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Case report

Lung transplantation for bronchiectasis due to smoke inhalation☆,☆☆

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ARTICLE INFO

Article history:
Accepted 11 January 2011

1. Introduction

Smoke inhalation is a challenging clinical problem due to the thermal injury to the respiratory tract and exposure to a large number of inhaled toxins that can lead to the potential development of bronchiectasis. Bronchiectasis is a syndrome characterized by airway dilatation and bronchial wall thickening [1,2]. There are numerous conditions associated with the development of bronchiectasis (Table 1). The pathophysiology of bronchiectasis includes chronic airway inflammation accompanied by chronic infection associated with the presence of a variety of bacterial or fungal infectious agents. Excessive production of abnormal mucus and impaired respiratory secretion clearance are the typical manifestations of the disease. Defects of host defense mechanisms often heighten susceptibility to bronchial wall injury and chronic infection. Bronchial wall and mucosal damage further impairs host defense mechanisms. The resulting clinical consequences are chronic cough and expectoration of abnormal mucus, airway obstruction, and persistent respiratory tract infections.

Bronchiectasis and bronchioloitis obliterans are complications that can result from smoke inhalation injury [3–6]. Sluzker et al. reported the development of bronchiectasis 12 years after smoke inhalation with the onset of hypercapnic respiratory failure and cor pulmonale 20 years following the injury [5]. Bronchiectasis is an indication for lung transplantation [7]. To my knowledge, there are no reported cases of lung transplantation for bronchiectasis resulting from smoke inhalation injury. This report discusses the pathophysiology of smoke inhalation injury while presenting the successful outcome of a young adult female who underwent bilateral sequential lung transplantation for bronchiectasis due to a smoke inhalation injury.

2. Case report

35-year-old female underwent a bilateral sequential lung transplant for progressive bronchiectasis after experiencing a lung injury from a house fire when she was 10-years-old. She and her younger sibling escaped the fire as a result of assistance from a neighbor. She suffered second and third degree burns to 30% of her body, primarily the middle torso and upper and lower extremities. The face and neck were relatively spared except for a few small blisters on her lips and singed nasal hair and eyebrows. She had significant carbonaceous sputum and secretions. She required intubation 24 h after admission due to acute respiratory with the eventual development of acute respiratory distress syndrome (ARDS). Her initial lung exam identified diffuse rhonchi and wheezing with crackles noted on

☆ There was no funding source needed for development of this manuscript. The author has no significant conflicts of interest with any companies or organizations whose products or services may be discussed in this article.
☆☆ Approval by the institutional review board (IRB) at the University of Kentucky was not required.
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doi:10.1016/j.burns.2011.01.011

Table 1 – List of specific causes of bronchiectasis.

Airway adenoma
Allergic bronchopulmonary aspergillosis
Alpha-1-antitrypsin deficiency
Aspiration/gastroesophageal reflux
Bronchial cyst
Bronchocentric granulomatosis
Bronchomalacia
Cartilage deficiency (Williams–Campbell syndrome)
Connective tissue disease
Chronic rejection following organ transplantation (bone marrow, lung and heart–lung transplantation)
Cystic fibrosis
Ectopic bronchus
Endobronchial teratoma
Foreign-body aspiration
HIV/AIDS
Inherited immunodeficiency disorders (IgG deficiency, IgA deficiency, leukocyte dysfunction, CXCR4 mutation, CD40 deficiency, CD40 ligand deficiency, and others)
Inflammatory bowel disease
Inhalation of smoke, toxic fumes, and dusts
Interstitial lung disease
Laryngeal papillomatosis
Nontuberculous mycobacteria
Postinfectious (bacterial, fungal, acid-fast bacteria, Mycoplasma)
Postoperative mucoid impaction
Primary ciliary dyskinesia
Pulmonary (intralobar) sequestration
Pulmonary artery aneurysm
Relapsing polychondritis
Right middle lobe syndrome (can occur in lingula)
Sarcoidosis
Tracheobronchial amyloidosis
Tracheobronchomegaly (Mounier–Kuhn syndrome)
Tracheoesophageal fistula
Yellow-nail syndrome
Young's syndrome

