RELATIONSHIP BETWEEN ABNORMAL Q WAVES IN LEAD aVL AND ANGIOGRAPHIC FINDINGS

—A Study to Redefine “High Lateral” Infarction—

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To re-evaluate the concept of “high lateral” myocardial infarction, angiographic findings were analysed in 29 patients with remote infarction and abnormal Q waves in lead I or aVL but no abnormal Q waves in other leads and no prominent R wave in lead V1.

All patients except one showed asynergy in the anterior segment on right anterior oblique left ventriculogram. Asynergies in other segments were, if present, mild or small. Critical narrowing was present in the diagonal branches or in their “parent” arteries (that is, the left anterior descending artery or left main trunk) in 28 patients but in the circumflex artery in only 3 patients.

Thus, the traditional concept that a “high lateral” infarction is myocardial necrosis of the basal aspect of the left ventricle, receiving its blood supply from the circumflex artery, needs correction. “High lateral infarction” corresponds to necrosis of the area between the obtuse margin and the inter-ventricular groove ordinarily supplied by the diagonal branches of the left anterior descending artery. Such infarctions are expressed by asynergy of the anterior segment rather than the posterior segment.

THE term “high lateral infarction” was first used by Wilson et al.¹ in describing the infarct of a patient with an abnormal Q wave in lead I but without abnormal Q waves in the standard precordial leads. Since then, abnormal Q waves in leads I and/or aVL in myocardial infarction have been considered to mean necrosis of the “high lateral” wall of the left ventricle. However, despite progress in angiocardiology, nuclear cardiology, echocardiography and other methods which can identify the exact site of most myocardial infarctions, the concept of “high lateral” infarction remains vague, and the

meaning of abnormal Q waves in leads I and aVL after myocardial infarction is still not clear. We studied patients with abnormal Q waves in leads I and aVL unassociated with electrocardiographic findings suggestive of anterior, inferior or posterior infarction to examine the concept of “high lateral” infarction using coronary arteriography and left ventriculography.

METHODS

Patients:

Out of the 5,000 patients having coronary arteriography between March 1975 and December 1986 in our laboratory, study patients were selected according to the following criteria:

1. The patient had a typical history of an acute myocardial infarction (that is, chest pain

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TABLE I ANGIOGRAPHIC FINDINGS OF ALL 29 PATIENTS

Case	Asynergies on Ventriculogram		Coronary Arteriogram	
	Anterior Segment	Other Segments	Narrowing	Collaterals
1	Dyskinesis	None	DIA : 99%	RCA → DIA, OM → DIA
2	Hypokinesis	None	DIA : 90% CX : 70%	None
3	Akinesis	Apex : Hypokinesis	DIA : 100%	RCA → DIA
4	Akinesis	None	DIA : 99%	RCA → DIA
5	Akinesis	None	DIA : 75% 2DIA : 75%	None
6	Akinesis	None	DIA : 99%	RCA → DIA
7	Hypokinesis	None	DIA : 100%	LAD → DIA
8	Hypokinesis	None	DIA : 75%*	None
9	Akinesis	None	2DIA : 100% LAD : 80% RCA : 75%	LAD → 2DIA
10	Dyskinesis	None	DIA : 90%	None
11	Dyskinesis	None	DIA : 99%	LAD → DIA
12	Dyskinesis	None	DIA : 100%	None
13	Akinesis	None	DIA : 80%*	None
14	Dyskinesis	None	DIA : 99% LAD : 60%	None
15	Hypokinesis	None	2DIA : 100%	DIA → 2DIA
16	Akinesis	None	DIA : 100%	OM → DIA
17	Dyskinesis	None	DIA : 99%	None
18	Hypokinesis	Apex : Hypokinesis	DIA : 80%	None
19	Hypokinesis	Apex : Hypokinesis Septum : Hypokinesis	LAD : 90%	None
20	Hypokinesis	Apex : Hypokinesis Septum : Hypokinesis	LAD : 90%	None
21	Akinesis	None	LAD : 100%	RCA → LAD
22	Akinesis	Apex : Akinesis Septum : Hypokinesis	LAD : 100%	RCA → LAD
23	Hypokinesis	Apex : Akinesis	2DIA : 90% LAD : 70% RCA : 90%	None
24	Hypokinesis	Posterior and Inferior Segment : Hypokinesis	CX : 100% RCA : 60%	RCA → CX
25	Akinesis	None	LMT : 100%	RCA → LAD, CX
26	Akinesis	None	LMT : 100%	RCA → LAD, CX
27	Akinesis	Posterior Segment : Akinesis Septum : Akinesis	LMT : 99% RCA : 90%	RCA → LAD, CX
28	Hypokinesis	Posterior Segment: Hypokinesis Apex : Hypokinesis	LAD : 90% CX : 90%	None
29	None	Apex : Hypokinesis	LAD : 90%	None

