Received: 23 November 2023 | Revised: 22 April 2024 | Accepted: 28 April 2024

DOI: 10.1002/ccr3.8899

CASE REPORT

Clinical Case Reports Open Access WILEY

Right ventricular dysfunction after pericardiectomy for tuberculous constrictive pericarditis: A case report

Natânia Ferreira Duarte | **Stella de Aguiar Trigueirinho Ferreira** | **Daniel Abdalla Added Filho** | **Carlos Henrique Lopes Vidal** | **Roger Sales Lima** | **Ana Vitória Vitoreti Martins** | **Rafael Oliveira Castro** | **Arthur Cicupira Rodrigues de Assis** | **Paulo Rogério Soares** | **Thiago Luis Scudeler**

Emergency Department of Instituto do Coração (InCor), Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil

Correspondence
Thiago Luis Scudeler, Instituto do Coração (InCor), Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, Avenida Doutor Eneas de Carvalho Aguiar 44, AB, Sala 114, Cerqueira César, São Paulo 05403-000, Brazil.
Email: thiago.scudeler@fm.usp.br

Key Clinical Message

This case report provides a peculiar case of tuberculous constrictive pericarditis (TCP) who presented with right ventricular dysfunction after pericardiectomy. Right ventricular dysfunction is one of the main postoperative complications after pericardiectomy. Rapid and accurate identification of right ventricular dysfunction confirmed by transthoracic echocardiography (TTE), associated with the rapid initiation of diuretics and inotropic therapy is necessary for the patient's complete recovery.

Abstract

TCP is a condition characterized by chronic inflammation and fibrosis of the pericardium. Pericardiectomy is the standard treatment for patients with constrictive pericarditis and persistent symptoms. One possible surgical complication is right ventricle (RV) failure. We report a case of a 44-year-old man who developed RV failure after pericardiectomy for TCP. A 41-year-old man with no medical history was referred to our hospital due to progressive dyspnea associated with edema of the lower limbs and significant weight loss (30 kg) over the past 5 months. TTE revealed significant pericardial thickening and mild pericardial effusion with normal RV function. Chest X-ray showed moderate bilateral pleural effusion. The patient underwent pericardiectomy and bilateral pleural drainage. Histopathological examination showed tuberculosis granulomas with caseous necrosis, and antituberculosis medication was initiated. Postoperative TTEs showed normal RV function and mild pericardial thickening. The patient was discharged home after successful postoperative recovery. Three weeks later, the patient was admitted to the emergency department with dyspnea and hypoxemia. TTE revealed RV systolic dysfunction. Chest CT showed a recurrence of moderate pleural effusion, this time loculated, with restrictive atelectasis of the adjacent lung parenchyma. Diuretics and inotropic

therapy were initiated, and the patient underwent lung decortication after confirmation of tuberculous empyema. The patient experienced significant clinical improvement. TTE before discharge showed a decreased RV chamber size with improved RV systolic function. The patient was discharged in a stable condition 30 days after admission with a low dose of oral furosemide. Four months after discharge, he remained asymptomatic with good functional status. Pericardiectomy for TCP may carry the risk of developing RV dysfunction. Furthermore, TCP itself may be associated with other complications, such as empyema. We emphasize the importance of conducting a thorough clinical evaluation for patients with TCP, particularly those undergoing pericardiectomy, to mitigate potential adverse outcomes.

