Original article

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Electrocardiographic changes in patients with acute myocardial infarction caused by left main trunk occlusion

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Background **Left main trunk (LMT) occlusion is a rare angiographic finding. The primary purpose was to investigate the ECG pattern of patients with angiographic confirmation of LMT as culprit lesion.**

Methods **Forty-eight patients (32 men; mean age 73 years) with angiographic total (21 patients; 44%) or subtotal (27 patients; 56%) occlusion of the LMT were analyzed. Twenty patients (42%) were found to have concomitant coronary lesions. Eighteen patients (38%) required intra-aortic balloon pump support.**

Results **A significant left axis deviation was observed in 26 cases (54%) and an intraventricular conduction delay in 32 patients (67%). The average QRS width and QTc interval were 122 and 433 ms, respectively. ST-segment elevation in lead aVR occurred in 32 patients (67%). ST-segment elevation in leads V2–5 and in leads I and aVL [classic left anterior descending (LAD) type] was frequently observed (18 cases; 35%). In six patients (13%), a partial LAD type was observed with ST-segment elevation in leads I and aVL, but not in peripheral leads. The remaining patients showed ST-segment elevation only in peripheral leads (12 cases), in inferior leads III + aVF (six cases), whereas six patients had no ST-segment elevation. Diffuse ST-segment depression only (>6 leads) was observed in 15 cases (31%). Eighteen patients (38%) died during hospitalization.**

Conclusion **ST-segment elevation in lead aVR or a LAD-type pattern can be associated with LMT disease. Other relevant aspects are the presence of cardiogenic shock at admission, the left axis deviation and marked prolongation of QTc interval and QRS width.**

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Introduction

Decision-making in acute coronary syndrome (ACS) is generally based on a standard 12-lead ECG that represents the simplest examination for diagnosis. There is a specificity of more than 90% between the ST-segment elevation and the culprit artery considering the left anterior descending (LAD) artery, right coronary artery (RCA) and left circumflex artery (LCX).[1–5]

Left main trunk (LMT) occlusion is a rare angiographic finding. The true incidence is unknown and usually underestimated considering that most patients do not survive to have a hospital admission. In patients with acute myocardial infarction (MI) who underwent primary percutaneous coronary intervention (PCI), incidence of LMT occlusion was reported between 1.5 and 2.4%.[6–8] To date the characteristic ECG findings are not well defined as lethal arrhythmias or atrioventricular block with hemodynamic compromise often occurs.[9,10] This unstable hemodynamics in absence of preexisting intercollaterals can lead to cardiac arrest before the patient arrives at the hospital and thus a poor prognosis.[9,11–13] With the implementation of hub and spoke networks, there is an increasing interest in specific ECG patterns to diagnose LMT infarction to institute coronary interventions without any delay. In recent years, numerous studies reported that ST-segment elevation of more than 1 mm in lead aVR has been correlated with a higher rate of LMT/three-vessel disease and worse prognosis,[5,13–19] but few published studies focused their attention on ECG features in LMT infarctions.[10,18] Moreover, several studies examined the role of ST-deviation in lead aVR during exercise testing, finding a significant prognostic value in the detection of LMT and/or LAD artery disease.[20–24] Even nowadays, there is still no consistent method for diagnosis and management of this very high-risk subgroup of patients.

The purposes of this study were to investigate the ECG pattern of patients with angiographic confirmation of LMT as culprit lesion; to evaluate the effect of primary angioplasty; and to report clinical features, prognostic determinants and in-hospital cardiovascular mortality.

Methods

We retrospectively studied the medical files of all patients admitted to the emergency department and/or

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to the coronary care unit of our Institution from January 2007 through December 2016 and receiving the initial diagnosis of ACS. Forty-eight patients (32 men, 16 women; mean age 73 years) with angiographic total or subtotal occlusion of the LMT were included in the study. We excluded patients with previous MI and cardiac surgery, and lack of ECG recording close to the time of PCI. Patients had to fulfill all inclusion criteria and none of the exclusion criteria. Data on clinical history, physical examination, ECG and blood tests were retrieved from the hospital records and were entered in a dedicated database. The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki.

