Cardiology Research and Cardiovascular Medicine
Richards B, et al. Cardiolog Res Cardiovasc Med 8: 242.
www.doi.org/10.29011/2575-7083.100242
www.gavinpublishers.com

OPEN ACCESS

GAVIN PUBLISHERS

Review Article

Idiopathic Chylopericardium in a 7-Year-Old Female with Latent Tuberculosis: A Case Report and Literature Review

Blythe Richards[1], Farrah Gaston[1], Girish Deshpande[1,2], Joe Baker[1], Mustafa Bakir[1,2*]

[1]University of Illinois College of Medicine Peoria, Peoria, Illinois, 530 NE Glen Oak Avenue, Peoria, IL 61637, Fax 309-655-2565 USA

[2]Children's Hospital of Illinois, OSF HealthCare, Peoria, Illinois 3 Lurie Children's Hospital, Chicago, Illinois USA

***Corresponding author:** Mustafa Bakir, University of Illinois College of Medicine Peoria, Peoria, Illinois, 530 NE Glen Oak Avenue, Peoria, IL 61637, Fax 309-655-2565 USA

Citation: Richards B, Gaston F, Deshpande G, Baker J, Bakir M (2024) Idiopathic Chylopericardium in a 7-Year-Old Female with Latent Tuberculosis: A Case Report and Literature Review. Cardiol Res Cardio vasc Med 9:242. DOI:https://doi.org/10.29011/2575-7083.100242

Received Date: 28 March, 2024; **Accepted Date:** 02 April, 2024; **Published Date:** 05 April, 2024

Abstract

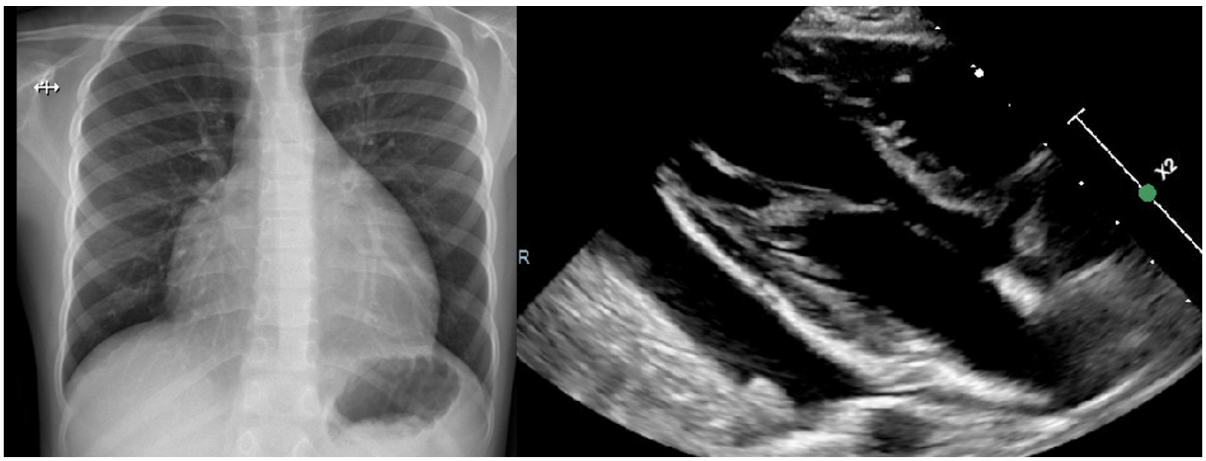
Diagnosing idiopathic (primary) chylopericardium is an extensive process that involves ruling out all potential secondary causes. Pericardiocentesis and fluid analysis are essential for diagnosis and additionally offer symptomatic relief. Management of idiopathic chylopericardium necessitates pinpointing and surgically correcting the lymphatic anomaly contributing to fluid accumulation. This case describes the diagnosis and management of idiopathic chylopericardium in a previously healthy 7-year-old female. Initial diagnosis was made with chest x-ray (CXR), transthoracic echocardiogram (TTE), and computed tomography (CT), followed by pericardiocentesis and pericardial drain placement. Pericardial fluid analysis revealed high triglycerides. Additional laboratory workup was largely unremarkable except for a positive QuantiFERON-TB Gold test. While disseminated tuberculosis infection can cause pericarditis and pericardial effusion, latent infection is unlikely to result in chylopericardium. Diagnosis of abnormal lymphatic perfusion and leakage into the pericardial sac was eventually established with dynamic contrast-enhanced magnetic resonance lymphangiogram (DCMRL). Non-target embolization with lip iodol administered via bilateral inguinal lymph nodes was initially performed, but chylopericardium recurred. The patient subsequently underwent selective lymphatic embolization targeting an abnormally dilated lymphatic channel in the upper mediastinum. Repeat lymphangiogram immediately after selective embolization showed continued abnormal mediastinal lymphatic perfusion through diminutive lymphatic channels, which ultimately necessitated thoracic duct embolization. The patient had diminished pericardial effusion at three weeks post-embolization. She has been monitored with serial echocardiograms, with the most recent showing only a tiny/small amount of pericardial effusion. Continuous surveillance in the coming months and years will be essential to confirm successful embolization.