1 | INTRODUCTION

Tuberculosis is an infectious disease characterized by the occurrence of granulomatous inflammation with necrosis, predominantly located in the lung tissue.[1] Pericardial involvement is a condition that affects 1%–2% of patients diagnosed with tuberculosis, but may account for up to 30% of pericarditis cases in endemic countries,[2] and has a high mortality rate (17%–40% in 6 months).[3]

Constrictive pericarditis is a condition characterized by inflammation, fibrosis and loss of elasticity of the pericardium, resulting in restricted ventricular filling and diastolic dysfunction. This leads to increased ventricular interdependence and dissociation of intracardiac and intrathoracic pressures during ventilation. Clinically, insidious signs and symptoms of systemic venous congestion predominantly appear. It is a relatively rare condition with varied causes.[4,5] Progression to constrictive pericarditis is reported in up to 30% of cases of tuberculous pericarditis.[6] Pericardiectomy is the standard treatment for patients with symptomatic constrictive pericarditis.[7] However, an intrinsic risk to surgery is RV dysfunction, a consequence of myocardial atrophy,[8] and the abrupt increase in venous return leading to volume overload, mainly in the RV. Echocardiography is an effective method to monitor these post-pericardiectomy hemodynamic changes.

We present a case involving a 41-year-old male patient who developed RV dysfunction 2 weeks after undergoing pericardiectomy for TCP.

2 | CASE HISTORY/EXAMINATION

A 41-year-old man with no medical history was referred to our hospital due to progressive dyspnea associated with edema of the lower limbs, pleural effusion, and significant weight loss (30 kg) over the past 5 months. Physical examination revealed hypophonetic heart sounds, jugular stasis at 45°, hepatojugular reflux, decreased breath sounds bilaterally with signs of respiratory effort, and moderate pitting edema on both pretibial areas.

3 | DIAGNOSIS AND TREATMENT

TTE showed pericardial thickening with adhesion, dilated inferior vena cava (28 mm in diameter with respiratory variation less than 50%), preserved left and right ventricular function, end-diastolic volume, and end-systolic volume (Figure 1A,B; Table 1; Videos S1,S2). Furthermore, TTE showed the presence of (1) ventricular septal shift, (2) medial mitral shift and velocity of 11 cm/s, and (3) the hepatic vein expiratory

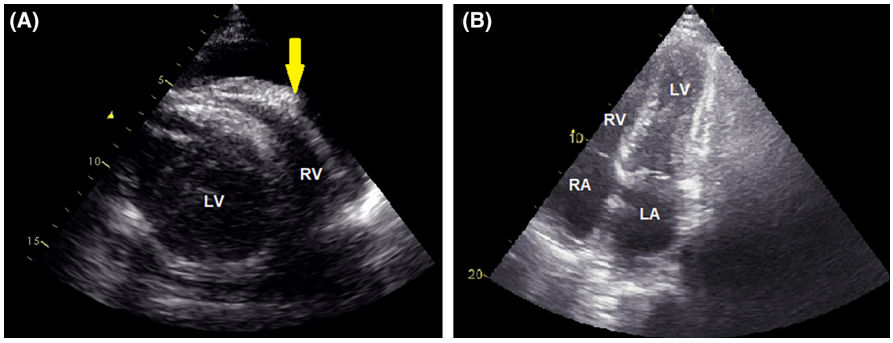

FIGURE 1 Preoperative TTE showed normal RV systolic function and small sized RV. (A) Parasternal short axis view; (B) Apical four-chamber view. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle. yellow arrow: significant pericardial thickening.

diastolic reversal ratio of 0.85, confirming the diagnosis of constrictive pericarditis. The preoperative chest X-ray showed moderate bilateral pleural effusion (Figure 2A). The patient underwent thoracentesis, and the findings, which were nonspecific, did not allow the identification of the etiology of the pleural effusion.

Considering the echocardiographic findings of constrictive pericarditis and the presence of symptoms, pericardiectomy via median sternotomy with cardiopulmonary bypass and bilateral pleural drainage was performed. Approximately 1500 mL of fluid was removed from the left side and 200 mL from the right side. The procedure involved the complete removal of the anterior pericardium between the two phrenic nerves, the removal of the diaphragmatic pericardium, and part of the posterior pericardium. The anatomopathological examination revealed chronic granulomatous pleuritis and chronic granulomatous pericarditis with extensive areas of caseous necrosis. Antituberculosis therapy was initiated (rifampicin 150 mg, isoniazid 75 mg, pyrazinamide 400 mg, and ethambutol 275 mg of five tablets daily for 2 months, followed by 4 months of rifampicin 150 mg and isoniazid 75 mg of five tablets daily).