All patients immediately underwent coronary angiography: data about all angiograms were independently reviewed by two experienced investigators and are reported in Table 1. The infarct-related artery was identified by the absence of anterograde coronary blood flow (total occlusion). The presence of a residual anterograde blood flow (stenosis >70% and <99%) has been considered as subtotal occlusion of LMT. The angiographic pattern consisted of subtotal occlusion of the LMT in 27 patients (56%) and total occlusion in 21 patients only (44%) considering the high prehospitalization mortality. Twenty patients (42%) were found to have other coronary artery disease: a single-vessel and two-vessels disease over the LMT in 14 and 6 patients, respectively. The immediate coronary intervention consisted of percutaneous transluminal coronary angioplasty only in two cases and associated with stent delivery in the other cases (one stent in 38 patients and two/more stents in 8 patients). Patients included in the series were treated with both bare metal stents (BMS) or drug-eluting stents (DES) according to the year of admission. Eighteen patients (38%) required intra-aortic balloon pump support to stabilize the patients for acute pulmonary edema or cardiogenic shock. Detailed in-hospital and follow-up data, including age, sex, angiographic results, number of diseased vessels and in-hospital mortality were collected and reported separately in the group with total (Table 2) or subtotal (Table 3) LMT occlusion.

Table 1 Clinical and angiographic characteristic and operative data of the study population

Variable	All patients, $n = 48$
Sex	
Men, *n* (%)	32 (67%)
Female, *n* (%)	16 (33%)
Age at diagnosis (years – SD)	73 (±11 years)
Cardiovascular shock at presentation	26 (54%)
Endotracheal intubation	12 (24%)
Procedural approach	
Femoral access	31 (65%)
Radial access	12 (25%)
Femoral + radial access	5 (10%)
LMT involvement	
Total occlusion	21 (44%)
Subtotal occlusion	27 (56%)
Number of diseased vessels	
LMT only	28 (58%)
LMT + 1 vessel	14 (29%)
LMT + 2 vessels	6 (13%)
Treated vessels	
LMT	40 (83%)
LMT + CX	4 (8%)
LMT + LAD	2 (4%)
LMT + LAD + CX	2 (4%)
Intra-aortic balloon pump	18 (38%)
Percutaneous transluminal coronary angioplasty	
Without stents	2 (4%)
+1 stent	38 (79%)
+2 or more stents	8 (17%)
Type of stents	
Bare metal stents	29 (60%)
Drug-eluting stents	17 (35%)
Temporary pacemaker	3 (6%)

CX, circumflex; LAD, left anterior descending; LMT, left main trunk.

Standard 12-lead surface ECG (paper speed: 25 mm/s, calibration: 1 mV = 10 mm) was recorded as soon as possible (transmitted from Territorial Health Emergency or after admission). Standard criteria were used for the diagnosis of right bundle branch block (RBBB) and left bundle branch block (LBBB). QTc interval was measured according to Bazzet's equation (QTc = QT divided by RR root), and ST-segment elevation or depression at the most prominent point was measured 0.06 s after the J point, and a significant ST-segment change was defined as more than 0.05-mV deviation from the baseline. Inverted T waves were considered present if the T wave was biphasic or negative and at least 0.1 mV in two contiguous leads.

The clinical study end points were cardiovascular death (i.e. composite of cardiac death, fatal stroke and fatal bleeding) during the index hospitalization. Cardiac death was defined as death from cardiac cause, sudden death or any death without another known cause. Stroke was defined as sudden onset of a focal neurologic deficit lasting more than 24 h. Major bleedings were defined as bleeding requiring transfusion or surgery, decrease in hemoglobin of more than 5 g/dl and intracranial hemorrhage. Minor bleedings were defined as local hematoma and any other clinically relevant bleeding that did not meet criteria for severity. Out-of-hospital data concerning vital status of patients and cause of death were obtained by telephone interviews or independently from the Emilia-Romagna Regional Health Agency through analysis of hospital discharge records and municipal civil registries, thus relying on the treating physicians' diagnoses.

