Table. Use of Ciprofloxacin by Travelers (N = 70)

Measure Taken	Number	% of All Filled Prescriptions
Used all of the ciprofloxacin for:		
Travelers' diarrhea	2	2.9
Sore throat, fever	1	1.4
Used part of the ciprofloxacin for:*		
Travelers' diarrhea	5	7.1
Gas, bloating, or upset stomach	2	
Did not use any ciprofloxacin, but:		
Stored unused pills	40	57.1
Disposed of pills	13	18.6
Donated pills	2	
Unknown	2	
Other	3	4.3
Gave to a friend	1	
Gave to dog	1	
Lost	1	

* Of these 7 patients, 6 stored and 1 disposed of the remaining pills.

did not address the use of adjunctive treatment for travelers' diarrhea, including bismuth subsalicylate, loperamide, and probiotics. These agents were used by almost half of the patients in Hill's study, obviating the need for antimicrobial therapy in many patients and contributing to the amount of unused antibiotics (3).

The disposal of unused ciprofloxacin is a previously unappreciated aspect of this clinical problem. Studies have shown that 10% to 40% of oral antibiotics are either wasted or used in potentially dangerous ways, such as self-medication, particularly for upper respiratory tract infections (4-6). The role of travel clinics in this alarming phenomenon has not been well described. How unused medications are "disposed" needs to be addressed. Patients could be informed with useful measures, such as revised medication labels to describe intended use and disposal procedures, targeted discussion at travel clinic follow-ups, and programs to recycle unused and unexpired medications.

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CHYLOTHORAX AS THE FIRST MANIFESTATION OF CONSTRICTIVE PERICARDITIS

To the Editor:

Chylothorax is defined as an accumulation of chyle in the pleural space due to disruption of the thoracic duct or one of its major divisions (1). We describe a 35-year-old white man with a 4-month history of bilateral chylothorax who presented with recent-onset ascites following thoracic duct ligation.

The patient's past history included aortoplasty for aortic coarctation at the age of 5 years. At admission, he appeared pale and ill. The physical examination revealed jugular venous distention at the angle of the jaw (8 cm above the sternal angle) while he was sitting. Heart sounds were normal. There was massive intra-abdominal free fluid. There was slight hepatomegaly and pitting edema of the legs. Blood chemical levels were normal except for a slight elevation in alkaline phosphatase and γ-glutamyl transpeptidase levels. Tuberculin skin test was negative. Paracentesis yielded a milky fluid with the following biochemical composition: triglycerides, 1080 mg/dL; cholesterol, 89 mg/dL; total protein, 3.8 g/dL; and lactate dehydrogenase, 108 U/L. Cytological and microbiologic examinations of peritoneal fluid were unremarkable. A chest radiograph showed minimal cardiac silhouette enlargement without pericardial calcification and moderate left pleural effusion. Abdominal echography disclosed a dilated inferior vena cava, which did not change in diameter during respiration. Computed tomographic (CT) scan of the thorax revealed a 1-cm thick pericardium (Figure) with some pericardial effusion and left pleural effusion. Right and left heart catheterization displayed an early diastolic dip followed by a mid-through-late diastolic plateau of the pressures in both ventricles. Cardiac index was reduced to 1.2 L/min/m^2. Pericardiectomy showed a thick and encasing pericardium. Microscopic examination revealed nonspecific chronic pericarditis. Ascites progressively resolved, and the patient had an uneventful recovery and was asymptomatic 24 months later.

Pleural effusion may be present in constrictive pericarditis (2). In our patient, errors in diagnosis and the physical examination led to delayed treatment. After a preliminary CT scan ruled out masses as an intra-thoracic cause of lymphatic obstruction, the past cardiovascular procedure be-

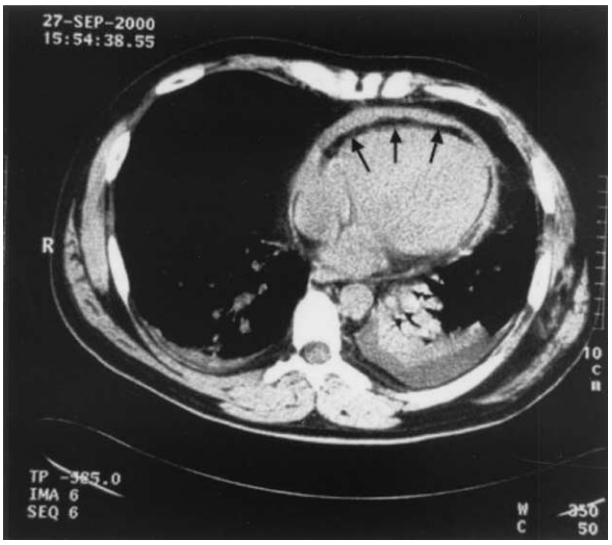

Figure. Computed tomographic scan (axial view) of the chest showing left pleural effusion and marked thickening of the pericardium (arrows).

came suspect. This assumption led to surgical ligation of the thoracic duct, which resulted in massive chyloperitoneum. Previous cardiovascular surgery is associated with pleural chylous effusion in less than 0.5% of cases (3). It is most common after resection of a coarcted aorta (4). However, in our patient there was an interval of 30 years between heart surgery and chylothorax onset, making this diagnosis very unlikely even if anatomical venolymphatic junction modification following surgical correction may have played a role in the generation of chylous effusion after high pressure development in the venous system. Experimentally induced constrictive pericarditis is followed by thoracic duct hypertension and dilation (5). The diameter of the duct may increase to up to four times its normal size and lymph flow may increase up to 12 times the normal rate in heart failure, as has been described in previous reports of peritoneal chylous effusion in heart disease and constrictive pericarditis (6). In our patient, free peritoneal fluid, which was ruled out at the time of pleural effusion onset, resulted because of the need for alternative drainage following thoracic duct ligation.

Although the etiology of constrictive pericarditis has not been established, constrictive pericarditis should be considered in the differential diagnosis of chylothorax.

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