The postoperative chest X-ray (fourth postoperative day) is illustrated in Figure 2B. Postoperative TTEs demonstrated preserved biventricular function and mild pericardial thickening (Figure 3A,B; Table 1; Videos S3,S4). The patient was discharged from the hospital 8 days after the pericardiectomy.

Three weeks after the surgery, he was admitted to the emergency department with dyspnea and hypoxemia. Physical examination again revealed jugular stasis at 45° and hepatojugular reflux, decreased breath sounds bilaterally, and moderate pitting edema on both pretibial areas. He presented with a heart rate of 93 beats per minute, blood pressure of 119/85 mm Hg, and body temperature of 36.3°C. The respiratory rate was 31 breaths per minute with 89% peripheral oxygen saturation without supplementary oxygen. The electrocardiogram (ECG) on admission showed sinus rhythm with nonspecific ST-T repolarization abnormalities (Figure 4).

A new TTE showed preserved LVEF, severe RV dysfunction, mild tricuspid and mitral regurgitation,

TABLE 1 Echocardiographic variables of right ventricular systolic function before and after pericardiectomy.

Echocardiographic parameters	TTE1	TTE2	TTE3	TTE4
TAPSE (cm)	22	NE	10.4	17
FAC (%)	35	NE	17	32.4
Tricuspid ring S´wave (cm/s)	11	NE	6	10.5

Abbreviations: FAC, fractional area change; NA, not evaluated; TAPSE, tricuspid annular plane systolic excursion; TTE, transthoracic echocardiography; TTE 1, preoperative TTE; TTE 2, 4 days postoperative TTE; TTE3, TTE on admission of the second hospitalization (21 days postoperative); TTE 4, TTE of the second hospitalization (44 days postoperative).

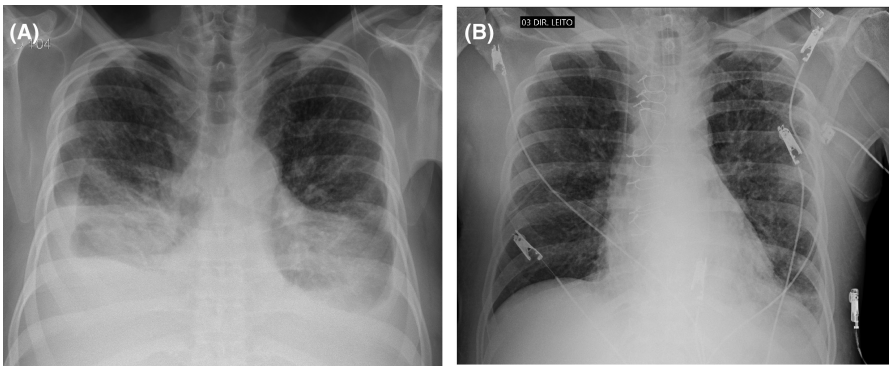

FIGURE 2 (A) Preoperative chest X-ray posteroanterior view showing moderate bilateral pleural effusion; (B) Postoperative chest X-ray posteroanterior view showing absence of pleural effusion.