Statistical analysis

Categorical data are expressed as proportions and continuous variables reported as mean ± SD. Differences among the qualitative data were analyzed by Student's *t* test and Chi-square test. The results are reported as relative risks with 95% confidence intervals. Differences with a value of *P* less than 0.05 were considered significant. The data were recorded in a Microsoft Excel 2003 spreadsheet (Microsoft Inc., Redmond, Washington, USA).

Table 2 Characteristics of patients with acute total occlusion of the left main trunk

No.	Sex, age (years)	Cardiogenic shock	Concomitant coronary lesions (no. of vessels[a])	IABP	Treatment	Treated lesion	Number/type of stent	Temporary PM	EI	Death
1	F, 75	Yes	No	Yes	PTCA + stent	LMT	1/BMS	No	No	No
2	F, 85	Yes	No	No	PTCA	LMT	–	No	Yes	Yes
3	M,81	Yes	No	Yes	PTCA + stent	LMT	1/BMS	No	No	No
4	M,87	Yes	No	Yes	PTCA + stent	LMT	1/BMS	Yes	No	Yes
5	F, 87	Yes	No	Yes	PTCA + stent	LMT	1/BMS	No	No	Yes
6	F, 78	No	No	No	PTCA + stent	LMT	1/BMS	No	No	Yes
7	M, 63	Yes	No	Yes	PTCA + stent	LMT	1/BMS	No	Yes	No
8	M, 76	Yes	No	Yes	PTCA + stent	LMT	1/DES	No	No	No
9	M, 69	Yes	No	Yes	PTCA + stent	LMT	1/DES	No	Yes	No
10	M, 71	Yes	Yes (1)	Yes	PTCA + stent	LMT, LAD	1/BMS	No	No	No
11	M, 86	Yes	No	No	PTCA + stent	LMT	1/BMS	No	No	Yes
12	M, 65	Yes	No	Yes	PTCA + stent	LMT	1/BMS	No	No	Yes
13	F, 55	Yes	No	Yes	PTCA + stent	LMT	1/DES	No	No	No
14	F, 80	Yes	No	No	PTCA + stent	LMT	1/DES	No	Yes	Yes
15	M, 64	Yes	Yes (2)	No	PTCA + stent	LMT, LAD, CX	1/BMS	No	No	No
16	M, 49	No	No	No	PTCA	LMT	–	No	No	Yes
17	M, 66	Yes	No	Yes	PTCA + stent	LMT	1/BMS	No	Yes	Yes
18	M, 63	Yes	No	No	PTCA + stent	LMT	1/DES	Yes	Yes	Yes
19	M, 87	Yes	No	No	PTCA + stent	LMT	1/DES	No	No	Yes
20	M, 81	No	Yes (1)	No	PTCA + stent	LMT, CX	1/DES	No	No	No
21	F, 76	Yes	No	Yes	PTCA + stent	LMT	1/DES	Yes	Yes	Yes

BMS, bare metal stents; CX, circumflex; DES, drug-eluting stents; EI, endotracheal intubation; IABP, intra-aortic balloon pump; LAD, left anterior descending; LMT, left main trunk; NA, not available; PM, pacemaker; PTCA, percutaneous transluminal coronary angioplasty. [a]No. of vessels over the LMT.

Results

Baseline clinical and laboratory findings and in-hospital management are listed in Table 1. The mean age of the study population was 73 years (range 49–89), and 32 patients (67%) were men.

