BRIEF REPORT

# Beware the Left-Sided Effusion

*Michael W. Felz, MD, and Jenay Neely, DO*
*Augusta, Georgia*

A left-sided pleural effusion is an infrequent clinical occurrence compared with bilateral or right-sided effusions. After coronary bypass surgery, a patient presented with dyspnea and an enlarging left pleural effusion erroneously attributed to congestive heart failure and refractory to medical management. Thoracentesis generated a clinical surprise, allowed specific therapy, and produced evidence for a rarely reported complication of coronary artery bypass grafting. The management of chylothorax and the differential diagnosis of left-sided pleural effusions are reviewed.

**KEY WORDS.** Pleural effusion; coronary artery bypass; chylothorax. *(J Fam Pract 1997; 45:519-522)*

A unilateral left-sided pleural effusion presents a diagnostic challenge. Depending on the clinical setting, thoracentesis is of paramount importance in differentiating transudative (congestive heart failure, nephrosis, cirrhosis) from exudative (infection, infarction, inflammation, neoplasm, trauma) causes. While congestive heart failure (CHF) is a frequently diagnosed clinical syndrome often associated with transudative bilateral or right-sided effusions, rarely does CHF result in purely left-sided effusions.[1] We present a case of a symptomatic left-sided effusion masquerading as CHF until therapeutic thoracentesis allowed definitive, but unanticipated, diagnosis.

## Case Report

A 50-year-old white woman presented to the Medical College of Georgia (MCG) emergency department with a 1-month history of slowly worsening shortness of breath, fatigue, and weakness 2 months after coronary bypass grafting. The shortness of breath was worse on exertion, progressively limited her routine daily activities, and had become intolerable in the past week. She had moderate orthopnea that required using three pillows. The patient's primary care physician was treating the patient with digoxin and increasing doses of furosemide and aldactone for presumed congestive heart failure. There was no improvement in her symptoms, despite diuresis and weight loss.

Serial chest films demonstrated a slowly enlarging left-sided effusion. The patient denied chest pain, lower extremity swelling, cough, fever, chills, or other systemic symptoms.

*Submitted, revised, August 13, 1997*
*From the Department of Family Medicine, Medical College of Georgia, Augusta. Requests for reprints should be addressed to Michael W. Felz, MD, Department of Family Medicine Medical College of Georgia, HB 4032, Augusta, GA 30912-3500.*

Prior medical history included coronary artery disease, poorly controlled type 1 diabetes mellitus, and hyperlipidemia. She had undergone an uncomplicated 2-vessel coronary artery bypass grafting (CABG) 2 months prior, with grafts to the left anterior descending artery using the left internal thoracic artery and to the right coronary artery from the aorta using saphenous vein. The pleura was not opened for harvesting of the left internal thoracic artery. Postoperatively, a small left pleural effusion was regarded as consistent with the usual recovery phase from coronary bypass surgery. Postprocedure left ventricular ejection fraction was 47%. Chest tubes were removed on postoperative day 1 after drainage was clear and measured less than 30 cc. Her medications at the time of this admission consisted of aspirin 325 mg daily, digoxin 0.25 mg daily, furosemide 40 mg 3 times a day, spironolactone 25 mg twice a day, potassium chloride 20 mEq daily, isosorbide dinitrate 20 mg twice a day, nifedipine XL 60 mg daily, colestipol HCl 600 mg daily, and 80 units per day of insulin 70/30.

On physical examination, the patient was found to be an obese woman in moderate respiratory distress with a temperature of 98° F, a heart rate of 102 beats per minute, labored respirations of 28 breaths per minute, blood pressure of 118/70 mm Hg, and oxygen saturation of 97%. Chest examination was remarkable for absent breath sounds to the mid-left lung field with diminished tactile fremitus, dullness to percussion, and egophony. No crackles were detected. Cardiac ascultation revealed a regular rate, tachycardia, crisp heart sounds, and no gallop. A median sternotomy scar was present from past surgery.

Chest radiograph revealed a large left-sided pleural effusion to the seventh rib. Electrocardiogram showed minor nonspecific ST-T wave changes, unchanged from past tracings. Echocardiogram demonstrated an ejection fraction of 56%, with no

pericardial effusion. White blood count was 7800/mm$^3$ with a normal differential. Serum chemistries were within normal limits with the exception of elevated glucose (222 mg/dL) and triglycerides (486 mg/dL) secondary to diabetes. Amylase, lipase, and CPK were within normal limits. Digoxin level was 0.94 ng/mL.

Our initial clinical impression was refractory CHF, although the left-sided effusion was atypical. Based on our experience, we would have expected right-sided or bilateral effusions with CHF. The normal ejection fraction also was inconsistent with CHF. Indeed, during evaluation in the emergency department, one of the authors (M.W.F.) remarked, "Beware the left-sided effusion."

