Clinical Case Reports International

Case Report
Published: 23 Jun, 2023

Biventricular Thrombosis Associated with Painless Myocardial Infarction: A Case Report and Literature Review

*Qu Y[1], Deng Y[1], Liu L[1], Wu C[2], Wei L[3], He X[1] and Sun Y[1]**

[1]Department of VIP & Geriatrics, Peking University Shenzhen Hospital, China

[2]Department of Cardiology, Peking University Shenzhen Hospital, China

[3]Department of B Ultrasonic, Peking University Shenzhen Hospital, China

Abstract

Background: Biventricular thrombosis, although rare, can be fatal. Early detection and timely intervention are crucial in determining the outcome of the disease.

Case Report: A 50-year-old male patient, with a history of active smoking and hypertension, was admitted due to slightly difficult breathing and edema in the feet for three days. Transthoracic echocardiography revealed mural thrombosis in both the left and right ventricular apex. Subsequent examination confirmed the presence of diabetes mellitus and old myocardial infarction. After adequate anticoagulation with warfarin, the thrombus resolved completely within 12 days.

Conclusion: The risk of biventricular thrombosis in patients with myocardial infarction may be underestimated, especially in those with painless myocardial infarction. Clinicians should focus on high-risk populations, such as anterior wall myocardial infarction combined with low left ventricular ejection fraction.

Keywords: Biventricular thrombosis; Anticoagulation; Left/right ventricular apex; Diabetes mellitus

OPEN ACCESS

Correspondence:
Yansun Sun, Department of VIP & Geriatrics, Peking University Shenzhen Hospital, 1120 Lianhua Road, Futian District Shenzhen, Guangdong, 518036 China,
E-mail: 1213217873@qq.com/ yansunsun@pkuszh.com

Received Date: *07 Jun 2023*
Accepted Date: *19 Jun 2023*
Published Date: *23 Jun 2023*

Citation:
Qu Y, Deng Y, Liu L, Wu C, Wei L, He X, et al. Biventricular Thrombosis Associated with Painless Myocardial Infarction: A Case Report and Literature Review. Clin Case Rep Int. 2023; 7: 1565.

Abbreviations

BT: Biventricular Thrombosis; NT- pro BNP: NT- pro Brain Natriuretic Peptide; CTNI: Cardiac Troponin I; TTE: Transthoracic Echocardiography; LAD: Left Anterior Descending Artery; PCI: Percutaneous Coronary Intervention; LVEF: Left Ventricular Ejection Fraction; LV: Left Ventricular; STEMI: ST-Segment Elevation Myocardial Infarction; INR: International Normalized Ratio; CMR: Cardiac Magnetic Resonance; NOACs: Novel Oral Anticoagulants; PE: Pulmonary Embolization; SE: Systemic Embolization; COVID-19: Coronavirus Disease 2019

Background

Biventricular Thrombosis (BT) is a rare but fatal complication that warrants attention due to its high risk of pulmonary and systemic embolism, resulting in elevated mortality rates. Incidence and epidemiological data on BT are still largely unknown, and the causes are multifactorial. Early detection and timely intervention are crucial in determining the outcome of the disease. In this report, we present a case of an Asian man with mild congestive heart failure, who was diagnosed with painless myocardial infarction and BT, and eventually achieved successful thrombosis resolution.

Case Presentation

Patient information

A 50-year-old Asian man, transferred from a community hospital, presented with slight shortness of breath upon waking from sleep, edema in his feet for three days, and a cough without production for the past month. He had a history of well-controlled hypertension for five years. There was no record of chronic illness before his current condition, no alcohol or drug abuse, active smoking.

