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CASE REPORT

Obstructive dysfunction of a mechanical mitral prosthesis due to pannus and thrombus: a case report

Disfunción obstructiva de prótesis mitral mecánica por pannus y trombo: reporte de un caso

Luis Alberto Morales Reinaga[1]
Daniel Saavedra Rodriguez[1]
Rommer Alex Ortega-Martinez[1]

[1] Hospital Obrero N°2, Caja Nacional de Salud. Cochabamba, Bolivia.

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Corresponding Author: Rommer Alex Ortega Martínez

ABSTRACT

Mechanical prosthetic valve dysfunction is a rare but highly serious complication, particularly in the mitral position. We present the case of a 40-year-old female patient with a history of rheumatic heart disease, mechanical mitral valve replacement, and suboptimal anticoagulation, who was admitted with severe dyspnea, acute pulmonary edema, and a focal neurological deficit consistent with ischemic stroke. Transthoracic echocardiography revealed increased transprosthetic gradients and restricted valve mobility, while transesophageal echocardiography confirmed a mass consistent with a thrombus associated with pannus, causing obstructive dysfunction of the mitral prosthesis. Despite intensive care unit management, ventilatory support, and the medical treatment initiated, the patient's clinical course was unfavorable, characterized by refractory acute pulmonary edema and progressive neurological deterioration; she passed away during hospitalization. This case highlights the importance of timely diagnosis and the differentiation between thrombosis.

Keywords: mechanical mitral prosthesis; prosthetic thrombosis; pannus; stroke; transesophageal echocardiography.

RESUMEN

La disfunción de una prótesis valvular mecánica constituye una complicación poco frecuente, pero de alta gravedad, especialmente en posición mitral. Se presenta el caso de una paciente de 40 años con antecedente de cardiopatía reumática, reemplazo valvular mitral mecánico y anticoagulación subóptima, que ingresó por disnea intensa, edema agudo de pulmón y déficit neurológico focal compatible con accidente cerebrovascular isquémico. El ecocardiograma transtorácico evidenció aumento de los gradientes transprotésicos y restricción de la movilidad valvular, mientras que el ecocardiograma transesofágico confirmó una masa compatible con trombo asociada a pannus, responsable de disfunción obstructiva de la prótesis mitral. A pesar del manejo en unidad de cuidados intensivos, el soporte ventilatorio y el tratamiento médico instaurado, la paciente presentó evolución clínica desfavorable, con edema agudo de pulmón refractario y progresión del compromiso neurológico, falleciendo durante su hospitalización. Este caso resalta la importancia del diagnóstico oportuno y de la diferenciación entre trombosis

Palabras clave: accidente cerebrovascular, ecocardiografía transesofágica, prótesis mitral mecánica; pannus; trombosis protésica

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INTRODUCTION

Mechanical valve prostheses have been a durable and effective strategy for valve replacement in selected patients, especially in young subjects with advanced valve disease and long life expectancy, but their success depends critically on sustained anticoagulation and adequate echocardiographic follow-up, as the prosthetic surface retains permanent thrombogenic potential (1,2). In this scenario, dysfunction of a mechanical mitral prosthesis continues to be an infrequent, but potentially devastating, complication, because it can be expressed as acute heart failure, acute pulmonary edema, ischemic stroke, or even in-hospital death (1,3). Although surgical and transcatheter techniques have expanded the therapeutic options for valvular heart disease, mechanical prostheses continue to require anticoagulation with vitamin K antagonists, and suspension or suboptimal control of treatment significantly increases the risk of prosthetic thrombosis (1,2). Rheumatic valve disease, although it has decreased in some settings, continues to represent a significant clinical burden in regions where barriers to early diagnosis, surgery, or longitudinal control of anticoagulation persist (2,4). From a pathophysiological perspective, prosthetic obstruction may be due to thrombus, pannus, or both; distinguishing them is essential, because the thrombus may respond to selected medical or interventional strategies, while pannus usually requires surgical resolution when it conditions significant obstruction (1,3). Transthoracic and transesophageal echocardiography are the central tools for evaluating morphology, mobility of prosthetic components, transvalvular gradients, and intraprosthetic masses, and their integrated interpretation allows the diagnosis to be guided more accurately (4,5). In this context, current guidelines recommend a multimodal and individualized approach to the study of prosthetic dysfunction, with an emphasis on clinical correlation, advanced cardiovascular imaging, and multidisciplinary decision-making (2,4). Therefore, this report describes the case of a patient with mechanical mitral prosthesis who developed obstructive dysfunction due to thrombus-associated pannus, with torpid evolution, acute refractory lung edema, and ischemic stroke, in order to highlight the importance of timely diagnosis, etiological differentiation, and clinical impact of this potentially fatal complication (1,2).

