

Late Cardiac Tamponade by Chylous Pericardial Effusion after Coronary Artery Bypass Surgery: Case Report

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ABSTRACT

Chylopericardium is an uncommon but serious complication after open heart surgery that often necessitates surgical treatment. We describe a case of continuous and severely symptomatic chylous pericardial effusion after coronary artery bypass grafting in which the diagnosis was established by lipid electrophoresis. Initial conservative management failed, and ligation of the thoracic duct and pericardial fenestration were finally required for a successful outcome.

CASE REPORT

A 74-year-old man with previous myocardial infarction was accepted for semiurgent CABG because of unstable angina pectoris. The angiogram revealed left main stenosis and proximal stenoses in the left anterior descending coronary artery (LAD) and the circumflex artery. The left ventricular ejection fraction was reduced to 0.30 to 0.35. The patient also had a history of alcohol abuse, with signs of moderate liver cirrhosis apparent in an abdominal ultrasound examination.

The patient underwent on-pump CABG, with a pedicled left internal mammary artery (LIMA) graft to the LAD and saphenous vein grafts to a diagonal branch and an obtuse marginal branch carried out in a routine fashion. The LIMA was harvested in a normal manner with no obvious damage to adjacent structures. The anterior mediastinal fat and thymic tissue was divided by cautery. There was no extensive dissection of the pericardium or behind the ascending aorta, and tapes around the great vessels were not used. The initial postoperative course was uneventful, with stable hemodynamics and a temporary episode of atrial fibrillation. There were no signs of perioperative myocardial infarction, infection, or a significant rise in liver enzymes. After 7 days, the patient was discharged to the referring hospital on furosemide, spironolactone, metoprolol, enalapril, aspirin, and simvastatin.

A transthoracic echocardiography evaluation performed 10 days postoperatively showed excess pericardial effusion, and a percutaneous pericardial drain was inserted. For the next 3 days, 1500 mL of yellowish, mildly sanguineous fluid was drained from the pericardium; the flow subsequently decreased from 400 to 500 mL daily to 30 mL daily. The drain was removed, and the patient was discharged again with plans for a prompt follow-up echocardiogram. Two days later, however, he was brought to the emergency room for severe dyspnea, a heart rate of 35 beats/minute and a blood pressure of 85/50 mm Hg. An emergency echocardiography examination showed cardiac tamponade, and a new pericardial drain was inserted. During the following 2 weeks, there was a continuous daily drainage of 500 mL despite diuretic and high-dose steroid treatment plus intravenous albumin supplementation. There were no signs of a general infection, and cultures of the clear and yellowish fluid were negative. The hemoglobin count was only 15 g/L in the fluid, and a cytologic examination revealed the presence of lymphocytic cells, but no malignant cells. Because of the initial suspicion of postcardiotomy pericarditis, a pericardial fenestration procedure was performed through a small left

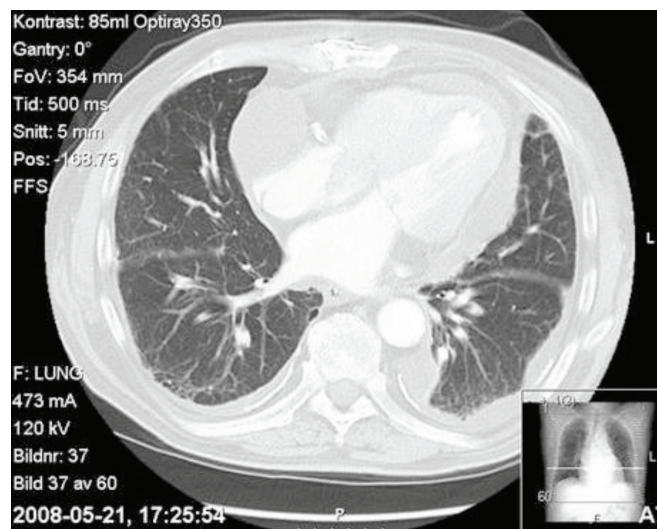


Figure 1. Computed tomography scan revealing a large pericardial effusion causing cardiac tamponade.