Abbreviations: DIA = 1st diagonal branch; 2DIA = 2nd diagonal branch; LAD = left anterior descending artery; RCA = right coronary artery; CX = circumflex artery; LMT = left main trunk.

*100% occlusion on angiogram in acute phase. Narrowings in LAD are proximal to the origin of the 1st diagonal branch in all subjects except for case 14.

TABLE II SUMMARY OF THE VENTRICULOGRAPHIC FINDINGS IN ALL 29 STUDY SUBJECTS

<i>Asynergy Present in the Anterior</i>	
Segment	-----28/29
<i>Dyskinesis or Akinesis in the</i>	
Anterior Segment	-----18/28
<i>Hypokinesis in the Anterior Segment</i>	
	-----10/28
<i>Asynergy solely in the Anterior</i>	
Segment	-----19/28
<i>Dyskinesis or Akinesis in Segment</i>	
Other than Anterior Segment	----- 3/28
<i>Dyskinesis or Akinesis limited</i>	
to Apex	----- 2/3
<i>Asynergy Absent in the Anterior</i>	
Segment	----- 1/29

lasting 30 min or more and elevated cardiac enzymes) that occurred at least 3 weeks before angiography.

2. The standard 12-lead electrocardiogram (ECG) of the patient showed an abnormal Q wave in lead aVL (QaVL), which may have been associated with an abnormal Q wave in lead I (QI), but there were no abnormal Q waves in leads V1 through V6, no decrease of R wave in lead V3 or V4 and no prominent R wave in lead V1.

Patients whose ECGs showed bundle branch blocks, early excitation, or hemiblocks were excluded. Patients with a clinical history suggestive of two or more infarctions were also excluded.

Twenty-nine patients met the above criteria.

ECG:

A standard 12-lead ECG was recorded within 48 hours of the angiography. An abnormal Q wave was defined as q wave of 30 msec or more.² For lead aVL, a QS pattern was considered as an abnormal Q wave only when an ECG recorded before the onset of the infarction showed an R wave. In lead V2 through V5, a q wave less than 30 msec was also considered as abnormal Q wave when it was wider and deeper than the one in the precordial leads on the left. A decrease in the R wave in lead V3 was defined as less than 0.2 mV and lower than the R wave in lead V2. It was defined in lead V4 as less than 0.4 mV and lower than the R wave in lead V3.³ A prominent R wave in lead V1 was defined as an r wave of 40 msec or more.

TABLE III SUMMARY OF CORONARY ANGIOGRAPHIC FINDINGS IN ALL 29 STUDY SUBJECTS

<i>Narrowing in Diagonal Branches</i>	
Present	-----19/29
<i>Narrowing in 1st Diagonal Branch</i>	
	-----16/19
<i>Narrowing in 2nd Diagonal Branch</i>	
	----- 4/19
<i>Narrowing solely in Diagonal Branches</i>	
	-----15/19
<i>Narrowing in Diagonal Branches</i>	
Absent	-----10/29
<i>Narrowing in Left anterior</i>	
Descending Artery	----- 6/10
<i>Narrowing in Left Main Trunk</i>	
	----- 3/10
<i>Narrowing in Circumflex Artery Present</i>	
	----- 3/29

Angiography:

Selective coronary arteriography and left ventriculography were performed after the administration of nitroglycerin or isosorbide dinitrate. Left ventriculography was recorded both in 30 degree right anterior oblique and in 60 degree left anterior oblique projections.