Case Presentation

A 40-year-old female patient with a history of rheumatic fever in youth. During pregnancy, at approximately 24 weeks of gestation, she consulted for progressive dyspnea and was evaluated in the emergency room, where the transthoracic echocardiogram showed severe mitral stenosis of probable rheumatic etiology. She was admitted for acute pulmonary edema and, once stabilized, underwent balloon mitral valvuloplasty in an external center.

After delivery, mitral valve replacement was performed with a bileaflet mechanical prosthesis, and the patient was indicated for chronic anticoagulation. The procedure was without complications, and she was subsequently discharged. The patient was followed up until April 2026, when she was readmitted to the emergency department of a private clinic for intense shortness of breath, orthopnea, and a focal neurological deficit of the right faciobrachial palsy type. On admission, she presented with frank orthopnea and significant dyspnea.

Physical examination revealed signs of decompensated heart failure, including dyspnea at rest, generalized weakness, lower extremity edema, palpitations, and a decreased prosthetic click on auscultation, a finding that raised suspicion of mitral prosthesis dysfunction. Oxygen saturation was 88%, and initial laboratory tests showed a prothrombin time of 15% and an INR of 1.5, values clearly subtherapeutic for a mechanical mitral valve.

A chest X-ray obtained during the initial evaluation showed cardiomegaly and bilateral interstitial infiltrates with a bat wing pattern, consistent with acute pulmonary edema (Figure 1).

The electrocardiogram showed sinus tachycardia at 150 beats per minute and incomplete right bundle branch block (Figure 2).

Given the suspicion of prosthetic involvement, a transthoracic echocardiogram (TTE) was performed, in which a hyperechogenic image was observed that prevented adequate visualization of one of the prosthetic discs, with a posterior acoustic shadow and no clear evidence of visible thrombi. Continuous Doppler on the mitral valve showed a velocity of 2.5 m/s, a mean gradient of 18 mmHg, and a hemipressure time of 230 ms, findings compatible with prosthetic obstructive dysfunction (Figure 3) and (Figure 4).

Figure 1. PA chest X-ray shows an increased cardiothoracic index (CTI), bilateral interstitial infiltrates in a butterfly pattern, osteosynthesis material, and history of sternotomy.

