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Bronchial Thermoplasty Management in Severe Asthma

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Abstract

Bronchial thermoplasty was first published in 2007 by Mayse et al. as a treatment for severe asthma. Studies such as AIR2, Seely et al., and others demonstrated improvements in quality of life, symptom reduction, and frequency of exacerbations in asthma patients treated with bronchial thermoplasty. It is crucial for clinicians to recognize this method as a potential treatment for severe asthma. This paper was prepared based on a literature review of journals published in the past five years. Bronchial thermoplasty was considered for patients with severe asthma that is not of the type 2 inflammatory nature. Severe asthma with type 2 inflammation is characterized by blood eosinophil count > 150 μl, FeNO > 20 ppb, sputum eosinophils > 2%, and asthma clinically controlled by allergens. Bronchial thermoplasty involves inserting a basket catheter into the airways, guided by bronchoscopy, to deliver thermal energy that reduces smooth muscle mass, decreases nerve fibers in the submucosa and smooth muscle, and suppresses airway hyperresponsiveness, thus alleviating airway obstruction in severe asthma. In conclusion, bronchial thermoplasty is a treatment for severe asthma that is not of the type 2 inflammatory nature. The mechanism of action of bronchial thermoplasty involves delivering thermal energy to the bronchial walls to widen the airways and suppress airway hyperresponsiveness.

Keywords: bronchial thermoplasty; severe asthma; asthma treatment

1. Introduction

Background

A population-based study conducted by Ronnebjerg et al. between 2009 and 2012 in Sweden evaluated 18,087 individuals aged 16–75 years and identified 744 patients with physician-diagnosed asthma. Among these, 9.5% (n = 71) were classified as having severe asthma. Within the severe asthma subgroup, 29.5% were receiving treatment consistent with Step 5 therapy according to the Global Initiative for Asthma (GINA) recommendations, whereas 70.4% were managed with Step 4 therapy. The authors further reported a higher prevalence of severe asthma among females (61.8%) compared with males, suggesting a potential sex-related difference in disease severity distribution.[(1)]

In a multinational cohort analysis by Wang et al., data were derived from the International Severe Asthma Registry between December 2014 and December 2017. The registry included patients from several countries, namely the United States, United Kingdom, South Korea, Italy, Singapore, Australia, and New Zealand. Among 4,990 patients with severe asthma, the majority were female (59.3%), predominantly White (72.6%), aged 55–79 years (52.1%), and classified as overweight or obese (70.4%). These findings highlight the demographic and clinical characteristics commonly associated with severe asthma in real-world clinical settings.(2)

Bronchial thermoplasty represents a non-pharmacological therapeutic modality for the management of severe asthma. This intervention employs controlled radiofrequency energy, which is converted into thermal energy to reduce airway smooth muscle mass. The underlying rationale is that attenuation of airway smooth muscle hypertrophy may decrease bronchoconstriction, thereby improving symptom control and enhancing health-related quality of life. Bronchial thermoplasty is primarily indicated for patients with severe asthma who remain inadequately controlled despite optimized pharmacological treatment in accordance with established guidelines.(3)

The Asthma Intervention Research 2 (AIR2) trial conducted in the United States in 2008 demonstrated significant improvements in Asthma Quality of Life Questionnaire (AQLQ) scores among patients with severe asthma who underwent bronchial thermoplasty compared with those receiving standard medical therapy alone, consisting of high-dose inhaled corticosteroids (ICS) and long-acting β2-agonists (LABA). Specifically, 80.9% of patients in the bronchial thermoplasty group achieved a clinically meaningful improvement in AQLQ scores relative to baseline, whereas 63.2% of patients in the control group demonstrated similar improvement. Additionally, the proportion of patients experiencing severe exacerbations was lower in the bronchial thermoplasty group (2.9%) compared with the control group (7.9%), underscoring the potential clinical benefits of this interventional approach.(4)

Bronchial thermoplasty has been approved by the Food and Drug Administration (FDA) since 2010 for adults with severe asthma inadequately controlled despite optimal medical therapy, and the 2023 update of the Global Initiative for Asthma (GINA) guidelines recognizes it as an alternative treatment option in selected patients with severe asthma.(5,6) First introduced in 2007 by Mayse and colleagues, bronchial thermoplasty has been evaluated in several clinical trials, including the AIR2 Trial, which demonstrated improvements in asthma-related quality of life, symptom control, and reductions in exacerbation frequency.(17,20) Therefore, increased clinician awareness is warranted, and a comprehensive understanding of its definition, mechanism of action, indications, and potential complications remains essential to inform its role in the management of severe asthma.

