num through the esophageal opening has been rewarded with complete resolution of mediastinitis.² An extra benefit has been the spontaneous healing and closure of the esophageal opening.

If the authors had used transesophageal irrigation on their patients with a failed esophageal closure, it is possible that better results would have been obtained. I continue to recommend transesophageal irrigation in patients with mediastinitis caused by esophageal perforation as the best option for this serious condition.

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Reply to the Editor:

Drs. Santos and Frater¹ reported a very interesting technique to treat patients with mediastinitis caused by esophageal perforation. They irrigated the mediastinum through the perforation in the esophagus. Profuse transesophageal irrigation of the mediastinum was achieved with orally ingested fluid. If the patient could not drink, the mediastinal irrigation was accomplished with a nasogastric tube positioned in the upper esophagus proximal to the perforation and connected to a bottle containing saline solution that was allowed to drip at a rate sufficient to keep the mediastinum clean. More precise guidelines were not available either in the original article¹ or in Dr. Santos' letter to the Editor.

Santos and Frater described eight patients who had a perforation not amenable to repair or a leak resulting from disruption of an attempted esophageal repair. It would be interesting to know how many primary repairs or other techniques were used during this time in their institution or whether these eight patients comprise their total experience with esophageal rupture. A critical examination of the series of patients presented by Santos and Frater¹ reveals principal differences compared with our patients.² Three of the eight patients had cervical esophageal rupture, which is a totally different condition from delayed septic perforation of the thoracic esophagus and has a very good prognosis even in delayed cases. The remaining five patients had rupture of the thoracic esophagus. All of our 34 patients² had a perforation of the thoracic esophagus with mediastinal sepsis, and the mean delay in diagnosis was 4 days, whereas the patients of Santos and Frater were operated on 26, 30, and 36 hours after rupture. This means that their diagnoses were made earlier and the delay in diagnosis may have been less than 1 day. The longer the diagnosis and treatment are delayed, the greater the mortality.³

Although the time factor and anatomic location are of critical importance in the prognosis of esophageal perforation, the size of the rupture also has an impact on the outcome. The size of the esophageal perforations in the report of Santos and Frater varied between 2.5 and 5 cm, whereas some of our patients had

tears measuring 10 cm.⁴ Taken all together, it is evident that the two series of patients are not comparable because of differences in the delay in diagnosis and the size of the perforations. Therefore no comparison of the reported therapeutic methods is possible.

I appreciate the interesting technique of transesophageal irrigation presented by Santos and Frater and hope that they report their results in delayed cases soon. However, I am dubious about its effectiveness. In delayed cases of mediastinitis the infection has spread far from the rupture in the mediastinum and pleural cavities. Open techniques (thoracotomy) allow exact placement of irrigation and drainage tubes, as well as careful cleansing of the mediastinum and both pleural cavities. Esophagectomy is necessary to remove the often gangrenous esophagus and to prevent the esophagomediastinal reflux of digestive gastric and duodenal juices, as well as intestinal bacteria. Cervical esophagostomy diverts the swallowed salivary enzymes and bacteria. We continue to use esophagectomy and mediastinal irrigation with local and intravenous antibiotics for the treatment of patients with delayed esophageal perforations with mediastinal sepsis. In early cases primary repair is the method of choice. If the perforation is located in the distal esophagus the primary repair site should be covered with gastric fundus and in more proximal perforations with omentum or pleura. Fibrin sealant (Tisseel, Immuno AG, Vienna, Austria) may be used for additional security.

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Regression of autoimmune thrombocythemia after resection of pulmonary plasma cell granuloma

To the Editor:

Plasma cell granuloma (PCG), a rare, nonneoplastic lesion of the lung, first was described by Bahadori and Liebow.¹ PCG accounts for fewer than 1% of all lung tumors. It occurs at all ages but predominantly in children and young adults; sexes and ethnic groups are equally affected. Clinical features, including evidence of a preceding or concurrent respiratory infection, are variable, but almost 70% of patients have no symptoms.² Associations with such immunologic and hematologic disorders as hypergammaglobulinemia involving immunoglobulin G, M, and A fractions, elevation of erythrocyte sedimentation rate,

Fig. 1. Chest radiograph shows a left lower lobe opacity.

and thrombocytosis have been reported.^{3,4} Chest radiographs usually demonstrate a single round lesion, more rarely showing double or multiple lesions. Computed tomographic scan often resembles bronchogenic carcinoma and the diagnostic accuracy of transthoracic fine-needle aspiration biopsy is also questionable.² The natural history of PCG of the lung is variable; it may resolve spontaneously, remain static for years, or continue to grow with possible aggressive impingement on the surrounding structures.² In fact, invasion of the mediastinum, the diaphragm, and the vessels has been described, as well as recurrence after surgical resection.⁵ A surgical approach is therefore advocated for diagnostic purposes as well as for complete excision of the lesion and prevention of local recurrence.

