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Electrocardiographic Case: Diagnosis of Acute Myocardial Infarction in the Presence of Left Bundle Branch Block

L F Hsu, R M L Kam, W S Teo

ABSTRACT

The electrocardiographic features associated with acute myocardial infarction (AMI) are often easily recognised. However, interpretation is made more difficult in the presence of confounding patterns such as a left bundle branch block (LBBB). This may result in missed cases which may otherwise have benefited from acute revascularisation therapy. Though not straightforward, the diagnosis of AMI in the presence of LBBB can be made with a reasonable amount of accuracy. We report a case of acute myocardial infarction with LBBB that was appropriately diagnosed and underwent acute revascularisation by angioplasty. A detailed knowledge of the typical electrocardiographic features associated with LBBB, especially the ST segment morphologies, is very important. This will greatly aid recognition of an evolving AMI and help us decide on the most appropriate therapy.

Keywords: acute myocardial infarction, diagnosis, electrocardiogram, left bundle branch block

INTRODUCTION

The 12-lead electrocardiogram (ECG) is a valuable tool in the evaluation of a patient with chest pain. Accurate interpretation of the ECG is essential for both diagnostic and therapeutic purposes. This is particularly important for patients with acute myocardial infarction (AMI), as timely diagnosis and institution of appropriate revascularisation therapy may be life-saving.

The classical ECG changes of AMI are often easily recognisable. However, these changes may be masked by certain ECG patterns, especially left bundle branch block (LBBB) and ventricular paced rhythms. In such patients, the diagnosis of AMI had been controversial in the past, with many ECG signs and criteria proposed but none gaining widespread acceptance[1]. This has now changed significantly with recent refinements in ECG interpretation, and the diagnosis of AMI can be made with a reasonable degree of accuracy.

Department of Cardiology
National Heart Centre
Mistri Wing
Third Hospital Ave
Singapore 169608

L F Hsu, MBBS, MRCP (UK)
Registrar

R M L Kam, MMed (Int Med), MRCP (UK), FAMS
Consultant

W S Teo, FRCP (Edin), FAMS, FACC
Senior Consultant

Correspondence to:
Dr W S Teo

CASE PRESENTATION

A 64-year-old man presented to the Emergency Department with severe, crushing central chest pain of 2 hours' duration, associated with diaphoresis. He had a 10-year history of double-vessel coronary artery disease on medical therapy, and had been asymptomatic for the past 8 years. In addition, he also had long-standing hypertension and hypercholesterolaemia on treatment, as well as a positive family history of ischaemic heart disease. Initial examination revealed a sweaty, obese man with bradycardia, his pulse rate being 52 beats per minute and regular. He was otherwise haemodynamically stable and not in cardiac failure.

The ECG on arrival (Fig 1) showed normal sinus rhythm with first degree atrioventricular block and LBBB. However, the ST segment and T wave changes were highly suggestive of AMI. There was definite ST segment elevation in leads II, III and aVF, ST segment depression in V2 and V3 and primary T wave changes in V5 and V6. He was immediately brought to the cardiac catheterisation laboratory where total occlusion of the right coronary artery (RCA) and partial 80% occlusion of the first diagonal branch of the left anterior descending artery (LAD) was found. Angioplasty with intracoronary stent application was performed successfully on the RCA lesion.

The patient recovered uneventfully from the AMI, which was confirmed by elevated cardiac enzyme levels. A subsequent myocardial perfusion study revealed evidence of an inferior infarct.

DISCUSSION

Numerous studies have demonstrated clearly that early restoration of myocardial perfusion results in reduced mortality and morbidity in patients with transmural or Q-wave AMI[2]. Either thrombolytic agents or primary coronary angioplasty may be employed depending on available facilities and expertise[3]. This survival benefit decreases progressively with increasing delay in starting treatment[2], hence rapid and accurate diagnosis of

