Coronary arteriography in complicated acute myocardial infarction; clinical and angiographic correlates

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From January 1979 to June 30, 1979, we performed coronary arteriography in more than 350 patients with acute and subacute myocardial infarction.

The purpose of this paper is to study 301 patients with myocardial infarction complicated in the acute and subacute period by (1) angina, (2) acute mitral regurgitation, (3) perforation of the interventricular septum, (4) ventricular fibrillation, (5) intractable recurrent ventricular tachycardia-fibrillation, (6) heart failure, and (7) cardiogenic shock (*Table 1*).

The studies were performed within 6 hours to 30 days after acute and subacute myocardial infarction and a few hours after the onset of complications. The coronary arteriograms were performed by the Sones technique without the aid of mechanical circulatory assistance, and there were no deaths associated with the studies.

Acute and subacute myocardial infarction was diagnosed by the clinical history, electrocardiogram, and serum enzyme levels. We consider only severe obstructions (>70% narrowing).

Angina after acute and subacute myocardial infarction

Two hundred thirty-six patients had angina within 30 days of an acute and subacute myocardial

Table 1. Complicated acute and subacute myocardial infarction; severity of the obstruction in the artery responsible for the infarction; 301 patients

	No. of patients	Total obstruction	Severe obstruction
Angina after acute myocardial infarction	236	151 (64%)	85 (36%)
Mitral insufficiency after acute myocardial infarction	15	12 (80%)	3 (20%)
Interventricular septal defect after acute myocardial infarction	19	13 (66.3%)	6 (33.7%)
Ventricular fibrillation	6	3 (50%)	3 (50%)
Intractable recurrent ventricular tachycardia—fibrillation	15	12 (80%)	3 (20%)
Heart failure	8	8 (100%)	
Cardiogenic shock	2	2 (100%)	

Table 2. Acute and subacute myocardial infarction complicated by angina; 236 patients

Acute	107 (45.4%)	Transmural infarction	215 (91%)
Subacute	129 (54.6%)	Nontransmural infarction	21 (9%)

Electrocardiographic location of the acute and subacute myocardial infarction by angiographic groups

	Group					
	I	II	III	IV	V	VI
Anteroseptal	27	24	13	17		
Anterior					33	
Anterolateral						5
Inferior	3	5	4	5	38	4
Posterior		4	1		9	
Lateral		6		1	14	1
Subendocardic anterior	4	6		5		
Subendocardic anterolateral	1	5	—	1	—	—
Total	35	50	18	29	94	10

Angiographic findings

Group	No. of patients
I	35
II	50
III	18
IV	29
V	94
VI	10

infarction (*Table 2*). Usually the pain was severe, repetitive, and appeared at rest. We identify six different angiographic groups.[1]

Group I. Severe obstruction of a coronary artery (≥70%) without collateral circulation producing an area of infarction smaller than the zone perfused by that artery, with a surrounding area of ischemia. The left ventriculogram shows an area of akinesia or hypokinesia smaller than the total zone irrigated by that artery.

Group II. The same anatomy as that in group I, but in addition there are one or more severe obstructions in other coronary arteries.

Group III. Total or subtotal obstruc-

tion of an artery that produces an infarction smaller than the area irrigated by that artery because of collateral circulation to the affected zone. The artery that provides the collateral circulation as well as the remainder of the coronary arteries are free of severe obstructions. In many cases, the collateral circulation limits the extension of the infarction, but is not sufficient to supply an adequate amount of oxygenated blood to the periinfarcted area that remains ischemic. The left ventriculogram shows a zone of hypokinesia or akinesia smaller than the total area perfused by the obstructed artery.

Group IV. The same anatomical findings as in group III, associated with severe obstruction in another artery. If this artery is the one that provides the collateral circulation, the periinfarction area is seriously jeopardized.

Group V. Severe or total obstruction of a coronary artery, which produces an extensive infarction (presumably corresponding to the total area perfused by that artery) associated with severe lesions in other coronary arteries. The ventriculogram shows an area of akinesia or dyskinesia corresponding to the area perfused by the obstructed artery.

Group VI. Subtotal or total obstruction of an artery with development of a ventricular aneurysm associated with mild or moderate obstructive lesions.

