from the thymus and had infiltrated the S³ segment of the left lung and the pericardium. However, no effusion was seen. Small metastatic nodules were also scattered on the surface of the myoecardium, and only a partial resection was performed. The tumor was confirmed to be malignant thymoma of epithelial origin. Histochecmially, the tumor cells were positive for keratin stain, and staining was negative for carcinoembryonic antigen, α-fetoprotein, and human chronic gonadotropin. The patient received 4,000 rad with Cobalt 60, and this resulted in the relief of chest pain and diminution of the tumor. Prednisolone was stopped 2 months later. Although the patient refused further chemotherapy, he remained symptom-free for 5 years up to the present time, without any complications such as myasthenia gravis, erythroid hypoplasia, or hypogammaglobulinemia.

**DISCUSSION**

The frequency of malignant thymoma is reported to be up to two-thirds that of benign thymoma.¹ ² Malignant thymoma is defined by invasive characteristics of the tumor to adjacent organs, and prognosis is more closely related to gross characteristics at operation than to histologic appearances. Some thymomas that were benign in their early periods can develop an invasive character in their late clinical courses.³ ⁴ Local invasion might occur into the pleura, pericardium, and lung. Reports of malignant thymoma, however, presenting with only pericarditis or tamponade are few,⁵ ⁶ and cytologic study of pericardial fluid has rarely been positive.⁷

Empiric antituberculosis treatment has been given with corticosteroids in life-threatening cases of suspected tuberculous pericarditis with good results.⁶ And large doses of corticosteroids that were used coincidentally for the pericarditis in the present case of malignant thymoma turned out to be lifesaving. Although the mechanism of action remains speculative, corticosteroids were reportedly effective in 11 of 13 thymoma cases.⁷

The treatment of malignant thymoma consists of radical extirpation followed by adjuvant or combination radiotherapy with or without chemotherapy.² ³ ⁴ In the present case, myocardial infiltration made radical extirpation of the tumor impossible. After relief of pericarditis with corticosteroids, radiotherapy was very effective for this thymoma.

Although infrequent, malignant thymoma may present solely as pericarditis, and the potential effectiveness of corticosteroids for such pericarditis should be noted.

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**REFERENCES**


**Hemothorax Following Uncomplicated Sclerotherapy for Esophageal Varices**

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Although pleural effusion as a complication of esophageal manipulation is well described in the literature, the fluid is usually nonhemorrhagic. We describe the first patient who had uncomplicated sclerotherapy with ethanolamine for esophageal varices, and on two occasions this patient developed left-sided bloody pleural effusion within 12-72 h after sclerotherapy. The effusion resolved spontaneously within 4 weeks. This case illustrates that hemothorax should be included in the pulmonary complications of sclerotherapy for esophageal varices.


**MANIPULATION**

Manipulation of the esophagus may lead to an exudative pleural effusion, sometimes with a very low pH pleural fluid if the procedure resulted in trauma to the esophagus. The effusion is usually a serous exudate. Bloody effusion has not been described as a complication of nontraumatic esophageal manipulation. We present a patient who developed bloody effusion on two occasions after sclerotherapy of the esophagus with ethanolamine for esophageal varices.

**CASE REPORT**

A 40-year-old white man arrived at the emergency department complaining of vomiting of blood, and dark stools for 3 days before presentation. He denied any pulmonary symptoms. There was a 40-pack-year history of smoking, as well as a long history of alcohol abuse.

On examination the patient appeared to be in no distress. His BP was 130/70 mm Hg and pulse 96/min. There was no postural change in BP or pulse rate. Chest examination was completely normal. Chest roentgenogram was normal. Hemoglobin was 56 g/L, platelet count 144,000/mL, and prothrombin and partial thromboplastin times were 14.4 s and 31.2 s, respectively.

On the day of admission, the patient underwent esophagogastroduodenoscopy, which revealed grade 3 esophageal varices without any active bleeding, and sclerotherapy with 14 mL of ethanolamine. There were no complications. The same procedure was repeated on the day after the admission without any complications.

Within 12 h after the second sclerotherapy, the patient developed a temperature of 39°C and left-sided pleuritic chest pain. Chest roentgenogram showed left pleural effusion. The patient was started on antibiotics. On the 5th hospital day, sclerotherapy was repeated, and the roentgenogram taken 12 h later showed increase in the size of the effusion. Thoracentesis revealed serosanguineous fluid that was an exudate by protein and lactate dehydrogenase criteria, and it did not show elevated amylase. The erythrocyte count was 40,000x10⁹/L, WBC count was

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11,700 X 10^6/L with 3 percent polymorphonuclear cells, 32 percent eosinophils, 62 percent monocytes, and 2 percent basophils. The patient became afebrile and the chest pain disappeared.