Cinefilms were recorded at 30 frames/sec on 35 mm film using an Arriflex camera mounted on an angiographic system (Philips Cardio-diagnost). For the names of the segments on left ventriculogram, the widely used nomenclature introduced by AHA committee⁴ was utilized but both the "anterobasal" and the "anterolateral" segments were called simply "anterior" segments in this report. Severe asynergy included akinesis and dyskinesis which were determined by quantitative measurements using a chord system.⁵

RESULTS

Left Ventriculographic Findings

Of the 29 patients thus selected, 28 (97%) had asynergy in the anterior segment (Table I and II), and 19 of these asynergies were severe ones (Table I). Among these 28, 19 patients had asynergy solely in the anterior segment and 9 patients had asynergies in other segments as well. However, 6 of these 9 patients showed only hypokinesis in segments other than the anterior segment. Two patients (cases 22 and 23) had akinesis limited to the apex and another patient (case 27) had akinesis in the anterior, septal and posterior segments.

Coronary Arteries

Among the 29 patients, 19 (66%) had critical narrowings in the first or second diagonal branches (15 in the first diagonal branch, 3 in the second diagonal branch, one in both first and second diagonal branches). Of these 19, 15 patients had narrowing exclusively in the diagonal branches (Table I and III). Of the remaining 10 patients, 9 had narrowings in the left anterior descending artery proximal to the origin of the first diagonal branch or in the left main trunk.

Thus, of all the 29 study patients, 28 (97%) had critical narrowing in the major diagonal branches themselves or in the arteries which gave rise to them.

On the other hand, only 3 (10%) patients had significant narrowing in the circumflex artery or in the obtuse marginal branch (Table I).

ECG

All 29 patients had QaVL and 5 of them (17%) showed concomitant QI. All had an inverted T wave in lead aVL. In 6 patients, precordial leads were also recorded one intercostal space above the standard leads and 5 of those 6 showed abnormal Q waves in some of these additional unipolar leads. None had abnormal Q waves in leads II, III or aVF.

DISCUSSION

The patient described by Wilson et al. in 1944¹ had a QR pattern in lead I and no abnormal Q waves in the standard precordial leads (lead aVL was not recorded). On autopsy, an infarction was found in the posterolateral wall of the left ventricle, and the authors introduced the term "high lateral" infarction for this condition. Two years later, they themselves reported 6 more cases of "high lateral" infarction diagnosed only by ECG findings, not pathological ones.⁶ All 6 patients had QaVL, 3 and QI and none had abnormal Q waves in the standard precordial leads but such waves were always found in the precordial leads recorded at higher intercostal spaces. One of these 6 patients had a large R wave in lead V1. In later reports, the findings in lead V1 or the additional precordial leads were not described and only QaVL, associated or unassociated with QI, was discussed as an electrocardiographic sign of "high lateral" infarction.⁷⁻¹¹

Although an ideal study would examine the pathological findings of many patients having

QaVL but not having other ECG findings suggestive of infarction, this has been impossible probably because the great majority of patients die from large infarctions, not from infarctions limited to the "high lateral" wall. In fact, although Myers et al.⁷⁻⁹ concluded that high lateral infarction involved the basal one-third of the lateral aspect of the left ventricle, most of their subjects had a large area of necrosis or multiple areas of necrosis and abnormal Q waves in many leads other than lead aVL. More recently, Warner et al.² studied the angiographic findings of 67 patients with abnormal Q waves in the "lateral" leads (leads I, aVL, V5 and V6) and concluded that these Q waves corresponded to infarction in the apex. However, the great majority of their subjects also had abnormal Q waves in other precordial leads or in inferior leads or had a prominent R wave in lead V1 and this made it difficult to analyse the exact relationship of the electrocardiographic and angiographic findings. In fact, among their 16 patients showing QaVL or QI without abnormal Q waves in leads V5 and V6, 11 had asynergies in the anterior segment but only 9 in the apex.

