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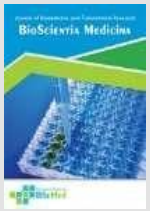

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Sternotomy and Pericardiectomy in a Patient with Constrictive Pericarditis and Multiple Comorbidities: A Case Report

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A B S T R A C T

Background: Constrictive pericarditis is a serious condition that can lead to heart failure. It is characterized by a thickened and scarred pericardium, which restricts the heart's ability to fill and pump blood effectively. The condition is often caused by infections, such as tuberculosis, but can also be caused by other factors, such as radiation therapy, heart surgery, or autoimmune diseases. A pericardiectomy, a surgical procedure to remove the thickened pericardium, is the definitive treatment for constrictive pericarditis. **Case presentation:** This case report describes a 57-year-old male patient with constrictive pericarditis and multiple comorbidities, including recent tuberculosis, pleural effusion, ascites, and cholelithiasis. The patient presented with symptoms of shortness of breath, swollen legs, and a swollen stomach. After a thorough evaluation, including imaging studies, the diagnosis of constrictive pericarditis was confirmed. The patient underwent a sternotomy and pericardiectomy procedure, which was successful in relieving his symptoms and improving his cardiac function. **Conclusion:** This case report highlights the successful management of constrictive pericarditis in a patient with multiple comorbidities. The case also emphasizes the importance of early diagnosis and timely intervention in patients with constrictive pericarditis.

1. Introduction

Constrictive pericarditis is a chronic inflammatory condition characterized by the thickening and rigidity of the pericardium, the fibroserous sac that envelops the heart. This pathological alteration of the pericardium impedes the heart's ability to expand and fill with blood during diastole, the relaxation phase of the cardiac cycle. The resulting restriction of ventricular filling leads to a constellation of clinical manifestations, including symptoms and signs of right heart failure. The pathophysiology of constrictive pericarditis is multifaceted and often involves a complex interplay of inflammatory processes, fibrosis, and calcification. The initiating factors can be diverse, ranging from infectious etiologies to non-infectious causes. In many cases, the precise etiology remains elusive, posing a challenge to diagnosis and management. The chronic inflammation within the pericardium triggers a cascade of events, ultimately leading to the deposition of collagen and other extracellular matrix components. This fibrotic process results in the thickening and stiffening of the pericardial layers, compromising their normal elasticity and distensibility. In some instances, the pericardium may also undergo calcification, further exacerbating the constrictive effect on the heart.[1,2]

The clinical presentation of constrictive pericarditis can be variable and may mimic other cardiac or

systemic conditions. Patients often present with symptoms of heart failure, such as dyspnea, fatigue, and peripheral edema. Dyspnea, or shortness of breath, is a common complaint, frequently exacerbated by exertion. Fatigue, a pervasive sense of weariness and lack of energy, can significantly impact a patient's quality of life. Peripheral edema, the accumulation of fluid in the interstitial spaces, typically manifests as swelling in the lower extremities, reflecting the increased hydrostatic pressure in the venous system due to impaired right ventricular filling. Physical examination findings in constrictive pericarditis may reveal signs of elevated central venous pressure, a hallmark of the condition. Jugular venous distension, the visible engorgement of the jugular veins in the neck, is a classic sign of increased venous pressure. Ascites, the accumulation of fluid in the peritoneal cavity, may also be present, contributing to abdominal distension. Hepatomegaly, the enlargement of the liver, and splenomegaly, the enlargement of the spleen, can occur due to chronic venous congestion. A pericardial knock, an early diastolic sound resulting from the abrupt cessation of ventricular filling by the rigid pericardium, may be auscultated on cardiac examination. The diagnostic evaluation of constrictive pericarditis requires a comprehensive approach, integrating clinical findings, electrocardiographic data, radiographic studies, and echocardiographic assessment. The electrocardiogram (ECG) may show non-specific abnormalities, such as low voltage, atrial fibrillation, or T-wave inversions. Chest radiography may reveal cardiomegaly, the enlargement of the heart, and pleural effusions, the accumulation of fluid in the pleural space surrounding the lungs.[3-5]

Echocardiography plays a pivotal role in the diagnosis of constrictive pericarditis. This non-invasive imaging technique provides valuable information regarding pericardial thickness, ventricular filling patterns, and the presence of constrictive physiology. Echocardiographic findings suggestive of constrictive pericarditis include pericardial thickening, abnormal septal motion, and respiratory variation in mitral and tricuspid valve inflow velocities. In some cases, further diagnostic evaluation may be warranted, such as cardiac catheterization or computed tomography (CT) or magnetic resonance imaging (MRI) of the chest. Cardiac catheterization can provide hemodynamic measurements, such as elevated right atrial pressure and equalization of diastolic pressures in all four cardiac chambers, which are characteristic of constrictive pericarditis. CT or MRI can provide detailed anatomical information regarding pericardial thickness and the presence of pericardial calcifications. The management of constrictive pericarditis typically involves a combination of medical and surgical interventions. Medical therapy is primarily aimed at managing the symptoms of heart failure, such as fluid overload, and may include the use of diuretics. However, definitive treatment often requires surgical intervention in the form of pericardiectomy, the surgical removal of the constricting pericardium.[6,7]

Pericardiectomy is a complex surgical procedure that aims to relieve the constrictive effect of the pericardium on the heart, thereby improving cardiac function and alleviating symptoms. The surgical approach typically involves a median sternotomy, a midline incision through the sternum, providing access to the mediastinum, the central compartment of the chest cavity. The thickened and scarred pericardium is carefully excised, freeing the heart from its constricting encasement. The postoperative care of patients undergoing pericardiectomy is crucial for ensuring a successful outcome. This includes meticulous monitoring of vital signs, respiratory function, and hemodynamic stability. Pain management, fluid and electrolyte management, and infection control are essential components of postoperative care. Early mobilization and physiotherapy are encouraged to prevent complications and promote recovery. The case presented in this report is particularly noteworthy due to the patient's history of pulmonary tuberculosis and the presence of multiple lymphadenopathy.

