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Emerging Variants of Bronchial Asthma and Innovative Therapeutic Modalities: From Pathophysiology to Precision Medicines

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Abstract

Background: *Bronchial asthma is a heterogeneous chronic inflammatory disease of the airways. Traditional classifications into allergic and non-allergic asthma have been challenged by the discovery of new phenotypes and endotypes defined by molecular, immunological, and clinical characteristics.*
Emerging variants include obesity-associated asthma, neutrophilic asthma, aspirin-exacerbated respiratory disease (AERD), and late-onset eosinophilic asthma.

Objectives: *This review aims to summarize recent evidence on newly recognized clinical and molecular variants of bronchial asthma and to highlight innovative treatment modalities, with a focus on biologics, small-molecule inhibitors, immunotherapy, and precision medicine strategies.*

Methods*: A narrative review of the literature was conducted using PubMed, Scopus, and Web of Science databases for studies published between 2010 and 2025. Studies were included if they described new phenotypes or endotypes of asthma and/or evaluated novel therapeutic interventions.*

Results: *Newly described asthma variants exhibit unique pathophysiological features and differential therapeutic responses. For example, neutrophilic asthma demonstrates steroid resistance but responds to macrolides and IL-17-targeted therapies. Biologic therapies—including anti-IgE, anti- IL-5, anti-IL-4/13, and anti-TSLP monoclonal antibodies—have transformed management of severe eosinophilic and allergic asthma. Novel interventions such as bronchial thermoplasty, JAK inhibitors, and microbiome-modulating therapies are under active investigation.*

Conclusion: *Recognition of asthma heterogeneity has redefined its diagnosis and treatment. Precision medicine approaches tailored to specific phenotypes and endotypes promise improved outcomes for patients with severe or treatment-resistant asthma. Continued research into molecular mechanisms and innovative therapies is essential to advance individualized care in bronchial asthma.*

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Introduction

Bronchial asthma is a chronic inflammatory disease of the airways characterized by variable airflow obstruction, airway hyperresponsiveness, and recurrent episodes of wheezing, cough, chest tightness, and dyspnea [1]. Globally, asthma affects more than 262 million people and accounts for nearly half a million deaths annually, posing a major public health burden [2]. Traditionally, asthma has been classified into allergic (extrinsic) and non-allergic (intrinsic) types. However, accumulating evidence over the last two decades has revealed that asthma is a highly heterogeneous disorder with multiple phenotypes and endotypes that differ in clinical presentation, immunological mechanisms, and therapeutic responses [3,4].

Heterogeneity of Asthma

Asthma heterogeneity is shaped by genetic predisposition, environmental exposures, and immune dysregulation [5]. Novel phenotypic variants include obesity-associated asthma, neutrophilic asthma, aspirin-exacerbated respiratory disease (AERD), late-onset eosinophilic asthma, and occupational/environmental asthma subtypes [6-10]. Each phenotype demonstrates distinct inflammatory patterns—eosinophilic, neutrophilic, mixed granulocytic, or paucigranulocytic—that influence disease severity and responsiveness to corticosteroids [11].

Molecular Endotypes

Advances in molecular profiling have further refined classification into "endotypes" based on underlying immunopathology. Type 2 (T2)-high asthma, driven by IL-4, IL-5, and IL-13 signaling, typically presents with eosinophilia, elevated fractional exhaled nitric oxide (FeNO), and good corticosteroid responsiveness [12]. In contrast, T2-low asthma, characterized by neutrophilic or paucigranulocytic inflammation, shows poor steroid response and remains a therapeutic challenge [13]. Emerging biomarkers such as periostin, blood eosinophils, and sputum cell differentials are being validated for guiding precision medicine [14,15].