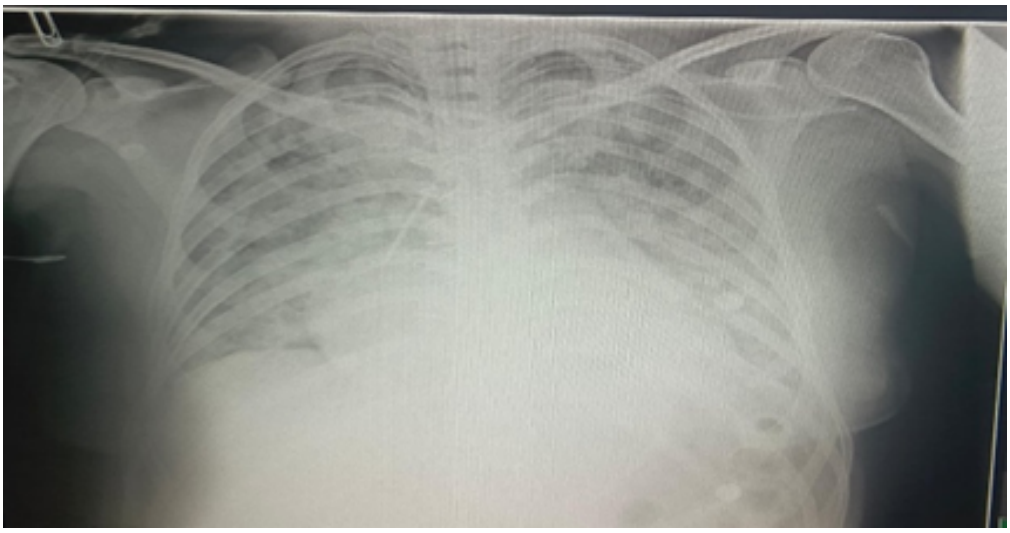
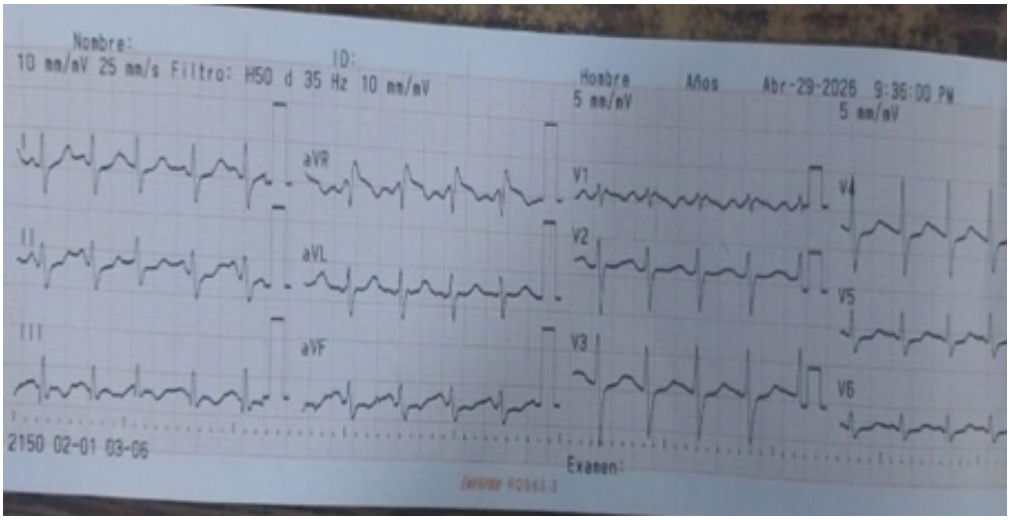


Figure 2. Electrocardiogram: sinus rhythm with HR 150 per minute, sinus tachycardia, incomplete right bundle branch block.



In the presence of one focal neurological event, a non-contrast CT scan of the head was performed, which showed a hypodense image in the left frontal region, compatible with an ischemic event and correlating with the clinical presentation of right facio-brachial deficit. Based on these findings, the diagnosis of ischemic stroke associated with mitral prosthetic dysfunction due to thrombus versus pannus was considered, leading to a transesophageal echocardiogram under sedation in the intensive care unit (Figure 5).

Transesophageal echocardiography (ETT) in the two-chamber view showed lack of mobility of the left hemidisc due to a heterogeneous annular mass that invaded the prosthesis, in addition to an intracavitary hypodense image with increased gradients, which confirmed obstructive prosthetic dysfunction. In another echocardiographic view, a hypodense image was observed on the atrial side of the prosthesis, compatible with thrombus, along with spontaneous contrast in the left atrium and left atrial appendage, a finding suggestive of low flow and high thromboembolic risk (Figures 6a and 6b).

During her hospital course, the patient showed a poor clinical response. She required invasive mechanical ventilation with sedation, inotropic support, and treatment for acute pulmonary edema; however, hemodynamic instability persisted, and she developed new respiratory deterioration upon any volume overload. Despite the measures instituted, the clinical course was torpid, and the patient ultimately did not survive.

Figure 3. ETT in a 4-chamber view in close-up shows a hyperechoic disc that is not clearly visualized and the presence of an acoustic shadow; no thrombi are observed.

Figure 4. Continuous Doppler on the mitral valve with velocity 2.5 ms, mean gradient 18 mmhg, THP 230 ms.

