Angiographic findings at different time intervals from hospital admission in first non-ST elevation myocardial infarction ☆

Jaume Figueras *, José A. Barrabés, Mireia Andrés, Imanol Otaegui, Rosa-Maria Lidón, David Garcia-Dorado

Hospital Universitari Vall d'Hebron, Universitat Autònoma de Barcelona, Barcelona, Spain

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Most patients with non-ST elevation myocardial infarction (NSTEMI) present a complicated plaque with local thrombosis [1,2] but an occluded culprit artery is, apparently, not a frequent finding [3–8]. It is unclear, however, whether the presence of an occluded artery may in part relate to the time interval to coronary angiography. The few studies reporting angiographic data have provided conflicting results which in part may be attributed to the inclusion of patients with previous myocardial infarction [3–8]. Thus, we investigated the frequency of occlusion of the culprit artery in patients with a first NSTEMI and its possible relationship to the time elapsed from admission with chest pain to angiography.

The 964 consecutive patients with a first NSTEMI admitted to our Cardiology Department from January 2000 to April 2011 in whom a coronary angiography was performed within 96 h from admission were prospectively included. Diagnosis of NSTEMI was based on anginal pain ≥30 min unresponsive to nitroglycerin associated with increased troponin I levels. Patients with ST elevation, left bundle branch block, previous myocardial infarction, cardiogenic shock or associated cardiac diseases were excluded. Coronary angiography was performed at the earliest convenience but the procedure was significantly delayed on weekends, at night, or by occupancy of the catheterization laboratory. According to time to catheterization, 4 groups were established: ≤6 h (n:148); 7 to 24 h (n: 269); 25 to 48 h (n:203); and 48 to 96 h (n:344). Among patients with multivessel disease, 86 without identifiable culprit artery were excluded. Thus, a total of 878 patients were finally included. The authors of this manuscript certify they comply with the Principles of Ethical Publishing in the International Journal of Cardiology.

NSTEMI occurred at rest in 797 patients (91%), mostly without apparent triggers, and the admission ECG showed a normal tracing in 205, ST depression in 504, and negative T waves in 169. The 4 groups presented similar clinical features although patients catheterized ≤6 h showed greater ST depression which, overall, involved most frequently the anterolateral leads. Ejection fraction was similar but the rate of coronary occlusion was the highest in ≤6 h group (Table 1, Fig. 1A). These differences were more apparent among patients with ST depression but a clear trend was also documented in those with normal ECG (Fig. 1B). There were 121 patients with anterior ST depression (V2 to V4) suggesting a true posterior infarction but only 36 were catheterized within 6 h (30%, Table 1). The most frequent culprit artery was the left circumflex.

Patients with an occluded culprit artery showed a higher peak of necrosis markers, larger ST depression, more extensive coronary disease and higher involvement of the left circumflex as culprit artery than those without (Table 2). A multivariate logistic regression analysis, however, disclosed that ≤6 h time to catheterization was the most significant independent marker of coronary occlusion (with reference to 48–96 h interval) (OR 3.01, 95% CI: 1.94–4.66, $p<0.001$) followed by anterior ST depression (2.09, 1.36–3.21, $p=0.001$), active smoking (1.53, 1.10–2.11, $p=0.011$), inferolateral ST depression (1.83, 1.08–3.11, $p=0.025$) and admission Killip class ≥II (1.61, 1.02–2.54, $p=0.042$).

The main contribution of this study is the documentation of a higher rate of thrombotic coronary occlusion in first NSTEMI patients with early catheterization than in those catheterized later. Occurrence of pain at rest in most patients and lack of response to nitroglycerin would already point towards a non-vasospastic coronary occlusion as a frequent initial phenomenon. Also, coronary thrombosis in non-ST elevation acute coronary syndrome patients is well documented (52–85%) [5,6,9,10], particularly when early coronary angioscopy is performed [10–13], and an early increased rate of thrombotic occlusion commonly occurs in STEMI patients [14,15] who also present their symptoms mostly at rest [16–18].

In an early study, De Wood et al. reported a 26% incidence of coronary occlusion in 341 patients with non-Q-wave myocardial infarction catheterized ≤24 h and increased to 37%, between 24 and 72 h, and to 42%, between 72 h and 7 days [3]. However, they had no data on earlier catheterizations and their late procedures were apparently guided by recurrence of pain. In more recent NSTEMI studies with catheterizations performed at an average of 27 h [4], 27 h [19], 36 h [20], 72 h [21], and 86 h [22], the rate of coronary occlusion varied little: 31%, 32%, 20%, 29% and 27% respectively, and was similar to ours. Only 2 reports, however, have analyzed very early angiography in these patients [8,23]. Keen et al. observed a 39% occlusion rate among 28 patients with non-Q-wave myocardial infarction studied within 6 h [8], and a 51% incidence had been documented by Koyama et al., in 125 patients with non-ST elevation acute coronary syndromes studied within 5 h [23]. Nevertheless, since 22% had no increases in myocardial enzymes [23] the occlusion rate for true NSTEMI was probably higher. Moreover, in contrast with previous studies [3,4,19–23], ours did not include patients with previous myocardial infarction — which may compound the angiographic interpretation.

