Bronchial Thermoplasty

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KEYWORDS

• Bronchial thermoplasty • Airway smooth muscle • Severe asthma

KEY POINTS

- Bronchial thermoplasty (BT) is a bronchoscopic procedure that involves the direct application of thermal energy to the airways and is performed at 3 separate sessions spaced 3 weeks apart.
- BT leads to the destruction of airway smooth muscle (ASM), because ASM plays an important role in the pathophysiology of asthma.
- The procedure is approved for patients with severe asthma and results in improvement in quality of life and decreased asthma exacerbations.

A video demonstrating BT accompanies this article at www.oto.theclinics.com

INTRODUCTION

Bronchial thermoplasty (BT) was approved by the Food and Drug Administration (FDA) in April 2010[1] and offers a novel add-on treatment of the management of severe asthma by reducing smooth muscle mass via direct application of thermal energy. Severe asthma is a widespread problem globally. There are 22.9 million Americans in the United States with asthma,[2] and severe persistent asthma constitutes up to 12%[3–5] yet disproportionately utilizes more direct and indirect health care dollars for asthma care.[6–8]

SEVERE ASTHMA

Severe persistent asthma consists of asthma symptoms throughout the day, nighttime awakenings often 7 times a week, need for short-acting β-agonists several times a day, and extreme limitations in daily activity as well as forced expiratory volume in 1 second (FEV_1) less than 60% predicted (without medications).[9] Risk factors for

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Abbreviations	
AIR	Asthma Intervention Research
ASM	Airway smooth muscle
ATS	American Thoracic Society
BT	Bronchial thermoplasty
FEV_1	Forced expiratory volume in 1 second
LABA	Long-acting β-agonist

severe asthma include being female (postpuberty), obesity, black race, significant secondhand smoke exposure, and comorbidities of gastroesophageal reflux, sinus infections, and pneumonia.[10] Although control is the ultimate goal in the management of asthma, complete control in many with severe persistent asthma is often elusive,[11] leading to several terms to describe this patient group, including *severe asthma*, *steroid-dependent or resistant asthma*, *difficult or poorly controlled asthma*, and *brittle or irreversible asthma*.[12] In 2000, the "Proceedings of the ATS Workshop on Refractory Asthma" agreed on the term, *refractory asthma*, to describe those asthma cases requiring a high amount of medications to maintain control or those cases of persistent symptoms, airflow obstruction, and frequent exacerbations despite high medication use.[12] Diagnosis requires all other diseases with similar presenting symptoms to have been ruled out, comorbid conditions adequately managed, and medication technique and adherence issues addressed.[12]

The lack of therapeutic effectiveness or response to traditional medications in this patient group raises the question of differing airway pathophysiology compared with mild or moderate asthmatics[12] and has led to a discussion of asthma being a heterogeneous disease with varying manifestations or responses to treatment.[13] Several distinct phenotypes are evolving when comparing clinical and physiologic features, prominence of biomarkers, age of disease onset, and response to therapy; however, additional research is needed for full characterization.[10,14,15] The movement from symptom-based therapy decisions to medications or interventions targeting the underlying physiologic features of the disease will pave the way to personalized treatment plans, with the ultimate goal of improved control for refractory asthmatics.

PATHOPHYSIOLOGY

One important target of current asthma therapy is ASM because it is critically involved in asthma pathogenesis. Acutely, ASM cell contraction causes bronchoconstriction. Chronically, hypertrophy and hyperplasia of ASM are responsible for airway remodeling.[9] Additionally, through interactions with other cells, such as mast cells, and by generating and releasing inflammatory mediators, ASM cells are responsible for some of the airway inflammation seen with asthma.[16] Medications, such as short-acting bronchodilators, long-acting bronchodilators, leukotriene inhibitors, and theophylline, play an important role in asthma management by relaxing the ASM. BT is used as a new approach to treatment by structurally modifying the airway and destroying smooth muscle. If ASM is to be destroyed, it is important to believe that ASM does not have a physiologic role. Investigators have debated if ASM has a beneficial or physiologic role or whether it is similar to a vestigial organ and serves no useful purpose.[17] This debate has not been settled; many researchers and clinicians have proposed possible roles for ASM.[18] Refractory asthmatics present a crucial opportunity to better understand the underlying pathophysiology of asthma and the