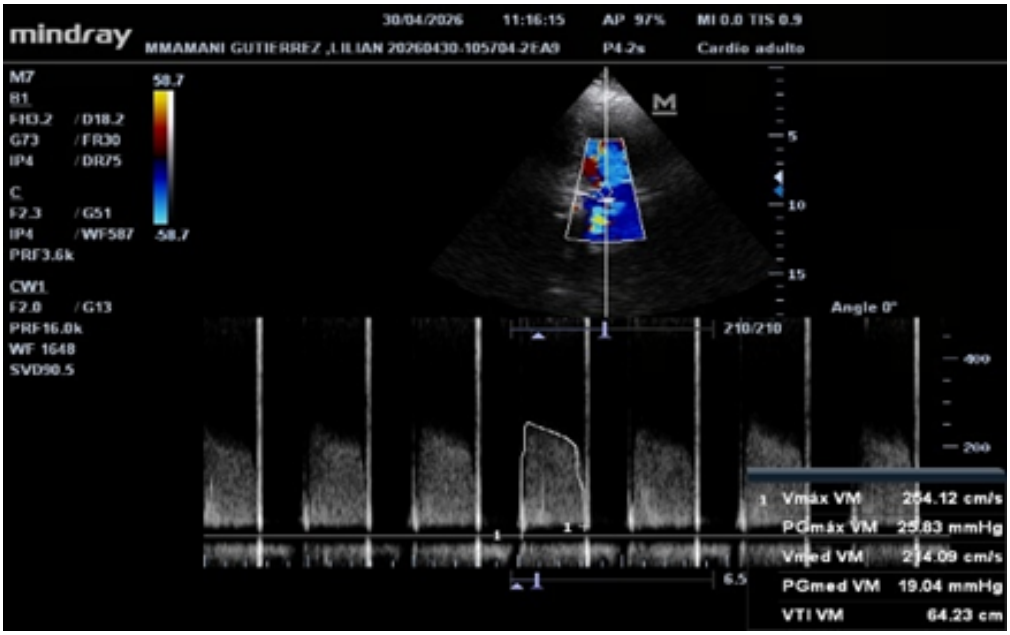


Figure 5. Skull tomography showing a hypodense image in the left frontal region, consistent with right faciobrachial paralysis on admission.

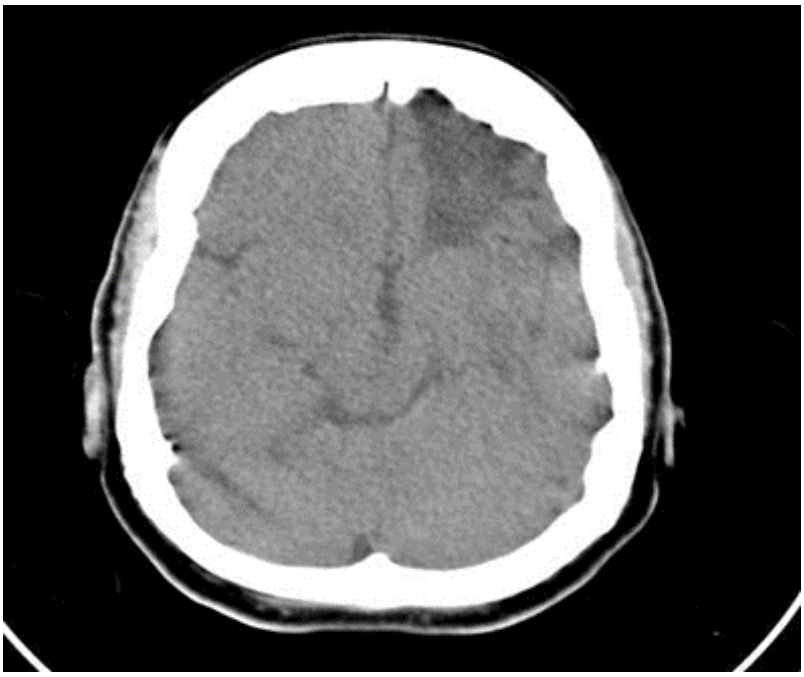


Figure 6a. TEE in the 2-chamber view at 51 degrees shows lack of mobility of the left hemidisc due to a heterogeneous annular mass invading the prosthesis, and a hypodense image within the prosthesis with aliasing and increased gradients.

