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Acute Q-Wave Versus Non Q-Wave Myocardial Infarction: Clinical and Regional Wall Motion Abnormality Studies

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Abstract

Clinical spectrum and echocardiography studies were performed in 30 patients divided into two groups: group I (QMI group) included 16 patients and group II (NOMI group) included 14 patients. Early echocardiography was done 24 hours after infarction and another one was done after 2 weeks. There was no difference between both groups as regard clinical data. There was a highly significant increased duration of symptoms before infarction ($p < 0.01$) and a significant increased number of ischaemic episodes after infarction ($p < 0.05$) in the NQMI group than QMI group. There was a highly significant increased CPK-MB enzyme in the QMI group than NQMI group ($p < 0.01$). There was no statistically significant difference between both groups as regard the segmental wall motion abnormalities early after myocardial infarction, but after 2 weeks there was a highly significant decreased wall motion score in the NQMI than the QMI groups ($p < 0.01$). There was a significant decrease in wall motion score (WMS) between early and late echocardiography ($p < 0.05$) in the NQMI group but there was a non significant difference between the two echocardiograms WMS in the QMI group. So, the present study suggested that NQMI is more unstable than the QMI in the clinical course. Improvement in regional wall motion suggested increased risk of future ischaemic events in the NQMI group. Serial echocardiographic imaging may be used to identify patients at risk for infarct extension in both QMI and NQMI patients.

Introduction

ACUTE myocardial infarction (AMI) has been divided into transmural myocardial infarction and non-transmural myocardial infarction by the evolution of pathologic Q-waves in the ECG. However, ECG has been shown to be incapable of distinguishing non-transmural infarction form transmural infarction by the use of Q-waves. So, a better terminology would be Q-wave infarction (QMI) and non-Q-wave infarction (NQMI) [1].

NQMI is associated with less myocardial necrosis and a lower in-hospital mortality rate when compared with the QMI, however, patients with NQMI are often

clinically unstable and are more likely to have post-infarction angina and recurrent myocardial infarction [2]. Patients with NQMI often have evidence of viable myocardium within the distribution of the infarct-related coronary artery and this tissue is at risk of future ischaemic events [3]. Serial studies of regional left ventricular function in control patients not given thrombolytic therapy for acute myocardial infarction have demonstrated spontaneous return of function in the area of presumed infarction [4].

The aim of this work was to study the evolution of wall motion abnormalities in a prospective series of patients with QMI and NQMI. Also, to study the clinical features of NQMI compared with those of MQI group.

Patients and Methods

The study group included 30 patients with the diagnosis of acute myocardial infarction (AMI) admitted to the coronary care unit at the El-Minia University Hospital between November 1992 and October 1993. The diagnosis of NQMI was based on the three following criteria: (1) Chest pain was compatible with AMI: (2) an abnormal elevation of serum cardiac enzymes CPK-MB; (3) an abnormal ECG i.e. persistent ST-segment depression, persistent T-wave inversion and absence of new pathologic Q-waves [1]. The diagnosis of QMI included not only the first two criteria but also the evolution of pathologic Q-waves.

All patients continuously received oral nitrates after IV nitroglycerin infusion, calcium antagonists, beta-blockers and baby aspirin 75 mg tablets. No thrombolytic therapy was given. Thorough history taking and clinical examination, ECG and serum cardiac enzymes were done for all patients. Postinfarction angina was defined as chest pain or attacks of chest oppression with ischaemic ECG changes 24 hours or later after pain onset [5]. We used Killip classification to assess functional class of the patients [6].

Two-Dimensions Echocardiography:

It was recorded with an interspace machine, model Apogee with transducer 3.5 MHz annular array sector scanner. Echocardiogram was undertaken 24 hours after admission (Echo I) and after 2 weeks (Echo II), to detect segmental wall motion abnormalities (SWA). We divided the left ventricle into 13 segments; anterior septum, posterior septum, anterolateral, posterolateral, inferior, posterior segments which were subdivided into apical and basal segments and the apex was the last segment [7].

