Clinical Case Report

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Severe bradycardia caused by the deviation of the laryngeal mask airway Supreme

A case report

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Abstract

Rationale: Classic laryngeal mask airway (LMA) has long been used for airway management. The LMA Supreme is a modified single-use version of the LMA Proseal, but it still remains some deficits such as the instable positioning that lead to easily sliding and the mask bowl full of air might lead to the reduced blood flow of the internal carotid artery. The carotid sinus is a baroreceptor that responds to the stretching of the arterial wall. Manual pressure of the carotid artery at the upper margin of the sternocleidomastoid muscle provoked bradycardia and hypotension.

Patient concerns: A previously fit and well 42-year-old woman presented with breast fibroma on the left side. No other disease history could be recorded. Her family history was negative for neuromuscular and autoimmune disease.

Diagnoses: The patient suffered from a severe bradycardia and hypotension when the LMA showed a shift. We presented with a hypothetical that the dislocated LMA may cause carotid sinus syndrome (CSS).

Interventions: The patient's heart rate (HR) gradually rosed up as soon as the LMA adjusted back to the normal position.

Outcomes: The patient was comfortable in the post anesthesia care unit (PACU) and had no adverse sequelae.

Lessons: The position of LMA Supreme should be confirmed throughout the surgery especially for the ones who has changed the position and the head was covered by surgical drapes.

Abbreviations: BP = blood pressure, bpm = beats per minute, CAS = carotid artery stenting, CSM = carotid sinus massage, CSS = carotid sinus syndrome, ECG = electrocardiogram, ETI = endotracheal intubation, HR = heart rate, LMA = laryngeal mask airway, NIBP = noninvasive blood pressure, OLP = oropharyngeal leak pressure, PACU = post anesthesia care unit, RR = respiratory rate, SpO_2 = pulse oximetry.

Keywords: bradycardia and hypotension, carotid sinus syndrome, the LMA Supreme

1. Introduction

Classic laryngeal mask airway (LMA) has long been used for airway management. Use of the LMA in general anesthesia has advantages over the endotracheal intubation (ETI) in decreasing patients' subjective and objective voice symptoms, reducing the duration of symptoms, and relieving the laryngopharyngeal symptoms.[1,2] The LMA Supreme is a modified single-use version of the LMA ProSeal and has improved features including an anatomically curved airway tube, a drain tube located within the center of the airway tube, and an oval-shaped cuff.[3,4] However, some deficits of it still remain, such as the instable positioning that lead to easily sliding; the mask bowl full of air might lead to the reduced blood flow of the internal carotid artery and superficial nerve damage.[5,6]

The carotid sinus is a baroreceptor that responds to stretching of the arterial wall such as provoked by an increase in intra-arterial pressure. Manual pressure of the carotid artery at the upper margin of the sternocleidomastoid muscle provoked bradycardia and hypotension. Carotid sinus syndrome (CSS) consists of a cardiovascular symptom complex resulting from excitation of a hyperactive carotid sinus reflex.[7,8]

It seems there is no correlation between the LMA and CSS. But here is a case where the patient suffered from a severe bradycardia and hypotension when the LMA showed a shift, as the heart rate (HR) rapidly decreased from 75 beats per minute (bpm) to 30 bpm accompanied with blood pressure (BP) reduced from

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110/75 mmHg to 50/30 mmHg. We present with a hypothetical that the dislocated LMA may cause CSS.

2. Case presentation

A previously fit and well 42-year-old woman presented with breast fibroma on the left side 1 month ago. She did not have any other significant medical and surgical history. She denied taking any drugs and alcohol drinking, was a non-smoker. No other disease history could be recorded. Her family history was negative for neuromuscular and autoimmune disease. She was scheduled for a resection of the breast fibroma.

The patient was kept fasting for 8 hours preoperatively and did not receive any premedication. In the operating room peripheral venous access was secured and monitoring with electrocardiogram (ECG), noninvasive blood pressure (NIBP) and pulse oximetry (SpO_2). Her preoperative vital signs were SpO_2 99% on room air, BP of 111/69 mmHg, HR at 74 bpm, and respiratory rate (RR) of 20 bpm. Preoperative airway assessment revealed interincisor gap was greater than 4 cm, head/neck movement was greater than 90°, ability to prognathic, a thyromental distance of 6.5 cm, body weight of 60 kg. Induction of anesthesia was accomplished with administration of IV fentanyl 200 μg, propofol 150 mg, and cisatracurium 10 mg; and a size 3 Supreme LMA was promptly inserted without difficulty, an intracuff pressure of 80 cm H_2O in the adult-sized LMA Supreme has been recommended to obtain a higher oropharyngeal leak pressure (OLP),[9,10] and the LMA's proper placement was confirmed by positive end-tidal CO_2 for tracing and bilateral breath sounds.

