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Right and Left Bundle Branch Block in Acute Inferior Myocardial Infarction

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ABSTRACT

An 88-year-old man presented with acute Q wave inferior, posterior and right ventricular myocardial infarction which was associated with intermittent complete right and left bundle branch block.

Keywords: myocardial infarction, right and left bundle branch block

INTRODUCTION

Complete right bundle branch block (RBBB) as well left bundle branch block (LBBB) in patients with Q wave inferior myocardial infarction has previously been reported[1,2]. However, to our knowledge, complete RBBB and LBBB occurring in the same patient presenting with acute Q wave inferior myocardial infarction has not been described.

CASE REPORT

An 88-year-old man who had previously been treated for hypertension and diabetes mellitus for the past 15 years presented with severe chest pain. Clinical examination revealed a systolic blood pressure of 50 mmHg by palpation (both the systolic and diastolic blood pressures could not be detected by the auscultatory method). The pulse rate was 100/minute and the peripheries of the upper and lower limbs were cold and clammy. Auscultation of the heart revealed that the heart sounds were soft. However, a third or fourth heart sound was not heard and there were also no cardiac murmurs. Crepitations were heard in the lower part of both lungs posteriorly. The 12-lead ECG showed acute Q wave inferior myocardial infarction together with complete RBBB (Fig 1). The chest X-ray showed bilateral pulmonary oedema. Intravenous frusemide (120 mg) was given and an intravenous infusion of dopamine (5mg/kg/min) was started. The patient was also ventilated with a respirator.

The serum electrolytes were normal but the creatinine and urea were both elevated at 132.00 mmol/L and 7.50 mmol/L respectively. The serum CK was 2787.00 u/L and the CK-MB was 282.00 u/L. Both these levels were markedly elevated based on the normal values in our laboratory. The PaO_2 and the $PaCO_2$, whilst the patient was on the respirator, were both within the normal limits. Pulmonary artery flotation catheter insertion, echocardiography and coronary angiography were all not performed.

The patient's condition deteriorated and he died five days after admission. Post-mortem examination was not performed.

Electrocardiographic analysis

Multiple consecutive ECGs were done during the patient's stay in the hospital. The first ECG recorded soon after admission to the hospital (Fig 1) shows complete RBBB and ST segment elevation in leads II, III and aVF with reciprocal ST segment depression in leads I and aVL indicating acute Q wave inferior myocardial infarction. Fig 2, which was recorded about one hour after Fig 1, shows complete LBBB, first degree atrio-ventricular block (PR interval = 0.24 second) and acute Q wave inferior myocardial infarction as reflected by ST segment elevation in the inferior leads. Fig 3, which was recorded about two hours after Fig 1, shows the classical ECG pattern of acute Q wave inferior infarction and acute right ventricular infarction, but without any evidence of

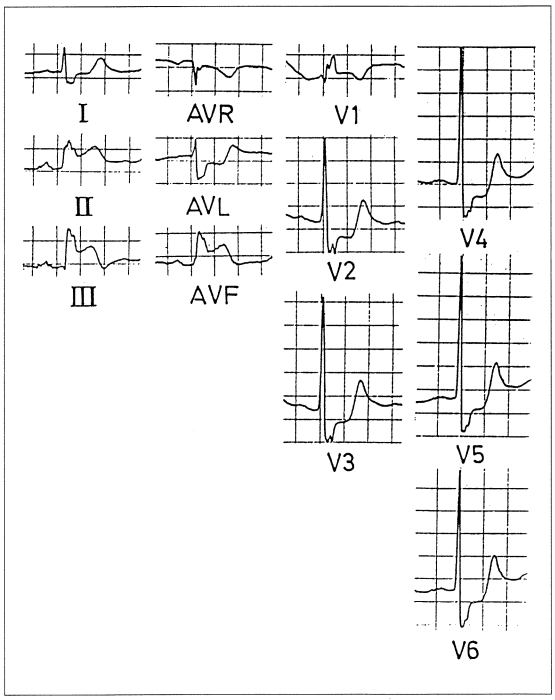

Fig 1 – ECG obtained soon after admission to hospital shows complete right bundle branch block and acute Q wave inferior myocardial infarction.

