SAS Journal of Medicine
Abbreviated Key Title: SAS J Med
ISSN 2454-5112
Journal homepage: https://saspublishers.com

OPEN ACCESS

Predictive Factors of Left Ventricle (LV) Dysfunction in Acute Myocardial Infarction (AMI): A Prospective Study

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DOI: 10.36347/sasjm.2021.v07i09.005 | **Received:** 28.07.2021 | **Accepted:** 03.09.2021 | **Published:** 08.09.2021

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Abstract **Original Research Article**

Left ventricular dysfunction (LV dysfunction) complicating myocardial infarction is common. Among patients with acute myocardial infarction (AMI), a strong relationship between the degree of LV dysfunction and mortality has been demonstrated. LV dysfunction is known to be a strong predictor of death in AMI with important implications for prognosis and treatment. The identification of patients at risk of developing LV dysfunction after AMI is relevant in the sense that it allows early and optimal management of heart failure for better quality of life. This prospective study of 75 cases of AMI complicated or not with LV dysfunction, hospitalized in the intensive care unit of the Mohammed V military training hospital in Rabat, covering a period of 6 months, aims to determine the predictive factors of LV dysfunction after an AMI in a Moroccan population group. It was found that male gender, delay in management, heart rate on admission, electrical territory, and the existence of crackling sounds on lungs at admission are predisposing factors for left ventricular systolic dysfunction. However, dyslipidemia is a protective factor. Early management of these patients would reduce the risk of reinfarction and death.

Keywords: Left ventricle dysfunction, predictive factors, myocardial infarction.

INTRODUCTION

Despite numerous diagnostic and therapeutic means, acute myocardial infarction (AMI) remains a frequent cause of morbidity and mortality today. Left ventricular systolic dysfunction after an AMI worsens the prognosis and increases mortality [1]. The identification of patients at risk of developing LV dysfunction after an acute myocardial infarction is of prognostic and therapeutic interest. Beta-blockers and vascular filling fluids that are potentially dangerous in the acute phase in AMI complicated by LV dysfunction can be avoided in the acute phase [2]. In the post-infarction phase, the timely introduction of treatment for heart failure, particularly aldosterone antagonists, would allow early control of symptoms and an improvement in quality of life [3]. Our work therefore aims to determine the predictive factors for the occurrence of left ventricular systolic dysfunction after an AMI.

METHODS

We conducted a prospective study of 75 cases of AMI complicated or not with LV dysfunction, hospitalized in the intensive care unit of the Mohammed V military training hospital in Rabat, covering a period of 6 months, from January 2021 to June 2021.

A total of 110 patients were admitted to the department during this period for chest pain. We included patients with AMI evolving less than 12 hours after the presumed onset of pain, or more.

We excluded patients with unstable angina without troponin elevation, patients admitted for NSTEMI, and patients already followed for heart failure with LV dysfunction. Of the 75 cases of AMI, 32 patients arrived less than 12 hours after the presumed onset of pain.

The data were collected in data sheets drawn up for this purpose and giving information on clinical, electrocardiographic, biological and echocardiogaphic elements (the collection was initiated from the patient's admission until his discharge.)

Left ventricular dysfunction was defined by an ejection fraction ≤ 40% on echocardiography and considered severe for a left ventricular ejection fraction (LVEF) of less than 30%.

Statistical analysis was performed with SPSS software. For comparisons of variables between the two groups, we used Fisher's F test and the median test.

The median test was used when the Fisher F-test was not suitable. Quantitative variables were expressed as means +/- standard deviations and qualitative variables as percentages. For both tests used, the significance threshold corresponds to a p-value of less than 0.05.

RESULTS

Characteristics of the general population

The mean age of the population was 61.04+/-2.056 years with 81.3% of male. Among the patients, 49.3% were diabetic, 41.3% were hypertensive, 49.3% were smokers. Dyslipidemia was found in 21.3% of cases and coronary heredity in 6.7% of cases. 28.2% of the patients were in acute heart failure with lungs crackling sound. The incidence of LV dysfunction was 45.3%, of which 12% had severe LV dysfunction.