We decided to consider separately two groups of patients considering the angiographic characteristics: subtotal/total occlusion of the LMT. Coronary risk factors included diabetes mellitus in 12% of patients, hypertension in 76%, hypercholesterolemia in 48% and current

Table 3 Characteristics of patients with subtotal occlusion of the left main trunk

No.	Sex, age (years)	Cardiogenic shock	Concomitant coronary lesions (no. of vessels[a])	IABP	Treatment	Treated lesion	Number/type of stent	Temporary PM	EI	Death
1	M, 70	No	No	No	PTCA + stent	LMT	1/DES	No	No	No
2	F, 87	No	Yes (2)	No	PTCA + stent	LMT	1/BMS	No	No	No
3	M, 83	No	No	No	PTCA + stent	LMT	1/BMS	No	No	No
4	F, 79	No	Yes (1)	No	PTCA + stent	LMT	1/DES	No	No	No
5	M, 76	No	Yes (1)	Si	PTCA + stent	LMT, LAD	2/BMS	No	No	No
6	M, 85	No	No	No	PTCA + stent	LMT	1/BMS	No	No	Yes
7	F, 72	Yes	Yes (1)	No	PTCA + stent	LMT	1/DES	No	No	No
8	M, 69	Yes	Yes (2)	Si	PTCA + stent	LMT, LAD, CX	4/DES	No	No	No
9	M, 65	Yes	No	Si	PTCA + stent	LMT	1/BMS	No	Yes	Yes
10	M, 80	Yes	No	No	PTCA + stent	LMT	1/BMS	No	No	No
11	M, 51	Yes	No	Si	PTCA + stent	LMT	1/BMS	No	No	No
12	F, 85	No	No	No	PTCA + stent	LMT	1/BMS	No	No	Yes
13	M, 56	Yes	Yes (2)	Si	PTCA + stent	LMT, LAD, CX	2/BMS	No	No	No
14	M, 74	Yes	Yes (1)	No	PTCA + stent	LMT, CX	2/BMS	No	No	No
15	M, 58	No	No	No	PTCA + stent	LMT	1/DES	No	No	No
16	F, 70	No	No	No	PTCA + stent	LMT	1/DES	No	No	No
17	M, 78	No	Yes (1)	No	PTCA + stent	LMT, LAD	2/DES	No	Yes	Yes
18	M, 55	No	No	No	PTCA + stent	LMT	1/BMS	No	Yes	Yes
19	M, 70	Yes	Yes (2)	No	PTCA + stent	LMT, LAD, CX	2/BMS	No	Yes	Yes
20	M, 54	No	Yes (1)	No	PTCA + stent	LMT, CX	3/BMS	No	No	No
21	M, 71	No	Yes (2)	No	PTCA + stent	LMT, LAD, CX	1/BMS	No	Yes	Coma
22	F, 88	No	Yes (1)	No	PTCA + stent	LMT, CX	2/BMS	No	No	No
23	M, 76	No	Yes (1)	No	PTCA + stent	LMT, CX	2/DES	No	No	No
24	F, 89	No	Yes (1)	No	PTCA + stent	LMT, CX	1/BMS	No	No	No
25	M, 67	No	Yes (1)	No	PTCA + stent	LMT, LAD	1/BMS	No	No	No
26	F, 71	No	Yes (1)	Si	PTCA + stent	LMT, LAD	1/DES	No	No	No
27	F, 85	No	Yes (1)	No	PTCA + stent	LMT, LAD	1/BMS	No	No	No

BMS, bare metal stents; CX, circumflex; DES, drug-eluting stents; EI, endotracheal intubation; IABP, intra-aortic balloon pump; LAD, left anterior descending; LMT, left main trunk; NA, not available; PM, pacemaker; PTCA, Percutaneous transluminal coronary angioplasty. [a]No. of vessel over the LMT.

