

Clinical Characteristics and Risk Factors of Left Ventricular Thrombus after Acute Myocardial Infarction: A Matched Case-control Study

Yue-Xin Jiang¹, Lin-De Jing², You-Hong Jia¹

¹Key Laboratory of Clinical Trial Research in Cardiovascular Drugs, Ministry of Health, State Key Laboratory of Cardiovascular Diseases, Fuwai Hospital, National Center for Cardiovascular Diseases, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing 100037, China
²Department of Cardiology, Qinghai Provincial People's Hospital, Xining, Qinghai 810007, China

Abstract

Background: Left ventricular thrombus (LVT) is reported to be a common complication in acute myocardial infarction (AMI) patients. And it has the potential to cause systemic embolism. This retrospective study was to present the current situation of LVT in clinical practice, as well as to evaluate the clinical characteristics and the risk factors of LVT after AMI.

Methods: LVT cases ($n = 96$) were identified from 13,732 AMI (non-ST elevation myocardial infarction was excluded) patients in Fuwai Hospital's electronic medical records system from January 2003 to January 2013. The controls ($n = 192$) were gender- and age-matched AMI patients without LVT during this period. A conditional logistic regression (fitted by the Cox model) was performed to identify the independent risk factors.

Results: The incidence of LVT after AMI was 0.7%. Univariate analysis indicated that the anterior myocardial infarction (especially extensive anterior myocardial infarction), lower left ventricular ejection fraction (LVEF), $LVEF \leq 40\%$, severe regional wall motion abnormalities (RWMA), pericardial effusion, and left ventricular aneurysm were all related to LVT after AMI. The independent risk factors obtained from the conditional logistic regression analysis were lower LVEF (odds ratio (OR) = 0.891, 95% confidence interval (CI): 0.828–0.960), extensive anterior myocardial infarction ($OR = 6.403$, 95% CI : 1.769–23.169), severe RWMA ($OR = 7.348$, 95% CI : 1.323–40.819), and left ventricular aneurysm ($OR = 6.955$, 95% CI : 1.673–28.921).

Conclusions: This study indicated that lower LVEF, extensive anterior myocardial infarction, severe RWMA, and left ventricular aneurysm were independent risk factors of LVT after AMI. It also suggested that further efforts are needed for the LVT diagnosis after AMI in clinical practice.

Key words: Left Ventricular Thrombus; Myocardial Infarction; Risk Factors

INTRODUCTION

Left ventricular thrombus (LVT) is a complication with a severe risk of brain and peripheral arterial embolization after acute myocardial infarction (AMI) that leads to a high rate of morbidity and mortality. Previous studies have reported that the incidence of LVT after AMI was between 20% and 60%.^[1-3] Moreover, the incidence is extremely high among extensive anterior wall myocardial infarction patients.^[4,5] In recent years, timely therapy during the acute phase of AMI, such as anticoagulation, thrombolytic, and primary percutaneous coronary intervention (PPCI), has reduced the incidence of LVT.^[6] Limited previous studies on LVT after AMI differed in the study population and study design, and

relevant studies could barely stand for the current situation of LVT after AMI since the limited overall sample size. We retrospectively scanned through a large sample size, 13,732

Address for correspondence: Dr. You-Hong Jia,
Key Laboratory of Clinical Trial Research in Cardiovascular Drugs,
Ministry of Health, State Key Laboratory of Cardiovascular Diseases,
Fuwai Hospital, National Center for Cardiovascular Diseases,
Chinese Academy of Medical Sciences and Peking Union Medical
College, No. 167, Beilishi Road, Xicheng District, Beijing 100037, China
E-Mail: jiayouhong@fuwai.cams.cn

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

© 2015 Chinese Medical Journal | Produced by Wolters Kluwer - Medknow

Received: 02-04-2015 **Edited by:** Xin Chen

How to cite this article: Jiang YX, Jing LD, Jia YH. Clinical Characteristics and Risk Factors of Left Ventricular Thrombus after Acute Myocardial Infarction: A Matched Case-control Study. Chin Med J 2015;128:2415-9.

