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In-Hospital Left Ventricular Thrombus following ST-Elevation Myocardial Infarction.

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Abstract

Background: In-hospital left ventricular (LV) thrombus following acute ST-elevation myocardial infarction (STEMI) has not been evaluated on a national scale and was the focus of this investigation.

Methods: We used the 2003 to 2013 Nationwide Inpatient Sample database to identify adults 18 years old with a principal diagnosis code of ST-elevation myocardial infarction. Patients were divided into two groups defined by the presence or absence of LV thrombus. Clinical characteristics and in-hospital outcomes were studied using relevant statistics. Multiple linear and logistic regression models were conducted to identify factors associated with LV thrombus.

Results: Of 1,035,888 STEMI patients hospitalized in the U.S from 2003-2013, 1,982 (0.2%) developed acute in-hospital LV thrombus. Compared to no LV thrombus, patients with LV thrombus were more likely to have in-hospital complications; acute ischemic and hemorrhagic stroke, acute renal failure, gastrointestinal bleed, cardiogenic shock, in-hospital cardiac arrest and mortality. They also had longer mean length of stay and higher hospital charges. Factors associated with LV thrombus included: anterior/anterolateral STEMI, acute or chronic heart failure

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“All authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation”

Conflict of Interest Statement: Aiham Albaeni, Khaled Chatila, Hind A. Beydoun, May A. Beydoun, Mohammad Morsy, and Wissam I. Khalife declare no conflict of interest.

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with reduced ejection fraction, atrial fibrillation, LV aneurysm, Left heart valvular disease, acute or chronic deep venous thrombosis/pulmonary embolism and alcohol abuse. Patients with LV thrombus were less likely to be female [AOR 0.66, 95% CI (0.51-0.84)].

Conclusion: The identification of factors associated with early development of LV thrombus following STEMI, will help direct resources for specific high-risk group and prompt cost-effective therapies. Gender variability in LV thrombus development warrant further investigations.

Keywords

Left ventricular thrombus; ST-elevation myocardial infarction; in-hospital complications; Resource utilization; Gender variation

Introduction

Left ventricular (LV) thrombus following ST-elevation myocardial infarction (STEMI) carries significant risk for catastrophic complications including stroke and death.¹⁻⁵ It may also increase resources utilization, length of hospital stay and overall health care burden. The general characteristics of STEMI patients who are at increased risk for developing LV thrombus has been reported in small studies.⁵⁻⁹ However, larger studies are likely to uncover further important information that will help in identifying patients who are more likely to develop acute post-myocardial infarction LV thrombus, and aid in directing resources and appropriate therapies. We sought to study factors associated with in-hospital development of LV thrombus following ST-elevation myocardial infarction using the Nationwide Inpatient Sample.

Methods

Data were obtained from the 2003-2013 Healthcare Cost and Utilization Project Nationwide Inpatient Sample. The database contains patient-level hospital discharge data provided by States that participate in the Agency for Healthcare Research and Quality's Project. Using a sampling probability of about 20%, the design of the Nationwide Inpatient Sample (NIS) is stratified and includes U.S. non-rehabilitation, community hospitals, with the target universe being all acute care hospital discharges in the United States.¹⁰⁻¹¹

We used the International Classification of Diseases, Ninth Edition Clinical Modification (ICD-9-CM) codes of ST-elevation myocardial infarction (410.01, 410.11, 410.21, 410.31, 410.41, 410.51, 410.61, and 410.81) to identify adults ≥ 18 years of age with a principal diagnosis of STEMI. These codes have been used in prior studies to identify patients with STEMI.¹²⁻¹⁴ The code 410.91 was not included as it may reflect NSTEMI.¹⁴ In order to ensure that STEMI was the primary reason for admission, STEMI ICD-9-CM codes with fifth digit subclassification of 0 (episode of care unspecified) or 2 (subsequent episode of care) were excluded. We also excluded patients who were transferred from another hospital (378,579 patients) to avoid duplication of records, and patients who did not have in-hospital imaging (Echocardiogram, Cardiac Magnetic Resonance Imaging, Cardiac Computed Tomography or Left Ventricular angiogram) that could detect LV thrombus (682,981 patients). This selection process yielded a final sample of 1,035,888 STEMI patients.