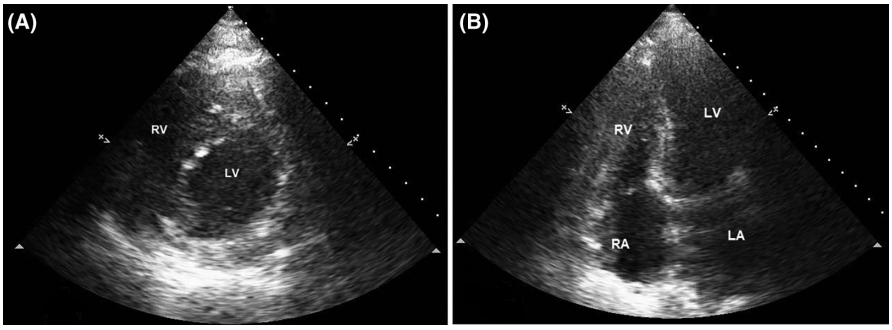

FIGURE 3 Postoperative TTE 2 showed marked dilatation and low ejection fraction of the RV. (A) Parasternal short axis view; (B) Apical four-chamber view. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

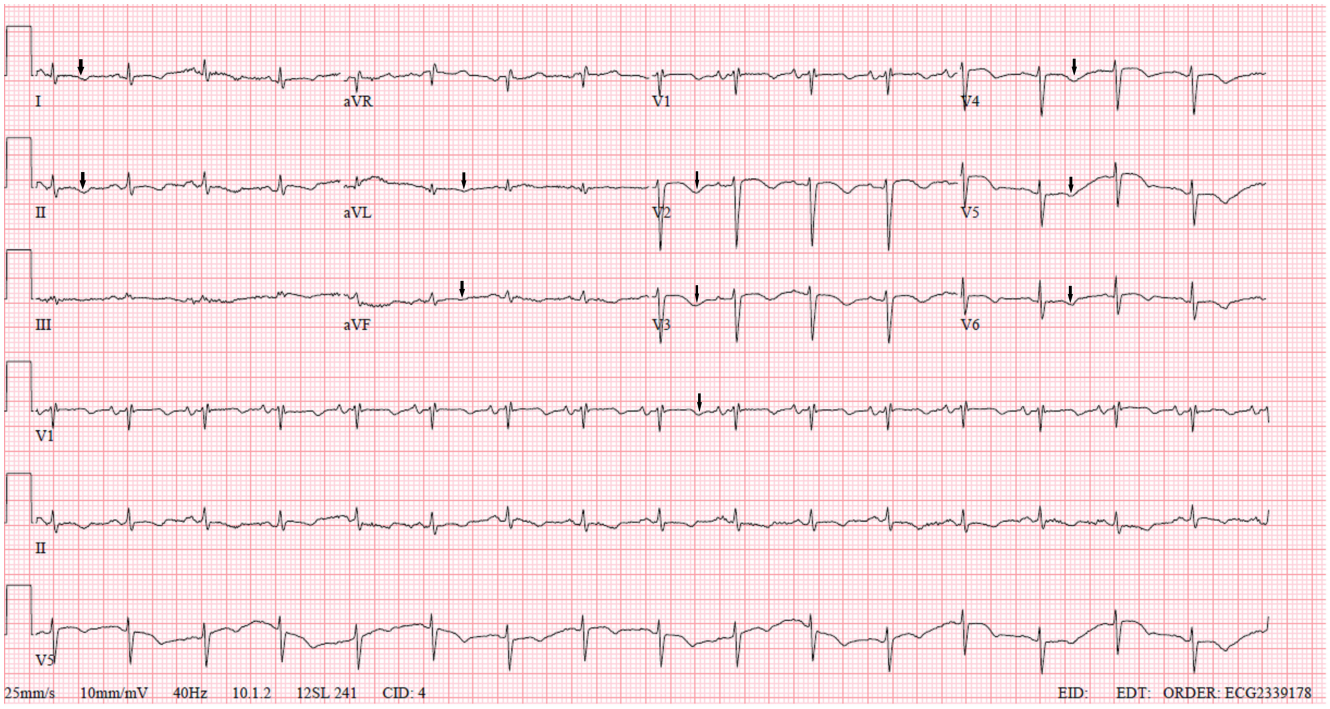

FIGURE 4 ECG on admission (2 weeks after pericardiectomy) showing sinus rhythm with nonspecific ST-T repolarization abnormalities (black arrows).