Table-1: Characteristics of the population

Characteristics	General population
Age	61.04+/-2.056
Male	81.3%
Diabetes	49.3%
High blood pressure	41.3%
Smocking	49.3%
Dyslipidaemia	21.3%
Coronary heredity	6.7%
Delay of less than 12 hours	42.7%
Acute heart failure	28.2%
Electrical territories :	
Anteroseptal	8%
Anteroseptoapical	18.7%
Extended anterior	18.7%
Inferior	16%
Inferobasal	16%
Inferior extended to the right ventricle	6.7%
Deep Septal	4%
High lateral	4%
Basal	5.3%
Circumferential	2.7%
Kinetic disorders such as akinesia:	53.3%
Site of akinesia	
anterior	35%
inferior	20%
septal	5%
Anterior and inferior	10%
Anterior and septal	22.5%
Inferior and septal	2.5%
Anterior, inferior and septal	5%
LVEF :	
Normal	54.7%
Moderate LV dysfunction	33.3%
Severe LV dysfunction	12%

Predictive factors for LV dysfunction

Age

In our population, the mean age was lightly similar between patients with LV dysfunction and patients without LV dysfunction (59.6+/-3.872 vs 61.56+/-2.430).

The age distribution of LVEF in the population is random as shown in the graph below, so there is no correlation between age and LVEF.

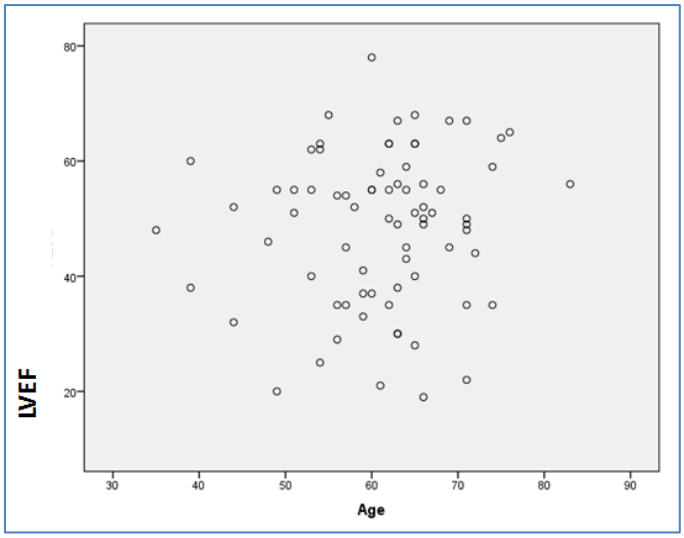

Fig-1: Distribution of the LVEF by age

Gender

In the population of patients with LV dysfunction, the proportion of men and women was 95% and 5% respectively.

In contrast, among patients with preserved LVEF, the proportion of men and women was 76.4% and 23.6% respectively. Male gender is therefore a predisposing factor for LV dysfunction in AMI with a significant p value of 0.018.

Cardiovascular risk factors

Diabetes, hypertension, smoking, are not correlated with LVEF in AMI with respective p values of 0.921, 0.569, 0.150 (see table 2).

On the other hand, dyslipidaemia is found more frequently in the population with preserved LVEF than in that with LV dysfunction (p: 0.027). A patient with dyslipidaemia would therefore be less prone to LV dysfunction in the AMI.

Electrical territories

Anterior territory

In the population with LV dysfunction, the proportion of patients with AMI in the anterior territory was 75% versus 34.5% in the population with preserved LVEF, this difference is statistically significant with a p value of 0.042. The anterior territory is therefore a predisposing factor for LV dysfunction in AMI.