Pulmonary tuberculosis, an infectious disease caused by *Mycobacterium tuberculosis*, can have long-term sequelae, including pulmonary fibrosis and bronchiectasis, which can contribute to respiratory symptoms. The patient's history of pulmonary tuberculosis raises the possibility of a complex interplay of respiratory and cardiac pathologies. The presence of multiple lymphadenopathies, as revealed by imaging studies, is also a significant finding. Lymphadenopathy, the enlargement of lymph nodes, can be indicative of a variety of conditions, including infections, inflammatory processes, and malignancies. In this patient, the lymphadenopathy raised concerns about recurrent tuberculosis or other granulomatous diseases, necessitating further investigation.[8-10] This case report provides a valuable opportunity to explore the challenges in diagnosing and managing constrictive pericarditis in the context of multiple comorbidities.

2. Case Presentation

A 57-year-old male from Surakarta, Indonesia, presented to the medical facility with a constellation of symptoms indicative of a complex underlying medical condition. The patient's primary complaint was shortness of breath, a symptom that significantly impacts daily functioning and quality of life. This dyspnea was accompanied by swelling in the lower extremities (pedal edema) and abdominal distension, further suggesting a systemic process rather than an isolated respiratory issue. The onset of these symptoms was insidious, spanning three months prior to presentation. This protracted course suggests a chronic or slowly progressive disease process. The patient reported a progressive worsening of his symptoms during this period, indicating a lack of spontaneous resolution or response to conservative measures. The patient's past medical history is significant, revealing a history of hypertension and pulmonary tuberculosis, diagnosed in 2008. Notably, the patient had been declared cured of tuberculosis. However, the prior history of pulmonary tuberculosis raises the possibility of long-term sequelae such as pulmonary fibrosis or bronchiectasis, which could contribute to his current respiratory symptoms. Furthermore, the patient had been previously hospitalized for isolated right heart failure attributed to constrictive pericarditis. This is a crucial aspect of his history, as constrictive pericarditis can lead to chronic venous congestion and subsequent fluid accumulation, potentially explaining the pedal edema and abdominal distension. The patient denied any history of diabetes mellitus, stroke, or kidney disease. However, it's important to note that the absence of a reported history does not definitively rule out these conditions, especially in a patient with multiple comorbidities. The patient's social history revealed that he was a former smoker but denied current tobacco use. He also denied alcohol consumption. Smoking is a well-established risk factor for cardiovascular and respiratory diseases, and its prior use could have contributed to the development or exacerbation of his current condition. While the patient denies current alcohol use, it is essential to consider the potential long-term effects of past alcohol consumption on liver function and overall health. The physical examination revealed several significant findings. The patient's blood pressure was recorded as 110/80 mmHg, which, while not overtly hypertensive, should be interpreted in the context of his history of hypertension. A normal blood pressure reading does not preclude the possibility of underlying cardiovascular dysfunction, especially in a patient with a history of right heart failure. The patient's heart rate was 80 beats per minute, which is within the normal range. However, the respiratory rate was elevated at 20 breaths per minute, suggesting respiratory distress. This tachypnea aligns with the patient's chief complaint of shortness of breath. Oxygen saturation was 95% on room air, indicating mild hypoxemia. While not severely hypoxic, this level of oxygen saturation suggests impaired gas exchange, possibly due to pulmonary congestion or parenchymal lung disease. Jugular venous pressure was elevated, a critical finding indicative of increased central venous pressure. Elevated JVP is a hallmark of right heart