Access this article online

Quick Response Code:



Website:
www.cmj.org

DOI:
10.4103/0366-6999.164869

AMI patients from January 2003 to January 2013 in Fuwai Hospital, to identify LVT cases in this study. The primary aim of this study was to present the current situation of LVT diagnosis in clinical practice, and to analyze the clinical characteristics and risk factors of LVT after AMI in Chinese patients.

METHODS

Study population and study design

This study was performed as a matched case-control study. All of the LVT cases and controls were identified from AMI patients who were admitted to Fuwai Hospital from January 2003 to January 2013, some of them were admitted for percutaneous coronary intervention treatment within 4 weeks after AMI. The controls were hospitalized AMI patients without LVT during the same period, and they were matched 2:1 with LVT cases for the variables of age, gender, and admission date. Some of the matched cases were excluded according to the exclusion criteria. The inclusion criteria of the LVT group and the control group were hospitalized AMI patients with or without LVT before discharge, respectively. The exclusion criteria were combination with previous myocardial infarction, non-ST segment myocardial infarction, arterial or venous thrombosis history, thrombotic hematological disorders, atrial fibrillation, dilated cardiomyopathy (DCM), severe valvular disease, and other related diseases. All of the patients involved in this study signed informed consent forms. This study was approved by the Research Ethics Board of Fuwai Hospital.

The data collection was based on the clinical information recorded in the case report forms in Fuwai Hospital's electronic medical records system. These records included the following: (1) The available medical data, including body mass index (BMI), combined diseases, infarction location, length of duration before hospital admission, ventricular fibrillation, and urgent revascularization (which included anticoagulation, PPCI, and coronary artery bypass grafting [CABG]); and (2) records of echocardiography, including left ventricular ejection fraction (LVEF), regional wall motion abnormality (RWMA), mitral regurgitation (MR), pericardial effusion, and left ventricular aneurysm.

Definitions

The diagnosis of AMI was made according to the WHO criteria as revised in 2000,^[7] in which a cardiac troponin rise accompanied by either typical ischemic chest symptoms, pathological Q waves, ST elevation or depression, or coronary intervention are diagnostic of MI. LVT was diagnosed before discharge and was based on the clear visibility of thrombus masses within the left ventricular cavity on echocardiography, coronary computed tomography angiography (CTA), cardiac magnetic resonance imaging (CMR), or left ventriculography. The duration before admission referred to the period (days) of the occurrence of AMI to the time of admission.

Echocardiography

Echocardiography was performed using commercially available ultrasound instruments. The LVEF was assessed by the echocardiography according to the modified Simpson's rule.^[8] The RWMA was assessed by the 16-segmental model according to the American Society of Echocardiography into five grades (normal, hypokinesia, akinesia, dyskinesia, and aneurysm).^[9] Severe RWMA was defined as akinesia or dyskinesia. A color Doppler evaluation was used to identify the presence of MR, and depending on the width and depth of the regurgitated jets, the presence of MR was classified into three grades of severity: (1) Mild; (2) moderate; and (3) severe.

Statistical analysis

Epi Info 3.0 (Centers for Disease Control and Prevention, USA) was used to set up the database. All of the analyses were performed using SPSS 19.0 (SPSS Inc., Chicago, IL, USA), with a nominal level of statistical significance defined as a two-tailed $P < 0.05$. The data were presented as the means \pm standard deviation (SD). The Chi-square test was used for the comparison of the nonparametric data, and the t -test was used to compare the continuous variables. A conditional logistic regression model (fitted by the Cox multivariate model) was performed to identify the independent risk factors of LVT after AMI. The odds ratio (OR) and the 95% confidence interval (CI) between the two groups were calculated.

