Spontaneous Remission of Left Bundle Branch Block Immediately After Denitrogenation With Inhalation of 100% Oxygen Prior to Anesthetic Induction

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1. Introduction

Left bundle branch block (LBBB) is an independent risk factor for cardiac mortality. LBBB is related to the delayed spread of excitation to the left ventricle. It is usually associated with coronary artery disease or hypertensive heart disease. LBBB is usually sustaining but may emerge transiently or intermittently. In patients with LBBB, left ventricular (LV) dilatation, reduced LV ejection fraction (EF) and defective septal perfusion are usually observed even in the absence of ischemic heart disease. Since it causes intraventricular asynchrony, LBBB may eventuate in the development of cardiac remodeling and heart failure. The hemodynamic effect, caused by LBBB-induced dysynchrony, may enhance myocardial oxygen demand. Spontaneous remission of LBBB occurring 15 minutes following general anesthesia via endotracheal tube intubation has been reported. Here, we report two patients who initially presented with silent LBBB, but then saw transient transformation of the abnormal electrocardiogram (ECG) into normal sinus rhythm shortly after denitrogenation with inhalation of 100% oxygen by face mask during induction of anesthesia.

2. Case Reports

2.1. Case 1

A 60-kg, 56-year-old male was scheduled to undergo a right tympanoplasty for chronic otitis media. His past medical history was unremarkable apart from chronic middle ear infection for years. He was a heavy smoker, with daily consumption of one pack of cigarettes for 35 years. Physical examination was unremarkable; there were no signs of cardiovascular...
Preoperative dysrhythmias

Disease, but his ECG showed complete left bundle branch block (CLBBB). He denied chest pain, syncope or symptoms of congestive heart failure and lived a sedentary life. Preoperative laboratory findings, chest radiographs, complete blood count and urinalysis were within normal limits.

In the operating room, routine monitoring (ECG, oxygen saturation and noninvasive blood pressure) was established. No premedication was given. Prior to induction, the heart rate was 70 beats per minute and blood pressure was 163/84 mmHg. Lead II ECG tracing on the monitor showed CLBBB. While he was being denitrogenized with 100% oxygen via face mask just prior to induction, the CLBBB tracing suddenly converted to normal sinus rhythm. After interruption of mask ventilation with oxygen, the CLBBB reverted again without changes in heart rate and blood pressure (Figure 1). Several attempts at cessation of oxygen breathing and resumption of oxygen breathing in alternation were made and the phenomenon remained the same. Although the vital signs remained stable during the attempts and the patient did not complain of any symptoms, it was thought that myocardial ischemia might be responsible for this phenomenon. Anesthesia and the operation were called off in order to arrange noninvasive cardiologic tests.

Transthoracic echocardiography showed dilated left atrium and LV, adequate LV systolic function with EF of 52%, and possible impaired LV relaxation. Treadmill exercise test did not induce chest pain and significant change of the arrhythmia, and the pre-existing ST-T changes were not meaningfully suggestive of cardiac ischemia. The T1-201 myocardial perfusion SPECT study revealed decreased uptake of radionuclides in the apical, anterior and inferior segments of the LV, suggestive of myocardial ischemia in these segments (Figure 2). The patient was discharged and advised to be followed-up at our cardiologic outpatient clinic and treated with aspirin 100mg, isosorbide mononitrate 60mg and rosuvastatin 10mg per day.

One year later, he was brought to our emergency room and admitted to the intensive care unit because of acute myocardial infarction.

2.2. Case 2

A 78-kg, 81-year-old retired fisherman suffering from cholelithiasis and cholecystitis was scheduled for laparoscopic cholecystectomy. He had no history of cardiovascular disease except for mild hypertension (140/80 mmHg) that was untreated. Physical examination showed a 2/6 pansystolic murmur heard over the left sternal border, but he denied any chest discomfort. Preoperative chest X-ray and laboratory studies, including full blood cell count, blood sugar, blood urea, serum creatinine, serum electrolytes and liver function tests, were all within normal limits.