Inferior territory

In the population with LV dysfunction, the proportion of patients with AMI in the inferior territory was 5% versus 50.9% in the population with preserved LVEF with a statistically non-significant difference (p: 0.059)

Time delays

The delay in consultation for patients without LV dysfunction and patients with LV dysfunction was 50.67+/-22,160 hours and 54.94+/-33,848 hours respectively. The distribution of LVEF in relation to time delays is random as shown in the graph below.

However, patients admitted less than 12 hours after the suspected onset of pain had less LV dysfunction than those admitted after 12 hours (49.1% vs 25%, p: 0.048).

The delay in admission beyond 12 hours from the presumed onset of pain is therefore a predisposing factor for LV dysfunction in AMI.

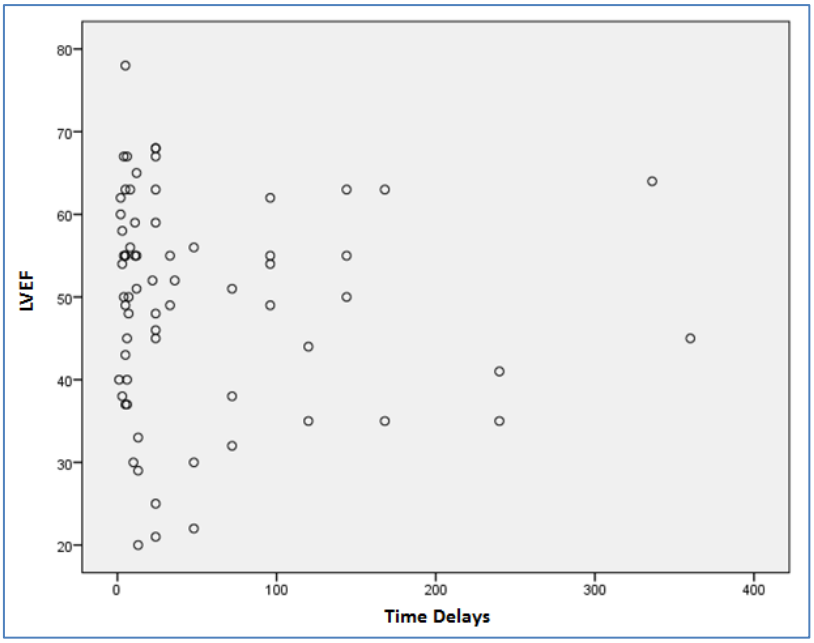

Fig-2: Distribution of LVEF by time delays

Hemodynamic parameters on admission

Heart rate

The mean heart rate was 77.40+/-4.408 bpm and 96.74+/-8.880 bpm in patients without LV dysfunction and in patients with LV dysfunction, respectively.

The distribution of LVEF by heart rate shows that LVEF decreases as heart rate increases with a statistically significant difference (p:0.003).

Heart rate is therefore correlated with LVEF in AMI.