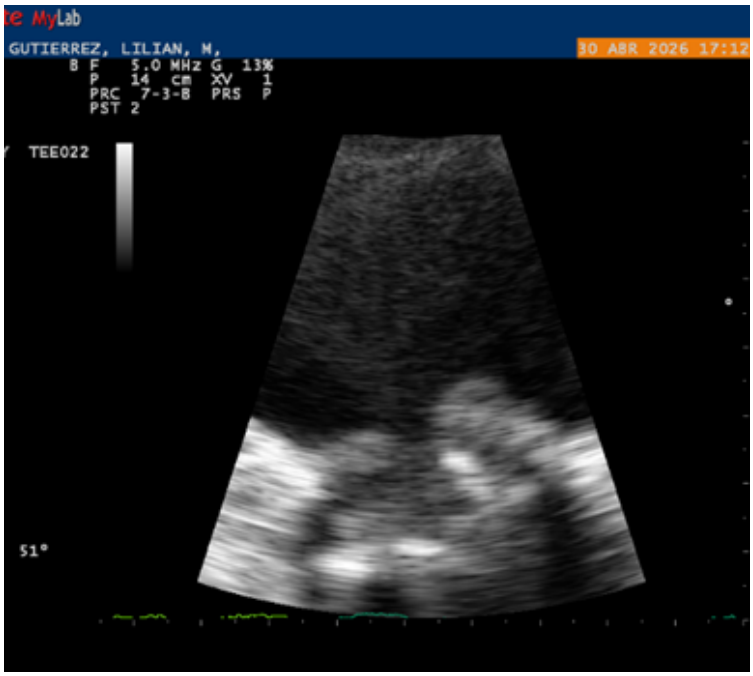
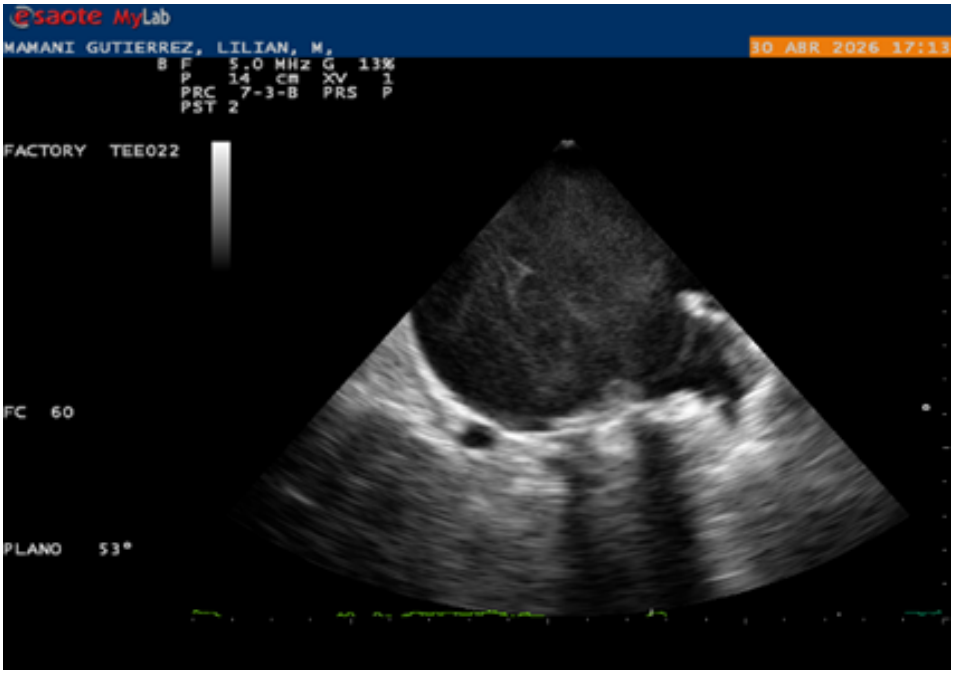


Figure 6b. TEE at 53 degrees, 2-chamber view with left atrial appendage: a hypodense image is observed on the atrial side, consistent with thrombus on the prosthesis, and spontaneous contrast in the left atrium (++) and smoke in the appendage with low velocities (-22 ms).