Thereafter, we identified patients with LV thrombus using the ICD-9 code 429.79 and divided the sample into two groups; STEMI patient with in-hospital LV thrombus and STEMI patients without in-hospital LV thrombus (Supplement eFigure1).

Inpatient outcomes of interest included: **(A)** Factors associated with the development of LV thrombus, **(B)** In-hospital complications following STEMI (Acute ischemic and hemorrhagic stroke, Acute renal failure, gastrointestinal bleed, Cardiogenic shock, In-hospital arrest, and Mortality), **(C)** Length of hospital stay (days), **(D)** Total hospital charges (US \$) adjusted for inflation using the 2018 consumer price index.

Patients' clinical characteristics and comorbidities were compared between the two groups and included: age, gender, race, hypertension, diabetes, coronary artery disease, acute and chronic heart failure, STEMI location (anterior/anterolateral, inferior/inferolateral/inferoposterior, lateral/posterior/unspecified), left ventricular aneurysm, left heart valvular disease (aortic stenosis, aortic regurgitation, mitral stenosis, and mitral regurgitation), atrial fibrillation, coronary artery disease, chronic renal failure, acute and chronic Deep Venous Thrombosis/Pulmonary Embolism (DVT/PE), hypocoagulable state (coagulation factors deficiency, thrombocytopenia), drug abuse, alcohol use, smoking, dyslipidemia, obesity, previous myocardial infarction, previous percutaneous coronary intervention, and previous coronary artery bypass grafting. Relevant co-morbidities that were not readily available in the Nationwide Inpatient Sample were obtained using the appropriate ICD-9-CM codes (Supplemental eTable 1).

Statistical analyses were performed using Stata 12.0, accounting for survey design complexity, sampling weights, primary sampling units and strata. Consequently, population estimates of proportions, means and regression coefficients were obtained. Standard errors were estimated using Taylor series linearization. First, patient demographics and co-morbidities were compared between STEMI patient with and without LV thrombus using Pearson χ^2 test for categorical variables and linear regression (1-way ANOVA) for continuous variables. Second, means and proportions of outcomes of interest were similarly compared. Third, multiple logistic regression models were conducted to examine factors associated with LV thrombus following STEMI. These models were adjusted for age, gender, race, atrial fibrillation, coronary artery disease, acute and chronic heart failure with reduced ejection fraction, acute and chronic heart failure with preserved ejection fraction, LV aneurysm, LV valvular disease, STEMI location, acute and chronic DVT/PE, percutaneous coronary interventions, the use of thrombolytics, hypo-coagulopathy, alcohol use, hypertension, diabetes, dyslipidemia, obesity, prior myocardial infarction, prior percutaneous coronary interventions, prior coronary artery bypass graft, and chronic renal failure. Fourth, factors associated with in hospital mortality and in hospital acute ischemic stroke were explored using univariate and multivariate logistic regression (supplement eTables 2,3). Fifth, gender differences of patients who had LV thrombus were studied using Pearson χ^2 test for categorical variables and linear regression (1-way ANOVA) for continuous variables. Sixth, due to the possibility that imaging might be under coded in the NIS, analysis of factors associated with LV thrombus was also performed on the entire sample without excluding patients without reported imaging and revealed similar results (Supplement eTable 8).

Categorical variables were expressed as n (%) and continuous variables as mean $\pm$ SEM. The OR/ β coefficients and 95% confidence interval were used to report the results of regression models. All P values were 2-sided and Type I error was set at 0.05.

Results

Our study included 1,035,888 STEMI patients admitted to U.S hospitals from 2003 to 2013. One thousand nine hundred and eighty-two patients (0.2%) developed in-hospital LV thrombus following STEMI. There were no differences in mean age (61.9 years vs. 61.5 years $p=0.58$) and race between patients who developed LV thrombus and those who did not. However, the LV thrombus group had fewer females compared to no LV thrombus group (24.7% vs. 30.6%, $P=0.01$).