Introduction

Pericardial effusion, the accumulation of fluid within the pericardial sac, is diagnosed when pericardial fluid surpasses the typical volume of less than 50 cc. When this fluid is primarily composed of chyle, a high-fat lymphatic fluid, the condition is termed chylopericardium. Chylopericardium is rare, accounting for less than 3% of all pericardial effusions. Identifying chyle involves fluid analysis revealing a triglyceride level greater than 500 mg/dL and a cholesterol: triglyceride ratio less than one [1]. The appearance of chyle varies, often resembling a milky white liquid, but it can occasionally exhibit hues of gold or brown [2]. Diagnosis of idiopathic (primary) chylopericardium is an extensive process that involves ruling out all potential secondary causes. Secondary causes are more common and are predominantly linked to trauma or surgical complications due to the location of the thoracic duct. Less common secondary causes include systemic infectious disease processes or autoimmune diseases. Disseminated tuberculosis is a well-documented cause of secondary chylopericardium [3]. Primary chylopericardium is typically associated with rare anatomical anomalies or malformations in the lymphatic system. Comprehensive data on common causes, prevalence, and incidence is elusive due to the scarcity of documented cases, with a thorough 65-year study finding only 104 cases of idiopathic chylopericardium across PubMed and Wanfang databases [2]. Hemodynamically stable patients, constituting approximately 40% of idiopathic chylopericardium cases, often remain asymptomatic, presenting a diagnostic challenge. In symptomatic patients, 44% of patients experienced dyspnoea, while 93% exhibited an enlarged cardiac silhouette on x-ray. Less commonly observed signs/symptoms at presentation included cough and jugular venous distention [2,4]. In one case of chylopericardium, chyloptysis, the expectoration of chyle due to leakage into the airway, occurred alongside chylopericardium. Both idiopathic chylopericardium and chyloptysis can occur with abnormal mediastinal/hilar lymphatic perfusion, suggesting potential under-recognition by healthcare providers due to the innocuous appearance of chyle in sputum. Manifestations of the condition vary: acute (< 1 week) and subacute (< 3 months) cases may be more symptomatic, while chronic disease (>3 months) tends to be asymptomatic due to the gradual accumulation of the effusion, which is generally tolerated well [5]. Detecting pericardial effusion in asymptomatic patients is typically incidental on chest x-ray, and diagnosis requires TTE, CT, or magnetic resonance imaging (MRI). Pericardiocentesis and fluid analysis are essential for diagnosing chylopericardium specifically and additionally offer symptomatic relief. Management of idiopathic chylopericardium requires pinpointing and correcting the lymphatic abnormality contributing to fluid accumulation either surgically or with a minimally invasive interventional radiology (IR) procedure [6-9].

Case

A seven-year-old female with an unremarkable past medical and surgical history initially presented to an outpatient clinic due to a persistent cough lasting over one month and one week of unexplained night sweats. Notably, she had family contacts that had recently traveled to Peru. The initial outpatient workup included a chest x-ray and echocardiogram. The chest x-ray showed clear lungs, but enlargement of the cardiac silhouette. Echocardiogram revealed the presence of >200 mL pericardial fluid in the pericardial sac without tamponade (Figures 1-2).



Figures 1-2: Initial CXR and echocardiogram demonstrating enlarged cardiac silhouette and pericardial effusion respectively

In response to these findings, she was directed to go to her local emergency department (ED). At the ED, a comprehensive initial laboratory workup was performed, including a complete blood count (CBC), complete metabolic panel (CMP), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), brain natriuretic peptide (BNP), and troponin, all of which were unremarkable. Following this, she was transferred to our hospital for further evaluation and treatment.

Upon admission to our hospital, the patient was started on furosemide (Lasix), and a chest CT scan was performed that confirmed the diagnosis of pericardial effusion without other mediastinal pathology (Figure 3).

