We conclude that a considerable cost savings and a low incidence of mortality, perioperative myocardial infarction, and prolonged intensive care unit stay can be attained in low-risk patients undergoing elective coronary artery surgery without preoperative PA catheterization. The question of whether an improvement in outcome can be achieved with PA monitoring cannot be answered from these data alone, and the large number of patients required probably precludes performing a prospective, randomized, controlled study.

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Chylothorax Following Celiac Plexus Block

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Neurolytic celiac plexus block is used to relieve intractable pain in selected cases.1,2 While major complications are rare, many risks and potential problems can occur, including paraplegia.3 In one series4 weakness and/or numbness in the T-10 through L-2 distribution was the most common finding (8%), followed by lower chest pain, failure of ejaculation in men, postural hypotension, the sensation of warmth or fullness of a leg, and urinary difficulties. Additionally, aortic or inferior vena cava puncture, kidney puncture, pneumothorax, puncture of viscera, abscess or cyst, and subarachnoid or intravascular injection with attendant sequelae are recognized hazards of this technique.5–8 We describe a previously unreported potentially serious complication of this therapy.

REPORT OF A CASE

A 75-year-old man with a history of pancreatic carcinoma was referred to our Pain Clinic for assessment and management of abdominal pain of 3 months duration. The diagnosis of adenocarcinoma of the head of the pancreas was made 18 months previously, at which time obstructive symptoms were treated surgically by roux en y choledochoenterostomy. Postoperative medical management consisted of 5-
fluorouracil, doxorubicin, and mitomycin chemotherapy. There had been no clinical evidence of recurrent disease until the onset of pain in the mid-epigastrium radiating through to the back, which was exacerbated by meals and relieved by antacids or an oral oxycodone preparation.

Essential hypertension was well controlled with hydrochlorothiazide and nadolol. He was chronically anemic and had a poor appetite, although body weight had been relatively steady over the prior 3 months. There were no known medical allergies and no history of tobacco or alcohol use.

On examination the patient had no external manifestations of disease except for pallor; he was not icteric. Abdominal examination revealed diffuse tenderness in the epigastric region. The liver edge was palpable four finger breadths below the right costal margin. Otherwise, general physical examination was unremarkable.

Gastroscopy revealed mild erosive gastritis. Computed axial tomography (CT) of the abdomen revealed mild enlargement of the pancreatic head. The retroperitoneal anatomy appeared normal. Measurement of serum amylose, electrolytes, hepatic enzymes, and bilirubin were normal; hemoglobin was 8.8 g/dl; platelet count was 151,000/mm²; prothrombin time and partial thromboplastin time were normal.

A diagnostic celiac plexus nerve block was performed with the patient in the right and left lateral positions sequentially. Under image intensifier direction 12-cm 22-gauge needles with obturators were placed 7.5 cm lateral to the midline at the L-3 level and directed beneath the 12th ribs in a medial cephalad direction to rest anterior to the L-1 vertebral body; 0.5% bupivacaine with 1/200,000 epinephrine, 5 ml, was injected on each side after negative aspiration tests. There were no acute complications, and the patient had pain relief that lasted 18 h. A neurolytic celiac plexus block was subsequently performed. The same directive technique was employed with the addition of anatomic demarcation by the water-soluble contrast metrizamide before injection of 4 ml 1% lidocaine, followed by 4 ml 6.7% aqueous phenol on each side.

The patient did well with no immediate problems and obtained significant pain relief. He continued to use one to two doses of 3 mg oxycodone with 325 mg acetaminophen per day for occasional breakthrough periprandial pain upon discharge from the hospital 48 h later. Six days after discharge he presented to the emergency room with acute intermittent pain in the left shoulder and anterior chest. Evaluation revealed a pleural effusion filling one-half of the left chest for which a thoracostomy tube was placed. Analysis of the milky effusion showed a protein of 2.89 mmol/l and triglycerides 29.28 mmol/l, consistent with chylous lymph. Culture of the effusion was negative, and there were no malignant cells seen. Chest tube drainage diminished daily, and the tube was removed after 10 days. The patient was discharged 2 days later, and to date his status remains satisfactory.