In univariate analyses, the LV thrombus group was more likely to have atrial fibrillation, chronic or acute heart failure with reduced ejection fraction, left heart valvular disease, left ventricular aneurysm, history of chronic or acute pulmonary embolism or deep venous thrombosis and anterior or anterolateral myocardial infarction (77% vs. 37.9% $P<0.001$). Compared to those with no LV thrombus, patients with LV thrombus were less likely to have prior coronary artery bypass grafts, prior or current (during hospital admission) percutaneous coronary interventions, and thrombolytics (table 1).

Compared to no LV thrombus group, patients with LV thrombus were more likely to have acute in-hospital ischemic stroke (5.9% vs. 0.9% $P<0.001$) in univariate analysis, and following adjustment [AOR 5.58, 95% CI (3.53-8.81)]. They also had higher in-hospital mortality (6.8% Vs 3.9%, $p=0.006$) in univariate analysis, but following adjustment the higher mortality was not significant [AOR 1.006, 95% CI (0.54-1.87)], and was related to other factors such as cardiogenic shock and cardiac arrest (supplement eTable 2).

LV thrombus group had higher hospital length of stay ($8.12 \pm$ SEM 0.36 days vs. $4.52 \pm$ SEM 0.03 days, $P<0.001$), and higher hospitalization charges ($\$119,463 \pm$ SEM $\$6,516$ vs. $\$85,352 \pm$ SEM $\$1,139$, $P<0.001$). Other in-hospital complications following STEMI are shown in table 2.

Factors associated with increased development of LV thrombus following STEMI included: anterior and antero-lateral STEMI as compared to inferior/inferolateral and posterior/lateral, reduced ejection fraction whether due to chronic [AOR 2.24, 95% CI (1.73-2.90)] or acute heart failure with reduced ejection fraction [AOR 1.64, 95% CI (1.15-2.34)], the presence of left heart valvular disease (mitral stenosis, mitral insufficiency, aortic stenosis and aortic insufficiency), the presence of LV aneurysm [AOR 5.32, 95% CI (2.63-10.75)], history of alcohol use, history of atrial fibrillation, chronic or acute deep venous thrombosis or pulmonary embolism as a surrogate to hypercoagulable state [AOR 2.30, 95% CI (1.28-4.16)].

On the other hand, the presence of thrombocytopenia and coagulation defects that are associated with higher bleeding risk were associated with decreased risk of LV thrombus [AOR 0.44, 95% CI (0.23-0.87)], female gender was also associated with decreased risk of LV thrombus both in the univariate and the multivariate analyses [AOR 0.65, 95% CI

(0.51-0.83)]. We further evaluated difference between males and females who had LV thrombus (total of 1,982 patients in supplemental eTable 4). There were not significant gender differences in the above identified factors that increased the risk of having LV thrombus. Furthermore, we have included those factors in addition to factors that were significantly different in univariate analysis (Age, Coronary artery disease and dyslipidemia) in the multivariate analysis model as showed in (table 3).

Discussion

In this large national database, the in-hospital development of LV thrombus following admission with ST-elevation myocardial infarction was 0.2%. The presence of LV thrombus was associated with increased in-hospital complications, length of hospital stay and hospitalization charges. Factors associated with LV thrombus included: anterior/anterolateral STEMI, acute or chronic heart failure with reduced ejection fraction, atrial fibrillation, LV aneurysm, left heart valvular disease, acute or chronic deep venous thrombosis/pulmonary embolism (DVT/PE), history of alcohol use, and male gender.

The incidence of LV thrombus following STEMI in our study was significantly lower than literature reports for various reasons. The Nationwide Inpatient Sample (NIS) is an administrative database that provides in-patient information; therefore, there is no follow-up period to detect more cases. Furthermore, the database spans a large number of participating acute care hospitals in the United States, reaching more than 4,500 hospitals in 46 states in 2011.¹¹ Therefore, a wide range of clinical practice, availability of diagnostic imaging (i.e. Cardiac Magnetic Resonance Imaging , contrast echocardiography, etc.), availability of experts to read those images, and dependency on coding with the possibility of under coding for LV thrombus or imaging are all expected factors to impact the reported incidence of LV thrombus following STEMI.