Global Burden and Unmet Needs

Despite advances in therapy, approximately 5–10% of asthma patients suffer from severe, treatment-resistant disease [16]. These patients account for a disproportionate share of healthcare utilization and costs [17]. Conventional therapies—including inhaled corticosteroids (ICS) and long- acting beta-agonists (LABA)—are insufficient in many cases, particularly in non-T2 asthma variants [18]. This unmet need has driven the development of novel biologics, small-molecule inhibitors, and interventional therapies.

New Therapeutic Modalities

Biologics targeting IgE (omalizumab), IL-5 (mepolizumab, reslizumab, benralizumab), IL-4/IL-13 (dupilumab), and thymic stromal lymphopoietin (tezepelumab) have demonstrated substantial efficacy in reducing exacerbations and improving lung function in severe eosinophilic and allergic asthma [19,20]. Novel small molecules such as Janus kinase (JAK) inhibitors and CRTH2 antagonists are under clinical evaluation [21]. Bronchial thermoplasty, a non-pharmacologic intervention that reduces airway smooth muscle mass, has shown benefit in select patients with severe refractory asthma [22].

The Promise of Precision Medicine

The recognition of distinct phenotypes and endotypes underscores the shift toward precision medicine in asthma management. Integrating clinical, molecular, and biomarker data enables tailored therapeutic approaches that optimize outcomes and minimize adverse effects [23]. Future strategies include microbiome-targeted therapies, epigenetic modulators, and personalized digital health tools for monitoring [24-26].

Thus, bronchial asthma should no longer be viewed as a single disease but as a spectrum of related disorders. Understanding these new variants and their underlying mechanisms is essential for designing effective treatment modalities and achieving long-term disease control. This review aims to provide an updated overview

of emerging asthma phenotypes and endotypes, highlight novel therapeutic strategies, and discuss implications for clinical practice and research.

Methodology

Study Design

This article was conducted as a narrative review with systematic elements, synthesizing evidence on emerging variants of bronchial asthma and novel therapeutic modalities. The methodology followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines where applicable.

Literature Search Strategy

A comprehensive literature search was performed in PubMed/MEDLINE, Scopus, and Web of Science databases to identify relevant publications between January 2010 and May 2025. Additional references were obtained by manual review of bibliographies from key review papers and clinical guidelines.

The following keywords and MeSH terms were combined using Boolean operators (AND, OR):

- "Asthma" OR "Bronchial Asthma"
- "Phenotype" OR "Endotype" OR "Variants"
- "Obesity-Associated Asthma" OR "Neutrophilic Asthma" OR "Aspirin-Exacerbated Respiratory Disease (AERD)" OR "Eosinophilic Asthma"
- "Biologic Therapy" OR "Monoclonal Antibodies" OR "Bronchial Thermoplasty" OR "Precision Medicine" OR "Novel Treatment"

Searches were limited to human studies, English language, and peer-reviewed journals.

Inclusion Criteria

Studies were included if they met the following criteria:

- Focused on newly identified clinical variants or molecular endotypes of bronchial asthma.
- Reported therapeutic outcomes of novel interventions (e.g., biologics, small molecules, bronchial thermoplasty).
- Study design: randomized controlled trials (RCTs), observational studies, meta-analyses, systematic reviews, or high-quality narrative reviews.
- Published between 2010–2025.

Exclusion Criteria

- Non-English publications.
- Case reports with fewer than 5 patients.
- Conference abstracts, letters to editors, and opinion pieces lacking original data.
- Studies addressing asthma in the context of COPD overlap without clear stratification.

Data Extraction and Analysis

Two independent reviewers (including the author) screened titles and abstracts for relevance. Full- text articles were retrieved for detailed evaluation. Extracted data included:

- Author(s), year, and country of study
- Population characteristics and sample size
- Asthma variant or endotype studied
- Intervention or treatment modality investigated
- Outcomes (exacerbation rate, lung function, biomarkers, quality of life)

Data were synthesized narratively, with emphasis on patterns across phenotypes/endotypes and comparisons of therapeutic effectiveness. Where available, results from randomized controlled trials and meta-analyses were prioritized to strengthen evidence.

