with pulmonary nodules. Presently, we feel that thoracoscopic resection only should be considered for small (less than 3 cm) lesions in the outer third of the pulmonary parenchyma. However, as experience is gained with endoscopic surgical techniques and with the continued development of improved instrumentation (such as endoscopic stapling devices), the indications for thoracoscopic pulmonary resections are likely to increase.

Currently, our approach to the evaluation of the peripheral pulmonary nodule consists of standard staging techniques that include computerized tomography and, when indicated, mediastinoscopy. When the diagnosis remains in question, our initial operative approach is thoracoscopic resection. The need for a more extensive resection can be determined based on the results of frozen-section pathologic analysis. Thoracotomy with its attendant morbidity can be avoided in patients with benign disease and in patients with metastatic lesions. A select group of patients with bronchogenic carcinoma and severe impairment of lung function also may be best treated by thoracoscopic lung resection as definitive operative therapy.

In summary, thoracoscopic resection of a benign lung nodule is described. Continued success with thoracoscopic resection is likely to have a significant impact on the operative management of select patients with pulmonary lesions.

REFERENCES

Reversible Cardiac Arrest Related to Late-Onset Coronary Spasm After a Positive Ergonovine Test*

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This report describes a patient with variant angina who had a positive ergonovine test successfully antagonized by intracoronary nitroglycerin and who developed a late recurrence of coronary spasm followed by reversible electromechanical dissociation. (Chest 1992; 102:1905-07)

The ergonovine provocative test is a useful tool in the evaluation of patients with chest pain and normal coronary arteries, due to its high sensitivity and specificity in the diagnosis of vasospastic angina.1-3 Although many years of experience have documented the safety of this test,1,3 a few cases of severe complications have been reported in the literature, including refractory coronary spasm,4-5 acute myocardial infarction,6 and death.7

We describe a case of a positive ergonovine test reversed by intracoronary nitroglycerin and followed, 20 min later, by recurrence of chest pain and reversible cardiac arrest due to electromechanical dissociation.

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Figure 1. Severe diffuse spasm of LAD (A, top), of RCA, and of PDA (B, bottom).
CASE REPORT

A 44-year-old woman came to our observation because of the recent onset of angina at rest. An inquiry into risk factors revealed mild hypertension and a smoking habit. The patient denied menstrual irregularities, as well as the use of the birth control pill. Findings from the physical examination were normal. A baseline ECG showed inverted T waves in all leads. During an episode of chest pain, positive T waves were documented in inferior leads. A maximal exercise stress test was negative for effort-related ischemia.

At cardiac catheterization, left ventricular function was normal (ejection fraction, 0.70). Coronary arteriography revealed nonsignificant (<50 percent) stenoses in the midportion of the left anterior descending coronary artery (LAD) and in the posterior descending coronary artery (PDA).

An ergonovine provocative test was performed following the guidelines of the American College of Physicians in separate doses at 5-min intervals until the total dose of 0.4 mg of methylergonovine was administered. At the end of the last dose, the patient experienced chest pain with ST-segment elevation in the monitored leads (1, 2, 3 and aVF). Coronary angiograms revealed marked diffuse constriction of the coronary vascular bed with severe stenoses in the midportion of the LAD (Fig 1A), in segment 2 of the right coronary artery (RCA), and at the origin of the PDA (Fig 1B).

Prompt intracoronary administration of nitroglycerin (TNG) selectively in the left (0.25 mg) and right (0.25 mg) coronary arteries resolved the spasm (Fig 2). The ECG changes and the patient's symptoms disappeared.

Twenty minutes later, the patient, just before being transferred to the ward, had a new episode of chest pain, followed by sudden loss of consciousness. Absence of the arterial pulse wave was documented through the femoral sheath, while electrical activity was present (electromechanical dissociation). The ECG showed widened QRS complexes and ST-segment elevation. Life support was started, and the patient was intubated. Intratracheal epinephrine (1 mg) was ineffective. After the administration of intracardiac epinephrine (3 mg), intracardiac calcium gluconate (10 mg), and intravenous nitroglycerin (1 + 1 mg), ventricular fibrillation ensued. Following DC shock, mechanical activity reappeared, with a blood pressure of 190/100 mm Hg, a heart rate of 120 beats per minute, and a normal ST segment. The total time of circulatory arrest was 10 min.

The patient was subsequently transferred to the intensive care unit in assisted ventilation and with stable hemodynamic parameters and completely recovered a few hours later, without any residual neurologic impairment.

In the following clinical course, there was no electrocardiographic, echocardiographic, or enzymatic evidence of myocardial infarction. The patient was discharged on oral therapy with calcium antagonists and nitrates and is now doing well.

DISCUSSION

Ergonovine is able to induce coronary spasm in a high percentage of patients with variant angina. Severe reactions to ergonovine have been described, including intracranial spasm, which can be lethal. The protocol of administration is crucial in determining a high sensitivity without an unnecessary increase in risk. Whereas the utilization of small separated doses of ergonovine is generally accepted, controversy exists about the total amount that has to be administered. We used a total dose of 0.4 mg, which had proved to be effective and safe in large series.