Table 4 Electrocardiographic characteristics of patients with acute total or subtotal occlusion of the left main trunk

No.	Sex, age (years)	Rhythm	Heart axis	PR	QRS	QT	QTc	RBBB	LBBB	LAHB	NIVCD	aVR > 1 mm	ST-D	ST-E
T1	F, 75	SR	33° (L)	183	87	426	388	No	No	Yes	No	No	DIII, aVF	DI, aVL, V2–V3
T2	F, 85	JR	75° (L)	–	188	470	504	No	Yes	Yes	No	Yes	DII, DIII, aVF	DI, aVL, V4–V6
T3	M, 81	SR	56° (L)	182	125	379	399	No	No	Yes	Yes	Yes	DII, DIII, aVF	DI, aVL
T4	M, 87	SR	33° (L)	194	142	476	442	No	No	No	Yes	Yes	DII, DIII, AVF, V4–V6	DI, aVL
T5	F, 87	AF	65° (R)	–	120	328	441	Yes	No	No	No	Yes	DII, DIII, aVF	DI, aVL, V1–V2
T6	F, 78	AF	6°	–	122	346	391	No	No	No	Yes	No	V5–V6	V1–V4
T7	M, 63	SR	13° (L)	138	126	338	427	No	No	No	Yes	Yes	DI–DIII, V3–V6, aVF	aVL, V1–V2
T8	M, 76	SR	56° (L)	168	146	460	431	Yes	No	Yes	No	Yes	DII, DIII, aVF	D1, aVL, V1–V3
T9	M, 69	AF	58° (L)	–	137	348	504	No	No	Yes	Yes	Yes	DII, V4–V6	V1–V2
T10	M, 71	SR	19°	158	82	360	407	No	No	No	No	Yes	DII, DIII, aVF, V2–V6	–
T11	M, 86	AF	37° (L)	–	80	376	420	No	No	No	No	No	DII, DIII, aVF	DI, aVL, V2–V6
T12	M, 65	JR	58° (L)	–	91	368	384	Yes[a]	No	No	No	No	DII, DIII, aVF	DI, aVL, V2–V4
T13	F, 55	SR	50° (L)	154	136	450	441	Yes	No	Yes	No	Yes	DII, DIII, aVF	DI, aVL, V2–V4
T14	F, 80	JR	75° (L)	–	182	460	502	No	Yes	Yes	No	Yes	DII, DIII, aVF	DI, aVL, V4–V6
T15	M, 64	AF	35°	–	116	350	490	No	No	No	Yes	Yes	DI, aVL	DII, DIII, aVF, V5–V6
T16	M, 49	JR	60° (L)	–	140	420	443	No	No	No	Yes	Yes	DII, DIII, aVF	DI, aVL
T17	M, 66	AF	53° (L)	–	150	375	446	Yes	No	Yes	No	Yes	DII, DIII, aVF	V2–V6
T18	M, 63	SR	24°	200	185	436	467	Yes	No	No	No	Yes	DII, DIII, aVF	DI, aVL, V1–V6
T19	M, 87	AF	50° (L)	–	115	330	390	No	No	Yes	Yes	Yes	DI, DII, aVL, V4–V6	–
T20	M, 81	AF	22°	–	125	385	414	No	No	No	Yes	No	V2–V6	–
T21	F, 76	SR	47° (L)	168	122	340	486	No	No	Yes	Yes	Yes	DII, DIII, aVF	DI, aVL, V2–V6
S1	M, 70	SR	73°	170	89	361	403	No	No	No	No	No	–	V4–V6 (<1 mm)