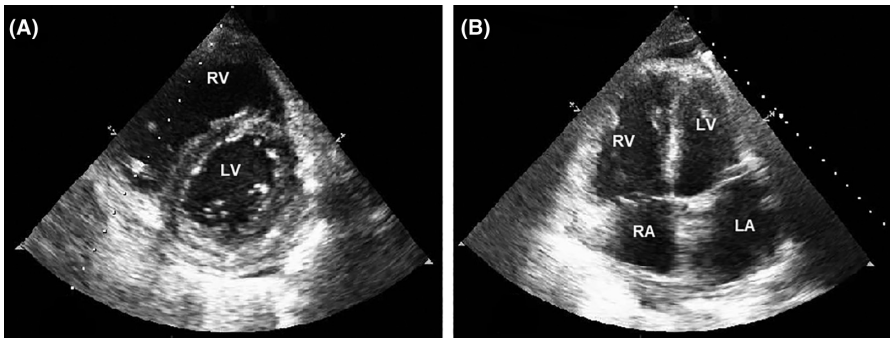

FIGURE 5 TTE of admission of second hospitalization (21 days postoperative) revealed a reduction in RV size with improved RV systolic function. (A) Parasternal short axis view; (B) Apical four-chamber view. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

and mild pericardial thickening (Figure 5A,B, Table 1, Videos S5,S6). Chest CT revealed a recurrence of moderate pleural effusion, now loculated, with restrictive atelectasis of the adjacent lung parenchyma (Figure 6). He developed respiratory failure and hemodynamic instability, requiring intubation. Dobutamine at 7.5 mcg/kg/min and diuretics were initiated. Corticosteroids and antibiotics were prescribed, and the loculated pleural effusion was drained. Dobutamine was gradually discontinued by day 15 of admission. TTE performed on day 44 revealed a decreased RV chamber size with improved RV systolic function (Figure 7A,B, Table 1, Videos S7,S8). He was discharged in a stable condition 30 days after admission with a low dose of oral furosemide. Four months after discharge, he remained asymptomatic with good functional status and normal exercise capacity.

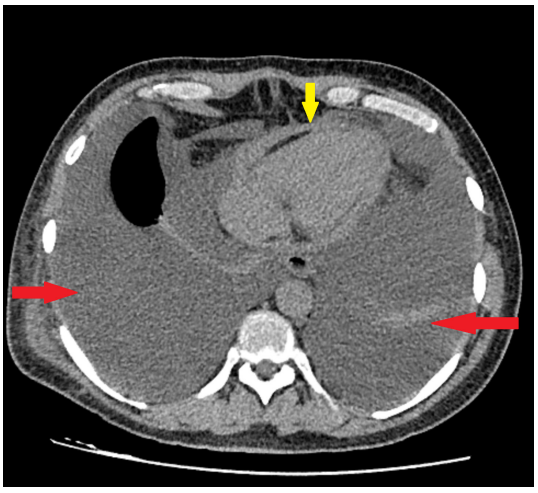

FIGURE 6 Chest CT showing moderate bilateral pleural effusion, and significant pericardial thickening. Yellow arrow: significant pericardial thickening; red arrow: bilateral pleural effusion.

