# Infected Chylopericardium: An Unexpected Cause of Cardiac Tamponade

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**Abstract**

A 22-year-old immunocompetent female with a history of small pericardial effusion while infant presented with fever and hemodynamic collapse four days after facial trauma. She was found to have cardiac tamponade secondary to infected chylopericardium from bacterial translocation. We report this very unusual case and review of the literature on chylopericardium infections.

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**Abstract:**

A 22-year-old immunocompetent female with a history of small pericardial effusion while infant presented with fever and hemodynamic collapse four days after facial trauma. She was found to have cardiac tamponade secondary to infected chylopericardium from bacterial translocation. We report this very unusual case and review of the literature on chylopericardium infections.

**Key words:**

- Pericardial Effusion
- Pericarditis
- Chylopericardium
- Tamponade

**Introduction:**

Chylopericardium is a pericardial effusion comprised of chyle carried by the thoracic duct from the intestinal tract to the bloodstream. Macroscopically, it appears milky white and opaque given its high triglyceride content. Its etiology is subdivided into primary (idiopathic) or secondary to trauma, recent thoracic or cardiac surgery, or due to congenital abnormalities such as lymphangiomatosis. Furthermore, pericardial analysis showing a high triglyceride level greater than 500 mg/dL, a cholesterol/triglyceride ratio less than

1, lymphocyte predominance, and negative culture confirms its diagnosis and allows for its differentiation with cholesterol pericarditis or purulent pericardial effusion. To our knowledge, a superimposed bacterial infection of a chylopericardium has not been described in the literature; although it seems to adequately respond to similar management to purulent effusions, associated progression to chronic pericarditis and rate of recurrence are yet to be elucidated.

**Case report:**

A 22-year-old immunocompetent female with a remote history of chronic right jaw osteomyelitis requiring reconstruction at age of 6 years old, and mild stable pericardial effusion first diagnosed at the age of 11, presented to our institution complaining of acute onset of fever, sharp chest pain and shortness of breath following a blunt left facial trauma four days prior.

The physical exam was remarkable for tachycardia and muffled heart tones. ECG showed diffuse concave ST-segment elevation (Figure 1 A). Complete blood count revealed leukocytosis (20.800/µl. with 86% neutrophilia). Given her otherwise stable vital signs a CT chest without contrast was performed demonstrating a large pericardial effusion in the absence of intrathoracic tumors; soon after, she became hemodynamically unstable, and a bedside transthoracic echocardiogram confirmed a large pericardial effusion with signs of cardiac tamponade (Figure 1 C-D) requiring emergency pericardiocentesis.

Using a Micropuncture technique (ref) access to the pericardial sac was obtained and a standard 8.5Fr pericardial drain was used to remove close to 900 ccs of brown thick and milky pericardial fluid (Figure 1 E). The fluid analysis demonstrated: a triglyceride level of 1298 mg/dL, cholesterol level of 103 mg/dL, cholesterol/triglyceride ratio of less than 1, absent cholesterol crystals, cytology was notable for lymphocytic predominance and negative for the presence of neoplastic cells. Microbiological analyses were significant for Streptococcus dysgalactiae subspecies equisilimis (SDSE), which is considered part of the normal oral, skin, and soft tissue flora[2]. In addition, the fluid analysis was negative for fungal microorganisms and acid-fast bacilli. She was treated using ceftriaxone 2g IV twice a day based on antimicrobial susceptibilities. The pericardial drain was kept for 4 days with a significant reduction in daily drainage and no accumulation of effusion on echocardiogram. However, the day after drain removal, she developed rapid re-accumulation of pericardial fluid, for this reason, a subxiphoid pericardial window was performed; severe pericardial inflammation was found, with loculated effusions that were drained. A sample of the pericardium was sent for pathology, which came back negative for malignancy.

After 10 in-hospital days, echocardiogram findings normalized, and the patient was discharged home to continue outpatient IV antibiotics for 4 weeks along with aspirin and oral colchicine therapy for 3 months. Although during the first six months of close follow-ups it seemed like the patient was directed towards a symptomatic, incessant, and chronic phase of pericarditis with thickening of the parietal pericardium by echocardiography; she responded well to a short course of oral prednisone. At the one-year follow-up, the patient remained asymptomatic with no further clinical symptoms or echocardiographic evidence of recurrent pericardial effusion.

**Discussion**

Pericardial effusions are usually associated with non-specific symptoms, yet a significant proportion of patients are asymptomatic, and its diagnosis constitutes an incidental finding[3] unless accompanied by acute pericarditis presenting with pericardial chest pain, pericardial rubs, and new widespread ST-elevations or PR depressions on ECG[1]. Cardiac tamponade physiology develops when the ability of the pericardium to stretch is surpassed by the speed of pericardial fluid accumulation.

When a large or rapidly evolving pericardial effusion is detected, it is pivotal to assess, hemodynamic significance, and associated diseases. In small, incidental, and asymptomatic effusions, conservative management is an appropriate initial approach, with drainage needed in cases of diagnostic dilemma or secondary to other causes (i.e., Malignancy, chronic infections such as tuberculosis). Drainage has also been suggested for cases non-responsive to medical treatment, or patients with large chronic and recurrent effusions, to

prevent potential complications such as infection or constriction[4]. Given the life-threatening consequences of cardiac tamponade, its treatment involves immediate drainage of the pericardial fluid, preferably by needle pericardiocentesis except in the case of purulent pericarditis where the surgical approach is indicated[5]. In cases associated with acute pericarditis, the drug choice is nonsteroidal anti-inflammatories (NSAIDs). Glucocorticoids should be used in low doses as a second line if NSAIDS fail, in patients with contraindications to NSAIDs, or in patients with associated comorbidities such as renal failure, pregnancy, or systemic inflammatory disease. [6,8]. Adjunctive low-dose colchicine helps reduce recurrence [7]. Unless pericardial tuberculosis.