The genesis of the pain can be different for each group. In groups I and III, with severe lesions in a single vessel, the pain probably originates from ischemic tissue surrounding the infarcted area. In groups II and IV, the pain can arise either from the infarcted area or from another ischemic zone or both.

In group V, the angina probably does not originate from the infarcted area, but from the ischemic regions produced by another artery.

The angina in group VI has several possible causes. (1) Patch areas of ischemic tissue within the zone of the acute necrosis, (2) increased oxygen consumption (acute ventricular aneurysm), and (3) underestimation of the severity of the obstruction in the other arteries.

Prognostic implications

In groups I and II, complete obstruction of the involved vessel can produce the extension of the previous infarction or a new infarction. We believe that in group III, the collateral circulation tends to improve oxygen supply to the ischemic area with time. Some of these patients had normal ergometric tests several months after the acute episode. Patients in group IV have the high risk of a new infarction and extension of the previous one if the artery that provides the collateral circulation became completely obstructed.

In group V, the infarcted area is large and a new infarct will cause a severe left ventricular impairment. In group VI, the prognosis is closely related to the evolution of an acute left ventricular aneurysm.

In groups III and VI, if the pain episodes are not associated with ventricular arrhythmias or pump failure, medical treatment is indicated.

We believe that postacute myocardial infarction angina is a distinct entity with characteristic clinical and angiographic findings and with a clear-cut indication for coronary arteriography.

Mitral regurgitation postacute myocardial infarction

Severe mitral regurgitation usually is a catastrophic complication of acute and subacute myocardial infarction, having an incidence of approximately 1%.[3] The mitral regurgitation is produced by rupture or dysfunction of a

papillary muscle. Papillary muscle dysfunction is more common than rupture and can produce mild to severe mitral regurgitation.[4]

Electrocardiographic location of infarction is inferior with rupture of the posterior papillary muscle and anterolateral muscle when the anteromedial muscle is affected.[5]

The clinical and angiographic findings of 15 patients are listed in *Table 3*. Thirteen patients had multiple-vessel obstructive lesions.

Two patients had single-vessel disease, the right coronary artery in one patient and circumflex in the other. Rupture or dysfunction of a papillary muscle occasionally can be produced by a single-vessel obstruction.

Cardiac catheterization should be associated with coronary angiography because of the high incidence of multiple-vessel disease in these patients. The studies should be performed in the acute phase to quantitate the hemodynamic alterations, the mitral regurgitation, delineate the coronary anatomy and the abnormalities of the left ventricle.

Table 3. Mitral insufficiency after acute myocardial infarction; 15 patients*

		No. of patients
Clinical findings		
Left ventricular failure		11
Acute pulmonary edema		3
Cardiogenic shock		1
Electrocardiographic location of the acute myocardial infarction		
Inferior wall		14
Anterior wall		1
Angiographic findings		
Multiple obstructions		13
2 vessels, RCA and Cx	1	
3 vessels, (LMT, 2 patients)	12	
Single obstruction		2
RCA	1	
Cx	1	

* Men, 13; women, 2. Mean age, 54 years; range 38 to 68 years.
RCA = right coronary artery, Cx = circumflex artery, LMT = left main trunk.

Interventricular septal defect postacute myocardial infarction

Rupture of the ventricular septum is a rare complication of acute and subacute myocardial infarction. It occurs in only 0.5% to 1% of patients with transmural myocardial infarction.[6] The superposition of a large left-to-right shunt upon acute and subacute myocardial infarction is usually a catastrophic

Table 4. Interventricular septal defect after acute myocardial infarction; 19 patients*

		No. of patients
Time between the acute infarction and the rupture of the ventricular septum		
24 hr		2
2 days		3
3 days		4
4 days		2
5 days		4
7 days		1
8 days		1
10 days		2
Clinical findings		
Congestive heart failure		7
Left ventricular failure		10
Cardiogenic shock		2
Electrocardiographic locations of the acute myocardial infarction		
Inferior wall		11
Anterior wall		8
Angiographic findings		
Multiple obstructions		11
2 vessels	8	
3 vessels	3	
Single obstruction		8
RCA	4	
ADA	4	
Left ventricular aneurysm		11
Anteroapical	6	
Diaphragmatic	5	

* Men, 13; women, 6. Mean age, 60 years; range, 43 to 81 years.
ADA = anterior descending artery.

event, and the frequent association of ventricular septal defect with acute ventricular aneurysm worsen the clinical and hemodynamic deterioration. Rupture is most frequently located in the lower portion of the septum. The defect is usually single and its size may vary from 1 mm to 4 cm.[7]

Table 4 lists the clinical, electrocardiographic, and angiographic findings in 19 patients. Eleven patients had multiple-vessel lesions. Single-vessel disease was present in eight patients, and the right coronary artery and the anterior descending artery were responsible for the acute and subacute myocardial infarction. These findings show that single obstruction of the right coronary artery or the anterior descending artery can occasionally produce perforation of the interventricular septum.

