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CASO CLÍNICO

Third-degree Atrioventricular Block and Asystole Induced by Double-Lumen Tube Intubation: A Rare but Risky Airway Management Complication

Bloqueio Auriculoventricular de Terceiro Grau e Assistolia Induzidos por Intubação com Tubo de Duplo Lúmen: A Abordagem de uma Complicação Rara e Arriscada

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Afiliação
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Keywords
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Palavras-chave
Abordagem da Via Aérea; Anestesia Geral; Bloqueio Auriculoventricular; Intubação Intratraqueal

ABSTRACT

Single-lung ventilation with a double lumen tube remains essential in thoracic surgery. Though these tubes insertion can be associated with multiple complications, from airway trauma or pulmonary shunt, potentially lethal complications can also occur, as we describe in the following case report. The authors believe the double lumen tube insertion in this case report induced a third-degree atrioventricular block and underline the lack of case reports and literature evidence regarding this potential complication. To our knowledge, no studies to date have reported third-degree A-V block occurring with double lumen tube insertion.

RESUMO

A ventilação de pulmão único com recurso a tubo de duplo lúmen ocupa um papel primordial na cirurgia torácica. Embora a inserção destes tubos possa estar associada a complicações variadas, desde trauma da via aérea ou *shunt* pulmonar, podem também ocorrer complicações potencialmente fatais, como descrevemos neste caso. Os autores consideram que a inserção do tubo de duplo lúmen no caso descrito induziu um bloqueio auriculoventricular de 3º grau e alertam para a escassez de casos clínicos descritos na literatura acerca desta complicação. Os autores desconhecem a existência de casos clínicos prévios a descrever bloqueio auriculoventricular de 3º grau com inserção de tubos de duplo lúmen.

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INTRODUCTION

Lung isolation with accomplished one-lung ventilation plays a key role in airway management for thoracic surgery.[1] Double-lumen tubes (DLT) used in these procedures can lead to serious problems such as airway trauma, pulmonary shunt or dislodgment of the endobronchial cuff. Our hypothesis is that DLT placement induced a third-degree atrioventricular (AV) block, though current absence of published case reports on this complication. Complete AV block is characterized by widening of QRS complexes and a ventricular rate between 30-40 bpm, regardless of atrial rate. This results in serious hemodynamic instability and is refractory to vagolytic medication. Clinical practice guidelines recommend early pacemaker implantation in intermittent documented bradycardia.[2]

We describe a case of two separate episodes of complete AV block after endotracheal intubation with a 39Fr DLT in a patient proposed for thoracic surgery with no previous cardiac medical history or medication. A permanent pacemaker was placed and recovery was complete. Informed consent for publication was obtained from the patient.

CASE REPORT

The patient was a 74-year-old man diagnosed with lung adenocarcinoma proposed for left lobectomy. He was a heavy smoker with chronic obstructive pulmonary disease, but denied previous cardiac symptoms, including syncope, tachycardia, or chest pain under at least 4 METs of daily activity and cardiac medical treatment. He had a preceding

epidural anesthesia without airway manipulation and without complications for transurethral resection of the bladder. Airway assessment revealed no signs of difficult airway management based on patient anatomy and computed tomography scan.

Preoperative exams were all normal, including a resting 12-lead electrocardiogram (ECG) with normal sinus rhythm and a heart rate of 67 beats/minute (Fig. 1).

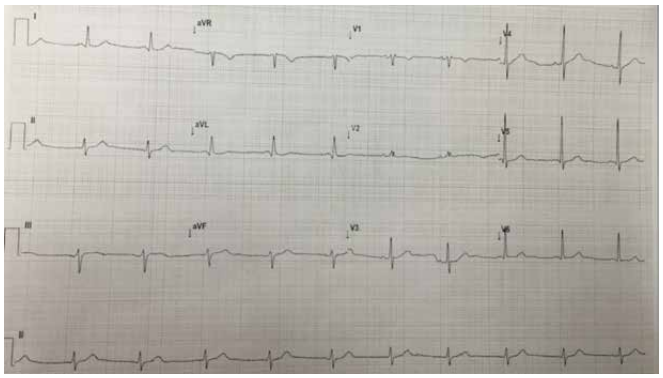

Figure 1. Patients' preoperative 12-lead electrocardiogram with normal sinus rhythm and a heart rate of 67 beats/minute

