vasospasm were not excluded with certainty in our patient, although there was no history of angina and the ECG was normal. Either of these entities can cause a wide spectrum of clinical syndromes ranging from transient silent myocardial ischemia to irreversible infarction. Patchy fibrosis and myofibrillar hypertrophy can result from diffuse small coronary vessel disease. Evidence for this process was not detected echocardiographically, and the flow of contrast medium during coronary angiography was brisk and not suggestive of small vessel disease. An endomyocardial biopsy can confirm the diagnosis of small coronary vessel disease; however, small vessel disease without an associated infarct region has not been reported to lead to LV thrombus. The same is true for patients with coronary vasospasm who are free of detectable LV dysfunction or necrosis. An endomyocardial biopsy may also be useful in diagnosing the early, insidious stages of endomyocardial fibrosis. This disease of uncertain etiology may have microscopic evidence of thrombus covering the outer layer of fibroed endocardium. Ventriculographic filling defects due to LV thrombus have been reported in patients with endocardial fibrosis, but these patients often have associated extensive left ventricular asynnergy. Our patient’s history, ECG, and echocardiogram failed to support the diagnosis of endomyocardial fibrosis, and LV wall motion was normal. This patient’s history of recurrent venous thromboses without apparent precipitating factors, associated now with apparently spontaneous thrombosis at an unusual anatomic site, raises the possibility of a primary or secondary hypercoagulable state. With or without confirmation of a diagnosis of a primary hypercoagulable state, lifelong prophylactic anticoagulant therapy is indicated under these circumstances, with careful attention to additive risk factors that might precipitate thrombosis. Diagnosis of a secondary hypercoagulable state might lead to amelioration of the thrombotic tendency through treatment of the underlying disorder. Such investigations in our patient to date are pending the termination of the postoperative anticoagulation period. We conclude that careful echocardiographic examination of all patients with arterial embolization is warranted, even in the absence of clinical evidence of cardiac disease.

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Treatment of Tracheobronchial Granular Cell Myoblastomas with Endoscopic Bipolar Cautery*


Granular cell myoblastomas of the trachea and right upper lobe bronchus were discovered incidentally during therapeutic bronchoscopy. Because of their propensity to cause airway compromise and distal atelectasis, ablation of both lesions was undertaken. This is the first reported case of bipolar cautery of GCM through a flexible fiberoptic bronchoscope. Small tumor size and lack of atelectasis permitted utilization of this technique. Long-term follow-up is necessary to compare this therapy with other nonsectional therapies.

(Chest 1989; 96:427-29)

Granular cell myoblastomas (GCM), first described by Abrikossoff in 1926, are Schwann cell tumors of the skin, breast, subcutaneous tissue, tongue, and rarely, the tracheobronchial tree. Classified as benign tumors, in the lung they present as a solitary pulmonary nodule, large obstructing endobronchial masses, or most commonly, as incidentally discovered small endobronchial lesions. The clinical recognition of this lesion is important for two reasons: resection must be performed for prevention or treatment of distal atelectasis and suppurration, and also to differentiate the observed lesion from bronchogenic carcinoma. Current treatment modalities include sleeve resection, lobectomy, pneumonectomy, external beam irradiation, and radon seed implantation. Curettage, electrocoagulation and laser therapy have been utilized through the rigid bronchoscope as alternative, nonsectional therapies. We describe the first case of multifocal (tracheal and right upper lobe bronchial) GCM ablated with bipolar cautery through the flexible fiberoptic bronchoscope.

Case Report
A 61-year-old black man was intubated nasotracheally after sustaining multiple rib fractures in a motor vehicle accident. On admission, diffuse wheezing and bilateral anterior chest wall tenderness were observed. Chest X-ray film demonstrated hyperinflation, left upper lobe bulla, bilateral rib fractures, right lower lobe density consistent with pulmonary contusion, and left lower lobe atelectasis. Therapeutic bronchoscopy was performed. Unexpectedly, lesions on the anterior wall of the trachea and a mass lesion of the right upper lobe bronchus were encountered. Cytologic brushings and washings yielded squamous metaplasia and reactive bronchial cells.

Because of concern for an endobronchial malignancy, repeat bronchoscopy was performed five days later. Nodular pink lesions were found on the anterior wall of the trachea (Fig 1). A separate

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FIGURE 1. Nodular granular cell myoblastomas of the anterior midportion of the trachea.

white-to-gray granular polypoid lesion was noted on the anterior wall of the right upper lobe bronchus, partially occluding the anterior segment of the lobe. Individually, tracheal lesions measured 0.5 to 1.0 mm; the right upper lobe lesion approximately 6 mm. The bronchial mucosa surrounding all lesions appeared indurated. Endoscopic biopsies of both lesions were reported as GCM (Fig 2).