Method

This literature review was conducted through a systematic search and selection of relevant and up-to-date sources from established academic databases, including Google Scholar, Scopus, and PubMed. The materials included peer-reviewed journal articles, textbooks, and scholarly publications issued within the past five years. Each identified source was critically appraised for methodological quality and relevance to the research topic prior to inclusion. The selected literature was subsequently organized into major thematic domains pertinent to the subject matter, followed by systematic analysis and comparison of findings to provide a comprehensive and balanced perspective. In preparing this manuscript, careful attention was given to the application of citation and referencing standards in accordance with the requirements of the target journal, ensuring full compliance with internationally accepted scientific publication guidelines.

Discussion

Definition and Epidemiology of Severe Asthma

Severe asthma, as defined by the European Respiratory Society (ERS) and the American Thoracic Society (ATS), refers to asthma that remains controlled only with high-dose inhaled corticosteroids (ICS) in combination with high-dose long-acting β_2-agonists (LABA), often requiring additional controller therapies such as tiotropium, leukotriene modifiers, or maintenance oral corticosteroids. Alternative definitions in the literature further emphasize that severe asthma is characterized by the need for sustained high-dose ICS–LABA therapy and clinical deterioration when treatment intensity is reduced.(7)

Clinically, severe asthma is marked by persistent symptoms and frequent exacerbations despite optimization of modifiable factors associated with treatment success. These factors include adherence to prescribed therapy, appropriate pharmacologic selection, correct inhaler technique, regular clinical follow-up, avoidance of known triggers, and adequate management of comorbid conditions. This terminology distinguishes severe asthma from "difficult-to-treat" asthma, which refers to uncontrolled disease primarily attributable to remediable factors such as incorrect diagnosis, ongoing smoking exposure, unmanaged comorbidities, poor adherence, or improper inhaler technique rather than true therapy-refractory pathophysiology.(7)

According to the 2023 report from the Global Initiative for Asthma (GINA), approximately 3.7% of all individuals with asthma meet criteria for severe asthma.(7) In a population-based study conducted in Sweden between 2009 and 2012, Ronnebjerg and colleagues evaluated 18,087 respondents aged 16–75 years and identified 744 individuals with asthma, of whom 9.5% (n=71) were classified as having severe asthma. Among these patients, 29.5% were receiving GINA Step 5 therapy, while 70.4% were managed with GINA Step 4 treatment. The study also reported a higher prevalence of severe asthma among women (61.8%) compared with men.(1)

Furthermore, an analysis by Wang et al., based on data from the International Severe Asthma Registry collected between December 2014 and December 2017 across the United States, United Kingdom, South Korea, Italy, Singapore, Australia, and New Zealand, included 4,990 patients with severe asthma. The majority of patients were female (59.3%), White (72.6%), aged 55–79 years (52.1%), and overweight or obese (70.4%). Approximately half of the cohort (50%) received maintenance oral corticosteroid therapy, and nearly one-quarter (24%) were treated with biologic agents, including anti-IgE and anti–interleukin-5 therapies. Most patients also received add-on leukotriene receptor antagonists (LTRA), followed by long-acting muscarinic receptor antagonists (LAMA), reflecting contemporary multidimensional management strategies for severe asthma.(2)

Airway Smooth Muscle Remodeling in Severe Asthma

Structural alterations of airway smooth muscle (ASM) represent a key feature of airway remodeling in severe asthma. A study conducted by James demonstrated that airway smooth muscle hypertrophy may occur in both non-severe and severe asthma; however, smooth muscle cell hyperplasia was observed exclusively in patients with severe asthma. The combined processes of hypertrophy and hyperplasia of ASM cells in severe asthma contribute to enhanced angiogenesis and increased deposition of extracellular matrix components. Excessive extracellular matrix accumulation promotes airway fibrosis and leads to increased airway resistance, thereby exacerbating airflow limitation.[8]

