Wall motion abnormalities in Q wave and non-Q wave myocardial infarction in isolated left anterior descending coronary artery disease

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Summary

Although the frequencies of transmural involvements of Q wave and non-Q wave myocardial infarction (MI) are similar, their clinical features are different in many aspects. In the present study, the wall motion abnormalities of 34 patients with Q wave MI and eight patients with non-Q wave MI, all with isolated left anterior descending artery (LAD) lesion, were compared using left ventriculography and two-dimensional echocardiography.

This study clearly demonstrated that the severity and distribution of asynergy were significantly greater in patients with Q wave MI than in those with non-Q wave MI.

1.   Akinesis or dyskinesis was observed in all 34 patients (100%) (151 of 544 segments) with Q wave MI, and in four of eight patients (50%) (eight of 128 segments) with non-Q wave MI (p<0.05).

2.   Apical aneurysm occurred exclusively in patients with Q wave MI.

3.   In non-Q wave MI, asynergy was localized at the papillary muscle level or the apex. At the chordal level, asynergy was observed in only one of eight cases with non-Q wave MI, and in 24 of 34 cases with Q wave MI (p<0.05).

These results suggest that the infarct size may be smaller in non-Q wave MI than in Q wave MI in patients with isolated LAD lesion.

Introduction

Recent pathological studies have shown that electrocardiographic evidence of abnormal Q waves may not necessarily indicate transmural myocardial infarction, and the absence of abnormal Q waves does not necessarily exclude transmural infarction[1,2]. Thus electrocardiography may not be reliable to predict transmural infarction.

Recent studies have demonstrated several different clinical aspects of Q wave and non-Q wave infarction. These include a higher incidence of re-infarction, smaller size of infarction as determined by MB-CPK activity, lower inpatient mortality and higher late mortality in non-Q wave infarction as compared to Q wave infarction[3~6]. However, the differences in wall motion abnormalities have not been extensively studied. The present study was therefore performed to compare wall motion abnormalities of the left ventricle in patients with Q wave and non-Q wave infarction who had isolated left anterior descending coronary artery (LAD) lesion.

Materials and methods

Study population

Since 1982, 220 patients with myocardial infarction received coronary angiography from one to three month after onset of infarction at our institution. Among them, 42 patients who had one vessel disease of the LAD were included in the present study. All the patients had chest pain typical of myocardial infarction, and serial electrocardiographic changes. In the majority of patients serum levels of MB-CPK, LDH, or GOT were elevated to twice that of the upper normal range. However, in three patients, elevation of serum enzyme could not be confirmed because they were referred to our institution more than one week after onset of their chest pain. In these three patients, thallium 201 scintigrams showed a perfusion defect in the corresponding region of the electrocardiographic QRS changes. Coronary angiography demonstrated complete obstruction or stenosis of grea-

ter than 50 percent of the luminal diameter of the LAD in all the patients. No significant stenotic lesion was observed in the right coronary artery or the left circumflex branch. The patients with stenosis of the LAD of less than 50% were excluded because of difficulties in determining the coronary lesion responsible for myocardial infarction.

These patients were categorized in two groups; Q wave myocardial infarction (MI) and non-Q wave MI. The electrocardiographic criteria of abnormal Q waves were the appearance of new Q waves greater than 0.04 seconds in duration in leads I, aV$_L$ or V$_{1-6}$. Active evolutions of the ST segment and T wave changes with or without minimal QRS changes were considered non-Q wave MI. The group of Q wave MI consisted of 34 patients (33 men and one woman); and the group of non-Q wave MI, eight patients (7 men and one woman).

