Original Research Article

Effect of Cigarette Smoking on Location of Infarction in Patients Presented with ST-Segment Elevation Myocardial Infarction

Abstract

Background: In an acute ST-elevation myocardial infarction, transmural myocardial ischemia causes myocardial damage or necrosis (STEMI). In order to establish myocardial ischemia damage, the current clinical definition of myocardial infarction (MI) in 2018 includes abnormal cardiac biomarkers. STEMI is linked to a greater risk of mortality in the hospital compared to non-ST segment elevation acute coronary syndromes. The study's purpose was to investigate and assess the effect of cigarette smoking on the location of infarction in people who had ST-segment elevation myocardial infarction.

Methods: The present case-control research investigated 100 individuals who had a first ST segment elevation acute myocardial infarction and got split into two groups based on the smoking status: group 1 (50 patients): Group 1 (50 patients) were smokers, whereas group 2 (50 patients) were nonsmokers.

Results: Smokers were significantly younger than non-smokers (53.86±7.77) years vs. (58.20 ± 7.80), P = 0. 006.Gender distribution: Smokers were significantly more likely to be male (82.0 % vs. 58.0 %, P = 0.009)**.** inferior STEMI was statistically significant difference among the two groups (P value =0.045*). Myocardial Infarction Localization by electrocardiogram was statistically significant different among the two groups (P value =0.045*). Ejection fraction (EF) was statistically significant different among the two groups (smoker and nonsmokers respectively 54.86 ± 8.58 % vs. 49.04 ± 6.61 %) (P value =0.001*).

Conclusions: Smoking raises STEMI risk, and smokers are more likely than non-smokers to develop coronary artery disease at an earlier age.

Keywords: Effect of cigarette smoking, location of infarction, ST-segment elevation myocardial infarction.

Introduction:

Myocardial infarction is a major cause of cardiovascular disease. The assessment of myocardial infarction incidence and case fatality are major drivers of coronary disease mortality reduction [1].

In an acute ST-elevation myocardial infarction, transmural myocardial ischemia causes myocardial damage or necrosis (STEMI) [2]. In order to establish myocardial ischemia damage, the current clinical definition of myocardial infarction (MI) in 2018 includes abnormal cardiac biomarkers [3].

In-hospital death rates for STEMI are higher than for non-ST segment elevation acute coronary syndromes [4].

The condition that causes the majority of acute coronary syndrome (ACS) instances is atherosclerosis. Acute thrombus obstructing an atherosclerotic coronary artery causes 90 percent of MIs. The rupture and erosion of plaque are thought to be the primary causes of coronary thrombosis. After plaque rupture or erosion, platelet activation and aggregation, activation of the coagulation cascade, and endothelial vasoconstriction ensue, leading in coronary thrombosis and occlusion.

Smoking or other tobacco use is one of the recognized modifiable risk factors for atherosclerosis [5] It is generally known that smokers are more susceptible to illnesses of the heart and blood vessels (cardiovascular disease) [8] as smoking damages blood vessels and can make them thicken and grow narrower. These changes can lead to tachycardia, hypertension and lastly thrombus formation [5, 6].

Smoking causes stroke and coronary heart disease, two of the main causes of mortality, and even persons who smoke fewer than five cigarettes per day can develop early symptoms of cardiac and vascular illness [5, 7].

There are regulations in place to reduce the number of smokers in society, and the results have been positive, with a reduction in the acute coronary syndrome incidence [8, 9].

This present research aims studying and assessing the consequences of cigarettes smoking on location of infarction in participating patients presented with ST-segment elevation myocardial infarction.

Patients and Methods:

This case control study got carried out on 100 patients presenting by new ST-segment elevation myocardial infarction older than 18 years old were recruited from cardiology department in Tanta University hospitals presented.

The patients were below 18 years old, presented with Non-ST Elevation myocardial infarction and had prior Percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG) were excluded.

Patients were split into 2 groups based on the smoking status: Group 1 (50 patients): Smokers. Group 2 (50 patients): Nonsmokers.

Patients were allocated to PCI according to catheterization lab availability after confirming diagnosis.