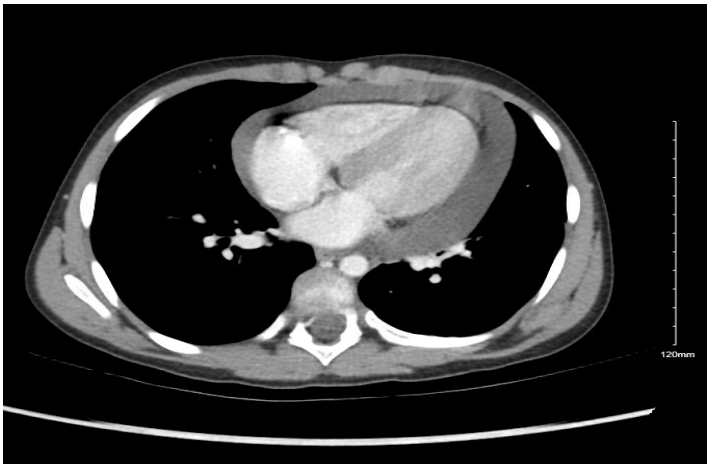


Figure 3: CT scan demonstrating large pericardial effusion

The working differential diagnosis included viral pericarditis, an unidentified autoimmune process, tuberculosis (TB), and malignancy. Antinuclear antibody (ANA) was drawn and was positive (1:80 with a speckled pattern). Thyroid stimulating hormone (TSH), free thyroxine (fT4), C3 complement, C4 complement, extractable nuclear antigen (ENA) panel, and creatinine kinase (CK) were also drawn and were all unremarkable. The QuantiFERON-TB Gold assay was found to be positive without other evidence of tuberculosis (TB) on the chest x-ray or CT. She was started on RIPE (rifampin, isoniazid, pyrazinamide, and ethambutol) therapy as well as ibuprofen and colchicine; however, the ibuprofen and colchicine were later discontinued due to potential interactions with TB treatment.

The patient was subsequently taken for cardiac catheterization, during which a pigtail drain was placed in the pericardial sac. Fifteen hundred milliliters (mL) of pericardial fluid was drained over the following 24 hours. The pericardial fluid was found to be exudative with a cholesterol level of 63 mg/dL and a triglyceride level of 431 mg/dL. Aerobic, anaerobic, and fungal cultures of the fluid were all negative, and the pericardial fluid was also negative for malignant cells. A purified protein derivative (PPD) skin test for tuberculosis was performed and was negative at 72 hours, despite the positive QuantiFERON-TB Gold test. A nasogastric tube (NGT) was placed for gastric lavage over three days, and all TB smears and cultures were negative. Adenosine deaminase was also found to be normal twice. The patient's mother, father, and brother were tested for TB and were negative.

Further workup was then performed, including rheumatoid factor (RF), antineutrophil cytoplasmic antibody (ANCA), anti-double stranded DNA (dsDNA), phospholipid panel, and immunoglobulin levels including IgA, IgG, and IgM, which were all within the normal range (WNL). Inflammatory markers were rechecked and remained WNL. T and B cell enumeration was drawn and was significant for low absolute CD4 T cell count (363/mm^3), and B cell count (CD19, 242/mm^3) significantly decreased. HIV serology was negative. Values can be seen in (Table 1).

Laboratory Test	Value	Normal Range
Rheumatoid factor (RF)	<15 IU/mL	<30 IU/mL
Antineutrophilic cytoplasmic antibody (ANCA) IFA screen	<1:20 titer	<1:20 titer
ANCA proteinase 3 antibody	<0.2 AI	<1.0 AI
ANCA myeloperoxidase antibody	<0.2 AI	<1.0 AI
Anti-double stranded DNA (dsDNA)	1 IU/mL	<5 IU/mL
Anticardiolipin immunoglobulin G (IgG)	<2.0 APL-U/mL	<20.0 APL-U/mL
Anticardiolipin IgM	<1.6 GPL-U/mL	<20.0 GPL-U/mL
Anticardiolipin IgA	<1.5 MPL-U/mL	<20.0 MPL-U/mL
Beta-2 glycoprotein IgA	<2.0 APL-U/mL	<20.0 APL-U/mL
Beta-2 glycoprotein IgG	<1.4 GPL-U/mL	<20.0 GPL-U/mL
Beta-2 glycoprotein IgM	<1.5 MPL-U/mL	<20.0 MPL-U/mL
CRP	0.03 mg/dL	<0.50 mg/dL
ESR	6 mm/h	3-13 mm/h
Total T (CD3)	59% of lymphs	60-76% of lymphs
Total T abs	591/mm(3)	1200-2600/mm(3)
Total B (CD19)	26 % of lymphs	13-27% of lymphs
Total B abs	265/mm(3)	270-860/mm(3)
Helper (CD4)	32% of lymphs	31-47% of lymphs
CD4 helper abs	315/mm(3)	650-1500/mm(3)
Suppressor (CD8)	18% of lymphs	18-35% of lymphs
Suppressor abs	183/mm(3)	370-1100/mm(3)
Natural killer	13% of lymphs	4-17% of lymphs
Natural killer abs	130/mm(3)	100-480/mm(3)
Help/Sup Ratio	1.7	-
Human immunodeficiency virus (HIV) 1&2 antibody & antigen	Non detected	Non detected