We present here a case report of PCG of the lung associated with autoimmune thrombocythemia that regressed after radical resection of the pulmonary lesion. We believe that the case we describe here is the first documented observation of such an association.

The patient was a 24-year-old nonsmoking man who had been well until the age of 7 years, when he had right basal bronchopneumonia. Ten years later, a chest radiograph demonstrated a pulmonary coin lesion of the left lower lobe in the absence of any clinical symptoms. The patient refused further diagnostic procedures. When the patient was 23 years old, routine laboratory examinations were remarkable for a platelet count of 54,000 cells/ml. A few months later, laboratory and immunologic screening showed a platelet count of 48,000 cells/ml with a decreased platelet-aggregation capacity and endogen adenosine diphosphate block. Results of direct Dixon test were positive for the presence of antiplatelet autoantibodies (1000 ng immunoglobulin G/10⁷ platelets, normal range 0 to 100 ng immunoglobulin G/10⁷ platelets). A myelogram showed an increased number of megakaryocytes. Coagulation test results were within normal limits. A chest radiograph (Fig. 1) integrated by computed tomographic scan of the thorax (Fig. 2) confirmed the presence of a left lower lobe opacity, the main diameter of which had increased from 2.5 to 3.0 cm, with evidence of pleural perinodular thickening. Results of bronchoscopy were negative. A left lower lobectomy was performed with-

Fig. 2. Computed tomographic scan of the thorax shows a round lesion (3 cm) in the left lower lobe, with evidence of pleural perinodular thickening.

out difficulties. Histologic diagnosis was PCG originating in the lung with a localized proliferation of mature plasma cells, reticuloendothelial cells, and intermediate forms supported by a stroma of granulation tissue infiltrating the visceral pleura. Although only 1 U platelet concentrate was transfused before the operation and no steroids were administered, platelet count was 310,000 cells/ml on postoperative day 10. The postoperative course was uneventful, but during follow-up the laboratory tests showed a reduction of the platelet count to 140,000 cells/ml 3 months after the operation. This value remains stable 24 months after operation. Results of direct Dixon test are actually negative for the presence of antiplatelet autoantibodies. The increased platelet count after removal of the tumor may suggest a correlation between the immunologic disorder and the presence of PCG of the lung. A secretory role of PCG has been previously supposed,^{1,4} but a hyperimmune response to the lesion itself may also explain detection of high immunoglobulin levels.⁴ The presence of autoantibodies in this patient and their decrease after surgical resection of the pulmonary lesion suggest an involvement of the immune system in this disorder.

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Malignant fibrous histiocytoma of the chest wall developing after pleuropneumectomy performed for tuberculous pyothorax: Report of an unusual case

To the Editor:

Reports indicate the onset of malignant fibrous histiocytoma (MFH) in the chest wall after old pleuritis or pyothorax,¹ radiotherapy,² or burns,³ but they rarely indicate that MFH develops after operations.⁴ We treated a patient who had contracted tuberculous pleuritis and tuberculous pyothorax and in whom MFH developed in the chest wall 16 years after pleuropneumectomy carried out for tuberculous pyothorax.

The patient was a 69-year-old man with cough and pyrexia. When he was 19 years old, complete rest had been prescribed for the treatment of left-sided tuberculous pleuritis. At the age of 53 years, he had undergone pleuropneumectomy for left-sided tuberculous pyothorax at our hospital. In March 1992, cough and fever (38° C) appeared. He was admitted to the department of internal medicine of our hospital and received combination drug therapy with isoniazid (INH), rifampicin (RFP), etambutol (EB), and various antibiotics for about 2 months. Despite these medications, remittent fever persisted.