If surgical treatment is considered, cardiac catheterization and coronary arteriography should be performed and concomitant coronary bypass and/or reconstruction of the left ventricle should be done if indicated.

Acute myocardial infarction complicated with primary ventricular fibrillation

Ventricular flutter or fibrillation is the most critical cardiac arrhythmia that complicates acute and subacute myocardial infarction. Its incidence is estimated to be about 10%.[8,9] *Table 5* shows the angiographic findings in six patients with an acute myocardial infarction complicated by an episode of primary ventricular fibrillation.

Intractable recurrent primary ventricular tachycardia-fibrillation

Primary recurrent ventricular tachycardia-fibrillation is one of the most serious complications of an acute myocardial infarction. The prognosis is very

Table 5. Acute myocardial infarction complicated by ventricular fibrillation; 6 patients

	No. of patients
Anterolateral wall	2
Anterior wall	1
Anteroseptal wall	1
Inferior wall	2
Angiographic findings	
Multiple obstructions	3
2 vessels, ADA, RCA	2
3 vessels	1
Single obstruction	3
ADA	1
RCA	2

Table 6. Intractable recurrent primary ventricular tachycardia-fibrillation complicating acute and subacute myocardial infarction; 15 patients

	Patients	
	No.	%
Appearance of arrhythmias in evolution of myocardial infarction		
1st week	2	
2nd week	1	
3rd week	2	
4th week	10	
Electrocardiographic location of the acute and subacute myocardial infarction		
Anterolateral wall	4	
Anterior wall	7	
Inferior wall	3	
Lateral wall	1	
Arrhythmias present in these patients		
Ventricular premature contractions	15	100
Ventricular tachycardia	12	80
Ventricular fibrillation	15	100
Ventricular flutter	2	13
Angiographic findings		
Multiple obstruction	11	
2 vessels: 3 patients		
3 vessels: 8 patients		
Single obstruction, ADA	4	
Acute anteroapical ventricular aneurysm	8	

Table 7. Heart failure; 8 patients

	No. of patients	
Electrocardiographic location of acute myocardial infarction		
Anterior wall		7
Inferior wall		1
Previous myocardial infarction		5
Angiographic findings		
Multiple obstruction		6
2 vessels	3	
3 vessels	3	
Single obstruction, ADA		2
Acute ventricular aneurysms		6

Table 8. Cardiogenic shock; 2 patients

Electrocardiographic location of acute myocardial infarction	
Anterior wall	2 patients
(All patients had a previous inferior wall myocardial infarction)	
Angiographic findings	
ADA, 100%	
1 patient, RCA, 95%	
Cx, 100%	
Multiple obstruction:	2 patients
ADA, 100%	
1 patient, RCA, 100%	
Cx, 40%	

poor either with pharmacological or electrical measures.[10] *Table 6* shows the clinical, electrocardiographic, and angiographic findings in 15 patients with this complication. Emergency coronary arteriography allows the detection of operable defects in many of these patients (coronary artery bypass or infarctectomy or both).

Heart failure

Table 7 shows the electrocardiographic and angiographic findings in eight patients with acute myocardial infarction complicated by left ventricular failure.

Coronary angiography is indicated in these patients to rule out a left ventricular aneurysm and to decide the adequate treatment.

Cardiogenic shock

Cardiogenic shock is one of the most serious complications of acute myocardial infarction. Depression of cardiac performance in acute myocardial infarction is directly related to extent of myocardial damage. Myocardial destruction in shock usually is over 40% of the left ventricular mass.[11] The left ventriculogram in these patients shows severe and diffuse dysynergy of the left ventricular myocardium.

Table 8 lists the electrocardiographic and angiographic findings in these patients.