Wall motion analysis

Biplane left ventriculography (LVG) was performed using right anterior oblique and left anterior oblique projections (30° RAO and 60° LAO). The left ventricular wall was divided into seven segments for wall motion analysis according to the classification of the American Heart Association[7]. Two-dimensional echocardiography (2DE) was recorded one week or less before cardiac catheterization. As shown in **Fig. 1**, the parasternal short-axis views were recorded at the levels of chordae tendineae, papillary muscles and the apex. The left ventricular wall was divided into 16 segments. The interventricular septal wall was bisected into anterior and posterior halves. A line was drawn from the posterior border of the interventricular septum to the farthest portion of the opposite left ventricular wall. The free wall segment anterior to this point was judged as the anterior segment. The posterior half of the left ventricular wall was divided into three nearly equal segments including and the lateral, posterior and inferior walls. The posterior wall was usually located between the two papillary muscles. The lateral wall included the anterolateral papillary muscle; the inferior wall included the

Level of the chordae   Level of the papillary   Level of the apex
tendineae   muscles
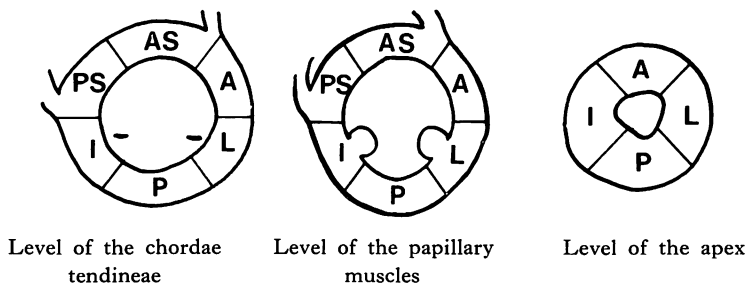
Fig. 1. Two-dimensional echocardiographic (2DE) views of the16-segment left ventricular model.

    Parasternal short-axis images at three different levels are shown. The left ventricle is divided into 6 segments at the chordae and the papillary muscle levels, and 4 segments at the apical level (see text).

    AS=anterior septum; A=anterior wall; L=lateral wall; P=posterior wall; I=inferior wall; PS= posterior septum.

posteromedial papillary muscle. In some cases, all 16 segments were not always observed satisfactorily using only the parasternal short-axis view, because the apical and lateral walls were not included in the view. Accordingly, additional apical approaches were used to visualize all of the segments. The severity of the wall motion abnormalities was assessed by three independent observers and graded as severe hypokinesis, akinesis or dyskinesis. Ventricular aneurysms were diagnosed when the endocardial contours bulged exteriorly during diastole.

Statistical analysis

The significance of categorical variables was analyzed using the chi-square test. The two groups were compared using the Student's t-test or a one-way analysis of variance. A probability of less than 0.05 was considered significant.

Results

1. Frequency of asynergy

The frequency of asynergy in patients with Q wave and non-Q wave MI was estimated (**Fig. 2**). When estimates of wall motion abnormalities differed among the same patients by 2DE and LVG, the more severe degree of asynergy (in the order of dyskinesis, akinesis and severe hypokinesis) was adopted. Although LVG was more sensitive in detecting apical asynergy than 2DE, the sensitivity of detecting asynergy in other areas was comparable between

the two methods. As shown in **Fig. 2**, akinesis or dyskinesis including aneurysmal formation was observed in all patients with Q wave MI. In non-Q wave MI, however, three cases showed hypokinesis and one had normal wall motion. Dyskinesis was observed in only one case (13%) which was significantly less than that of Q wave MI (68%, $p < 0.05$). The number of asynergic segments determined by 2DE was significantly greater in patients with Q wave MI (151 of 554 segments) than in those with non-Q wave MI (eight of 128 segments, $p < 0.05$).

2. Location of asynergy

Fig. 3 shows the frequency in the distribution of asynergy detected by 2DE. At the level of the chordae tendineae only one patient with non-Q wave MI showed wall motion abnormality (hypokinesis); whereas, 64 percent of patients with Q wave MI showed asynergy (hypokinesis or akinesis) at this level. In all patients with Q wave MI, asynergy was observed at the levels of the papillary muscle and the apex. This frequency was greatest at the segment of the anterior septum (97%). However, in patients with non-Q wave MI, asynergy was observed in three of eight patients at the papillary muscle level and in six of eight patients at the apical level. Less than 10 percent of patients with Q wave MI showed severe hypokinesis at the posterior or inferior segment.