RESULTS

Baseline between left ventricular thrombus cases and controls

Ninety-six LVT cases and 192 controls were enrolled by scanning through 13,732 patients with AMI in Fuwai Hospital's electronic medical records system from January 2003 to January 2013. According to the results of the scan, the incidence of LVT was 0.7%. Gender, age, BMI, smoking history, combined diseases (such as hypertension, diabetes mellitus, hyperlipidemia, cerebral stroke, or deep venous thrombosis [DVT] history, etc.), duration before admission, and the timing of the first imaging examination after admission were not significantly different (all $P > 0.05$) between the two groups. Eleven patients in the LVT group and 20 patients in the control group were admitted to the hospital within 24 h after experiencing chest pain, whereas the other patients were admitted for further therapy within 1-month after receiving brief treatment for their AMI. The percentages of triple vessel disease between the two groups had no significant difference ($P > 0.05$) [Table 1].

One patient in LVT group experienced abdominal pain and limb hemiplegia 20 days after thrombolysis. Emboli exfoliation to the brain was diagnosed after cerebral magnetic resonance imaging detection, and a superior mesenteric arterial embolism was diagnosed because of the typical symptoms.

Clinical outcomes between left ventricular thrombus cases and controls

The infarction location, ventricular fibrillation

Table 1: Baselines of LVT cases and controls

| Variables | Controls (<i>n</i> = 192) | LVTs (<i>n</i> = 96) | <i>P</i> |
|---|-------------------------------------|-------------------------------------|----------|
| Male, <i>n</i> (%) | 171 (89.1) | 86 (89.6) | 1.000 |
| Age, years, mean ± SD | 53.78 ± 11.02 | 54.03 ± 11.07 | 0.854 |
| BMI, mean ± SD | 25.35 ± 3.54 | 25.00 ± 3.19 | 0.416 |
| Smoking, <i>n</i> (%) | 124 (64.6) | 64 (66.7) | 0.793 |
| Hypertension, <i>n</i> (%) | 98 (51.0) | 41 (42.7) | 0.211 |
| Diabetes mellitus, <i>n</i> (%) | 45 (23.4) | 21 (21.9) | 0.882 |
| Hyperlipidemia, <i>n</i> (%) | 109 (56.8) | 53 (55.2) | 0.803 |
| Cerebral stroke, <i>n</i> (%) | 6 (3.1) | 5 (5.2) | 0.515 |
| DVT, <i>n</i> (%) | 1 (0.5) | 1 (1.0) | 1.000 |
| Duration before admission, days, mean ± SD | 11.60 ± 8.17 (<24 h, <i>n</i> = 28) | 13.45 ± 8.27 (<24 h, <i>n</i> = 20) | 0.073 |
| Implicated vessels (triple vessels), <i>n</i> (%) | 61 (33.2) | 21 (25.6) | 0.313 |

The duration before admission referred to the period (number of days) of the occurrence of acute myocardial infarction to admission. LVTs: Left ventricular thrombus; BMI: Body mass index; DVT: Deep venous thrombosis; SD: Standard deviation.

caused by the AMI, Killip classification, and urgent revascularization (including thrombolysis, PPCI, and CABG) were recorded and analyzed. The results demonstrated that the infarction location (both anterior wall and extensive anterior wall) showed a statistically significant difference between the two groups ($P < 0.001$) [Table 2].

Echocardiography between left ventricular thrombus cases and controls

The LVEF, severe RWMA, moderate to high levels of MR, pericardial effusion, and ventricular aneurysm were all analyzed to identify the univariate values. The results showed that the LVEF, LVEF $\leq 40\%$, severe RWMA, pericardial effusion, and left ventricular aneurysm were all significantly different between the two groups (all $P < 0.001$) [Table 3].

Characteristics of left ventricular thrombus cases

All of the LVT cases were detected by an imaging examination. Eighty-nine of the cases were detected by echocardiography, 3 were detected by CMR, 3 were detected by left ventriculography and 1 was detected by CTA. The median timing of the LVT detection was the 17th day after AMI, whereas 75% of the LVT cases were detected within 21 days after the AMI. One of the LVT was detected on the 1st day of chest pain. Almost all of the LVT masses were located in the apex of heart, except for one that was located in the cavity, which was considered to be a pedunculated thrombus associated with the wall. Most of the LVT masses were described as a single mural mass, except for one described as a cloudy area, one as multiple active masses and four as multiple mural masses.