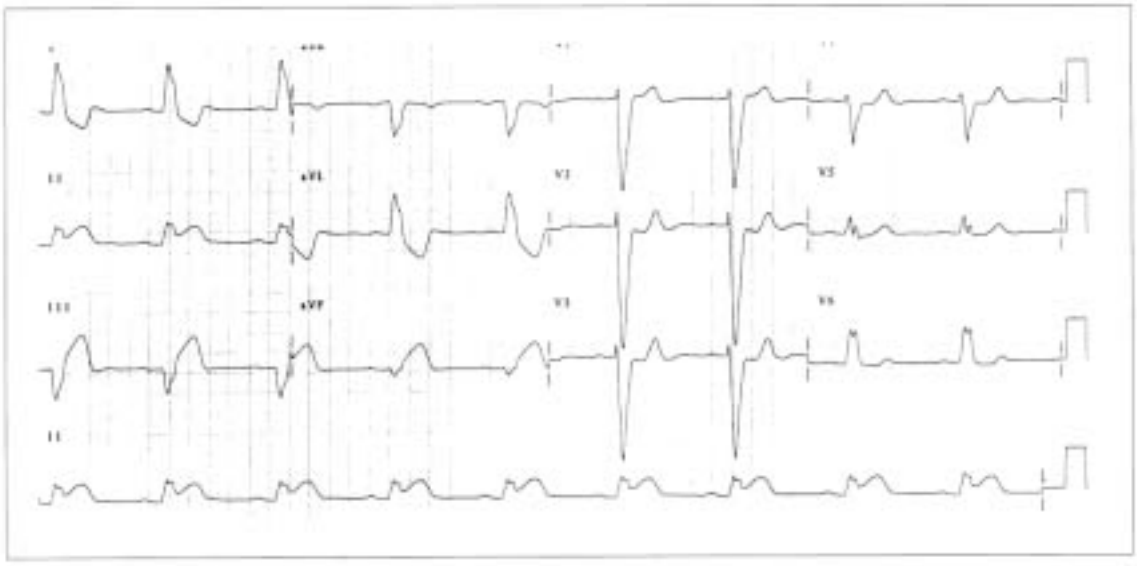

Fig 1 – ECG of patient, showing significant concordant ST elevation in lead II, ST depression in leads V_2 and V_3, and discordant ST elevation in lead III.

AMI is essential. The ECG is a valuable aid in this respect. However, the presence of LBBB may render its interpretation more difficult.

The correct diagnosis of AMI in the presence of LBBB is even more crucial as LBBB is associated with more severe underlying coronary artery disease and hence poorer cardiac function, and a higher incidence of cardiogenic shock and mortality[4]. They are thus at higher risk for adverse outcomes[5], but more importantly they have also been shown to derive significant benefit from timely revascularisation[2].

In a normal heart, the interventricular septum is activated from left to right. This produces an initial R wave in the right praecordial leads and a Q wave in leads I, aVL and the left praecordial leads. When complete LBBB is present, depolarisation proceeds from right to left, producing initial Q waves in the right praecordial leads and eliminating the normal Q waves in the leftward oriented leads. The QRS complexes are widened to greater than 0.12 seconds and are monophasic, with a predominantly negative QS complex in lead V_1 and a positive R wave in leads I, aVL, V_5 and V_6[6]. These changes are often used in published literature as criteria to define LBBB. The repolarisation process is also similarly affected, resulting in ST segment and T wave configurations which are discordant and directed away from the QRS complex. Thus, ST depression and T wave inversion are observed in leads I, aVL, V_5 and V_6, while ST elevation is seen in the right and anterior praecordial leads[7]. These features, which bear a striking semblance to the changes seen in AMI, are illustrated in Fig 2. Loss of this normal discordant relationship in patients with LBBB may imply the presence of an acute process, such as an AMI[8].

The diagnosis of AMI in the presence of LBBB had been the subject of considerable interest in the past. Early studies variously described changes in the QRS complexes, the presence of Q waves, R wave regression and primary ST segment and T wave changes as having diagnostic value[1,9]. Changes in the QRS complex included notching of the S wave in leads V_3 to V_5 (Cabrera's sign), notching of the upstroke of the R wave in leads I, aVL, V_5 and V_6 (Chapman's sign) and a terminal S wave in V_5 and V_6. Primary ST segment and T wave abnormalities are defined as the respective displacement of the ST segment or T wave in the same direction as the QRS complex, in contrast to their discordant relationship with the QRS complex in uncomplicated LBBB[7]. They have been found to have very high specificity (91%) but low sensitivity (25%) for detecting AMI[9]. In fact, primary T wave abnormalities, as seen in V_5 and V_6 in our patient, were present in 24% of patients with LBBB without AMI in one study[1]. On a practical basis, all these ECG signs were not entirely satisfactory and did not gain widespread acceptance[5,9].

Recently, Sgarbossa et al[10] developed and validated a clinical prediction rule based on a set of 3 ECG criteria which were found to have the most diagnostic value in the presence of LBBB. These criteria, based on simple ST segment changes, were ST segment elevation of 1 mm or more which was concordant (in the same direction) with the QRS complex, ST segment depression of 1 mm or more in lead V_1, V_2 or V_3 and ST segment elevation of 5 mm or more that was discordant (in the opposite direction) with the QRS complex. A scoring system was also developed (Table I) which allowed a highly specific diagnosis of AMI to be made.

