Cardiac Dysrhythmia Complicating Total Parotidectomy

Smita Prakash*, Ameeta Sahni**, Charu Bamba***, Chintamani****, Anoop Raj Gogia†

Abstract

Sudden severe dysrhythmias during anaesthesia can be life-threatening for the patient. We describe a case in which ventricular dysrhythmias and severe bradycardia occurred during dissection and mobilization of the deep lobe of the parotid gland during total parotidectomy under general anaesthesia. We believe that these dysrhythmias were caused by a trigemino-vagal reflex similar to the oculocardiac reflex, but with afferent innervation from mandibular division of the trigeminal nerve. The case report is presented to illustrate a possible existence and importance of reflex bradycardic responses that may occur during surgical procedures involving the parotid gland.

Introduction

Severe dysrhythmias have been reported during maxillofacial surgery, but the literature is sparse in describing these events. We describe a case in which ventricular dysrhythmias and severe bradycardia occurred during dissection and mobilization of the deep lobe of the parotid gland during total parotidectomy under general anaesthesia. The case report is presented to illustrate a possible existence and importance of reflex bradycardic responses that may occur during surgical procedures involving the parotid gland, and to propose a hypothesis of etiology.

Case Report

A 60 yr old, non smoker, 49kg, normotensive male was scheduled for left total conservative parotidectomy. History and physical examination were unremarkable. Routine investigations, including ECG, were within normal limits. Premedication included oral alprazolam 0.25mg the night before and on the morning of surgery.

In the Operation Room, standard monitoring was instituted. Baseline heart rate (HR) was 85 beats/ min and blood pressure (BP) 124/80mmHg. Anaesthesia was induced with propofol (90mg) and morphine (4.5mg). Orotracheal intubation was facilitated with vecuronium. Anaesthesia was maintained with isoflurane 0.6% in nitrous oxide (66%) and oxygen, and controlled ventilation. The HR and BP remained stable at induction (75-85 beats/ min and 150/80-120/70 mmHg, respectively). The surgeon proceeded with left total conservative parotidectomy (facial nerve preserved). For the initial 90 minutes the patient’s condition was stable, with EtCO2 ranging between 28 and 35mmHg, SpO2 >98%, BP between 120/80 and 100/60 mmHg, and sinus rhythm of 70-80 beats/ min. During dissection of the deep lobe using rubber slings to gently lift the facial nerve trunk and branches, there was a rise in BP to 175/108 mmHg, HR 99 beats/ min. Fentanyl 40µg iv was administered and isoflurane concentration was increased to 2%. There was a further increase in BP to 186/120 mmHg with HR 108 beats/ min. Metoprolol (1mg iv) was administered and the dose repeated after 5min. BP decreased gradually over the next 10min and stabilized between 130/90 to 110/80mmHg with a HR of 70-80 beats/ min. Isoflurane concentration was decreased to 0.6%. Approximately 30minutes later, during manipulation and delivery of the deep lobe of the parotid gland, ventricular ectopic beats (VEB) at approximately 20 beats per min were seen on the ECG that degenerated soon after into ventricular bigemini and monomorphic ventricular tachycardia (runs of three or more VEBs). The BP recorded was 90/66 mmHg, HR 41 beats/ min. Surgical stimulation was immediately halted, oxygenation and normocapnia confirmed, and inhalational anaesthetic agents were discontinued. (FiO2 1.0). Lignocaine (75 mg iv) was administered. HR was 48 beats/ min with persisting VEBs. Lignocaine 50 mg iv was repeated. Pulse could be detected by right radial artery palpation and by pulse oximetry. ECG now showed nodal rhythm (HR 46 beats/ min) with occasional VEBs. Atropine (0.6 mg iv) was administered and repeated (0.6mg) after two minutes as HR had decreased further to 39 beats/ min. As HR remained unresponsive to atropine, adrenaline 50µg iv was administered. HR promptly increased to 94 beats/ min with normal sinus rhythm. BP was 110/76 mmHg, and SpO2 was 99%. At 5 min following administration of adrenaline, BP was 120/81 mmHg with HR 48-50 beats/ min, sinus rhythm. The entire episode lasted approximately 10 min; throughout this period, systolic BP (recorded every 1 min) remained >90 mmHg and SpO2 >99%. Anaesthetics were re-instituted and surgery proceeded. The remainder of the anaesthetic period was uneventful. At the end of surgery neuromuscular block was antagonized with neostigmine and glycopyrrolate, and the trachea was extubated when the patient was awake. The total duration of surgery was 4h. The patient made a safe recovery with no untoward haemodynamic events in the postoperative period. Cardiology consultation was obtained. Postoperative serial 12 lead ECGs and echocardiogram was normal. Trop T test was negative at 8h postoperative. Blood biochemistry, including serum electrolytes, was within normal limits. The patient was started on metoprolol 50mg bd. Biopsy proved the tumor to be adenoid cystic carcinoma. Postoperative radiotherapy was advised. The patient was discharged on postoperative day 3 to continue follow up as an outpatient.