Independent risk factors of left ventricular thrombus after acute myocardial infarction

After the application of the conditional logistic regression model (fitting by the Cox multivariate model) with the above statistically significant variables, the independent risk factors of LVT after AMI were a lower LVEF ($OR = 0.891$, 95% $CI: 0.828-0.960$), extensive anterior myocardial infarction ($OR = 6.403$, 95% $CI: 1.769-23.169$), severe

Table 2: Clinical outcomes of LVT cases and controls, *n* (%)

| Variables | Controls (<i>n</i> = 192) | LVTs (<i>n</i> = 96) | <i>t</i> | <i>P</i> |
|--------------------------------|----------------------------|-----------------------|----------|----------|
| Infarction location | | | | |
| Anterior wall | 51 (26.6) | 36 (37.5) | 3.63 | <0.001 |
| Extensive anterior wall | 18 (9.4) | 58 (60.4) | 83.84 | <0.001 |
| Ventricular fibrillation | 5 (2.6) | 6 (6.3) | 2.32 | 0.189 |
| Killip classification ≥ 3 | 5 (2.6) | 7 (7.3) | 3.52 | 0.113 |
| Urgent revascularization | 66 (34.4) | 41 (42.7) | 1.90 | 0.196 |
| Thrombolysis | 41 (21.4) | 26 (27.1) | 1.18 | 0.302 |
| PPCI | 30 (15.6) | 18 (18.8) | 0.45 | 0.506 |
| CABG | 1 (0.5) | 0 (0) | 0.50 | 1.000 |

LVTs: Left ventricular thrombus; PPCI: Primary percutaneous coronary intervention; CABG: Coronary artery bypass grafting.

Table 3: Results of Echocardiography for LVT cases and controls

| Variables | Controls (<i>n</i> = 192) | LVTs (<i>n</i> = 96) | <i>t</i> | <i>P</i> |
|---|----------------------------|-----------------------|----------|----------|
| LVEF, %, means ± SD | 56.53 ± 8.95 | 42.50 ± 9.67 | 12.21 | <0.001 |
| LVEF $\leq 40\%$, <i>n</i> (%) | 17 (8.9) | 40 (41.7) | 43.41 | <0.001 |
| Severe RWMA, <i>n</i> (%) | 2 (1.0) | 47 (49.0) | 104.07 | <0.001 |
| Moderate to high level MR, <i>n</i> (%) | 6 (3.1) | 6 (6.3) | 1.57 | 0.223 |
| Pericardial effusion, <i>n</i> (%) | 2 (1.0) | 15 (15.6) | 24.51 | <0.001 |
| Left ventricular aneurysm, <i>n</i> (%) | 6 (3.1) | 62 (63.5) | 134.03 | <0.001 |

Severe RWMA was defined as akinesis or dyskinesis according to the echocardiography. LVTs: Left ventricular thrombus; LVEF: Lower left ventricular ejection fraction; RWMA: Regional wall motion abnormalities; MR: Mitral regurgitation; SD: Standard deviation.

RWMA ($OR = 7.348$, 95% $CI: 1.323-40.819$), and left ventricular aneurysm ($OR = 6.955$, 95% $CI: 1.673-28.921$) [Table 4].

DISCUSSION

Our study indicated that the independent risk factors of LVT after AMI were a lower LVEF, extensive anterior myocardial

Table 4: Independent risk factor analysis for LVT after acute myocardial infarction

| Variables | Regression coefficient | SE | OR | 95% CI | P |
|---|------------------------|-------|-------|--------------|-------|
| LVEF | -0.115 | 0.038 | 0.891 | 0.828–0.960 | 0.002 |
| Severe RWMA | 1.994 | 0.875 | 7.348 | 1.323–40.819 | 0.023 |
| Extensive anterior wall myocardial infarction | 1.857 | 0.656 | 6.403 | 1.769–23.169 | 0.005 |
| Left ventricular aneurysm | 1.939 | 0.727 | 6.955 | 1.673–28.921 | 0.008 |

Severe RWMA was defined as akinesis or dyskinesis according to the imaging diagnosis. LVEF: Lower left ventricular ejection fraction; RWMA: Regional wall motion abnormalities; MR: Mitral regurgitation; SE: Standard error; OR: Odds ratio; CI: Confidence interval; LVT: Left ventricular thrombus.

infarction, severe RWMA and left ventricular aneurysm. However, the existence of moderate to high level of MR showed no difference between the LVT group and the control group in our study, whereas other researches differed in whether MR is responsible for the LVT formation.