Repeated thoracentesis at the department of internal medicine yielded no materials sufficient for bacteriological examination. Inasmuch as no definitive diagnosis was established, he was referred to our department for open chest biopsy on May 15, 1992. After he was transferred to our department, laboratory examination revealed an erythrocyte count of $348 \times 10^4/\text{mm}^3$, a hematocrit value of 27.1%, and a hemoglobin level of 8.9 gm/dl, indicating anemia. Anemia had progressed during the 2 months after the initial examination at the department of internal medicine. In addition, marked inflammation was apparent: the leukocyte count was $9800/\text{mm}^3$, erythrocyte sedimentation rate 143 mm/hr, and C-reactive protein 22.6 mg/dl. The purified protein derivative test was $0/22 \times 20$ mm. Roentgenogram of the chest showed that the left lung was absent because of past pleuropneumectomy. The left sixth rib was absent, and the seventh rib was deformed. Comparison with the previous chest x-ray films indicated that the defect and deformity were due to the operation carried out 16 years previously. A computed tomographic scan of the chest visualized marked deformity of the thorax and ribs. A wall with a uniform thickness on both the pleural and mediastinal sides surrounded the thoracic cavity, which contained no obvious protruding tumor. By gallium 67 scintigraphy, an image of abnormal accumulation was observed

in the area corresponding to the left thoracic cavity. In summary, no abscess could be aspirated by thoracentesis, fever did not subside after antibiotic administration, and the chest roentgenogram showed no air fluid level. These findings did not suggest bacterial infection, and the reaction to the purified protein derivative test was too weak to suggest active tuberculosis. Because the patient became anemic in a short period of time, a malignant tumor of the chest wall was suspected, and open chest biopsy was performed on June 5, 1992.

A left posterolateral incision revealed the intercostal spaces to be narrowed and to show yellowish white thickening. A portion of the parietal pleura was resected through the narrowed fifth intercostal space. Prompt pathologic examination revealed sarcomatoid malignant cells. Thoracotomy was done in the fifth intercostal space to collect tissue from the thoracic cavity. Malignant cells were also present in this area. The yellowish white intercostal thickening involved the other intercostal spaces and ribs. The tumor in the thoracic wall was thought to have invaded the mediastinal side. Because a curative operation was judged to be impossible because of the extensive lesion, part of the third, fourth, and fifth ribs, the neighboring parietal pleura, and the tissue in the thoracic cavity were collected to end the surgical procedure. Specimens of the parietal pleural tissue were stained with hematoxylin and eosin. Low-power magnification of the specimens visualized a tumor in which spindle cells grew while forming a storiform pattern. The tumor appeared to be a polymorphic sarcoma marked by atypical giant cells. High-power magnification of the image revealed tumor cells consisting of fusiform fibroblastic cells and slightly oval histiocytic cells. These cells were positive in α_1 -antichymotrypsin staining but were negative in all of cytokeratin (KL-1), epithelial membrane antigen (EMA) and S-100 protein staining. These findings led to a histologic diagnosis of MFH. Histologic findings of the resected bone also showed invasion of the bone marrow by the MFH.

Postoperatively, three courses of CYVADICT therapy (cyclophosphamide, vincristine, doxorubicin, and dactinomycin) were given. Subsequently, the chemotherapy could not be continued because of debilitation. The patient died of general debilitation caused by tumoral fever 5 months after the operation. Consent for autopsy was not obtained.

The common sites of occurrence of MFH include the proximal regions of the extremities, such as the thigh, buttock, upper arm and shoulder, and the posterior peritoneum. The chest wall is a rare site of origin.⁵ Reports indicate that MFH develops 8 to 14 years after radiotherapy,² 16 to 25 years after burns,^{3,6} and in the cicatricial wound 6 to 8 years after the operation.⁴ It also has occurred 10 months after the onset of bone marrow infarction⁷ and in the area having a vascular prosthesis 1 year later.⁸ Malignant tumors frequently occur in the chest wall and thoracic cavity after chronic pleuritis and pyothorax. Epithelial tumors include squamous cell carcinoma, and nonepithelial tumors include malignant lymphoma, liposarcoma, angiosarcoma, rhabdomyosarcoma, malignant mesothelioma, and MFH. Myoui and associates¹ found soft tissue sarcoma originating in the chest wall or thoracic cavity in two of 134 patients with tuberculous pyothorax and reported that the prevalence was about 1000 times that among normal individuals. They also reported that 65% of patients with MFH originating in the thoracic cavity had histories of chronic pleuritis or pyothorax, drawing attention to the relationships between histories of these disorders and the onset of MFH. The reported interval between