Adopted the mode of volume-control ventilation with the breath parameters: FiO_2 100%, oxygen flow 2 L/min, tidal volume 8 ml/kg, RR 12 bpm, I/E 1: 2, maintaining $PETCO_2$ 35 to 40 mmHg. Anesthesia maintenance: continual intravenous pump injection of propofol 6 to 8 mg/kg/h and intermittent intravenous injection of fentanyl and cis-atracurium to maintain the anesthesia. After the surgery started, the operating table was adjusted to be inclined to the right, with the operating side higher than the other side. During the surgery, the vital signs were stable, with BP remaining at 97 - 112/60 - 75mmHg, HR at 75 to 85 bpm, SpO_2 100%. The whole operation was successful and cost 35 minutes, with only a small amount of blood loss.

At the completion of surgery, the intravenous anesthetic was stopped. After the surgeon removed the surgical drapes, the HR of the patient suddenly dropped from 75 bpm to 30 bpm and the BP dropped from 110/75 mmHg to 50/30 mmHg. 0.5 mg Atropine and 5 mg dopamine were immediately injected. As it had no effect, another 0.5 mg of Atropine was injected again in 1 minute. The HR and BP still stayed low level. No more anomaly was observed in SpO_2 and $PETCO_2$. It was spotted that the patient's head turned to the right (the operation table was adjusted to be inclined to the right, as shown in Fig. 1), and the LMA was seriously deflected to the right due to the traction of the surgical drapes (as shown in Fig. 1). The mask bowl full of air was right oppressing the thyroid cartilage level of the left neck.

The patient's HR gradually rose up as soon as the LMA adjusted back to the normal position. For the next 10 minutes her HR remained greater than 100 bpm, and BP rose to normal level. The patient quickly met extubation criteria and the LMA was removed uneventfully.

She was comfortable in the post anesthesia care unit (PACU) and had no adverse sequelae. In order to confirm our hypothesis, the carotid sinus massage (CSM) was proceeded when her preoperative vital signs were SpO_2 100% on room air, BP of 127/61 mm Hg, HR at 87 bpm. The result was positive, her HR turned down to 41 bpm, BP reduced by more than 50 mm Hg, and a fit of dizziness came over her.[11]

After being observed in the PACU for an hour the patient returned to the ward. The patient was reluctant to do further examinations to confirm our diagnosis on the first postoperative day. Two days later the patient was discharged from our hospital.

3. Discussion

The carotid sinus, located just superior to the bifurcation of the carotid artery controls the HR and BP via receptors located

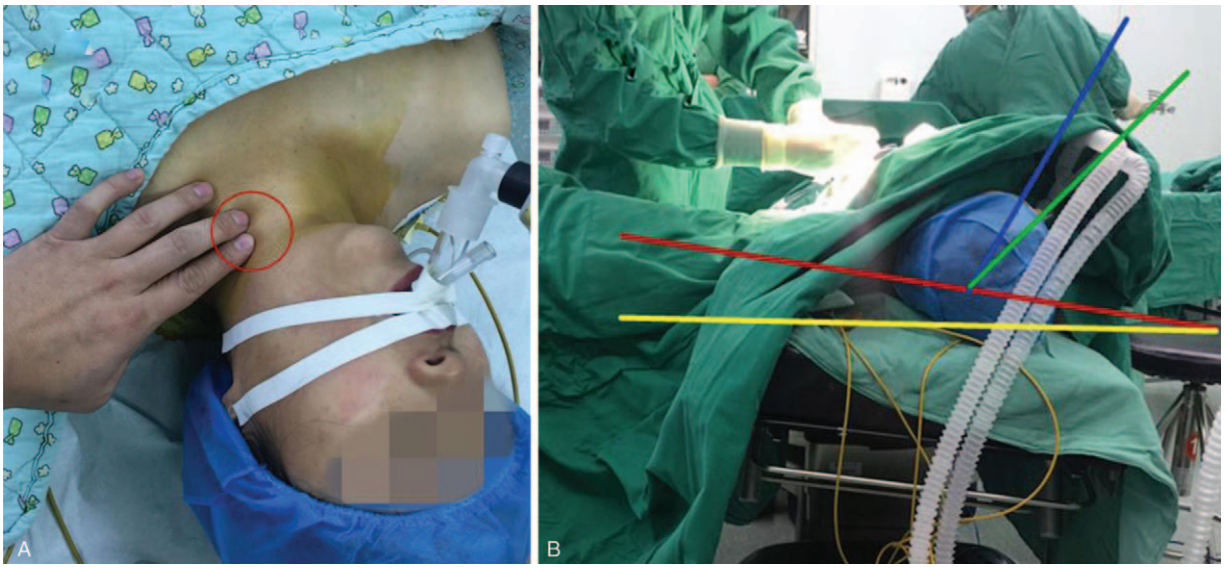

Figure 1. A. The patient's head turned to the right side, the laryngeal mask airway (LMA) largely deflected (Red circle: the left edge of LMA was palpable on the left side of patient's neck) to the left and stimulated the carotid sinus directly. B. The yellow line was horizontal line; the red line was patient's horizontal line; the blue line was patient's head sagittal line; the green line was LMA's sagittal line.