Our study involved 96 LVT cases and 192 matched controls to balance the selection bias. All of cases were scanned from the 13,732 AMI patients of Fuwai Hospital's electronic medical records system. All of the patients were treated with anticoagulation and dual-antiplatelet therapy after AMI onset, and 37.2% underwent urgent revascularization. The incidence of LVT in this study was 0.7%, which was lower than the incidences in other prospective studies (5–8%).^[5,10] It may be a reflection of the low detection rate of LVT after AMI in real clinical practice. The reason for this lower incidence in this study may also be that the timing of the echocardiography detection after AMI onset, as well as during admission, was hardly consistent in a retrospective study. Moreover, the length of the time of hospitalization in this study was not sufficient to monitor the LVT formation. Thus, a silent occurrence of LVT may exist after discharge.

The mechanism of LVT after AMI is still unclear, with a hypothesis that includes dysfunction of the coagulation and myocardial injury, as well as hemodynamic abnormalities.^[4] An extensive area of myocardial injury stimulates a large quantity of fibrin, erythrocytes, and platelets to adhere to the exposed collagen, which impels a coagulation cascade and results in the formation of a thrombus. In our study, the percentage of extensive anterior wall myocardial infarction was much higher than that of other infarction locations among the LVT patients, possibly because extensive myocardial injury is related to a larger explosion of coagulation, which induces a greater amount of platelet adhesion. Additionally, severe RWMA is usually accompanied by an extensive myocardial infarction, which results in a higher incidence of LVT.

The LVEF, as well as the ventricle vortex flow because of the RWMA and formation of the ventricular aneurysm, is comprised in the mechanism of hemodynamic abnormality after AMI. In our study, the average LVEF in the LVT group was lower than that in the controls ($[42.50 \pm 9.67]\%$

vs. $[56.53 \pm 8.95]\%$) in the univariate analysis; a higher LVEF also showed a protective effect in the multivariate analysis ($OR = 0.891$, 95% CI : 0.828–0.960). Previous studies indicated that an LVEF $\leq 40\%$ was an independent risk factor of LVT after AMI.^[11,12] However, we used an LVEF $\leq 40\%$ rather than the LVEF in the conditional logistic model, which yielded no identifiable statistically significant differences. The reason for this result may be that when the LVEF $\leq 40\%$, the variable for the extensive anterior myocardial infarction and severe RWMA might be both closely related to each other.

In this study, most of the LVT were formed in the left ventricular apex (95 of the 96 cases), and 91.2% of the LVT masses were formed within the ventricular aneurysm. This phenomenon was also reported in another study. This may be a reason that the apex is the reverse point of the left ventricular inflow and outflow tracts, which can cause a ventricle vortex flow, even the deposition of blood, especially after the aneurysm formation.^[13,14] Moreover, when the RWMA develops into akinesis or dyskinesis, a circulation disorder will act to increase the incidence of LVT, which was also indicated by this study.

The studies with DCM patients reported by Kalaria *et al.*^[15] and Ozdemir *et al.*^[16] inferred that severe MR may have a protective role in LVT formation, as it was identified only in the non-LVT group. However, other studies with AMI patients reported by Ascione *et al.*^[17] and Van Dantzig *et al.*^[18] showed that MR was not protective for LVT and that a moderate to high level of MR may facilitate LVT formation. In contrast to the studies described above, a moderate to high level of MR had no effect on the formation of LVT. It has been acknowledged that accurate and timely treatment, such as reperfusion and drug therapy in the early phase of AMI, has an effect on improving the level of MR.^[19] As the patients involved in this retrospective study had received different therapies before admission and the lengths of time after the AMI symptoms before admission were not the same, their MR levels might differ, making it difficult to ascertain the relationship between the level of MR and the formation of LVT.