To determine wall motion score (WMS) we used Hegar scoring system [8], where a hypokinetic, an akinetic and dyskinetic segments were assigned a score of 1,2 and 3, respectively.

Statistical analysis of the results was undertaken with chi--square test and Student's (t) test.

Results

Thirty patients were included in this study and were divided into two groups: group I, (QMI) included 16 patients, (14 males and two females) with age ranges from 49 to 62 years and a mean of 54.9±1.04 years and group II (NQMI) included 14 male patients with age ranging from 48 to 62 years and a mean of 54.7±1.19 years (Table 1).

Table II shows that there were no differences between both groups as regard

age, sex factors, history of angina and Killip classification. There was a highly significant increased duration of symptoms ($p < 0.01$) and a significantly increased post-infarction angina ($p < 0.05$) in the NQMI group than the QMI group but there was a highly significant increased level of cardiac enzymes ($p < 0.01$) in the QMI group than the NQMI group.

The individual results of the echocardiographic studies in the patients with acute QMI and NQMI are shown table (3). It shows the comparison between Echo II and between both groups as regard segmental wall motion abnormalities (akinetic and hypokinetic) and wall motion score. There was no significant difference between Echo I and Echo II in QMI group but there was a highly significant decrease in number of hypokinetic segments in Echo II than Echo I of NQMI group ($p < 0.01$). There was no significant difference between akinetic segments in Echo I and Echo II of QMI group. There was a highly significant increased number of akinetic segments in both Echo I and Echo II of the QMI patients ($p < 0.01$) than NQMI paients and a highly significant increased number of hypokinetic segments in both Echo I and Echo- II of the NQMI patients ($p < 0.01$) than the QMI patients. There was a significant decrease of wall motion score (WMS) in the Echo II than the Echo I of the NQMI patients ($p < 0.05$) and there was a significant decrease of WMS in Echo II of NQMI patients ($p < 0.01$) than Echo II of QMI patients.

Figs. 1 and 2 show the number of segments with WMA among each segment in the QMI and NQMI groups and its distribution in Echo I and Echo II. There were more abnormal segments in the anterior septum; anterolateral wall and posterolateral wall than other areas of left ventricle in the QMI patients but in the NQMI patients all the segments were nearly equally affected. It also shows that in the Echo II there was improvement in the wall motion in the apical segments more than the basal segments.

Table (1): Age and Sex Distribution in both Groups (QMI and NQMI).

		Age (yers)		Sex	
	n	Range	Mean ± S.E	Male	Female
QMI	16	49-62	54.9±1.04	14	2
NQMI	14	48-62	54.7±1.19	14	

Table (2): Comparison between the Clinical Data in QMI and NQMI Group.

	QMI n = 16	NQMI n = 14	*p*
Age (years): Mean ± S.E	54.9±1.04	54.7±1.19	M.S.
Risk factors:			
Hypertension	7 (43%)	5 (36%)	M.S.
Diabetes	5 (31%)	7 (50%)	M.S.
Smoking	12 (75%)	9 (64%)	M.S.
Family history	4 (25%)	5 (36%)	M.S.
History of angina	9 (56%)	10 (71%)	M.S.
Duration of symptoms (months):			
Mean ± S.E.	7.75±1.7	17.28±3.2	< 0.01
Post infarction angina	4 (25%)	9 (64%)	< 0.05
Killip class I	12 (75%)	11 (78%)	M.S.
Killip class II	4 (25%)	3 (21%)	M.S.
CPK-MB (IU/L) Mean ± S.E	83.3±3.8	36.5±1.9	< 0.01

Table (3): Comparison between Echo I, Echo II Segmental Wall Motion Abnormality and Wall Motion Score (WMS) between QMI and NQMI groups.