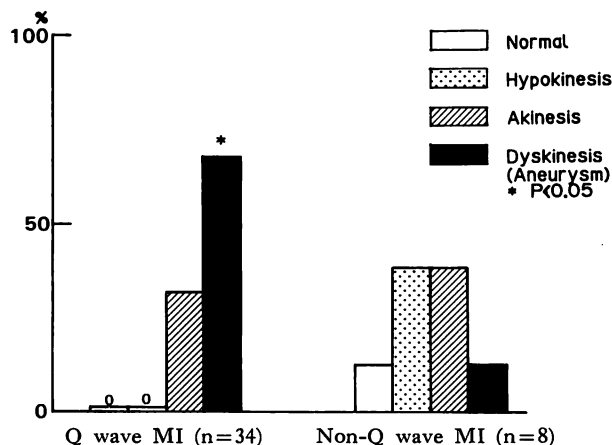The frequency of the location of asynergy

Fig. 2. Frequency of asynergy in patients with Q wave and non-Q wave myocardial infarction (MI).

Level of the chordae   Level of the papillary   Level of the apex
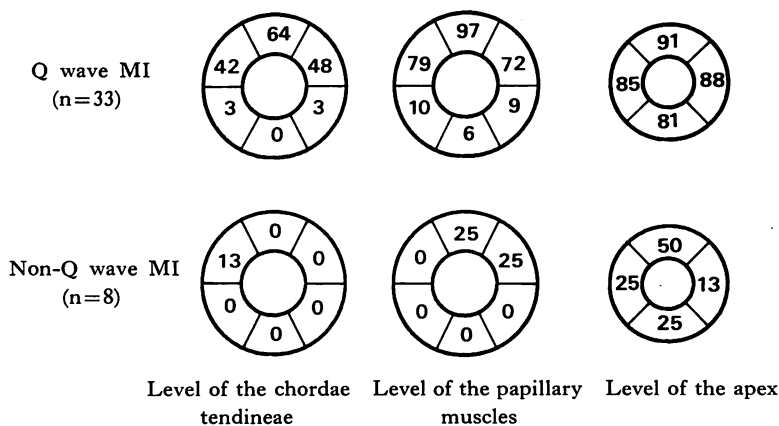tendineae   muscles

Fig. 3. Frequency and distribution of asynergy determined by 2DE in patients with Q wave and non Q wave MI.

Each circle represents the short-axis view of the left ventricle, and the segment model is the same as in Fig. 1. The percentage of patients with asynergy is shown for each segment.

as diagnosed by LVG is shown in **Fig. 4.** A marked difference was observed at the antero-lateral segment, where asynergy was observed in all cases except for one with Q wave MI compared to only one case with non-Q wave MI (p<0.05). At the apex, however, all cases with Q wave MI and seven of eight cases with non-Q wave MI showed asynergy. This indicated that the apex is the segment where asynergy

occurs exclusively in single vessel infarction of the LAD, both in Q wave and non-Q wave MI. However, apical aneurysm developed only in patients with Q wave MI (Q wave MI: 38%, non-Q wave MI: 0%).