Table 1: Further laboratory workup

Repeat echocardiogram continued to show pericardial effusion without tamponade. The pigtail drain was removed five days after placement, and a repeat x-ray was performed two days later showing re-accumulation of her pericardial effusion. The patient was discharged on a low-fat diet (<5 g/day) with instructions to continue furosemide, RIPE therapy, and famotidine for stress ulcer prophylaxis.

Around two months later, dynamic contrast-enhanced magnetic resonance lymphangiography (DCMRL) was performed at a larger academic institution, which was significant for a mildly dilated abnormal lymphatic channel coursing anteriorly from the upper third of the duct toward the lower neck with subsequent enhancement of dysplastic lymphatic channels in the supraclavicular fossae bilaterally, lower neck and upper to mid mediastinum, as well as eventual contrast accumulation within the pericardial sac indicative of a leak (Figure 4).

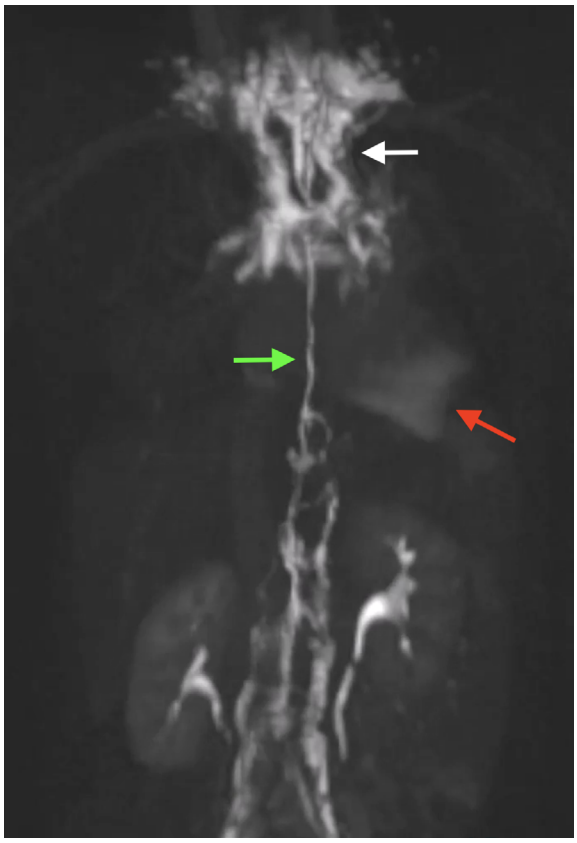
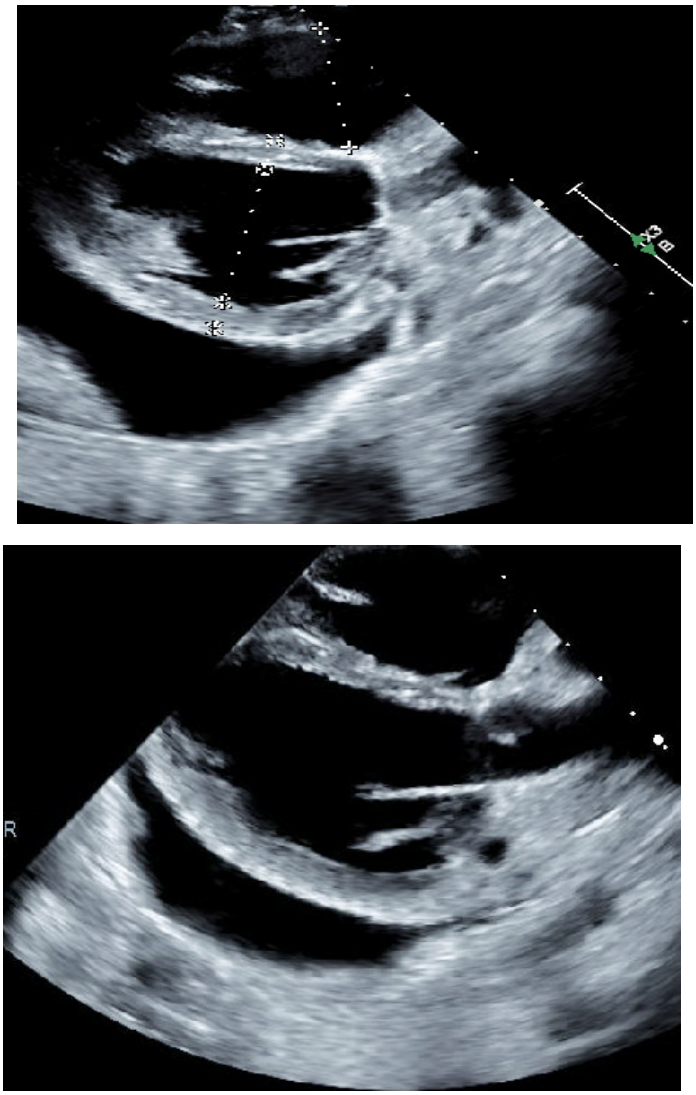