S2	F, 87	SR	13°	128	72	322	425	No	No	No	No	Yes	DII, DIII, aVF, V3–V6	V1
S3	M, 83	SR	27°	96	123	261	400	No	No	No	Yes	No	DIII, aVF	aVL, V2–V6
S4	F, 79	SR	37°	167	85	400	390	No	No	No	No	No	–	V2–V5
S5	M,76	SR	75°	205	141	359	458	Yes	No	No	No	Yes	DII, DIII, aVF, V4–V6	V1–V2
S6	M, 85	AF	32°	–	39	369	444	No	No	No	No	Yes	DI, DII, aVL, V2–V6	–
S7	F, 72	SR	39° (L)	200	132	353	425	No	No	No	Yes	Yes	DII, DIII, aVF, V3–V6	–
S8	M, 69	SR	63° (L)	136	98	486	430	No	No	No	No	Yes	DII, DIII, aVF, V3–V6	DI, aVL
S9	M, 65	SR	75° (L)	136	140	364	457	Yes	No	Yes	No	No	DII, DIII, aVF	DI, aVL, V2
S10	M, 80	SR	83°	138	112	320	458	No	No	No	Yes	Yes	DI, DII, aVL, V4–V6	DIII, aVF
S11	M, 51	SR	35° (L)	152	102	318	410	No	No	No	No	Yes	DII, DIII, aVF	DI, aVL, V1–6
S12	F, 85	AF	38°	–	70	346	411	No	No	No	No	No	–	V1–V4[b]
S13	M, 56	SR	26°	144	124	447	454	No	No	No	Yes	Yes	DI, DII, aVL, 54–V6	DIII, aVF
S14	M, 74	SR	30° (L)	178	130	348	412	No	No	No	Yes	Yes	DII, DIII, aVF, V3–V6	DI, aVL
S15	M, 58	SR	20°	160	110	330	440	No	No	No	No	Yes	DIII, aVF	DI, aVL, V1–V6
S16	F, 70	SR	79° (L)	188	169	390	440	Yes	No	Yes	No	Yes	DI, aVL, V2–V6	–
S17	M, 78	SR	28° (L)	178	82	358	402	No	No	No	No	No	DII, DIII, aVF	DI, aVL, V4–V6
S18	M, 55	AF	19°	–	162	340	540	No	No	No	Yes	No	DIII, aVF	DI, aVL, V1–V6
S19	M, 70	AF	67° (L)	–	185	370	420	Yes	No	Yes	No	Yes	DII, DIII, aVF, V1–V2	DI, aVL
S20	M, 54	SR	18°	180	129	344	400	No	No	Yes	No	Yes	V1–V4	DI, aVL, V5–V6
S21	M, 71	SR	51°	217	126	360	420	No	No	No	Yes	No	DI, aVL, V2–V6	DII, DIII, aVF
S22	F, 88	SR	4° (L)	156	150	424	464	No	Yes	No	No	No	DI, aVL	V4–V6
S23	M, 76	SR	62°	205	97	350	380	No	No	No	No	Yes	DI, aVL, V2–V6	DIII, aVF
S24	F, 89	SR	50°	192	126	380	402	No	No	No	No	Yes	DI, aVL, V2–V6	DII, DIII, aVF
S25	M, 67	SR	21° (L)	176	86	352	383	Yes[a]	No	No	No	No	–	V3–V6
S26	F, 71	SR	33°	188	184	476	546	No	Yes	No	No	Yes	DI–DIII, aVF, aVL, V5–V6	V1–V4
S27	F, 85	SR	50° (L)	180	103	406	398	No	No	Yes	No	No	–	V2–V6 [b]