3. Coronary lesions

Fig. 5 shows the frequency of segment and the complete coronary artery obstruction responsible for myocardial infarction, and the
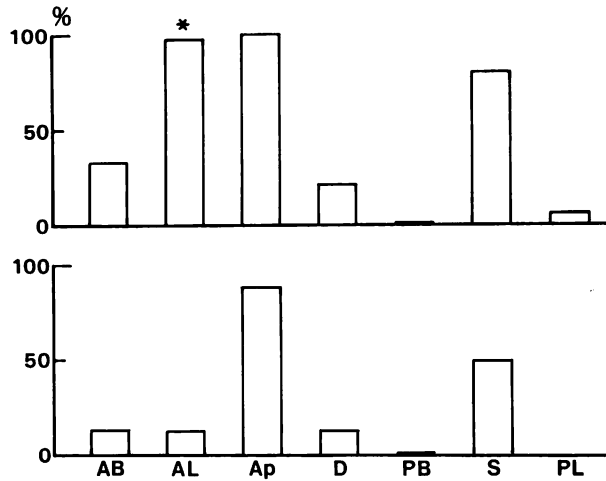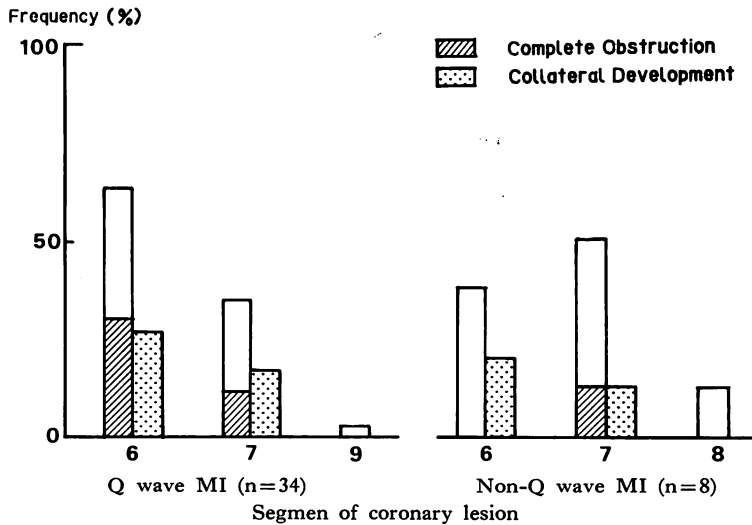
Fig. 4. **Frequency in the location of asynergy determined by left ventriculography in patients with Q wave** (upper) **and non-Q wave** (lower) **MI.**

The percentage of patients with asynergy is calculated for each segment.

AB=anterobasal; AL=anterolateral; Ap=apical; D=diaphragmatic; PB=posterobasal; S=septal; P=posterior.

* $p < 0.05$ vs non-Q wave MI

Fig. 5. **Frequency of location and complete obstruction of the coronary lesion, and development in the collateral vessels in patients with Q wave and non-Q wave MI.**

development of collateral vessels to the diseased arteries. In all cases, no other critical coronary lesion was observed. The number of cases with complete obstruction was greater among Q wave MI (14 of 34 patients, 42%) than in non Q wave MI (one of eight patients, 13%, $p < 0.05$). The segment of the coronary obstruction tended to be more proximal of the LAD (most frequent at segment 6) in Q wave MI than in non-Q wave MI (most frequent at segment 7). The degree of collateral development was similar in the two groups.

Discussion

The present study demonstrated that in patients with acute myocardial infarction with isolated LAD disease, the degree and extent of asynergy were greater in Q wave MI than in non-Q wave MI, in which asynergy was localized nearly at the anteroseptal or anterolateral wall between the papillary muscle levels and the apex. Also, left ventricular aneurysms exclusively evolved in Q wave MI. These results may indicate that the size of infarction may be smaller in non-Q wave MI than in Q wave MI. This is consistent with previous studies which showed that serum enzyme levels and the incidence of congestive heart failure were less in non-Q wave than in Q wave MI[3-5].

In this study, patients with isolated LAD disease were selected to eliminate the influences of additional ischemic area due to other diseased vessels. The difference in wall motion abnormalities between Q wave and non-Q wave MI could thus be clarified. The results might have been different if multivessel disease had been included, because large circumferential subendocardial infarction without abnormal Q waves can occasionally accompany severe heart failure. Such cases involve multivessel disease[1,8].

The reason for relatively less severe wall motion abnormalities and lower incidence of ventricular aneurysms in non Q-wave MI is not clear. However, this may be explained by the lower frequency of complete coronary occlusion and the distal site of coronary obstruction in non-Q wave MI.