	Akinetic segments			Hypokinetic segments			WMS (Mean±S.E)		
	Echo I	Echo II	*p*	Echo I	Echo II	*p*	Echo I	Echo II	*p*
QMI	47 (22%)	44 (21%)	N.S	14 (7%)	5 (2%)	N.S	6.80± 0.82	6.31± 0.58	N.S
NQMI	22 (21%)	13 (7%)	N.S	42 (23%)	20 (10%)	< 0.01	6.07± 0.39	3.57± 0.36	< 0.05
p	< 0.01	< 0.01		< 0.01	< 0.01		N.S	< 0.01	

N.S = Not significant

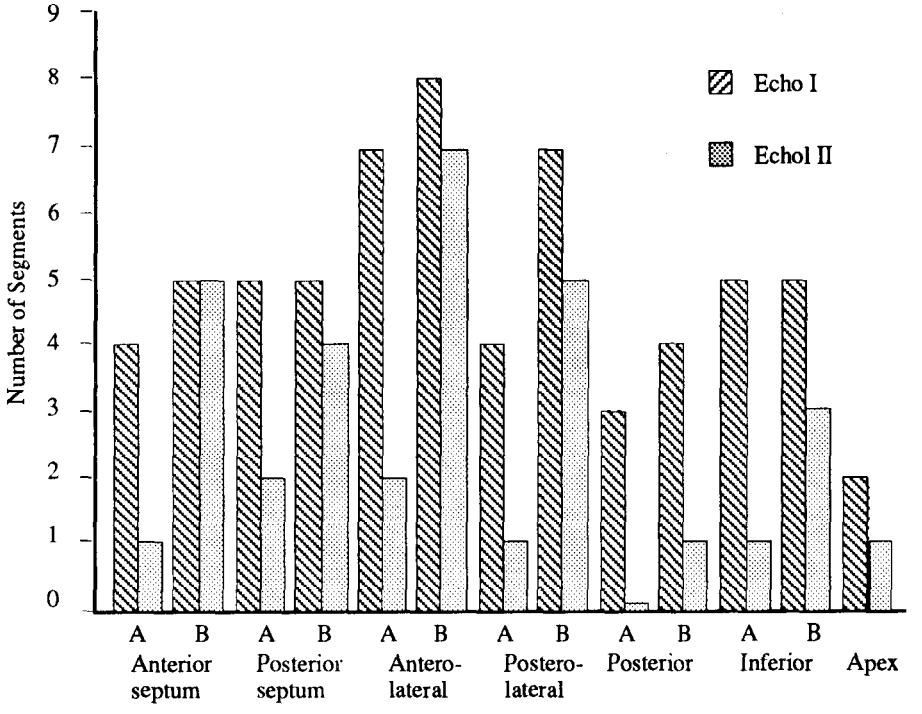

Fig. (1): Number of segments with WMA affecting each segment among NQMI group.