Figure 4: DCMRL demonstrating aberrant thoracic duct (red arrow = contrast in the pericardial sac, green arrow = normal thoracic duct, white arrow = abnormal upper mediastinal lymphatic perfusion).

The patient was subsequently scheduled for embolization. Serial TTEs were performed showing pericardial effusion without tamponade leading up to the procedure, which occurred approximately one month after MRL. Nontarget embolization of the lymphatic system was performed with lipiodol injection via bilateral inguinal lymph nodes with pericardial drain placement in the same setting. Post-procedure TTE revealed a small posterior pericardial effusion. Approximately two weeks later, a repeat echocardiogram was performed, which showed re-accumulation of a moderate circumferential pericardial effusion increased from the post-procedure TTE but decreased from prior (Figures 5-6).



Figures 5-6: Pre-embolization echocardiogram followed by echocardiogram two weeks later demonstrating reduced re-accumulation of fluid.

The patient subsequently underwent repeat conventional lymphangiogram with successful cannulation of the thoracic duct and placement of a pericardial drain. The abnormal dilated lymphatic channel in the upper mediastinum was again identified, and embolization of this channel was accomplished with n-butyl

cyanoacrylate (n-BCA) glue. Following selective embolization, repeat contrast injection in the thoracic duct showed several diminutive lymphatic channels arising from the midportion of the thoracic duct resulting in continued abnormal mediastinal lymphatic perfusion. These ducts were too small to cannulate, so thoracic duct embolization was performed (Figure 7).

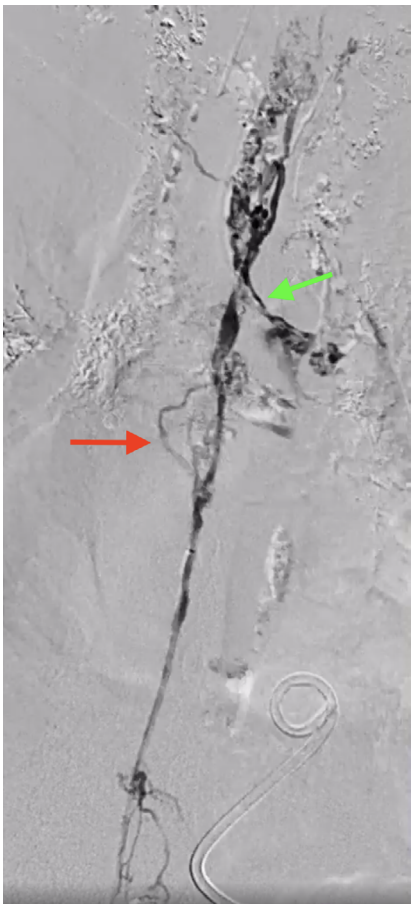


Figure 7: Digital subtraction angiogram showing a glue cast in the abnormal upper mediastinal lymphatic channel (green arrow) and additional abnormal lymphatic channels arising from the mid thoracic duct (red arrow).

