Clinical, Hemodynamic and Angiographic Findings in 94 Patients with Old Myocardial Infarction

Comparison with 79 Patients with Coronary Artery Disease and No Myocardial Infarction

Carlo VIGORITO, M.D., Paolo RUSSO, M.D., Federico PISCIONE, M.D.,
Sandzo BETOCCHI M.D., Pietro GIUDICE, M.D.,
Domenico MICELI, M.D., Giulio BONZANI, M.D.,
Giuseppe GIUFFRIDA, M.D., and Mario CONDORELLI, M.D.

SUMMARY

Clinical, hemodynamic, coronary arteriographic, and ventriculo-graphic findings of 94 patients (pts) with coronary artery disease (CAD) and old myocardial infarction (MI) have been described and compared with those of 79 pts with CAD and no previous MI.

Pts with old MI presented a more compromised functional and hemodynamic status, with a higher incidence of NYHA class III-IV pts (40%), symptoms of left ventricular (LV) failure (31%), cardiomegaly (70%), higher left ventricular end-diastolic pressure (LVEDP) (19.6 ± 9.6 mmHg, $p < 0.05$), compared with 32%, 22%, 54% and 16.0 ± 6.8 mmHg respectively in CAD pts with no MI.

MI pts presented more severe CAD, higher incidences of three vessel disease (56%, $p < .005$) and of left anterior descending (LAD) (34%) and right coronary artery (RCA) (36%) occlusions, compared with 34%, 6%, and 9% respectively in pts with no MI.

Furthermore, pts with old MI showed more diffuse and severe LV segmental wall contraction abnormalities, with higher frequencies of LV aneurysm (31%) and of pts with asynergy of more than 2 LV segments (26%), compared with 1% and 3% respectively in pts with no MI.

Within the group with old MI, LVEDP was higher in pts with anterior (A) (22.0 ± 11 mmHg, $p < .02$) and anterior+diaphragmatic (A+D) (21.5 ± 7.9 mmHg, $p < .05$) MI, compared with pts with diaphragmatic (D) MI (16.6 ± 7.1 mmHg); furthermore, LVEDP increase was significantly correlated with the severity and the extent of LV asynergy.

In A and D MI, LAD and RCA stenoses or occlusions were prevalent respectively; pts with A+D MI showed larger numbers of coronary stenosis (3.05) and occlusion (1.05) per patient. The coronary artery supplying the infarcted area showed stenosis ($\geq 75\%$) in 95.6% and was normal in 2.2% of cases.

Thus, pts with CAD and old MI, and particularly of A+D and A

From the Istituto di Patologia Medica, University of Naples, II Medical School, Naples, Italy.

Address for reprint: Carlo Vigorito, M.D., Via G. Vigorito 6, 84100 Salerno, Italy.

Received for publication September 16, 1980.

MI, show a more compromised hemodynamic status and more severe degrees of CAD and of LV segmental wall contraction abnormality, which are responsible for their poor prognosis, compared to pts with CAD but no old MI.

Additional Indexing Words:

Coronary arteriography Myocardial infarction Coronary artery disease

MYOCARDIAL infarction (MI) is one of the most severe complications of coronary artery disease (CAD), being associated with a high immediate and long term mortality and with an impairment of functional capability in patients (pts) surviving the acute event. Indeed, after MI, the mortality is around 5% per year, and the incidences of left ventricular (LV) failure, angina, and recurrent MI are 2, 5, and 3 to 9% per year, respectively.¹⁾

Furthermore, sudden death has also been frequently described after MI.²⁾ The poor long term prognosis and the reduced functional status of these pts are related to the LV functional impairment, the latter reflecting the extension of the infarcted myocardial area. Several studies have described the high degree of severity of CAD and of hemodynamic and LV functional compromise in pts with old MI.^{3),7)}

This prompted us to reexamine the clinical, hemodynamic, coronary arteriographic, and ventriculographic data of pts with old MI undergone cardiac catheterization in our laboratory and to compare these data with those of pts found to have significant CAD, but without evidence of old MI.

MATERIALS AND METHODS

Study population

One hundred seventy-three pts showing a significant ($\geq 50\%$) stenosis of one of the three major coronary arteries (main left coronary artery, MLCA; left anterior descending, LAD; circumflex coronary artery, Cx; right coronary artery, RCA) at coronary arteriography in the last 2 years have been included into the study. Pts with associated valvular, congenital or other cardiac disease have been excluded.