The literature reports of LV thrombus following STEMI ranged widely from 3-8% in the post-PCI era¹⁵⁻¹⁸ to 15-30% in the pre-PCI era.¹⁹⁻²² The lower incidence in the post-PCI era could be attributed to multiple factors, namely: the wide and prompt use of PCI for the treatment of STEMI which contributed to smaller infarct size and improved cardiac perfusion, the use of dual antiplatelet therapy, the increased adherence to guideline-directed therapies for heart failure with reduced ejection fraction which improved cardiac remodeling and contractility. In addition, the wide variability between studies in the incidence of LV thrombus could be due to different study designs, sample sizes, follow-up periods, and modalities used to diagnose LV thrombus which have wide differences in sensitivity and specificity.¹⁶ Echocardiography is widely available but has a low sensitivity of 35-40% in detecting LV thrombus, which improve to 60% with contrast use, and a specificity of 90%. On the other hand, delayed enhancement Cardiac Magnetic Resonance (CMR) has the highest accuracy for detecting LV thrombus (sensitivity of 88% and specificity of 99%),^{16,22} but is not widely available in all hospitals. Weinsaft et al reported that 33% of LV thrombus that were detected by CMR were actually missed by echocardiography¹⁶. In addition, timing of imaging performance and the natural history of LV thrombus development will likely to affect the reported incidence. Studies evaluating the natural history of LV thrombus development after myocardial infarction have reported variable

results¹⁷⁻²². But, in general the majority of LV thrombus develop within one to two weeks after the acute event^{17,20,22}, and those who develop thrombus after 4-6 months are usually patients with low ejection fraction or large anterior/anterolateral STEMI¹⁷. A recent study by Cambroner-Cortinas et al using CMR for LV thrombus detection, with one year follow up, revealed 33% (9 out of 27) new LV thrombus cases at 6 months¹⁷. All those factors were likely to contribute to the low LV thrombus incidence following STEMI in our study. Despite previously mentioned limitations inherent in the database, smaller studies of highly selected STEMI patients (anterior and anterolateral) might overestimate the incidence of LV thrombus.

LV thrombus following STEMI is associated with increased risk of embolic complications, requiring the use of anticoagulation in addition to dual antiplatelet therapy in the setting of acute STEMI. The current American Heart Association and European Society of Cardiology recommendations depend on observational and retrospective data with regards to the type and length of treatment,^{23,24} but in general, anticoagulation is recommended for 3-6 months with the aim to decrease the length of triple therapy to minimize the risk of bleed. The presence of thrombus increases the risk of stroke by 4-folds as reported by Maniwa et al³; similarly, in our study, after adjusting for Atrial fibrillation and other factors that increased hypercoagulable state, the presence of LV thrombus increased the risk of stroke by 5- folds (Supplement eTable 3).

In our study, LV thrombus group was also more likely to suffer from other in-hospital complications related to their severity of illness, lower cardiac function, and the use of anticoagulation, such as: GI bleed, hemorrhagic conversion of stroke, acute renal failure, inhospital cardiogenic shock and arrest. The higher in-hospital mortality rate reported in the LV thrombus group was mainly driven by increased incidence of cardiogenic shock, cardiac arrest, stroke, acute renal failure and other complications rather than the thrombus itself, as shown in multivariate analysis (supplement eTable 2). The higher in hospital complications in the LV thrombus group and possibly the need for anticoagulation could certainly contributed to longer hospital length of stay and higher hospital charges.