Clinical findings and diagnostic assessment

Physical examination revealed a blood pressure of 151/111 mmHg, pulse rate of 126 beats/

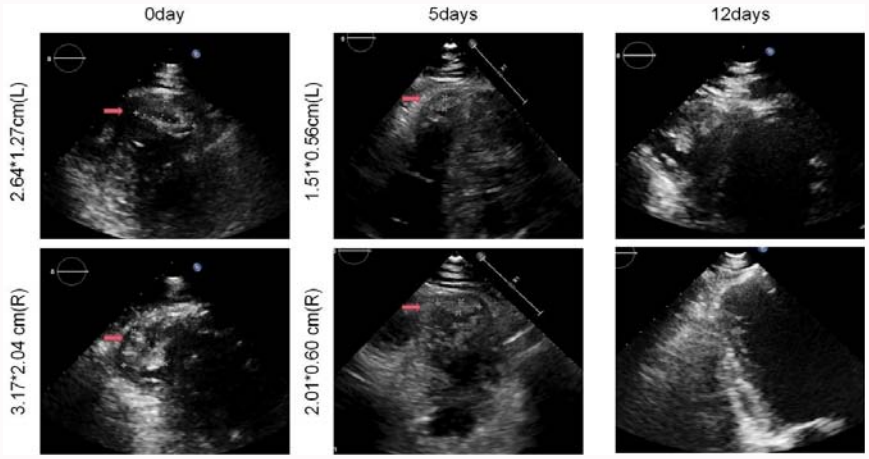
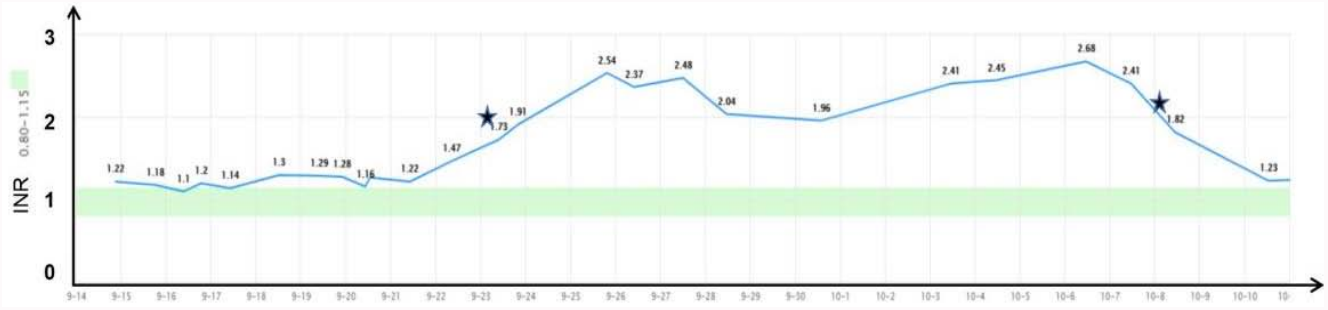


Figure 1: Echocardiographic study of apical four-chamber views obtained, (a) On admission, the left ventricle is significantly dilated with apical thinning. The left and right ventricles have prominent apical masses with thrombotic features (red arrows); (b) the intracardiac thrombus on day 5 becomes smaller; (c) the intracardiac thrombus on day 12 completely resolves after adequate anticoagulation with warfarin.



Supplementary Figure 1: Trajectories of INR over time.

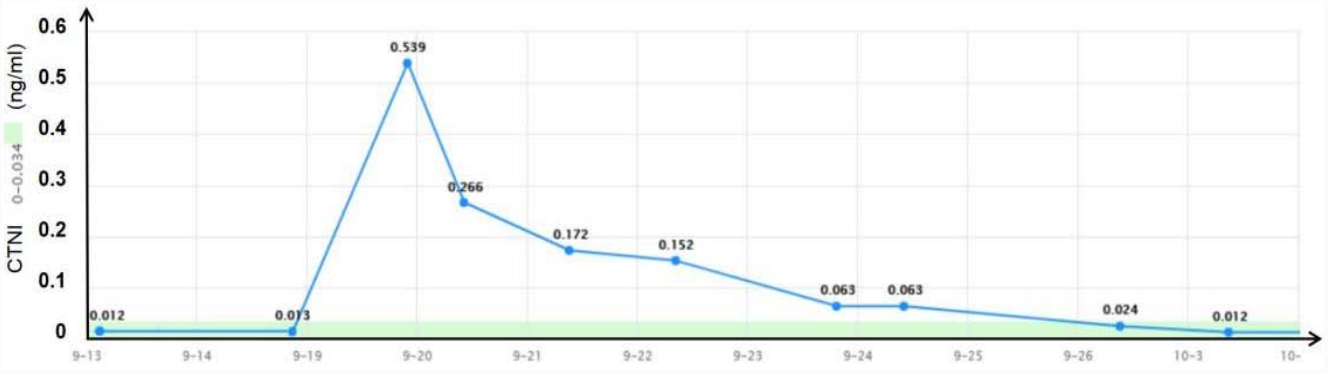


Figure 2: Trajectories of CTNI over time.