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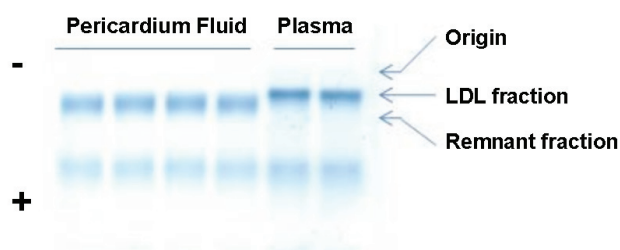


Figure 2. Lipid electrophoresis of pericardial fluid and plasma. A remnant fraction in the pericardial fluid with the mobility of a very low-density lipoprotein strongly supported chylous involvement. LDL, low-density lipoprotein.

anterolateral incision, which relieved the patient's symptoms. The patient was discharged again with a minimal residual pericardial effusion and normal laboratory test results, including those for electrolytes, hemoglobin, and liver enzymes.

A week later, a computed tomography scan demonstrated the recurrence of pericardial fluid of 4 to 7 cm that affected right atrial and ventricular filling (Figure 1). Minimal fluid in the left pleura indicated a failure of the fenestration procedure, and a new pericardial drain yielded continuous drainage of 500 mL/day. At this point, a lipid analysis of the pericardial fluid was performed on the suspicion of chylous involvement. The levels of cholesterol, triglycerides, and apolipoproteins were analyzed with routine methods used at the Karolinska University Hospital laboratory. Lipoproteins were separated by electrophoresis on buffered (pH 7.5) agarose gels (Hydragel 7 LIPO + Lp(a); Sebia, Evry, France), followed by staining with Sudan black with the semiautomated Hydrasys instrument (Sebia). The Table presents the concentrations of triglycerides, cholesterol, and apolipoproteins measured in pericardial fluid and plasma. The cholesterol-triglyceride ratio in plasma was different from that in pericardial fluid (1.30 and 0.99, respectively). The lipoprotein separation showed an absence of chylomicrons in both pericardial fluid and plasma, but a clear remnant lipoprotein fraction with a distinct mobility difference was identified in the pericardial fluid. Conversely, the plasma had a lipoprotein fraction with the electrophoretic mobility of low-density lipoprotein (Figure 2).

Because these findings strongly suggested a chylous pericardial effusion and because the patient had experienced a 20-kg weight loss, further treatment consisted of restricted oral intake of water only, total parenteral nutrition, and somatostatin therapy for 2 weeks. The patient showed no signs of improvement, however. The underlying pathology was assumed to be an iatrogenic injury to lymphatic vessels with subsequent leakage into the pericardium. A lymphangiography evaluation could not be performed at our institution, however, and the exact location of the presumed fistula therefore could not be determined.

Finally, at 2.5 months after the CABG operation, the patient underwent reoperation through a right-sided posterolateral thoracotomy, with open ligation of the thoracic duct in the posterior mediastinum adjacent to the diaphragm.

Levels of Apolipoproteins in Pericardial Fluid and Plasma

	Pericardial Fluid	Plasma (Reference Interval)
Triglycerides, mg/dL	62.0	77.1 (40-230)
Cholesterol, mg/dL	61.9	100.5 (150-301)
Apolipoprotein B, g/L	<0.35	0.52 (0.50-1.70)
Apolipoprotein E, mg/L	21	28 (23-63)
Apolipoprotein A-I, g/L	0.64	1.03 (1.10-1.80)

In addition, a wide fenestration of the pericardium was performed over the right heart. The thoracic duct was easily identified as a whitish structure by giving cream to the patient through the nasogastric tube approximately 30 minutes before skin incision. We continued fast and total parenteral nutrition early in the postoperative period and observed a subsequent decrease in pericardial fluid drainage, despite the patient having been allowed to eat after 4 days. The patient's subsequent course was uncomplicated, and echocardiography and chest radiography evaluations showed a marked reduction in fluid accumulation. Biopsies of the pericardium from the final operation showed no signs of mesothelioma or other malignancy. The patient was discharged in good condition after 11 days and remained asymptomatic during follow-up. After 6 months, his x-ray was normal, and he was doing well.