Our results indicate that patients with myocardial infarctions who exhibit QaVL, occasionally associated with QI, but who do not show ECG findings suggestive of anteroapical infarction or posterior infarction, almost always have critical narrowing in the diagonal branches, or in the left anterior descending artery in segments proximal to the origins of the major diagonal branches or in the left main trunk, the latter two of which are the "parent" arteries of the first ones. And the circumflex artery does not seem to contribute to the electrocardiographic or ventriculographic findings in such subjects. Also, in these subjects, asynergy is commonly present in the anterior segment on the right anterior oblique left ventriculogram and asynergies seen in other segments are, if present, mild or limited to small areas.

This finding conflicts with the traditional view that QaVL in such subjects indicates infarction due to occlusion of the circumflex artery or the obtuse marginal branch, which would be suggested by the first report by Wilson et al.¹ However, Dunn et al.¹² studying 84 patients with an isolated narrowing in the circumflex artery, reported that QaVL was rare in such cases. They also noted that many of these subjects had myocardial perfusion defects in the posterolateral walls but few in the "anterolateral" walls.

Movahed et al¹³ studying the ECGs of 31 patients with a first infarction associated with perfusion defects and wall motion abnormalities in the posterolateral walls, reported that QaVL was never seen in the 22 patients without concomitant perfusion defects in the "anterolateral" walls on antero-posterior scintigrams.

Thus, at least in the absence of a prominent R wave in lead V1 or abnormal Q waves or decreased R waves in the standard precordial leads, QaVL seems to correspond to myocardial necrosis in the area supplied by the diagonal branches, and these infarcts are expressed as asynergy in the anterior segment on the right anterior oblique left ventriculogram. Also, since in general the diagonal branches supply the area between the obtuse margin and the interventricular groove but do not supply the "basal" aspect of the left ventricle near the atrioventricular groove, the traditional view that "high lateral" infarction indicates a relatively limited necrosis of the "basal lateral" wall should also be altered. Movahed et al¹³ also described that QaVL was observed in only 9 subjects whose thallium defects and wall motion abnormalities extended into the anterolateral wall from the posterolateral wall. In fact, the first case reported by Wilson et al¹ itself had extensive posterolateral necrosis, not small necrosis limited to the "basal lateral" wall and showed prominent R waves in leads V1 and V2. Thus, the traditional concept regarding "high lateral" infarction that a relatively small area of necrosis in the basal lateral wall gives rise to QaVL is only an illusion. The term "high lateral" infarction should be used in a subject with a myocardial infarction who shows an "isolated" QaVL, as seen in this report. This, in turn, suggests myocardial necrosis in the area ordinarily perfused by the diagonal branches.

QaVL associated with abnormal Q waves in the precordial leads may have a different significance from the "isolated" QaVL reported here. This is because necrosis in the area supplied by the left anterior descending artery itself, that is, the anterior wall near the interventricular septum, may give rise to QaVL and asynergy in the anterior segment in the absence of an infarction of the area perfused by the diagonal branches. And it remains to be seen whether QaVL can be a reliable index of a large anterior infarction involving the whole area perfused by the left anterior descending artery in subjects with abnormal Q waves in the precordial leads.

Finally, it is also unclear whether a myocardial

infarction caused by an occlusive disease of the diagonal branches always expresses itself as "isolated" QaVL and exclusive anterior asynergy. This is because an occlusion of a large diagonal branch would give rise to necrosis of the myocardium ordinarily perfused by the left anterior descending artery itself or the obtuse marginal branch. This might be expressed as ECG and ventriculographic findings suggestive of anterior or posterior infarction as well. In fact, we previously reported 8 patients with myocardial infarction caused by occlusion of the first diagonal branch and, more than half of them showed some changes in the QRS complexes in the standard precordial leads suggestive of anterior infarction.¹⁴ Moreover, many patients were excluded from this study who had a critical narrowing solely in a diagonal branch because they had also abnormal Q waves or decrease of the R waves in the standard precordial leads.

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