Because of continuous gastrointestinal bleeding, sclerotherapy was repeated on the 8th hospital day. Roentgenogram of the chest obtained within 72 h showed moderate-sized left pleural effusion. Thoracentesis and closed pleural biopsy were done. The fluid was essentially similar as on the previous occasion, with high erythrocyte cell count (80,000 X 10^6/L) and high eosinophil count of 69 percent (total WBC count was 9,100 X 10^6/L). A pleural biopsy showed pleural thickening, vascular congestion and focal hemorrhage, proliferation of fibroblasts and endothelial cells, and infiltration by lymphocytes and plasma cells. Bacterial cultures, auraminorhodamine stains, and subsequent tuberculous cultures were negative.

The patient remained asymptomatic, afebrile, and was discharged on the 10th hospital day. When seen in follow-up within 1 month of discharge, the chest examination was normal and the roentgenogram of the chest showed complete resolution of left-sided pleural effusion.

**DISCUSSION**

This case shows that hemothorax may be a complication of sclerotherapy for esophageal varices. Nonhemorrhagic pleural effusion after variceal sclerotherapy is not uncommon. In a retrospective study of 223 patients undergoing this treatment, Zeller et al. found the incidence of pleural effusion to be 27 percent. Other studies demonstrate an even higher incidence of pleural effusion, up to 50 percent.1,5 In their series of 65 patients undergoing sclerotherapy, Saks et al. found 31 (48 percent) episodes of pleural effusion; in 35 percent of the cases the fluid was an exudate. These effusions are relatively small and tend to involve either side with equal frequency. There seems to be a relationship between the amount of sclerosant used and the incidence of pleural effusion. Furthermore, many patients with significant pleural effusion also complain of chest pain. Occasionally, the effusion can be a transudate, probably reflecting fluid overload after overzealous resuscitation.

It is likely that the pleural effusion resulting from variceal sclerosis is secondary to necrotizing periesophageal inflammation and hemorrhage resulting in mediastinitis. This accounts for an equal incidence of right- and left-sided pleural effusions noted in most of the published series. Spread of inflammation is also favored by the lack of serosae covering of the esophagus. Using computed tomography, Mauro et al. found evidence of mediastinitis in most patients receiving sclerotherapy. Chylothorax resulting from the thoracic duct injury after sclerotherapy has been reported.5

As far as we are aware, this is the first documented case of hemorrhagic pleural effusion after variceal sclerosis. We have established a clear relationship between the onset or increase in size of the effusion and the timing of sclerotherapy. Appearance of the fluid, high erythrocyte count, and increased eosinophil count all confirm the hemorrhagic nature of the effusion.

The pathogenesis of this hemorrhagic effusion is not entirely clear. It is possible that it may simply reflect the severity of inflammation after para-variceal extravasation of the sclerosant. Alternatively, this patient could have abnormally dilated vessels on the outer wall of the esophagus, secondary to portal hypertension. If that is the case, sclerotherapy might have led to bleeding from one of the vessels, with subsequent accumulation of blood in the pleural space. Independently of the precise mechanism involved, this case serves to raise the awareness of hemothorax as a potential complication of sclerotherapy for esophageal varices.

**REFERENCES**


**Relief of Superior Vena Cava Syndrome Due to Fibrosing Mediastinitis Using the Palmaz Stent**

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Relief of superior vena cava (SVC) syndrome due to non-neoplastic mediastinal disease presents a formidable challenge. Long-term patency of surgically created bypass grafts has been poor, and the morbidity associated with these procedures is substantial. We report a case of SVC syndrome, caused by fibrosing mediastinitis, treated with Palmaz balloon expandable intravascular stents. Intravascular stents are a promising alternative for relief of non-neoplastic SVC obstruction.

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**SVC=superior vena cava**

While superior vena cava (SVC) obstruction most often is caused by intrathoracic malignancy, non-neoplastic etiologies continue to account for about 20 percent of cases.1,6 Fibrosing mediastinitis is the most common non-neoplastic cause of SVC obstruction, causing about 10 percent of cases overall.1,2 Treatment of SVC syndrome due to non-neoplastic disease presents the challenge of maintaining long-term patency of the SVC in these patients with otherwise normal life expectancies. The efficacy of surgical bypass for SVC obstruction has been disappointing in this regard, leading to controversy about the optimal treatment for these patients.3,4 We report the use of intravascular stents to treat SVC syndrome due to non-neoplastic obstruction.

**CASE REPORT**

A 44-year-old man was admitted to the hospital for treatment of SVC obstruction. He lived in the Ohio River Valley. He was

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