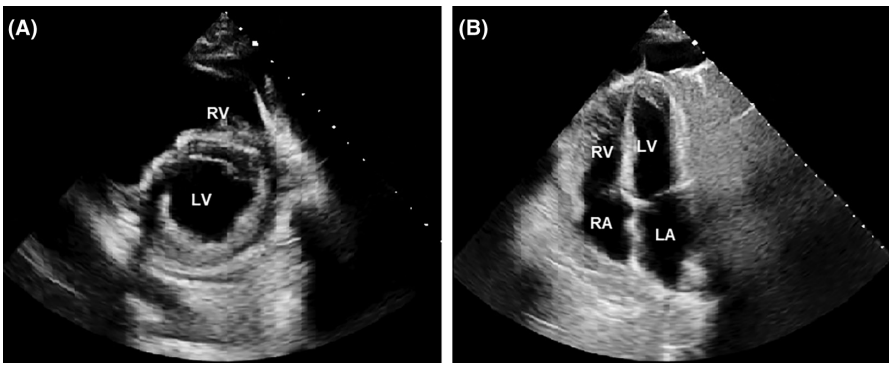

FIGURE 7 TTE of the second hospitalization (44 days postoperative) showing RV less dilated than seen previously. (A) Parasternal short axis view; (B) Apical four-chamber view. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

4 | DISCUSSION

Tuberculosis is the most common cause of constrictive pericarditis, accounting for 38%–83% of cases in endemic countries.[9] Transient constrictive pericarditis occurs in 10% of tuberculosis-related pericarditis, while progression to chronic constrictive pericarditis occurs in 20%–50% of patients despite antituberculosis treatment.[9] Furthermore, chronic constrictive pericarditis can persist for several years, leading to heart failure.[9] Although the right and left ventricular diastolic pressures are equalized in this disease, symptoms of RV dysfunction are dominant.

TCP typically presents with dense fibrosis that progressively impairs diastolic filling of the heart. Therefore, pericardiectomy in combination with antituberculosis drugs is the recommended treatment for TCP and persistent symptoms.

The main advantages of pericardiectomy are (1) improvement of symptoms, (2) a better chance of a complete cure than with medication, and (3) preventing constriction from worsening and damaging the heart muscle. On the other hand, bleeding complications, atrial arrhythmias, kidney failure, RV dysfunction, and death are potential postoperative complications. Pericardiectomy can lead to increased left ventricular end-diastolic volume and improved Frank-Starling reserve, providing an increase in exercise stroke volume, cardiac output, and aerobic capacity.[10] Bozbuga et al. showed an improvement in the functional status in 88% of patients undergoing pericardiectomy after a 1-year follow-up.[11] A meta-analysis that investigated the effects of pericardiectomy in patients with TCP showed that patients had a significant improvement in the New York Heart Association (NYHA) grade 1 year following pericardiectomy (HR 8.04, 95% CI [5.20–12.45], $I^2 = 0\%$).[12]

The mortality rate in patients with chronic constrictive pericarditis undergoing pericardiectomy varies between 4% and 8%.[13,14] However, it is important to note that the mortality associated with pericardiectomy has markedly decreased over the past decades.[15]

RV dysfunction is one of the main postoperative complications after pericardiectomy.[10,16] However, this problem commonly occurs during the early postoperative phase. Azzu A et al. showed that approximately 80% of patients have evidence of RV dysfunction early post-pericardiectomy.[16] The main pathophysiological mechanisms possibly involved in RV dysfunction after pericardiectomy are myocardial atrophy secondary to prolonged constriction as well as a rapid increase in venous return to the right heart after pericardial decompression.[17] This external constriction leads, over time, to a reduction in myocardial mass due to low diastolic volumes. For this reason, when the ventricle is exposed to a greater volume postoperatively, ventricular dilation occurs, and cardiac output decreases. Thus, it is important to choose the ideal period for pericardiectomy, given that if the surgical option is postponed for a long time, the greater the risk of irreversible ventricular remodeling and the greater the probability of low output syndrome.[18] Another post-pericardiectomy complication is tricuspid insufficiency, which occurs due to sudden RV dilation.