The new criteria for diagnosing myocardial infarction according to the European Society of Cardiology (ESC) guidelines 2017 [26] are discovery of a spike and/or decline in cardiac biomarkers (ideally troponin) with a minimum one value above the upper reference limit's 99th percentile, as well as indication of myocardial ischemia with a minimum one of the following [10]:

1. Ischemia symptoms (e.g., chest discomfort, angina equivalent and silent ischemia).
2. Electrocardiogram (ECG) alterations symptomatic of new ischemia (new ST-T changes or new left bundle branch block (LBBB).
3. Development of pathological Q-wave changes in the ECG.
4. New proof of viable myocardial loss or a new regional wall motion abnormality on imaging.

Studied groups had been through the following:

1. An informed consent taken from all participants.
2. Full history taking including:

Gender, age, history of risk factors for coronary artery disease (CAD) as: Diabetes Mellitus, hypertension, smoking, as well as past history and family history of CAD.

3. Full clinical examination:

- Heart rate, blood pressure, and breathing rate are all vital indicators.
- *General examination*: with attention to height, weight, body mass index (BMI), patient look, decubitus, cyanosis, jaundice, with special attention to signs of heart failure (e.g., congested neck veins and orthopnea).
- *Local cardiac examination*: abnormal pulsation, Heart sounds & murmurs.

4. Resting 12 leads ECG:

Standard 12-lead ECG was obtained within 10 minutes of first medical contact (FMC) according to ESC guidelines 2017 including:

- limb leads I, II, III, aVR, aVL, aVF, and chest leads from V1 to V6 for all patients on admission to the hospital [11].
- Right pericardial leads (V3R, V4R, V5R, V6R) and posterior chest leads (V7 to V9) were done for some patients to detect posterior wall and right ventricular infarction [11].

5. Baseline laboratory tests:
    - including serum urea and creatinine, cardiac enzymes include serum troponin and CK-MB and hemoglobin level.
6. Echocardiography:

The study was performed using (a GE vivid seven cardiac ultrasound phased array system with tissue Doppler imaging using M4S transducer 4 MHz).

Two-dimensional echocardiographic assessment was done during admission after successful PCI in left lateral decubitus position to:

- Assess left ventricular LV systolic function using Simpson's method in the apical 4 & apical 2 views also left ventricular volumes were assessed (End diastolic volume and end systolic volume) [12].

7. Reperfusion through: percutaneous intervention for Infarct related artery (IRA)

8-Predischarge 12-lead electrocardiograph.

Statistical analysis

SPSS v26 was used for statistical analysis (IBM Inc., Chicago, IL, USA). The mean and standard deviation (SD) of quantitative variables were investigated among the two groups via an unpaired Student's t-test. Also, the Chi-square test or Fisher's exact test were conducted to evaluate qualitative variables that were reported as frequency and percentage (percent). Statistical significance was defined as a two-tailed P value < 0.05.

Results

As shown in Table 1, smokers compared to non-smokers were significantly younger and more likely to be male. Table 1

Table 1: Demographic data of the study.

Demographics	**Group I Smokers (n = 50)**		**Group II Non-smokers (n = 50)**		**Test of Sig.**	**P. value**
Age (years)					T test	
Min. – Max.	36 – 70		45 – 71		T = 2.788	0. 006*
Mean ± SD.	53.86 ± 7.77		58.20 ± 7.80			
Sex	NO	%	NO	%	X2	
Male	41	82.0%	29	58.0%	6.857	0.009*
Female	9	18.0%	21	42.0%		

χ^2: **Chi square test** t: **Student t-test**
p: p-value to compare the two groups
Group I: Smoker patients with acute ST elevation myocardial infarction. **Group II:** Non-Smoker patients suffering acute ST elevation myocardial infarction.

Smokers were significantly less often hypertensive than non-smokers, but no significant differences were seen in diabetes mellitus or in family history of coronary artery diseases. Table 1

Table 2: Comparing the two participating groups in regards to risk factors

	G I smokers	**G II non smokers**	**X^2**	**P-value**

HTN	**No**	**N**	32	22	4.026	0.045*
		%	64.0%	44.0%		
	Yes	**N**	18	28		
		%	36.0%	56.0%		
DM	**No**	**N**	37	28	3.560	0.059
		%	74.0%	56.0%		
	Yes	**N**	13	22		
		%	26.0%	44.0%		
Family Hx	**No**	**N**	43	37	2.250	0.134
		%	86.0%	74.0%		
	Yes	**N**	7	13		
		%	14.0%	26.0%		

χ^2: **Chi square test**, p: p value to compare the two groups, *: Statistically significant at $p \leq 0.05$, HTN: Hypertension, DM: Diabetes Mellitus, Family Hx: Family History.