failure and suggests impaired right ventricular function or increased resistance to right atrial filling. Lung sounds were clear to auscultation bilaterally, which is somewhat unexpected given the patient's respiratory symptoms. However, clear lung sounds do not exclude the possibility of interstitial lung disease or other pulmonary pathologies. The absence of adventitious sounds such as crackles or wheezes does not rule out pulmonary congestion, especially if it is mild or localized. Abdominal examination revealed a soft and non-tender abdomen, but with fullness noted bilaterally. This finding, combined with the pedal edema and elevated JVP, strongly suggests fluid accumulation in the peritoneal cavity (ascites), a common complication of right heart failure and constrictive pericarditis. The molecular rapid test for *Mycobacterium tuberculosis* was negative, confirming the patient's declared cure of tuberculosis. However, it is essential to consider the possibility of non-tuberculous mycobacterial infections or other respiratory pathogens, especially in an immunocompromised individual. A chest X-ray revealed cardiomegaly and bilateral pleural effusions. Cardiomegaly, or an enlarged heart, is a common finding in patients with chronic heart failure. Pleural effusions, or fluid accumulation in the pleural space surrounding the lungs, are often a consequence of increased hydrostatic pressure due to heart failure or impaired lymphatic drainage. The electrocardiogram (ECG) showed sinus rhythm with low voltage. Low voltage on ECG can be indicative of several conditions, including pericardial effusion, pulmonary emphysema, and obesity. In this patient, given the history of constrictive pericarditis and the presence of pleural effusions, low voltage could be attributed to these factors. An echocardiogram revealed thickened pericardium and signs of constrictive physiology. This is a crucial finding, confirming the diagnosis of constrictive pericarditis. Thickening of the pericardium restricts the heart's ability to expand during diastole, leading to impaired ventricular filling and ultimately causing heart failure symptoms. The echocardiogram also demonstrated thickening of the abdominal fascia wall and omentum favoring an extruded omental hernia in the right side of the abdomen. This finding could be related to the patient's abdominal distension and requires further investigation. Computed tomography (CT) scan of the abdomen and pelvis revealed multiple lymphadenopathy in the retroperitoneal, bilateral paracolic, and bilateral inguinal regions, suspected of tuberculosis-related adenopathy. This finding is concerning, given the patient's history of tuberculosis, despite the negative molecular rapid test. It raises the possibility of recurrent tuberculosis or other granulomatous diseases. The CT scan also showed multiple calcifications in the pericardium, consistent with the known constrictive pericarditis. Additionally, it showed bilateral pleural effusions, left inguinal hernia, and hepatosplenomegaly. Hepatomegaly and splenomegaly, or enlargement of the liver and spleen, respectively, are often indicative of chronic venous congestion and can be seen in patients with right heart failure. Based on the comprehensive evaluation, a clinical diagnosis of constrictive pericarditis was established. This diagnosis is supported by the patient's history of right heart failure attributed to constrictive pericarditis, the physical examination findings of elevated JVP and abdominal distension, and the echocardiographic evidence of thickened pericardium with constrictive physiology. Recurrent tuberculosis was considered as a differential diagnosis, given the patient's history of tuberculosis and the presence of multiple lymphadenopathies on the CT scan. However, the negative molecular rapid test for *Mycobacterium tuberculosis* makes this less likely. Other potential diagnoses include pleural effusions, ascites, cardiomegaly, and hernia. These findings are likely secondary to the underlying constrictive pericarditis and right heart failure (Table 1).

The patient, a 57-year-old male from Surakarta, Indonesia, with a complex medical history including constrictive pericarditis, underwent a surgical intervention in Bandar Lampung, Lampung, Indonesia. The procedure was meticulously

documented, encompassing a series of steps aimed at addressing the patient's diagnosed conditions. This section will delve into the procedural details, elucidating the rationale and techniques employed during the surgical intervention. The initial phase of the procedure involved a median sternotomy, a standard surgical approach for accessing the thoracic cavity. This began with a meticulous skin incision. A midline incision was made, extending from the sternal notch, a readily identifiable anatomical landmark at the superior border of the manubrium, to the xiphoid process, the cartilaginous structure at the inferior end of the sternum. This incision, carefully executed through the skin and subcutaneous tissue, provided the initial access to the underlying skeletal structures. The depth and length of the incision were precisely controlled to minimize tissue trauma while ensuring adequate exposure for subsequent steps. Following the skin incision, the sternotomy itself was performed. This involved the longitudinal division of the sternum, the bone forming the anterior midline of the chest wall. A specialized surgical instrument, a sternal saw, was utilized to achieve this division. The sternal saw, designed to minimize bone fragmentation and thermal injury, allowed for a clean and controlled sternal split. The division of the sternum was performed with utmost care, ensuring the integrity of the surrounding tissues and minimizing the risk of complications such as mediastinal bleeding or injury to underlying structures. With the sternum divided, the surgical field was expanded, and the mediastinum, the central compartment of the thoracic cavity, was exposed. This allowed for a thorough exploration of the mediastinal structures, including the pericardium, the fibroserous sac surrounding the heart. The exploration phase was crucial for confirming the preoperative diagnosis of constrictive pericarditis and for assessing the extent of pericardial involvement. Visual inspection of the pericardium provided valuable information regarding its thickness, degree of calcification, and the presence of adhesions to surrounding structures. The core of the surgical intervention was the pericardiectomy, the surgical removal of the pericardium. This procedure was undertaken to relieve the constrictive effect of the thickened and scarred pericardium on the heart, thereby improving cardiac function and alleviating the patient's symptoms. The initial step in the pericardiectomy was adhesiolysis, the meticulous separation of adhesions between the pericardium and the heart. Adhesions, formed as a result of chronic inflammation and scarring, can tether the pericardium to the epicardium, the outermost layer of the heart. These adhesions were carefully lysed using a combination of sharp and blunt dissection techniques. Sharp dissection, employing surgical instruments such as scissors or scalpels, was used to divide dense adhesions, while blunt dissection, utilizing instruments such as dissectors or swabs, was used to gently separate looser adhesions. The goal of adhesiolysis was to free the heart from the constricting pericardium without causing injury to the delicate myocardial tissue. Following adhesiolysis, the thickened and scarred pericardium was excised. This involved the careful removal of the affected portions of the pericardium, leaving behind a thin layer of residual pericardium to prevent direct contact between the heart and surrounding structures. The extent of pericardial excision was determined based on the degree of pericardial involvement and the intraoperative findings. The excised pericardium was sent for histopathological examination to confirm the diagnosis and to rule out other pathological processes. In addition to the pericardiectomy, the surgical intervention also addressed the patient's bilateral pleural effusions, the accumulation of fluid in the pleural space surrounding the lungs. This was achieved through pleurectomy, the surgical removal of a portion of the pleura, the serous membrane lining the thoracic cavity. The right pleura was opened first, and a significant amount of serous fluid, approximately 1100 cc, was drained. This drainage relieved the compressive effect of the pleural effusion on the right lung, allowing for improved lung expansion and ventilation. The left pleura was then opened, although the volume of fluid drained from the left pleural space was not specified. The pleurectomy