Discussion

Dysfunction of a mechanical mitral prosthesis represents a most feared complication after valve replacement, not only because of its ability to produce acute hemodynamic obstruction, but also because it can trigger decompensated heart failure, systemic embolism, and death in a very short period if not recognized in time (6,7). In our patient, the combination of severe dyspnea, orthopnea, acute pulmonary edema, decreased prosthetic click, hypoxemia, and subtherapeutic INR early pointed to obstructive prosthetic dysfunction, which coincides with the presentation form described in recent series and reports, where respiratory deterioration and signs of low output are usually the first clinical manifestations (6,8). The history of insufficient anticoagulation acquires special relevance because prosthetic thrombosis is a complication directly related to inadequate control of anticoagulant therapy and continues to be a frequent cause of obstruction in mechanical valves (6,9). One of the most important contributions of this case is that it forces us to insist on the distinction between thrombus and pannus, a difference that is not always simple in clinical practice and that can coexist in the same patient. Contemporary studies have shown that both processes can occur simultaneously in a non-negligible proportion of cases, which complicates diagnosis and also therapeutic decision-making (10,11).

In the case we present, transthoracic echocardiography showed an increase in gradients and restriction of prosthetic motion, while transesophageal echocardiography allowed visualization of a heterogeneous annular mass associated with an image compatible with thrombus, findings that strengthened the suspicion of mixed obstruction due to pannus and thrombus (12,13). This diagnostic sequence is consistent with the literature, which recognizes echocardiography as the indispensable initial tool, although often insufficient by itself to define the exact etiology of the obstruction (12,14). Compared with other clinical reports, our case shares the diagnostic difficulty described by several authors when the mitral prosthesis is partially immobilized and the clinical picture is already severe at the time of consultation. Barrio Alonso et al. reported a case of mitral mechanical dysfunction in which multislice computed tomography helped tilt the diagnosis toward pannus, emphasizing that the value of advanced imaging lies not only in detecting obstruction but in defining its anatomical origin to change therapeutic management (10).

Similarly, Faraj et al. described a mitral prosthetic obstruction in which the differentiation between thrombus and pannus was central to deciding treatment, highlighting that thrombosis usually has a window of reversibility if identified early, whereas pannus, being proliferative fibrous tissue, hardly responds to isolated medical management (11). Our case is one that confirms this premise: the diagnostic delay and the coexistence of both lesions favored a torpid clinical course and limited the possibilities of functional rescue.

The neurological presentation of this patient adds a particular element of severity. The coexistence of ischemic stroke and prosthetic dysfunction suggests embolism of cardiac origin in the context of underlying valve thrombosis, a pattern also described in other reports of mechanical valves with suboptimal anticoagulation (15,16). In this scenario, the therapeutic dilemma is real: intensifying anticoagulation or thrombolysis may favor recanalization but increases the risk of hemorrhagic transformation of the cerebral event, especially when acute ischemic injury is already present (6,15). For precisely this reason, contemporary guidelines emphasize that the approach must be individualized according to hemodynamic stability, valve location, thrombus size, presence of systemic embolism, and bleeding risk, always within a multidisciplinary team (7,17,18). From a practical standpoint, this case also invites reflection on the long-term monitoring of patients with mechanical prostheses. The literature agrees that early diagnosis is likely the most important factor in changing the outcome, since when obstruction is identified before severe respiratory decompensation or an embolic event, the chances of success increase significantly (6,7,12). In contrast, when the patient arrives with refractory acute pulmonary edema, hypoxemia, and neurological compromise, as occurred here, the therapeutic window narrows considerably and mortality remains high despite correct treatment (10,11,19).

In relation to treatment, prosthetic thrombus obstruction may respond to intensive anticoagulation or thrombolysis in selected situations, but pannus does not usually subside with pharmacological measures and often requires surgical replacement or definitive structural intervention (6,7,20). In this case, the coexistence of pannus and thrombus, together with hemodynamic instability and the concomitant neurological event, made decision-making particularly complex and reinforced the need for an early surgical strategy if the patient's conditions allowed it (10,11,20). The impossibility of reversing respiratory and neurological deterioration in a timely manner illustrates one of the most difficult scenarios in clinical cardiology: the moment when prosthetic anatomy, anticoagulation, and the patient's physiological reserve combine to leave very little therapeutic margin.