Among the two groups, we discovered no statistically significant difference in pain (P-value = 0.461). The difference in IRA was statistically significant among the two groups (P-value =0.013). We found no statistically significant difference among the two groups in terms of intervention type (P-value = 0.558). Between the two groups, we discovered no statistically significant difference in TIMI (P-value = 0.222). Table 1

Table 3: Comparing the two participating groups based on Clinical Presentation and according to angiographic procedure.

			G I smokers	**G II non smokers**	**X^2**	**P-value**
TCP	**No**	**N**	3	5	0.5643	0.461
		%	6.0%	10.0%		
	Yes	**N**	47	45		
		%	94.0%	90.0%		
ATCP	**No**	**N**	47	45	0.543	0.461
		%	94.0%	90.0%		
	Yes	**N**	3	5		
		%	6.0%	10.0%		
SOB	**No**	**N**	45	42	0.796	0.372
		%	90.0%	84.0%		
	Yes	**N**	5	8		
		%	10.0%	16.0%		
LAD	**No**	**N**	30	20	4.001	0.046*
		%	60.0%	40.0%		
	Yes	**N**	20	30		

		%	40.0%	60.0%		
LCX	**No**	**N**	45	44	0.102	0.749
		%	90.0%	88.0%		
	Yes	**N**	5	6		
		%	10.0%	12.0%		
RCA	**No**	**N**	25	37	6.112	0.013*
		%	50.0%	74.0%		
	Yes	**N**	25	13		
		%	50.0%	26.0%		
PTCA only	**No**	**N**	48	49	0.344	0.558
		%	96.0%	98.0%		
	Yes	**N**	2	1		
		%	4.0%	2.0%		
Stent Implantation	**No**	**N**	2	1	0.344	0.558
		%	4.0%	2.0%		
	Yes	**N**	48	49		
		%	96.0%	98.0%		
Final TIMI Flow	**1**	**N**	0	1	3.010	0.222
		%	.0%	2.0%		
	2	**N**	2	0		
		%	4.0%	.0%		
	3	**N**	48	49		
		%	96.0%	98.0%		

TCP: typical chest pain; ATCP: atypical chest pain; SOB: shortness of breath; LAD: left anterior descending artery; LCX: left circumflex artery; RCA: right coronary artery; PTCA: percutaneous transluminal coronary angioplasty; TIMI: Thrombolysis in Myocardial Infarction.
χ^2: **Chi square test**
p: p value to compare the two groups
*: Statistically significant at $p \leq 0.05$

Heart rate was insignificant difference among the two groups (P-value =0.716). The systolic blood pressure appeared to be statistically significant difference among the two groups (P-value =0.006*). The Diastolic blood pressure was statistically significant difference among the two groups (P-value =0.030*). Smokers have considerably greater hemoglobin and hematocrit levels than non-smokers (14.30 ± 1.74mg/dL vs. 13.10 ± 1.71mg/dL, P = 0.001 and 42.50 ± 4.28% vs. 39.50 ± 4.65, P = 0.001, respectively). Smokers had considerably greater mean platelet levels than non-smokers (290.48± 97.84 109/L vs. 235.28 ± 73.14 109/L, P = 0.002). Smokers had considerably lower mean creatinine levels than non-smokers (0.98 ± 0.25mg/dL vs. 1.15 ± 0.30mg/ dL, P=0.003). Admission blood glucose was also lower in smokers (136.66 ± 41.45mg/dL vs. 174.28 ± 80.17mg/ dL, P = 0.004) 5- Cardiac Troponin were lower in smokers' group than nonsmokers (0.76 ± 0.90 ng/ml vs. 1.36 ± 0.97 ng/ml, P < 0.002*). Smokers had a lower frequency of hyperlipidemia,

particularly LDL, than nonsmokers (92.12 ± 16.78 mg/dl vs. 101.98 ± 29.30mg/dl, P < 0.042*). Table 1

Table 4: Comparing the two participating groups based on vital signs and according to laboratory investigation