We recognized that ours is not a randomized study and inclusion of a larger number of patients would have increased the number of those with very early angiography. Indeed, lack of randomization possibly introduced a certain bias towards selecting high-risk patients for early angiography although their modest number was more often attributable to logistic constraints that prevented performance of emergent catheterizations. Overall, however, clinical, electrocardiographic, hemodynamic and angiographic characteristics of patients with early catheterization were quite similar to those of patients catheterized later which indicates a comparable TIMI risk score [24]. In addition, a possible transmural posterior infarction by ST depression limited to V2–V4 [25] – and hence, with higher chance for an occluded culprit artery – accounted for only 26% of patients catheterized within 6 h.

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* Corresponding author at: Unitat Coronària, Servei de Cardiologia, Hospital General Universitari Vall d'Hebron, Universitat Autònoma de Barcelona, P. Vall d'Hebron 119-129, 08035 Barcelona, Spain. Fax: +34 93 2746002.
E-mail address: 5751jfb@gmail.com (J. Figueras).

Table 1
Clinical, electrocardiographic and angiographic characteristics of patients with a first NSTEMI according to time to catheterization (mean ± SD, or n, %).

	All (n:878)	≤6 h (a) (n:141)	7–24 h (b) (n:244)	25–48 h (c) (186)	49–96 h (d) (n:307)	p value for a trend
Age, years	65 ± 12	66 ± 12	63 ± 11	64 ± 12	65 ± 12	0.097
Female (%)	259 (30)	29 (21)	75 (31)	53 (29)	102 (33)	0.024
Hypertension (%)	532 (61)	88 (62)	145 (59)	115 (62)	84 (60)	0.824
Diabetes (%)	248 (28)	35 (25)	67 (28)	54 (22)	92 (37)	0.240
Cholesterol >230 mg/dl (%)	471 (54)	76 (54)	140 (57)	91 (49)	164 (54)	0.544
Active smoking (%)	315 (36)	52 (37)	100 (41)	66 (36)	97 (32)	0.063
COPD (%)	171 (20)	26 (18)	51 (21)	34 (18)	60 (20)	0.979
PVD (%)	134 (15)	20 (14)	40 (16)	22 (12)	52 (17)	0.643
CVA (%)	57 (7)	12 (9)	7 (3)	11 (6)	27 (9)	0.203
Angina, exercise %	480 (55)	78 (55)	134 (55)	112 (60)	112 (51)	0.420
No or ≤2 days	417 (48)	68 (48)	116 (48)	81 (44)	152 (50)	0.805
3–30 days	136 (16)	20 (14)	45 (18)	29 (16)	42 (14)	0.441
>30 days	320 (36)	53 (38)	81 (33)	76 (41)	110 (36)	0.857
Angina, rest %	345 (39)	49 (35)	113 (46)	71 (38)	112 (37)	0.453
No or ≤2 days	640 (73)	111 (79)	168 (69)	137 (74)	224 (73)	0.660
3–30 days	150 (17)	18 (13)	51 (21)	32 (17)	49 (16)	0.962
>30 days	85 (10)	12 (9)	25 (10)	16 (9)	32 (10)	0.663
Killip class I (%)	770 (88)	113 (80)	221 (91)	163 (88)	273 (89)	0.086
Peak Tn I, μg/l	8.0 ± 18.1	10.6 ± 24.2	4.4 ± 10.5	8.5 ± 19.9	9.3 ± 18.4	0.004
Admission ECG, %						
Normal, %	221 (25)	31 (22)	53 (22)	60 (32)	77 (25)	0.226
ST depression, %	504 (57)	96 (68)	137 (56)	100 (54)	171 (56)	0.045
–Maximum, mm	−1.0 ± 1.3	−1.7 ± 1.4	−1.0 ± 1.0	−0.9 ± 1.0	−0.9 ± 1.0	<0.001
–∑, mm	−3.7 ± 4.8	−6.1 ± 6.3	−3.3 ± 4.4	−3.2 ± 4.3	−3.1 ± 3.9	<0.001
–n. of leads	2.7 ± 2.7	3.5 ± 2.9	2.6 ± 2.7	2.5 ± 2.7	2.5 ± 2.6	0.002
