Multivessel spontaneous coronary artery dissection: clinical features, angiographic findings, management, and outcomes

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Background: Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome. The rate of SCAD patients with multivessel (MV) involvement varies between series (6–13%)1,2. MV SCAD might be potentially associated to a worse prognosis due to a higher ischemic burden compared with patients with single-vessel (SV) involvement. However, comparative data between patients with MV versus SV SCAD is lacking.

Methods: The Spanish multicentre nationwide SCAD registry prospectively included 389 consecutive patients from 34 university hospitals. Patients were classified according to the number of affected vessels in two groups: SV or MV SCAD. In-hospital major adverse event (MAE) was defined as a composite of death, myocardial reinfarction, unplanned revascularization, cardiogenic shock, ventricular arrhythmia or stroke. A major cardiac or cerebrovascular adverse event (MACCE) was defined as a composite of death, myocardial reinfarction, unplanned revascularization, SCAD recurrence or stroke.

Results: A total of 41 patients (10.5%) presented MV SCAD (Table 1). There were no significant differences between groups regarding age, sex and distribution of most cardiovascular risk factors, with a non-significant trend towards more hypertension in the MV group (49% vs 34%, p=0.06). MV SCAD patients had more often previous history of hypothyroidism (22% vs 11%, p=0.04) and anxiety disorder (32% vs 16%, p=0.01), with a trend towards more fibromuscular dysplasia (50% vs 29%, p=0.12) among those patients screened. MV SCAD patients presented more often with NSTEMI (73% vs 52%, p=0.01). Regarding angiographic findings, MV SCAD patients presented more frequently focal type 3 lesions (19% vs 6%, p<0.01) and fewer type 1 double-lumen lesions (12% vs 21%, p=0.04). The rate of lesions with an impaired initial Thrombolysis In Myocardial Infarction (TIMI) flow 0–1 was lower (14% vs 29%, p<0.01) in MV SCAD. In both groups, most patients were treated conservatively (71% vs 79%, p=NS). We found no significant differences between groups in MAE during admission. At long-term follow-up (median 29 months), there were no significant differences in MACCE between groups (18% vs 12%, p=0.28). However, the rate of stroke was higher in patients with MV SCAD, both in-hospital (2.4% vs 0%, p<0.01) and at follow-up (5.1% vs 0.6%, p=0.01). This finding could be explained by the basal differences found in hypertension and fibromuscular dysplasia between MV and SV SCAD patients.

Conclusions: Patients with MV SCAD have some distinctive clinical and angiographic features. We found no significant differences in our primary composite outcomes, both in-hospital and at long-term follow-up, between patients with SV and MV SCAD. Rate of stroke was significantly higher in patients with MV SCAD.

	Global (N=389, 441 lesions)	Single vessel (n=348)	Multivessel (n=41, 93 lesions)	p Value
Age, years	54 ± 11	54 ± 12	55 ± 10	0.82
Sex (female)	344 (88%)	305 (88%)	39 (95%)	0.15
Hypertension	139 (36%)	119 (34%)	20 (49%)	0.06
Connective tissue disease	2 (0.5%)	2 (0.6%)	0	0.62
Chronic inflammatory disease	18 (5%)	16 (5%)	2 (5%)	0.93
Fibromuscular dysplasia	34/106 (32%)	27/92 (29%)	7/14 (50%)	0.12
Presentation as STEMI	156 (40%)	147 (42%)	9 (22%)	0.01
Presentation as NSTEMI	211 (54%)	181 (52%)	30 (73%)	0.01
Affected coronary artery*				
Left main	10 (2%)	7 (2%)	3 (3%)	0.48
LAD	196 (44%)	162 (47%)	34 (37%)	0.08
LCX	141 (32%)	108 (31%)	33 (35%)	0.41
RCA	94 (21%)	71 (20%)	23 (25%)	0.36
Proximal segment involvement*	59 (13%)	47 (14%)	12 (13%)	0.87
Multi-segment (>1 Syntax segment)*	92 (23%)	79 (23%)	13 (29%)	0.35
Lesion length (mm)*	37 ± 24	37 ± 24	37 ± 16	0.97
Angiographic classification*				
Type 1	84 (19%)	73 (21%)	11 (12%)	0.04
Type 2a	162 (37%)	125 (36%)	37 (40%)	0.49
Type 2b	109 (25%)	89 (26%)	20 (22%)	0.41
Type 3	38 (9%)	20 (6%)	18 (19%)	<0.01
Type 4	48 (11%)	41 (12%)	7 (8%)	0.24
Initial TIMI flow*	2.2 ± 1.1	2.1 ± 1.1	2.5 ± 0.9	0.01
Initial management				0.22
Conservative	305 (78%)	276 (79%)	29 (71%)	
PCI	84 (22%)	72 (21%)	12 (29%)	
Left ventricular ejection fraction	57 ± 9	57 ± 9	58 ± 10	0.58
Treatment at discharge				
Aspirin	354 (93%)	316 (92%)	38 (95%)	0.55
DAPT	221 (58%)	193 (56%)	28 (70%)	0.10
Beta-blocker	304 (79%)	274 (80%)	30 (75%)	0.44
ACEIs/ARB	196 (51%)	174 (51%)	22 (51%)	0.62
Statins	288 (75%)	256 (75%)	32 (80%)	0.47
In-hospital MAE (n=389)	25 (6%)	21 (6%)	4 (10%)	0.35
Death	7 (2%)	6 (1.7%)	1 (2.4%)	0.74
Myocardial re-infarction	11 (3%)	10 (2.9%)	1 (2.4%)	0.87
Unplanned revascularization	17 (4%)	15 (4.3%)	2 (4.8%)	0.59
Ventricular arrhythmia	5 (1.3%)	4 (1.1%)	1 (2.4%)	0.48
Cardiogenic shock	7 (1.8%)	5 (1.4%)	2 (4.8%)	0.11
Stroke	1 (0.3%)	0	1 (2.4%)	<0.01
MACCE at follow-up (n=355)	46 (13%)	39 (12%)	7 (18%)	0.28
Death	9 (2.5%)	8 (2.5%)	1 (2.6%)	0.96
Myocardial re-infarction	27 (8%)	22 (7%)	5 (13%)	0.17
Unplanned revascularization	22 (6%)	20 (6%)	2 (5%)	0.80
SCAD recurrence	7 (2%)	5 (1.6%)	2 (5.4%)	0.12
Stroke	4 (1.1%)	2 (0.6%)	2 (5.3%)	0.01

* Analysis performed per lesion.

Table 1. Characteristics and outcomes

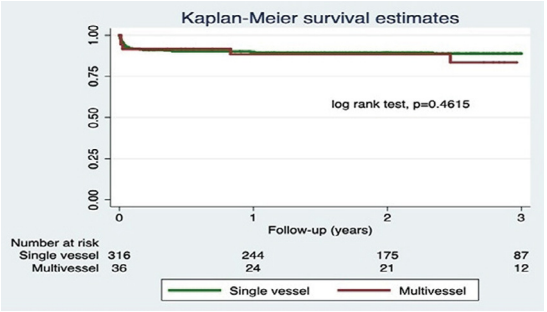

Figure 1. MACCE-free survival curves

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