AF, atrial fibrillation; JR, junctional rhythm.; LAHB, left anterior hemiblock; LBBB, left bundle branch block; NA, not available; NIVCD, nonspecific intraventricular conduction delay; RBBB, right bundle branch block; SR, sinus rhythm; ST-D, ST-segment depression; ST-E, ST-segment elevation. [a] Incomplete. [b] With Q waves in the same site and inferior.

smoking in 52%, whereas 21 patients (44%) had a history of preinfarction angina. The ratio of underlying disease and risk factors, such as diabetes mellitus, hypertension, hyperlipidemia and smoking, was NS by different among the two groups. Twenty-six patients (54%) were in cardiogenic shock at presentation.

As presented in Table 4, 32 patients had sinus rhythm at presentation, whereas 11 atrial fibrillation, one flutter and four junctional rhythm. The average QRS width and QTc interval were 122 and 433 ms, respectively, and the QRS axis showed a significant left axis deviation: 26 cases (54%). The left axis deviation was associated with left anterior hemiblock (LAHB) in 14 cases. An intraventricular conduction delay (QRS width >110 ms) was observed in 32 patients with a mean QRS width of 140 ms: a RBBB occurred in nine cases (28%), a LBBB in four cases and nonspecific intraventricular conduction delay in 19 cases (59%). Abnormal Q wave was seen in two patients in leads V1–V4 and V2–V6. ST-segment elevation in lead aVR occurred in 32 patients (67%) and represented the most frequent finding in patients with LMT disease. A diffuse ST-segment depression (in >6 leads) was observed in 15 cases (31%). The ECG patterns are reported in Table 5. An anteroseptal and lateral infarction appearance with ST-segment elevation in leads V2–5 and in leads I and aVL (classic LAD type) was the most frequently observed (18 cases; 35%), with

Table 5 ECG patterns in patients with acute myocardial infarction caused by left main trunk occlusion

ECG patterns	No.	%
Classic LAD type[a]	18	37.5
Partial LAD type[b]	6	12.5
ST-segment elevation only in peripheral leads	12	25
ST-segment elevation in inferior leads (III + aVF)	6	12.5
No ST-segment elevation	6	12.5
Diffuse ST-segment depression (>6 leads)	15	31.3
ST-segment elevation in lead aVR	32	66.7

LAD, left anterior descending. [a] ST-segment elevation in leads V2–5 and in leads I and aVL. [b] ST-segment elevation in leads I and aVL, but not in peripheral leads.

ST-segment depression in leads II, III and aVF via a reciprocal change was observed in all of this type (Table 5).

Eighteen patients (38%) died in the cardiac catheterization laboratory or during hospitalization due to profound shock, pump failure or refractory ventricular fibrillation, whereas one patient remained in coma status at last follow-up.

Discussion

The LMT as the culprit artery in ACS rapidly triggers cardiogenic shock with severe left ventricular dysfunction, ventricular fibrillation, pulmonary edema and acute respiratory failure, with high mortality before reaching the emergency department.[9,25,26] This is particularly true in most of the cases with acute total occlusion of the artery. However, with the improvement of operative networks for ACS consisting in telemedicine equipment and 24-h catheter facility availability, new treatments and logistic systems of care, more patients with complete/subtotal occlusion of the LMT are seen in clinical practice. In this scenario, it is well known that electrocardiography plays an important role in prognostic stratification and management of patients with non-ST-segment elevation MI (NSTEMI).[27] The current study shows that angiographic total or subtotal occlusion of the LMT is frequently associated with ST-segment elevation in lead aVR (67% of the cases) and with anteroseptal/lateral infarction appearance with ST-segment elevation in leads V2–5 and in leads I and aVL (so-called LAD type; 35% of the cases).

Different ECG patterns related to occlusion of the LMT have been described.[8,10,13,28,29] These patients presented with either NSTEMI or ST-segment elevation MI (STEMI) patterns. The typical pattern in NSTEMI cases consisted of global ischemia ECG appearance, with widespread ST-segment depression in seven or more leads and reciprocal ST-segment elevation in lead aVR and often V1.[11,17,28,30–33] When occlusion is total and transmural, it corresponds to the clinical syndrome STEMI and shows a corresponding pattern in the ECG. Hirano *et al.*[10] on their analysis reported that the ECG features of LMT infarctions can be classified into two main groups: one with RBBB with marked left axis deviation [RBBB + left axis deviation (LADEV) type] or northwest axis, and the other with an anteroseptal and lateral infarction appearance with ST-segment elevation in leads V2–5 and in leads I and aVL (LAD type). The RBBB + LADEV types and the LAD types occurred in 37.1 and 51.4%, respectively.[10] In our series, only eight patients had the RBBB + LADEV pattern (17%), whereas 50% of the patients had the LAD types (partial or complete). Fiol *et al.*[8] reported that the most typical ECG pattern, in a small series of complete occlusion of LMT without collateral circulation, was ST-segment elevation in the precordial leads from V2 to V4 through V6 and in leads I and aVL, as well as ST depression in the inferior leads often with RBBB and LAHB. As reported in Table 4, few patients of our series presented with this long-LAD pattern of ST-segment elevation.