		Range			Mean	±	S. D	t. test	p. value
SBP	G I	90	–	180	126.40	±	22.88	2.793	0.006*
	G II	100	–	200	139.04	±	22.38		
DBP	G I	60	–	120	82.00	±	15.08	2.201	0.030*
	G II	70	–	115	87.80	±	10.93		
MAP	G I	70	–	140	96.80	±	17.36	2.536	0.013*
	G II	80	–	136.67	104.88	±	14.36		
HR	G I	50	–	115	80.32	±	16.61	0.365	0.716
	G II	48	–	120	79.08	±	17.33		
S Chol.	G I	113	–	267	183.88	±	35.27	1.480	0.142
	G II	120	–	312	196.06	±	46.29		
LDL	G I	60	–	133	92.12	±	16.78	2.065	0.042*
	G II	60	–	183	101.98	±	29.30		
HDL	G I	35	–	73	54.40	±	9.44	3.010	0.003*
	G II	34	–	63	48.92	±	8.76		
S. creat	G I	0.5	–	2	0.98	±	0.25	3.087	0.003*
	G II	0.7	–	2	1.15	±	0.30		
Urea	G I	19	–	50	26.86	±	7.29	2.527	0.013*
	G II	18	–	80	32.84	±	15.06		
RBS	G I	84	–	300	136.66	±	41.45	2.948	0.004*
	G II	99	–	500	174.28	±	80.17		
Hb	G I	10	–	19	14.30	±	1.74	3.482	0.001*
	G II	10	–	17	13.10	±	1.71		
HCT	G I	33	–	50	42.50	±	4.28	3.357	0.001*
	G II	29	–	47	39.50	±	4.65		
PLT	G I	130	–	570	290.48	±	97.84	3.195	0.002*
	G II	90	–	500	235.28	±	73.14		
CKMB	G I	20	–	450	90.18	±	98.91	0.217	0.829
	G II	19	–	1300	96.64	±	186.28		
Tn	G I	0.03	–	5	0.76	±	0.90	3.216	0.002*
	G II	0.1	–	5	1.36	±	0.97		

t: **Student t-test**; p: p value to compare the two groups; *: Statistically significant at $p \leq 0.05$. SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean Arterial Pressure; HR: Heart rate; S Chol: Serum Cholesterol level; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; S Creat: Serum Creatinine level; RBS: Retained Blood Syndrome; Hb: Hemoglobin; HCT: Hematocrit level; PLT: Platelet count; CK-MB: Creatine Kinase Myocardial Band; Tn: Troponin level.

Myocardial Infarction Localization by electrocardiogram was statistically significant difference among the two groups (P-value =0.045*). Ejection fraction (EF) assessment by Echocardiography was statistically significant difference among the two groups (smoker and nonsmokers respectively 54.86 ± 8.58 % vs. 49.04 ± 6.61 %) (P-value =0.001*). Table 1

Table 5: Comparing the two participating groups based on Myocardial Infarction Localization by electrocardiogram and EF

			G I Smokers	G II Non smokers	X^2	P-value
Inferior	**No**	**N**	21	31	4.006	0.045*
		%	42.0%	62.0%		
	Yes	**N**	29	19		
		%	58.0%	38.0%		
Anterior	**No**	**N**	29	19	4.006	0.045*
		%	58.0%	38.0%		
	Yes	**N**	21	31		
		%	42.0%	62.0%		
Lateral	**No**	**N**	43	44	0.088	0.766
		%	86.0%	88.0%		
	Yes	**N**	7	6		
		%	14.0%	12.0%		
Posterior	**No**	**N**	45	49	2.837	0.092
		%	90.0%	98.0%		
	Yes	**N**	5	1		
		%	10.0%	2.0%		
ECHO EF (%)	Min. – Max.		35 – 71	35 – 67	t= 3.802	0.001
	Mean ± SD.		54.86 ± 8.58	49.04 ± 6.61		

□2: Chi square test ; p: p value to compare the two groups; *: Statistically significant at $p \leq 0.05$. EF: Ejection fraction.

Discussion

According to our findings, smokers with STEMI are more likely to have an inferior than an anterior myocardial infarction. Tobacco use has a variety of impacts on the cardiovascular system, making smokers more likely than nonsmokers to suffer coronary artery disease at an earlier age [13, 14].

Several studies have shown that smoking is highly linked to suffering coronary artery disease (CAD) early in life [15, 16]. According to the Framingham Heart Study, young smokers had a three-fold greater risk of coronary artery disease than nonsmokers. Increased plasma Lp (a) levels are well recognized to be linked to a significant risk of early CAD [17-20].

In both studies, smokers had a greater danger of inferior myocardial infarction [21]. In another research by Grines et al., the occurrence of inferior STEMI reached 60 percent in smokers and 53 percent in non-smokers, with the smoker group having a considerably greater frequency. Despite the fact that the total occurrence of inferior STEMI was greater in their study, the findings on the link between smoking and inferior STEMI were identical to ours [22].