min, jugular venous distention, bilateral pulmonary rales, displaced cardiac impulse, an S3 gallop, and bipedal 2+ pitting edema. However, laboratory workup showed diabetes with a high fasting blood glucose level of 9.3 mmol/L, mild congestive heart failure with an NT-pro Brain Natriuretic Peptide (NT- pro BNP) level of 5176 pg/ml, Cardiac Troponin I (CTNI) <0.05 ng/ml. Electrocardiogram revealed sinus tachycardia, complete right bundle branch block, and an old anterior myocardial infarction. A chest computed tomographic scan showed mild pulmonary congestion and small bilateral pleural effusion.

Transthoracic Echocardiography (TTE) revealed a large left atrium and ventricle, hypokinesis of the left ventricle's apical and mid segments, and a large mural thrombus (2.64 cm × 1.27 cm) at the apex of the left ventricle (Supplementary Video 1). The patient also had systolic dysfunction, with an estimated Left Ventricular Ejection Fraction (LVEF) of 38.9%. Additionally, another large mural thrombus (3.17 cm × 2.04 cm) was found in the right ventricular apex (Figure 1, Supplementary Video1, 2), with moderate tricuspid regurgitation and a maximum systolic pulmonary arterial pressure of 41 mmHg, and minimal pericardial effusion.

Therapeutic interventions

Immediate subcutaneous enoxaparin was bridged with 1 mg/kg two times per day, followed by oral administration of warfarin

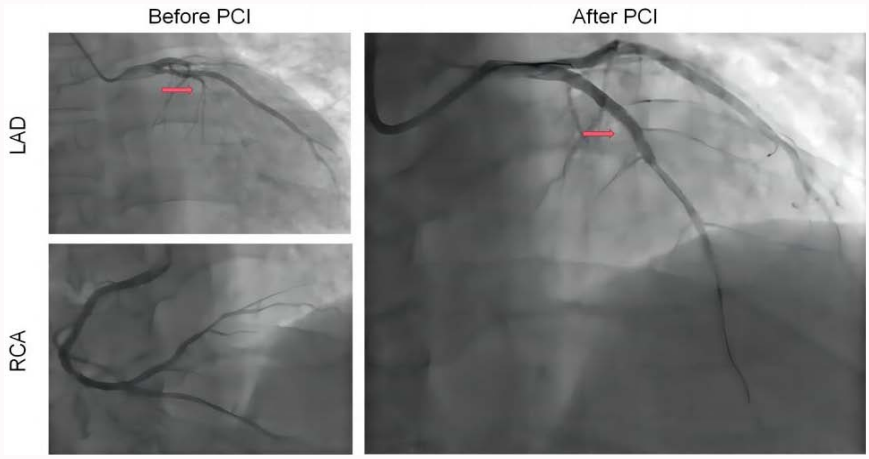
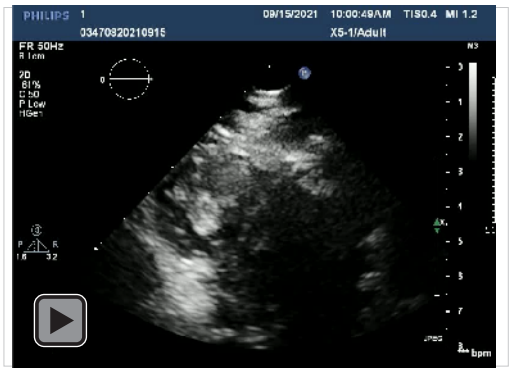


Figure 3: Coronary angiography before and after a stent insertion.