The significance of ST-segment change in lead aVR in LMT infarction has been underestimated and considered only as an aspect related to changes from the left lateral side (i.e. leads I, aVL, V5 and V6) until the last decade.[5,15] Recent studies focused their attention on this topic and revealed that ST-segment elevation in lead aVR is frequent in LMT obstruction.[10,13,17–19,34–36] A large collaborative international meta-analysis including 22 740 patients with left main disease and three-vessel disease reported that an extent ST-segment elevation in lead aVR on 12-lead ECG was one of the most powerful predictors of disease.[37] Taglieri *et al.*[13] reported that patients with diffuse ST-segment depression and ST-segment elevation in lead aVR showed the highest rate of LMT or LMT/3-vessel disease, with an increased risk of culprit LMT disease, as well as an increased risk of cardiovascular death. At multivariate analysis, this pattern was independently associated with an increased risk of overall LMT disease with an adjusted odds ratio of 3.82 ($P < 0.001$).[13] The presence of ST-segment elevation in lead aVR was frequently observed also in our selected series of patients with LMT disease.

The electrogenesis and ECG appearance in LMT occlusion are attributable to numerous factors, but usually the ST vector identify basal interventricular septum as the anatomical area subject of transmural ischemia.[4,17,19,34,36] However, a simultaneous ST-segment elevation in lead V1 could be present as the effect induced by ischemia in anterior vectors is offset by posterior vectors.[10,34] On the other hand, the absence of ST-segment elevation in lead V1 despite proximal occlusion of LAD is justified by the presence of a large conal branch of the RCA, able to perfuse the high septum instead of first septal branch.[38] Hirano *et al.*[10] clearly reported that ST-segment elevation in aVR in LMT infarction can be explained by 'severe ischemia in the basal part of the septum and in the lateral part of the heart, which interferes with the blood flow in the LAD and LCX and produces another injury induced electric current toward the right upper part of the heart'.

Moreover, the ST change in lead aVR indicates that the culprit lesion is located proximal to the first septal branch,[35] underlies transmural ischemia of the basal septum[13,28,35] and can be distinguished considering the association with ST-segment elevation in V1.[28,39] In our ECG analysis, ST-segment elevation in lead aVR was associated with ST-segment elevation in lead V1 in nine cases only, confirming the results of previously reported studies about that severe cardiac ischemia, such as LMT infarction and extended anterior infarction in LAD occlusion, can elevate the ST-segment in lead aVR but not lead V1.[28,39]

LMT infarction may initially induce severe ischemia in the anteroseptal and posterolateral sites of the heart, which would lead to significant left axis deviation,[7] decreased cardiac output and relative ischemia in the RCA area. Delays in the depolarization and repolarization process are caused by the severe myocardial acidosis and ischemia in the entire heart, and this is clinically evident as QTc and QRS prolongation.[40,41]

In a large study of more than 100 patients with NSTE, it has been reported that short-term and long-term mortality was significantly associated with the ECG pattern of widespread ST-segment depression and ST-elevation in lead aVR.[13] Fiol *et al.*[8] reported seven patients with total LMT occlusion, observing an in-hospital mortality of 80%. In our study, despite the severe clinical state of our patients at admission, most of them survived with an in-hospital mortality of 38%. However, due to the retrospective nature of the study, the series included patients treated with both BMS or DES. This should be considered a limitation in the survival analysis, considering that in the DES era, a more favorable outcome for LMT disease has been reported.[42]

Conclusion

The present findings suggest that evaluation of ST-segment elevation in lead aVR or a LAD-type pattern (sometimes with intraventricular conduction disturbance such as RBBB) on admission ECG may be useful to improve risk stratification and management of patients with ACS, as they could be associated with LMT disease. Other relevant aspects are the presence of cardiogenic shock at admission, the left axis deviation and marked prolongation of QTc interval and QRS width.

In these cases, we suggest the urgent need for a PCI or surgical treatment.

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Conflict of interest

There are no conflicts of interest.

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