Dilatation of Bronchial Stenoses Due to Sarcoidosis Using a Flexible Fiberoptic Bronchoscope*

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Stenosis of the trachea and bronchi can complicate many diseases and lead to significant pulmonary complaints. Unfortunately, steroids rarely yield satisfactory results in reversing symptoms. We describe six patients with symptomatic airway stenosis from sarcoidosis, all of whom were refractory to steroid therapy. By using a Fogarty embolectomy catheter inserted through the inner channel of a flexible bronchoscope, we were able to dilate the stenotic areas under direct vision. Patients had significant subjective improvement following dilatation and no significant complications occurred. We believe this technique represents an improvement on previously described methods because it can easily access the upper lobes and more distal segments and can be performed at the bedside. (Chest 1994; 106:677-80)

Tracheal stenosis, bronchial stenosis, or both can complicate many diseases, including bronchogenic carcinoma, tuberculosis, sarcoidosis, and more recently as a consequence of lung transplantation.1-9 If consideration is not given to the possibility of bronchostenosis, this entity can be missed or the symptoms attributed to other diseases. Bronchostenosis can mimic asthma with wheezes or stridorous sounds or can cause recurrent infections and progressive dyspnea on exertion.

Patients with sarcoidosis have been referred to our institution for this problem. In fact, two of these patients presented with a prior diagnosis of asthma. The treatment of sarcoid bronchostenosis is usually with corticosteroids, but with mixed results at best.2-7,10 Alternatively, mechanical bougie dilation via a rigid bronchoscope10 has had limited success because the dilations are limited to the proximal airways.

We describe a new technique of dilatation in six patients with sarcoidosis. All patients were symptomatic and refractory to corticosteroid therapy. The procedure, done with a flexible fiberoptic bronchoscope and a Fogarty embolectomy catheter, can be performed at the bedside with local anesthesia and sedation, an advantage over previously described methods that used rigid bronchoscopes under general anesthesia or required fluoroscopy.

METHODS

There was an evolution to the described procedure that began using angioplasty catheters in the operating room. It was quickly realized that these expensive catheters were unable to adequately withstand the forces exerted and easily broke. Additionally, with the patient intubated, there were limitations to the maneuverability of the fiberoptic bronchoscope. For these reasons, the following method evolved.

The bronchoplasty procedure is performed using a 5-French Fogarty embolectomy catheter (Baxter Healthcare Corporation, Santa Ana, Calif) that easily fits through the inner channel of a fiberoptic bronchoscope (Olympus P-10) (diameter, 2.0 mm). After routine use of local anesthesia and intravenous sedation, the bronchoscope is positioned near the stenotic opening. The catheter is placed through the inner channel and guided into the opening. Normal saline solution, 1.5 ml, will inflate the balloon to a diameter of 11 mm. The balloon is inflated until firm resistance is felt on the syringe plunger (which will vary depending on the size of the opening) and held for 30 s. The balloon is deflated and withdrawn from the bronchoscope channel, if needed, to allow for irrigation and suctioning. The dilatation is repeated until maximal benefit is derived. The balloon on this catheter is clear, which makes it difficult to visualize when inflated in the airways. To overcome this problem, a few drops of methylene blue are mixed with the saline solution to produce a very visible balloon.

CASE 1

A 40-year-old black woman originally presented to the medical system in 1983 when she was evaluated for headaches and photophobia. Chest radiograph at the time of hospital admission revealed bilateral hilar adenopathy with some evidence of interstitial disease. Bronchoscopy revealed normal airways, but non-caseating granulomas were seen on tranbronchial biopsy specimens. She was started on a regimen of 60 mg of prednisone that was tapered over 1 year.

