and it has been hypothesized that local mast cell activation may lead to coronary spasm. The effects of mediators such as leukotrienes and prostaglandins released during anaphylaxis may be important in regulation of coronary vascular tone. Leukotrienes C₄, D₄, and E₄ are coronary vasoconstrictors and negatively inotropic agents in both human and animal cardiac tissue.

As more cases of IA are being recognized, it is important to provide appropriate instructions and treatment for the patient. The pharmacologic management has been recommended in treatment of idiopathic anaphylaxis. Patients with infrequent episodes are treated immediately with epinephrine, prednisone, and an antihistamine (hydroxyzine, diphenhydramine, or chlorpheniramine) for each episode of anaphylaxis. It is essential that patients carry epinephrine at all times, having received instructions for self-administration. Patients with frequent episodes are treated with maintenance therapy with antihistamines, sympathomimetics such as albuterol or ephedrine (in absence of cardiac contraindication), and prednisone. This pharmacologic management, in addition to avoidance of nonessential medications, especially aspirin, has been shown to reduce the severity and recurrent episodes of life-threatening anaphylaxis. On theoretic grounds, β-adrenergic antagonists should be withheld from cases of IA as well.

REFERENCES

A case is presented of a woman with bilateral spontaneous pneumothorax and pulmonary metastases originating from an ovarian granulosa cell tumor. It is the first known case report of an association between these two entities described in the English literature. (Chest 1990; 98:503-05)

Spontaneous pneumothorax is a common cause of acute pulmonary insufficiency that is usually caused by rupture of a bulla on the surface of the upper lobe of the lung. However, a pneumothorax also can be caused by some underlying pulmonary disease, particularly those causing fibrosis or cystic disease. Another important etiology of spontaneous pneumothorax is malignancy, originating from either the lung or a pulmonary metastasis from another area of the body.

Textbooks and reviews discussing the etiology of pneumothorax frequently neglect to mention metastatic neoplasms. This report describes a case of bilateral spontaneous pneumothoraces caused by pulmonary metastases from an ovarian tumor. It represents the first case report in the English literature of an association between pneumothorax and ovarian malignancy. It is hoped that this article will emphasize the importance of including metastatic neoplasms in the differential diagnosis of spontaneous pneumothorax.

CASE REPORT
A 67-year-old white woman presented to the emergency room of The Staten Island Hospital complaining of the sudden onset of dyspnea and right-sided chest discomfort. A chest radiograph revealed nearly complete collapse of the right lung, a right pulmonary nodule, and an estimated 30 percent left pneumothorax.

Bilateral closed-tube thoracostomies was performed. Her past medical history was significant for hypertension and hypercholesterolemia. The patient also had a history of a right ovarian granulosa cell tumor (stage IAii) for which she had undergone a total abdominal hysterectomy and bilateral salpingooophorectomy 17 years prior to this admission. Nine years prior to this admission, she had a workup for bilateral pulmonary nodules, which were believed to be benign. Four years later, she suffered a right spontaneous pneumothorax (15 percent) which was treated conservatively.

A CT scan of the chest, performed shortly after admission, revealed multiple nodules of the right middle and lower lobes. A CT scan of the abdomen failed to demonstrate any signs of intra-abdominal disease.

Bilateral Spontaneous Pneumothoraces Caused by Metastatic Ovarian Granulosa Cell Tumor

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![Figure 1. Low-power micrograph of subpleural peripheral metastatic tumor nodule in lung parenchyma (hematoxylin and eosin, original magnification × 100).](image-url)
Bilateral Spontaneous Pneumothoraxes (Davidson, McGlin, Goldberg)

Median sternotomy and bilateral pleurodesis were performed on hospital day 5. Multiple pulmonary nodules ranging in size from 1 mm to 2 cm were palpable throughout the parenchyma of both lungs and visible under the surface of the visceral pleurae. They were composed of soft, pale tan-gray translucent glistening tissues. Excisional wedge biopsies of four of these lesions were performed. Gross examination of these biopsies showed the nodules to be well circumscribed, with a bulging fish-flesh-like cut surface. The two largest were subpleural (Fig 1). Microscopic examination showed that these nodules, both interstitial and intrapleural, were composed of a biphasic tumor of (1) mostly sarcomatoid small, oval cells in a diffuse or haphazard pattern with indistinct borders, scant pale cytoplasm and occasional nuclear grooves (Fig 2), and (2) a rare larger cell element in a solid-tubular pattern (Fig 3). Microfolicular structures with empty central spaces were focally present (Fig 2), and only rare mitotic figures were identified (0.12/10 HPF in 500 HPF counted). There was no significant tumor cell lipid on frozen section material and immunohistochemistry studies were 1+ positive for vimentin and negative for chromogranin, S-100 protein, desmin, alpha-1-antitrypsin, and carcinoma embryonic antigen. These morphologic findings are diagnostic of metastatic granulosa cell tumor. The two largest nodules showed central degenerative changes and hemorrhage, without any evidence of pulmonary or interstitial emphysema in the surrounding lung parenchyma.

