The Enigma of Intermittent Left Bundle Branch Block: A case report

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Abstract: *A 45 yr old male was diagnosed as a case of acute coronary syndrome presenting as intermittent left bundle branch block (LBBB). He was thrombolysed with streptokinase (STK) and his rhythm reverted back to normal sinus rhythm within six hour post STK. He was subsequently sent home after six days of coronary care unit admission only to be readmitted within five days of discharge with severe chest pain and reappearance of LBBB. He underwent coronary angiogram on his second admission which revealed 95% left anterior descending (LAD) and 50% right coronary artery (RCA) block. A stent was placed in mid LAD and the patient has been asymptomatic since then. We conclude that patients with symptomatic intermittent complete LBBB should never be ignored and should undergo coronary angiogram at the very outset if facilities are available.*

Keywords: *Intermittent left bundle branch block, acute myocardial infarction, coronary angiogram, thrombolysis, stent*

Introduction

Men with left bundle branch block (LBBB) have a substantially increased risk of coronary death, mainly due to sudden death outside the hospital setting. It is also known that degeneration of the specific conduction system increases with advancing age, resulting in a rise in the prevalence of bundle branch block in older individuals. Our insight into the patho-physiological relationship between left bundle branch block and organic heart disease remains superficial.1,2,3 For example, it is unknown whether left ventricular dysfunction precedes left bundle branch block or whether the reverse is the case.4 Intermittent LBBB with changing heart-rate dependency and heart-rate-dependent supranormal conduction in the left bundle branch may be seen in some cases. Masking of the electrocardiogram (ECG) features in acute myocardial infarction (AMI) in LBBB patients makes the diagnosis difficult.

Case Report

We report the case of a 45 year old man who presented with severe shortness of breath of sudden onset associated with central chest pain at our medical college during regular morning out-patient clinic (OPD). His breathlessness started at around 9.30 AM, on the same day, minutes before he had central chest pain. He was sweating, restless, and looked fatigued. Besides being obese (body mass index >30) there were no other relevant history. While he was an avid tea drinker, he was a non-alcoholic and a non-smoker. There were no previous health records. Physical examination revealed nothing significant except sinus tachycardia and loud first heart sound. Immediate 12 lead ECG revealed complete LBBB. Since there was no base line ECG to compare with, and with no other previous ECGs, we presumed it to be a fresh onset LBBB (Fig.1). We suspected it to be due to acute myocardial infarction based on Sgarbossa et al ECG algorithm on LBBB's ECGs, which is ST elevation more than or equal to 1 mm in leads with a positive QRS, ST-depression.

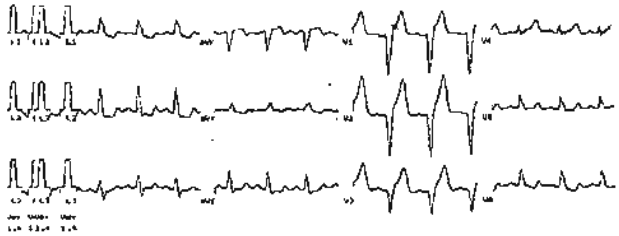

Fig 1: ECG showing complete LBBB.

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be more than or equal to 1 mm in VI to V3, and ST elevation more than or equal to 5 mm in leads with a negative QRS.8,10 Even though Troponin T was negative, considering the time frame he presented in (within 2 hour of chest pain), we did not consider it worthwhile to wait for the test to be positive and then start the treatment. He was put on streptokinase (STK) 1.5 million units intravenous drip in 100 ml of normal saline at around 11.30 AM in cardiac care ward. He was also put on regular myocardial infarction protocol as designated by American heart association and American College of Cardiology. Follow up 12 lead ECG were recorded at first, second, fourth, sixth and twelve hours of STK infusion on first day and subsequently every four hourly next day even as patient was on continuous ECG monitoring. To our satisfaction his LBBB reverted back to normal. ECG at six hour post STK, did not show any sign of infarction. Patient was asymptomatic by this time except for some breathing difficulty for which he was kept on oxygen mask infusion at 3 liters/minute. His chest pain was gone. His respiratory rate had come down to 24/min. from 40/ minute at the time of admission. JVP now normal, with no signs of heart failure. Cardiac examination revealed normal heart sound with no murmurs. Two dimensional transthoracic echocardiography (2DTTE) was carried out three days post admission, which did not revealed any wall motion abnormality or any increase in pulmonary artery systolic pressure and no significant valve regurgitation. There was mild left ventricular hypertrophy with grade 1 left ventricular diastolic dysfunction which was checked by pulmonary tissue Doppler studies. His follow up ECG showed normal sinus rhythm still did not show any features of MI. Considering the clinical improvement he was discharged on day 6 with advice of follower visits on designated OPD days. He was put on anti-angina and anti-cardiac failure drugs in to aspirin, clopidogrel and atorvastatin. He was discharged with a diagnosis of acute coronary syndrome. To our astonishment he came back to us within five days of discharge with severe shortness of breath, chest pain and was readmitted to CCU.