Key words: balloon dilatation; bronchoplasty; bronchostenosis; sarcoid

FVC=forced vital capacity; LLL=left lower lobe; LUL=left upper lobe; RLL=right lower lobe; RUL=right upper lobe

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She did well until 1990 when she noted progressive dyspnea on exertion with intermittent coughing and wheezing. She was treated with bronchodilators and intermittent steroids with some relief. In June 1992, the patient had rapid progression of her dyspnea and wheezing. She was restarted on a regimen of bronchodilators and given steroid bursts with marginal improvement. She had three hospital admissions between July 1992 and September 1992 for severe wheezing and shortness of breath. Despite the addition of theophylline, cromolyn, and intermittent steroid bursts, she became progressively more dyspneic and was essentially housebound in the 3 months prior to National Jewish Center referral. She had audible inspiratory and expiratory wheezing on examination without clubbing or cyanosis. She achieved only 21 percent of her workload on exercise testing due to dyspnea limitation. The FEV$_1$ and FVC were 1.32 L and 2.53 L, respectively.

Bronchoscopy revealed granular mucosa in the main-stem bronchi along with stenosis of the left lower lobe (LLL) takeoff, stenosis of lingular takeoff, approximate 1-mm opening of the posterior segment of the left upper lobe (LUL), stenosis of right lower lobe (RLL) takeoff, and stenosis of the posterior segment of the right upper lobe (RUL). The patient underwent bronchoplasty of involved segments over a 3-day period with marked dilatation of all involved segments. Figure 1 demonstrates the stenotic opening and postdilatation appearance of the posterior segment, LUL. Postprocedure, the patient was clinically improved and could walk around the hospital without problems. Repeated exercise testing showed that the patient could reach 38 percent of her workload which was limited by leg fatigue, not dyspnea. The FEV$_1$ and FVC were 1.72 L and 3.24 L, respectively. Presently the patient is doing well with FEV$_1$/FVC of 1.69 L/3.15 L, 6 months postprocedure.

**Case 2**

A 31-year-old black woman was referred for evaluation of progressive dyspnea. She had been diagnosed as having sarcoidosis following mediastinal biopsy 3 years earlier. Spirometry showed a FEV$_1$ and FVC of 1.58 L and 2.16 L, respectively. A 7-month course of high-dose prednisone therapy (40 mg) failed to improve her dyspnea, although there was some resolution of hilar adenopathy on chest radiograph. Her steroid trial was complicated by a 45-kg weight gain.

On bronchoscopy she had a stenosis at the lingular takeoff, a diffuse narrowing of the LUL takeoff, stenosis of the superior and basilar segments of the LLL, and stenosis of the superior segment of the RLL. Biopsy specimens showed nonnecrotizing granulomas with patchy fibrosis consistent with sarcoidosis. Spirometry revealed FEV$_1$/FVC of 1.79 L/2.02 L.

Bronchoplasty of the involved areas was performed as described above. The size of the stenotic areas improved, but full dilatation was not obtained. However, subjective improvement in her dyspnea occurred with increase in walking distance. Spirometry findings were unchanged. Repeated bronchoplasty was performed at 1 and 3 months to try to obtain more complete dilatation, but with minimal success. One year postprocedure, the patient is stable in regard to her symptoms. The FEV$_1$ is 1.89 L and FVC is 2.53 L.

**Case 3**

A 36-year-old white woman presented with progressive dyspnea on exertion over 18 months. Prior to the onset of symptoms, she had been well without pulmonary complaints. She had been diagnosed as having asthma and treated with inhaled and oral β-agonists, prednisone, and theophylline, without much improvement. Additionally, supplemental oxygen was required (PaO$_2$=45 mm Hg). The patient appeared normal at rest, but had inspiratory stridor with exercise. The FEV$_1$/FVC was 1.35 L/2.12 L.

On bronchoscopy, there was only a pinpoint opening to the anterior segment and narrowing of the posterior segment of the LUL. The lingular takeoff was absent. The superior segment of the LLL was also markedly narrowed. The RUL had significant stenosis of the apical and posterior segments. The right middle lobe takeoff was patent, but the openings to both the lateral and medial segments were pinpoint. The superior segment of the RLL was stenotic with other segments appearing normal. Endobronchial biopsy specimens revealed noncaseating granulomas consistent with sarcoidosis.