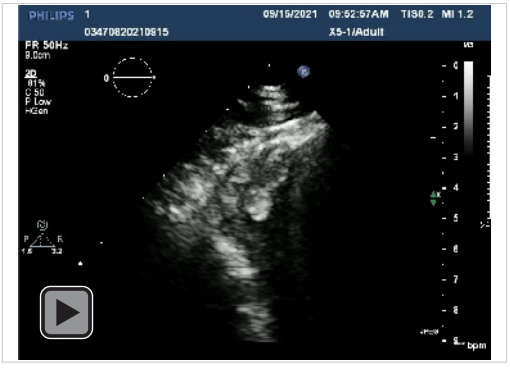
Table 1: Summary of case report characteristics of myocardial infarction combined with biventricular thrombosis (*:0 for No, #:1 for Yes, ^:/ for Absence).

Myocardial Infarction Sites	LVEF (%)	PE/SE	Mobile Thrombosis	Thrombosis Remain	Death	References
Anterior Infarction	37%	0/0*	1#	1	0	Friedman [3]
Old Anterior and Inferior Infarction	/^	0/0	1	1	1	Paul [4]
Posteroinferior and Lateral Infarction	35%	0/1	0	1	0	Espinola-Zavaleta [5]
Apical Infarction	/	1/1	0	0	0	Keeble [6]
Old Anteroseptal Infarction	10%	1/0	0	1	1	Ida [7]
Acute Right Ventricular and Inferior Infarction	20%	0/0	0	0	/	Kaya [8]
Old Anteroseptal Infarction	<20%	1/0	0	0	/	Mujer [9]
Anterolateral Infarction	17%	1/0	1	1	1	Soltani [10]

LVEF: Left Ventricular Ejection Fraction; PE: Pulmonary Embolization; SE: Systemic Embolization



Supplementary Video 1: Biventricular thrombosis in left and right ventricular apex.



Supplementary Video 2: Thrombosis in the right ventricular apex.

potassium. However, seven days later, the INR ranged between 1.2 and 1.6, and the patient suddenly developed hemoptysis, chest tightness, and hypoxemia while standing to urinate, with a blood pressure of 127/84 mmHg, pulse of 112 beats/min, and Spo2 of 92%. No significant changes were noted on the electrocardiogram, and TTE showed no signs of right ventricular pressure overload. Despite high suspicion of pulmonary embolism, computed tomography pulmonary angiography was not performed due to the patient's refusal. On the second day, increased CTNI levels of 0.576 ng/ml were detected (normal range: 0-0.034 ng/ml) (Figure 2). The patient no longer had symptoms of hemoptysis, and the warfarin continued to be adjusted to make the International Normalized Ratio (INR) reach 2-3.

Follow-up and outcomes

Subsequently, the patient was transported to the catheterization laboratory. Coronary angiography demonstrated 100% occlusive thrombosis in the proximal Left Anterior Descending artery (LAD) (Figure 3), a drug-eluting stent was implanted. Serial echocardiographic assessments were performed, and the thrombi gradually decreased in size at five days after adequate anticoagulation and disappeared at 12 days after INR reach 2-3 (Figure 1, Supplementary Figure 1).

Discussion

Biventricular Thrombosis (BT) is a rare but extremely dangerous

disease whose epidemiological, etiological and clinical features have not been thoroughly studied. Despite the high prevalence of myocardial infarction, our literature analysis revealed that only 7 case reports of BT associated with MI were available in full.

Epidemiology

Since the number of BT cases due to MI is extremely rare, we refer to published knowledge on the epidemiology of left ventricular thrombus in myocardial infarction. The incidence of Left Ventricular (LV) thrombus was 6.3% to 15% in patients with ST-Elevation Myocardial Infarction (STEMI), and 19.2% in those with anterior STEMI and LVEF less than 50%, after the widespread use of Percutaneous Coronary Intervention (PCI) [1,2]. This indicates that BT maybe more common in patients with anterior STEMI and lower LVEF after PCI, just as what we found in our case. Considering the high prevalence of MI, it is possible that the incidence of BT is underestimated. Among seven reported cases associated with BT, 3 cases were old myocardial infarction and 5 cases of acute MI, and the features of them were list (Table 1).

