Case Report

Cardiac arrest during alveolar recruitment manoeuvre under general anaesthesia for laparoscopic surgery

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Summary

A 67-year-old woman with no history of cardiovascular disease, undergoing an elective laparoscopic cholecystectomy, experienced severe bradycardia and cardiac arrest immediately following an alveolar recruitment manoeuvre under general anaesthesia. Prompt cardiopulmonary resuscitation restored cardiac output within 2–3 min. Postoperatively, she remained stable and was discharged following 24 h of monitoring. The cardiac arrest was likely triggered by vagal nerve stimulation and activation of intrinsic cardiac reflexes by the alveolar recruitment manoeuvre. The event emphasises a rare, but significant, risk of the routine management of pulmonary atelectasis.

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Introduction

Atelectases affect 85–100% of patients undergoing general anaesthesia with tracheal intubation [1, 2]. Atelectasis has been demonstrated to occur after just 5 min of general anaesthesia [1]. The four main explanations for atelectasis formation are ciliary dyskinesis, increased alveolar oxygen concentration, ablation of the sigh/deep breath and decreased availability of active alveolar surfactant [3, 4]. Recruitment of collapsed alveoli by recruitment manoeuvres improves alveolar gas exchange and arterial oxygenation. These manoeuvres reduce postoperative pulmonary complications associated with considerable morbidity and mortality [3, 5]. In patients without cardiovascular compromise, alveolar recruitment manoeuvres are considered safe and are conducted as a standard procedure before termination of general anaesthesia for laparoscopic surgeries [3, 6].

We report a case of cardiac arrest secondary to alveolar recruitment in a woman with no history of cardiovascular disease undergoing elective laparoscopic cholecystectomy.

Report

A 67-year-old woman was scheduled to undergo an elective laparoscopic cholecystectomy after multiple admissions with upper abdominal pain due to gall bladder polyps in the span of 1 year. The patient was 170 cm tall and weighed 65 kg. She was considered an ASA physical status 1 with no history of regular medications. She was an ex-smoker who stopped smoking 40 years ago and had no history of alcohol consumption. Thirteen years ago, she had been treated for breast cancer with a lumpectomy and axillary clearance, chemotherapy and radiotherapy. At the time of the cholecystectomy, there was no residual complaint after the treatment and no further concerns for malignancy.

Standard intra-operative monitoring was performed with 3-lead ECG, non-invasive blood pressure measurement (NIBP) and pulse oximetry. Train of four monitoring was used to assess neuromuscular blockade. The patient's pre-operative observations were: NIBP 160/90 mmHg, heart rate 96 beats.min^{-1} and oxygen saturations of 97%. Anaesthesia was induced and maintained using total intravenous anaesthesia with propofol and remifentanil. Tracheal intubation was facilitated with rocuronium and a 140 mg bolus of propofol. Continuous infusion of phenylephrine was started after 30 min due to a slow but continuous decrease in blood pressure. During the surgery, pneumoperitoneum with up to 12 mmHg was established and ceased before suturing of the port holes. During surgery, the patient was in a head-up position until the gall bladder was removed when the position was changed to neutral. The surgical procedure went as planned and total operative time was 83 min.

Before termination of anaesthesia, an alveolar recruitment manoeuvre was performed according to our departmental guidelines with peak airway pressure of 35 cmH_2O for 20 s. Immediately after the recruitment manoeuvre the patient became increasingly bradycardic progressing rapidly to cardiac arrest. The ECG sequence was later confirmed with vital capacity monitor recording. Cardiopulmonary resuscitation (CPR) was initiated immediately by the surgical team. An in-hospital cardiac arrest call was activated and the anaesthetist in charge was called to the operating room. During CPR, automated external defibrillator pads were placed on the chest. Cardiac rhythm was analysed after the first round of CPR and electrical activity was observed with a rate of 75–80 beats.min^{-1}. Return of spontaneous circulation was established in 2–3 min. The emergence of anaesthesia was uneventful, and the patient's trachea was safely extubated. In the postoperative period, the patient was transferred to the intensive care unit for further investigations and observation. The patient remained stable with acceptable vital parameters and without any chest pain in the postoperative period. At 1.5 and 6 h following the cardiac arrest, blood was sampled for troponin T measurement and a 12-lead ECG was performed. Troponin T was 16 ng.l^{-1} after the event and remained unchanged after 6 h. The two 12-lead ECGs showed sinus rhythm with a heart rate of 67 and 81 beats.min^{-1}, respectively, normal axis and transmission, narrow complexes and no T-wave abnormalities. The patient was subsequently transferred to the cardiology department for further observation and was discharged after 24 h of uneventful cardiac telemetry.