Ninety-four pts presenting with the commonly accepted⁸⁾ electrocardiographic criteria for old MI, accompanied or not by the anamnestic and enzymatic criteria composed the MI group. Mean age was 52.1 (range 31–68 years) and mean duration of symptoms 46.7 months in this group. Mean time distance from MI was 6 months (range 2–9 months). The most commonly encountered location was diaphragmatic (D), followed by the antero-septal (AS) MI and the other various types of anterior MI.

Seventy-nine pts, presenting with no clinical, enzymatic or electrocardiographic evidence of old MI, composed the group with no MI. Mean age was 52.8 (range 28–69 years) and mean duration of symptoms 35.8 months in this group. Differ-

ences in age and duration of symptoms between the groups were not statistically significant.

Small differences between the 2 groups existed also as far as incidences of coronary risk factors (Table I). Male/female ratio was 46:1 in pts with old MI and only 3.4:1 in pts with CAD but no old MI. Unstable angina was the most common type of clinical presentation in the 2 groups (Table II), followed by symptoms of LV failure or arrhythmias in pts with MI and stable angina in pts without MI. Furthermore, pts in III-IV NYHA class⁹⁾ and with cardiomegaly on chest X-ray were most commonly found in the group of pts with MI than in the group without MI. However, none of these differences was statistically significant.

Methods of study

All pts were studied with the Sones technique.¹⁰⁾ Cardiac output was measured by the thermodilution method.¹¹⁾ Pressures were recorded by Statham P23Db transducers on an OTE photographic recorder.

All ventriculographies were obtained after nitroglycerin administration in the right and often in the left oblique projection. Coronary injection was then performed in multiple oblique projections. Angiographic images were recorded by a General Electric Fluoricon 300 image intensifier and a 105 Spot camera at 6-8 frames/sec. Severity of CAD was graded according to presence of single (SVD),

Table I. Incidences of Coronary Risk Factors in Patients with Old Myocardial Infarction (MI) and without Old MI (no MI)

	MI		no MI	
	Pts. No.	%	Pts. No.	%
Male	92/94	98	61/79	77
Female	2/94	2	18/79	23
Smoking	72/83	87	57/71	80
Systemic hypertension	16/50	32	24/57	42
Diabetes mellitus	11/49	22	10/57	18
Hyperlipidemia	21/53	40	16/52	31
Familiar history	26/48	54	21/51	41

Table II. Clinical Presentation, NYHA Class, Cardiomegaly on Chest X-ray in Patients with Old Myocardial Infarction (MI) and without Old MI (no MI)

	MI		no MI	
	Pts. No.	%	Pts. No.	%
Stable angina	23/94	24	22/79	29
Unstable angina	42/94	45	40/79	51
Dyspnea, arrhythmias	29/94	31	17/79	22
NYHA I-II	56/93	60	52/77	68
NYHA III-IV	37/93	40	25/77	32
Cardiomegaly	36/53	70	37/68	54

double (DVD) or triple (TVD) vessel CAD; evaluation of LV segmental wall motion was quantitatively performed by the Herman criteria.¹²⁾ The following LV segments have been considered for analysis: antero-basal, antero-lateral, apical, diaphragmatic, posterobasal, postero-lateral, and septal. Pts have then been subdivided in pts with normal LV motion, pts with asynergy of one, two, more than two (or diffuse) LV segments and pts with LV aneurysm. Statistical analysis was performed by means of the chi-square and Student's "t"-test.¹³⁾

RESULTS

Hemodynamic data

LV end-diastolic pressure (LVEDP) was significantly higher in pts with old MI than in those without MI (Table III), while no significant differences were found in cardiac index (CI) and LV dP/dt max. Within the group with MI, LVEDP was significantly higher in pts with anterior (A) MI and anterior+diaphragmatic (A+D) MI than in those with only diaphragmatic (D) MI (Table IV), with no significant differences in CI and LV dP/dt max between subgroups.

