Asian Journal of Cardiology Research

5(2): 36-41, 2021; Article no.AJCR.72715

Myocardial Infarction Revealing Thrombosis of Mechanical Prosthesis in Mitral Position

Désiré Massimbo[1*], Hicham Faliouni[2], Soumaila Nikiema[2], Zouhair Lakhal[2] and Aatif Benyass[1]

[1]Departement of Non-Invasive Explorations, Heart Center of Military Hospital Mohammed V, Faculty of Medicine and Pharmacy of Rabat, Mohammed V University Rabat, Morocco.
[2]Department of Intensive Care and Catheterization, Heart Center of Military Hospital Mohammed V, Faculty of Medicine and Pharmacy of Rabat, Mohammed V University Rabat, Morocco.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

Editor(s):
(1) Dr. Sam Said, Hospital Group Twente, Netherlands.
Reviewers:
(1) Marcos Mello Moreira, University of Campinas, Brazil.
(2) Arda Özyüksel, Biruni University, Turkey.
Complete Peer review History: https://www.sdiarticle4.com/review-history/72715

Case Report

Received 20 June 2021
Accepted 23 August 2021
Published 26 August 2021

ABSTRACT

Aims: This case demonstrates that coronary embolism secondary to prosthetic valve thrombosis, although rare, can occur in patients years after mitro-aortic valve replacement despite adequate anticoagulation.
Presentation of Case: We report the case of a 52-year-old man with a mechanical prosthesis in mitral and aortic position who presented to the emergency department with a recent anterior post-myocardial infarction complicated by reccurent angina. Coronary angiography revealed a complete occlusion of the middle segment of the anterior interventricular artery by a fresh thrombus, despite an INR within the recommended target for mechanical prostheses in the mitral position. The etiological workup was in favor of a thrombosis of the mitral prosthesis. He was successfully treated with antiglycoprotein IIb-IIIa in combination with unfractionated heparin.
Discussion: Myocardial infarction by coronary embolism secondary to prosthetic valve thrombosis is a rare event. A few authors have reported patients with mechanical valve prostheses whose thrombosis caused myocardial infarction in the contexte of inadequate anticoagulation. Case reports in the setting of optimal anticoagulation are even more rare. To date, there is no consensus

**Corresponding author: Email: desire.massimbo@yahoo.fr;*

on the treatment of coronary embolism, although there are many therapeutic options. In this case, 72 hours of intravenous anticoagulation with glycoprotein IIb-III a inhibitors and unfractionated heparin were sufficient to recanalize the anterior interventricular artery with a satisfactory control coronary angiography.

Conclusion: Coronary embolism in mechanical prosthesis wearers in the context of adequate anticoagulation is possible and need to be rapported and explored.

Keywords: Myocardial; infarction; thrombosis; prothesis.

1. NTRODUCTION

Coronary embolism is a rare cause of myocardial infarction, it is important to know this entity and its predisposing factors. One of the most important factors is thromboembolism due to mechanical heart valve prostheses.

Coronary embolism in mechanical prosthesis wearers in the context of inadequate anticoagulation, although exceptional, has been reported in the literature [1,2]. This case report relates the history of a patient with a double mechanical mitral and aortic prosthesis who presented with an acute myocardial infarction following a coronary embolus from mitral prosthesis thrombosis despite optimal anticoagulation.

2. PRESENTATION OF CASE

A 52-year-old man, former smoker, with no other modifiable cardiovascular risk factors, presented to the emergency department 15 hours after the onset of infarct-like chest pain.

In his medical history, he was operated in 2007 for a double mitro-aortic valve replacement by mechanical prosthesis following a tight mitral stenosis associated with a severe aortic insufficiency with favorable outcomes. Since then, he has been under vitamin K antagonist treatment with good compliance and medical follow-up.

On admission, blood pressure was normal with persistent chest pain. The clinical examination was also normal with an audible prosthetic click in mitral and aortic position.

The per-critical electrocardiogram showed necrosis Q waves associated with ST-segment elevation in the anterior leads (Fig. 1), indicating a post-infarction anterior myocardial injury.