The response to ergonovine in uncomplicated positive tests occurs within the first minutes after the administration of the drug. The same holds true in reported cases of severe complications.

In the case described, coronary spasm did occur early after the completion of the test but recurred 20 min later with dramatic clinical features.

Cases of electromechanical dissociation due to spontaneous or provoked coronary spasm rarely have been reported in the literature. This event is most likely a consequence of widespread ischemia due to severe three-vessel or left main coronary spasm.

Although no angiographic documentation of recurrence of spasm was obtained in our case because of the late onset, symptoms and ECG changes before cardiac arrest are strong supportive evidence that spasm was again present.

The intracoronary administration of nitroglycerin is the treatment of choice in antagonizing the effects of ergonovine on coronary vasculature, and in our experience, no case of refractoriness to this treatment ever occurred; however, this possibility is described in the literature, and the utilization of nitroprusside or nifedipine is suggested in these cases.

Our report emphasizes that in spite of the efficacy in the resolution of acute spasm, intracoronary nitroglycerin is
insufficient to prevent the late recurrence of the spasm because of nitroglycerin's short pharmacologic action. Therefore, the use of a combination of intracoronary nitroglycerin and sublingual long-acting nitrates is, in our opinion, advisable if intense and prolonged neutralization of ergonovine action is desired.

Observation of the patient for 15 min after the last dose of ergonovine is recommended by some authors who observed rare cases of recurrence of coronary spasm up to 10 min after induced spasm. Our report suggests that, especially in the case of a strongly positive test, observation of the patient in the catheterization laboratory has to be prolonged even when successful neutralization of spasm has been achieved, because it can recur several minutes later.

REFERENCES

Hilar and Mediastinal Lymphadenopathy with Hypersensitivity Pneumonitis Induced by Penicillin*

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A 54-year-old Japanese man demonstrated a sulbactam-induced hilar and mediastinal lymphadenopathy with hypersensitivity pneumonitis. A positive lymphocyte stimulation test for sulbactamin and a decreased CD4/CD8 ratio of lymphocytes in BAL fluid suggested that an alteration in cell-mediated mechanisms was responsible for the patient's symptoms. (Chest 1992; 102:1907-09)

Sulbactam, a derivative of the basic penicillin nucleus, inhibits β-lactamase. The use of sulbactam in combination with β-lactamase-sensitive antibiotics can potentiate its effect. Based on this concept, sulbactam tosilate (Unasyn), a prodrug double ester of ampicillin and sulbactam joined via a methylene linkage, has been developed.

Pulmonary hypersensitivity to antibiotics has been reported to manifest interstitial pneumonitis or eosinophilic infiltrates. In this report, we describe a rare case of sulbactam-induced pulmonary disease in which the prominent manifestation was hilar and mediastinal lymphadenopathy.

CASE REPORT

A 54-year-old Japanese male office worker was referred to Tokyo Medical College Hospital on November 7, 1990, for evaluation of hilar enlargement on a chest roentgenogram. In mid-October 1990, sulbactamin, an antiflussive, and an antiinflammatory drug were prescribed because of fever (about 40°C) and a nonproductive cough. A mild skin rash appeared mainly on the trunk two days after the medication was initiated, and all drugs were discontinued. On October 30, complaining of occasional cough and low-grade fever, he visited another physician. Coincidentally, sulbactam was again prescribed along with a mucolytic agent (Mucoisolvan). On October 31, a marked pruritic skin rash became prominent over the entire body, and sulbactam was discontinued. On November 1, he started to complain of exacerbated cough and sense of fever.

Although the skin eruptions gradually improved, bilateral hilar enlargement appeared on a chest roentgenogram on November 2 and became distinctive on November 6. The patient did not sense significant respiratory distress during this period, although a temporal visual disturbance occurred twice in the left eye. In addition to the above-mentioned drugs, he had taken cimetidine (Tagamet) for the treatment of gastritis for about two months before this episode.

His medical history revealed the patient suffered from pulmonary tuberculosis at the age of 27 years. He occasionally felt mild paresthesia of the tongue after oral penicillin. An elder brother had previously developed penicillin anaphylaxis and shock.

Physical examination, the skin over the patient's entire body demonstrated a mild urticaria-like rash. On the forehead, the skin over the temporal artery was particularly reddish and edematous bilaterally. The supravacular, axillary and inguinal lymph nodes were swollen but not tender. Auscultation of the lungs revealed a small amount of fine crackles in the bilateral basilar area. Cardiac examination was unremarkable. The edge of the liver was palpated 3 cm below the costal margin. The spleen was not palpable. Urine examination was normal. Complete blood cell counts showed leukocytosis (9,000/mm3) with significant eosinophilia (34 percent) and atypical lymphocytes (4 percent). Although it was 3,300/mm3 two months earlier, the lymphocyte count ranged from 1,800 to 3,200/mm3 during admission. (It recovered to 3,500/mm3 after...