Beyond structural remodeling, ASM cells actively participate in the pathophysiology of severe asthma through immunomodulatory mechanisms. Airway smooth muscle is capable of inducing airway hyperresponsiveness by releasing pro-inflammatory cytokines and chemokines that augment smooth muscle contraction and alter relaxation kinetics. Furthermore, ASM expresses muscarinic (M) receptors that respond to parasympathetic (cholinergic) neural stimulation. Muscarinic receptors are also present in airway mucous glands. Parasympathetic signaling through these receptors mediates bronchoconstriction, mucus hypersecretion, airway inflammation, and structural remodeling, collectively contributing to the persistent airflow obstruction characteristic of severe asthma.[9]

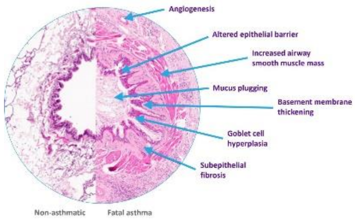

Figure 1. Airway Smooth Muscle Remodeling in Severe Asthma[10]

Mechanism of Action of Bronchial Thermoplasty

Bronchial thermoplasty is a non-pharmacological intervention designed to reduce airway smooth muscle (ASM) mass through the controlled delivery of radiofrequency energy, which is converted into thermal energy and applied to the airway wall. The procedure is performed bronchoscopically by introducing a specialized catheter that delivers radiofrequency energy to targeted segments of the bronchial wall. This technique was first performed in 2007 and subsequently reported by the Washington University School of Medicine, Division of Pulmonary and Critical Care Medicine.[20] Since 2010, bronchial thermoplasty has been approved as a safe therapeutic modality for severe asthma by the Food and Drug Administration (FDA).[5]

Bronchial thermoplasty is indicated for adults aged 18 years and older with severe asthma that remains inadequately controlled despite high-dose inhaled β_2-agonists and long-term corticosteroid therapy. The procedure reduces airway smooth muscle mass via bronchoscopic insertion of a basket-shaped catheter that delivers high-frequency electrical energy to narrowed bronchial segments. In addition to decreasing ASM bulk, bronchial thermoplasty has been shown to reduce submucosal nerve fibers and attenuate airway hyperresponsiveness, thereby contributing to improved airway stability and reduced bronchoconstrictive responses in patients with severe asthma.[4]

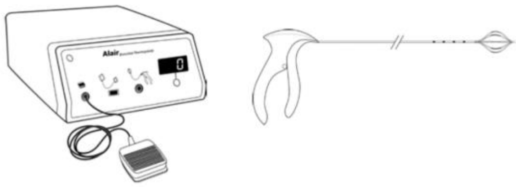

Figure 2. Bronchial Thermoplasty[11]

Bronchial thermoplasty is performed using the Alair Bronchial Thermoplasty System, which comprises a radiofrequency controller, a footswitch, a dispersive (grounding) electrode, and the Alair catheter. The Alair catheter is compatible with a bronchoscope featuring an internal diameter of ≥ 2 mm and an external diameter of ≤ 5 mm, enabling access to distal bronchi with a luminal diameter of ≥ 3 mm. The distal tip of the catheter is equipped with four expandable electrodes that deploy in a basket configuration to establish circumferential contact with the airway wall. Radiofrequency energy generated by the controller is converted into controlled thermal energy and delivered through the catheter electrodes to the bronchial wall at a target temperature of 65°C for 10 seconds per activation. This localized thermal application results in a sustained reduction in airway smooth muscle mass. Consequently, the diminished smooth muscle burden leads to decreased airway contractility during asthma exacerbations, thereby reducing both the frequency and severity of asthma attacks.[9]