day 3, which was the day she met diagnostic criteria for ARDS. The initial chest X-ray identified a right middle lob infiltrate. Serial bronchoscopies were performed with the initial one demonstrating a pseudomembrane with carbonaceous material present, which was cleared. A tracheostomy was placed due to prolonged mechanical ventilation 1.5 months after intubation. The burns were treated with split thickness skin grafts, which healed very nicely. Respiratory cultures 2 weeks after admission and on multiple occasions thereafter isolated pansensitive *Pseudomonas aeruginosa*. Due to intermittent fevers during the prolonged hospitalization, she was treated with 2 separate courses of ticarcillin and tobramycin. She was decannulated 1 month after the tracheostomy was placed; however, she continued to require supplemental oxygen and was discharged home on 1 L/min (LPM) by nasal cannula. Computed tomography (CT) of the chest was performed during the hospitalization that reported airway dilatation, but an actually copy of the film is not available. Her total hospitalization was 3 months with 2 months of that being in the intensive care unit (ICU). Her discharge medications in 1985 included theophylline 300 mg twice daily, cimetidine 200 mg four times daily, and bronkosol 0.5 mL in 2 mL of normal saline along with percussion and postural drainage.

From a pulmonary function standpoint, spirometry demonstrated very severe airway obstruction immediately. Her forced vital capacity (FVC) was 0.76 L (35% predicted) and her forced expiratory volume in 1 s (FEV_1) was 0.43 L (24% predicted). Over the next 8 years, her pulmonary function remained consistently in the range of 50–60% predicted for the FVC and 20–30% predicted for the FEV_1. When at baseline, she had no supplemental requirement but did require 2 LPM by nasal cannula during times of acute illnesses requiring hospitalization. Her FVC and FEV_1 measurements would drop to 25% predicted and 15%, respectively, during an acute exacerbation of bronchiectasis. Eventually, she was discharged from the pediatric pulmonary service and then was lost to followup.

A local primary physician was providing care during her young adult years and eventually referred her to be evaluated for lung transplantation at 31 years of age. Her quality of life was significant declining because of severe dyspnea with a supplemental oxygen requirement of 2 LPM. Upon evaluation,

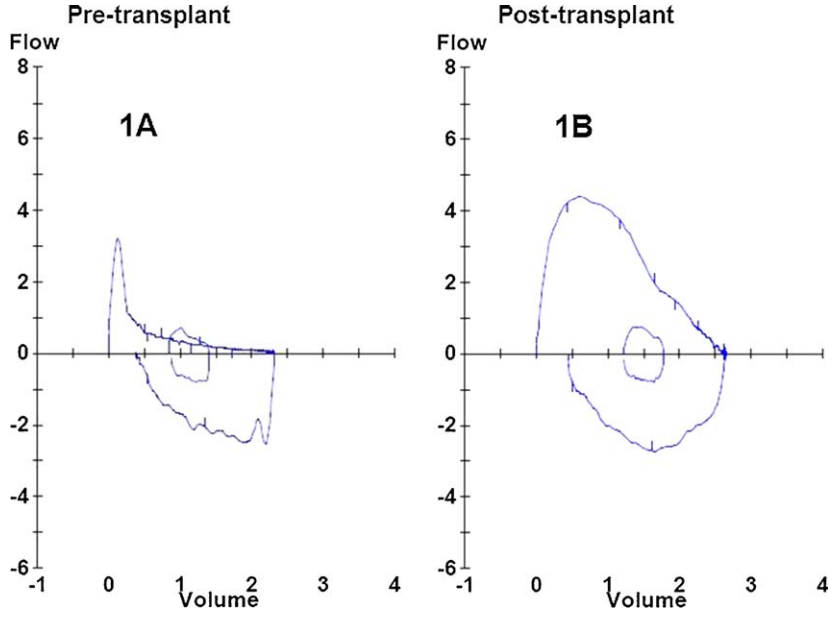

Fig. 1 – Flow–volume curves. (A) Pre-lung transplant and (B) post-lung transplant.