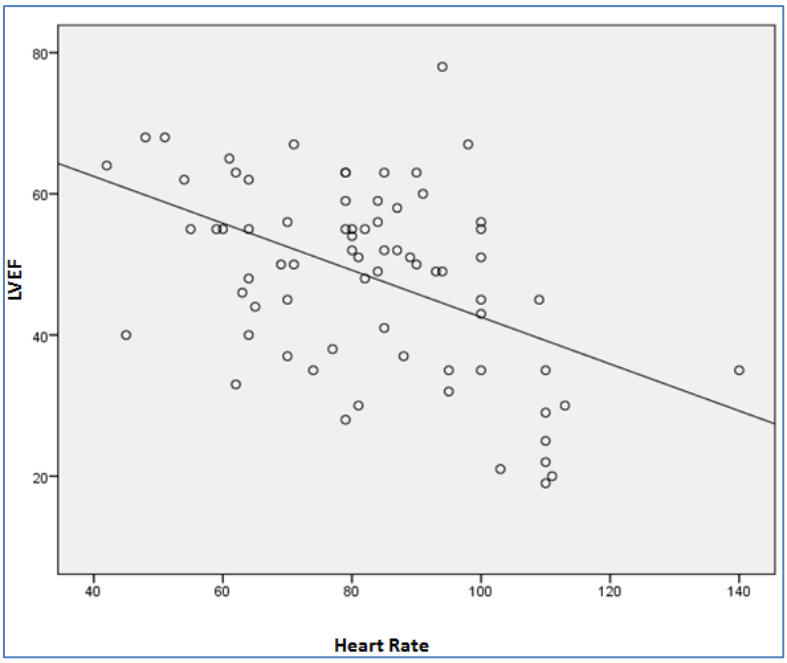

Fig-3: Distribution of LVEF by heart rate

Systolic blood pressure (SBP)

SBP is approximately the same in both population groups (131.67+/-5.388 mmHg vs 129.26+/-9.948 mmHg). In addition, the distribution is random, so SBP is not correlated with LVEF.

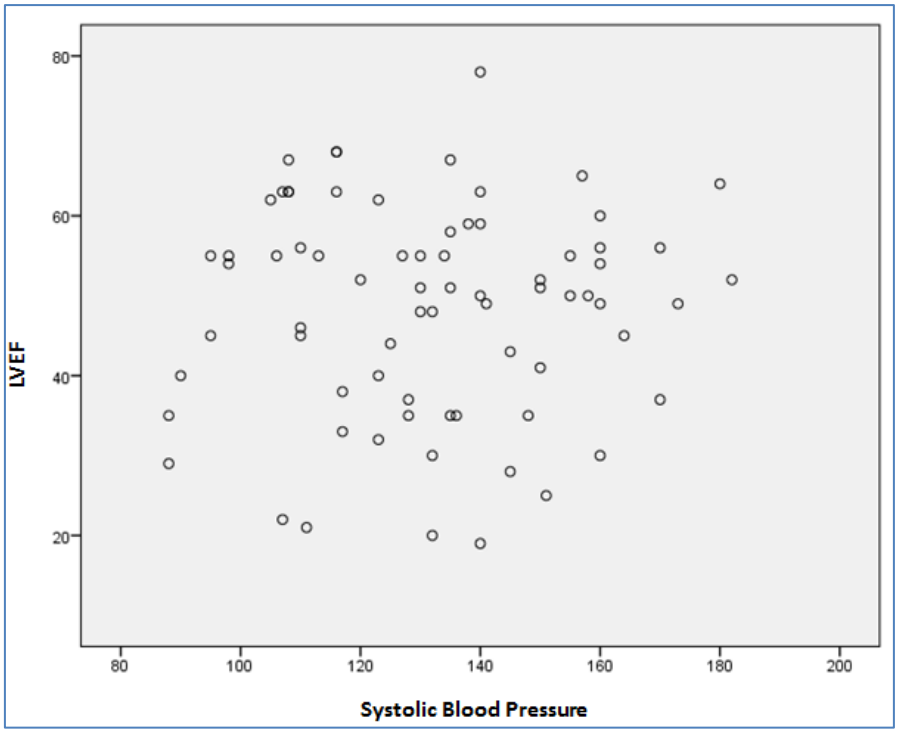

Fig-4: Distribution of LVEF by SBP

Crackling sound on lungs at admission

In the group of patients without dysfunction, 5.3% of patients had crackles on admission compared to patients with LV dysfunction of whom 54.5% had crackles, this difference is statistically significant with a p value of 0.000.

Troponin levels on admission

The mean troponin level of patients with LV dysfunction and patients without LV dysfunction was 45092.9+/-34846.338 ng/l and 32778+/-16147.634 ng/l respectively. The distribution of LVEF versus troponin is random (see graph below).