On the surgery day, no premedication was used and he had a heart rate of 72 beats/minute with normal sinus rhythm and a blood pressure of 140/96 mmHg. He was monitored with noninvasive arterial blood pressure evaluated every 3 minutes, continuous ECG, pulse oximetry and bispectral index (BIS). General anesthesia was induced with fentanyl 0.15 mg, propofol 150 mg and rocuronium 50 mg. Under stable hemodynamic conditions and BIS of 47, the patient was intubated with a Robertshaw 39Fr left-sided DLT by direct laryngoscopy without difficulties. Immediately after tracheal cuff insufflation and confirmation of proper tube position by fiberoptic bronchoscopy, the heart rate decreased to <40 bpm with a rhythm suggestive of 3rd degree AV block . This bradycardia with no response to 1 mg atropine, rapidly evolved to asystole. After 2 cycles of advanced life support the patient reverted to spontaneous circulation and displayed supraventricular tachycardia (186 bpm). Amiodarone infusion of 300 mg was administered over 10 minutes, but after this he was hemodynamically unstable requiring noradrenaline perfusion to maintain mean arterial pressure (MAP) above 65 mmHg. A radial arterial line was placed and the DLT was exchanged for a single-lumen 8.0 mm ID endotracheal tube (SLT), by direct laryngoscopy without further rhythmic events. The patient was transported to the intensive care unit (ICU) and extubated a few hours later. Serial troponin measurements showed an initial rise to 115 ng/L with a fall to 52 ng/L in the day after the event. He was proposed for a cardiac catheterization which displayed no signs of coronary lesions and the patient´s surgery was rescheduled for 1 week later.

On the day of surgery, no premedication was administered. Standard monitoring systems, including ECG, pulse-oximetry, and non-invasive blood pressure, as well as a radial arterial line, were placed in the operating room before the anesthetic induction. Initial patient´s ECG pattern remained sinus rhythm at a rate of 72 bpm. General anesthesia was induced with fentanyl 0.15 mg, propofol 170 mg and rocuronium 50 mg. The pattern of the ECG remained unchanged with a blood pressure of 89/50 mmHg. The patient was intubated by direct laryngoscopy with a 39Fr left-sided DLT without difficulties and immediately after proper placement confirmation, the ECG pattern converted to complete AV block with heart rate of 36-42 bpm and a blood pressure of 59/44 mmHg. A perfusion of noradrenaline was initiated (5 $\mu g min^{-1}$) to maintain MAP above 65 mmHg. Surgery was again postponed, the DLT was exchanged to SLT and the patient transferred to the ICU. Later, at the ICU a 12-lead ECG revealed normal sinus rhythm with a heart rate of 99 bpm, normal axis and no ischemic changes. The patient was extubated in the ICU with stable vital signs and proposed for implementation of a permanent pacemaker in VDDR mode.

After 2 weeks, surgery was rescheduled and the patient was submitted to a left lobectomy under general anesthesia and DLT placement. The pacemaker was activated after intubation and the patient remained on pacemaker rhythmic during the intra-operative period. Both the anesthesia and surgery were uneventful, with a smooth anesthetic recovery with stable hemodynamics.

DISCUSSION

In all patients with cardiac conduction abnormalities, our main concerns rely on the relative risk for progression to complete AV blockade, requiring urgent pacing, due to hemodynamic instability and cardiac arrest risk in the absence of escape rhythms, as occurred with our patient.[3] Paroxysmal AV block is thought to be related to vagotonic stimulation, coronary artery disease, degenerative changes in the cardiac conduction system, electrolyte disturbances, surgical manipulation and drugs, among others[4,5] (Table 1).

Usually, the anatomic levels of blockade can be distinguished by QRS complex sizes'. In our case, the patient developed a broad QRS complex in both situations, suggesting a location distal to the bundle of His[2], confirmed by the absence of response to atropine. Nonetheless, for definite diagnosis, electrophysiological studies are needed.

Our patient had no preoperative risk factors for development of intraoperative heart block. He had no preexisting heart disease or chronic medication that prolongs AV node or AV conduction. ECG and laboratory tests were normal, excluding underlying conduction system disease and electrolyte or metabolic imbalance.