Coronary arteriography

Out of 94 pts with MI, 11 (12%) had SVD, 25 (27%) had DVD, 53 (56%) TVD, and 5 (5%) MLCA disease (Fig. 1). Of these latter 5, 1 pre-

Table III. Hemodynamic Data (mean $\pm$ SD) in 94 Patients with Old Myocardial Infarction (MI) and in 79 Patients without Old MI (no MI)

	LVEDP mmHg	CI L/min/M ²	LV dP/dt max mmHg/sec
MI	19.6 $\pm$ 9.6	3.20 $\pm$.86	1761 $\pm$ 690
No MI	16.0 $\pm$ 6.8*	3.20 $\pm$.95	1838 $\pm$ 646

* $p < .005$ (vs MI)

LVEDP=left ventricular end-diastolic pressure; CI=cardiac index; LV dP/dt max=maximal rate of rise of LV pressure.

Table IV. Hemodynamic Data (mean $\pm$ SD) in 94 Patients with Old MI of Different Locations

	LVEDP mmHg	CI L/min/M ²	LV dP/dt max mmHg/sec
D MI	16.6 $\pm$ 7.1	3.12 $\pm$.75	1783 $\pm$ 708
A MI	22.0 $\pm$ 11*	3.23 $\pm$.94	1721 $\pm$ 643
A+D MI	21.5 $\pm$ 7.9**	3.17 $\pm$.86	1823 $\pm$ 811

* $p < .02$; ** $p < .05$ (vs D MI)

D=diaphragmatic; A=anterior; other abbreviations as in Table III.

Fig. 1. Incidences of single (SVD), double (DVD), and triple (TVD) vessel coronary artery disease and of stenosis of main left coronary artery (MLCA) in 94 pts with old myocardial infarction (MI) and 79 pts without MI (no MI). * $p < .005$, MI vs no MI pts.

Fig. 2. Frequencies of significant ($\geq 50\%$) involvement and of complete occlusion of left anterior descending (LAD), right (RCA), and circumflex (Cx) coronary artery in 94 pts with old myocardial infarction (MI) and in 79 pts without old MI (no MI).

sented additionally TVD, 3 DVD, and 1 SVD. Total number of diseased coronaries per patient was 2.5 in this group. The difference in incidence of TVD between the 2 groups was statistically significant ($p < .005$).

LAD was the artery most commonly involved and, with RCA, also most commonly occluded in MI group (Fig. 2). Indeed, LAD was involved in 91 (97%), RCA in 79 (84%), and Cx in 60 (64%) of the 94 pts with MI; within the 79 pts without MI, LAD was involved in 72 (91%), RCA in 55 (70%), and Cx in 34 (43%) pts. Thus, the frequency of involvement of each major coronary artery was slightly, although not significantly, higher in pts with MI compared with pts without MI. Complete occlusion of the LAD was found in 32 pts (34%), of RCA in 36 pts (38%), and of Cx in 5 pts (5%) with MI; in pts without MI LAD was occluded in 5 (6%), RCA in 7 (9%), and Cx in 8 (10%) cases. Complete occlusion of MLCA has not been observed. Thus, 59 of 94 (63%) pts with MI presented at least one occlusion, and of these 46 (49%) one, 12 (13%) two, and 1 (1%) three occlusions (Fig. 3), giving a total of 1.24 occlusions/pt. Twenty of the 79 pts without MI

Fig. 3. Incidences of 1, 2, and 3 coronary artery occlusion in 94 pts with old myocardial infarction (MI) and in 79 pts without old MI (no MI).

Fig. 4. Significant stenoses ($\geq 50\%$) and complete occlusions of left anterior descending (LAD), right (RCA), circumflex (Cx), and main left (MLCA) coronary artery in 34 diaphragmatic (D), 43 anterior (A), and in 17 A+D myocardial infarctions (MI).

(25%) presented a single occlusion, and none 2 or 3 coronary occlusions (1.0 occlusion/pt).

