CASE REPORT

Postoperative rate induced left bundle branch block after craniotomy

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ABSTRACT

Rate induced left bundle branch block (LBBB) is a rare peri-operative phenomenon. We encountered rate related LBBB in a 72 year old patient who had undergone a craniotomy.

Acute coronary event was ruled out by doing serial troponin-I levels and absence of new onset regional wall motion abnormalities on echocardiogram. The electrocardiographic changes reverted to normal after controlling the rate with β blockers. Further cardiac evaluation was advised but the patient and family opted for a conservative medical management considering his age and co-morbidities.

Key words: Bundle-Branch Block, Coronary Artery Disease, Electrocardiography, Postoperative Period

Citation: Rayani BK, Narayanan H, Saifuddin MS, Nair AS. Postoperative rate induced left bundle branch block after craniotomy. Anaesth Pain & Intensive Care 2016;20(3):334-337

Received: 28 April 2016; **Reviewed:** 30 April & 23 August 2016; **Accepted:** 15 September 2016

INTRODUCTION

New onset left bundle branch block (LBBB) is an ominous electrocardiographic (ECG) change which warrants further evaluation. The reason could be acute coronary event, coronary vasospasm, underlying structural heart disease or idiopathic.

The management depends on the cause of LBBB. If LBBB develops postoperatively after a non-cardiac surgery, a cardiologist has to be involved early.

CASE REPORT

A 72 years old male weighing 68 kg, having a left parieto-occipital 4×3.4 cm lesion with moderate perifocal oedema was posted for craniotomy and excision of tumour. He was a known hypertensive for last 10 years on tab amlodepine 5 mg once daily. He was diagnosed with type II diabetes mellitus with blood sugars under control with oral hypoglycaemic agents. Other laboratory tests were unremarkable. 12 lead electrocardiogram was within normal limits (Figure 1). His resting two dimensional echocardiogram (2D ECHO) revealed good LV/RV function with ejection fraction of

55%. Dobutamine stress echo (DSE) was done for risk stratification which was negative for inducible ischemia. The cardiologist cleared the patient for surgery under high risk in view of age, co-morbidities and major surgery. After obtaining a high risk consent for major surgery and after explaining possibilities of ventilatory support, major cardiovascular events, seizures, neurodeficit ;the patient was taken to operation theatre after confirming 6 hours of fasting. Anesthesia was induced after premedication with 2 mg midazolam, 100 μg fentanyl using 150 mg propofol. Airway was secured with 8 mm cuffed endotracheal tube after achieving neuromuscular blockade with 7 mg vecuronium. We monitored oxygen saturation with pulse oximeter, ECG with lead II, V5, V6; end tidal carbon dioxide; and arterial BP with a left radial arterial cannula. We cannulated the right subclavian vein with a 7 French double lumen central venous catheter to monitor central venous pressure and for

possible vasopressor use. Surgery lasted for 4 hours during which a blood loss of 400 ml occurred. Post operative medications were: mannitol 20 gm 8th hourly, phenytoin sodium 100 mg 8th hourly and dexamethasone 4 mg 8th hourly along with injection ceftriaxone 2 gm twice daily and pantoprazole 40 mg once daily. After ventilating for 2 hours in surgical intensive care unit, an uneventful tracheal extubation was done.

There was no post-operative neurodeficit on examination. 12 hours later, an abnormal rhythm with broad QRS complex was noted on cardioscope with heart rate of 140/min. 12 lead ECG was taken that revealed a new onset left bundle branch block not present in preoperative ECG (Figure 2). On

enquiring the patient, he didn't complain of chest pain or shortness of breath although he experienced palpitations. Cardiologist was asked to review who did a screening 2D ECHO which revealed LVEF of 54%, paradoxical septal motion, concentric left ventricular hypertrophy with no regional wall motion abnormality. Troponin I was ordered immediately and 6 hours later which was <0.01 ng/ml on both occasions suggesting no acute coronary event. Cardiologist advised rate control with tab metoprolol 25 mg twice daily, aspirin 150 mg once daily, atorvastatin 40 mg