Results

Emerging Variants of Bronchial Asthma

Recent clinical and molecular research confirms that asthma is not a single entity but a heterogeneous syndrome. Distinct phenotypes and endotypes demonstrate unique clinical profiles, inflammatory pathways, and treatment responses. Among the most important:

- **Obesity-Associated Asthma:** characterized by systemic inflammation, altered lung mechanics, and poor corticosteroid response.
- **Neutrophilic Asthma:** defined by Th17-driven neutrophilia, frequent exacerbations, and resistance to ICS.
- **Aspirin-Exacerbated Respiratory Disease (AERD):** triad of asthma, chronic rhinosinusitis with nasal polyps, and NSAID hypersensitivity.
- **Late-onset Eosinophilic Asthma:** adult-onset, severe, with persistent airway eosinophilia.
- **Occupational/Environmental Asthma:** triggered by workplace allergens/irritants.

Table 1: Major Emerging Variants of Asthma

Variant	Pathophysiology	Clinical Features	Prognosis	Refer ences
Obesity- associated	Adipokine im-balance, systemic inflammation	Dyspnea, poor control, female predominance	Chronic, poor ICS response	[6],
[35]				
Neutrophilic	IL-17 driven,	Steroid resistant,	Severe, limited therapy	[7],
AERD	Dysregulated ara-chidonic acid	NSAID intolerance, polyps, severe asthma	Severe but respon-sive to leukotriene antagonists	[8]
Late-onset eosino-philic	IL-5 mediated air-way eosinophilia	Adult-onset, fre-quent exacerbations	Biologics highly effective	[9],
[30]				
Occupationa	Allergen/irritant induced	Work-related symp-toms	Improves with exposure	[10]

Biomarkers for Precision Classification

Biomarkers are essential for stratifying asthma variants and guiding therapy. Blood eosinophils, FeNO, peri-ostin, and urinary leukotriene E4 have predictive value. Novel markers such as transcriptomic signatures and microbiome profiles are under study.

Table 2: Biomarkers of Asthma Variants

Biomar+E10+A1:E11	Associated Variant	Diagnostic Role	Clinical Utility	Refere nces
Blood eosinophils	Eosinophilic, AERD	≥300 cells/μL predicts response to anti-IL-5	Stratifies biologic therapy	[12],
				[30]
FeNO	T2-high eosino-philic	Indicates IL-13 activity	Guides ICS and dupilumab use	[14],
				[15]
Periostin	Eosinophilic	Serum marker of airway	Emerging for	[15]
Sputum neutrophils	Neutrophilic	Indicates IL-17 activity	Predicts steroid	[7]
Urinary LTE4	AERD	Marker of leukot-riene overproduc-tion	Monitors aspirin desensitization	[8]
Multi-omics (RNA-seq, microbiome)	All endotypes	Endotype discovery	Future personal-ized therapy	"[24], [25]"

Novel Therapeutic Modalities

New therapies have transformed asthma management, particularly for severe variants. Biologics now form the backbone of precision medicine in asthma.

Table 3: Innovative Therapies for Severe and Variant Asthma

Therapy	Target	Indicated Variant	Efficacy	Limitations	References
Blood eosinophils	Eosinophilic, AERD	≥300 cells/ µL predicts response to anti-IL-5	Stratifies biologic therapy	[12],	[19]
Mepolizumab/ Reslizumab	IL-5	Eosinophilic, AERD	Improves FEV1, ↓	Costly, parenteral	[30], [33]
Benralizumab	IL-5Rα	Severe eosinophilic	Complete eosinophil depletion	Limited access	[31]
Dupilumab	IL-4/13	Eosinophilic,	↓ Exacerbations, ↑	Conjunctivitis	[20]
Tezepelumab	TSLP	Broad	Effective across T2-	Long-term data	[32]
Macrolides (azithromycin)	Neutrophil inflammation	Neutrophilic	↓ Exacerbations	Resistance risk	[7]
Bronchial	Smooth muscle	Severe	Improves QoL	Invasive	[22]
JAK inhibitors	JAK-STAT	T2-low	Under investigation	Safety concerns	[21]

Comparative Outcomes Across Variants

Therapeutic outcomes vary significantly by phenotype/endotype, reinforcing the role of precision treatment strategies.