## MANAGEMENT

Because of the patient's respiratory embarrassment, a diagnostic and therapeutic thoracentesis was performed in the left ninth intercostal space. Anticipating the removal of transudative fluid, we were surprised to obtain thin, opaque, milky liquid (Figure). A total of 1200 cc of chylous fluid was removed within 30 minutes, with marked improvement in the patient's symptoms. A repeat chest radiograph demonstrated partial clearance of the effusion. Laboratory analysis of the fluid is displayed in the Table.

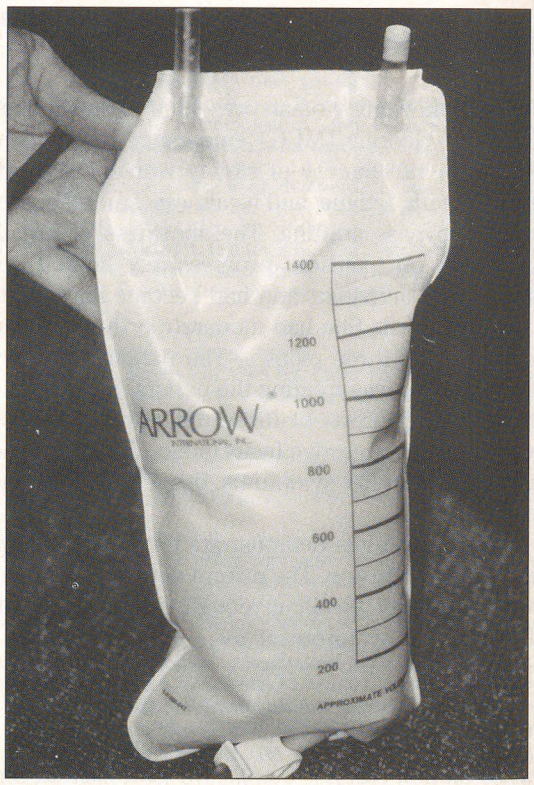
The patient was admitted to MCG hospital with a chylothorax of unknown etiology. Markedly elevated triglycerides in the exudative effusion were diagnostic of chylothorax. The thoracic surgery department was consulted promptly regarding the source of chylous fluid. The surgical opinion raised the possibility of iatrogenic leakage from the thoracic duct secondary to trauma during CABG or postoperative central line placement. Recommendations from surgical consultants were to attempt conservative management with dietary fat restriction, and to repeat therapeutic thoracentesis if the effusion recurred. Recurrent symptomatic chylothoraces, if persistent over a total postoperative observation period of 6 months, would necessitate invasive surgical ligation of the thoracic duct.

The following day, mild dyspnea recurred with partial reaccummulation of the effusion seen on chest film. Repeat thoracentesis was performed with the removal of an additional 1000 cc of chylous fluid, again with improvement in dyspnea and resolution of effusion confirmed by chest film. The patient was discharged home the following day, asymptomatic, and without recurrence of the pleural effusion for more than 24 hours, as demonstrated by radiographs.

Over a follow-up period of 2 months, the patient required five additional thoracenteses, each of progressively diminishing volume. A cumulative total of 5400 cc of chyle was removed on an outpatient basis for symptomatic relief of recurrent left-sided chylothoraces. The chylothorax then spontaneously resolved without further surgical intervention. There has been no symptomatic or radiologic recurrence over a 12-month observation period. No immunologic or nutritional sequelae of the removal of more than 5 L of chyle have been observed to date. Nutritional status, as assessed by body weight, lymphocyte count, total protein, and albumin, has likewise been uncompromised on follow-up.

**FIGURE**

**A surprising amount of chylous fluid — 1200 cc — was removed by initial thoracentesis within 30 minutes.**



## Discussion

### Literature Review

The management of postthoracotomy chylothorax is controversial.[2-3] The rarity of the syndrome, coupled with the lack of controlled studies, limits authoritative recommendations.[4-6] Some experts advocate immediate thoracotomy for thoracic duct repair or pleuroperitoneal shunting to prevent ongoing loss of chyle and eventual nutritional and immunologic deterioration. Video-assisted thoracoscopy (VAT) has recently been utilized for thoracic duct ligation,[7] fibrin occlusion,[8] clipping,[9] oversewing,[10] or talc pleurodesis.[11] Others, however, recommend conservative treatment with dietary fat restriction, total parenteral nutrition, and periodic thoracentesis or chest tube drainage for 2 to 6 weeks before resorting to operative intervention.[12-13] In an exhaustive review of 132 cases of postoperative chylothorax in the world literature spanning 50 years, Sieczka and Harvey[14] found that the majority were related to malignant thoracic disease, with only 7 (5%) associated with benign cardiovascular procedures. They pointed out a 21% mortality rate, largely because of underlying malignancy, and suggested prompt surgical repair within 2 weeks of diagnosis. Milsom et al,[15] reviewing 20 cases (16 patients were younger than 1-year-old) treated over a 10-year span at a large university, reported an 83% mortality rate for nonoperative management in this predominantly pediatric age group compared with a 14% mortality rate among those treated with thoracic duct ligation or shunting. Of the 4 adults in the series, 2 had lung carcinoma. Ferguson et al[16] documented 13 cases of chylothorax in 3589 thoracotomies (0.36% incidence) in an 8-year period. Nine of these patients had primary operations for malignant disease and 10 required reoperation for chylothorax. The mean duration of chylous leakage was 37 days. Four patients, all with cancer, died with persistent leaks. These case series indicate that chylothoraces from malignant disease of the thorax are more likely to need invasive therapy than those of benign origin.