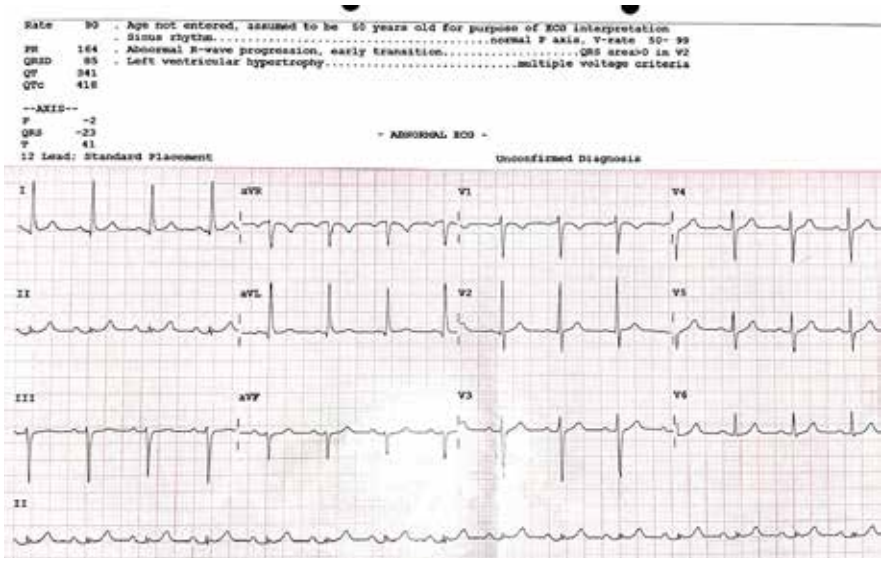

Figure 1: Preoperative 12 lead ECG – normal sinus rhythm. Left axis deviation and left ventricular hypertrophy by voltage criteria noted on ECG.

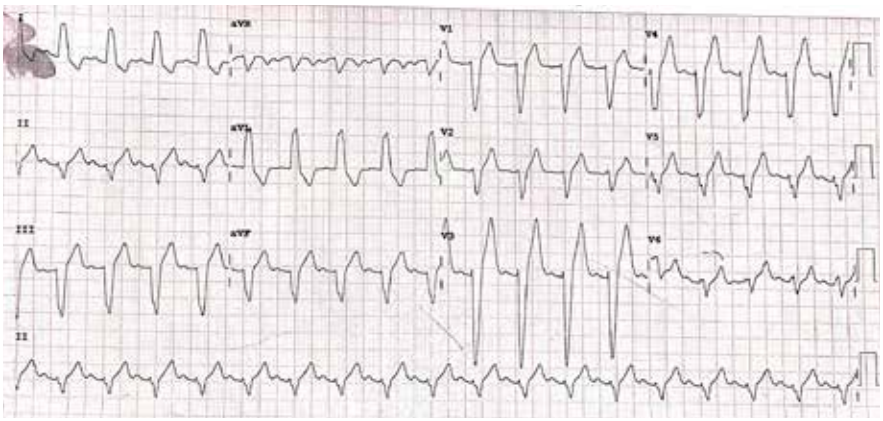

Figure 2: 12 lead ECG showing LBBB morphology.

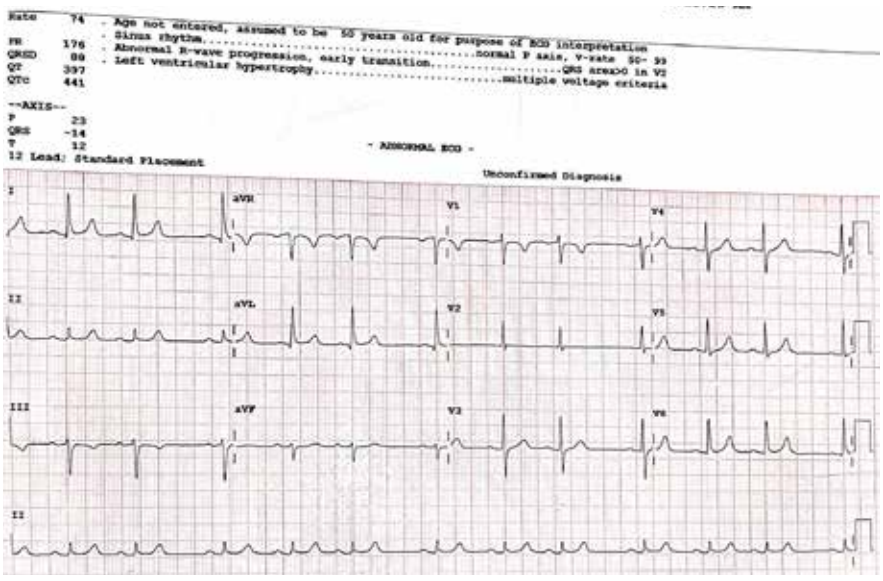

Figure 3: 12 lead ECG after starting metoprolol: normal sinus rhythm with no ST-T changes.

once daily and injection enoxaparine 40 mg twice daily. After discussing with the surgeon, we decided to administer 40 mg enoxaparine subcutaneously only as acute coronary event was ruled out. The heart rate reduced from 140 to 84/min after metoprolol administration which also reverted the LBBB morphology (Figure 3). The ECG changes appeared again after 8 hours which reverted after another dose of tab metoprolol 25 mg. After this episode his heart rate was under control with no further episodes of palpitations till discharge.