ECG revealed reappearance of complete LBBB pattern. He was not in heart failure but in angina pain. He was again put on intravenous nitrates and morphine. This time STK was not given as he had already received it eleven days back. With the appearance of a second episode of LBBB, we carried out a coronary angiogram to rule out coronary spasm or a thrombus in the coronary arterial system. His angiogram was done the day after, which revealed two vessel block, 95% mid LAD and 50% RCA branch. (Figure 2,3,4)

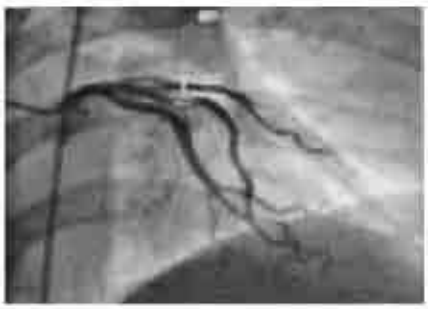


Fig 2: Coronary angiogram showing mid LAD (95%) block.

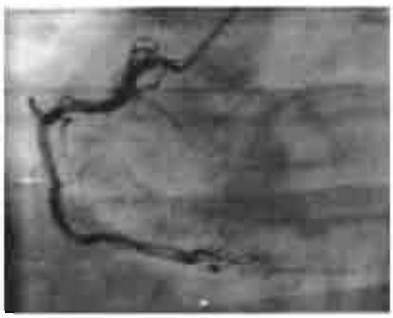


Fig 3: Coronary angiogram showing 50% RCA block.

He was subsequently put on a stent at mid LAD. The patient has been asymptomatic since then with no recurrence of LBBB on subsequent follow up.

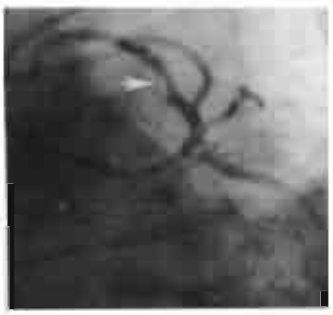


Figure 4 a, b. Coronary angiogram images, pre and post angioplasty with stent in mid LAD showing TIMI 3 flow.

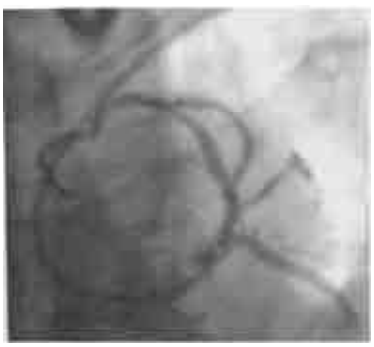
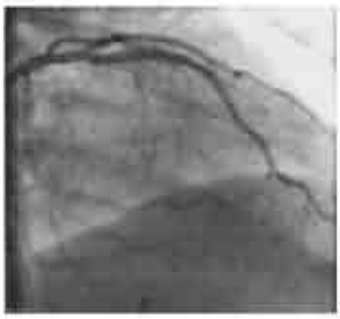


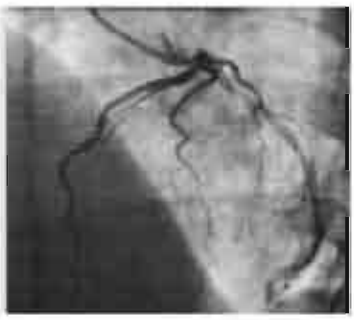
Fig 4 a: Coronary angiogram images, pre angioplasty with stent in mid LAD showing TIMI 3 flow.

Figure 5 a, b : Post PICA with stent to LAD showing TIM 13 flow.

(a)



(b)



Discussion

As opposed to right bundle branch block, left bundle branch block has been associated with organic heart diseases caused by high blood pressure, coronary artery disease, aortic valve stenosis, and cardiomyopathy. This may result in missed cases which may otherwise have benefited from acute revascularization therapy. Invariably, this results in delays in the provisioning of thrombolysis to these patients despite the mounting body of evidence which demonstrates that patients with AMI who present with LBBB have greater in-hospital mortality than those who do not.5,6,7 In our patient who had intermittent LBBB, the diagnosis of acute myocardial infarction was difficult but not impossible when the three criteria suggested by Sgarbossa et al were followed. We did not believe in waiting and went ahead with revascularization because prognosis of acute myocardial infarction improves by early revascularization. Difficulties in interpreting the ECG in these patients can therefore delay treatment and compromise their prognosis. Currently, thrombolytic treatment is under-utilized in patients with LBBB and AMI, and those who are thrombolysed endure lengthy delays before treatment.

Conclusions

It is our belief that complete LBBB should never be ignored and patients who presents with intermittent LBBB should undergo coronary angiogram as 2D TTE may fail to reveal any regional wall motion abnormality.

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