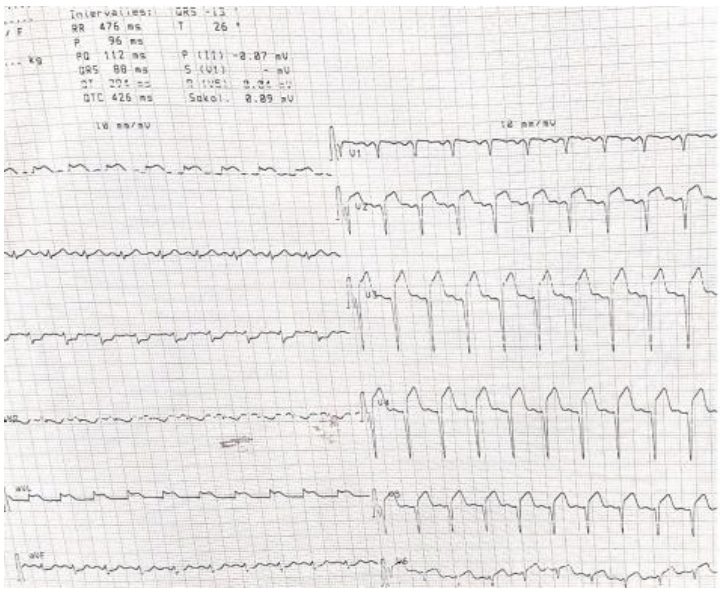

Fig. 1. Necrosis Q waves associated with ST-segment elevation in the anterior leads
Blue Arrow: Necrosis Q wawes

Because of the persistence of the symptomatology, the patient was admitted to the cardiac catheterization room after premedication with antithrombotics. Coronary angiography performed via the right radial approach revealed a thrombus in the middle interventricular artery (Fig. 2 A). The circumflex artery as well as the right coronary network was free of lesions. The therapeutic decision was to dissolve the thrombus with antiglycoprotein IIb-IIIa in combination with unfractionated heparin. The control coronary angiography after three days of anticoagulation showed complete dissolution of the thrombus in the middle interventricular with angiographically healthy coronaries (Fig. 2 B).

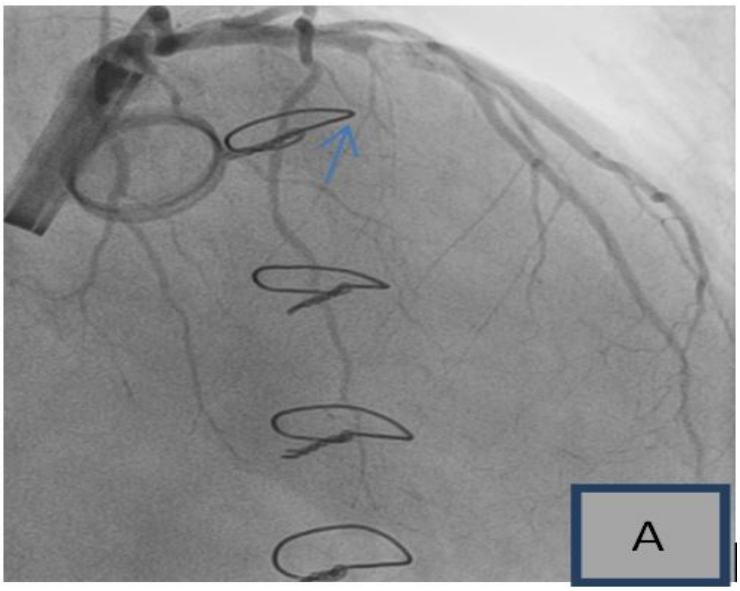

Fig. 2 A. Thrombus in the middle left artery descending (LAD)
Blue Arrow: Thrombus (in white) inside middle LAD

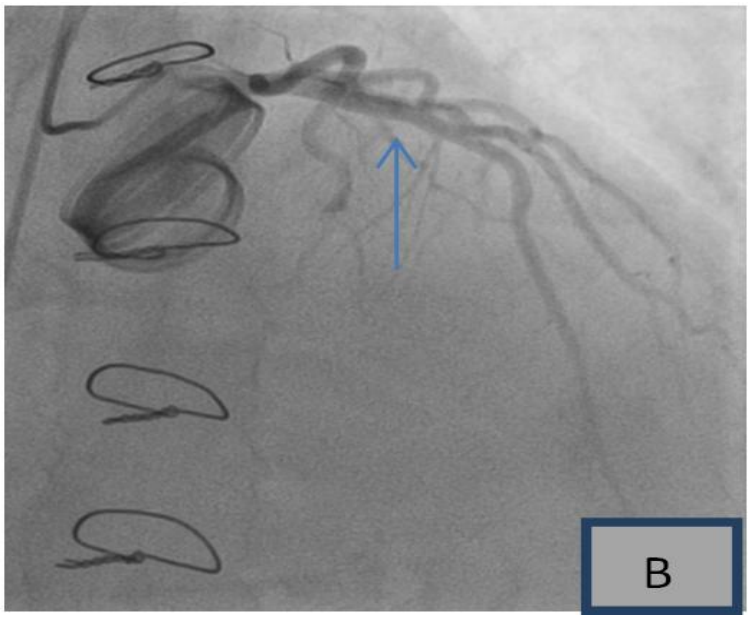

Fig. 2 B. Dissolution of the thrombus after treatment
Arrow: Permeable artery after treatment

Biological workup revealed a positive troponin. However, the patient's INR on admission to the hospital was 3.4 within the target recommended for mechanical prostheses in the mitral position. The metabolic and infectious workup was normal.