Following three separate episodes of bronchoplasty over 3 weeks, the patient had marked improvement in her symptoms, although results of spirometry remained unchanged. Her PaO$_2$ increased to 72 mm Hg. Treatment with all medications, including steroids and oxygen, was discontinued without adverse effect. More than 3 years postbronchoplasty, the patient subjectively reports minimal dyspnea on two flights of stairs. She is employed on a full-time basis.
CASE 4

A 40-year-old white woman developed dyspnea following the delivery of her first child 12 years previous. After the birth of her second child 1 year later, she again developed dyspnea and was given the diagnosis of asthma. She was started on a regimen of theophylline and underwent desensitization. Over the ensuing 11 years she had recurrent episodes of bronchitis with worsening of her dyspnea. She could walk only 4.5 m without dyspnea. Six months of high-dose steroid therapy did not improve her clinical status. The FEV₁ and FVC were 1.72 L and 2.66 L, respectively.

Bronchoscopy revealed a trachea narrowed to approximately 1 cm in diameter along with severe narrowing of the left and right main-stem bronchi. Endobronchial biopsy specimen was consistent with sarcoidosis. After bronchoplasty of the trachea and main-stem bronchi, the patient’s symptoms subsided and she was able to exercise without difficulty. The FEV₁/FVC was 2.36 L/2.98 L. Treatment with all medications was successfully tapered off. Symptoms started to recur after 1 year and repeated bronchoplasty of the right main-stem and bronchus intermedius reversed the dyspnea. She remains asymptomatic 6 months following the repeated bronchoplasty. The FEV₁ and FVC are 2.16 L and 3.02 L, respectively.

CASE 5

A 36-year-old black woman had a 9-year history of slowly progressive tracheobronchial narrowing. In February 1982, she noted the onset of upper airway stridorous breathing and breathlessness. Two weeks later, she developed symptoms of severe upper airway obstruction necessitating an emergency tracheostomy. In August 1982, she underwent tracheal resection of a subglottic stenotic area. From 1983 to 1988, she had frequent episodes of acute bronchitis requiring steroids and antibiotics. A chest radiograph in 1989 revealed RUL atelectasis. She had progressive dyspnea on exertion and recurrent respiratory tract infections over the next 2 years with deterioration of her exercise tolerance. In April 1991, she was hospitalized with RLL atelectasis and pneumonia. Spirometry at that time revealed an FEV₁ and FVC of 1.24 L and 1.99 L, respectively. Although there was a high degree of suspicion that the stenosis was due to sarcoidosis, only chronic inflammation and scarring were seen on biopsy specimens. Fifty milligrams of prednisone daily did not improve her symptoms.

At bronchoscopy the patient had diffuse narrowing of her trachea about 5 cm distal to the vocal cords. The RUL bronchus was obliterated and the bronchoscope could not be passed into the bronchus intermedius. Following the bronchoplasty on two successive days the patient felt markedly improved. Subjectively she was less dyspneic at rest and could perform housework without becoming short of breath. Results of her spirometry were unchanged. She has had repeated bronchoplasty procedures about every 9 months as her symptoms return.

CASE 6

This 32-year-old white man presented with a 6-month history of progressive shortness of breath. He worked as a mail carrier and had been limited by exertional dyspnea. He denied any other pulmonary symptoms. Chest radiograph revealed a RUL infiltrate with primarily right hilar adenopathy. Pulmonary function tests showed FEV₁/FVC of 2.56 L/1.40 L.

Bronchoscopy revealed marked narrowing of the various segmental bronchi. Biopsy specimens showed non-necrotizing granulomas consistent with sarcoidosis. The patient underwent bronchial dilatation on two occasions with significant improvement of his exertional dyspnea. Following bronchoplasty, the FEV₁ and FVC were 2.83 L and 4.66 L, respectively.