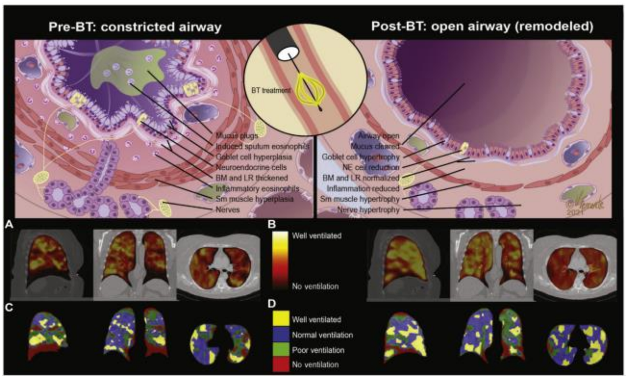

Figure 3. Changes in the respiratory tract after bronchial thermoplasty[12]

Severe asthma is characterized by airway narrowing and structural remodeling, including epithelial hyperplasia, airway smooth muscle hypertrophy and hyperplasia, goblet cell hyperplasia, thickening of the basement membrane and lamina reticularis, and persistent eosinophilic airway inflammation (Figure 3, left panel: pre–bronchial thermoplasty). Bronchial thermoplasty has been shown to reduce airway smooth muscle mass, decrease epithelial mucin production, attenuate basement membrane thickening, and diminish neuroendocrine and epithelial nerve fiber density (Figure 3, right panel: post–bronchial thermoplasty). These structural and cellular modifications are associated with functional improvement in ventilation among patients with severe asthma. Imaging findings support these observations, as demonstrated by xenon-enhanced magnetic resonance imaging (Xe-MRI) and proton MRI, which reveal improved ventilation patterns following bronchial thermoplasty (Figure 3, lower panel: proton MRI before and after bronchial thermoplasty; volumetric computed tomography combined with MRI illustrating ventilation changes before and after intervention).[12]

In a prospective study conducted in Italy between March 2013 and June 2014, Facciolongo and colleagues performed endobronchial biopsies in 12 patients with severe asthma at baseline (T0), 1 month (T1), 2 months (T2), and 12 months (T12) after bronchial thermoplasty, with additional immunohistochemical analyses. The findings demonstrated an early and sustained reduction in epithelial nerve fibers and airway smooth muscle cells, persisting for up to one year following the procedure, thereby providing histopathological evidence of the long-term structural effects of bronchial thermoplasty.[13]

Indications for Bronchial Thermoplasty

Bronchial thermoplasty was approved in 2010 by the Food and Drug Administration (FDA) for the treatment of patients with severe asthma who meet specific eligibility criteria. These include: (1) age ≥ 18 years; (2) persistent uncontrolled asthma despite treatment with inhaled corticosteroids (ICS) and long-acting β_2-agonists (LABA); (3) post-bronchodilator forced expiratory volume in one second (FEV_1) variability of less than 15% from baseline; (4) absence of respiratory infection or asthma exacerbation within the preceding two weeks; (5) no implanted electronic medical devices, such as pacemakers or defibrillators; (6) no known hypersensitivity to medications commonly used during bronchoscopy, including atropine, lidocaine, or benzodiazepines; (7) no history of coagulation disorders; and (8) non-smoking status, or former smokers who have abstained for at least one year with a cumulative smoking history of fewer than 10 pack-years.[9]

The 2023 update of the Global Initiative for Asthma (GINA) guidelines incorporates bronchial thermoplasty as a potential treatment consideration in selected patients with severe asthma. Specifically, bronchial thermoplasty may be considered in individuals with severe asthma who do not exhibit type 2 inflammatory characteristics. Severe asthma with type 2 inflammation is defined by biomarkers such as blood eosinophil counts >150 cells/μL, fractional exhaled nitric oxide (FeNO) levels >20 ppb, sputum eosinophils >2%, or clinically allergen-driven disease. Assessment of blood eosinophils and FeNO should be repeated at least three times, ideally 1–2 weeks after completion of oral corticosteroid therapy or during periods of lowest corticosteroid dosing, to ensure accurate phenotypic classification prior to therapeutic decision-making.[7]

