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Case Report

Vagal Stimulation Causing Intra-operative Cardiac Arrest, A Major Dilemma Whether to Proceed or to Defer Surgery: A Case Report

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Abstract

Vagal stimulation under general anesthesia can lead to life-threatening bradycardia and cardiac arrest. Here we present two cases of cervical carcinoma and tongue carcinoma, developing intra-operative cardiac arrest due to severe vagal stimulation, treated and resuscitated as per Advanced Cardiac Life Support protocol followed by clinical evaluation along with 12 lead electrocardiogram, arterial blood gases and screening echocardiogram, and completion of surgery. Also, we stress that intra-operative cardiac arrest is a dreadful consequence of severe vagal bradycardia leading to asystole, which needs to be recognized early and treated promptly followed by thorough clinical assessment and a decision regarding whether to proceed or abandon the surgery. This kind of cardiac arrest due to severe vagal stimulation which is resuscitated with minimal intervention may not call for deferring the surgical procedure if the evaluation done clinically together with tools of electrocardiogram, arterial blood gases, and screening echocardiogram are within the normal range.

Introduction

The incidence of cardiac arrest during general anesthesia is 0.03%.[1] During general anesthesia excessive vagal stimulation can cause life-threatening bradycardia progressing to cardiac arrest.[2] Painful stimulus of the bronchial, pharyngeal, laryngeal, esophageal mucosa and peritoneum stretch can increase the vagal activity leading to severe bradycardia and cardiac arrest. Even venous cannulation, neuraxial, and regional anesthesia techniques have been attributed to vasovagal syncope.[3] Prompt treatment with intravenous atropine, sympathomimetic drugs, fluids, and leg elevation may help in effective resuscitation of the patient.[4] A pacemaker may be considered for patients where severe bradycardia is frequent and non-responsive to medical treatment. We hereby describe two cancer patients developing vagal stimulus induced life-threatening bradycardia, leading to cardiac arrest in the intra-operative period, resuscitated with standard protocols followed by clinical evaluation together with tools of electrocardiogram (ECG), arterial blood gases, and echocardiogram (ECHO) followed by completion of surgery (Table 1), showing this can be a logical step forward. This approach needs further validation.

Case presentation

The study was performed in accordance with the ethical standards of the institution with which we are affiliated, and with the Declaration of Helsinki (as revised in 2013). Written informed consent was obtained from the patients for publication of this case report. Institutional ethical approval was not required for a case report. This report complied with the CARE guidelines and the completed CARE checklist is attached (Supplemental File 1).

Keywords: Intra-operative arrhythmia; Intra-operative cardiac arrest; Electrocardiogram; Peri-operative dilemma; ROSC; Cardiopulmonary resuscitation.

Abbreviations: ACLS, Advanced Cardiac Life Support; ASA, American Society of Anesthesiologists; ECG, electrocardiogram; ECHO, echocardiogram; HR, heart rate; MAP, mean arterial pressure; ROSC, return of spontaneous circulation.

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Table 1. The summary of clinical condition and management of the two cases described in the case report

Case	Age	Sex	Comorbidities	Surgery	Cause of Vagal stimulation leading to cardiac arrest	Post cardio pulmonary resuscitation				Postoperative outcome	One-year follow-up
						Clinical parameters	Arterial blood gases	Electrocardiogram	Echocardiography		
I	59	Female	Nil	Radical hysterectomy with bilateral pelvic lymph node dissection	Liver superior surface palpation	MAP > 75 on noradrenaline infusion 0.2 mcg/kg/min which was tapered to 0.05 mcg/kg/min	No significant abnormality	Normal sinus rhythm without ST changes	No regional wall abnormality, ejection function ~55%	Extubated and shifted to post anesthesia care unit with noradrenaline support of 0.05 mcg/kg/min. Repeat ECG and serial cardiac enzymes were unremarkable	No report of any cardiac sequelae
II	55	Female	Hypothyroid (Controlled)	Local excision of tongue with neck dissection	Neck dissection near carotid body	No requirement of inotropic support	No significant abnormality	Sinus tachycardia ~120 beats/min	No regional wall abnormality, ejection function ~55%	Patient extubated in recovery as per surgical plan. Serial ECG and cardiac enzymes levels were unremarkable	No report of any cardiac sequelae