within the arterial adventitia. The afferent limb of the carotid sinus reflex begins with these receptors.[12] Myelinated nerve fibers emerge from these menisci as spiral fibers and from the sinus nerve of Hering, a branch of the glossopharyngeal nerve. Other fibers may follow the hypoglossal nerve, vagus nerve or cervical sympathetic nerves to the medulla. The efferent fibers descend in the vagus and cervical sympathetic nerves to the cardioinhibitory and vasomotor centers.[13,14] CSS consists of a cardiovascular symptom complex resulting from excitation of a hyperactive carotid sinus reflex. Weiss and Baker[15] classified three types of responses leading to carotid sinus stimulation. As per current guidelines, CSM was performed first on one and then on the other carotid sinus, with a positive on either side indicating hypersensitivity.[16] Previous work has shown that CSM is quite reproducible,[17] and that the sensitivity increases in the upright position.[18] The massage was performed for 5 seconds while the subject was tilted upright on a tilt table at 70°[18] as per previous CSS studies.[19] The vasodepressor and cardioinhibitory response to CSM was defined as the maximum change in systolic BP and RR interval observed during both massages. Cardioinhibitory response, which is expressed as bradycardia and asystole, vasodepressor response, characterized by profound hypotension without bradycardia, and cerebral response, which is an interference with the circulation of the ipsilateral cerebral hemisphere circulation.[20]

Considering the obvious delayed effect of Atropine and the rapid recovery of the HR and BP after the position of the LMA was corrected, it was considered that it was the oppression of the mask bowl on the carotid sinus that caused the CSS. The reason of bradycardia in this case was assumed to be the oppression of carotid sinus from the LMA Supreme. This mechanoreceptive baroreceptor respond to stretch or distortion of it, thereby causing bradycardia and/or hypotension as an efferent response.

There are so many reasons to cause this phenomenon, but we have ruled out. Bradycardia and hypotension are commonly encountered adverse effects of total intravenous anesthesia with propofol and opioids. The hemodynamic instability caused by these anesthetics regimen could be critical.[21] These adverse effects are at least partly the result of the centrally mediated vagotonic and/or sympatholytic action of opioids[22,23] and the centrally mediated sympatholytic action of propofol.[24] Although muscle relaxants are designed to specifically block nicotinic cholinergic receptors at the neuromuscular junction, many alter HR by binding to muscarinic cholinergic receptors. It is well recognized that vecuronium induces bradycardia because of its direct action or the increase in vagal tone induced by surgical procedures.[25,26] However, Cisatracurium, the one we used nondepolarizing muscle relaxant in this case, does not induce significant cardiac effects.[27] Other drugs may cause bradycardia, either alone or in combination with others, such as the β- blockers, Digitalis and NDHP-CCBs but which this patient had not received. In this case, bradycardia and hypotension occurred very suddenly, and the intravenous anesthetic was stopped before all these happened.

There are some operations, which give rise to bradycardia and hypotension, are familiar to everybody. The oculocardiac reflex and biliary-cardiac reflex are well-known phenomenons mediated via the trigeminal nerve (afferent limb) and the vagus nerve (efferent limb). However, no correlating operations had been done when the emergency occurred in this case.

CSS is common in clinical work. There are cases reporting that patients with head and neck tumors, including thyroid cancer, cervical lymphoma, and internal aneurysms are accompanied with it. Radiotherapy is also reported as a risk factor as it can lead to the tissue fibrosis.[28,29] The carotid artery stenting (CAS) has also been widely reported the trigger of hemodynamic disorders.[30] But the case that the deviation of the LMA causes the CSS has never been reported before.

There are some tips that we can learn from this case: 1. A comprehensive assessment is a key element to protect the safety of patients, the one who accompanies with history of faint should be paid enough attention to. 2. The position of LMA Supreme should be confirmed throughout the surgery especially for the ones who has changed the position and the head was covered by surgical drapes. 3. The reinforced LMA and ETI can get better performance in the operations that the patients need to change position. 4. CSS might be a possible reason when inexplicable severe bradycardia with or without the decrease of BP occur. Releasing the carotid sinus from the oppression is the key to rescue. 5. Why bradycardia and hypotension occurred at the end of the surgery? We considered that the anesthesia depth was too low to inhibit nervous reflex due to stopping all anesthetics more than 5 minutes.

4. Conclusion

This case report describes what we believe to be the first reported case of the CSS induced bradycardia and hypotension due to the LMA Supreme displaced.

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