DISCUSSION

Exercise induced LBBB is a rare event observed during 0.5-1 % of exercise stress tests. Rate induced LBBB are of two types. The first one is exercise induced which is called as acceleration induced LBBB which manifests when the heart rate increases to a particular number. The other one is deceleration induced where LBBB is seen when the heart rate drops to a particular number.[1] The possible causes of such LBBB could be structural heart disease (cardiomyopathy, congenital heart disease, valvular heart disease), slow flow through coronaries, coronary vasospasm. The condition is possible even in presence of normal coronaries. However, another hypothesis is that the changes could be due to coronary microcirculation ischemia which cannot be detected n routine coronary angiography. Observational studies have shown greater possibilities of death and major cardiovascular events in patients developing LBBB during exercise or induced by rate. However, patients with normal coronaries presenting with exercise induced LBBB have shown to do well compared to patients having documented abnormality in the cardiovascular system.[2]

Further evaluation involves 2D ECHO, cardiac enzymes, coronary angiogram (CAG). 2D ECHO usually reveals paradoxical motion of interventricular septum. This paradoxical septal motion is responsible for the chest discomfort experienced by the patient.[3] The presence of regional wall motion abnormality can be seen on 2D ECHO. Troponins are useful in identifying an acute coronary event which warrants antiplatelets, statins and a CAG to quantify coronary artery disease. Definitive

management can be planned if CAG is positive for coronary artery disease (CAD) i.e. revascularization, percutaneous or surgical. These patients can be absolutely asymptomatic during the rate related ECG change. However, a CAG might still be useful to rule out CAD, to access state of flow across the coronaries or to rule out coronary vasospasm also known as Prinzmetal's angina.[4]

Management depends on the cause of LBBB. A proven CAD is managed is by using β blockers, angiotensin converting enzyme inhibitors, angiotensin receptor blockers, antiplatelets, statins and revascularization in indicated patients. In low flow states or in Prinzmetal's angina, diltiazem is used. Anderson et al used non-pharmacological method to treat a patient with exercise induced LBBB having normal coronaries on CAG. They treated the rhythm abnormality with exercise training. The patient initially had chest discomfort with LBBB at a heart rate of 108/min, which with exercise training was later experienced at 150/min.[5]

Our patient had a DSE done preoperatively which was negative for inducible ischemia at a maximum heart rate of 150 /min. However, there was no LBBB noted at that time but was noted and documented in the post-operative period. We presume it could be due to surgical stress or coronary microcirculatory changes. The family didn't evaluate the patient further owing to his age and associated co-morbidities and opted for conservative medical management.

Conflict of interest: Nil declared by the authors

Authors' contribution: BKR: Concept & design of manuscript; HN: Manuscript preparation; MSS: Manuscript preparation & bibliography; ASN: Manuscript preparation / review

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My Most Unforgettable Experience

Fourteen years back!

You are doing a case in Africa. There is no air conditioning and it is only fanning you can do to keep from fainting. There is one oxygen tank in the hospital and your drug selection is pentothal, ketamine and diazepam. That is it! The surgeon is doing a mastectomy without cautery and the rusted suction machine is working intermittently. The African provider doesn't use the anesthesia machine anymore because he can't fix the leak. Ketamine, it's a good drug, but not for total general anesthesia. I look around and there is no Ambu bag, no LMA and one laryngoscope blade but he doesn't like to intubate. We start the case and there is bleeding. A lot of bleeding and a lot of sweating. We need blood and we need it now. I call the lab thinking I'm going to loose my first patient in Africa. Surprisingly the blood arrives and there is no name on the bag. It came from a relative. Direct cross-matched.

This was 14 years ago. The stories are numerous but things have not remained the same. We now have a modern operating room and can run 4 tables with all the bells and whistles we have in the USA. While much of Africa works as described above, we do not. Check out our new machines. Join Kenyarelief.org (a CRNA centric group). Take the trip of a life time and use your skills abroad.

Email Molly Shaw CRNA at molly@kenyarelief.org