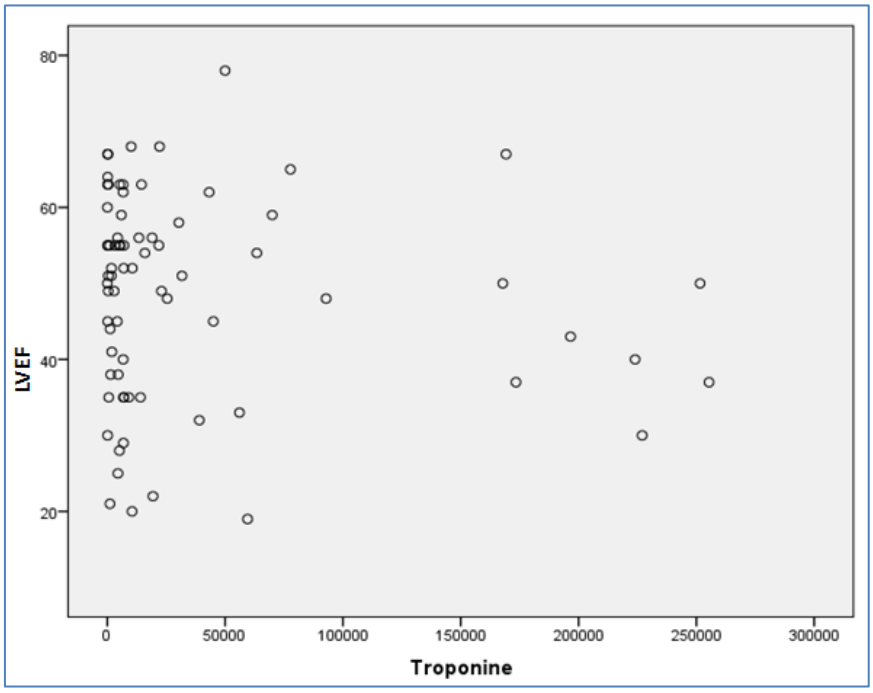

Fig-5: Distribution of LVEF by troponin levels

Table-2: Predictive factors for LV dysfunction (LVEF less than 40% in AMI)

Predictive factors	General population	No LV dysfunction i.e LVEF>40%	LV dysfunction i.e LVEF<40%	P Value
	Number (%)	Number (%)	Number (%)	
	Total number =75	Total number= 41	Total number = 34	
Gender (M/F)	60 (81.3)/ 15 (18.7)	31 (76.4)/ 10 (23.6)	32 (95)/ 2(5)	0.018
Diabetes (Yes/No)	37 (49.3)/ 38 (50.7)	20(49.1)/ 21(50.9)	17 (50) /17 (50)	0.921
High blood pressure (Yes/No)	31 (41.3)/ 44 (50.7)	16 (40)/ 25 (60)	15 (45)/ 16(55)	0.569
Smocking (Yes/No)	37 (49.3) / 38 (50.7)	19 (46.3)/22 (53.7)	20 (57.9) /14(42.1)	0.150
Dyslipidaemia (Yes/No)	16 (21.3)/ 59 (78.7)	10 (23.6)/ 31 (76.4)	5 (15)/29 (85)	0.027
Delay of less than 12h(Yes/No)	32 (42.7) / 43 (57.3)	49.1%/50.9%	9(25)/ 25 (75)	0.048
Anterior electrical territory (Yes/No)	34 (45.3) / 41 (54.7)	14 (34.5)/ 27 (65.5)	25 (75)/ 9(25)	0.042
Inferior electrical territory(Yes/No)	29 (38.7)/ 46 (61.3)	21 (50.9) / 20 (49.1)	2 (5)/ 32 (95)	0.059
Crackling sound on lungs at admission (Yes/No)	21 (28.2) / 54 (71.8)	2 (5.3) / 39 (94.7)	19 (54.5)/ 15 (45.5)	0.000
Age	61.04+/-2.056	61.56+/-2.430	59.60+/-3.872	0.402
Delay in consultation	51.68+/-18.612	50.67+/-22.160	54.94+/-33.848	0.775
average troponin levels	36106.64+/-15006.66	32778+/-16147.634	45092.9+/-34846.338	0.794
Systolic blood pressure	131.67+/-5.388	131.67+/-5.388	129.26+/-9.948	0.599
Heart rate	82.5+/-4.454	77.40+/-4.408	96.74+/-8.880	0.000