As a limitation of this report, it should be noted that definitive anatomical confirmation of the exact proportion between pannus and thrombus can be difficult when surgical correlation or high-resolution multislice tomography are not available, and this may lead to some diagnostic uncertainty despite the significant contribution of echocardiography (10,12,14). Likewise, the severe clinical course and fatal outcome limited the possibility of documenting the response to a definitive therapeutic strategy, which is an inherent limitation of several case reports in critical scenarios. However, precisely that limitation reinforces the value of the case as a clinical warning: a mechanical prosthesis that changes in auscultation, shows increasing gradients, and is associated with hypoxemia or neurological deficit should be considered a cardiological emergency until proven otherwise.

In terms of recommendation, this report supports close monitoring of anticoagulation, patient education about adherence, early echocardiographic evaluation upon any clinical change, and rapid activation of a multidisciplinary team to define surgery or advanced intervention when significant obstruction exists (6,7,12,17). Together, this case shows that mitral prosthetic dysfunction can evolve with considerable aggressiveness and that, even with intensive support, the outcome can be fatal if the condition is diagnosed late or if neurological and respiratory damage is already irreversible. Therefore, early diagnosis, etiological differentiation, and timely indication of surgical or interventional treatment remain the pillars for modifying the prognosis in this entity (6,7,10,11,20).

