Men commonly carry their inhaler in their pants pocket, women in their purses. For convenience, they store it in the “ready” position. The cap will not fit the mouthpiece end at the top (Bronkometer), allowing for coins (pennies, nickels, dimes) to lodge inside. The patient, during inhalation, inhales deeply with maximally opened vocal cords. It is the ideal situation to allow unobstructed passage into the airways. It may be well to warn patients of the possibility that foreign material may lodge in the mouthpiece. Perhaps it would be advisable for the drug companies to make both ends of the mouthpiece the same size (this is true of the metaproterenol [Alupent] inhaler). The patient could then keep the cap on and prevent access to the inside of the mouthpiece. Even then, however, the patient should be instructed to keep the cap on when storing the inhaler in his pocket.

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Pulmonary Complications of Oral BCG

To the Editor:

During the past decade, the use of BCG as an adjuvant cancer therapy mode has increased markedly. The pulmonary sequelae of intraleral scarification technique of administration include granuloma formation and miliary diseases. Few reports of the complications of therapy with oral BCG exist. Although interstitial pneumonia is described, to our knowledge a documented case of BCG air-space pneumonia has not been reported. Our patient

Figure 1. Lateral chest x-ray film showing metallic object in right mainstem bronchus.

Figure 1. Roentgenogram eight months postoperation showing patchy, inhomogeneous consolidation in the left lower lobe. Illustrates the potential of oral BCG to produce such an illness.

Case Report

A 58-year-old white man was begun on a course of monthly oral BCG therapy following left upper lobectomy for a well-differentiated bronchogenic adenocarcinoma. By the third month of the BCG regimen, skin testing with 5 TU resulted in 20 mm induration with blister formation. Cough, productive of white sputum, and left pleuritic pain began eight months postoperatively and one week after the last administered BCG. Chest x-ray examination (Fig 1) revealed a patchy, inhomogeneous consolidation in the left lower lobe. There was no resolution of symptoms with administration of ampicillin for a two-week period. Two sputum samples obtained four weeks after the last oral dose of BCG were positive on smear for acid-fast bacillus and subsequently grew a Mycobacterium species (3+4+ growth). Its culture characteristics were those of the BCG species; furthermore, both the patient’s organism and the BCG organism used for his immunotherapy were of the same phage type. Following institution of isoniazid, ethambutol and rifampicin, there was complete clinical and radiologic resolution of the pneumonia over a two-month period.

Discussion

The failure of resolution of the symptoms with ampicillin therapy and the time course of improvement (radiologically and clinically) with the antituberculosis medications, as well as the positive sputum cultures all support the diagnosis of BCG pneumonia. BCG pneumonia should enter into the differential diagnosis of air-space pneumonia in the patient receiving oral BCG immunotherapy.

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Tracheal Stenosis Due to Sarcoidosis

To the Editor:

Sotenic involvement of the large airways is an unusual complication of sarcoidosis, with bronchial stenosis recently being described.1,2 To our knowledge we report the first case of tracheal stenosis due to sarcoidosis.

CASE REPORT

A 48-year-old black woman was referred to Massachusetts General Hospital for surgical repair of tracheal stenosis due to sarcoidosis. Initial complaints of a decrease in voice strength 30 years prior to admission prompted a non-diagnostic evaluation by a local physician. One year later, skin lesions appeared on her extremities which were biopsied and revealed non-caseating granulomas consistent with sarcoidosis. She received a course of corticosteroids with improvement of her skin lesions but no change in her diminished voice strength. Twenty-five years prior to admission, a chest roentgenogram revealed a diffuse interstitial pattern with nodules. Ten years later, her chest roentgenogram appeared unchanged, but she continued to experience decreasing strength of her voice, and three years prior to admission she experienced her first episode of stridulent breathing. She was only minimally improved with antibiotics and a short course of beclomethasone and systemic corticosteroids. Two years prior to admission, her chest roentgenogram revealed emphysematous changes and bullae in addition to a diffuse interstitial pattern. Tracheal tomograms revealed narrowing of the trachea. Her pulmonary function tests were consistent with an obstructive process. Over the next one and a half years prior to admission she had progressive stridor becoming more difficult to manage with antibiotics and bronchodilators.

On admission to our hospital additional history obtained was negative for smoking, asthma, previous bronchoscopy, or intubation. Physical examination revealed minimal inspiratory stridor. Her skin tests were positive for mumps and negative for PPD. Results of laboratory tests and arterial blood gases were essentially normal. Pulmonary function tests again revealed severe obstructive lung disease. Tracheal tomograms revealed more severe narrowing of the trachea and left mainstem bronchus when compared to films two years earlier (Fig 1). During bronchoscopy, an extensive 5-6 mm narrowing of the trachea was observed and tracheal dilatation was performed. Her inspiratory stridor improved and she was discharged on a prolonged course of corticosteroids and bronchodilators with repeated tracheal dilations anticipated in the future. Sputum cultures for mycobacteria and fungi remained free of growth for over a three-month period.

DISCUSSION

Tracheal involvement due to sarcoidosis has been limited to the description of tracheal dystonia, a weakening of the membranous wall.3 Our patient experienced at least a 30-year history of sarcoidosis with symptoms initially of laryngeal involvement. In the three years prior to admission progressive tracheal stenosis developed manifested by inspiratory stridor and dyspnea, with serial tracheal tomograms demonstrating progressive stenosis of the trachea. This entity appears distinct from tracheal dystonia and may represent a form of end-stage tracheomalacia where the trachea loses its supporting elastic and cartilagenous structures.4

Unlike reported cases of bronchial stenosis due to sarcoid, which has been noted to arrest following treatment with high dose corticosteroids,5 our patient with tracheal stenosis failed to stabilize and deteriorated in spite of several short courses of systemic corticosteroids. Therapy in these patients may be limited to prolonged courses of corticosteroids combined with repeated tracheal dilatations.

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