Although cardiopulmonary bypass is associated with higher rates of postoperative complications, in our case, it was necessary to facilitate a complete pericardiectomy. Studies have demonstrated the benefit of complete pericardiectomy in terms of long-term survival and symptom improvement.[19–21]

The prognosis after surgery depends on age, the degree of myocardial involvement, the severity of liver dysfunction caused by the constriction, the preoperative NYHA functional class, the presence of extensive pericardial calcifications, which condition the incomplete removal of the pericardium, or of pericarditis caused by exposure to radiation.[22]

Over the next 4 months, he had no recurrence of symptoms while using antituberculosis therapy. In post-pericardiectomy follow-up, it is necessary to identify patients who are at greater risk of complications. The development of post-pericardiectomy complications is not uncommon in patients with constrictive pericarditis. RV dysfunction warrants prompt recognition and aggressive treatment for heart failure.

We report a peculiar case of a patient with TCP who presented RV dysfunction 2 weeks after pericardiectomy. RV dysfunction occurred in the late post-pericardiectomy

phase, although it is more common early post-pericardiectomy. Rapid and accurate identification of RV dysfunction confirmed by TTE, associated with the rapid initiation of diuretics and inotropic therapy, in addition to drainage of the pleural effusion complicated by empyema and administration of appropriate antibiotics, allowed the prompt and complete recovery of the patient.

5 | CONCLUSIONS

Tuberculosis is a disease associated with the development of empyema and constrictive pericarditis. Acute RV dysfunction is a potentially fatal event that necessitates the administration of diuretics and inotropic support, along with hemodynamic assistance. The presence of empyema requires drainage for both therapeutic and diagnostic purposes. Recognizing and managing acute RV dysfunction are crucial steps in preventing life-threatening outcomes, and therapeutic approaches aim to address the hemodynamic challenges associated with this condition.

AUTHOR CONTRIBUTIONS

Natânia Ferreira Duarte: Conceptualization; formal analysis; investigation; methodology; resources; visualization; writing – original draft; writing – review and editing. **Stella de Aguiar Trigueirinho Ferreira:** Conceptualization; data curation; formal analysis; investigation; resources; supervision; validation; visualization; writing – original draft; writing – review and editing. **Daniel Abdalla Added Filho:** Conceptualization; formal analysis; investigation; methodology. **Carlos Henrique Lopes Vidal:** Conceptualization; formal analysis; investigation; methodology. **Roger Sales Lima:** Conceptualization; formal analysis; investigation; methodology; visualization. **Ana Vitória Vitoreti Martins:** Conceptualization; formal analysis; investigation; methodology; validation; visualization. **Rafael Oliveira Castro:** Conceptualization; data curation; formal analysis; investigation; methodology; validation; visualization. **Arthur Cicupira Rodrigues de Assis:** Conceptualization; data curation; formal analysis; investigation; methodology; validation; visualization. **Paulo Rogério Soares:** Conceptualization; data curation; formal analysis; investigation; methodology; supervision; validation; visualization. **Thiago Luis Scudeler:** Conceptualization; data curation; formal analysis; investigation; methodology; resources; supervision; validation; visualization; writing – original draft; writing – review and editing.

FUNDING INFORMATION

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

CONFLICT OF INTEREST STATEMENT

The authors declare that there is no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal'spatient consent policy.