Some studies have indicated that immediate anticoagulation measures were able to reduce the incidence of LVT among AMI patients with extensive RWMA. Therefore, anticoagulation treatment is recommended for AMI patients who have a low bleeding risk.^[20,21] In addition, the European Society of Cardiology guidelines for the management of AMI in patients in Europe advise that timely dual-antiplatelet therapy with warfarin should be administered to patients with LVT for at least 3 months (Grade IIa B).^[22] However, there is still no evidence of reliable treatments or of biomarkers to prevent or predict LVT formation, embolism and therapeutic evaluations.

Limited by the retrospective nature of this matched case-control research, we used a selected study population in our study, and selection bias may exist for the patients involved in this study. And since this study covered the AMI patients from January 2003 to January 2013, prehospital and in-hospital treatment imbalance might be exist according to the changes of ST-segment elevation myocardial

infarction (STEMI) guidelines throughout these years, even though the changes are unclear with LVT appearance in other studies. And according to the limited sample size in this study, it is impossible for sub-group analysis based on the admitted time. Thus, it is also difficult to tell the efficiency of different treatment in preventing LVT appearance. At the same time, because of the different lengths of the AMI's duration of symptoms and treatment before admission, it is difficult to analyze the particular timing of LVT's formation or the biomarkers of LVT. Moreover, due to the improvements in treating left ventricular dysfunction and hemodynamic failure in AMI patients, the incidence of LVT in this study might be lower than in other reports.

In conclusion, this study demonstrated that the incidence of LVT was higher in AMI patients with a lower LVEF, extensive anterior myocardial infarction, severe RWMA and left ventricular aneurysm. Thus, AMI patients who have these risks factors should be closely monitored by physicians to avoid the formation of LVT or embolism caused by LVT. Additionally, further studies are needed to evaluate the protective roles of reperfusion, anticoagulation, and antiplatelet therapy in reducing the incidence of LVT and their related complications. Furthermore, the incidence of LVT after AMI in this retrospective study, though bias might exist, reflects the absence of awareness of LVT diagnosis in clinical practice, which need further efforts to improve.

Financial support and sponsorship

This work was supported by grants from the National Science and Technology Major Project (No. 2012ZX09303-008-001) from the Ministry of Science and Technology of China and the Key Laboratory Construction Project from the Ministry of Health of China.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

- Jordan RA, Miller RD, Edwards JE, Parker RL. Thrombo-embolism in acute and in healed myocardial infarction. I. Intracardiac mural thrombosis. *Circulation* 1952;6:1-6.
- Lanzillo C, Di Roma M, Sciahbasi A, Minati M, Maresca L, Pendenza G, *et al.* Cardiac magnetic resonance detection of left ventricular thrombus in acute myocardial infarction. *Acute Card Care* 2013;15:11-6.
- Rabbani LE, Waksmonski C, Iqbal SN, Stant J, Sciacca R, Apfelbaum M, *et al.* Determinants of left ventricular thrombus formation after primary percutaneous coronary intervention for anterior wall myocardial infarction. *J Thromb Thrombolysis* 2008;25:141-5.
- Stokman PJ, Nandra CS, Asinger RW. Left ventricular thrombus. *Curr Treat Options Cardiovasc Med* 2001;3:515-521.
- Osherov AB, Borovik-Raz M, Aronson D, Agmon Y, Kapeliovich M, Kerner A, *et al.* Incidence of early left ventricular thrombus after acute anterior wall myocardial infarction in the primary coronary intervention era. *Am Heart J* 2009;157:1074-80.
- Solheim S, Seljeflot I, Lunde K, Bjørnerheim R, Aakhus S, Forfang K, *et al.* Frequency of left ventricular thrombus in patients with anterior wall acute myocardial infarction treated with percutaneous coronary intervention and dual antiplatelet therapy. *Am J Cardiol* 2010;106:1197-200.
- Alpert JS, Thygesen K, Antman E, Bassand JP. Myocardial

infarction redefined – A consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. *J Am Coll Cardiol* 2000;36:959-69.