procedure aimed to prevent the reaccumulation of pleural fluid and to promote the apposition of the lung to the chest wall. To further optimize pulmonary function, a lung release procedure was performed. This involved freeing the lungs from any adhesions or restrictions that might impede their expansion. The right lung was carefully freed from the pleura, ensuring its complete expansion. Similarly, the left lung was released from any pleural adhesions, allowing it to expand fully and participate in gas exchange. The lung release procedure was crucial for improving the patient's respiratory mechanics and alleviating his symptoms of dyspnea. Following the completion of the pericardiectomy, pleurectomy, and lung release procedures, the surgical field was meticulously inspected for hemostasis, ensuring the absence of active bleeding. The sternum was then closed using sternal wires, providing rigid fixation and promoting bone healing. The sternal wires, strategically placed to distribute tension evenly across the sternal edges, ensured the stability of the chest wall and prevented sternal dehiscence. The wound was closed in layers, approximating the subcutaneous tissue and skin edges. This layered closure technique minimized the risk of wound infection and promoted optimal wound healing. A subxiphoid drain was placed to facilitate the drainage of any residual fluid or air from the mediastinum and pleural spaces. The drain was secured to the skin and connected to a closed drainage system, allowing for continuous monitoring of drainage volume and characteristics. The patient was then transferred to the postoperative care unit for close monitoring and management. Postoperative care focused on pain control, respiratory support, and prevention of complications such as infection, bleeding, and arrhythmias. The subxiphoid drain was monitored for drainage volume and characteristics, and removed once drainage was minimal. The patient's respiratory status was closely monitored, and supplemental oxygen was administered as needed. This detailed description of the "Procedure of Treatment" provides a comprehensive understanding of the surgical intervention performed on the patient. The meticulous execution of each step, from the initial sternotomy to the final wound closure, reflects the surgical team's commitment to providing optimal care. The procedural details, elucidated in this section, highlight the complexity and precision of the surgical intervention, contributing to a thorough understanding of the patient's management (Table 2).

Following the intricate surgical intervention for constrictive pericarditis, bilateral pleural effusions, and associated complications, the patient's postoperative care was meticulously planned and executed. This section will delve into the details of the patient's immediate and early postoperative management, as well as the comprehensive follow-up care provided, aiming to illustrate the multifaceted approach to recovery and long-term health. Immediately following the procedure, the patient was transferred to the Intensive Care Unit (ICU) for close observation and specialized care. This transfer was crucial given the complexity of the surgery and the potential for postoperative complications. The ICU setting provided the necessary resources for continuous monitoring of vital signs, respiratory function, and hemodynamic stability. Upon admission to the ICU, the patient was placed on mechanical ventilation to support breathing. This was particularly important given the extensive nature of the thoracic surgery and the potential for respiratory compromise. Mechanical ventilation allowed for precise control of respiratory parameters, ensuring adequate oxygenation and carbon dioxide removal. The ventilator settings were adjusted based on the patient's arterial blood gas analysis and clinical status, aiming to optimize respiratory function while minimizing the risk of ventilator-associated lung injury. Hemodynamic support was a cornerstone of the immediate postoperative care. The patient's heart rate, blood pressure, and cardiac output were closely monitored. Inotropic support, using medications that enhance cardiac contractility, was provided as needed to maintain hemodynamic stability. This was crucial given the potential for postoperative hypotension or low cardiac output, which could compromise organ