DISCUSSION

In patients with acute myocardial infarction, left ventricular dysfunction is a well established predictor of mortality. Estimates of left ventricular function have formed the basis of short-term [4, 5] and long-term [6] prognosis. More recent studies [7-11] have confirmed these observations. The incidence of LV dysfunction is high. In our study, the incidence of left ventricle dysfunction was 45.3%. In the Pedros S. Mateus *et al*. [12], which evaluated the impact of cardiovascular risk factors in the occurrence of LV dysfunction in AMI, the incidence of LV dysfunction was 55.8%. In our prospective series, we evaluated the parameters that predispose to LV dysfunction in AMI. We found that gender, dyslipidaemia, time to management, admission heart rate, electrical territory, existence of Crackling sounds on lungs on admission were factors correlated with LVEF in AMI. Male sex, high heart rate, anterior electrical territory, as well as the existence of Crackling sound on lungs correlated with impaired LV function. Male gender may predispose to LV dysfunction due to the association with several other comorbidities and the frequent association of risk factors including smoking [13]. An elevated heart rate reflects a high mechanical load on the LV as well as an increase in myocardial oxygen consumption that may aggravate ischaemia and precipitate LV dysfunction [14]. The anterior territory is most often vascularised by the left anterior descending artery which provides vascularisation to a large part of the LV myocardium, and its obstruction would correlate with a greater volume of LV infarcted myocardium, hence the predisposition to LV dysfunction [15]. Crackling sounds on lungs indicate diastolic or systolic LV dysfunction. However, dyslipidaemia and a delay in management of less than 12 hours are protective factors in our study. Indeed, the earlier the management, the less extensive and transmural the infarction. Dyslipidaemia could protect against LV dysfunction by the fact that patients with dyslipidaemia are more often followed up and benefit from a correction of cardiovascular risk factors and comorbidities. In the Pedros S. Mateus *et al*. [12], they did not find an association between age, sex and hypertension. Diabetes and smoking were found to be predisposing to LV dysfunction (diabetes OR: 3.73). As in our study, dyslipidaemia appeared to be a protective factor with an odds ratio of 0.37, 95% CI, 0.15-0.88. In the Prashant D. *et al*. Study [16] which also assessed predictors of LV dysfunction in AMI, age, renal function at admission, anterior electrical territory were correlated with LV dysfunction in AMI. A predictive score for LV dysfunction has been established by the same authors including: history of coronary disease, mechanical ventilation, creatinine level, ST+ elevation, and inferior territory. (see table 3) A score of greater than or equal to 1 was associated with a probability of LV dysfunction with a sensitivity of 97% and a specificity of 43%.

Table-3

Score for the prediction of depressed left ventricular function*	
Criteria	Score
History of coronary artery disease†	+4
Mechanical ventilation	+4
Creatinine (mg/dl)	
1.0–1.5	+3
>1.5	+6
Maximum ST-segment elevation >2 mm	+3
Inferior ST-segment elevation	−7

CONCLUSION

Left ventricular systolic dysfunction after AMI is associated with significant morbidity and mortality. Identifying patients at risk of developing LV dysfunction will have prognostic and therapeutic impact. In our series evaluating the parameters that predispose to LV dysfunction in AMI, male gender, dyslipidaemia, time to management, heart rate on admission, electrical territory, existence of Crackling sound on lungs at admission are factors correlated with LV dysfunction.

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