**DISCUSSION**

The cisterna chyli is classically described as lying anterior to the first and second vertebrae to the right of the aorta extending a length of 5–7 cm. However, the actual specific anatomy and location is quite variable. In an individual, the structure may be completely absent or multilocular, or there may be multiple discrete cisternae. The thoracic duct originates from either lymphatic confluentes or the cisterna(e) and ascends in proximity to the vertebral column traversing the diaphragm via the aortic hiatus posterolateral to the aorta and medial to the right crus of the diaphragm at the T-12 level. This duct continues between the azygos vein and aorta, passing to the left in the mediastinum usually around the T-5 level. Finally, this duct empties into the left jugulosubclavian venous system. A needle passing anterior to the vertebral bodies could easily penetrate the lymphatic structures.

Perhaps such a retroperitoneal injury may lead to a subdiaphragmatic collection of lymph, which is then transported into the pleural cavity via diaphragmatic lymphatics. The flow is unidirectional consequent to valves within the lymphatic vessels. Perhaps the relatively negative intrathoracic and transpleural pressure gradient, augmented by pulmonary excursions, serves as an abdominal-to-thoracic pump in this setting. Regardless of actual mechanisms, physiologically, such an egress definitely takes place. Since the total flow of chyle averages 2–4 l/day leakage can result in potentially massive effusions.

The detailed anatomy of the chylous drainage system was described by Mascagni in 1781, and subsequently 40 cases of chylothoraces secondary to tumor were described by Burgebuhr in 1895. This process was thought to be untreatable and frequently fatal until Lampson demonstrated that ligation of the thoracic duct was both feasible and effective. A convenient classification of chylothorax is as follows: congenital (in the neonatal period due to birth trauma and/or anatomic anomaly); traumatic (surgical, with reported 0.25–0.5% incidence after cardiovascular and thoracic surgery); nonsurgical, with the development of chylothorax typically two to 10 days after penetrating or blunt trauma; and atraumatic (associated with malignancy).

A tentative diagnosis is made by the usual signs, symptoms, and roentgenographic findings of pleural effusion in association with the clinical situation. The most frequently cited presenting symptom is dyspnea. The specific diagnosis is made by determination of the composition of the effusion. Chylous lymph is unique in that its fat content is greater than that found in plasma and its protein content is approximately one-half that of plasma.

Treatment consists of repeated thoracenteses or chest tube drainage. Concomitant medical therapy includes nutritional upkeep with a high calorie, high-protein, medium-chain triglyceride, low-fat diet supplemented with fat-soluble vitamins. This serves to replete losses and decrease lymph flow, promoting fistula closure and/or clot formation. It is the potential nutritional deficit coupled with losses of other plasma proteins, electrolytes, and lymphocytes that increases morbidity and mortality with prolonged leakage. Spontaneous closure occurs in approximately 50% of cases treated conservatively. Failing this, surgical ductal ligation is indicated.

The sequence of events as outlined in our case report and the proximity of the major lymphatic conduits to the needle placement leads one to strongly suspect that the development of the chylous effusion was a direct consequence of this procedure. Review of the literature reveals...
neither a report of chylothorax nor allusion to potential damage to the lymphatic system in reference to celiac plexus block.

Interestingly, the radiology literature reveals that chylothorax developing secondary to puncture of the cisterna chyli or thoracic duct is now a recognized although infrequent complication of high translumbar aortography.\textsuperscript{17,18} The percutaneous approach for this diagnostic procedure\textsuperscript{19} closely resembles that for performing celiac plexus block.

The possibility that the chylothorax in the case presented resulted from the patient's primary disease rather than caused iatrogenically cannot be absolutely excluded. Against this conclusion, however, is the relatively rapid resolution of the process without recurrence or other symptoms such as chylous ascites, the absence of an appreciable obstructing mass on CT scan, and the overall rarity of such a situation.\textsuperscript{10} Another possible explanation could be erosion of the lymphatic endothelial wall by the neurolytic agent. Again, this mechanism has not been described and, without a specimen to examine, only speculation is in order.

In summary, a case of chylothorax following a neurolytic celiac plexus block is presented with a review and discussion of the possible pathophysiology and management involved. Though we are not aware of any technical measures that could be used to avoid this complication, this case report reinforces the need for follow-up of patients having such therapies and the need to include the relationships of the major lymphatic structures in anatomic descriptions of this particular procedure.

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