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CASA REPORT

CASE REPORT: IMPORTANCE OF POSTERIOR LEAD ECG

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ABSTRACT

A 50-year-old man with history of diabetes who presented with chest pain and subtle electrocardiographic (ECG) abnormalities. Only with the aid of additional posterior ECG leads (V7-9) was a posterior wall acute myocardial infarction identified and confirmed by raising cardiac enzyme s. Later the ECG changes were also seen in the inferior wall. This case shows the importance of posterior ECG leads (15-lead ECG) in increasing the diagnostic yield of a conventional ECG in patients presenting with symptoms of an acute coronary syndrome by showing a posterior myocardial infarction and need for reperfusion therapy.

Keywords: gastric carcinoma, risk factors, alcohol, smoking, H.pylori, Screening Endoscopy

1.INTRODUCTION

ST-segment elevation by electrocardiography remains the hallmark for the diagnosis of acute myocardial infarction (MI), it helps in decision making and allows for early treatment to these patients. Here is a report of an unusual case of acute MI with ST-segment elevation in posterior ECG leads, in a patient with chest pain and ST segment depression anteriorly in the conventional 12-lead ECG, who was finally diagnosed to have an acute inferior wall myocardial infarction, which was reperfused successfully by thrombolysis.

A 50-year-old male with a history of diabetic for 10 years on medications was presented to the casualty with severe chest discomfort. He complained of retrosternal heaviness which started while doing house hold work 3 hours earlier, lasted for about 10 minutes, and had no radiation or other accompanying symptoms. The discomfort increased when he went for a walk, lasted for another 30 minutes, then he was brought to the casualty by his relatives.

On examination, his pulse rate was recorded at 72/min and his blood pressure was 100/60mm of hg. On auscultation heart sounds were normal and other systems no abnoramlities were detected. The 12-lead ECG showed sinus rhythm and minimal ST-segment depression in the anterior precordial leads V1 to V3 (Figure 1). As the patient was being examined in the casualty, he experienced chest pain again and the 12-lead ECG showed subtle, ST-segment depression in the anterior precordial progressed. Then, additional leads were taken (posterior leads V7, V8 & V9) .which demonstrated clear ST-segment elevation of 1 mm suggesting acute myocardial injury of the posterior wall. The enzymes were sent and their raising trentd was noted. Patinet was given aspirin and clopidogrel as stat doses. During the next ECG we noticed there was ST elevation in the inferior leads also. As he was not affordable for angiography or PCI we thrombolysed him with Streptokinase. echocardiographic examination showed an ejection fraction estimated at 50% and hyokinesia in the posterior and inferior segments.

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2.DISCUSSION

Though there was a tremendous development in the cardiology the ECG is the que. Urgent reperfusion therapy, with thrombolysis or with primary percutaneous coronary intervention, has improved the prognosis of patients with acute MI. It is indicated in patients with chest pain or discomfort of <12- hour duration who have persistent ST-segment elevation or new LBBB, hence an basic investigation like a 12 lead ECG is important in myocardial infarction. An earlier retrospective study of 100 consecutive cases of autopsy-proven acute MIs demonstrated that only 53% of them were correctly diagnosed ante-mortem. In many patients ST depression in the anterior precordial leads suggestive of posterior myocardial infarction is misdiagnosed. An erroneous or a belated diagnosis mostly plagues acute posterior-wall MI, since the ST-elevation of the posterior wall is displayed as ST-depression in the

anterior ECG leads and only when a concurrent ST elevation is noted in the inferior leads and/or a tall R wave is recorded in the right precordial leads (V1-2), a correct diagnosis may be derived. Heuy et al4 observed acute ST-segment elevation in only 48% of patients with LCX occlusion (versus 71% in RCA and 72% in LAD occlusion) and found that 38% of patients with a LCX-related infarct had no significant ST changes on admission. Similarly, Blanke et al found that 56% of patients with