DISCUSSION

Chylopericardium secondary to open heart surgery is a very rare complication and has been described in only a few case reports to occur after coronary bypass, valve, and atrial septal defect procedures [Sharpe 1999; Kumar 2004; Sachithanandan 2008]. Suggested etiologies are iatrogenic disruption of mediastinal lymph vessels when dissecting the fat and thymic tissue anteriorly, or the pericardium posteriorly (ie, snaring the aorta or venae cavae). Another possible site of injury is to the thoracic duct proximally during LIMA harvesting. Obstruction of drainage in the lymphatic system may occur by subclavian venous thrombosis caused by central venous catheters. Chylopericardium is a feared complication, because it may lead to prolonged drainage and hospitalization, hemodynamic compromise, malnutrition, immunologic deficiency, need for repeat interventional procedures, and death [Dib 2008].

The diagnosis of chylopericardium rests on the detection of chylous fluid in the pericardium, based on results of laboratory analysis of lipid contents, cytologic findings of a lymphocytic predominance, and negative cultures. The diagnosis is usually verified by analyzing fluid appearance and triglyceride content. Classically, the fluid is milky-white with a triglyceride concentration >500 mg/dL and a ratio of cholesterol to triglycerides of <1.0 [Dib 2008]. Nevertheless, the diagnosis has also been established in cases with lower triglyceride levels (down to 181 mg/dL) [Sasaki 2002; Nanjo 2004; Dib 2008]. In patients with chylothorax, the chylous pleural effusion may be yellow or green, turbid or serous, instead of milky-white [Nair 2007].

Our case is unusual and atypical, because we had a patient with a never-ending pericardial effusion, which was clearly a hemodynamic threat. The fluid was never milky-white, and triglyceride levels were much lower than ever reported for chylopericardium. The lipoprotein electrophoretic analysis, however, excluded the presence of circulating chylomicrons in both the plasma and the pericardial fluid, suggesting that the sampling was done when the patient was not in the postprandial dyslipidemic phase. Thus, lower triglyceride levels were to be expected. Furthermore, the electrophoretic analysis demonstrated the presence of a lipoprotein fraction in the pericardial fluid with a mobility similar to that of a very low-density lipoprotein remnant. Analysis of the apolipoprotein content of the pericardial fluid also demonstrated a larger ratio of apolipoprotein E and apolipoprotein B than observed in plasma, suggesting enrichment in triglycerides and a possible intestinal origin of the lipoprotein fraction. In our case, the triglyceride levels in the pericardial fluid were similar to the cholesterol levels, and the cholesterol-triglyceride ratio was <1 . Given that cholesterol levels are normally higher than triglyceride levels in chronic inflammation, this finding made pericarditis unlikely. The combination of these lipid analysis findings, the negative cultures, and an abundance of lymphocytes in the fluid gave us no plausible explanation for this serious and long-standing complication other than chylous involvement. Conservative therapy and a limited surgical procedure failed, and the problem was finally resolved with ligation of the thoracic duct and a wider pericardial fenestration, further indicating that the patient had experienced chylous pericardial effusion.

Treatment of chylopericardium is conservative, surgical, or both. Conservative treatment involves pericardiocentesis and drainage, a medium-chain triglyceride diet or total parenteral nutrition, and somatostatin therapy. Some case reports of postoperative chylopericardium have described the success of this approach [Sharpe 1999; Sachithanandan 2008], but a wider analysis of different causes of chylopericardium found that conservative management failed in $>50\%$ of cases [Dib 2008]. No clear predictors of failure of conservative therapy exist, and many cases require subsequent surgical intervention. Indications for surgery include hemodynamic compromise,

recurrence after conservative treatment, or a leakage sufficiently large to cause metabolic and immunologic disturbances [Dib 2008]. The most commonly reported approach is ligation of the thoracic duct with the creation of a wide pericardial window to allow sufficient drainage and possibly avoid future constrictive pericarditis. The operation is performed preferably through the right chest by an open access or by video-assisted thoracoscopic surgery. Mass ligation of the thoracic duct near the diaphragm is recommended for the best results [Dib 2008], and the duct can be easily identified by preoperative administration of cream directly through the nasogastric tube after intubation, as in the present case.

In conclusion, chylopericardium after cardiac surgery is very rare but can lead to hemodynamic compromise and a prolonged postoperative course. The diagnosis may not be straightforward and should include an extensive analysis of the pericardial fluid. If conservative therapy fails, surgery with thoracic duct ligation and wide pericardial fenestration may be necessary.

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