**TABLE**

**Laboratory Analysis of Chylous Fluid from Patient with Left-sided Pleural Effusion**

| | Fluid | Serum |
|---|---|---|
| WBC, mm$^3$ | 5850 | 7800 |
| Differential, % | | |
| Segmented | 2 | 49 |
| Lymphocytes | 88 | 36 |
| Monocytes | 5 | 8 |
| Eosinophils | 0 | 7 |
| Glucose (mg/dL) | 248 | 222 |
| Amylase (U/mL) | 27 | 26 |
| LDH (U/mL) | 155 | 191 |
| Total protein (g/dL) | 6.3 | 6.2 |
| Cholesterol (mg/dL) | 79 | 226 |
| Triglycerides (mg/dL) | 2,535 | 486 |
| Gram stain | No organisms | — |
| Bacterial cultures | Negative | — |
| Cytologies | Negative | — |

### Case Commentary

In our case, conservative management with a low-fat diabetic diet and periodic thoracenteses (seven total) resulted in complete resolution of the large chylothorax within 8 weeks of initial detection, and 4 months after successful coronary bypass surgery. The specific anatomic site of chylous leakage was never determined in our patient. We speculate that, since the effusion was slow to accumulate and symptomatic compromise was delayed for 2 months after thoracotomy, a tiny leak from a traumatized minor lymphatic tributary occurred. It is conceivable that occult trauma to lymphatic collaterals during dissection of the internal thoracic artery, or during cautery of small vessels in the superior left mediastinum, could have allowed gradual accumulation of chyle and symptomatic onset at a time remote from thoracotomy. A minute lymphatic rent could also occur even during careful retraction of the left subclavian vein near the usual confluence with the thoracic duct and its collaterals. A small leak could also be potentially self-limited, as was the case in our patient, and never require operative repair. In contrast, if the main thoracic duct itself had been significantly lacerated, we believe that the chylothorax would have accumulated much more rapidly, been symptomatic sooner, and proved refractory to conservative therapy.

In retrospect, the outpatient diagnosis of CHF warranted more definitive evaluation. The absence

of unexplained weight gain, peripheral edema, crackles on lung examination, an $S_3$ gallop, and jugular venous distention should have left the diagnosis of CHF in doubt. The lack of angina or ECG evidence of recent myocardial infarction, and the normal heart size on serial chest films, were persuasive evidence against post-CABG ischemic cardiomyopathy and resultant CHF as the cause of the unilateral effusion. Finally, a timely outpatient assessment of left ventricular function by echocardiography[17] would have revealed a normal ejection fraction, as was documented at hospital admission, and rendered the diagnosis of CHF highly improbable. Documentation of normal left ventricular function in the first few weeks after CABG would have prompted more definitive investigation of the exact cause of the pleural effusion, and perhaps earlier therapeutic intervention, sooner than 2 months after bypass.

To our knowledge, this case represents the eighth report in the world literature of a patient with chylothorax following internal thoracic artery implantation for coronary artery bypass.[10,13,18-23] In the previous seven cases, four required thoracotomy for resolution while three responded to conservative management, and all survived. Because of the potential for compromise in respiratory, nutritional, and immunologic function, prompt recognition and management of chylothoraces are essential, especially when a left-sided effusion attributed to CHF fails to respond as expected. A strictly left-sided effusion, depending on the clinical situation, is far more likely to result from exudative disorders such as pneumonia, tuberculosis, pulmonary embolus, malignancy, immune-mediated syndromes, esophageal rupture, pancreatitis, subphrenic abscess, pneumothorax or chylothorax (collectively) than from a transudative syndrome such as CHF.[24] Our case is unique in that left-sided effusions are relatively uncommon, chylothorax is an unusual finding on thoracentesis, and chylous effusion is extremely rare after median sternotomy and internal thoracic artery dissection for coronary artery bypass. Furthermore, the syndrome responded to conservative therapy, and management was performed chiefly by primary care clinicians utilizing frequent consultation with thoracic surgeons. As in so much of bedside clinical medicine, a terse adage applies in this case: "Beware the left-sided effusion."