- Cheitlin MD, Armstrong WF, Aurigemma GP, Beller GA, Bierman FZ, Davis JL, *et al.* ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography: Summary article: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASE Committee to Update the 1997 Guidelines for the Clinical Application of Echocardiography). *Circulation* 2003;108:1146-62.
- Schiller NB, Shah PM, Crawford M, DeMaria A, Devereux R, Feigenbaum H, *et al.* Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. *J Am Soc Echocardiogr* 1989;2:358-67.
- Gianstefani S, Douiri A, Delithanasis I, Rogers T, Sen A, Kalra S, *et al.* Incidence and predictors of early left ventricular thrombus after ST-elevation myocardial infarction in the contemporary era of primary percutaneous coronary intervention. *Am J Cardiol* 2014;113:1111-6.
- Shacham Y, Leshem-Rubinow E, Ben Assa E, Rogowski O, Topilsky Y, Roth A, *et al.* Frequency and correlates of early left ventricular thrombus formation following anterior wall acute myocardial infarction treated with primary percutaneous coronary intervention. *Am J Cardiol* 2013;111:667-70.
- Chiarella F, Santoro E, Domenicucci S, Maggioni A, Vecchio C. Predischarge two-dimensional echocardiographic evaluation of left ventricular thrombosis after acute myocardial infarction in the GISSI-3 study. *Am J Cardiol* 1998;81:822-7.
- Delemarre BJ, Visser CA, Bot H, Dunning AJ. Prediction of apical thrombus formation in acute myocardial infarction based on left ventricular spatial flow pattern. *J Am Coll Cardiol* 1990;15:355-60.
- van Dantzig JM, Delemarre BJ, Bot H, Visser CA. Left ventricular thrombus in acute myocardial infarction. *Eur Heart J* 1996;17:1640-5.
- Kalaria VG, Passannante MR, Shah T, Modi K, Weisse AB. Effect of mitral regurgitation on left ventricular thrombus formation in dilated cardiomyopathy. *Am Heart J* 1998;135 (2 Pt 1):215-20.
- Ozdemir N, Kaymaz C, Daglar E, Karakaya O, Akçay M, Ozkan M. Severe mitral regurgitation may prevent mural thrombus formation within the left ventricle with systolic dysfunction. *Jpn Heart J* 2002;43:495-503.
- Ascione L, Antonini-Canterin F, Macor F, Cervasato E, Chiarella F, Giannuzzi P, *et al.* Relation between early mitral regurgitation and left ventricular thrombus formation after acute myocardial infarction: Results of the GISSI-3 echo substudy. *Heart* 2002;88:131-6.
- Van Dantzig JM, Delemarre BJ, Bot H, Koster RW, Visser CA. Usefulness of mitral regurgitation in protecting against left ventricular thrombus after acute myocardial infarction. *Am J Cardiol* 1995;75:1270-2.
- American College of Emergency Physicians; Society for Cardiovascular Angiography and Interventions, O'Gara PT, Kushner FG, Ascheim DD, Casey DE Jr, Chung MK, *et al.* 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol* 2013;61:e78-140.
- Reeder GS, Lengyel M, Tajik AJ, Seward JB, Smith HC, Danielson GK. Mural thrombus in left ventricular aneurysm: Incidence, role of angiography, and relation between anticoagulation and embolization. *Mayo Clin Proc* 1981;56:77-81.
- Turpie AG, Robinson JG, Doyle DJ, Mulji AS, Mishkel GJ, Sealey BJ, *et al.* Comparison of high-dose with low-dose subcutaneous heparin to prevent left ventricular mural thrombosis in patients with acute transmural anterior myocardial infarction. *N Engl J Med* 1989;320:352-7.
- Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC), Steg PG, James SK, Atar D, Badano LP, Blömstrom-Lundqvist C, *et al.* ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *Eur Heart J* 2012;33:2569-619.