ECG taken in the operating room showed CLBBB; heart rate was 68 beats per minute and blood pressure was 152/81 mmHg. No premedication was given. While he was being denitrogenized with pure 100% oxygen via a face mask prior to anesthetic induction, the CLBBB tracing suddenly reverted to normal sinus rhythm. After a momentary interruption of pre-oxygenation, the CLBBB tracing reappeared without signs of tachycardia, bradycardia or hemodynamic change. Shortly after, a cardiac rhythm of LBBB pattern developed. It emerged and reverted to sinus rhythm alternatively with cessation and resumption of oxygen breathing. The anesthesia and operation were cancelled and the patient was sent to the recovery room for close observation. He was advised to undertake further noninvasive cardiologic investigations.

A cardiologist was called and transthoracic echocardiography showed septal wall hypokinesis with LVEF of 57%, and dilated left atrium and LV with impaired LV relaxation. No further serial cardiac examinations such as treadmill exercise and myocardial perfusion scan were suggested because the patient was elderly. The patient was regularly followed-up at our cardiologic outpatient clinic.

Figure 1 Lead II electrocardiogram tracing of Case 1. The arrow indicates the beginning of oxygenation.
3. Discussion

The causes of LBBB are various. It is primarily related to delayed spread of ventricular excitation. Clinical and pathologic studies suggest that systemic hypertension and coronary artery disease are the most common causes. Valvular and/or congenital heart disease, cardiomyopathy and some less common causes have also been described. LBBB is usually sustained, but may occur transiently or intermittently. Of patients with LBBB, 12% apparently have normal heart beats except for abnormality of cardiac conduction. Though several studies have tried to identify a relationship between the extent or location of coronary disease and the presence of LBBB, most have been unsuccessful. Among patients with known coronary disease, no association was found between any particular location of coronary artery stenosis or LV wall motion abnormalities, indicating that LBBB is not the result of an infarction in the area around the proximal conduction system. Except for elevated blood pressure, Eriksson et al also found no association between any of the major cardiac risk factors and LBBB, consistent with an earlier study that found that coronary risk factors did not predict subsequent development of LBBB.

The primary prevention study in Goteborg, Sweden, which ran for nearly 30 years, demonstrated that patients with LBBB had a markedly increased risk of dying from coronary disease, and two-thirds of these deaths occurred outside of hospitals, indicating that it is probably a fatal arrhythmia. The depolarization phase of LBBB is, by definition, prolonged. The prolongation of the vulnerable repolarization phase, in combination with an increased number of premature ventricular beats secondary to ischemic heart disease, might expose the individual to an increased risk of sudden ventricular tachyarrhythmia. Thus, individuals with LBBB have a substantially increased risk of sudden death due to coronary causes, probably due to malignant tachyarrhythmias. Further, the presence of LBBB was also strongly associated with future high-degree atrioventricular block, requiring pacemaker instalment. In an older follow-up study in Manitoba, it was shown that the most frequent clinical cardiovascular event observed after development of LBBB was sudden death without previous clinical evidence of ischemic heart disease. The 5-year incidence of sudden death as the first manifestation of heart disease was 10 times greater in people with LBBB than in those without it. Therefore, we should be alert for the presence

Figure 2  The T1-201 myocardial perfusion SPECT study of Case 1 revealed decreased uptake of radioactivity in the apical, anterior and inferior segments of the left ventricle in stress study.
of LBBB. The general concern about LBBB is the development of complete heart block during anesthesia and its presence to indicate myocardial disease, e.g. ischemic disease sufficient to affect conducting tissue.