Table 4: Comparative Outcomes of Novel Therapies Across Asthma Variants

Variant	Most Effective Therapy	Exacerbation Reduction	Lung Function	QoL Improveme	References
Obesity- associated	Weight loss, lifestyle + biologics under study	Moderate	Modest	Moderate	[6]
Neutrophilic	Macrolides, anti–IL-17	Variable	Minimal	Limited	[7],
AERD	Leukotriene antagonists,	Significant	Significant	Strong	[8],
Late-onset eosinophilic	Anti-IL-5, anti-IL-4/13	Strong	Strong	Strong	[30],[32]
Occupational	Exposure avoidance + ICS	Variable	Moderate	Strong	[10]

Figure 1: Conceptual Framework of Asthma Heterogeneity

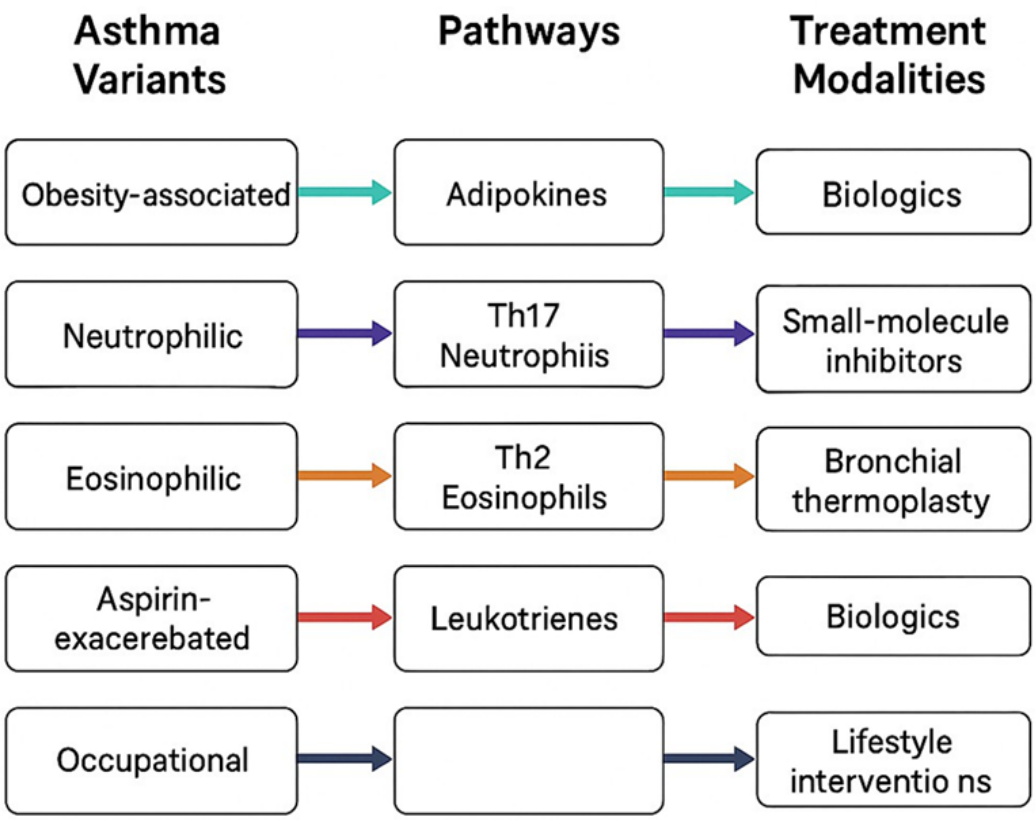
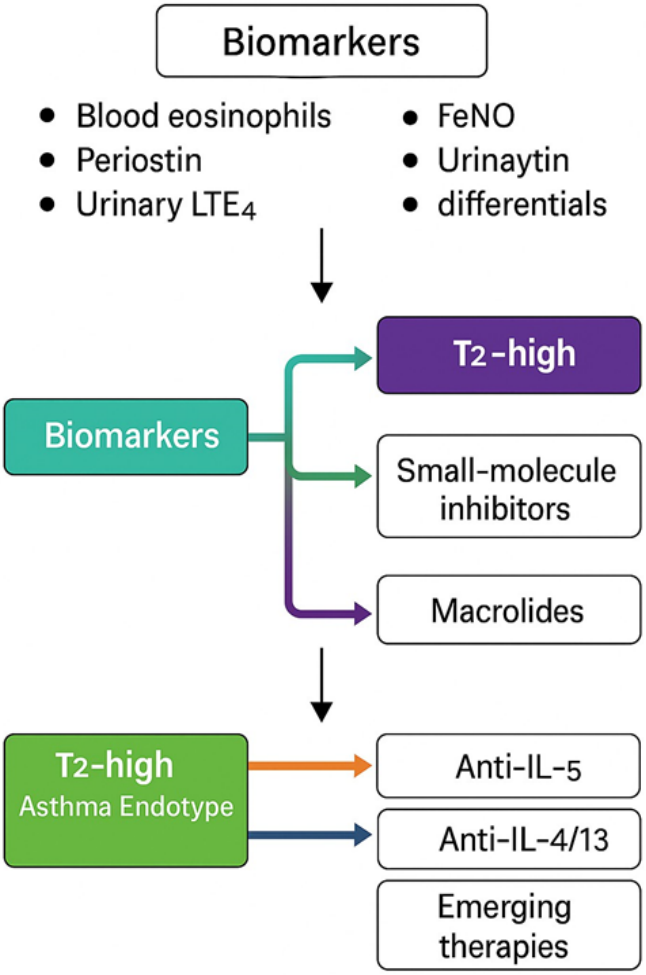