LCX occlusion had 'non-classic electrocardiographic abnormalities'.5 they supported the concept that the ECG pattern of true posterior acute MI in the absence of classic changes in the inferior leads was highly specific and predictive of LCX/RCA disease. In patients suspected of having an acute MI, particularly if the affected area is the far postero portion of the left ventricle, three additional ECG chest leads (posterior V7, V8, and V9 leads) are useful in detecting and localizing myocardial injury or necrosis. The posterior wall of the left ventricle is not directly represented by any standard ECG lead. This is the reason why posterior MIs are characterized as 'dead angle infarctions' of the ECG,6 they are often misdiagnosed and as a result the patients are undertreated. It has been reported that 5% of non-Q-wave MIs are retrospectively diagnosed as posterior MIs in which reperfusion therapy would have been justified if additional ECG chest leads had been recorded.7 A posterior MI is suspected in the 12-lead ECG when
there is ST-segment depression in leads V1 V3, ST-segment elevation in leads I, aVL, V5 and V6 and can be confirmed by placing extra leads in the high lateral V7, V8 and V9 positions. For these three additional leads the electrode is placed over the posterior axillary line (V7), over the midscapular line (V8) and halfway between the midscapular line and the spine (V9), all at the same level as V6, providing a 15-lead ECG. ST-segment elevation of >1mm in these high lateral and posterior leads is suggestive of posterior MI,8 But because of the greater distance between the infarcted area and the leads in the posterior-wall MI, it is suggested that an elevation of 0.5 mm is sufficient to justify the diagnosis of posterior MI and the decision for aggressive treatment.6 The majority of patients with the ECG abnormalities suggestive of posterior MI have critical stenosis or occlusion of the LCX coronary artery, while significant
RCA disease is less prevalent.9 Moreover, in isolated posterior MIs the infarct-related artery is found to be the LCX coronary artery,3,10,11 suggesting that in the appropriate clinical setting posterior leads may help to discern LCX from RCA occlusion. Patients with inferior or lateral MIs with concomitant posterior wall involvement, have a larger-sized infarct with an increased risk of complications and are in dire need for reperfusion therapy. In keeping with this notion, in a group of patients with ST-segment elevation recorded only in leads V7-V9, isolated posterior MI was found to be complicated up to 69% with mitral regurgitation which was moderate to
severe in one third of patients.11 The high percentage of electrocardiographically undiagnosed acute MI, especially due to LCX occlusion, remains an issue that cannot be satisfactorily addressed by additional posterior ECG leads. Yet in a cohort of patients the percentage of ECG-undiagnosed LCX occlusion was reduced from 50 to 39% when the criterion of ST-segment elevation of 2 mm in V1 through V6 and 1 mm in V7 through V9 was used.12 Zalenski et al13 studied posterior (V7 to V9) and right ventricular (V4R to V6R) leads to assess their accuracy compared with standard 12-lead ECG and found that these leads increased sensitivity for acute MI by 8.4% but decreased specificity by 7%. A total
of 149 patients admitted with suspected acute MI or unstable angina were studied on admission with 12-lead versus 15-lead ECG showing increased sensitivity of ST elevation for acute MI from 47.1% to 58.8% respectively, with no decrease in specificity. It was also found that 22% of patients negative for ST-segment elevation on 12-lead ECG were positive on 15-lead ECG and there was a 6-fold increase in the odds of
meeting the ECG criteria for thrombolytic therapy.14 Nevertheless, in a study investigating the use of a 15-lead ECG in
comparison with a 12-lead ECG in every patient presenting at the emergency room with chest pain, no change in diagnosis or management was proved although physicians had a more complete anatomic picture of the acute coronary syndrome.15

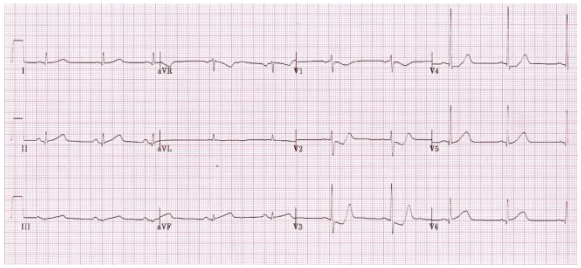
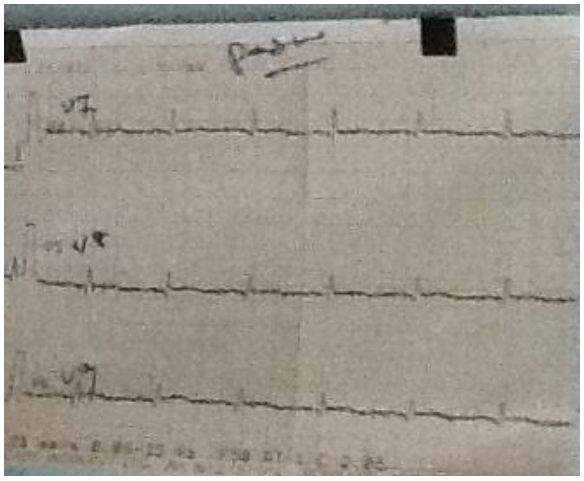


Fig: ECG showing only ST segment depression in the anterior chest leads(V7, V8 & V9)



ECG showing posterior leads with ST elevation

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