Pathophysiology

Thrombus formation involves the three components of Virchow's triad, which include blood stasis, endothelial injury, and hypercoagulability. Myocardial infarction has acute endothelial exposure with associated loss of myocardial contractility, and particularly anterior myocardial infarction with low LVEF, often involves the apex and can lead to blood stasis and endothelial injury [1]. Blood stasis can be triggered by myocardial akinesis or dyskinesis, while endothelial injury can cause inflammation and hypercoagulability by exposing subendothelial tissue and collagen to the circulating blood, leading to the formation of thrombus [11]. A 32-year-old male was reported to have an acute myocardial infarction combined with thrombosis that occurred after cocaine ingestion [5]. The mechanisms of cocaine-induced myocardial infarction include coronary artery spasm and unstable plaques [12]. Myocardial infarction occurs when fragile, but not necessarily narrow atherosclerotic plaques are disrupted under hemodynamic stress [13]. In our cases, as seen in the literature, anterior myocardial infarction with low LVEF supported the formation of BT.

Acquired and hereditary risk factors often trigger hypercoagulability, accelerating thrombosis and embolism, such as Factor V Leiden mutation, antiphospholipid antibody syndrome, heparin-induced thrombocytopenia, and anticoagulant protein deficiency. There has been one reported case of Coronavirus Disease 2019 (COVID-19) complicating anterior wall MI leading to BT formation, where COVID-19-induced hypercoagulability has been widely discussed [14]. Our patient had no history of long-term medication use and showed no evidence of hereditary hypercoagulability, suggesting that decreased ventricular wall contraction caused by myocardial infarction was the key factor in thrombus formation.

Diagnosis

Cardiac Magnetic Resonance (CMR) is considered the gold standard for diagnosing intracardiac thrombi, but it's not commonly used in clinical practice. Instead, BT is usually found incidentally during routine TTE. Out of the seven cases reported, only one was diagnosed with BT through CMI [6], while the others were diagnosed through TTE. This patient underwent a comprehensive evaluation at a community hospital, but incomplete testing may have led to missed diagnoses. During the further TTE exam, low echogenicity intracardiac masses were observed, along with a decreased Left Ventricular Ejection Fraction (LVEF) and weakened ventricular wall contraction. This finding is consistent with the five other cases reported where Myocardial Infarction (MI) was associated with BT. All of these patients had a significant drop in LVEF, with four of them having an LVEF below 30% (excluding the three cases where LVEF was not mentioned) (Table 1). This supports the idea that decreased ventricular wall contraction resulting from MI is one of the key factors in BT formation, caused by slow blood flow and stasis.

Anterior STEMI, proximal LAD infarction, and LVEF below 30% are strong predictors of LV thrombus. However, thrombus formation can also occur in patients with an LVEF greater than 30% or in those with diabetes, which is a major cause of asymptomatic myocardial infarction. Therefore, health education should remind patients to watch their juice intake, as consuming more than 100% of daily recommended fruit juice intake can increase the risk of developing diabetes [15].

Therapy

Anticoagulant therapy remains the cornerstone of treatment for thrombotic diseases. According to the 2021 American Heart Association/American Stroke Association stroke prevention guidelines, in patients with stroke or transient ischemic attack and LV thrombus, systemic antagonists, such as vitamin K antagonists, are recommended for treating left ventricular thrombus after MI for three months (Class I), with a target INR value of 2 to 2.5 [16]. In our case treatment, we used bridging low-molecular- weight heparin anticoagulation with warfarin, and achieved good results. Currently, new oral anticoagulant drugs are started to be use in the treatment of left ventricular thrombus [17], and their efficacy in BT may need to be verified in left ventricular thrombus before they can be widely promoted.

Prognosis

The characteristics of thrombus mobility, including thrombus mobility and protrusion, have been used to identify patients at high risk of embolism. Among seven patients with myocardial infarction, three described thrombus mobility, and there were seven cases of pulmonary or systemic embolism, with four cases of residual thrombus and three deaths after the follow-up period ended (Table 1). We reported that the patient's thrombus was resolved after 12 days of full anticoagulation with warfarin, with no evidence of embolism and a good prognosis.

Conclusion

Currently, the risk of BT in patients with myocardial infarction may be underestimated, especially in those with painless myocardial infarction. Clinicians should focus on high-risk populations, such as anterior wall myocardial infarction combined with low LVEF.

Funding

Shenzhen Science and technology program: JCYJ20210324110004012 and JCYJ20220531094006013. The manuscript editing and polishing was supported by the funding.