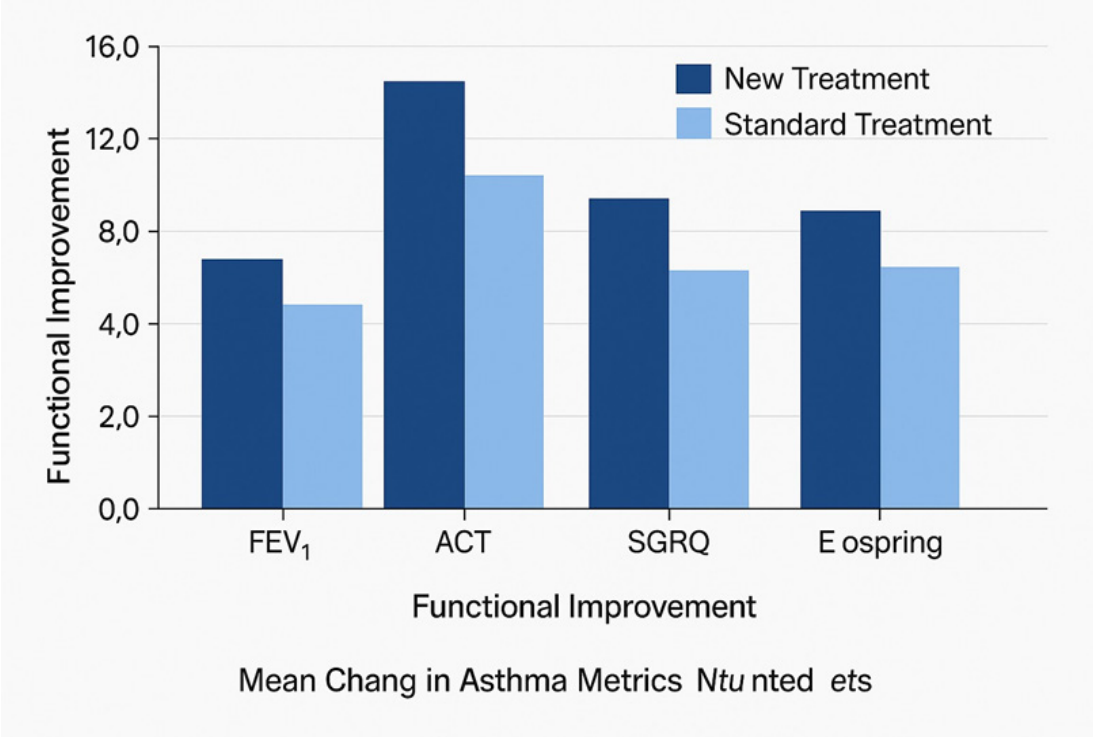
Diagram illustrating emerging variants of bronchial asthma (obesity-associated, neutrophilic, eosinophilic, aspirin-exacerbated, occupational) and their corresponding immunological pathways. Links are shown to novel treatment modalities such as biologics, small-molecule inhibitors, bronchial thermoplasty, and lifestyle interventions.