The transthoracic ultrasound completed by the transesophageal ultrasound showed the presence of a mobile filiform element on the atrial side of the mitral prosthesis measuring 14 mm, well visualized in 3D (Fig. 3A and 3B). The mechanical prosthesis in the mitral position was free of stenotis (mean gradient at 5 mmHg) and leakage. The aortic mechanical prosthesis was also non stenotic (Vmax 1.02 m/s) and non leaking without suspicious elements. The left ventricle was the site of anteroseptal and inferoseptal wall akinesia with mild systolic dysfunction (LVEF 45%).

3. DISCUSSION

In patients with severe symptomatic valve disease, valve replacement with mechanical or biological prostheses remains the current standard treatment [3]. Mechanical valve replacements are more thrombogenic than bioprostheses, and patients who receive them require long-term anticoagulation to prevent acute thromboembolic events of various locations [4].

Coronary embolism is rare and is an uncommon cause of acute myocardial infarction (AMI) compared with atherosclerosis [5]. Indeed, in a study including 419 patients with autopsied infarcts, only 13% showed evidence of coronary embolism [6].

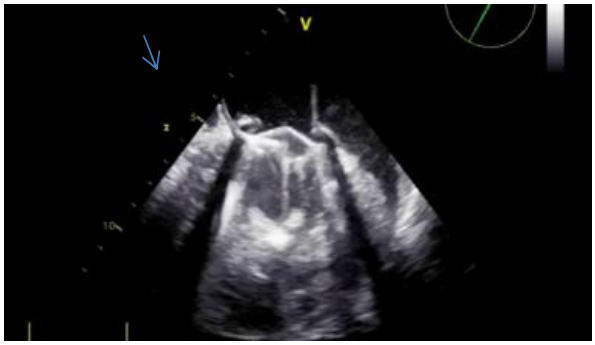

Fig. 3A. Filiform element on the atrial side of the mitral prosthesis ETO
Blue Arrow: Filiform thrombus

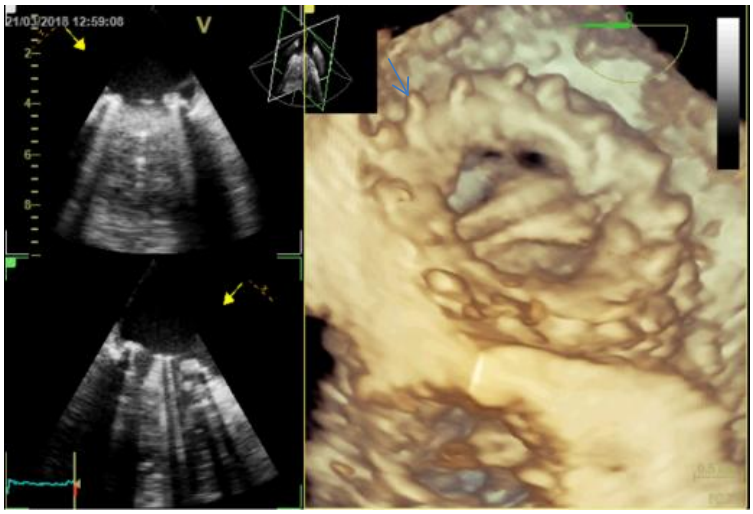

Fig. 3 B. ETO 3D Mitral prothesis thrombus
Blue arrow: Visible thrombus in 3D ETO

When coronary emboli are present, they are mostly in the left coronary network because of the volume of left coronary flow and the morphology of the aortic valve [6]. The causes of coronary embolism are numerous and include infective endocarditis [7], atrial thrombus in the setting of atrial fibrillation [8], atrial myxomas [9], calcific emboli of calcified aortic stenosis [10], as well as mechanical or biological prostheses (in aortic or mitral positions) in the setting of suboptimal anticoagulation [11].

In our case, the coronary embolism was revealed on angiography as a thrombus localized to the middle segment of the middle interventricular artery, despite an INR in the range recommended for mechanical prostheses in mitral position. As recommended in patients with suspected coronary embolism, a transesophageal ultrasound was performed [12]. It ruled out infective endocarditis and any other potential endocardial source of embolism and showed the presence of a mobile filiform element on the atrial side of the mitral prosthesis measuring 14 mm, well visualized in 3D.

To date, there is no consensus on the treatment of coronary embolism, although there are many therapeutic options. In the early phase of a myocardial infarction, thrombolysis or transluminal coronary angioplasty are two options. However, if the source of the embolism is infectious vegetation, thrombolysis is contraindicated because of the risk of distal embolization causing complete distal obstruction in a smaller branch [13].

Percutaneous transluminal coronary angioplasty with stent placement can be performed successfully as demonstrated by Sial and al. in patients with embolic myocardial infarction [14].