Table I – Criteria and scoring system for diagnosis of AMI in LBBB

Criteria	Score
1. ST segment elevation 1 mm or more concordant with QRS	1
2. ST segment depression 1 mm or more in V1, V2 or V3	3
3. ST segment elevation 5 mm or more discordant with QRS	2

A total score of 3 or more is highly suggestive of AMI

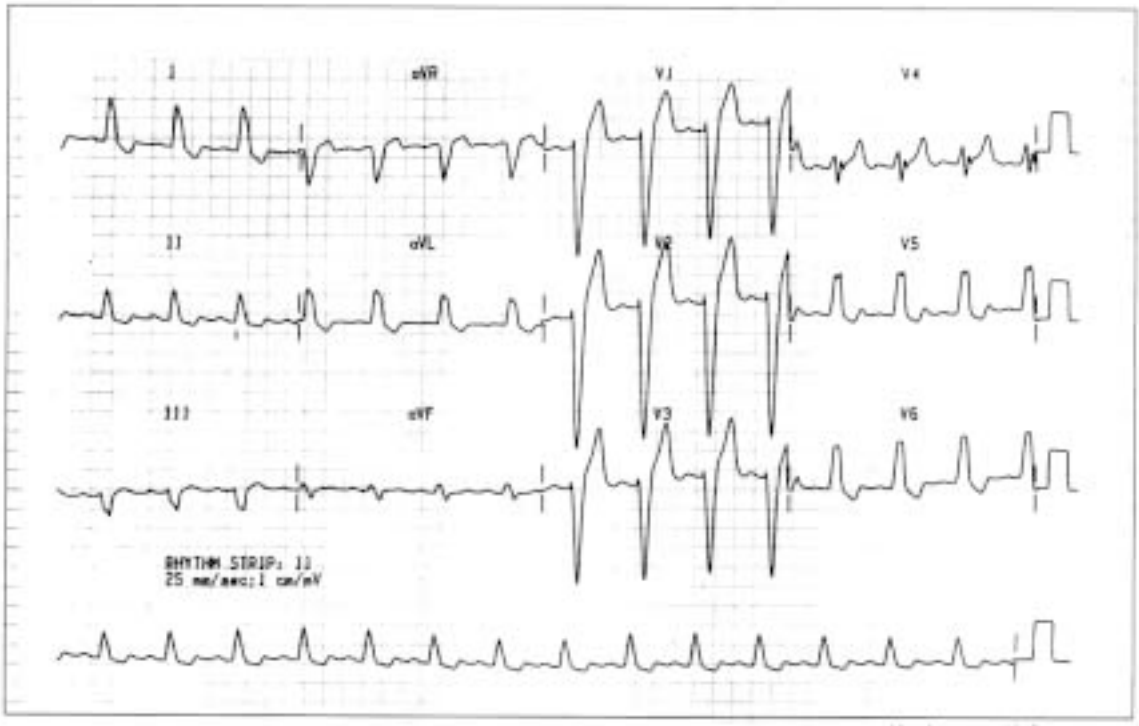

Fig 2 – ECG showing uncomplicated LBBB. The QRS and ST segments demonstrate the typical changes associated with LBBB.

Our patient's ECG demonstrated all 3 criteria. There was concordant ST segment elevation of 2.5 mm in lead II, ST segment depression of 1 mm in leads V_2 and V_3 and discordant ST segment elevation in lead III. This resulted in a total score of 13, which was very strongly suggestive of AMI. The ST segment elevation in leads II, III and aVF suggested an inferior infarction, which was later confirmed by myocardial perfusion scanning. Based on the ECG information, urgent revascularisation in the form of primary angioplasty was successfully performed.

CONCLUSION

Using data from published studies, the incidence of LBBB in patients with AMI varies from 0.5% to 2%[9,10]. New-onset LBBB or ischaemic changes superimposed on chronic LBBB are easy to recognise when compared with a previous ECG. Unfortunately, such information is usually not available, and the clinician will have to make the diagnosis and select treatment based only on the ECG information available at the time. As a result, some patients in this group may not receive early revascularisation therapy due to nondiagnostic ECGs. A detailed knowledge of the ECG changes found in uncomplicated LBBB, together with the criteria proposed by Sgarbossa et al[10] may enable the clinician to better identify these patients and offer appropriate therapy.

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