Prior studies have reported factors associated with increased incidence of LV thrombus following STEMI, mainly factors that increase blood stasis, endothelial injury and hypercoagulability (Virchow triad) such as: anterior myocardial infarction, low ejection fraction, apical akinesis and hypokinesia, large infarct size, and LV aneurysm.^{5,6,9,16,17,22} Similarly, in our study, anterior and anterolateral STEMI and LV aneurysms were associated with increased incidence of LV thrombus. Acute and chronic heart failure with reduced ejection fraction were used as surrogates for low EF in the database and were significantly associated with LV thrombus. In addition, the presence of atrial fibrillation, acute or chronic deep venous thrombosis or pulmonary embolism as surrogates for hypercoagulable state were also associated with LV thrombus.

Prior smaller STEMI studies have shown a non-significant trend for more LV thrombus following STEMI in males compared to females,^{6,7} and probably did not reach statistical significance due to small sample size. In the current study, despite similarity between males and females who develop LV thrombus (supplement eTable 4), females were significantly

less likely to have LV thrombus following STEMI in multivariate analysis (table3). The unadjusted proportion of STEMI patients who developed LV thrombus was higher in females compared to males in the age group 18-44 as shown in figure 1. This finding could be partially explained by increased hypercoagulable state in females under age 45 due to higher estrogen levels and the use of contraceptives.^{25,26} However, such finding could not be further investigated due to database limitations (lack of medications and laboratory measures). Gender variability in the development of LV thrombus following STEMI warrant further investigations.

The use of alcohol in our study was associated with increase risk of LV thrombus, a finding that could be related to alcohol induced complications such as cardiomyopathy, liver disease, pancreatic disease, polyneuropathy and decreased mobility that could lead to increase venous stasis and hypercoagulable state. A study by Zoller et al, found an association of alcohol intake with increased incidence of venous thromboembolism even in the absence of alcohol related complications.²⁷ Such finding could partially explain the increased hypercoagulable state in alcohol users, however literature reports on alcohol impact on venous thromboembolism has been conflicting^{28,29} and require further investigations.

This study should be interpreted in light of the following limitations. First, the Nationwide Inpatient Sample is an administrative database; therefore, the validity of the data highly depends on the accuracy of coding, and the possibility of under coding of LV thrombus or images used to diagnose it could not be excluded. Second, the database contains only inpatient information; therefore, follow-up after hospital discharge is lacking, which will limit the ability to detect more cases of LV thrombus. Third, our study sample included a wide variety of US hospitals that spanned 46 states with differences in resources and expertise availability, therefore different imaging procedures with variable sensitivity and specificity were used to diagnose LV thrombus. In addition, timing of such imaging plays an important role in detecting LV thrombus as shown by Khoury et al where 50% of LV thrombus cases were detected on the second echocardiogram performed before discharge³⁰. Therefore, the possibility of missing cases of LV thrombus if an echo is performed too early following STEMI could not be eliminated.

Fourth, the database does not provide information about medications that could affect inpatient outcomes, or inpatient laboratory tests that are associated with increased incidence of LV thrombus such as CRP and fibrinogen levels. A study by Shacham Y et al had shown that elevated admission CRP and fibrinogen levels in anterior wall STEMI patients were associated with early development of LV thrombus³¹. Fifth, the severity of valvular heart disease (mitral or aortic) could not be assessed using an administrative database, and some of the ICD-9 codes (for example ICD-9 code 396.2) could be used to code for both aortic stenosis and mitral regurgitation. Therefore, we created a variable called (left heart valvular disease) that account for all mitral and aortic valvular diseases (stenosis and insufficiency), a fact that will limit our ability to explain the association with LV thrombus development. Finally, similar to other observational studies, the presence of residual confounding by measured or unmeasured confounders could not be completely eliminated.

In summary, our study may provide further help in identifying STEMI patient who are at higher risk of developing in-hospital LV thrombus, and therefore guide cost-effective utilization of diagnostic procedures. Further research is needed to investigate gender variability of LV thrombus following STEM.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights:

- LV thrombus Following STEMI increases in-hospital complications.
- Early identification of LV thrombus will help direct resources for specific high-risk group.
- LV thrombus increases with anterior STEMI, HFrEF, atrial fibrillation and alcohol abuse.
- Female patients were less likely to develop LV thrombus following STEMI.