The patient was admitted to the intensive care unit (ICU) post-procedure (institutional standard following lymphatic embolization) and did well. Pericardial drain output was minimal, and the drain was removed on post-procedure day 2. The patient subsequently developed a low-grade fever, which was treated with a 5-day course of cephalexin. She was discharged home on post-procedure day 4. Follow-up echocardiogram showed no pericardial effusion (Figure 8).

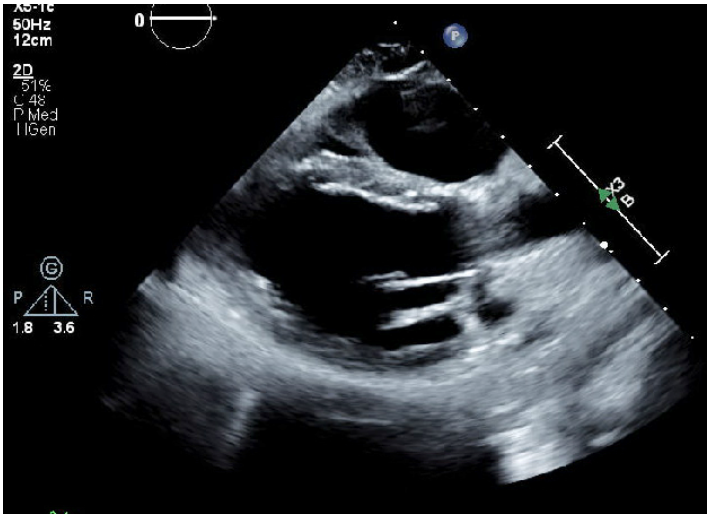


Figure 8: Echocardiogram two weeks post-final embolization demonstrating resolution of pericardial effusion.

Throughout this time, RIPE therapy was de-escalated to rifampin and isoniazid only for the treatment of latent TB infection (LTBI), as the aberrant thoracic duct was found to be the cause of her chylopericardium. The patient has been monitored with serial echocardiograms, with the most recent showing a tiny/small pericardial effusion.

Discussion

In this report, we present a rare case of idiopathic chylopericardium in a 7-year-old female with LTBI. Idiopathic chylopericardium was found to be secondary to congenital aberrant lymphatic channels draining into the pericardial space, and she was successfully treated with thoracic duct embolization. Overall, it is estimated that the prevalence of pericardial effusion in the general population is up to 6.5% [10]. Less than 3% of identified pericardial effusions are found to be chylopericardium, with only about half of these being idiopathic in etiology [11,12]. The first instance of idiopathic chylopericardium recorded in literature was in 1888 and was first described in detail by Dr. Groves in 1954, and it continues to be an exceptionally rare diagnosis [5, 11, 13]. A contributing factor to the scarcity of literature describing this condition is likely the fact that up to 40% of individuals with chylopericardium are asymptomatic, and patients who do have symptoms typically do not show them until roughly 70 months after the initial onset of effusion [14,15]. A systematic review of cases of chylopericardium showed that occurrence did not lean in favour of one gender, and the age of diagnosis ranged from 18 to 68 years with a mean age of 36 years [9]. In the paediatric population, secondary chylopericardium can be caused by systemic infection,

malignancy including lymphoma, and most commonly trauma or surgery [16]. Idiopathic chylopericardium can rarely be caused by thoracic lymph vessel anomalies as is seen in our case [17,18].

It is known that chylopericardium can be caused by mediastinal tuberculosis; however, there are no known case reports of idiopathic pericardium in patients with latent tuberculosis [3]. Since our patient did not have a history of TB and her CT was negative for parenchymal findings of current or past TB, we ruled out this etiology as a cause of chylopericardium and treated the patient for LTBI. Treatment of idiopathic chylopericardium varies. Conservative management such as a diet favouring medium-chain triglycerides over long-chain triglycerides, serial pericardiocentesis, or somatostatin infusion has demonstrated limited long-term efficacy, with roughly two-thirds of patients failing to respond [19]. While pericardiocentesis alleviates symptoms, the recurrence of chylopericardium might necessitate repeated procedures. Additionally, utilizing a pericardial drain during a patient's clinical stay results in substantial chyle loss (100-1500 mL/day), potentially causing nutritional deficiencies and immunodeficiency in chronic cases [20]. Integrating a medium-chain triglycerides (MCTs) diet has emerged as a crucial non-invasive management strategy. MCTs bypass intestinal lacteals that are directly absorbed into the portal system, thereby reducing the postprandial increase in lymphatic flow and decreasing pericardial chyle accumulation in select cases. Notably, there was a decrease in effusion size with this diet in two cases, one involving a 5-year-old female and another a 29-year-old female, with both exhibiting aplasia of the thoracic ducts leading to chylopericardium. However, it should be noted that the authors of these cases recommended considering ligation as a definitive treatment due to the continued presence of pericardial effusions [20,21]. Early surgical intervention emerged as a more effective option due to the low success rate of conservative management with associated risks including pericarditis and cardiac tamponade [2,7]. Most authors propose an initial conservative approach of as little as two weeks before considering surgery (thoracic duct ligation) or minimally invasive therapy (percutaneous lymphatic embolization).