perfusion and delay recovery. Pain management was a critical aspect of the patient's immediate postoperative care. Adequate pain control was essential to ensure patient comfort and facilitate early mobilization. Pain was assessed using a standardized pain scale, and analgesics were administered as needed. The choice and dosage of analgesics were carefully titrated to balance pain relief with the potential for side effects, such as respiratory depression or sedation. Fluid management was meticulously carried out to prevent fluid overload or dehydration. Careful monitoring of fluid balance was crucial to maintain hemodynamic stability and prevent complications. Intravenous fluids were administered as needed, and the patient's urine output was closely monitored. This was particularly important given the patient's history of heart failure and the potential for postoperative fluid shifts. Electrolyte monitoring was another essential component of the immediate postoperative care. Regular monitoring of electrolytes, such as sodium, potassium, and magnesium, was performed to identify and correct any imbalances. Electrolyte abnormalities can lead to cardiac arrhythmias and other complications, highlighting the importance of meticulous electrolyte management. Infectious control was paramount in preventing postoperative infections. Strict aseptic techniques were followed during all procedures, including dressing changes and line insertions. Prophylactic antibiotics were administered as appropriate to minimize the risk of surgical site infections or other infectious complications. As the patient's respiratory function improved, they were gradually weaned from mechanical ventilation. This process involved progressively reducing the level of ventilatory support, allowing the patient to assume more of the work of breathing. The weaning process was closely monitored, and arterial blood gas analysis was performed to assess the patient's ability to maintain adequate oxygenation and ventilation. Drainage management was continued throughout the early postoperative period. The subxiphoid drain was monitored for drainage output and air leaks. It was removed once drainage had decreased and air leaks had resolved. This allowed for the prevention of complications such as mediastinitis or pneumothorax. Ambulation was encouraged early in the postoperative period to prevent complications such as deep vein thrombosis and pneumonia. Early mobilization was facilitated by physical therapy and nursing staff, who assisted the patient with sitting, standing, and walking. Physiotherapy played a crucial role in the patient's recovery. Chest physiotherapy was provided to help clear secretions and improve lung expansion. Exercises were tailored to the patient's individual needs and abilities, aiming to enhance respiratory function and prevent pulmonary complications. Nutritional support was provided to promote healing and recovery. Adequate nutritional intake is essential for wound healing, immune function, and overall recovery. Nutritional support was provided through oral intake or enteral feeding, depending on the patient's tolerance and clinical status. Once the patient was stable, they were transferred from the ICU to a general ward for ongoing monitoring and care. This transition marked a significant step in the patient's recovery, indicating their improved clinical status and reduced need for intensive care. Continued monitoring of vital signs, respiratory function, and hemodynamic stability was performed on the general ward. This allowed for the early detection of any potential complications and ensured the patient's ongoing stability. Wound care was an essential component of the patient's postoperative management. The surgical wound was regularly inspected for signs of infection or dehiscence. Dressings were changed as needed, using aseptic techniques to minimize the risk of infection. Medications management was continued on the general ward. The patient's medications were reviewed and adjusted as necessary, based on their clinical status and laboratory findings. This included the continuation of analgesics, antibiotics, and other medications as needed. Patient education was provided to the patient and their family regarding postoperative care. This included education on wound care, medication management, and activity

restrictions. Patient education empowered the patient and their family to actively participate in the recovery process and ensured a smooth transition to home care. The patient was scheduled for regular outpatient visits to monitor their progress and assess for any complications. This allowed for ongoing evaluation of the patient's clinical status and the early detection of any potential issues. Cardiac rehabilitation was recommended to help the patient regain their strength and improve their cardiovascular health. Cardiac rehabilitation programs are tailored to individual patient needs and typically include exercise training, education, and counseling. This was crucial for optimizing the patient's long-term cardiovascular health. Long-term laboratory monitoring was performed for any treatment of constrictive pericarditis or other complications. This included regular blood tests to monitor kidney function, liver function, and electrolyte levels. Imaging studies, such as echocardiograms or chest X-rays, were also performed as needed to assess the patient's cardiac and pulmonary status. This detailed account of the "Procedure of Post Treatment and Follow-up" provides a comprehensive understanding of the patient's recovery process. The meticulous planning and execution of each step, from the immediate postoperative care in the ICU to the long-term follow-up, highlight the multidisciplinary approach to patient management. The emphasis on monitoring, rehabilitation, and education underscores the commitment to optimizing the patient's long-term health and well-being (Table 3).

Table 1. Summary of patient findings.

Characteristic	Data
Demographics	
Age	57 years
Gender	Male
Location	Surakarta, Indonesia
Anamnesis	
Chief complaint	Shortness of breath, swollen legs, and swollen stomach
Duration of symptoms	3 months
Aggravating factors	Sleeping position
Alleviating factors	Sitting position
Other symptoms	None
Past medical history	Hypertension, pulmonary tuberculosis (2008, declared cured), recent hospitalization for isolated right heart failure due to constrictive pericarditis
Social history	Former smoker, no history of diabetes mellitus, stroke, kidney disease, or alcohol use
Physical examination	
Blood pressure	140/90 mmHg
Heart rate	80 beats per minute
Respiratory rate	20 breaths per minute
Oxygen saturation	95% on room air
Jugular venous pressure	Elevated
Lung sounds	Clear to auscultation bilaterally
Abdomen	Soft and non-tender
Legs	Edematous bilaterally
Laboratory	
Molecular rapid test for *Mycobacterium tuberculosis*	Negative
Imaging	
Chest X-ray	Cardiomegaly and bilateral pleural effusions
Electrocardiogram	Sinus rhythm with low voltage
Echocardiogram	Thickened pericardium and signs of constrictive physiology
Computed tomography (CT) scan of the abdomen	Thickening of the ileocaecal lumen wall and omentum forming an omental cake image in the right iliac region accompanied by ascites and multiple lymphadenopathy in the mesenteric, bilateral parailiac and bilateral inguinal regions suspected of TB peritonitis; multiple calcifications in the pericardium; cholecystitis accompanied by cholelithiasis; bilateral pleural effusion; left inguinal hernia; and hepatosplenomegaly
Clinical diagnosis	
Constrictive pericarditis	
Recent tuberculosis	
Pleural effusion	
Ascites	
Cholelithiasis	

Table 2. Procedure of treatment.