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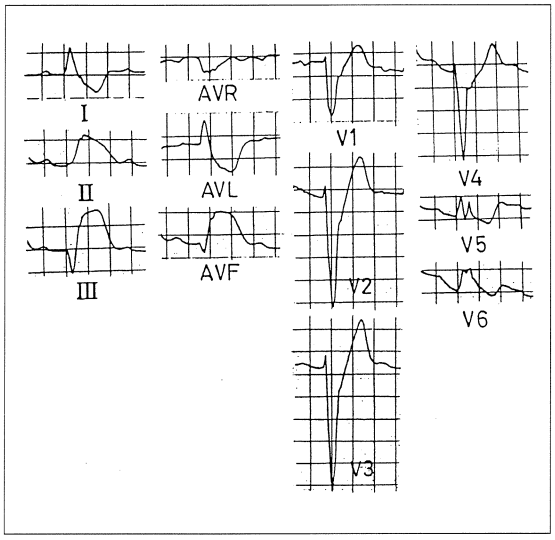

Fig 2 – ECG obtained about one hour after Fig 1 shows complete left bundle branch block, first degree atrio-ventricular block (PR interval = 0.24 second) and acute Q wave inferior myocardial infarction.

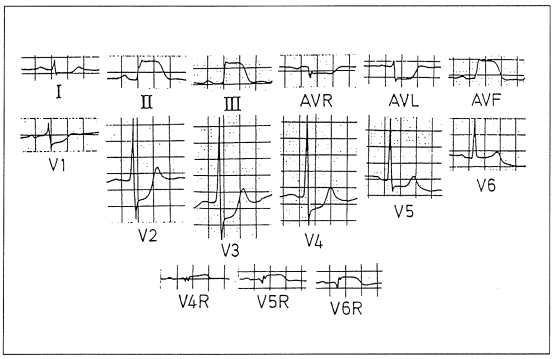

Fig 3 – ECG recorded about two hours after Fig 1 shows acute Q wave inferior and right ventricular myocardial infarction. Both the tall R waves in V_1 to V_3 and ST segment depression in V_1 to V_4 are most likely a reflection of posterior myocardial infarction.

bundle branch block. The tall R waves in leads V1 to V3 and the ST segment depression in leads V1 to V4 are most likely a reflection of co-existing posterior infarction. Subsequent ECGs showed evolving patterns of inferior and posterior infarction and intermittent complete LBBB. Transient 2:1 atrio-ventricular block was also seen during the patient's illness, but there was no occurrence of complete heart block.

DISCUSSION

Several previous studies, which have described bundle branch block (BBB) in acute myocardial infarction, have included both anterior as well as inferior myocardial infarction patients[3,4]. Therefore in these early reports, the incidence of BBB in patients with inferior myocardial infarction alone cannot be determined.

In 1995, Hod et al reported that in 2,215 consecutive patients with acute inferior Q wave myocardial infarction, 108 (4.9%) exhibited complete BBB. Right bundle branch block occurred in 85 (3.8%) and LBBB in 23 (1.0%) of these patients[1]. The time of appearance of the BBB was new in 30 patients (28%), old in 45 patients (42%) and undetermined in 33 patients (30%). Twenty-three (21%) of the 108 patients with BBB developed complete heart block compared to 9% of patients without BBB. In another study reported by Ricon et al, the incidence of BBB in acute inferior myocardial infarction was 8.9%[2] and this was higher than the incidence in the study reported by Hod et al[1]. However, the ratio of patients with RBBB with LBBB in both these studies was similar.

Bundle branch block in acute Q wave inferior myocardial infarction has been shown to be an independent risk factor for increased mortality[5]. In the study by Hod et al, the in-hospital and 5-year mortality rates were 22% and 33% respectively compared to mortality rates of 13% and 23% respectively in patients without BBB. However, the increased in-hospital mortality was seen only in patients with new BBB[1].

The aetiology of either RBBB or LBBB in acute Q wave inferior myocardial infarction is at present unclear. The most plausible explanation is that such patients have more extensive coronary artery disease or concomitant involvement of the left anterior descending artery[1]. In our patient, intermittent RBBB and LBBB were observed and this has not been previously described. The clinical presentation of acute pulmonary oedema and hypotension strongly suggest severe left ventricular dysfunction. However, since neither echocardiography, coronary angiography nor post-mortem study was performed, it is not possible to be certain of the exact extent of anatomical damage to the left and right ventricles nor the coronary artery lesions which had been responsible for the acute myocardial infarction and the bundle branch block.

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