Fig. 1:
Proportion of LV thrombus case per 10,000 cases of ST-elevation myocardial infarction by gender.

Table 1:

Demographic and Clinical Characteristics of ST elevation Myocardial Infarction in the NIS Years 2003 to 2013

Variables	No LV thrombus	LV thrombus	P Value
	n = 1,033,906	n = 1,982	
Age, mean (SE)	61.5 (0.1)	61.9 (0.7)	0.58
Age group			0.86
18-44 years	93,860 (9.1%)	162 (8.2%)	
45-54 years	238,991 (23.1%)	444 (22.4%)	
55-79 years	588,467 (56.9%)	1,166 (58.8%)	
80 years	112,588 (10.9%)	210 (10.6%)	
Female	316,922 (30.6%)	489 (24.7%)	0.01
White	644,205 (62.3%)	1,222 (61.7%)	0.98
Black	61,834 (6.0%)	136 (6.9%)	
Hispanic	64,332 (6.2%)	128 (6.4%)	
Asian/Pacific Islander	18,134 (1.8%)	37 (1.9%)	
Native American	3,600 (0.3%)	*	
Other/missing	241,802 (23.4%)	454 (22.9%)	
Comorbidities			
Alcohol abuse	32,276 (3.1%)	119 (6.0%)	<0.001
Smoking	104,640 (10.1%)	173 (8.7%)	0.37
Drug abuse	22,411 (2.2%)	50 (2.5%)	0.61
Obesity	116,209 (11.2%)	173 (8.7%)	0.10
Hypertension	598,167 (57.9%)	1,051 (53.0%)	0.06
Diabetes mellitus	258,945 (25%)	474 (24%)	0.60
History of Coronary artery disease	893,393 (86%)	1,604 (81%)	0.001
Chronic renal failure	45,358 (4.4%)	116 (5.9%)	0.15
Dyslipidemia	584,141 (56.5%)	953 (48.1%)	<0.001
Coagulopathy	33,119 (3.2%)	47 (2.4%)	0.33

Variables	No LV thrombus	LV thrombus	P Value
	n = 1,033,906	n = 1,982	
Atrial Fibrillation	108,026 (10.4%)	371 (18.7%)	<0.001
Chronic HFrEF	166,411 (16.1%)	796 (40.1%)	<0.001
Acute/Chronic HFpEF	8,289 (0.8%)	42 (2.1%)	0.002
Acute HFrEF	32,165 (3.1%)	217 (10.9%)	<0.001
Left Valvular Disease	80,965 (7.8%)	270 (13.6%)	<0.001
LV aneurysm	2360 (0.2%)	44 (2.2%)	<0.001
Prior CABG	271,68 (2.6%)	19 (1.0%)	0.03
Prior PCI	93,527 (9.0%)	116 (5.8%)	0.02
Prior MI	72,237 (7.0%)	115 (5.8%)	0.36
Acute/chronic DVT/PE	9,229 (0.9%)	57 (2.9%)	<0.001
Thrombolytics	31,205 (3.0%)	24 (1.2%)	0.03
PCI	800,712 (77.4%)	1,406 (70.9%)	0.002
DES	464,975 (45.0%)	808 (40.8%)	0.09
BMS	291,749 (28.2%)	548 (27.6%)	0.81
CABG	85,942 (8.3%)	158 (7.9%)	0.80
STEMI location			<0.001
Anterior/Anterolateral	392,024 (37.9%)	1,527 (77.0%)	
Inferior/Inferolateral/Inferoposterior	555,262 (53.7%)	330 (16.7%)	
Lateral/posterior/unspecified	86,620 (8.4%)	125 (6.3%)	

SE: standard error, %: percentage.

* : indicates very small number. NIS: nationwide inpatient sample, LV: left ventricular, HFrEF: heart failure with reduced ejection fraction, HFpEF: heart failure with preserved ejection fraction, CABG: coronary artery bypass grafting, PCI: percutaneous coronary intervention, MI: myocardial infarction, DVT/PE: Deep venous Thrombosis/Pulmonary Embolism, DES: Drug eluting Stent, BMS: bare metal stent, STEMI: ST-elevation myocardial infarction.

