CLINICAL INFORMATION

Intermittent left bundle branch block – reversal to normal conduction during general anesthesia

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Abstract
Background and objectives: Transient changes in intraoperative cardiac conduction are uncommon. Rare cases of the development or remission of complete left bundle branch block under general and locoregional anesthesia associated with myocardial ischemia, hypertension, tachycardia, and drugs have been reported. Complete left bundle branch block is an important clinical manifestation in some chronic hypertensive patients, which may also be a sign of coronary artery disease, aortic valve disease, or underlying cardiomyopathy. Although usually permanent, it can occur intermittently depending on heart rate (when heart rate exceeds a certain critical value).
Case report: This is a case of complete left bundle branch block recorded in the preoperative period of urgent surgery that reverted to normal intraoperative conduction under general anesthesia after a decrease in heart rate. It resurfaced, intermittently and in a heart-rate-dependent manner, in the early postoperative period, eventually reverting to normal conduction in a sustained manner during semi-intensive unit monitoring. The test to identify markers of cardiac muscle necrosis was negative. Pain due to the emergency surgical condition and in the early postoperative period may have been the cause of the increase in heart rate up to the critical value, causing blockage.
Conclusions: Although the development or remission of this blockade under anesthesia is uncommon, the anesthesiologist should be alert to the possibility of its occurrence. It may be benign; however, the correct diagnosis is very important. The electrocardiographic manifestations may mask or be confused with myocardial ischemia, factors that are especially important in a patient under general anesthesia unable to report the characteristic symptoms of ischemia.
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PALAVRAS-CHAVE
Defeitos da condução
cardíaca;
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Bloqueio completo de
ramo esquerdo;
Anestesia geral

Bloqueio intermitente de ramo esquerdo – reversão para condução normal durante anestesia geral

Resumo

Justificativa e objetivos: Alterações transitórias da condução cardíaca no intraoperatório são pouco frequentes. Foram reportados raros casos de desenvolvimento ou remissão de bloqueio completo de ramo esquerdo sob anestesia (geral e locorregional), associados a isquemia do miocárdio, hipertensão, taquicardia e fármacos. O bloqueio completo de ramo esquerdo é uma manifestação clínica importante em alguns hipertensos crônicos, pode também significar doença arterial coronária, doença valvular aórtica ou cardiomiopatia subjacentes. Embora habitualmente permanente, pode ocorrer na forma intermitente dependente da frequência cardíaca (quando a frequência cardíaca excede determinado valor crítico).

Relato de caso: Este é um caso de bloqueio completo de ramo esquerdo registrado no pré-operatório de cirurgia urgente que reverteu para condução normal no intraoperatório sob anestesia geral após diminuição da frequência cardíaca. Ressurgiu, de forma intermitente e dependente da frequência cardíaca, no pós-operatório imediato, acabou por reverter novamente à condução normal de forma sustentada durante vigilância em unidade semi-intensiva. O estudo com marcadores de necrose muscular cardíacos foi negativo. A dor do quadro cirúrgico urgente e pós-operatório imediato pode ter estado na origem da subida da frequência cardíaca até ao valor crítico e causado bloqueio.

Conclusões: Embora o desenvolvimento ou a remissão desse bloqueio sob anestesia sejam incomuns, o anestesiologista deverá estar alertado para a possibilidade da sua ocorrência. Pode ter caráter benigno, contudo o diagnóstico correto é muito importante. As manifestações eletrocardiográficas podem ser confundidas com ou encobrir isquemia miocárdica, fatos de especial importância num paciente sob anestesia geral incapaz de referir sintomatologia característica de isquemia.

Introduction

A complete left bundle-branch block (LBBB) development or remission in patients under anesthesia is uncommon.[1–4] The heart rate-dependent intermittent block may be benign[5,6]; however, the correct diagnosis is very important. The electrocardiographic manifestations may be confused with or mask a myocardial ischemia,[5,7] facts of particular importance in a patient under general anesthesia unable to describe the characteristic symptoms of myocardial ischemia.

We report a case of complete preoperative left bundle branch block that reverted to normal conduction after a decrease in heart rate during surgery under general anesthesia and which resurged intermittently and dependent on heart rate in the immediate postoperative period.

Clinical case

A 73-year-old female patient suspected of iatrogenic perforation of the sigmoid colon during colonoscopy with polypectomy was referred for urgent exploratory laparotomy. Pre-anesthetic evaluation revealed a medical history of bronchial asthma, hypertension, type 2 diabetes mellitus, gastritis, hiatal hernia, obesity, and depressive syndrome. Anesthetic history included general anesthesia for hysterectomy and locoregional anesthesia for varicose vein surgery, with no known complications. Physical examination revealed only abdominal pain, with no signs of peritoneal irritation or significant dysfunction of other organs or systems. Laboratory tests showed leukocytosis ($21,100\,mm^{-3}$), with no other distinct changes in blood count, renal and hepatic functions, or ionogram. Arterial Blood gas analysis showed no changes. ECG showed sinus rhythm with 82 bpm and a pattern compatible with complete left bundle branch block (LBBB) (Fig. 1). The patient remained without chest pain and with blood pressure and heart rate (HR) values within normal range from hospital admission to arrival in the operating room.