References

1. Bulluck H, Chan MHH, Paradies V, Yellon RL, Ho HH, Chan MY, et al. Incidence and predictors of left ventricular thrombus by cardiovascular magnetic resonance in acute ST-segment elevation myocardial infarction

treated by primary percutaneous coronary intervention: A meta-analysis. J Cardiovasc Magn Reson. 2018;20(1):72.

2. McCarthy CP, Vaduganathan M, McCarthy KJ, Januzzi JL, Jr., Bhatt DL, McEvoy JW. Left ventricular thrombus after acute myocardial infarction: Screening, prevention, and treatment. JAMA Cardiol. 2018;3(7):642-9.
3. Friedman HZ, Buda AJ. Biventricular thrombus formation in association with acute myocardial infarction: Diagnosis by two-dimensional echocardiography. J Clin Ultrasound: JCU. 1986;14(4):315-8.
4. Paul V, Foster CJ, Brownlee WC. Cross-sectional echocardiographic demonstration of biventricular thrombus. Int J Cardiol. 1988;18(2):266-70.
5. Espinola-Zavaleta NG, Vargas-Barron J, Vazquez-Neira J, Romero-Cardenas A, Keirns C. Echocardiographic diagnosis of biventricular thrombi in a cocaine-induced myocardial infarction. Echocardiography. 1998;15(5):499-502.
6. Keeble W, VonderMuhll I, Paterson I. Biventricular apical thrombi demonstrated by contrast-enhanced cardiac MRI following anteroapical STEMI and unsuccessful reperfusion therapy. Can J Cardiol. 2008;24(8):E51-E53.
7. Ida H, Hirata K, Takahashi T, Hirosawa T. Thrombi in all four cardiac chambers in a patient with an old anteroseptal myocardial infarction and atrial flutter. BMJ Case Rep. 2014;2014.
8. Kaya A, Hayıroğlu M, Keskin M, Öz A, Aydın BA. Giant biventricular thrombi presenting with acute myocardial infarction. Indian Heart J. 2016;68 Suppl 2(Suppl 2):S153-S155.
9. Mujer M, Kandola SK, Saleh Y, Herzallah K. Rare case of biventricular thrombi complicating pulmonary embolism. BMJ Case Rep. 2019;12(5):e229698.
10. Soltani M, Mansour S. Biventricular thrombi associated with myocardial infarction in a patient with COVID-19. Can J Cardiol. 2020;36(8):1326.e1329-1326.e1311.
11. Camaj A, Fuster V, Giustino G, Bienstock SW, Sternheim D, Mehran R, et al. Left ventricular thrombus following acute myocardial infarction: JACC state-of-the-art review. J Am Coll Cardiol. 2022;79(10):1010-22.
12. Almeida I, Miranda H, Santos H, Santos M, Paula S, Chin J. Cocaine-associated myocardial infarction: Features of diagnosis and treatment. J Electrocardiol. 2021;67:11-12.
13. Mittleman MA, Mintzer D, Maclure M, Tofler GH, Sherwood JB, Muller JE. Triggering of myocardial infarction by cocaine. Circulation. 1999;99(21):2737-41.
14. Hadid T, Kafri Z, Al-Katib A. Coagulation and anticoagulation in COVID-19. Blood Rev. 2021;47:100761.
15. Drouin-Chartier JP, Zheng Y, Li Y, Malik V, Pan A, Bhupathiraju SN, et al. Changes in Consumption of Sugary Beverages and Artificially Sweetened Beverages and Subsequent Risk of Type 2 Diabetes: Results From Three Large Prospective U.S. Cohorts of Women and Men. Diabetes Care. 2019;42(12):2181-9.
16. Kleindorfer DO, Towfighi A, Chaturvedi S, Cockroft KM, Gutierrez J, Lombardi-Hill D, et al. 2021 Guideline for the prevention of stroke in patients with stroke and transient ischemic attack: A guideline from the American Heart Association/American Stroke Association. Stroke. 2021;52(7):e364-e467.
17. Jones DA, Wright P, Alizadeh MA, Fhadil S, Rathod KS, Guttmann O, et al. The use of novel oral anticoagulants compared to vitamin K antagonists (warfarin) in patients with left ventricular thrombus after acute myocardial infarction. Eur Heart J Cardiovasc Pharmacother. 2021;7(5):398-404.