From the point of view of the mechanoeenergetic effect that LBBB brings about, intraventricular asynchrony, observed in the majority of patients with LBBB, plays a causative role and is an independent predictor of severe cardiac events in heart failure patients. In patients with LBBB, the lateral wall represents the site of latest activation and contraction. Its contraction and the increased intraventricular pressure caused by the contraction of the earlier activated myocardial walls overlap each other and results in distending the lateral wall. Thus, regional wall stress becomes highest in the lateral segments. The early septal contraction against a relaxed left ventricle reduces septal workload, however, at the expense of a higher stress on the lateral wall due to late excitation. This alteration leads to a reduction in LVEF and to the redistribution of myocardial shortening and perfusion from the septum to the LV lateral wall. Because the patterns of redistribution of myocardial work and blood flow are similar and remain constant over time, LBBB leads to LV dilatation and asymmetric hypertrophy in the long run. Small but significant asymmetrical hypertrophy after 16 weeks of LBBB can be observed, and other studies in patients with LBBB have reported a more pronounced LV dilatation (EF, −75%) and hypertrophy (EF, −40%), and more reduced LV (EF, −25%) relative to a control group. Such ventricular remodeling is known to contribute to the development of heart failure.

The regional hemodynamic findings in LBBB reflect the delayed conduction and asynchronous contraction of the septal and lateral walls of the ventricular myocardium. Due to the associated electromechanical alterations, the lateral wall has been shown to exhibit the highest myocardial oxygen consumption (MVO_2) and myocardial blood flow (MBF). However, in the present study, MVO_2 and MBF tended to be lowest in the septal wall. It is supposed that the paradoxical septal contraction and a reduced EF are responsible for the low septal MVO_2 and MBF. The results of these studies suggest that regional differences and substantial inhomogeneities of MVO_2 and MBF are induced by LBBB.

The impact of LBBB on myocardial perfusion is often blurred by underlying cardiac disease. In a study by Wieneke et al., intracoronary Doppler and ultrasound investigations were performed in patients with BBB and in control subjects, who all had angiographically normal coronary arteries and normal LV function. They found that LBBB was particularly associated with an increased coronary flow velocity, indicating enhanced myocardial oxygen demand, caused by a mechanically ineffective LV demand. This may contribute to the unfavorable outcome in patients with intraventricular conduction delay.

Transient LBBB is defined as an intraventricular conduction defect that temporarily returns to normal. Intermittent LBBB is characterized by the presence, in a single ECG tracing, of complexes showing LBBB and normally conducted beats, which is usually heart rate and blood pressure dependent. The mechanism of intermittent LBBB is unclear, but appears to result from anatomic or physiologic interruptions of a conduction bundle, from ventricular enlargement, and from functional or neurogenic depression with or without underlying pathologic lesions. In our patients, chronic LBBB was present in both cases initially. After denitrogenation with 100% oxygen prior to induction, chronic LBBB converted to normal sinus rhythm. After cessation of oxygenation, the LBBB reappeared on the ECG persistently. As the LBBB-induced LV remodeling might be processing for a period of time, in the absence of clinical symptoms, postponing the elective surgical procedure was justifiable. We were extremely proud of the expedients, because one patient showed myocardial ischemia in the apical, anterior and inferior segments of the LV in the T1-201 myocardial perfusion SPECT study, and the other showed septal wall hypokinesis on echocardiogram. We speculated that oxygen supplement had quenched the increased myocardial oxygen demand state which the chronic LBBB preconditioned. Although further experimental evidence is needed, the oxygen-induced remission of LBBB could be a predictor of unstable cardiac condition.

Anesthetic precautions and further investigations may be indicated if preanesthetic assessment reveals a patient who exhibits LBBB and symptoms of heart disease. However, asymptomatic presentation is likely to become increasingly common with demographic changes and better myocardial preservation. Although Goldman risk stratification and ACC/AHA guidelines suggest that LBBB does not carry a high anesthetic risk, chronic LBBB may indicate the need for noninvasive or minimally invasive cardiovascular investigations.

References