Figure 2: Biomarker-Guided Precision Medicine in Asthma



Flowchart showing how biomarkers (blood eosinophils, FeNO, periostin, urinary LTE4, sputum differentials) stratify asthma endotypes (T2-high vs. T2-low) and direct therapeutic selection (anti IgE, anti-IL-5, anti-IL-4/13, anti-TSLP, macrolides, or emerging therapies).

Figure 3: Comparative Outcomes of Biologics



Bar chart comparing the relative reduction in exacerbation rates and improvements in lung function (FEV1) among patients treated with omalizumab, mepolizumab, benralizumab, dupilumab, and tezepelumab.

Discussion

This review highlights the evolving understanding of asthma as a heterogeneous disease composed of distinct phenotypes and endotypes, each with unique immunological, clinical, and therapeutic implications. The emergence of obesity-associated asthma, neutrophilic asthma, aspirin-exacerbated respiratory disease (AERD), and late-onset eosinophilic asthma illustrates how clinical heterogeneity is underpinned by diverse molecular pathways [1-4]. This heterogeneity necessitates precision medicine approaches that align therapeutic interventions with the underlying biology of each variant [5,6].

Asthma Heterogeneity and Variants

Traditional classifications of asthma into allergic and non-allergic forms are no longer sufficient. For instance, obesity-associated asthma is influenced not only by mechanical restriction but also by adipokine-mediated systemic inflammation, leading to poor corticosteroid responsiveness [7,8]. Neutrophilic asthma, often associated with environmental triggers and infection, demonstrates Th17-driven inflammation and resistance to inhaled corticosteroids, highlighting the need for macrolides and novel IL-17–targeting strategies [9,10]. Similarly, AERD exemplifies the intersection of immunological and metabolic pathways, with dysregulated arachidonic acid metabolism producing excessive leukotrienes and severe disease [11]. The late-onset eosinophilic variant, in contrast, exhibits robust response to biologics targeting IL-5 and IL-4/13, emphasizing the clinical utility of endotype-based therapy [12–14].

Biomarkers and Precision Medicine

The integration of biomarkers such as blood eosinophils, fractional exhaled nitric oxide (FeNO), periostin, and urinary leukotriene E4 into clinical practice has transformed asthma management. These biomarkers facilitate accurate stratification of patients into T2-high and T2-low endotypes, guiding biologic therapy [15–17]. However, T2-low asthma remains a major challenge due to the lack of reliable biomarkers and effective targeted treatments. Emerging technologies, including transcriptomics, proteomics, and microbiome analyses, may uncover novel predictive signatures for these difficult-to-treat patients [18,19].