Discussion

Cardiac dysrhythmias are associated with maxillofacial surgery. The reported incidence of either asystole or bradycardia in patients undergoing maxillofacial orthognathic or temporomandibular surgery is 1.6%. Episodes of bradycardia...
or asystole and other dysrhythmias have been reported during elevation of zygomatic fractures, surgical disimpaction of a fractured maxilla, temporomandibular joint irrigation, and mobilization of bony tissues for Le Fort I and II osteotomies. The exact nature of these dysrhythmias is not well documented but appears to be vagally induced.\(^1\)

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Surgical procedures performed in the region of the cranial nerve areas, especially the trigeminal nerve (CN V), can lead to vagally induced bradycardia and/ or other dysrhythmias.\(^2\)

The ocularocaridial reflex (OCR) is a well established phenomenon, mediated afferently via the ophthalmic division of the trigeminal nerve (V1) and efferently by the vagus nerve. Noxious stimulation of trigeminal divisions other than the ophthalmic division can also trigger life-threatening dysrhythmias. The trigeminocardiac reflex (TCR) is the sudden onset of dysrhythmias and hypotension during manipulation of any of the branches of the trigeminal nerve. The trigeminal nerve and cardioinhibitory vagus nerve constitute the afferent and efferent pathways of the reflex arc. Afferent pathways other than the ciliary nerves (OCR) most frequently involved in TCR are zygomaticotemporal and superior alveolar nerve (branches of the maxillary nerve) and the auriculotemporal and the inferior alveolar nerve (branches of the mandibular division of trigeminal nerve).\(^5\)

The proposed hypothesis for the etiology of dysrhythmias is as follows: the parotid gland is innervated by the auriculotemporal nerve, which is derived from the posterior branch of the mandibular nerve (V3). (Figure 1) Afferent input is carried by these nerves via the gasserian ganglion to the sensory nucleus of the trigeminal nerve, forming the afferent pathway of the reflex arc. Short internuncial fibers in the reticular formation connect with the efferent pathway in the motor nucleus of the vagus nerve.\(^6\) Cardioinhibitory efferent fibers arising from the motor nucleus of the vagus nerve terminate in the myocardium and lead to a negative chronotropic and inotropic response.

Though the facial nerve passes through the parotid gland, it does not innervate the gland. Traction on the facial nerve during deep lobe dissection may be implicated in the causation of these dysrhythmias. This appears unlikely as the facial nerve trunk and its branches were lifted gently. There was no postoperative facial nerve palsy. Review of the literature did not reveal any instance of dysrhythmias arising during facial nerve manipulation.

Our patient was not hypertensive and had no structural heart disease. Previous medical records documented normal blood pressure values. Intraoperative blood pressure was labile. The patient developed hypertension (186/120 mmHg) and tachycardia (108 beats/ min). Pain and light anaesthesia were considered. The hypertensive event was managed satisfactorily by increasing the concentration of isoflurane (up to2%), and administration of fentanyl (40µg) and metoprolol (intermittently, total 2mg). Ventricular dysrhythmias and severe bradycardia occurred suddenly in response to dissection, manipulation and traction of the deep lobe of the parotid gland. The haemodynamic parameters recorded just prior to occurrence of dysrhythmias were normal (HR 79 beats/ min, BP 112/80 mmHg), with isoflurane concentration at 0.6% in nitrous oxide (50%) and oxygen. Cessation of the surgical stimulus did not disrupt the dysrhythmia, bradycardia did not respond to atropine 1.2 mg, and we had to resort to administration of epinephrine 50 µg that resulted in restoration of normal sinus rhythm.

The ventricular tachycardia observed was monomorphic. We are unable to comment on the occurrence of R on T phenomenon, as the cardiac rhythm was observed on the monitor and an ECG recording could not be obtained.

Narcotics may augment vagal tone via their inhibitory action on the sympathetic nervous system.\(^3\) In the present case, fentanyl can be implemented in the causation of these dysrhythmias. The quantity of this drug 30min after iv administration in the present case, was, however, very small, and probably contributed negligibly to vagal activity. Metoprolol may have predisposed to the development of TCR in our patient. Intravenous administration of 0.5 mg and 1.5 mg metoprolol before the induction of anaesthesia and after tracheal intubation, respectively, had advantages with regard to improvement of haemodynamic stability and decreased incidence of myocardial ischaemia.\(^7\) However, this dose was associated with a high incidence of bradycardia caused by peritoneal traction.\(^2\)

In summary, we reported a case of cardiac dysrhythmias occurring during total parotidectomy possibly due to stimulation of the auriculotemporal nerve that mediated the trigeminovagal reflex. Clinically this report emphasizes that vagal reflexes can be elicited not only by afferent stimuli from V1 (OCR), but also by stimulation of other divisions of the trigeminal nerve. Clinicians must be aware of the potential for TCR during surgery on the parotid gland and be prepared to treat the possible life threatening consequences.

References