Our patient's postoperative course was uncomplicated, except for a prolonged air leak on the left side. The right chest tubes were removed on the fourth postoperative day and the left tubes were removed several days later. The patient was discharged and has continued to do well during the past 11 months of follow-up.

**Discussion**

Primary spontaneous pneumothorax usually is caused by rupture of a bulla on the surface of the upper lobe of the lung. Secondary spontaneous pneumothorax has been associated with many underlying pulmonary diseases, particularly those causing fibrosis or cystic disease. These include cystic fibrosis, Marfan's syndrome, eosinophilic granuloma, scleroderma, sarcoidosis, lymphangioleiomyomatosis, idiopathic pulmonary fibrosis, and some of the pneumonoises.

At least 76 cases of metastatic lung lesions and 12 cases of primary lung cancers associated with spontaneous pneumothorax had been reported in the literature before 1976. The metastatic lesions include sarcomas, Wilms' tumor, teratomas, malignant melanoma, lymphomas and carcinomas. Sarcomas appear to be the most frequently cited causes of spontaneous pneumothorax caused by metastatic lesions. Uterine and cervical malignancies have been associated with pneumothorax, but there has not been a similar association reported involving an ovarian tumor. Evans et al, in a review of 118 granulosa cell tumors, reported three lung recurrences, neither of which resulted in pneumothorax.

Spontaneous pneumothorax is still a rare complication of pulmonary metastases. Dines et al reviewed 1,143 cases of pneumothorax and found only five cases that could be attributed to pulmonary metastases, and all of these were sarcomas. These cases of pneumothorax are thought to be caused by tumor necrosis causing cavitation. Rupture of the necrotic tumor creates a communication between a bronchus and the pleural cavity causing pneumothorax. An alternate theory has tumor metastases at the lung periphery producing bronchiolar obstruction and a ball-valve effect. Alveoli become overdistended and eventually rupture and pneumothorax results. The first theory seems to explain the pathogenesis of the pneumothorax in our patient, since the pathologic examination did not uncover evidence of pulmonary or interstitial emphysema in the lung parenchyma but did note tumor necrosis.

Any agent which causes or promotes tumor necrosis and cavitation would increase the risk of pneumothorax. Treatment of tumors with chemotherapeutic agents and radiation have been reported to cause pneumothorax in patients with lung metastases.

Textbooks and reviews discussing the etiology of pneumothorax frequently neglect to mention metastatic neoplasms. Our patient had a past medical history of a malignant disease and previous pneumothorax associated with pulmonary nodules. The nodules, thought to be benign, were not considered as an etiologic factor until the patient presented to our institution with a potentially catastrophic event. It was not until her past medical record was meticulously reviewed and the lesions were seen intraoperatively that the cause became more clear. It is our view that pulmonary metastases should always be included in the differential diagnosis of spontaneous pneumothorax.

**Figure 2.** High-power micrograph of tumor showing sarcomatoid pattern of small oval tumor cells. Note the microfolicular structure, nuclear grooving in the center of the micrograph, and the uniform oval to angular nuclei with a delicate chromatin pattern (hematoxylin and eosin, original magnification × 400).

**Figure 3.** High-power micrograph of tumor showing solid-tubular pattern of larger tumor cells. Note the peripheral nuclei and central abundant cytoplasm in the tubular structures (hematoxylin and eosin, original magnification × 400).
Acute Coronary Occlusion After Recent Coronary Angioplasty

Association with Exercise and Successful Treatment with Intracoronary Thrombolysis

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A case of coronary occlusion occurred seven days after successful percutaneous transluminal coronary angioplasty. The acute complication occurred shortly after a negative exercise stress test and was resolved with intracoronary urokinase. (Chest 1990; 98:505-07)

PTCA = percutaneous transluminal coronary angioplasty;
LAD = left anterior descending coronary artery

Early exercise stress testing after percutaneous transluminal coronary angioplasty (PTCA) is widely performed in order to verify the functional result of the procedure. Major complications derived from this approach have been reported in the literature with extreme rarity. We describe a case of coronary occlusion at the site of previous PTCA which occurred shortly after exercise stress testing and was successfully treated with intracoronary thrombolysis.

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Figure 1. Left anterior oblique coronary angiogram showing a 90 percent stenosis in the LAD proximal to the second diagonal branch before (top) and after (bottom) percutaneous transluminal coronary angioplasty.