Discussion

We describe an episode of severe bradycardia leading to cardiac arrest after alveolar recruitment manoeuvre under general anaesthesia. Since the patient had no cardiovascular conditions pre-operatively and no abnormalities were observed postoperatively, we consider that the cardiac arrest was provoked by the alveolar recruitment manoeuvre. The recruitment manoeuvre can lead to stimulation of the vagus nerve by overstretching of the peripheral airways and in addition, can lead to a reduction in preload secondary to an increase in intrathoracic pressure. These events can trigger intrinsic cardiac reflexes such as the Bezold-Jarisch reflex, reverse Bainbridge reflex and pacemaker stretch reflex, which can influence cardiac rhythm. The Bezold-Jarisch reflex, triggered by ventricular mechanoreceptors, increases parasympathetic activity, leading to bradycardia and reduced peripheral resistance. The reverse Bainbridge reflex also causes bradycardia by detecting decreased atrial stretch, which signals the brainstem to increase parasympathetic outflow. The pacemaker stretch reflex, involving the sinoatrial node, adjusts the heart rate based on the mechanical stretch of pacemaker cells, with increased stretch raising the heart rate and decreased stretch leading to bradycardia [7].

In addition to the recruitment manoeuvre, several factors could have contributed to impaired venous return and/or bradycardia in this case. The surgery was conducted laparoscopically with increased intrathoracic pressure compared to a laparotomy, due to the pneumoperitoneum. While increased intra-abdominal pressure can affect venous return, the pneumoperitoneum ceased before the recruitment manoeuvre, making it unlikely to have influenced venous return at the time of the bradycardia. Another potential factor for impaired venous return was hypovolemia. The patient consumed two cups of fluid 3 h before surgery and received 600 ml of fluid during the procedure, with no blood loss. We estimated the fluid requirement during surgery to be approximately 2 ml.kg^{-1}.h^{-1} suggesting that hypovolemia is not a likely contributor to the bradycardia. The patient was anaesthetized with remifentanil, which can induce bradycardia. At the time of cardiac arrest, the patient had been receiving remifentanil for approximately 1 h and maintained a stable heart rate of 65–75 beats.min^{-1}. Although propofol can cause bradycardia during induction, it is not typically associated with bradycardia during continuous infusion. No reversal agents for neuromuscular blockade or anticholinergic drugs were administered during the procedure or CPR.

A severe reduction in heart rate leading to asystole due to the above-mentioned mechanisms is possible, but extremely rare. Although rare, it has been established through several case reports that asystole is a risk during spinal anaesthesia, possibly through the mechanisms discussed [8].

To the best of our knowledge, this is the first report of a cardiac arrest following alveolar recruitment manoeuvre during laparoscopic surgery. We want to highlight the risk of this potentially devastating complication of a routine procedure.

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References

1. Brismar B, Hedenstierna G, Lundquist H, Strandberg A, Svensson L, Tokics L. Pulmonary densities during anesthesia with muscular relaxation—a proposal of atelectasis. *Anesthesiology* 1985; **62**: 422–8.
2. Lundquist H, Hedenstierna G, Strandberg A, Tokics L, Brismar B. CT-assessment of dependent lung densities in man during general anaesthesia. *Acta Radiologica* 1995; **36**: 626–32.
3. Hartland BL, Newell TJ, Damico N. Alveolar recruitment maneuvers under general anesthesia: a systematic review of the literature. *Respiratory Care* 2015; **60**: 609–20.
4. Hedenstierna G, Rothen HU. Atelectasis formation during anesthesia: causes and measures to prevent it. *Journal of Clinical Monitoring and Computing* 2000; **16**: 329–35.
5. Shander A, Fleisher LA, Barie PS, Bigatello LM, Sladen RN, Watson CB. Clinical and economic burden of postoperative pulmonary complications: patient safety summit on definition, risk-reducing interventions, and preventive strategies. *Critical Care Medicine* 2011; **39**: 2163–72.
6. Rothen HU, Neumann P, Berglund JE, Valtysson J, Magnusson A, Hedenstierna G. Dynamics of re-expansion of atelectasis during general anaesthesia. *British Journal of Anaesthesia* 1999; **82**: 551–6.
7. Lacey JR, Dubowitz JA, Riedel B. Asystole following spinal anaesthesia: the hazards of intrinsic cardiac reflexes. *Anaesthesia Reports* 2022; **10**: e12198.
8. Kopp SL, Horlocker TT, Warner ME, et al. Cardiac arrest during neuraxial anesthesia: frequency and predisposing factors associated with survival. *Anesthesia and Analgesia* 2005; **100**: 855–65.