Among its causes, chylopericardium is a rare entity of pericardial effusion that can be primary (idiopathic), accounting for 3% of non-traumatic chylothorax; and more commonly secondary to trauma, malignancy, thoracic surgery, infection such as tuberculosis, congenital lymphangioleiomyomatosis or lymphangiectasia, and less commonly due to thrombosis of the vena cava or subclavian vein. Pericardial fluid analysis usually shows a milky appearance with high triglyceride levels, cholesterol to triglyceride ratio of less than one, lymphocytic predominance, and negative cytology for malignancy. Clinically chylopericardium can variate from an asymptomatic patient to signs of cardiac tamponade. It has a documented prevalence of 0.22% and 0.5% in post-operated pediatric patients, it is not well documented in the adult population

Purulent effusion is usually manifested as a febrile disease that should be managed aggressively, if untreated carries a high rate of mortality, intravenous empiric antibiotics, and urgent drainage is crucial[5]. Purulent effusions are often heavily loculated and as such, subxiphoid pericardiotomy should be considered. It is differentiated from chylopericardium by the neutrophilic predominance on cellular content, significantly lower triglyceride levels, and causative agent identified on culture. In our case, we hypothesize that the patient had primary chylopericardium which has been stable on multiple echocardiograms, and given her recent history of facial trauma, oropharyngeal bacteria translocation led to hematogenous dissemination into the pericardial sac, as no other sources of infection were identified. Non-hematological spread, such as Ludwig Angina (spreading to the mediastinum) was considered in the differential but ruled out with the CT scan and physical exam.

Although lymphangiography-lymphangioscintigraphy was not performed, the pericardial fluid analysis met all the criteria for chylopericardium. She was initially diagnosed with pericardial effusion at age of 6, therefore, we believe that the most probable etiology is a primary chylopericardium. Chylopericardium is taught to be bacteriostatic in nature and rarely is heard of being infected in non-immunocompromised patients. To our knowledge, this would be the first time a case of infected spontaneous chylopericardium is described. Superinfection of this fluid, confirmed by cultures growing SDSE, caused her acute illness.

SDSE is an emerging human pathogen closely related to Streptococcus pyogenes. Although SDSE is regarded as less virulent, an upsurge incidence of cases with severe clinical manifestations has been documented [9]. Given its rarity, there is no evidence-based data to guide the management of this entity, however in our experience, it seems to adequately respond to similar management to purulent effusion, respondent to IV antibiotics and anti-inflammatory agents, nevertheless, its associated outcomes and complications are yet to be elucidated.

**Conclusion:**

To our knowledge, this is the first reported case in the available medical literature of a chylopericardium with superimposed bacterial infection, successfully managed with antibiotics, surgical drainage, and prolonged use of anti-inflammatories. Although sharing similarities with purulent effusion, outcomes, and complications are yet to be elucidated.

**Disclosures:** Authors have no relationships with the industry. This work is not under consideration in any other journal.

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**Figures:**

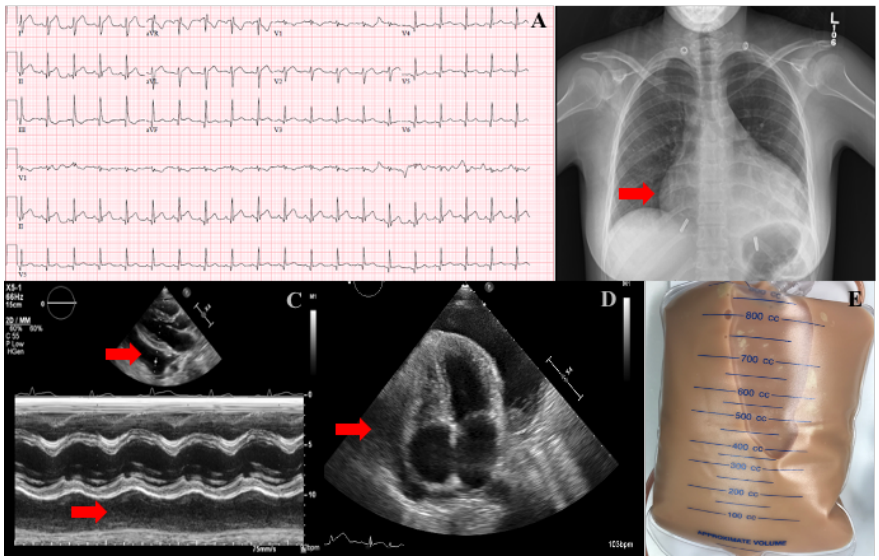


**Figure:** (A) 12 lead ECG depicting widespread concave ST-segment elevation (B) Chest XR showing

globular enlargement of cardiac shadow (C) M-mode echocardiogram demonstrating pericardial effusion (arrows) D) 2D echocardiogram, four-chamber view with right ventricular diastolic collapse (E) Milky brown pericardial fluid appearance.