Smokers had a greater risk of inferior STEMI than anterior STEMI, according to our findings. The greater rate of inferior STEMI, in which the right coronary artery (RCA) is the infarct-related artery in most of the cases, may be explained by the dominating thrombogenic etiology of STEMI in smokers [23]. The RCA has fewer branches than the left coronary artery, which may make it easier for big clots to develop in this conduit. RCA also has a less turbulent flow, which, together with its greater diameter, may make it more prone to thrombus development [24].

However, more scientific investigations are required to determine the specific mechanism by which smoking raises the risk of inferior STEMI. Various researches have looked at the influence of smoking in the distribution of coronary lesions, with mixed results [25-28].

Regarding left ventricular involvement and dysfunction in patients with ST elevation myocardial infarction in our study there was significant difference among the two groups as the ejection fraction was significantly higher in the smokers group. This result is supported by Mark E. Hands et al study who found in comparison to inferior infarctions of equal enzymatic size, anterior infarctions have a lower global left ventricular ejection fraction, which is associated with more left ventricular regional wall abnormalities. The latter is most likely due to a larger degree of left ventricular necrosis in the anterior infarct group, as shown by QRS scoring [29].

In the study of FH Zimmerman et al, a history of smoking is very common, with 73 percent to 90 percent of young patients with myocardial infarction reporting it [16, 30-35]. According to the findings, current smoking is linked to myocardial infarction in young people [36]. Both the current study and prior investigations found that young patients suffering myocardial infarction outperformed elderly individuals [33, 35-37] ([208],[211],[214],[215]) hypertension and diabetes were shown to be more prevalent in older age groups.

Tobacco use activates the sympathetic nervous system, which leads to a rise in heart rate and blood pressure [38]. Elevated oxygen demand is accompanied by vasoconstriction, resulting in a reduction in oxygen supply [39]. In our investigation, we found no significant difference in admission heart rate among the two groups, yet smoking caused increases in plasma norepinephrine levels in Grassi, G., et al study and other studies, which is consistent with other investigators' findings [40, 41]. This might be due to nicotine's direct effects on sympathetic nerve terminals, which increase catecholamine release.

Tobacco use also promotes LDL cholesterol oxidation [42] endothelial function is harmed as a result of this [43]. In smokers, an upsurge in inflammatory factors and atherogenesis, as well as enhanced platelet aggregation and a hypercoagulable state, all play a role in the etiology of coronary disease [44]. Both smokers and non-smokers groups showed increase in LDL but in nonsmokers group the LDL was significantly higher.

These results, on the other hand, might imply that smokers and nonsmokers have different pathways for producing myocardial infarction. As stated in our study's findings, The mean hemoglobin and hematocrit levels in smokers were significantly elevated more than in nonsmokers, with a statistically significant difference among the two groups. We can explain this by the hypoxemia resulted from carbon monoxide in cigarette smoking, which leads to an elevation in red blood cell mass. According to some studies, a rise in hemoglobin levels in smokers' blood might constitute a compensatory mechanism. Carbon monoxide binds to Hb

to generate carboxy hemoglobin, an inactive form of hemoglobin with no potential to deliver oxygen. Carboxyhemoglobin also moves the left side of the Hb dissociation curve, reducing Hb's capacity to supply oxygen to the tissue. Smokers uphold a greater hemoglobin level than non-smokers to compensate for the diminished oxygen delivery capacity [45].

The findings of our study on hemoglobin and hematocrit levels are backed up by a study by Malenica M, Prnjavorac B, Bego T, et al., which found that smokers' hemoglobin levels were considerably higher than non-smokers' regardless of gender [46]. Also , In a research conducted by Lakshmi et al. [47] In smokers, the hematocrit and hemoglobin levels were much increased, and the RBC count grew dramatically as the intensity of smoking increased.

In a previous research, teenagers who had just started smoking had a higher platelet count [48]. Platelet activity is much elevated in smokers than in nonsmokers, according to several research. The study found a relationship between cigarette smoking and platelet formation, suggesting that smokers may be more prone to developing an acute occlusive platelet thrombus in a sick and stenotic coronary artery than nonsmokers [22]. The elevated amounts of red blood cells and platelets seen in smokers imply that they may have a hypercoagulable condition that promotes coronary thrombosis, according to our findings.