Correlation between coronarographic findings and site of myocardial infarction (Fig. 4)

Of 34 pts with D MI, RCA was involved in all cases (100%) and occluded in 21 cases (62%), LAD involved in 31 (91%), and occluded in only 2 (6%) cases, Cx involved in 19 (56%) and occluded in 5 cases (15%), MLCA involved in only 2 cases (6%) without occlusions, giving a total of 2.52 coronaries involved and 0.8 coronaries occluded per patient. In the group of 43 pts with A MI (including 3 pts with lateral MI) LAD was involved in all cases (100%) and occluded in 21 (49%), RCA involved in 28 (65%) and occluded in 6 cases (14%), Cx involved in 25 (58%) with no occlusions, and MLCA involved in only one case (2%), giving a total of 2.25 coronaries involved and 0.6 coronaries occluded per patient in this group. Of 17 pts with A+D MI both LAD and RCA were involved in all cases (100%) and occluded in 9 (53%), while Cx and MLCA were involved in 16 (95%) and 2 cases (12%) respectively, without occlusions, giving in this last group a

total of 3.05 coronaries involved and 1.05 coronaries occluded per patient.

Left ventriculography (Fig. 5)

Of the 94 pts with MI, 8 (8%) presented a normal ventriculography, 14 (15%) asynergy of one LV segment, 19 (20%) asynergy of two LV segments, 24 (26%) asynergy of more than two segments, and 29 (31%) a LV aneurysm. Twenty-six (34%) of the 79 pts without MI had a normal LV contraction pattern, 24 (32%) showed an isolated area of asynergy, 15 (20%) two, 10 (13%) more than two areas of asynergy, and only one patient (1%) a LV aneurysm.

Hemodynamic-left ventriculography correlations (Table V)

LVEDP was significantly higher in pts with 2 segments or diffuse LV asynergy and with LV aneurysm than in pts with normal LV wall motion. LVEDP in pts with only 1 segment LV asynergy was above the normal value for our laboratory (15.0 mmHg), but was not significantly higher than in pts with normal left ventriculogram. Pts with diffuse LV asynergy showed a statistically lower value of CI, compared to pts with normal LV wall motion. No statistical differences were observed in LV dP/dt max between subgroups.

Fig. 5. Incidences of normal left ventricular (LV) motion and of asynergy of one, two or more than two (diffuse) LV segments and of LV aneurysm (AN) in 94 pts with old myocardial infarction (MI) and 79 pts without old MI (no MI).

Table V. Correlation between Hemodynamic Data (mean $\pm$ SD) and LV Wall Motion (LVWM) in 94 Patients with Old MI

	LVEDP mmHg	CI L/min/M ²	LV dP/dt max mmHg/sec
Normal	12.0 $\pm$ 4.3	3.3 $\pm$.76	2025 $\pm$ 664
Asynergy (1 segment)	15.7 $\pm$ 8.2	3.2 $\pm$ 1.1	1867 $\pm$ 945
Asynergy (2 segments)	18.6 $\pm$ 8.5*	3.2 $\pm$.86	1727 $\pm$ 769
Asynergy (diffuse)	24.3 $\pm$ 11.3**	2.5 $\pm$.91**	1729 $\pm$ 725
LV aneurysm	21.7 $\pm$ 7.3***	3.2 $\pm$.68	1611 $\pm$ 599

* $p < .05$; ** $p < .001$; *** $p < .005$ (vs Normal LVWM)

Abbreviations as in Table III.

DISCUSSION

The higher, although not statistically significant, incidence of risk factors for CAD in pts with old MI, compared with pts without MI, is found in epidemiological studies demonstrating a correlation between these factors and morbidity and mortality for cardiovascular events.¹⁴⁾ In particular, the higher male-female ratio in pts with MI confirms that sex is one of the major risk factor for MI.

The most common reason for indication to catheterize MI pts was history of unstable angina (45% of pts), especially post-infarction angina,¹⁵⁾ but many pts (31%) presented with other symptoms, such as dyspnea, and signs of cardiac failure, indicating a significant compromission of LV contractile function; this is also confirmed by the 70% incidence of cardiomegaly and the 40% incidence of pts in III-IV NYHA class, higher than those observed in pts without MI, and is in accord with the observations of reduced LV function following MI.⁴⁾

LVEDP was found to be elevated in pts with old MI in our study, as in earlier observations.^{4),16)} The elevation of LVEDP seems to be related to the extension of MI, since a direct correlation was found in our population between LVEDP and the extent of LV asynergy (Table V). Other authors had reported an abnormally high LVEDP in pts with old MI only when the asynergic LV segment was over 15–30% of LV circumference.^{17),18)} This is also confirmed in our study by the higher LVEDP found in pts with A and A+D MI than in pts with usually smaller D MI.^{19),20)}