Case 1

A 59-year-old female patient with cervical carcinoma was posted for radical hysterectomy with bilateral pelvic lymph node dissection under general anesthesia. She did not have any comorbidities except history of pulmonary tuberculosis three years back for which she got treated with anti-tubercular drugs and cured. Her biochemical investigations, ECG, and chest x-ray were all within normal limits. She was wheeled to the operating room, all standard American Society of Anesthesiologists (ASA) monitors were attached, and a thoracic epidural was placed in thoracic 9–10 interspace, followed by induction of general anesthesia with a 2 mcg/kg injection of fentanyl, a 2 mg/kg injection of propofol, and a 1 mg/kg injection of rocuronium along with endotracheal intubation with a 7.5 mm internal diameter cuffed endotracheal tube using standard protocols. Anaesthesia was maintained with sevoflurane (1–1.5%) in oxygen (50%) and nitrous oxide (50%) targeting a mean alveolar concentration of 1. In the intra-operative period, when the surgeons were palpating the superior surface of the liver, as per institutional surgical protocols for liver metastasis, the patient developed severe bradycardia with heart rate (HR) <40 beats/minute, treated immediately with intravenous 0.6 mg of atropine together with simultaneous withdrawal of stimulus but the patient went into asystole. Immediately cardiopulmonary resuscitation was started according to Advanced Cardiac Life Support (ACLS) protocol. With one dose of intravenous adrenaline (1 mg) together with 2 cycles of 120 seconds chest compressions, restoration of spontaneous circulation (ROSC) was achieved within 5 minutes. Patient was started on noradrenaline at 0.2 mcg/kg/minute to maintain mean arterial pressure (MAP) >75 mmhg. An arterial blood gas was sent which reported pH-7.33, pCO_2-43mmHg, pO_2-211mmHg, HCO_3-21.9 mmol/L and lactate-1.7 mmol/L. A 12-lead ECG and a screening ECHO were performed on table, which did not reveal any electrical or mechanical abnormalities in the heart (ECG: normal sinus rhythm without ST changes, ECHO: no regional wall abnormality, ejection function ~55%). It was decided to proceed with the surgical procedure. The remaining intra-operative period was uneventful, and noradrenaline was tapered to 0.05 mcg/kg/min. At the end of the procedure, the patient was extubated and shifted to the post-anesthesia care unit with noradrenaline support of 0.05mcg/kg/min. In the post-anesthesia care unit repeat ECG and serial cardiac enzymes did not reveal any abnormality. She was referred to a cardiologist post hospital discharge for further cardiological management. Upon a one-year follow-up, the patient did not report any cardiovascular sequel.

Case 2

A 55-year-old female patient was posted for wide local excision of tongue with neck dissection for right lateral border of tongue carcinoma. She was known to have hypothyroidism, well-controlled with levothyroxine 50 mcg once daily. The patient was shifted to the operating room and all standard ASA monitors were applied. General anesthesia was induced with with a 2 mcg/kg injection of fentanyl, a 1.5 mg/kg injection of propofol, and a1 mg/kg injection of vecuronium followed by nasotracheal intubation with 7.0 mm Portex cuffed endotracheal tube. Anesthesia was maintained with isoflurane (0.8–1%) in oxygen (50%) and nitrous oxide (50%) gases targeting a mean alveolar concentration of 1. In the intra-operative period during neck dissection, the patient developed bradycardia with a HR of 35–40 beats/minute. Immediately a 0.6 mg intravenous injection of atropine was given together with

withdrawal of surgical stimulus. The patient did not respond to the initial treatment and went into cardiac arrest. Cardiopulmonary resuscitation was initiated according to ACLS protocol. One cycle of chest compressions with a single 1 mg dose of adrenaline was given to achieve ROSC in 3 minutes. The patient did not require inotropic support following ROSC. We evaluated the patient with ECG (showing sinus tachycardia 120 beats/min), screening echocardiography (no regional wall abnormality, ejection function ~50–55%) and arterial blood gases analysis (found to be without any significant abnormality). The surgery was completed uneventfully. Recovery was uneventful. The patient was shifted with an endotracheal tube with T-piece for oxygen support (as per the pre-operative surgical plan) to the postanesthesia care room. Serial ECG and cardiac enzymes levels in the postanesthesia care room were unremarkable and the patient remained stable. The next morning the patient was extubated and shifted to a ward. She was referred to a cardiologist post-hospital discharge. The patient did not report any cardiac sequelae at one-year follow-up.

Patient's perspective

Both the patients were satisfied with the peri-operative care, the treatment and clinical outcome.