Procedure	Description
Sternotomy	
1. Incision	A midline incision was made through the skin and subcutaneous tissue, extending from the sternal notch to the xiphoid process.
2. Sternotomy	The sternum was divided longitudinally using a sternal saw.
3. Exploration	The pericardium was exposed and inspected.
Pericardiectomy	
1. Adhesiolysis	Adhesions between the pericardium and the heart were carefully lysed using sharp and blunt dissection.
2. Pericardiectomy	The thickened and scarred pericardium was excised.
Pleurectomy	
1. Right pleurectomy	The right pleura was opened, and 1100 cc of serous fluid was drained.
2. Left pleurectomy	The left pleura was opened.
Lung release	
1. Right lung release	The right lung was freed from the pleura and expanded well.
2. Left lung release	The left lung was freed from the pleura and expanded well.
Closure	
1. Sternal closure	The sternum was closed with sternal wire.
2. Wound closure	The wound was closed in layers.
3. Drain placement	A subxiphoid drain was placed.

Table 3. Procedure of post-treatment and follow-up.

Post-treatment and follow-up	Description
Immediate postoperative care	
1. Intensive Care Unit (ICU) Admission	The patient was admitted to the ICU for close monitoring of vital signs, respiratory function, and hemodynamic stability.
2. Mechanical Ventilation	The patient was initially placed on mechanical ventilation to support breathing and ensure adequate oxygenation.
3. Hemodynamic Support	Continuous monitoring of the patient's heart rate, blood pressure, and cardiac output was performed. Inotropic support or other interventions were provided as needed to maintain hemodynamic stability.
4. Pain Management	Adequate pain control was provided to ensure patient comfort and facilitate early mobilization.
5. Fluid Management	Careful monitoring of fluid balance was crucial to prevent fluid overload or dehydration. Intravenous fluids were administered as needed, and the patient's urine output was closely monitored.
6. Electrolyte Monitoring	Regular monitoring of electrolytes, such as sodium, potassium, and magnesium, was performed to identify and correct any imbalances.
7. Infection Control	Strict aseptic techniques were followed to minimize the risk of infection. Prophylactic antibiotics were administered as appropriate.
Early postoperative period	
1. Weaning from Mechanical Ventilation	The patient was gradually weaned from mechanical ventilation as their respiratory function improved.
2. Chest Tube Management	The chest tube was closely monitored for drainage output and air leaks. It was removed when drainage had decreased and air leaks had resolved.
3. Ambulation	Early ambulation was encouraged to prevent complications such as deep vein thrombosis and pneumonia.
4. Physiotherapy	Chest physiotherapy was provided to help clear secretions and improve lung function.
5. Nutritional Support	Adequate nutritional support was provided to promote healing and recovery.
Transfer to ward	
1. Continued Monitoring	Once the patients were stable, they were transferred to the ward for ongoing monitoring and care.
2. Wound Care	The surgical wound was regularly inspected, and dressings were changed as needed.
3. Medication Management	The patient's medications were reviewed and adjusted as necessary.
4. Patient Education	The patient and their family were provided with education on postoperative care, including wound care, medication management, and activity restrictions.
Follow-up care	
1. Regular Outpatient Visits	The patient was scheduled for regular outpatient visits to monitor their progress and assess for any complications.
2. Cardiac Rehabilitation	Cardiac rehabilitation was recommended to help the patient regain their strength and improve their cardiovascular health.
3. Long-Term Monitoring	Long-term monitoring was necessary to assess for any recurrence of constrictive pericarditis or other complications.

3. Discussion

The patient presented with a constellation of symptoms, including shortness of breath, pedal edema, and abdominal distension, which are consistent with the clinical manifestations of right heart failure. These symptoms had been progressively worsening over a period of three months, suggesting a chronic or slowly progressive underlying pathology. The insidious onset of symptoms is a common feature of constrictive pericarditis. The gradual thickening and stiffening of the pericardium lead to a progressive restriction of ventricular filling, resulting in a gradual elevation of systemic venous pressures. This elevation of venous pressure manifests clinically as peripheral edema, ascites, and jugular venous distension. Dyspnea, or shortness of breath, is another common symptom, which can be attributed to pulmonary congestion and reduced cardiac output. The patient's past medical history was significant for hypertension and pulmonary tuberculosis, diagnosed in 2008. While the patient had been declared cured of tuberculosis, the prior history of pulmonary tuberculosis raised the possibility of long-term sequelae such as pulmonary fibrosis or bronchiectasis, which could contribute to his current respiratory symptoms. Pulmonary tuberculosis can sometimes lead to pericardial involvement, either through direct extension or hematogenous spread, which could potentially contribute to the development of constrictive pericarditis. However, in this case, the molecular rapid test for *Mycobacterium tuberculosis* was negative, suggesting that active tuberculosis was unlikely. The patient also had a history of hospitalization for isolated right heart failure attributed to constrictive pericarditis. This is a crucial aspect of his history, as it indicates a previous diagnosis of constrictive pericarditis and suggests a recurrence or exacerbation of the condition. Constrictive pericarditis can be a chronic and progressive disease, and even after treatment, recurrence can occur. The physical examination revealed several significant findings, including an elevated respiratory rate, mild hypoxemia, elevated jugular venous pressure, and abdominal fullness. These findings are consistent with the clinical manifestations of right heart failure and support the diagnosis of constrictive pericarditis. The clear lung sounds on auscultation were somewhat unexpected, given the patient's respiratory symptoms and the presence of pleural effusions. However, clear lung sounds do not exclude the possibility of interstitial lung disease or other pulmonary pathologies.[11-13]