Table 2:

In-hospital Outcomes in Patients with ST Elevation Myocardial Infarction in the NIS Years 2003 to 2013

Variables	No LV thrombus	LV thrombus	P Value
	n = 1,033,906	n = 1,982	
Acute Stroke	9,065 (0.9%)	117 (5.9%)	<0.001
Hemorrhagic conversion	3,333 (0.3%)	37 (1.8%)	<0.001
Acute renal failure	63,304 (6.1%)	189 (9.5%)	0.005
Gastrointestinal bleed	43,607 (4.2%)	124 (6.3%)	0.04
Cardiogenic shock	75,567 (7.3%)	319 (16.1%)	<0.001
In-Hospital cardiac arrest	16,636 (1.6%)	64 (3.2%)	0.008
Mortality	40,557 (3.9%)	136 (6.8%)	0.006
Length of hospital stay, mean ($\pm$ SE)	4.52 (0.03)	8.12 (0.36)	<0.001
Total hospital charges, mean ($\pm$ SE)	\$85,352 (\$1,139)	\$119,463 (\$6,516)	<0.001

SE: standard error, %: percentage. NIS: nationwide inpatient sample, LV: left ventricular.

Table 3:

Association of Select Factors with LV thrombus in patient with ST Elevation Myocardial Infarction in the NIS Years 2003 to 2013

Variables	Unadjusted OR (95% CI)	P Value	Adjusted OR (95% CI)	P Value
Age	1.002 (0.99-1.01)	0.57	0.99 (0.98-1.001)	0.08
Female	0.74 (0.59-0.93)	0.01	0.65 (0.51-0.83)	0.001
Alcohol abuse	1.99 (1.33-2.97)	0.001	1.74 (1.14-2.65)	0.01
Atrial Fibrillation	1.97 (1.53-2.55)	<0.001	1.55 (1.16-2.06)	0.003
STEMI Location				
Anterior/anterolateral	Reference	-	Reference	-
Inferior/inferolateral	0.15 (0.12-0.20)	<0.001	0.18 (0.14-0.24)	<0.001
Lateral/posterior	0.37 (0.24-0.57)	<0.001	0.38 (0.25-0.57)	<0.001
Chronic HFrEF	3.50 (2.86-4.28)	<0.001	2.24 (1.73-2.90)	<0.001
Acute HFrEF	3.83 (2.81-5.24)	<0.001	1.64 (1.15-2.34)	0.006
LV valvular disease	1.85 (1.37-2.51)	<0.001	1.40 (1.02-1.92)	0.04
LV aneurysm	9.96 (5.06-19.58)	<0.001	5.32 (2.63- 10.75)	<0.001
DVT/PE	3.27 (1.84-5.82)	<0.001	2.30 (1.28-4.16)	0.006
PCI	0.71 (0.57-0.88)	0.002	0.89 (0.70-1.12)	0.31
Thrombolytics	0.40 (0.17-0.96)	0.04	0.43 (0.18-1.04)	0.06
Thrombocytopenia/Coagulopathy [†]	0.73 (0.39-1.38)	0.34	0.44 (0.23-0.87)	0.02

OR: odds ratio, NIS: Nationwide Inpatient Sample, OR: odds ratio, CI: confidence interval, STEMI: ST-elevation myocardial infarction, HFrEF: heart failure with reduced ejection fraction, LV: left ventricular, DVT/PE: Deep venous Thrombosis/Pulmonary Embolism, PCI: percutaneous coronary intervention.

Additional variables used for adjustment: Race, Hypertension, Diabetes, coronary artery disease, obesity, Dyslipidemia, prior myocardial infarction, prior PCI, prior CABG, Chronic kidney disease. Heart failure with preserved ejection fraction.

[†]Thrombocytopenia/Coagulopathy indicates primary and secondary thrombocytopenia, hypo-coagulable states due to factors deficiency (i.e. Factor IIIV, Factor IX, Factor X, Factor XI, afibrinogenemia). Von Willebrands Disease.