Contraindications to Bronchial Thermoplasty

Contraindications to bronchial thermoplasty are classified as absolute and relative. Absolute contraindications include: (1) the presence of implanted electronic devices such as pacemakers, defibrillators, or other electronic implants; (2) a history of acute myocardial infarction within the preceding six weeks; (3) severe cardiopulmonary disease rendering the patient unable to tolerate bronchoscopy; (4) documented hypersensitivity to anesthetic agents required for the bronchoscopic procedure; (5) uncorrectable coagulation disorders; and (6) prior completion of bronchial thermoplasty treatment. Relative contraindications include: (1) inability to discontinue anticoagulant or antiplatelet therapy due to underlying medical conditions; (2) uncontrolled asthma associated with severe impairment of pulmonary function; (3) a history of near-fatal asthma exacerbations; and (4) other significant comorbidities that remain inadequately controlled. Careful patient selection and comprehensive pre-procedural assessment are therefore essential to optimize safety and clinical outcomes in candidates for bronchial thermoplasty.[9]

Bronchial Thermoplasty Procedure

Bronchial thermoplasty is performed by advancing a basket catheter into the airway under bronchoscopic guidance. The bronchial thermoplasty catheter is introduced through the working channel of the bronchoscope, and radiofrequency energy is delivered via expandable electrodes located at the distal tip of the catheter. This radiofrequency energy is converted into controlled thermal energy and applied to the bronchial wall at approximately 60°C for 10 seconds per activation. Energy delivery is initiated in the most distal accessible airways and sequentially applied in a stepwise manner toward the proximal segments to ensure systematic treatment coverage.(3)

The procedure is typically conducted in three separate sessions. The first session targets the right lower lobe, followed by treatment of the left lower lobe during the second session. The final session involves both upper lobes. Current recommendations advise against treating the right middle lobe due to its relatively long and narrow bronchus, as thermal application in this region may increase the risk of chronic injury and predispose to right middle lobe syndrome secondary to post-procedural inflammation. Careful adherence to procedural protocols is essential to optimize safety and therapeutic outcomes.(3)

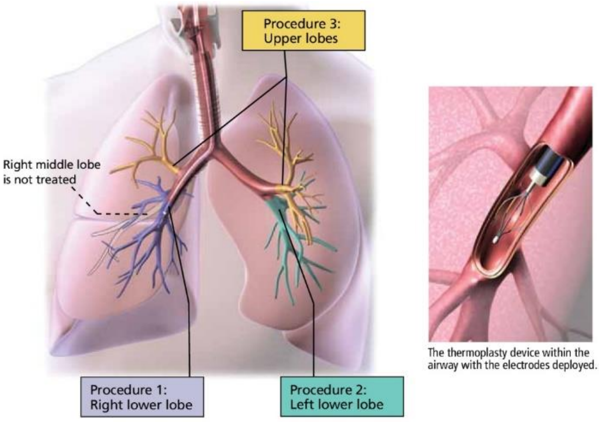

Figure 4. Bronchial thermoplasty procedure (14)

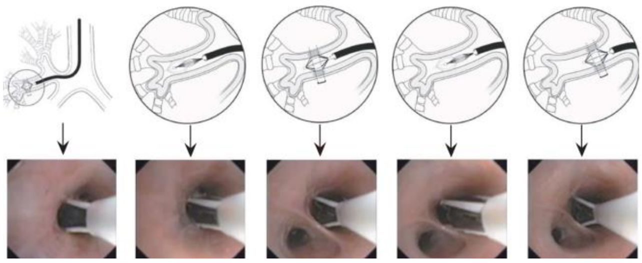

Figure 5. The process of delivering heat energy through the Alair catheter electrode (20)

Clinical Outcomes of Bronchial Thermoplasty

To date, no global registry has established the total number of patients with severe asthma who have undergone bronchial thermoplasty. Clinical efficacy has generally been evaluated using objective and patient-reported outcome measures, including changes in airway luminal volume, Mini Asthma Quality of Life Questionnaire (mini-AQLQ) scores, and the frequency of severe exacerbations. In a study conducted in Australia, Langton et al. (2019) utilized computed tomography (CT) imaging to assess airway volume following bronchial thermoplasty and demonstrated a significant increase in luminal airway volume in treated regions compared with untreated control lungs.(15)

Similarly, Seely and colleagues evaluated 25 patients with persistent severe asthma who underwent bronchial thermoplasty at El Camino Hospital between January 2011 and November 2013. At one-year follow-up, significant improvements were observed in mini-AQLQ scores, accompanied by a reduction in asthma medication use. Overall, 88% of patients demonstrated clinically meaningful improvement in quality-of-life measures and decreased pharmacologic requirements one year after the procedure.(16)