Whether myocardial infarction was transmural or subendocardial in our cases of non-Q wave MI is not clear. It has been speculated that myocardial infarction without evolving abnormal Q waves is localized at the subendocardium. However, recent pathological studies demonstrated that there is no consistent correlation between transmural involvement of infarction and abnormal Q waves[1,2,8]. Experimental studies showed that obstruction of a small coronary artery results in subendocardial infarction[9], and that echocardiography is relatively insensitive in detecting wall motion abnormalities of small (less than six percent of left ventricular mass)[10] or subendocardial infarction (less than 20 percent of transmural involvement)[11]. At greater than their " threshold " levels, akinesis or dyskinesis appeared[10,11]. Therefore, it is possible that in patients of non-Q wave MI showing only hypokinesis or no apparent asynergy (four of eight cases), myocardial necrosis is localized at the subendocardium.

In assessing wall motion abnormalities we excluded cases with mild or moderate hypokinesis, because; 1) hypokinesis is nonspecific for myocardial infarction[12] ; 2) normal values are widely distributed[13] ; and 3) interobserver variability in these evaluations is relatively large. The severity of asynergy was determined by visual inspection by three independent observers. Since this impression diagnosis might have inherent limitations, several investigators recently used computer-aided determination of segmental asynergy. However, there is also a significant limitation in determining the endocardial or epicardial contours using still-frame images[14]. This effect is relatively large in the lateral fields of sector images of 2DE. Furthermore, another potential problem consists of determining the central reference point of the left ventricular cavity. Both the floating and the fixed methods may distort evaluations of segmental asynergy[13,15]. Therefore, motion images were used for analysis and fortunately there was minimal interobserver difference in determining asynergy.

In this study, few patients showed hypokinesis at the inferior and posterior walls at the choradae or papillary muscle levels. These segments are not perfused from the LAD. The mechanism of this remote asynergy is not clear. However, since coronary angiography is not entirely reliable for estimating the severity of coronary lesions[16], it is possible that some significant coronary lesions might have been present in other vessels. Other investigators have also previously reported a similar frequency of hypokinesis at the posterior or inferior walls of patients with single LAD lesion[14].

Although the cardiac apex is almost exclusively involved in isolated LAD lesion, the true apex is sometimes not imaged in apical approaches of 2DE, and the degree of asynergy tends to be underestimated. Therefore, our evaluation was made using both 2DE and LVG.

左前下行枝一枝病変例における Q 波心筋梗塞と非 Q 波心筋梗塞との左室壁運動異常の相違

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要　　約

Q 波を伴う梗塞 (Q MI) と伴わない梗塞 (non-Q MI) とでは，貫壁性心筋梗塞の頻度に差がないにも拘らず，臨床所見が異なることが近年報告されている．今回我々は，左前下行枝に一枝病変を有する Q MI 34 例と non-Q MI 8 例について，左室造影および断層心エコー図を用いて，左室壁運動異常の相違を比較した．

Non-Q MI 群では，Q-MI 群に比べ，壁運動異常の程度は明らかに小さかった．Akinesis ないし dyskinesis は Q MI の全例 (151/544 分節) にみられたが，non-Q MI では 8 例中 4 例 (8/128 分節) (p<0.05) にみられたにすぎなかった．心室瘤は non-Q MI にはみられず，Q MI の 38%

にみられた．Non-Q MI の 1 例では壁運動異常を認めなかった．Non-Q MI においては，腱索レベルでの運動異常は Q MI に比べて有意に少なく (1/8 例，vs. 24/34 例，p<0.05)，多くは乳頭筋レベルないし心尖部に限局していた (7/8 例 vs. 33/34 例).

以上の結果は，冠動脈一枝病変例においては，non-Q MI の心筋梗塞巣の拡がりは Q MI のそれよりも狭いことを示唆するものと考えられる．

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