

Left Bundle Branch Block Under General Anaesthesia in an Athlete's Heart

D T Kumaravadivel, MD*, **A Z Nor Azian, MMed (Anes)***, **S Thiruselvi, MMed (Anes)****, **J Abdul Aziz, MMed (Anes)***

*Department of Anaesthesiology and Intensive care Unit, Hospital Teluk Intan, 36000 Teluk Intan, Perak, Malaysia, **Senior lecturer, International Medical University, 126 Jalan 19/155b, 57000 Kuala Lumpur

SUMMARY

Left bundle branch block (LBBB) during anaesthesia is uncommon. During general anaesthesia, LBBB may be related to hypertension or tachycardia and its acute onset makes the diagnosis of acute myocardial ischemia or infarction difficult. We would like to present a case report of a healthy patient who developed LBBB intra operatively. Acute LBBB should lead to suspicion of acute coronary syndrome until proven otherwise. Inability to exclude an acute cardiac event resulted in postponement of surgery twice after general anaesthesia was administered. Cardiological investigation of our patient showed physiological left ventricular hypertrophy (LVH), "athlete's heart" which was the most likely cause of the LBBB under anaesthesia.

KEY WORDS:

Anaesthesia, arrhythmia, heart, conduction, athlete's heart

INTRODUCTION

LBBB, occurring during general anaesthesia, makes the diagnosis of acute myocardial infarction difficult. We report a patient with acute LBBB that appeared after induction of anaesthesia. Cardiological investigation of our patient showed an athlete's heart which is a physiological adaptation to athletic activities. To the best of the authors' knowledge, incidence of LBBB under general anaesthesia in athlete's heart has not been reported yet.

CASE REPORT

A 22-year-old, 75kg, male, a case planned for endoseptoplasty and bilateral functional endoscopic sinus surgery under general anaesthesia. A detailed pre-anaesthetic check up done, found him to be healthy and well. The patient was induced with fentanyl (100microgramIV) followed by a sleep dose of propofol (150mgIV) and atracurium (35mgIV) was used to facilitate endotracheal intubation. His blood pressure (BP) was 160/82mmHg and his heart rate (HR) was 90 beats per minute (bpm) prior to induction. The patient was intubated and ventilated with nitrous oxide, oxygen (50%, 50%) and 1% sevoflurane. Immediately, junctional rhythm was noted on the cardiac monitor. The procedure was abandoned after 20 minutes of observation showed no change to a sinus rhythm although the patient was hemodynamically stable (BP ranging 120-

150/60-85 mmHg, HR 55-70 bpm). After emergence it was noted that his heart reverted back to sinus rhythm. The patient was referred to the physicians for further investigations. All biochemical, electrolytes, hematological, and echo findings were within normal limits. His electrocardiogram (ECG) showed sinus rhythm with left ventricular hypertrophy (LVH). Our patient's BP during ward stay was also normotensive ranging 120-130/ 70-85 mmHg. Two weeks later the patient was rescheduled for the same procedure.

This time patient was induced with fentanyl (100microgramIV) followed by cardio stable drug, etomidate (14mgIV) and atracurium (35mgIV). His BP was 150/80mmHg and the HR was 90 bpm before induction. Intubation and ventilation was smooth and maintained with nitrous oxide, oxygen (50%, 50%) and 1-2% sevoflurane. One minute later we noted T inversion that progressed to ST depression and widened QRS complex on the ECG monitor. ECG taken at the 10th minute showed junctional rhythm LBBB (FIG 1). Post induction BP ranged from 120-140/ 60-75 mmHg, HR 50-65 bpm and peripheral oxygen saturation (SPO2) 98-100%. Pharmacological acceleration (using atropine 0.4 mg IV) of heart rate was done, but the LBBB did not return to sinus rhythm. He was referred again to the medical team and surgery was postponed again. Immediately after anaesthesia was discontinued, the LBBB returned to normal sinus rhythm and patient was well and asymptomatic. The patient was monitored in the recovery bay and repeat ECG showed normal sinus rhythm. Further cardiological investigations: holters monitoring, Bruce protocol exercise stress test, and electrophysiological study showed no abnormalities. However during the second echocardiogram conducted in the cardiology centre LVH was detected and he was scheduled for cardiac magnetic resonance imaging (MRI). Cardiac MRI showed an athletic heart, LVH with normal volume and function of left ventricle.

DISCUSSION

In recent years there has been increasing awareness and reports about sudden death amongst athletes. In highly trained athletes, it is often critically important to determine whether LVH is an expression of a physiologic and benign adaptation to training or a pathologic process with a risk of sudden death, due to hypertrophic cardiomyopathy.