Discussion

Here we describe two cases of sudden severe bradycardia decompensated into cardiac arrest in middle aged females without any comorbidities during the intra-operative period. The reflex cardiovascular depression associated with vasodilation and bradycardia is known as vasovagal syncope, Bezold-Jarisch reflex, and noncardiogenic syncope.[3] Cardiac arrest in the operating room or post-anesthesia care unit is known to have better survival.[5] Cardiac arrest with initial rhythm of bradycardia were more likely to survive to hospital discharge and have favourable neurological outcomes.[6] Also, asystole was found to be most common rhythms in anesthesia-attributed cardiac arrest and was associated with relatively good prognoses.[5] These findings suggest that intra-operative cardiac arrest is due to reversible causes and is amenable to favourable outcomes with prompt corrective action.[7] Reflex bradycardia is one of the causes of intra-operative cardiac arrest among other causes, such as hemorrhage, cardiac issues, hypoxia, acid base imbalance, electrolyte imbalance, and drugs.[6,8,9] A study done by Rocco and Vandam observed a reflex fall in blood pressure with or without a decrease in heart rate on excessive stimulation of the upper abdomen parietal peritoneum.[10] Also, a study showed persistent association of decrease in blood pressure and variable changes in heart rate with mesenteric stretch due to inhibition of sympathetic activity and increased vagal tone.[11] Coeliac plexus reflexes have been attributed to these hemodynamic changes during abdominal surgeries.[1] During head and neck surgeries, severe bradycardia and cardiac arrest has been explained with different distinct mechanisms such as direct vagal stimulation, trigeminocardiac reflex, and baroreceptor reflex.[12] Increased vagal tone, athletic build, excessive tissue handling in neurovascular-rich areas, and fluid-electrolyte imbalance have been observed as risk factors for sudden intra-operative cardiac arrest-complicated bradycardia.

Widely available literature has established various procedures like laryngoscopy, neck surgery, abdominal palpation, laparoscopy gas inflation, electroconvulsive therapy, eye surgery *etc*.[1] as important predisposing factors for bradycardia which may end up as sinus arrest in the intra-operative period. Various drugs used in anesthesia such as vecuronium, atracurium, halothane, fentanyl, and succinylcholine have also been attributed to exaggerating vagal reflex.[1] Anticholinergic premedication may prevent this vagal reflex.[1] These findings were explained with an increase in vagal tone and a consequent reduction in cardiac contractility and output.[1]

The available literature does not address the issue of whether to abandon or to proceed with the surgery in the event of successful resuscitation post intra-operative cardiac arrest. This report highlights such two cases where patients were resuscitated following severe bradycardia resulting in cardiac arrest and evaluated with clinical parameters, arterial blood gas values, ECG and screening echocardiography. The proposed surgeries were completed successfully with no peri-operative adverse events.

We recommend the reassessment of the patient once resuscitation is done by means of clinical assessment, ECG, screening echocardiography, and arterial blood gas. If the reassessment does not show any significant abnormality then we recommend to proceeding with the surgery in cases of semi-urgent or urgent surgeries and to postponing the surgery in case of purely elective surgeries.

Clinical perspectives

Future research should focus on randomized control trials or studies reflecting the feasibility of proceeding with the proposed surgery after resuscitation from intra-operative cardiac arrest. There is a paucity of literature regarding consensus on whether to proceed or to defer the surgery after intra-operative bradycardia progressing to cardiac arrest.

Conclusions

Two cases of intra-operative vasovagal cardiac arrest have been described. Vagal bradycardia can happen at any point in the intra-operative period and requires swift and precise treatment; nonresponders may end up with vaso-vagal arrest which usually responds well with cardiopulmonary resuscitation measures. Following revival, the feasibility of proceeding with surgery needs to be evaluated and should be considered on a case-by-case basis, though further research is required for evidence-based recommendations.

Supporting information

Supplementary material for this article is available at https://doi.org/10.14218/ERHM.2021.00063.

Supplemental File 1. CARE Checklist.

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Conflict of interest

The authors declare no conflicts of interest.

Author contributions

AK contributed to the clinical concept; AK and AG performed the anesthesia described in this report and wrote and revised the manuscript; FR helped revise the manuscript; UH wrote and revised the manuscript. All authors have made a significant contribution to this study and have approved the final manuscript.

Ethical statement

The study was performed in accordance with the ethical standards of the institution with which we are affiliated and with the Declaration of Helsinki (as revised in 2013). Written informed consent was obtained from the patients for publication of this case report. Institutional ethical approval was not required for a case report.

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