Advances in Therapeutics

The therapeutic landscape of asthma has shifted significantly with the advent of biologics. Omalizumab, targeting IgE, has long been established in allergic asthma, while mepolizumab, reslizumab, and benralizumab have proven effective for eosinophilic asthma by suppressing IL-5 signaling [20-23]. Dupilumab, an IL-4Rα antagonist blocking both IL-4 and IL-13, further expands treatment for T2-high asthma, while tezepelumab, targeting thymic stromal lymphopoietin (TSLP), offers the potential for efficacy across both T2-high and T2-low phenotypes [24,25]. Non-biologic innovations such as bronchial thermoplasty have also shown benefit in carefully selected patients with severe refractory asthma [26]. Nevertheless, biologics are limited by high costs, injection requirements, and variable responses, underscoring the need for predictive biomarkers and long- term data [27].

T2-Low and Neutrophilic Asthma: The Unmet Need

While T2-high asthma management has advanced substantially, treatment of T2-low asthma lags behind. Neutrophilic asthma is particularly problematic due to corticosteroid resistance and the lack of approved targeted therapies [28]. Macrolides have shown some benefit, but concerns about antimicrobial resistance limit their widespread use [29]. Ongoing trials of JAK inhibitors and anti– IL-17/IL-23 biologics may provide new options [30]. Additionally, therapies modulating airway microbiota and epigenetic pathways are promising areas for future research [31,32].

Implications for Clinical Practice

The recognition of asthma variants highlights the importance of individualized assessment. Clinicians must consider not only clinical symptoms but also inflammatory profiles and biomarker data when determining treatment. This precision medicine approach can optimize outcomes, minimize adverse effects, and reduce healthcare costs associated with uncontrolled severe asthma [33]. Importantly, management should also integrate non-pharmacological strategies such as weight reduction in obesity-associated asthma and allergen avoidance in occupational asthma [34].

Future Directions

Future research should prioritize the integration of "omics" data (genomics, transcriptomics, metabolomics, microbiome) to refine asthma endotyping. Large, longitudinal cohort studies are needed to validate novel biomarkers and assess long-term safety and efficacy of emerging therapies. Additionally, health system interventions should ensure equitable access to biologics, as cost and availability remain significant barriers, particularly in low- and middle-income countries [35].

Summary of Discussion

Asthma is a complex, heterogeneous disease that requires a paradigm shift in management. Identifying and targeting new variants such as obesity-associated, neutrophilic, AERD, and late- onset eosinophilic asthma through biomarker-driven precision medicine has already improved patient outcomes. However, significant gaps remain, particularly in the treatment of T2-low asthma. Continued research into molecular mechanisms, novel therapeutics, and integrated care models will be critical to achieving personalized asthma control worldwide.

Conclusion

Bronchial asthma is no longer understood as a single disease entity but rather as a spectrum of phenotypically and endotypically distinct disorders. Recognition of variants such as obesity- associated asthma, neutrophilic asthma, aspirin-exacerbated respiratory disease, and late-onset eosinophilic asthma has transformed both clinical understanding and therapeutic strategies.

Biomarker-driven precision medicine has already demonstrated substantial improvements in patient outcomes, particularly with biologic therapies targeting IgE, IL-5, IL-4/13, and TSLP.

However, significant challenges remain, especially in T2-low and neutrophilic asthma, where corticosteroid resistance and limited therapeutic options persist. Emerging modalities—including small-molecule inhibitors, microbiome-based interventions, epigenetic therapies, and bronchial thermoplasty—represent promising avenues for the future.

The evolution of asthma care now depends on integ-

rating clinical phenotyping, molecular profiling, and novel therapeutics into personalized management strategies. Such an approach will optimize disease control, reduce exacerbations, and improve quality of life for patients worldwide. Bridging gaps in access and ensuring equitable implementation of precision medicine will be critical in reducing the global burden of asthma.

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