Table 1. Possible causes for AV block

Possible causes for AV block
1. Idiopathic (50% cases)
- Lenegre's disease - Lev's disease
2. Vagotonic stimulation
- Direct laryngoscopy and endotracheal intubation - Baroreceptor reflex activation with hypertension - Rectal/cervical dilation - Oculocardiac and Bezold-Jarish reflex - Peritoneal retraction
3. Drugs
- AV node blockers: beta-blockers, non-dihydropyridine calcium channel antagonists, digitalis, antiarrhythmic drugs (most commonly amiodarone, quinidine, procainamide, disopyramide) - Drugs related to vagal stimulation: anticholinesterases - Drugs related to prolonged QT interval: antiarrhythmic drugs, droperidol, ondansetron, metoclopramide - Anesthetic drugs: propofol, volatile anesthetics, opiates, succinylcholine
4. Imbalances
- Blood gases - Hypothermia - Electrolyte disturbances: hyponatremia, hypocalcemia, hyperkalemia - Hyperthyroidism and hypothyroidism - Surgical manipulation (open heart surgery, TAVI*, catheter ablation, percutaneous closure of ventricular septal defects)
5. Structural diseases with degenerative change in conduction system
- Coronary artery disease - Inflammatory and infiltrative heart diseases: cardiac neoplasms, trauma, endocarditis, cardiomyopathies and myocarditis, congenital heart diseases
6. Hereditary disease
- Familial AV block: transmitted as an autosomal dominant trait. (genetic locus at chromosome 19q13 and chromosome 3p21, where the cardiac sodium channel, SCN5A, is encoded)
*TAVI - transcatheter aortic valve implantation

Intraoperative risk factors associated with development of AV heart block include drugs, increased vagal tone or autonomic imbalance. We ruled out hypoxia-induced bradyarrhythmia by pulse oximetry (the minimum peripheral oxygen saturation recorded was 95%) and adequate ventilation revealed by capnography. Possible anesthetic causes of AV node blockade include medications frequently used, such as droperidol, ondansetron and metoclopramide with potential to prolong the QT interval, and also propofol[4] and opioids, which have vagotonic properties. Notwithstanding, evidence is still lacking and studies suggest that anesthetics only have significant negative dromotropic effects on AV conduction when used in high doses (propofol >8 mg/kg).[6]

Endotracheal intubation and endobronchial intubation usually cause similar cardiovascular and arousal responses determined by transient hypertension and tachycardia and increased BIS values.[8] It is suggested that DLT intubation using video laryngoscopy can help reduce this cardiovascular response to classic laryngoscopy intubation.[9] Bronchial blockers combined with conventional SLT as an alternative to DLT may also provoke less hypertension.[10] Nevertheless, direct laryngoscopy and endotracheal intubation is also a known cause of vagal stimuli,[7] despite few ancient reports about bradycardia or asystole. A vagal reflex occurs by activation of afferent parasympathetic nerve fibers during stimulation of the lower pharynx, larynx, trachea and epiglottis. The magnitude of which depends on the duration of intubation and the force exerted on the tongue during laryngoscopy, which were both insignificant in our patient. The DLT, with its increasing diameter, has an even greater risk of autonomic imbalance. In both episodes of AV block, withdraw of the double lumen tube and insertion of SLT, was followed by recovery, supporting a cessation of vagal stimulation.

We therefore accept that the most possible cause for the AV block and asystole in our case report was the vagotonic stimulation by DLT intubation, since other causes were excluded. Unfortunately, we did not consider the use of bronchial blockers as an alternative approach to assure single-lung ventilation. Therefore, we can not figure out if cardiovascular response would have been different using this strategy.

In order to properly diagnose a patient with AV block, examinations include an ECG, 24-hour Holter monitoring, echocardiography and electrophysiologic testing. In our case only coronary angiography was made and did not reveal significant results, excluding structural disease as a possible cause for disturbances on the cardiac conduction system. As an electrophysiological study was not done, it is unknown whether the AV block in our patient was explained by a direct electrophysiological effect or an autonomic one. A postoperative echocardiography was done and revealed no structural relevant heart diseases.

Management of AV block includes pharmacological and electrical therapy. Guidelines for management recommend implantation of a permanent pacemaker in all asymptomatic patients (class IIa recommendation) and symptomatic patients with type III AV block (class I recommendation).[11] Our multidisciplinary team decided for a permanent pacing, but alternatively a provisory pacemaker could have been used in this intermittent documented bradycardia associated with a identified possible causal etiology. This measure would have avoided postponing of the second surgery but not the risk of further episodes in the case of future urgent or emergent surgical reintervention.

In conclusion, it is the objective of the authors to report and highlight the event of endotracheal intubation and particularly double-lumen tube intubation as an intraoperative risk factor for atrioventricular heart block and asystole. Airway management demands appropriate vigilance and awareness of common potential complications on airway and breathing, but must comprise other rare and unexpected circulatory and cardiac complications.

Ethical Disclosures
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