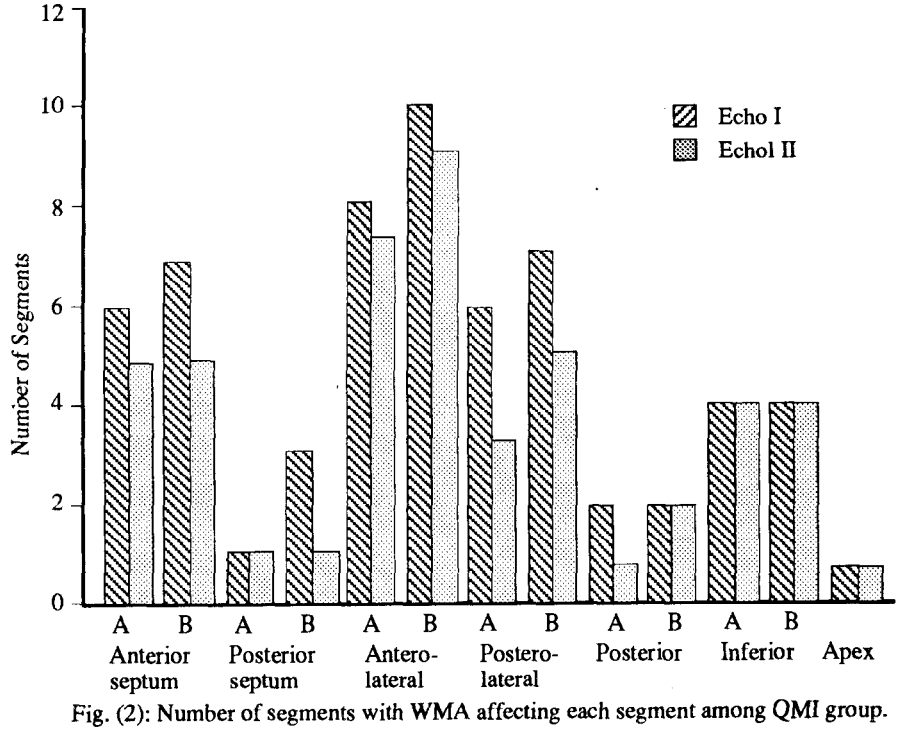

Fig. (2): Number of segments with WMA affecting each segment among QMI group.

Discussion

In the studies comparing autopsy findings with ECG changes in patients dyeing from AMI, there was a poor correlation between the presence of Q-waves and the thickness of myocardium involved by the infarction [9,10]. Therefore, Spodick [1] has suggested that infarcts, should be designated according to the presence or absence of Q-waves. In the present study, we used QMI and NQMI as better terms and compared the clinical features and echocardiographic study in both groups.

In our study, there were no differences statistically between the QMI groups with respect to age, sex, risk factors, functional classification and history of angina. These data are in accord with other reports [11,12], except the history of angina. They reported that incidence of angina was higher in the NQMI than in the QMI patients. This difference may be due to the similar clinical parameters between both groups including the risk factors of coronary atherosclerosis in our study. But we found a significant difference between both groups with respect to duration of symptoms before infarction and presence of post infarction angina in the NQMI patients than the QMI patients. Other investigators [13] reported that in hospital post-infarction angina occurred in 55% of patients with the NQMI and this ratio was significantly higher than that of QMI patients (21%). On the other hand, it was thought that the collateral vessels which supplied the peri-infarction zone prevented the extension of myocardial infarction and that the salvaged myocardial area might be susceptible to subsequent ischaemia [14].

Our results revealed that there was a significant increased CPL-MB cardiac enzyme in the QMI group than the NQMI group and this is similar to the results of other studies that have also shown a lower CPK-enzyme peak in the patients with NQMI [15]. The lower peak CPK and CPK-MB levels likely represent smaller infarctions [16].

In this study, we found that there was increased number of akinetic segments in the QMI group and increased number of hypokinetic segments in the NQMI group. There was a significant reduction of hypokinetic segments in the NQMI group after 2 weeks. These results correlate with other studies [17]. Also, there was a significant decreased WMS in the NQMI patients than the QMI patient after 2 weeks. This indicates that there were increased areas of ischaemia in the NQMI group which improved after treatment. We found that the number of abnormal segments were more localized in anterior septum, anterolateral, posterolateral wall among the QMI group but in the NQMI group, there were more or less similar distribution in all segments of left ventricle. This result suggested that the NQMI patients had mostly multivessel disease and collateral vessels. We found that the improvement in the wall motion were more in the apical segments which indicated that there were increased areas of ischaemia in the NQMI patients liable to subsequent ischaemia and recurrent infarction [13].

In conclusion, there were no clinical differences between the QMI and the NQMI patient except for duration of symptoms to give time to develop more coronary collateral vessels. On the other hand, patients with the NQMI had more frequent post-infarction angina and had wall motion improvement. These results suggest that the NQMI patients had mostly multivessel disease and more collateral vessels that may prevent much viable myocardium

from necrosis at the onset. These results suggested that serial echocadiography in initial acute myocardial infarction may be used to identify patients at high risk for infarct extension in both NQMI and QMI patients.

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