Due to the urgent nature of the surgery and the hemodynamic stability, it was decided not to postpone the surgical procedure for further study of electrocardiographic alterations.

On admission to the operating room, the patient presented with blood pressure of 123/69 mmHg, HR 83 bpm, peripheral oxygen saturation 96%, and LBBB. Under the American Society of Anesthesiologists monitoring standards, induction of general anesthesia with propofol bolus ($2\,mg\cdot kg^{-1}$) and infusion of remifentanil ($0.5\,\mu g\cdot kg^{-1}$ for one minute, followed by $0.1\,\mu g\cdot kg^{-1}\cdot min^{-1}$) and neuromuscular block with rocuronium ($0.6\,mg\cdot kg^{-1}$), after which the orotracheal intubation was performed with a cuffed endotracheal tube of 7.5 mm internal diameter, uneventfully. An episode
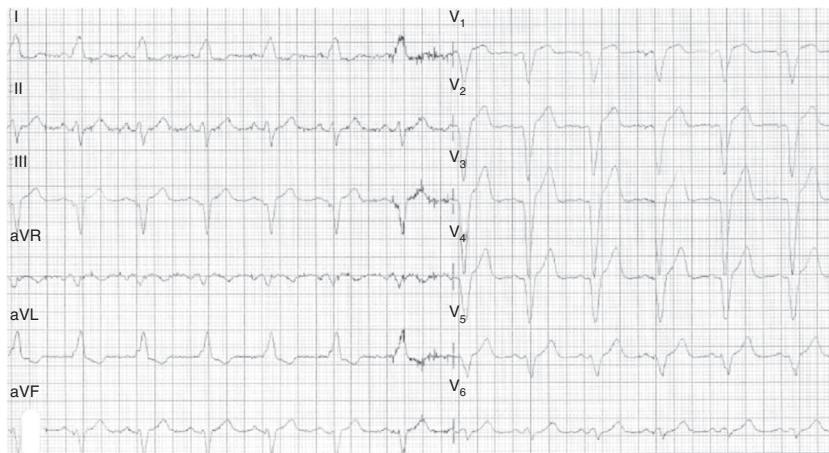
Figure 1   Preoperative 12-lead ECG with a LBBB-compatible pattern.
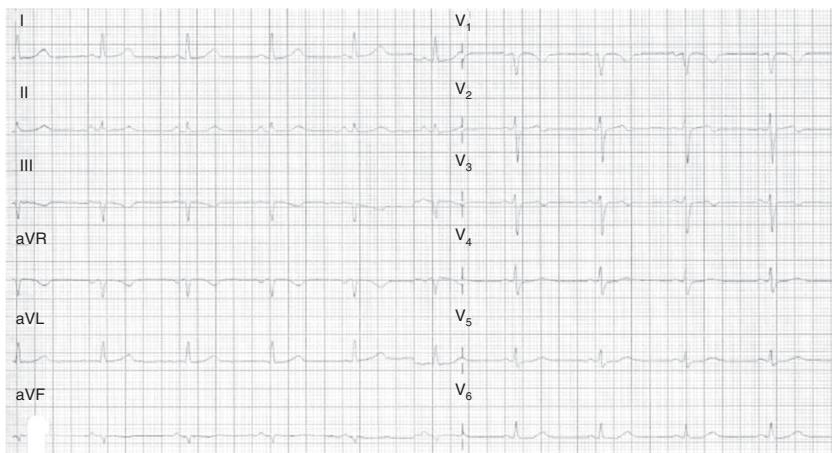
Figure 2   Intraoperative 12-lead ECG with normal conduction, without LBBB.

of bronchospasm followed the administration of intravenous hydrocortisone (100 mg) and salbutamol (400 μg) and ipratropium bromide (40 μg) via expander chamber. For maintenance of anesthesia, oxygen, air, and sevoflurane (FiO$_2$ 35%, end-tidal sevoflurane concentration of 1.8%) and remifentanil infusion (0.1–0.2 μg·kg^{-1}·min^{-1}) were used. Ventilation was mechanically controlled. Analgesia was performed with paracetamol (1 g) and tramadol (150 mg) and antiemetic prophylaxis with intravenous dexamethasone (4 mg at induction) and ondansetron (4 mg at the end of surgery).