Imaging studies played a critical role in confirming the diagnosis of constrictive pericarditis. The chest X-ray revealed cardiomegaly and bilateral pleural effusions. Cardiomegaly is a common finding in patients with chronic heart failure, reflecting the compensatory dilatation of the heart chambers. Pleural effusions are also common in heart failure, resulting from increased hydrostatic pressure in the pulmonary circulation. The electrocardiogram showed sinus rhythm with low voltage. Low voltage on ECG can be indicative of several conditions, including pericardial effusion, pulmonary emphysema, and obesity. In this patient, given the history of constrictive pericarditis and the presence of pleural effusions, low voltage could be attributed to these factors. The echocardiogram was the most important imaging study in confirming the diagnosis of constrictive pericarditis. It revealed a thickened pericardium and signs of constrictive physiology, including abnormal septal motion and respiratory variation in mitral and tricuspid valve inflow velocities. These findings are characteristic of constrictive pericarditis and provide strong evidence for the diagnosis. The echocardiogram also demonstrated thickening of the abdominal fascia wall and omentum, favoring an extruded omental hernia in the right side of the abdomen. This finding could be related to the patient's abdominal distension and requires further investigation. The computed tomography (CT) scan of the abdomen and pelvis revealed multiple lymphadenopathy in the retroperitoneal, bilateral paracolic, and bilateral inguinal regions, suspected of tuberculosis-related adenopathy. This finding is concerning, given the patient's history of tuberculosis, and raises the

possibility of recurrent tuberculosis or other granulomatous diseases. The CT scan also showed multiple calcifications in the pericardium, consistent with the known constrictive pericarditis. Additionally, it showed bilateral pleural effusions, left inguinal hernia, and hepatosplenomegaly. Hepatomegaly and splenomegaly are often indicative of chronic venous congestion and can be seen in patients with right heart failure.[14-16]

The surgical intervention in this case was multifaceted, addressing the patient's constrictive pericarditis, pleural effusions, and associated complications. The procedure involved a median sternotomy, pericardiectomy, pleurectomy, lung release, and closure. The median sternotomy provided excellent exposure of the mediastinum, allowing for a thorough assessment of the pericardium and surrounding structures. The pericardiectomy involved the meticulous excision of the thickened and scarred pericardium, freeing the heart from its constricting encasement. This procedure is the definitive treatment for constrictive pericarditis, aiming to relieve the hemodynamic impairment and alleviate symptoms. A pleurectomy was performed to address the patient's bilateral pleural effusions. The removal of a portion of the pleura allowed for the drainage of fluid and prevented its reaccumulation. The lung release procedure aimed to improve lung expansion and ventilation by freeing the lungs from any adhesions or restrictions. The surgical technique employed was meticulous, emphasizing careful dissection and hemostasis. The goal was to minimize tissue trauma and prevent complications. The procedural details, as documented in the table, highlight the complexity and precision of the surgical intervention.[17,18]

The patient's postoperative course was carefully managed in the intensive care unit (ICU) and then on the general ward. The immediate postoperative care focused on hemodynamic support, respiratory support, pain management, fluid and electrolyte management, and infection control. Mechanical ventilation was provided initially to support breathing, and hemodynamic support was provided to maintain adequate blood pressure and cardiac output. Pain management was a critical component of postoperative care. Adequate pain control facilitated early mobilization and prevented complications. Fluid and electrolyte management was essential to maintain hemodynamic stability and prevent imbalances. Infection control measures were implemented to minimize the risk of postoperative infections. As the patient's condition improved, he was gradually weaned from mechanical ventilation and transferred to the general ward. On the ward, continued monitoring, wound care, medication management, and patient education were provided. Early mobilization and physiotherapy were encouraged to prevent complications and promote recovery. The patient was scheduled for regular outpatient visits for follow-up care. Cardiac rehabilitation was recommended to help the patient regain strength and improve cardiovascular health. Long-term laboratory monitoring was performed for any treatment of constrictive pericarditis or other complications.[19,20]

4. Conclusion

This case report elucidates the complexities of managing constrictive pericarditis in a patient with a significant burden of comorbidities, including a history of pulmonary tuberculosis, pleural effusion, ascites, and cholelithiasis. The patient's presentation with symptoms of shortness of breath, pedal edema, and abdominal distension underscored the challenges in diagnosis, necessitating a comprehensive evaluation. The diagnostic process integrated clinical assessment, laboratory investigations, and advanced imaging techniques, with echocardiography playing a pivotal role in confirming the diagnosis of constrictive pericarditis by demonstrating pericardial thickening and signs of constrictive physiology. The surgical management, involving sternotomy and pericardiectomy, was successful in alleviating the patient's symptoms and improving cardiac function. The meticulous surgical technique, including adhesiolysis, pericardial excision, pleurectomy, and lung release, underscores the importance of a precise

and thorough approach in addressing this complex condition. Postoperatively, the patient received comprehensive care, including mechanical ventilation, hemodynamic support, pain management, and infection control, highlighting the significance of multidisciplinary management in optimizing patient outcomes. The successful outcome in this case underscores the importance of early diagnosis, timely surgical intervention, and meticulous postoperative care in patients with constrictive pericarditis and multiple comorbidities.