This article was accepted: 16 November 2012

Corresponding Author: T Kumaravadivel Dharmalingam, Department Of Anesthesiology and Intensive Care, Hospital Teluk Intan, Jalan Changkat Jong, 36000 Teluk Intan, Perak Email: dtkumar123@yahoo.com

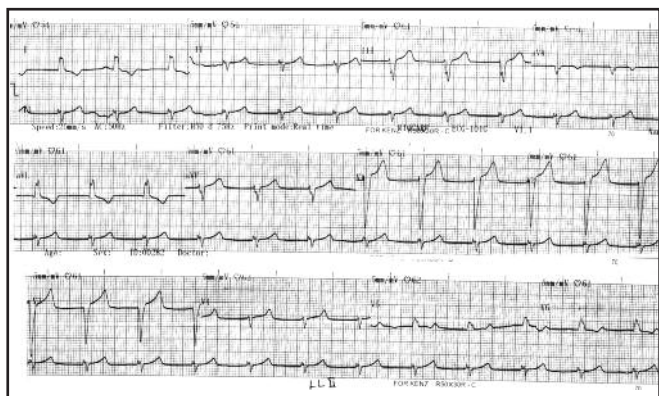


Fig. 1 : 12-Lead ECG during developed junctional rhythm LBBB under anaesthesia

We present a case report of a patient who developed junctional rhythm LBBB under anaesthesia. It was very difficult for us to decide on continuing the anaesthesia as we were unable to exclude the pathological condition. When LBBB occurs acutely under general anaesthesia, it is difficult to exclude acute ischemia or myocardial infarction¹ especially in a case like ours where the patient is a young and otherwise healthy individual.

Studies² show that transition from normal conduction to abnormal intraventricular conduction during anaesthesia may be related to alterations in heart rate by only 1 or 2 beat/min. In our patient, pharmacological acceleration (using atropine 0.4 mg IV) of heart rate did not change intraventricular conduction.

Usage of higher concentrations of sevoflurane (8%) for induction has been reported to precipitate arrhythmias in susceptible individuals³. Although sevoflurane was the only similar agent used for both events, only 1-2% was used in our patient.

In our patient, cardiological investigations did not show any pathological condition. He is an active badminton player and plays 12 hours per week since primary school days. It appears to be just a benign condition called physiological LVH that caused the conduction defect under anaesthesia.

Physiological LVH or "athlete's heart" is the normal response to healthy exercise or pregnancy which results in an increase in the heart's muscle mass and pumping ability. Trained athletes (Rowers, cyclists, and cross-country skiers) have hearts that have left ventricular mass up to 60% greater than untrained subjects⁴.

ECG changes in athletes are common and usually reflect structural and electrical remodeling of the heart as an adaptation to regular physical training⁵. It is imperative that ECG abnormalities resulting from intensive physical training and those potentially associated with an increased cardiovascular risk be properly defined.

The difference between physiologic LVH and hypertrophic cardiomyopathy are the appropriately increased size of the left ventricular internal dimension, and the normal systolic and diastolic left ventricular function. Cardiac MRI provides highly reproducible and accurate assessments of the structure (diameters, volumes, mass) and function of both the ventricles. In our patient, cardiac MRI was used to differentiate between physiological and pathological LVH.

In our patient, we believe that his LBBB under anaesthesia is a consequence of his "Athlete's Heart", a physiological left ventricular hypertrophy. This raises a question of whether young athletic individuals planned for surgery routinely need a cardiology referral and workup. Would there be a difference in anesthetic management if it is confirmed? Having said that, I would like to say that with a probable diagnosis of "Athlete's heart", the anaesthetist would be able to be more prepared and not hesitate to proceed with the surgery thus avoiding unnecessary repeated exposure to anaesthesia and cancellation of surgery which carry their own disadvantages for the patient as well as the hospital.

REFERENCES

1. Eldeman DM, Hurlbert BJ. Intermittent left bundle branch block during anesthesia *Anesthesia and Analgesia* 1980; 59: 628-30.
2. A Tyagi, A K Sethi, V Agarwal, M Mohta. Rate-dependent left bundle branch block during anaesthesia *Anesthesia and intensive care*.2004; 32(5): 715-8.
3. Pankaj Kundra, V Vinodhadevi, and G Arimanickam. Sevoflurane-induced arrhythmia in an adult and a child. *J Anaesthesiol Clin Pharmacol*. 2011 Apr-Jun; 27(2): 269-71.
4. John Rawlins, Amit Bhan, and Sanjay Sharma, Left ventricular hypertrophy in athletes *European Journal of Echocardiography* 2009; 10: 350-6.
5. Fagard R. Athlete's heart. *Heart* 2003; 89: 1455-61.