









# Cardiac functional recovery after acute coronary syndrome: a comparison between smokers and non-smokers

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**Introduction:** Smoking is a well-established risk factor for the development of acute coronary syndrome (ACS).<sup>1</sup> Beyond its role in disease onset, smoking has been associated with adverse outcomes during follow-up.<sup>2</sup> While several studies have examined the impact of smoking cessation on cardiac recovery, few have directly compared smokers and non-smokers with respect to functional cardiac recovery following ACS.<sup>3</sup> The aim of this study was to evaluate differences in cardiac functional recovery between smokers and non-smokers after ACS.

**Patients and Methods:** This retrospective study included patients hospitalized with ACS between January and December 2023. Patients were classified as smokers or non-smokers. Variables included demographics, comorbidities, MACE score, troponin, stenosis extent, hypokinesia, diastolic dysfunction, and echocardiographic changes. Analyses were performed using SPSS 26. Categorical variables were compared with Fisher's exact or Chi-square tests, and continuous variables with Student's t-test or Mann-Whitney U test. A two-tailed  $p < 0.05$  was considered significant.

**Results:** The study included 42 patients. The mean age of smokers was 61.4 years (SD 12.1) and 67.0 years (SD 7.9) for non-smokers (Student's t-test,  $p = 0.09$ ; 95% CI: -0.8 to 11.9). Among smokers, the median pack-years was 33 (IQR 24–50). Baseline and clinical characteristics (**Table 1**) and echocardiographic

**TABLE 1. Baseline and clinical characteristics according to smoking status.**

	N (%)			P*
	Smokers	Non-smokers	Total	
Gender				
Male	13 (44.8)	16 (55.2)	29 (69)	0.32
Female	8 (61.5)	5 (38.5)	13 (31)	
Coronary stenosis				
Single-vessel disease	10 (58.8)	7 (41.2)	17 (40.5)	0.35
Multivessel disease	11 (44)	14 (56)	25 (59.5)	
Improved hypokinesia	3 (37.5)	5 (62.5)	8 (19)	0.7†
Improved diastolic dysfunction	1 (33.3)	2 (66.7)	3 (7.1)	>0.99†
Hyperlipidemia	17 (53.1)	15 (46.9)	32 (76.2)	0.5
Hypertension	13 (46.4)	15 (53.6)	28 (66.7)	0.51
Diabetes mellitus	5 (83.3)	1 (16.7)	6 (14.3)	0.18†
COPD	2 (100)	0 (0)	2 (4.8)	0.49†

\* $\chi^2$  test; †Fisher's exact test

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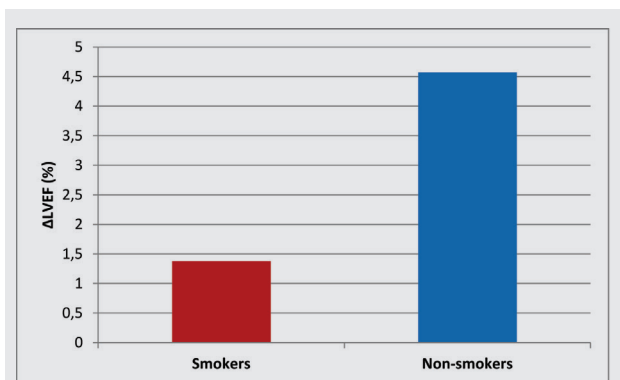


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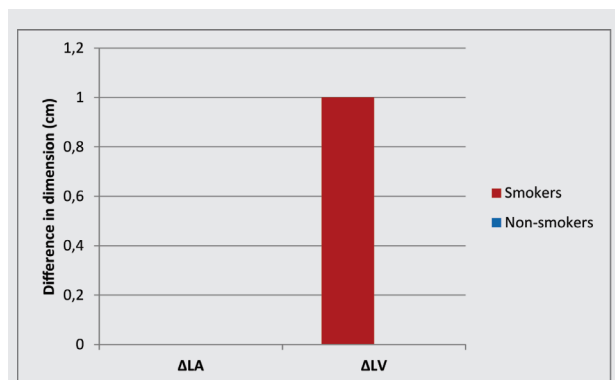
**TABLE 2. Comparison of cardiac recovery between smokers and non-smokers after acute coronary syndrome.**

	Median (IQR)		P*
	Smokers	Non-smokers	
<b>LVEF (%)</b>			
Baseline value	55 (45-62)	57 (45-60)	0.6
Follow-up value	60 (52-62)	60 (49-67)	0.6
<b>Left ventricle (cm)</b>			
Baseline value	51 (45-53)	52 (46-56)	0.7
Follow-up value	54 (46-56)	52 (48-54)	0.8
<b>Left atrium (cm)</b>			
Baseline value	42 (38-43)	42 (36-45)	>0.9
Follow-up value	42 (38-43)	39 (35-43)	0.2

\* Mann Whitney U test



**FIGURE 1. Change in left ventricular ejection fraction (LVEF; %) over time (initial vs. follow-up) in smokers and non-smokers.**



**FIGURE 2. Change in left ventricular (LV) and left atrial (LA) dimensions (cm) over time (initial vs. follow-up) in smokers and non-smokers.**

graphic parameters (**Table 2**) showed no significant differences between groups. Although non-smokers showed greater improvement in left ventricular ejection fraction (LVEF) (mean  $\Delta$  4.6%, SD 6.9) compared to smokers (mean  $\Delta$  1.4%, SD 13.3), the difference was not statistically significant (Student's t-test,  $p = 0.3$ ; 95% CI: -3.5 to 9.8) (**Figure 1**). A slight, non-significant increase in left ventricular (LV) dimension was observed in smokers (median  $\Delta$  1 cm, IQR -2 to 4; Mann-Whitney U test,  $p = 0.4$ ), suggesting a possible trend toward adverse LV remodeling (**Figure 2**). Left atrial (LA) dimension remained unchanged in both groups.

**Conclusion:** This study found no statistically significant difference in cardiac recovery after ACS between smokers and non-smokers. However, a trend toward less favorable remodeling in smokers was observed. Given the small sample size and self-reported smoking status, further larger studies are needed to confirm these findings and clarify the clinical impact.

**LITERATURE**

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