Intraoperatively, after about 30 min of anesthesia, LBBB reverted to normal conduction, at which point HR reached 74 bpm after a gradual reduction, thus remaining during the 2 h of surgery. Mean arterial pressure varied between 73 and 80 mmHg and HR between 65 and 74 bpm during this period. At the end of the surgery, but still before the anesthesia reversal, a 12-lead ECG was performed, which confirmed absence of LBBB (Fig. 2).

The patient was then awakened, and the neuromuscular block reversed with sugammadex (200 mg).

Upon arrival at the post-anesthesia care unit (PACU), the monitoring showed blood pressure of 125/66 mmHg; HR 86 bpm and ECG with LBBB pattern, which was maintained intermittently and dependent on HR $\geq$ 75 bpm over 2 h of surveillance. During this period, two boluses of intravenous morphine (2 mg) were administered for pain management. There were no changes in blood pressure during the blockade periods.

After a joint evaluation by the PACU anesthesiologist and the physician responsible for the semi-intensive care unit, the patient was admitted to this unit for clinical, laboratory, and electrocardiographic surveillance. The patient remained asymptomatic throughout the stay in this unit, the markers of cardiac muscle necrosis were negative, and serial ECGs showed no blockade. An emergency cardiologist evaluated the results and established the diagnosis of intermittent LBBB, with no need for particular care. The patient was taken then to the general surgery care unit, and the remaining postoperative period was uneventful.

Discussion and conclusions

Right or left bundle-branch block is related to conduction impairment and electrical impulse delayed propagation in the bundle of His corresponding branch.[8]

Branch block may occur in several situations. LBBB is an important clinical manifestation in some chronic hypertensive subjects. It may also imply coronary artery disease, aortic valve disease, or underlying cardiomyopathy.[9] In young adults it is often a benign finding,[6,10] but in the elderly it may represent progressive myocardial degeneration affecting the conductive system.[9] Delayed intraventricular conduction may also be caused by extrinsic factors that decrease conduction, such as hyperkalemia or drugs (antiarrhythmics, tricyclic antidepressants, and phenothiazines).

Impaired conduction in the bundle of His (or bundle branches) is electrocardiographically translated into a QRS interval prolongation ($\geq$120 ms in complete branch block); QRS vector is directed to the myocardial region where depolarization is delayed.[8] Left bundle branch block changes the early and late stages of ventricular depolarization. Septal depolarization abnormally occurs from right to left, and the main QRS vector is oriented to the left and backwards.[8] Thus, a LBBB generates complex, elongated, and predominantly negative QS in the right precordial leads and complex and fully positive R (absence of physiological Q-waves) in lead V6.[8] In addition to these depolarization changes, the blockade is also characterized by secondary changes in ventricular repolarization. Although depolarization and repolarization cause deflections of opposite polarity at the cellular level, under normal conditions the QRS complex and the T-wave have the same polarity, as depolarization and repolarization waves occur, at least in part, in opposite directions in the heart.[7] In LBBB the depolarization and repolarization sequences are altered, so that the two waves have almost parallel directions.[7] Moreover, the delayed left bundle branch conduction associated with changes in the depolarization and repolarization sequence prevents the occurrence of the refractory period of most myocytes simultaneously and allows the occurrence of a delayed depolarization of the left ventricle lateral wall and early repolarization of the right ventricle at the same time.[7] On the ECG, these changes are shown by the QRS complex and T-wave discordance (T-wave typically has opposite polarity to that of the last QRS deflection) and by ST segment elevation or depression.[7] These secondary changes in repolarization caused by the LBBB may be confused with or obscure primary changes in repolarization, such as acute myocardial infarction (AMI).[5,7]

The blockade is usually permanent, but it may occur transiently when reverts to normal conduction—even temporarily—or intermittently when both the blockade and normal conduction are observed on the same ECG trace.[1,3] A large proportion of patients with intermittent block eventually develops a permanent block.[5,11] The cause of this intermittent block may be organic or functional. The exact mechanism of an intermittent blockade is unclear, but it appears to result from anatomical or physiological disruptions in a conductive branch either by ventricular hypertrophy or dilation,[3,9] or by functional or neurogenic depression, with or without conductive tissue underlying lesions.[3,9] The intermittent form was also associated with some drugs[2,4,5] and tachycardia.[1]