DISCUSSION

Sarcoidosis, a granulomatous disease of unknown etiology, usually involves the lungs and classically presents with restrictive physiology. Obstructive disease is common in sarcoidosis, however, and is thought to occur in one of four ways: compression of bronchi by enlarged hilar nodes, cicatricial obstruction of airways due to fibrosis in advanced disease, endobronchial sarcoid causing bronchial and segmental stenosis, and airway hyperreactivity presumably due to active sarcoidosis. The review by Olsen et al. of 99 patients with sarcoidosis revealed 8 with bronchial or segmental stenosis, while 9 of 64 patients with sarcoidosis had airway stenosis in the series of Stjernberg and Thunell. Progression of stenoses can lead to severe dyspnea on exertion and create pulmonary “cripples” even in patients with relatively little parenchymal disease. Fortunately, the response of airway stenoses to steroids is uncertain.

Despite the awareness of bronchial stenosis due to sarcoid, no attempts at dilatation were reported until 1981. Iles used a rigid bronchoscope for a bougie dilatation on four patients with multiple bronchial stenoses, two with sarcoidosis, one with berylliosis, and one from unknown causes. All patients presented with stridor, dyspnea on exertion, and frequent chest infections and all were refractory to corticosteroid therapy. He effected improvement in all patients without significant complication, although some required repeated dilatation. However, he was able to reach only the lower lobes with the rigid scope. Other types of endobronchial dilations have been tried. Kazuyoshi and associates used the flexible fiberoptic bronchoscope to pass a J-wire across a left main-stem stenosis due to tuberculosis and dilate it with an angioplasty catheter under fluoroscopy. Keller and Frost used a pediatric bronchoscope to directly visualize placement of an angioplasty catheter in patients with anastomotic stenosis following lung transplantation.

Our method utilizes the flexibility of the fiberoptic bronchoscope to reach upper lobe and distal segments and still perform dilatation under direct visualization, something not possible with the rigid bronchoscope. In addition, fluoroscopy or external devices added to bronchoscopy are not needed. Although stenosis of upper lobe segments may not contribute significantly to dyspnea in upright patients because of their smaller contribution to gas exchange, dilatation can reduce the incidence of atelectasis and postobstructive infections which are common problems.

Relieving fixed airway obstruction in patients with sarcoidosis is of significant importance. Unlike pa-
tients with obstructing malignant lesions, these patients tend to be younger and have a much longer life expectancy. Airway stenosis is often progressive and refractory to treatment and, as shown by the patients in this and other published series, quite debilitating. Unfortunately, the use of corticosteroids is not reliable in effecting improvement. The improvement in symptoms following dilatation was dramatic in most cases. Only three of our patients have required repeated dilatation on a long-term basis after initial therapy. Our longest postbronchoplasty follow-up is in a patient with excellent exercise tolerance 3 years after dilatation.

Complications or problems from this procedure are minimal. Localized postdilatation bleeding is cleared easily with saline solution washes and is no more prominent than bleeding from endobronchial biopsies. A sensation of pain or discomfort occurs, at times, when the balloon is inflated. This sensation is unpredictable and even in the same individual does not occur in all areas of dilatation. If the segment is a "pinpoint" opening, the biopsy forceps used in the closed, nonbiopsy position is needed to dilate the opening enough to allow the balloon catheter to be placed within the stenosed segment for dilatation. A given area of stenosis may dilate markedly with one 30-s balloon inflation or may need multiple attempts to achieve acceptable dilatation. Achievable airway diameter of greater than 5 mm can often be obtained. However, even if less is obtained, marked clinical improvement occurs as resistance to airflow involves the radius to the fourth power. Objective improvement in pulmonary function testing was not observed, but improvement in exercise testing was usually seen and subjective improvement was uniform. Lastly, we empirically placed patients whose airways appear very inflamed and friable on a 2-day regimen of oral prednisone postprocedure to help decrease the edema and inflammation produced by the bronchoplasty.

In conclusion, we have described an easy and safe method for dilatation of bronchial and segmental stenosis in patients with endobronchial sarcoidosis. Unlike methods previously described, it can be done at the bedside, using local anesthesia with sedation, and under direct visualization giving access to all lobes and segments.

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REFERENCES