**ACKNOWLEDGMENT**

We are grateful to Dr Joseph W. Rubin for comments on surgical aspects of the text.

**REFERENCES**

1. Haselton PS. Pleural disease. In: Hazelton PS, ed. Spencer's pathology of the lung. 5th ed. New York: McGraw-Hill, 1996; 1131-1210.
2. Valentine VG, Raffin TA. The management of chylothorax. Chest 1992; 102:586-91.
3. Robinson CLN. The management of chylothorax. Ann Thorac Surg 1985; 39:90-5.
4. Cevese PG, Vecchioni R, D'Amico DF, et al. Postoperative chylothorax. J Thorac Cardiovasc Surg 1975; 69:966-71.
5. Witz JP, Roeslin N, Miech G. Chylous discharge in the thorax, with reference to 8 cases. [In French] Ann Chir Thorac Cardiovasc 1976; 15:191-8.
6. Kostiainen S, Meurala H, Mattila S, Appelquist P. Chylothorax: clinical experience in nine cases. Scand J Thor Cardiovasc Surg 1983; 17:79-83.
7. Kent RB III, Pinson TW. Thoracoscopic ligation of the thoracic duct. Surg Endosc 1993; 7:52-3.
8. Inderbitzi RGC, Krebs T, Stirnemann P, Althaus U. Treatment of postoperative chylothorax by fibrin glue application under thoracoscopic view with use of local anesthesia. J Thorac Cardiovasc Surg 1992; 104:209-10.
9. Janssen JP, Joosten HJM, Postmus PE. Thoracoscopic treatment of postoperative chylothorax after coronary bypass surgery. Thorax 1994; 49:1273.
10. Wood MK, Ullyot DJ. Chylothorax. J Thorac Cardiovasc Surg 1994; 108:1156.
11. Graham DD, McGahren ED, Tribble CG, Daniel TM, Rodgers BM. Use of video-assisted thoracic surgery in the treatment of chylothorax. Ann Thorac Surg 1994; 57:1507-12.
12. Marts BC, Naunheim KS, Fiore AC, Pennington DG. Conservative versus surgical management of chylothorax. Am J Surg 1992; 164:532-35.
13. Chaiyaroj S, Mullerworth MH, Tatoulis J. Surgery in the management of chylothorax after coronary artery bypass with left internal mammary artery. J Thorac Cardiovasc Surg 1993; 106:4:754-6.
14. Sieczka EM, Harvery JC. Early thoracic duct ligation for postoperative chylothorax. J Surg Oncol 1996; 61:56-60.
15. Milsom JW, Kron IL, Rheuban KS, and Rodgers BM. Chylothorax: an assessment of current surgical management. J Thorac Cardiovasc Surg 1985; 89:221-7.
16. Ferguson MK, Little AG, and Skinner DB. Current concepts in the management of postoperative chylothorax. Ann Thorac Surg 1985; 40:542-5.
17. Felner JM, Martin RP. Clinical application of echocardiography. In: Schlant RC, Alexander RW, O'Rourke RA, et al, eds. Hurst's The Heart. 8th ed. New York, NY: McGraw Hill Co. 1994; 398-422.
18. Davies MJ, Spyt TJ. Chylothorax and wound lymphocele formation as a complication of myocardial revascularization with the internal thoracic artery. J Thorac Cardiovasc Surg 1994; 108:1155-6.
19. Di Lello F, Werner PH, Kleinman LH, Mullen DC, Flemma RJ. Life-threatening chylothorax after left internal mammary artery dissection: therapeutic considerations. Ann Thorac Surg 1987; 44:660-1.
20. Weber DO, Del Mastro P, Yarnoz MD. Chylothorax after myocardial revascularization with internal mammary graft. Ann Thorac Surg 1981; 32:499-502.
21. Zakhour BJ, Drucker MH, Franco AA. Chylothorax as a complication of aortocoronary bypass: two case reports and a review of the literature. Scand J Thorac Cardiovasc 1988; 22:93-5.
22. Czarnecki DJ, Kehoe ME, Tector AJ. Lymphangiographic evaluation of chylothorax after myocardial revascularization [letter]. AJR 1988; 151:1255.
23. Kshetiry VR, Rebello R. Chylothorax after coronary artery bypass grafting [letter]. Thorax 1982; 37:954.
24. Connors AE Jr, Altose MD. Pleural inflammation and pleural effusion. In: Baum GL, Wolinski E, eds. Textbook of pulmonary diseases. 5th ed. Boston, Mass: Little, Brown, and Co. 1994; 1853-68.