ORCID

Thiago Luis Scudeler https://orcid.org/0000-0001-5685-3633

REFERENCES

1. Dheda K, Barry CE, Maartens G. Tuberculosis. *Lancet*. 2016;387:1211-1226.
2. Klein AL, Cremer PC. Ephemeral effusive constrictive pathophysiology. *JACC Cardiovasc Imaging*. 2018;11(4):542-545.
3. Isiguzo G, Du Bruyn E, Howlett P, Ntsekhe M. Diagnosis and Management of Tuberculous Pericarditis: what is new? *Curr Cardiol Rep*. 2020;22(1):2.
4. Maisch B. Management of pericarditis and pericardial effusion, constrictive and effusive-constrictive pericarditis. *Herz*. 2018;43(7):663-678.
5. Welch TD, Oh JK. Constrictive pericarditis. *Cardiol Clin*. 2017;35:539-549.
6. Chang SA. Tuberculous and infectious pericarditis. *Cardiol Clin*. 2017;35:615-622.
7. Karima T, Nesrine BZ, Hatem L, Skander BO, Raouf D, Selim C. Constrictive pericarditis: 21 years' experience and review of literature. *Pan Afr Med J*. 2021;38:141.
8. Beckmann E, Ismail I, Cebotari S, et al. Right-sided heart failure and extracorporeal life support in patients undergoing pericardiectomy for constrictive pericarditis: a risk factor analysis for adverse outcome. *Thorac Cardiovasc Surg*. 2017;65:662-670.
9. López-López JP, Posada-Martínez EL, Saldarriaga C, et al. Tuberculosis and the heart. *J Am Heart Assoc*. 2021;10(7):e019435.
10. Borlaug BA, Reddy YNV. The role of the pericardium in heart failure: implications for pathophysiology and treatment. *J Am Coll Cardiol HF*. 2019;7(7):574-585.
11. Bozbuga N, Erentug V, Eren E, et al. Pericardiectomy for chronic constrictive tuberculous pericarditis: risks and predictors of survival. *Tex Heart Inst J*. 2003;30(3):180-185.
12. Yadav S, Shah S, Iqbal Z, et al. Pericardiectomy for constrictive tuberculous pericarditis: a systematic review and meta-analysis on the etiology, Patients' characteristics, and the outcomes. *Cureus*. 2021;13(9):e18252.
13. Park YH. Novel imaging parameters for right ventricular dysfunction after pericardiectomy in constrictive pericarditis. *J Cardiovasc Imaging*. 2021;29(4):373-374.
14. Choi MS, Jeong DS, Oh JK, Chang SA, Park SJ, Chung S. Long-term results of radical pericardiectomy for constrictive

pericarditis in the Korean population. *J Cardiothorac Surg*. 2019;14(1):32.
15. Gillaspie EA, Stulak JM, Daly RC, et al. A 20-year experience with isolated pericardiectomy: analysis of indications and outcomes. *J Thorac Cardiovasc Surg*. 2016;152(2):448-458.
16. Yu HT, Ha JW, Lee S, et al. Transient right ventricular dysfunction after pericardiectomy in patients with constrictive pericarditis. *Korean Circ J*. 2011;41(5):283-286.
17. Azzu A, Morosin M, Antonopoulos AS, Capoccia M, Rosendahl U, Mohiaddin R. Cardiac decompression by pericardiectomy for constrictive pericarditis: multimodality imaging to identify patients at risk for prolonged inotropic support. *J Cardiovasc Imaging*. 2021;29(4):361-372.
18. Raizada V, Sato K, Alashi A, et al. Depressed right ventricular systolic function in heart failure due to constrictive pericarditis. *ESC Heart Fail*. 2021;8(4):3119-3129.
19. Avgerinos D, Rabitnokov Y, Worku B, Neragi-Miandoab S, Girardi L. Fifteen-year experience and outcomes of pericardiectomy for constrictive pericarditis. *J Card Surg*. 2014;29(4):434-438.
20. Gatti G, Fiore A, Ternacle J, et al. Pericardiectomy for constrictive pericarditis: a risk factor analysis for early and late failure. *Heart Vessel*. 2020;35(1):92-103.
21. Szabó G, Schmack B, Bulut C, et al. Constrictive pericarditis: risks, aetiologies and outcomes after total pericardiectomy: 24 years of experience. *Eur J Cardiothorac Surg*. 2013;44(6):1023-1028.
22. Unai S, Johnston DR. Radical pericardiectomy for pericardial diseases. *Curr Cardiol Rep*. 2019;21(2):6.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Duarte NF, de Aguiar Trigueirinho Ferreira S, Filho DAA, et al. Right ventricular dysfunction after pericardiectomy for tuberculous constrictive pericarditis: A case report. *Clin Case Rep*. 2024;12:e8899. doi:10.1002/ccr3.8899