The Asthma Intervention Research 2 Trial (AIR2), conducted across six centers in the United States with follow-up beginning in 2008, included patients aged 18–65 years with severe asthma receiving high-dose inhaled corticosteroids (≥1000 µg/day beclomethasone equivalent) and long-acting β_2-agonists (≥100 µg/day salmeterol equivalent). Participants were randomized to bronchial thermoplasty or control management. The study demonstrated a lower incidence of severe exacerbations in the bronchial thermoplasty group (2.4%) compared with the control group (7.4%), providing further evidence of the procedure's effectiveness in reducing clinically significant exacerbations in patients with severe asthma.(4)

Adverse Effects and Complications of Bronchial Thermoplasty

Bronchial thermoplasty may be associated with short-term adverse events, primarily related to transient airway inflammation following thermal energy delivery. These include bronchial wall edema, increased mucus production, and bronchospasm. Such events are generally self-limited and typically respond to standard medical management with bronchodilators and systemic or inhaled corticosteroids. In addition to these acute effects, bronchial thermoplasty induces structural airway modifications, including reduction of airway smooth muscle mass, decreased epithelial mucin expression, attenuation of basement membrane thickening, and a reduction in bronchial neuroendocrine nerve fibers. These changes are considered therapeutic mechanisms rather than complications, as they contribute to long-term improvement in airway stability and hyperresponsiveness.(17)

With the emergence of biologic therapies for severe asthma, targeted monoclonal antibody treatments have become integral components of disease management. Agents directed against interleukin-5 (IL-5), such as Mepolizumab and Benralizumab, as well as therapies targeting IL-4, IL-13, and thymic stromal lymphopoietin (TSLP), have expanded therapeutic options for patients with type 2 inflammatory phenotypes. Given that bronchial thermoplasty is an invasive procedure, current clinical practice generally prioritizes biologic therapy in appropriately selected patients before considering bronchial thermoplasty as an alternative intervention.(17)

The Research in Severe Asthma (RISA) trial conducted in the United States in 2008 reported that 27% of patients who underwent Bronchial Thermoplasty required at least one hospitalization following the procedure, whereas no hospital admissions were observed in the control group. Hospitalizations in the intervention arm were primarily attributed to asthma exacerbations or partial collapse of the lower lung lobes occurring within 1–2 days after treatment.(4) Similarly, the Asthma Intervention Research 2 study conducted in the United States in 2008 demonstrated that 8.4% of patients who underwent bronchial thermoplasty required post-procedural hospitalization, predominantly due to asthma exacerbations or segmental atelectasis, compared with 2% in the control group. However, no statistically significant increase in long-term adverse events was observed in the bronchial thermoplasty group after completion of treatment, and improvements in respiratory symptoms were documented following therapy.[(4)]

A study conducted by Chaudhuri et al. between 2018 and 2019 in the United States evaluated thoracic computed tomography (CT) findings in 260 patients with severe asthma who underwent bronchial thermoplasty. Thoracic CT imaging revealed that 7% of patients developed mild bronchiectasis after the procedure. This airway dilation was transient in nature and was associated with a reduction in airway resistance.[(18)] Furthermore, a study by Soo et al. conducted in Malaysia between 2012 and 2018 reported clinical outcomes among patients with severe asthma treated with bronchial thermoplasty. Of the 20 patients included, 12 (71%) experienced mild to moderate exacerbations during the first week following the procedure. Two patients (10%) developed severe exacerbations requiring intubation. Additional complications included atelectasis secondary to sputum retention. No post-procedural mortality was reported.[(19)]

Conclusion

Bronchial Thermoplasty is an interventional therapeutic modality for severe asthma that utilizes radiofrequency energy, which is converted into thermal energy, to reduce airway smooth muscle mass. By targeting and diminishing airway smooth muscle, this procedure addresses a key pathophysiological component of airway hyperresponsiveness.

Bronchial thermoplasty has been demonstrated to improve patients' quality of life by reducing asthma-related symptoms and decreasing the frequency of exacerbations. In addition, the intervention contributes to an increase in bronchial lumen diameter and enhances overall ventilatory function in patients with severe asthma.

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