Percutaneous coronary angioplasty without stenting is also an alternative, but it may not achieve an optimal angiographic result and TIMI flow.

Aspiration thrombectomy has been described by some authors and is an effective management strategy [15]. Thrombus dissolution with anticoagulant and high-dose glycoprotein inhibitors if the risk of bleeding is low has also been shown to be effective.

Given all these possibilities, the choice of treatment must be based on the practitioner's expertise and depend on the patient's clinical condition and comorbidities.

In this case, 72 hours of intravenous anticoagulation with glycoprotein IIb/IIIa antagonist (tirofiban) in combination with unfractionated heparin were sufficient to recanalize the anterior interventricular artery with a satisfactory control coronary angiography without bleeding complications. We do not use fibrinolytics such as streptokinase or urokinase.

4. CONCLUSION

Coronary embolism secondary to prosthetic valve thrombosis, although rare, can occur in patients years (or months or weeks) after mitro-aortic valve replacement despite adequate anticoagulation. To date, there is no consensus on the treatment of coronary embolism, although treatment options are numerous. Thrombus dissolution with anticoagulant and high-dose glycoprotein inhibitors if the risk of bleeding is low has been shown to be effective.

CONSENT

This document is submitted with the patient's consent.

ETHICAL APPROVAL

It is not applicable.

ACKNOWLEDGEMENTS

We thank the patient who made this case report available.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Iakobishvili Z, Eisen A, Porter A. Acute coronary syndromes in patients with prosthetic heart valves – a case series. Acute Cardiac Care. 2008;10:148–51.
2. Yuce M, Yavuz F, Sari I. A rare cause of myocardial infarction: Coronary embolism in a patient with proshtetic mitral valve trombosis. East Mediterr Health J. 2011; 17:887–88.
3. Khan SS, Trento A, DeRobertis M. Comparaison sur vingt ans du remplacement des tissus et de la valve mécanique. J Thorac Cardiovasc Surg. French. 2001 août;122:257–69.

4. Hammermeister KE, Sethi GK, Henderson WG. Une comparaison des résultats chez les hommes 11 ans après le remplacement d'une valve cardiaque avec une valve mécanique ou une bioprothèse. Étude coopérative des Anciens Combattants sur la cardiopathie valvulaire. N Engl J Med 6 Mai. French. 1993;328 :1289–96.
5. Cheng TO. Embolie coronarienne. Int J Cardiol 24 Juillet. French. 2009;136:1–3.
6. Prizel KR, Hutchins GM, Bulkley BH. Embolie coronarienne et infarctus du myocarde. Ann Intern Med. French. 1978;88 :155–61.
7. Glazier JJ, McGinnity JG, Spears JR. Embolie coronaire compliquant l'endocardite valvulaire aortique: Traitement avec mise en place d'un stent intra-coronaire. Clin Cardiol. French. 1997;20:885–88.
8. Noto N, Osaka T, Yamanaka O, Kobayashi S. [Un cas d'infarctus aigu du myocarde dû à une embolie coronarienne de thrombus auriculaire gauche avec fibrillation auriculaire]. Kokyu To Junkan. French. 1990;38:483–87.
9. Taşdemir K, Andaç MH, Ceyran H, Yasim A. Myxomes provoquant des emboles coronaires entraînant un infarctus aigu du myocarde. Asiatique Cardiovasc Thorac Ann. French. 1999;7:150–52.
10. Mansur AJ, de Miranda RC. Sténose aortique calcique se présentant comme un infarctus aigu du myocarde. J Cardiovasc Surg. French. 1990;31:310–12.
11. Sial JA, Ferman MT, Saghir T, Rasool SI. Embolie coronarienne provoquant un infarctus aigu du myocarde chez un patient avec prothèse valvulaire mitrale: prise en charge réussie par angioplastie. J Pak Med Assoc. French. 2009;59:409–11.
12. Fermeture par cathéter Meier B. du foramen ovale perméable. Circulation. French. 2009;120:1837–41.
13. Dogan M, Acikel S, Aksoy MMN, et al. Coronary saddle embolism causing myocardial infarction in a patient with mechanical mitral valve prosthesis: Treatment with thrombolytic therapy, International Journal of Cardiology. 2009; 135(2) :e47–e48.
14. Akbar Sial J, Ferman M, Saghir T. Coronary embolism causing acute myocardial infarction in a patient with mitral valve prosthesis: Successful management with angioplasty. J Pak Med Assoc. 2009; 59(6):409-11.
15. Hung WC, Wu CJ, Chen WJ, Yang CH, Chang JP. Transradial intracoronary catheter-aspiration embolectomy: For acute coronary embolism after mitral valve replacement, Texas Heart Institute Journal. 2003;30(4) :316–318.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle4.com/review-history/72715