In one case, a 15-year-old female with chylopericardium caused by a lymphatic vessel anomaly that was unresponsive to conservative management underwent thoracic duct ligation at 23 years of age, which successfully resolved the effusion. However, one year later, subsequent pericardiectomy and mitral valve repair became necessary to address pericarditis-induced fibrosis, underscoring the risks associated with chronic uncontrolled disease [9]. In another study of 49 patients who underwent surgical thoracic duct ligation, recurrence was reported in only 2 of 49 cases. This ligation approach involved left or right thoracotomy followed by thoracic duct dissection and ligation near the diaphragm [2]. Another case series of four patients who underwent video-assisted thoracic surgery targeting ligation of the thoracic duct just above the diaphragm illustrated favourable outcomes and a low recurrence rate, with a mean hospital stay of seven days and no recurrence at 24 months [13].

Current methods employ a less invasive approach that involves catheterization of the cisterna chyli/thoracic duct, similar to the one employed in our patient. This typically involves first identifying abnormal anatomy via DCMRL, followed by catheterization of the thoracic lymphatic system and subsequent targeted or non-targeted embolization. Access to the cisterna chyli/thoracic duct is usually transabdominal or retrograde transvenous following lymphatic contrast administration via inguinal lymph nodes. These approaches have been shown to provide similar efficacy to more invasive forms of lymphatic access and are feasible in the paediatric population [22]. In one case of a 16-year-old male with idiopathic chylopericardium, a minimally invasive approach was utilized for thoracic duct embolization, and the patient exhibited rapid recovery with no recurrence of pericardial fluid on TTE post-procedure at 2 weeks or 7 months [18]. Embolization has also been shown to be an effective treatment approach. Embolization involves deploying embolic material (typically n-BCA glue, coils, or both) directly into the thoracic duct (non-selective embolization) or into an abnormal lymphatic channel (selective embolization) [23]. One study demonstrated a 75% clinical success rate in managing nontraumatic chylous effusions, including nontraumatic chylopericardium, with clinical success being measured as no recurrence of effusion [17]. Embolization with access via catheterization was ultimately the most effective treatment for our patient, with resolution of pericardial effusion on postoperative imaging. In patients with iatrogenic chylopericardium, octreotide, a somatostatin analogue, reduces lymph production and has demonstrated promising outcomes in managing post-surgical chylothorax. One study reported complete resolution of postoperative chylopericardium in a 46-year-old woman following a 2-week subcutaneous octreotide regimen of 100 µg three times daily (TID) [24]. Due to the scarcity of reported cases and even less paediatric data, we were unable to find sources detailing the use of octreotide in idiopathic chylopericardium [24].

Conclusion

Idiopathic chylopericardium is rare, and clinicians should have a high level of clinical suspicion when other causes have been ruled out. For idiopathic (primary) chylopericardium, there are several challenges in both diagnosis and treatment that make the clinical course long and arduous. Once the determination of chylopericardium is established by echocardiogram and pericardiocentesis with fluid evaluation, an extensive work-up is required to rule out a secondary cause such as infection or malignancy. If no obvious cause is found, it is important to obtain MRL to look for lymphatic anomalies that could lead to

chylopericardium. Conservative management is usually not successful. Definitive treatment involves thoracic duct ligation or embolization. This case raises awareness of the diagnosis of idiopathic chylopericardium and highlights the importance of MRL in diagnosis, as well as the variation in treatment strategies. Further research needs to be done on the embryology leading to lymphatic anomalies resulting in chylopericardium, as well as the potential association with latent tuberculosis.