Negative T waves, %	153 (17)	14 (10)	54 (22)	26 (14)	59 (19)	0.220
ST depression site[a]						
Anterior, %	121 (14)	36 (26)	38 (16)	11 (6)	36 (12)	<0.001
Anterolateral, %	208 (24)	46 (33)	52 (21)	53 (29)	57 (19)	0.011
Lateral, %	126 (14)	11 (8)	35 (14)	28 (15)	52 (17)	0.210
Inferolateral, %	71 (8)	15 (11)	21 (9)	10 (5)	25 (8)	0.351
Inferior,%	13 (2)	2 (1)	2 (1)	2 (1)	8 (3)	0.066
Ejection fraction, %	57.5 ± 9.7	56.7 ± 11.2	58.0 ± 9.6	57.0 ± 8.8	57.8 ± 9.6	0.501
Number vessels = >70% (%)						0.002
0	95 (11)	7 (5)	22 (9)	26 (14)	40 (13)	
1	352 (40)	59 (42)	95 (39)	62 (33)	136 (44)	
2	200 (23)	28 (20)	63 (26)	43 (23)	66 (22)	
3	129 (15)	27 (19)	36 (15)	27 (15)	39 (13)	
LM	102 (12)	20 (14)	28 (12)	28 (15)	26 (9)	
Culprit coronary artery						0.403
–<50% stenosis	80 (9)	5 (4)	20 (8)	23 (12)	32 (10)	
–LAD + Dg	290 (33)	39 (28)	95 (39)	60 (32)	96 (31)	
–CF + Mg + Itm	332 (38)	70 (50)	90 (37)	64 (34)	108 (35)	
–RCA + PD + PL	124 (14)	17 (12)	24 (10)	24 (13)	59 (19)	
–LM	52 (6)	10 (7)	15 (6)	15 (8)	12 (4)	
100% occlusion	265 (30)	78 (55)	69 (28)	39 (21)	79 (26)	<0.001
Treatment prior to admission, %						
–Beta blockers	201 (23)	41 (29)	72 (30)	38 (21)	50 (16)	0.001
–Nitrates	87 (10)	17 (12)	26 (11)	22 (12)	22 (7)	0.729
–ACE inhibitors	201 (23)	35 (25)	54 (22)	43 (23)	69 (23)	0.092
–Aspirin	168 (19)	39 (28)	42 (17)	31 (17)	56 (18)	0.081
–Clopidogrel	19 (2)	4 (3)	6 (3)	2 (1)	7 (2)	0.629
–Calcium antagonists	112 (13)	13 (9)	26 (11)	24 (13)	49 (16)	0.022
–Statins	257 (29)	48 (34)	72 (30)	50 (27)	87 (28)	0.259
In-hospital treatment, %						
–Beta blockers	733 (84)	90 (64)	204 (84)	162 (88)	277 (90)	0.001
–Nitrates	864 (99)	136 (97)	240 (98)	182 (98)	306 (100)	0.013
–ACE inhibitors	102 (12)	17 (12)	33 (14)	22 (12)	30 (10)	0.266
–Aspirin	874 (100)	140 (99)	244 (100)	184 (100)	306 (100)	0.895
–Heparin	865 (99)	138 (98)	241 (98)	181 (98)	305 (99)	0.290
–Clopidogrel	236 (27)	56 (40)	85 (35)	47 (25)	48 (16)	0.001
–IIb/IIIa inhibitors	132 (15)	19 (14)	40 (16)	43 (23)	30 (10)	0.197
–Calcium antagonists	58 (7)	11 (8)	21 (9)	10 (5)	16 (5)	0.125
–Statins	180 (21)	20 (14)	76 (31)	41 (22)	43 (14)	0.032
In-hospital revascularization:						
PCI, %	542 (62)	107 (76)	152 (62)	108 (58)	175 (57)	0.001
CABG, %	141 (16)	20 (14)	47 (19)	33 (18)	41 (13)	0.365
In-hospital complications, %						
–Reinfarction	12 (1.4)	2 (1.4)	5 (2.0)	3 (1.6)	2 (0.7)	0.576
–Death	28 (3.2)	10 (7.1)	10 (4.1)	4 (2.2)	4 (1.3)	0.001
–Cardiac death	17 (1.9)	7 (5.0)	5 (2.0)	3 (1.6)	2 (0.7)	0.004

Abbreviations: COPD: chronic obstructive pulmonary disease; CVA: cerebro-vascular accident, PVD: peripheral vascular disease; LM: left main; LAD: left anterior descending; Dg: diagonal branch; CF: left circumflex; Mg: marginal branch; Itm: intermediate branch; RCA: right coronary artery; PL: posterolateral branch; PD: posterior descending branch. ACE: angiotensin converting enzyme; PCI: primary coronary intervention; CABG: coronary artery bypass grafting.

[a] They are not mutually excluded.