Over the ensuing six days, the patient’s clinical condition improved and a third bronchoscopy was performed just prior to extubation for tumor ablation. Each lesion was repeatedly cauterized with 2 s bursts of 30 watts utilizing a bipolar electrode system (BICAP, Pulmonary Therapeutic System, Microvasive Inc, Milford, MA) through a flexible fiberoptic bronchoscope (Pentax Inc, Orangeburg, NY). Following the procedure, no tumor was visible—the trachea, the right upper lobe bronchus and anterior segment of the right upper lobe were widely patent. No other bronchoscopic follow-up was obtained prior to hospital discharge.

DISCUSSION

Once believed to be of skeletal muscle origin, much controversy has existed over the histogenesis of GCM. Electron microscopic evidence has indicated that this lesion is derived from neural cells of the Schwann cell line. Grossly, GCM range from several millimeters to 6.5 cm in size, can be white or pink, nodular or polypoid, and vary from submucosal irregularities to obstructing masses. These tumors rarely invade the pulmonary parenchyma, but may extend into the peribronchial tissues. Cytologically, it is associated with bronchial squamous metaplasia, and on biopsy, it displays closely packed, large, granular acidophilic cells with small oval to round nuclei, as seen in Figure 2.

Female sex, fifth decade of life and black race are the most frequently cited demographic factors for the development of GCM. As of 1986, only ten tracheal and 45 bronchial GCM have been described; the oldest patient with a tracheal lesion was 56 years old. Along with being the oldest described patient with tracheal GCM, our case also exhibited an additional bronchial tumor. Though multicentric tumors located in the skin, breast, tongue, and subcutaneous tissue are well described, multifocal tracheobronchial GCM are distinctly unusual. Ostermiller et al described a 7 percent incidence of multiple endobronchial occurrences.

The majority of these benign tumors are small, asymptomatic and are detected incidentally at bronchoscopy, surgery, or autopsy. Bulky tracheal lesions tend to present with dyspnea, chest pain, and stridor. Obstructing lesions in the major bronchi may present with hemoptysis, focal wheezing, and signs of recurrent pneumonia. The solitary pulmonary nodule, asthma, tracheobronchitis, airway obstruction, endobronchial lesions with atelectasis and many causes of hemoptysis need to be considered in the differential diagnosis of GCM. Abnormalities on computed tomography and flow volume loops may provide preliminary data prior to diagnosis by flexible fiberoptic bronchoscopy.

The multiple therapeutic modalities that have been employed to treat GCM have met with variable success. Clearly, sleeve resection, lobectomy, or pneumonectomy are indicated for tumors causing proximal airway compromise or atelectasis with distal suppuration. Resectional surgery is also suggested for larger (8 mm) GCM because of high recurrence rates with all endoscopic therapies.

Bipolar cautery has been used widely for ablation of gastrointestinal lesions. To date, it has been used seldom in pulmonary medicine and just recently was adapted for use with the flexible fiberoptic bronchoscope. Electric current is applied directly to burn, desiccate and vaporize obstructing endobronchial tissue by a bipolar cautery probe which is more flexible than a laser bundle. Rare reported complications of bipolar cautery include easily controlled bronchial hemorrhage, tracheal fires associated with high concentrations of inspired oxygen during general anesthesia, and electroshock of the endoscopist who discharges the electrode while in direct contact with the bronchoscope. Indications include palliation of upper lobe endobronchial tumors, cautery of vascular lesions, and treatment of tracheotomy suture granulomas. This technique was particularly well suited to our patient because he had multiple lesions, the uncertain natural history of asymptomatic tumors, underlying chronic obstructive pulmonary disease, and proximity to thoracic trauma. We propose bipolar cautery as quick, useful, safe, efficacious and less expensive nonresectional therapy for GCM ablation. Daniel et al have suggested yearly follow-up bronchoscopy for five years to screen for recurrence. More reported cases of GCM treated with bipolar cautery are required for critical analysis of this new therapeutic option.