References

1. Luo X, Zhang Z, Wang S, Gu X, Wang X (2018) Chyloptysis with chylopericardium, a rare case and mini-review. BMC Pulmonary Medicine, 18:21.
2. Yu X, Jia N, Ye S, Zhou M, Liu D (2018) Primary chylopericardium: A case report and literature review. Exp Ther Med.15: 419-425.
3. Rajagopala S, Kancherla R, Ramanathan RP (2018) Tuberculosis-Associated Chylothorax: Case Report and Systematic Review of the Literature. Respiration. 95: 260-268.
4. Han Z, Li S, Jing H, Liu H (2015) Primary idiopathic chylopericardium: a retrospective case series. Boston Medical Center Surgery. 15: 61.
5. Lazaros G, Vlachopoulos C, Lazarou E, Tsioufis K (2021) New Approaches to Management of Pericardial Effusions. Current Cardiology Reports. 23:106.
6. Akamatsu H, Amano J, Sakamoto T, Suzuki A (1994) Primary chylopericardium. Annals of Thoracic Surgery. 58:262-6.
7. Dib C, Tajik AJ, Park S, Kheir ME, Khandieria B, et al. (2008) Chylopericardium in adults: a literature review over the past decade (1996-2006). J Thorac Cardiovasc Surg.136:650-6.
8. Uchikawa T, Ohtani K, Muramatsu K, Sonoda H, Shiose A, et al. (2018) Constrictive Pericarditis and Worsening Mitral Annular Disjunction After Long-Term Chylopericardium. Circulation Heart Failure Journal. 11:e004698.
9. Wurnig PN, Hollaus PH, Ohtsuka T, Flege JB, Wolf RK (2000) Thoracoscopic direct clipping of the thoracic duct for chylopericardium and chylothorax. Annals of Thoracic Surgery. 70:1662-5.
10. Argulian, Edgar. Vogel, Birgit. Evaluation of Pericardial Effusion, British Medical Association Journal. ISSN: 1756-1833
11. Rochefort MM. (2022) Review of chylopericardium. Mediastinum. 6:3.
12. Bewick DJ, Johnstone DE, Landrigan PL. (1984) Primary chylopericardium associated with allergic alveolitis. Canadian Medical Association Journal. 130:1577-1579.
13. Dib C, Tajik AJ, Park S, Kheir MEL, Khandieria B, et al. (2008) Chylopericardium in adults: A literature review over the past decade (1996–2006), The Journal of Thoracic and Cardiovascular Surgery, 136:650-6.
14. Tan JX, Fu Y, Chen J (2013) Primary idiopathic chylopericardium: a case report. Cardiology in the Young. 23:773-5.
15. Yüksel M, Yildizeli B, Zonüzi F, Batirel HF (1997) Isolated primary chylopericardium. European Journal of Cardiothoracic Surgery. 12:319-21.
16. Densupsoontorn NS, Jirapinyo P, Wongarn R, Thamonsiri N, Nana A, et al. (2005) Management of chylothorax and chylopericardium in pediatric patients: experiences at Siriraj Hospital, Bangkok. Asia Pacific Journal of Clinical Nutrition. 14:182-7.
17. Haddad RN, Dautry R, Bonnet D, Malekzadeh-Milani S (2023) Transvenous retrograde thoracic duct embolization for effective treatment of recurrent plastic bronchitis after fontan palliation. Catheter Cardiovascular Interventions. 101:863-869.
18. Rivera-Beltrán S, Ortíz VN, Díaz R, Hernández JA (2013) Transabdominal ligation of the thoracic duct with pericardial-peritoneal shunting in a case of primary idiopathic chylous pericardial effusion. Journal of Pediatric Surgery. 48:1434-7.
19. Svedjeholm R, Jansson K, Olin C (1997) Primary idiopathic chylopericardium--a case report and review of the literature. European Journal of Cardiothoracic Surgery. 11:387-90.
20. Mewis C, Kühlkamp V, Sokiranski R, Karsch KR (1997) Primary chylopericardium due to partial aplasia of the thoracic duct. European Heart Journal. 18:880-1.
21. Valentine VG, Raffin TA. (1992) The management of chylothorax. CHEST Journal. 102:586-91.
22. Srinivasan A, Smith C, Krishnamurthy G, Escobar F, Biko D, et al. () Characterization and treatment of thoracic duct obstruction in patients with lymphatic flow disorders. Catheter Cardiovascular Interventions. 101:853-862.
23. Nadolski GJ, Itkin M (2013) Thoracic duct embolization for nontraumatic chylous effusion: experience in 34 patients. CHEST Journal. 143:158-163.
24. Szabados E, Toth K, Mezosi E (2011) Use of octreotide in the treatment of chylopericardium. Heart & Lung: The Journal of Cardiopulmonary and Acute Care. 40:574-5.