5. References

1. Egwu C, Scullion M, Gingles C, Kerrigan S. Constrictive pericarditis requiring pericardiectomy: an unusual first presentation of extra-articular rheumatoid arthritis-a case report. Eur Heart J Case Rep. 2024; 8(9): ytae428.
2. Miyazaki T, Kamimura D, Wakamatsu M, Konishi M, Matsumura A, Teshigawara H, et al. Rare severe constrictive pericarditis complication in Erdheim-Chester disease: a case report and literature review. J Clin Exp Hematop. 2024; 64(3): 232–6.
3. Kaya Ç, Yalta K. Is constrictive pericarditis associated with long COVID, its vaccine, or both? Balkan Med J. 2024; 41(5): 411–2.
4. Tarasov RV, Krasnikova EV, Cadovnikova SS, Prokhodtsov DN, Khozikov AS, Bagirov MA. A case of treatment of chronic constrictive pericarditis of tuberculous etiology. Vmirvz. 2024; 14(4): 92–8.
5. Benjanuwattra J, Feazell JD, Dressman SA, Harris DM. Constrictive pericarditis with concurrent aortic insufficiency: The diagnostic challenge. J Cardiovasc Magn Reson. 2025; 27(101735): 101735.
6. Chen A, Melvin R. Novel use of parametric mapping in the assessment of acute pericarditis and hemopericardium in the setting of constrictive pericarditis. J Cardiovasc Magn Reson. 2025; 27(101719): 101719.
7. Teixeira BL, Albuquerque F, Cardoso I, Ferreira V, Fiarresga A, Cardoso F, et al. Overcoming diagnostic challenges in constrictive pericarditis: why volume is important? Int J Cardiovasc Imaging. 2025; 41(1): 157–60.
8. Pan J, Villines TC. Linking physiology to findings on invasive and non-invasive tests for the diagnosis of constrictive pericarditis. J Cardiovasc Comput Tomogr. 2025; 19(1): 56–7.
9. Ono K, Nomura T, Shoji K, Kato Y, Wada N. A very rare phenotype of immunoglobulin G4-related disease that was manifested as constrictive pericarditis: a case report. Eur Heart J Case Rep. 2025; 9(1): ytae689.
10. Lee HN, Hyun J, Jung SH, Kim JB, Lee JE, Yang DH, et al. CT-derived myocardial strain measurement in patients with chronic constrictive pericarditis. J Cardiovasc Comput Tomogr. 2025; 19(1): 48–55.
11. Dos Santos VM, Tedeschi AP, Modesto LC, Modesto JC. Constrictive pericarditis associated with COVID-19 or vaccination. Balkan Med J. 2025; 42(1): 87–8.
12. Beseliia KM, Magomedova ZM, Eldzharkieva FK, Dolgaya MS, Shchekochikhin DY, Pershina ES, et al. Potential of magnetic resonance imaging in diagnostics of transient constrictive pericarditis. Kardiologiia. 2025; 65(1): 67–72.
13. Ren Y, Dong Y, Fan X, Xu H, Yin S. Development of rapid evaluation scale for pericardiectomy of tuberculous constrictive pericarditis by 12-lead electrocardiogram. J Cardiothorac Surg. 2025; 20(1): 27.
14. Luebbe ST, Youmans Q, Provias T, Lewis A, Cremer PC, Obeidin F, et al. Primary pericardial mesothelioma causing constrictive pericarditis. JACC Case Rep. 2025; (103204): 103204.

15. Tribak M, Adanho C-M, Elmourabit Y, Leghlimi LH, Soufiani A, Agoumy Z, et al. Chronic constrictive pericarditis with rheumatic mitral regurgitation. J Surg Case Rep. 2025; 2025(2): rjae424.
16. Tsunematsu O, Takeda S-I, Yamaguchi M, Kaneko M, Oe K, Murakami Y, et al. Constrictive pericarditis in a hemodialysis patient who presented with rapidly progressive pericardial calcification: a diagnostic challenge—case report. Case Rep Nephrol Dial. 2025; 1–12.
17. Takahashi T, Teodorescu DL, Kniola W, Luthringer D, Singh S. Extensive pericardial metastasis of angiosarcoma: Diagnostic challenge in a young case with effusive constrictive pericarditis. Am J Cardiol. 2025; 243: 40–4.
18. Yoshinaga T, Takaki J, Nakata K, Hirota T, Hidaka H, Horibe T, et al. Pericardiectomy using an ultrasonic scalpel in patients with constrictive pericarditis. Interdiscip Cardiovasc Thorac Surg. 2025; 40(2).
19. Nuaimi M, Shaw B, Ubosy S, Iyyani M, Shaw R, Madruga M, et al. Constrictive pericarditis as a post-cardiac surgery complication. Am J Case Rep. 2025; 26: e945294.
20. Jia B, Yan S, Luo Y, Cheng J, Cheng J, Fei J, et al. Post-pericardiectomy ECMO for constrictive pericarditis: a case series and literature review. BMC Anesthesiol. 2025; 25(1): 110.