developed into pulse less ventricular tachycardia which proceeded to ventricular fibrillation. Electrical defibrillation was performed with 200 J of energy. Post defibrillation there was sinus tachycardia with HR 140/min. with ST elevation, B.P. of 230/150 and SpO₂ 94%. Meanwhile, a central line in the right internal jugular vein, an arterial line (left radial artery) and a wide bore peripheral line was secured. Possible causes of asystole were ruled out by doing blood gases which ruled out any acidosis, there was no hypoxia due to any ventilatory malfunction and total dexmedetomidine drug infused was less than 0.5 mic/kg. Nitroglycerine infusion (25 mg/50 ml NS) was started to stabilize the B.P. As patient showed some window of stability, surgeon completed the last surgical steps with closure and dressing. Patient was then shifted to ICU on ventilatory support with a low dose noradrenaline infusion to maintain the blood pressure; with adrenaline effect weaning off and hypotension ensuing in. Total Intraoperative fluid input was 1500 ml, minimal blood loss with urine output of 150 ml. Postoperative 12 lead ECG showed ST-T depression of anterior-lateral leads. On 2D echo left ventricular ejection fraction (LVEF) was 40% with hypokinesia of basal part of inferior wall and basal anterolateral wall. Patient was assessed neurologically, and no neuro deficits were found on postoperative day one and she was extubated on postoperative day two without any need for any non-invasive ventilatory support. Meanwhile, all infusions were tapered based on haemodynamics. Patient was discharged on postoperative day six to continue follow up as an outpatient. Patient was asked to follow up after three months for repeat cardiac assessment.

Discussion

Trigemino-cardiac reflex (TCR) a known phenomenon during maxillofacial surgeries; is a brainstem reflex that manifests as sudden onset of hemodynamic perturbation in BP and HR during stimulation of any branches of the trigeminal nerve [6].

In TCR the sensory nerve endings of the trigeminal nerve send neuronal signals via the Gasserian ganglion to the sensory nucleus of the trigeminal nerve, which form the afferent pathway of the reflex arc. The afferent pathway then continues with the short internuncial nerve fibres in the reticular formation, which then connect with efferent pathway in motor nucleus of the vagus nerve [2]. Cardioinhibitory efferent fibres arising from vagal motor nucleus ending in the myocardium leads to negative chronotropic and ionotropic effects. TCR may occur with mechanical stimulation of any of the central or peripheral branches of the trigeminal nerve anywhere along its course. The reaction may subside with removal of the stimulus. But in some patients it may lead to severe bradycardia, asystole, and arterial hypotension with need of intervention. The risk

factors known to increase the incidence of TCR include hypercapnia; hypoxemia; light general anaesthesia; children; nature of the provoking stimulus and drugs like potent narcotic agents; beta-blockers; and calcium channel blockers [7].

Oral and maxillofacial surgical procedures can induce the development of this reflex, which leads to significant changes in the heart rate and sinus rhythms [8]. Incidence of either bradycardia or asystole is 1.6% in patients undergoing maxillofacial orthognathic or temporomandibular surgery [9].

As the parotid gland is innervated by the auriculotemporal nerve, which is derived from post branch of mandibular nerve (V3), our hypothetical etiology is that during deep lobe dissection the TCR was stimulated leading to severe bradycardia and asystole. Traction of the facial nerve during deep lobe dissection was also considered. But review of literature does not reveal dysrhythmias arising during facial nerve manipulation [10]. Patient was not paralyzed due to nerve monitoring and adequate depth of anaesthesia was maintained using intravenous and inhalational agents with a BIS 40-45. So any lighter plane of anaesthesia was also ruled out.

Our patient was haemodynamically stable 85 min. into the surgery. Heart rate was around 65/min. Patient suddenly developed severe bradycardia with drop in heart rate to 30/min. during deep lobe dissection which rapidly progressed to systole. Heart rate remained non-responsive to removal of surgical stimulus and to vagolytic drugs. Hence cardiac massage was started with intravenous epinephrine and 100% oxygen.

On review of literature, we found cases of severe cardiac dysrhythmias during parotidectomy but no case of asystole was reported. In these reported cases the bradycardia was unresponsive to vagolytic drugs but did response to either intravenous dopamine [11] or bolus ephedrine [10]. Neither of these cases had neuromonitoring and hence there was no need of intravenous infusions of dexmedetomidine and propofol. No bolus of dexmedetomidine drug was given; in fact total dose administered was only around 40mic in infusion at the rate of (0.3 mic/kg/hr) till the event. Dexmedetomidine is known to cause bradycardia but the dose we used was minimal and unlikely to cause severe bradycardia. Probably local anaesthetic infiltration of the nerve concerned could have prevented this incidence [12].

Conclusion

Parotidectomy with nerve monitoring is becoming increasingly popular. We would like to underline the possibility of TCR leading to severe bradycardia leading to asystole during handling of deep lobe of parotid gland. Bradycardia during this type of surgery should be taken seriously by being extra vigilant during deep lobe handling, continuous and meticulous monitoring of ECG for immediate recognition and effective management.

Clinical Message

Anaesthetists and surgeons should be aware of the potential and possibility of TCR during dissection of deep lobe of parotid gland and also be prepared to prevent or treat the possible life-threatening complications.

Abbreviations: TCR: Tri-Gemino-Cardiac reflex, HR: Heart Rate, BP: Blood Pressure, BIS: Bi-Spectral Index, Et CO₂: End tidal carbon dioxide, CPR: Cardio-pulmonary Resuscitation, ROSC: Return of spontaneous circulation, LVEF: Left ventricular ejection fraction

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his/her consent for his/her images and other clinical information to be reported in the Journal. The patient understands that his/her name and initials will not be published, and due efforts will be made to conceal his/her identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

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