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FIGURE 2. Granular cell myoblastoma of the right upper lobe with closely packed, granular acidophilic cells containing small oval to round nuclei and associated squamous metaplasia.
T Wave Inversion Associated with Severe Theophylline Toxicity*
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Adverse cardiovascular effects are commonly seen in severe theophylline poisoning. Primary ST-T wave changes have not been described previously. We report T wave inversion associated with severe theophylline toxicity in a 33-year-old woman with no evidence of organic heart disease. The T wave inversion resolved after treatment. Physicians should be alerted to possible T wave abnormalities in patients with severe theophylline poisoning. (Chest 1989; 96:429-31)

Theophylline is a widely used agent in the treatment of bronchospastic disorders. Toxicity generally occurs at levels greater than 25 μg/ml. Severe theophylline poisoning is characterized by cardiac arrhythmias, seizures, hypotension and cardiovascular collapse. To our knowledge, primary T wave abnormalities have not been reported previously in connection with theophylline toxicity. We report T wave inversion associated with severe theophylline toxicity.

CASE REPORT
A 33-year-old white woman with a history of asthma since childhood presented to Hahnemann University Hospital on February 1, 1986. The night prior to admission she developed progressive dyspnea and wheezing, and took a total of 2,400 mg of theophylline over eight hours in an attempt to ameliorate her symptoms. The patient then became nauseous, tremulous, and experienced palpitations which brought her to the emergency room. She denied chest pain or dizziness. Her only medication was theophylline 300 mg twice daily. She smoked one pack of cigarettes daily. There is no history of intravenous drug use, but the patient admits to occasional cocaine use, which she claimed to have had taken two weeks prior to admission.

On presentation, she was afebrile with blood pressure of 94/60 mm Hg. The pulse rate was 80/min with a respiratory rate of 28/min. She appeared anxious, but was alert, oriented and in no respiratory distress. Physical examination was remarkable only for a fine resting tremor and symmetric hyperreflexia.

The admission theophylline level was 50.4 μg/ml. Serum potassium, calcium, phosphorus, and magnesium levels were normal. A room air arterial blood gas determination revealed a pH of 7.50; Pco2, 29 mm Hg; and Po2, 100 mm Hg. Her chest x-ray film revealed only slight hyperinflation. The ER electrocardiogram revealed normal sinus rhythm at 62/min with T wave inversion in leads 2, 3, aVF, and V1 through V6 (see Fig 1). No dysrhythmia occurred during emergency room cardiac monitoring.

Eighteen hours after treatment with activated charcoal and magnesium citrate, her theophylline level had decreased to 1.9 μg/ml. A repeat electrocardiogram at that time revealed almost complete resolution of the initial T wave inversion (Fig 1). Serial cardiac enzymes were negative for an acute myocardial infarction. Findings from a two-dimensional echocardiogram including Doppler was normal. Pulmonary function testing revealed normal spirometry, but a decreased FEV1 25-75% suggesting small airways disease.

A test for urinary benzylecgonine (metabolite of cocaine) was negative. Follow-up ECGs remained unchanged. Three days after admission, the patient was asked to hyperventilate, during which time simultaneous ABC analysis and ECG were performed. There were no significant ST-T wave changes at a pH of 7.48 and Pco2 32 mm Hg. During treadmill exercise tolerance testing, the patient exercised into stage 3 of the Bruce protocol achieving only 60 percent of her predicted maximum heart rate. The exercise electrocardiogram showed normal physiologic ST-T wave changes without arrhythmia. Coronary arteriography revealed normal coronary arteries.

COMMENT
Studies of the electrophysiologic effects of theophylline have shown that it decreases both sinoatrial conduction time and His-Purkinje conduction interval. These changes may be caused by direct action of theophylline or by associated increases in sympathetic tone, but were not associated with T wave abnormalities.

Primary T wave inversion has been associated with myocardial ischemia, drug effect, electrolyte imbalance, central nervous system disease and hyperventilation. Hyperventilation is unlikely to be the cause of T wave inversion in our patient since an electrocardiogram performed during hyperventilation showed no significant T wave changes. Central nervous system disease can be associated with marked ECG waveform abnormalities, which are thought to be due to altered autonomic tone or centrally induced structural cardiac changes. Electrical stimulation of the animal brain has resulted in inversion and increased amplitude of the T wave. It is possible that the central nervous system effects of toxic levels of theophylline are responsible for the T wave changes seen in our patient.

It is interesting that our patient did not have tachycardia while theophylline toxic. We postulate that our patient may have underlying sinus node dysfunction or perhaps had relative autonomic dysfunction secondary to chronic cocaine use. Chronic cocaine use has been associated with decreased tyrosine hydroxylase activity and decreased beta-adrenergic

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