LVEDP is inferior to other hemodynamic parameters, not evaluated in the present study, such as LV ejection fraction, volumes and compliance, in reflecting LV functional impairment.^{5),21)} Morasky et al⁵⁾ found an elevated LVEDP only in CAD pts with TVD, but not in CAD pts with SVD and DVD, who had abnormal LV ejection fraction, volumes and compliance. Also Rackley et al²¹⁾ did not find a correlation between LVEDP and LV ejection fraction and volumes in pts with different types of heart disease. Our data show that, at least in our study population, LVEDP well reflects the presence and severity of LV asynergy; its elevation may be attributed either to LV failure or reduction of LV compliance^{22),23)} secondary to the severity of LV asynergy.

The particular extension and severity of CAD in pts with MI found in our study has also been previously reported.^{3),6),7),24)} Proudfit et al⁶⁾ found at least a >90% coronary stenosis and a complete coronary occlusion in 84% and 58%, respectively, of pts with old MI; these frequencies were superior to those found in pts without MI. They also found a good correlation between the

site of MI and the stenosis or occlusion in the anatomically corresponding coronary artery. Hamby et al⁴⁾ also demonstrated that pts with old MI, more frequently than CAD pts without MI, show severe multivessel CAD. In agreement with the results of these studies, our pts with MI demonstrated a higher frequency of multivessel disease, involving all 3 major coronary arteries, and a larger prevalence of coronary occlusions (LAD and RCA), compared to pts without MI. Furthermore, a good correlation was found between the site of MI and the stenosis or occlusion of the coronary artery anatomically distributing to the infarcted area; this confirms that reduction of coronary arterial flow plays a major role in the pathophysiology of MI.

The very frequent association of significant stenoses of coronary arteries supplying non infarcted myocardial areas has also important clinical implications; progression of stenosis to complete occlusion may lead to a second MI, which carries a high mortality risk. This justifies extended criteria of indication to coronary arteriography in pts with old MI, in order to disclose significant coronary narrowings and areas of not infarcted but jeopardized ischemic myocardium potentially savable from necrosis by coronary artery bypass surgery.

Similarly to other authors,⁴⁾ we found a particular severity of CAD in pts with old A+D MI, associated in all but one pt of our study with TVD, and with high frequency of LAD and RCA occlusion.

The severe impairment of segmental LV wall motion at left ventriculography in pts with old MI is a consequence of the higher gravity and extension of CAD in these pts. Previously, abnormal LV volumes,⁷⁾ ejection fraction,^{5),7)} and wall motion^{7),26)} had been described in pts with old MI.

In summary, our results confirm that pts with old MI, especially those with old A+D MI, show more compromised hemodynamic, coronary arteriographic and left ventriculographic findings, compared to pts without MI, which can readily explain the more compromised functional status and the worst prognosis of these pts.^{1),2),26),27)}

REFERENCES

1. Kannel WB, Sorlie P, McNamara PM: Prognosis after initial myocardial infarction. The Framingham study. *Am J Cardiol* **44**: 53, 1979
2. Hagstrom RM, Billings FT, Chapnick FM, Ball DT: Sudden death in survivors of myocardial infarction. *Circulation* **30** (Suppl 111): 111-91, 111-92, 1964
3. Baldrighi V, Passoni F, Forzani G, Grugni A, Campiglio P, e Fazzi O: Rilievi coronarografici in 224 pazienti affetti da infarto del miocardio. *Boll Soc Ital Cardiol* **17**: 837, 1972
4. Hamby RI, Hoffman I, Hilsenrath J, Aintablian A, Shanies S, Padmanabhan VS: Clinical, hemodynamic and angiographic aspects of inferior and anterior myocardial infarctions in