In several trials, smokers had a better therapeutic response following fibrinolysis [49, 50]. The observed impact can be attributed to smokers' greater levels of serum fibrinogen, which leads to an elevated fibrin content in thrombosis. 226 Elevated serum fibrinogen, platelet activity, and red blood cell bulk in these individuals also point to a hypercoagulable condition [44, 51].

In smokers, the hypercoagulable condition may cause coronary thrombosis [22, 51]. Smokers had more thrombus than plaque load in the TEAM-2 research and several other findings. After thrombolytic treatment, individuals were also more likely to have TIMI grade

3 flow [52, 53]. These data indicate the thrombogenic mechanism's dominance in the pathophysiology of STEMI in smokers [44, 54, 55].

The findings of the present research on the impact of smoking on the site of myocardial infarction are in line with Alemu et al findings. They looked examined the link between smoking status and the site of myocardial infarction in five other cohorts, as well as their own cohort [21].

In both studies, smokers had a greater risk of inferior myocardial infarction. 230 In another research by Grines et al., the commonness of inferior STEMI reached 60 percent in smokers, while 53 percent in non-smokers, with the smoker group having a considerably greater frequency. Despite the fact that the total incidence of inferior STEMI was elevated in their study, the findings on the link between smoking and inferior STEMI showed to be identical to ours [22].

Smokers had a greater risk of inferior STEMI than anterior STEMI, according to our findings. The greater rate of inferior STEMI, in which RCA is the infarct-related artery in most of the cases, may be explained by the dominating thrombogenic etiology of STEMI in smokers [21]. The RCA has fewer branches than the left coronary artery, which may make it easier for big clots to develop in this conduit. RCA also has a less turbulent flow, which, together with its greater diameter, may make it more prone to thrombus development [24].

However, more researches are required to determine the specific mechanism by which smoking raises the risk of inferior STEMI. Various researches have looked at the influence of smoking in the distribution of coronary lesions, with mixed results [25, 27, 28, 56, 57].

The most noteworthy finding was that the increased risk of coronary disease followed a distinct anatomic distribution, with individuals who smoked having a high tendency to suffer right coronary blockage. Smoking patients who have single-vessel disease exhibited a considerably greater rate of right coronary blockage than nonsmokers. Similarly, among two-

vessel patterns, smokers were less likely than nonsmokers to have a mix of anterior descending and circumflex lesions, i.e., the only form without right coronary involvement. As a result, smoking raises the likelihood of RCA lesions more than other types of vascular lesions [25].

The previous study also mentioned that the right coronary lesions were substantially more common than the other lesions in individuals with single-vessel disease. After accounting for other risk variables, smoking posed the greatest danger to the right coronary artery, albeit there was some overlap with the circumflex artery's confidence interval. The relative probability of anterior descending plus circumflex lesions was also considerably lower in the two patterns with right coronary artery involvement in the population with two-vessel disease. If you smoke and suffer one or two vessels disease, the right coronary artery is more subjected to be affected (and the anterior descending artery is proportionately less probable) than if you don't.

Koliaki et al [27] showed a link between smoking and the occurrence of a lesion in RCA, left circumflex artery (LCA), and left anterior descending artery (LAD), but not in the left main coronary artery (LMCA).

As a result, this may have an influence on disease development, as evidenced by studies showing greater vascular resistances and worse autoregulatory ability in the right coronary artery system compared to the left [58, 59].

In our study, there was a substantial difference among the two groups in terms of left ventricular involvement and dysfunction in patients having ST elevation myocardial infarction, with the smokers' ejection fraction being much greater. This finding is reinforced by research by Mark E. Hands et al, who found that anterior infarction had a worse global left ventricular ejection fraction than inferior infarction of equal volumetric size, as well as more left ventricular regional wall abnormalities. The latter is most likely due to a larger degree of

left ventricular necrosis in the anterior infarct group, as shown by the Quality Rating System (QRS) scoring [29].

Strengths & Limitations:

The concept of our research is strengthened by its type as a case control study which was fruitful in investigating and evaluating the effect of smoking on the location of infarction in patients with ST-segment elevation myocardial infarction. However, the sample size was ought to be larger and the shortage of time and funds has hindered some of our work and procedures.

Conclusions:

Smoking raises the risk of inferior ST-Elevation Myocardial Infarction, as we have said (STEMI). Smokers are more likely than non-smokers to develop coronary artery disease at an earlier age.

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Conflict of Interest: Nil

COMPETING INTERESTS DISCLAIMER:
Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

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