REFERENCES

1. Roudaut R, Serri K, Lafitte S. Thrombosis of prosthetic heart valves: diagnosis and therapeutic considerations. Heart. 2007;93(1):137-142. doi:10.1136/hrt.2005.071183. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC1861363/.
2. Dangas GD, Weitz JI, Giustino G, Makkar R, Mehran R. Prosthetic heart valve thrombosis. J Am Coll Cardiol. 2016;68(24):2670-2689. doi:10.1016/j.jacc.2016.09.958. Available from: https://pubmed.ncbi.nlm.nih.gov/27978952/.
3. Vahanian A, Beyersdorf F, Praz F, Milojevic M, Baldus S, Bauersachs J, Capodanno D, Conradi L, De Bonis M, De Paulis R, Delgado V, Freemantle N, Gilard M, Haugaa KH, Jeppsson A, Jüni P, Pierard L, Prendergast BD, Sádaba JR, Tribouilloy C, Wojakowski W; ESC/EACTS Scientific Document Group. 2021 ESC/EACTS Guidelines for the management of valvular heart disease. Eur Heart J. 2022 Feb 12;43(7):561-632. doi: 10.1093/eurheartj/ehab395. Erratum in: Eur Heart J. 2022 Jun 1;43(21):2022. doi: 10.1093/eurheartj/ehac051.
4. Zoghbi WA, Jone PN, Chamsi-Pasha MA, et al. Guidelines for the evaluation of prosthetic valve function with cardiovascular imaging: a report from the American Society of Echocardiography developed in collaboration with the Society for Cardiovascular Magnetic Resonance and the Society of Cardiovascular Computed Tomography. J Am Soc Echocardiogr. 2024;37(1):2-63. doi:10.1016/j.echo.2023.10.004. Available from:
5. https://pubmed.ncbi.nlm.nih.gov/38182282/
6. Ashraf H, Freeman WK. Echocardiographic assessment of prosthetic valves. Rev Cardiovasc Med. 2022;23(10):343. doi:10.31083/j.rcm2310343. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC11267339/.
7. Tiede DJ, Nishimura RA, Gastineau DA, Mullany CJ, Orszulak TA, Schaff HV. Modern management of prosthetic valve anticoagulation. Mayo Clin Proc. 1998 Jul;73(7):665-80. doi: 10.1016/S0025-6196(11)64893-3.
8. Barbetseas J, Nagueh SF, Pitsavos C, Toutouzas PK, Quiñones MA, Zoghbi WA. Differentiating thrombus from pannus formation in obstructed mechanical prosthetic valves: an evaluation of clinical, transthoracic and transesophageal echocardiographic parameters. J Am Coll Cardiol. 1998 Nov;32(5):1410-7. doi: 10.1016/s0735-1097(98)00385-4.
9. Montorsi P, De Bernardi F, Muratori M, Cavoretto D, Pepi M. Role of cine-fluoroscopy, transthoracic, and transesophageal echocardiography in patients with suspected prosthetic heart valve thrombosis. Am J Cardiol. 2000 Jan 1;85(1):58-64. doi: 10.1016/s0002-9149(99)00607-4.
10. Habib G, Cornen A, Mesana T, Monties JR, Djiane P, Luccioni R. Diagnosis of prosthetic heart valve thrombosis. The respective values of transthoracic and transoesophageal Doppler echocardiography. Eur Heart J. 1993 Apr;14(4):447-55. doi: 10.1093/eurheartj/14.4.447.
11. Barrio Alonso AI, López Suarez RY, Álvarez Cabo R, Ríos Gómez E. Dysfunctioning mechanical mitral valve prosthesis: when thrombus over pannus hinders diagnosis. Radiol (Engl Ed). 2024;66(1):90-93. doi:10.1016/j.rxeng.2022.11.008. Available from:
12. https://pubmed.ncbi.nlm.nih.gov/38365358/
13. Faraj R, Diallo TH, Dib H, et al. Mechanical prosthetic mitral valve obstruction: pannus or thrombus? A case report. Radiol Case Rep. 2023;18(8):2685-2688. doi:10.1016/j.radcr.2023.05.016. Available from:
14. https://pubmed.ncbi.nlm.nih.gov/37293521/
15. Deviri E, Sareli P, Wisenbaugh T, Cronje SL. Obstruction of mechanical heart valve prostheses: clinical aspects and surgical management. J Am Coll Cardiol. 1991 Mar 1;17(3):646-50. doi: 10.1016/s0735-1097(10)80178-0.
16. Gohlke-Bärwolf C. Anticoagulation in valvar heart disease: new aspects and management during non-cardiac surgery. Heart. 2000;84(5):567-572. doi:10.1136/heart.84.5.567.
17. Fernandes V, Olmos L, Nagueh SF, Quiñones MA, Zoghbi WA. Peak early diastolic velocity rather than pressure half-time is the best index of mechanical prosthetic mitral valve function. Am J Cardiol. 2002 Mar 15;89(6):704-10. doi: 10.1016/s0002-9149(01)02343-8.
18. Tong AT, Roudaut R, Ozkan M, Sagie A, Shahid MS, Pontes Júnior SC, Carreras F, Girard SE, Arnaout S, Stainback RF, Thadhani R, Zoghbi WA; Prosthetic Valve Thrombolysis-Role of Transesophageal Echocardiography (PRO-TEE) Registry Investigators. Transesophageal echocardiography improves risk assessment of thrombolysis of prosthetic valve thrombosis: results of the international PRO-TEE registry. J Am Coll Cardiol. 2004 Jan 7;43(1):77-84. doi: 10.1016/j.jacc.2003.08.028.
19. Guéret P, Vignon P, Fournier P, et al. Transesophageal echocardiography for the diagnosis and management of non-obstructive thrombosis of mechanical mitral valve prosthesis. Circulation. 1995;91(1):103-110. doi:10.1161/01.CIR.91.1.103.
20. Laffort P, Roudaut R, Roques X, et al. Early and long-term (one-year) effects of the association of aspirin and oral anticoagulant on thrombi and morbidity after replacement of the mitral valve with the St. Jude medical prosthesis. J Am Coll Cardiol. 2000;35(3):739-746. doi:10.1016/S0735-1097(99)00601-7.
21. Bonow RO, Carabello BA, Chatterjee K, et al. ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: executive summary. Circulation. 2006;114(5):450-527. doi:10.1161/CIRCULATIONAHA.106.176857.
22. Roudaut R, Lafitte S, Roudaut MF, Courtault C, Perron JM, Jaïs C, Pillois X, Coste P, DeMaria A. Fibrinolysis of mechanical prosthetic valve thrombosis: a single-center study of 127 cases. J Am Coll Cardiol. 2003 Feb 19;41(4):653-8. doi: 10.1016/s0735-1097(02)02872-3.
23. Roudaut R, Roques X, Lafitte S, Choukroun E, Laborde N, Madona F, Deville C, Baudet E. Surgery for prosthetic valve obstruction. A single center study of 136 patients. Eur J Cardiothorac Surg. 2003 Dec;24(6):868-72. doi: 10.1016/s1010-7940(03)00568-

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CONFLICT OF INTEREST

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