The HR-dependent intermittent branch block is the most commonly reported. It is related to a defect in intraventricular conduction, which occurs only when HR exceeds a certain critical value usually within the physiological values.[5,12] Increased HR and decreased RR interval may cause downward electrical impulses to find one of the conductive branches still in their refractory period and generate the blockage.[9] It persists until HR is slower than that critical one that caused the blockage; the RR interval at which the block occurs is 80–170 ms shorter than that in which conduction returns to normal (zone of linking).[5] A transition from normal conduction to branch block is sudden and may occur even with HR variations of only 1 or 2 bpm,[5,9] and the critical value depends on HR variation velocity: rapid accelerations cause blockage with lower HR, rapid decelerations cause reversal with higher HR.[5,9] Electrophysiological studies have shown that cells from the conductive branches of patients with HR-dependent LBBB have prolonged refractory periods.[5] With higher HR, their membrane potential does not decrease normally, and the hypopolarization they present provides a delayed electrical impulse conduction.[5]

Transient changes in cardiac conduction during the intraoperative period are uncommon.[1–4] There have been rare reports of LBBB development or remission under anesthesia (general and locoregional), which have been associated with myocardial ischemia, hypertension, tachycardia, HR variations without tachycardia, and drugs (lidocaine, trimethaphan, lithium, and atropine).[1,2,4,5,9] In some cases it was not possible to identify the cause of intermittent blockade.[3]

Acute pain leads to a typical neuroendocrine response, proportional to its intensity, mediated by the endocrine and sympathetic nervous systems. Its main cardiovascular effects are generalized vasoconstriction with increased peripheral vascular resistance and increased cardiac contractility and frequency.

In the case described here, the patient, although without known history of coronary artery disease, aortic valvular disease, or cardiomyopathy referred as one of her conditions an arterial hypertension with several years of evolution. Given the severe acute abdominal pain caused by the hollow viscera perforation presented by the patient preoperatively, as well as an immediate postoperative pain initially difficult to control, it is likely that the HR values measured, although within normal values, were above the baseline values. Intraoperatively, the sympathetic tone reduction by the analgesic drugs and anesthetics decreased the HR values, which remained between 65 and 74 bpm, possibly to a value similar or even lower than the patient's baseline value. In the semi-intensive care unit, the postoperative pain was already controlled and the HR was probably close to the baseline values. Thus, we hypothesized that the HR may have been intermittently above the patient's critical HR (75 bpm) and capable of triggering a LBBB in a heart with probable changes caused by diabetes and hypertension. Cardiac investigation showed no acute pathological condition.

We therefore consider a case of intermittent rate-dependent LBBB with a blockade present in the preoperative and early postoperative periods and intraoperative normal conduction.

Although frequently associated with AMI, certain studies point to a benign nature of this type of blockade, not associated with ischemia or changes in ventricular function.[5] However, the electrocardiographic ST-T changes associated with LBBB can be confused with or mask changes caused by AMI,[5] as the electrocardiographic interpretation of the

repolarization gradient between normal and abnormally perfused myocardium is the basis for the diagnosis of AMI.[7] This is an especially important fact for patients under general anesthesia, as they are unable to discribe the characteristic symptoms of myocardial ischemia. The diagnosis of AMI in this context, although challenging, is possible. Currently, the most accurate and reliable electrocardiographic signal for the diagnosis of AMI in the presence of LBBB is ST-segment elevation, which represents the sum of the repolarization primary changes from infarction and the changes secondary to the blockade.[7] In 1996, Sgarbossa[13] described a validated scoring system for the electrocardiographic diagnosis of AMI in patients with LBBB. Diagnosis is considered positive if three points are reached based on the following three criteria: ST-segment elevation of at least 1 mm in a lead with QRS complex and T-wave concordant (5 points); ST-segment depression of at least 1 mm in lead V1, V2, or V3 (3 points); ST-segment elevation of at least 5 mm in a lead with a QRS complex and T-wave discordant (2 points).[13] In 2012, Smith et al.[14] developed a modification to the Sgarbossa criteria based on the ST-segment deviation ratio with discordant S-wave or R-wave amplitude. Sgarbossa system is highly predictive of AMI in the presence of LBBB. The modified criteria appear to be useful for diagnosis; however, the manual calculation of ST/S or ST/R is time consuming.[7]

HR-dependent LBBB has also been confused with slow ventricular tachycardia and inappropriately treated.[1] Thus, the correct diagnosis of this particular change in cardiac conduction is of particular importance. Intraoperative maneuvers were described that, by triggering or interrupting the blockade through the HR alteration (valsalva, carotid massage, administration of atropine, neostigmine or propranolol), aided the diagnosis of HR-dependent LBBB. However, provocative maneuvers should be used with caution in patients with cardiovascular, cerebrovascular, or atrioventricular node disease.

Although the development or remission of this blockade under anesthesia is uncommon, anesthesiologists should be alerted to the possibility of its occurrence. In addition to using the Sgarbossa criteria and/or the maneuvers described above, it is advisable to perform a Holter monitoring after surgery.

Conflicts of interest

The authors declare no conflicts of interest.

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