- patients with angina pectoris. *Am J Cardiol* **34**: 513, 1974
5. Moraski RE, Russel RO Jr, Smith MK, Rackley CE: Left ventricular function in patients with and without myocardial infarction and one, two and three vessel coronary artery disease. *Am J Cardiol* **35**: 1, 1975
 6. Proudfit WL, Shirey EK, Sones MF Jr: Selective cinecoronary arteriography. Correlation with clinical findings in 1000 patients. *Circulation* **33**: 101, 1966
 7. Williams RR, Cohen PF, Vokonas PF: Electrocardiographic, arteriographic and ventriculographic correlations in transmural myocardial infarction. *Am J Cardiol* **31**: 595, 1973
 8. Rose GA, Blackburn H: Electrocardiographic reading codes (Appendix D). in *The Coronary Drug Project: Design, Methods and Baseline Results*. *Circulation* **47** (Suppl I): 39, 1973
 9. Kossman CE (Chairman): The Criteria Committee of the New York Heart Association. Diseases of the heart and blood vessels. Nomenclature and Criteria for Diagnosis, 6 Ed, Little, Brown & Co, Boston, 1964
 10. Sones FM Jr, Shirey EK: Cine coronary arteriography. *Mod Conc Cardiov Dis* **31**: 735, 1962
 11. Ganz W, Donose R, Marcus HS, Forrester JS, Swan HJC: A new technique for measurement of cardiac output by thermodilution in man. *Am J Cardiol* **27**: 392, 1971
 12. Herman MV, Heinle RA, Klein MD: Localized disorders in myocardial contraction. Asynergy and its role in congestive heart failure. *New Engl J Med* **277**: 222, 1967
 13. Snedecor GW, Cochran GW: *Statistical Methods*, Iowa University Press, Iowa, 1967
 14. Stamler J, Berkson DM, Lindenberg HA: Risk factors. Their role in the etiology and pathogenesis of the atherosclerotic diseases. in *The Pathogenesis of the Atherosclerotic Diseases*, ed by Weissler RV, Geer JC, Williams and Wilkins Co, Baltimore, p 67-69, 1972
 15. Bertolasi CA, Trongè JE, Carreno CA, Jalon J, Ruda Vega M: Unstable angina. Prospective and randomized study of its evolution, with and without surgery. Preliminary report. *Cardiol* **33**: 201, 1974
 16. Hamilton GW, Murray JA, Kennedy JW: Quantitative angiocardiology in ischemic heart disease. *Circulation* **45**: 1065, 1972
 17. Feild BJ, Russel RO Jr, Dowling JT, Rackley CE: Regional left ventricular performance in the year following myocardial infarction. *Circulation* **46**: 679, 1972
 18. Kitamura S, Kay JH, Krohn BG, Magidson O, Dunne EF: Geometrical and functional abnormalities of the left ventricle with a chronic localized non contractile area. *Am J Cardiol* **31**: 701, 1973
 19. Horan JC, Flowers NC, Johnson JC: Significance of the diagnostic Q wave of myocardial infarction. *Circulation* **43**: 428, 1971
 20. Miller RR, Olson HG, Vismara LA, Bogren HG, Amsterdam EA, Mason DT: Pump dysfunction after myocardial infarction. Importance of location, extent and pattern of abnormal left ventricular segmental contraction. *Am J Cardiol* **37**: 340, 1976
 21. Rackley CE, Hood WP, Rolett EL: Left ventricular end-diastolic pressure in chronic heart disease. *Am J Med* **48**: 310, 1970
 22. Braunwald E, Ross J Jr: The ventricular end-diastolic pressure: appraisal of its value in the recognition of ventricular failure in man. *Am J Med* **34**: 147, 1963
 23. Smith MK, Russel RO Jr, Feild BJ: Left ventricular compliance and abnormally contracting segments in post myocardial infarction patients. *Chest* **65**: 368, 1974
 24. Proudfit WL, Shirey EK, Sones MF Jr: Distribution of arterial lesions demonstrated by selective cinecoronary arteriography. *Circulation* **36**: 54, 1967
 25. Cheng TO: Incidence of ventricular aneurysm in coronary artery disease. *Am J Med* **50**: 340, 1971
 26. Bruschke AVG, Proudfit WL, Sones FM Jr: Progress study of 590 consecutive non surgical cases of coronary disease followed 5-9 years. I. Arteriographic correlations. *Circulation* **47**: 1147, 1973

27. Bruschke AVG, Proudfit WL, Sones FM Jr: Progress study of 590 consecutive non surgical cases of coronary disease followed 5-9 years. II. Ventriculographic and other correlations. *Circulation* **47**: 1154, 1973