# Why treating asthma and CRSwNP separately may fail your patients

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Asthma and chronic rhinosinusitis with nasal polyps (CRSwNP) have long been treated as distinct entities, managed by different specialists with often siloed approaches. But the clinical reality for many patients is a persistent overlap of symptoms and underlying pathology, pointing towards a more integrated understanding of airway disease. The presence of anosmia, a frequently overlooked symptom, may offer a key to bridging this diagnostic and therapeutic divide.

**KEY TAKEAWAYS**

- The Pivot: Anosmia in patients with asthma and CRSwNP signals a deeper, shared inflammatory process across the upper and lower airways, challenging the traditional separation of these conditions.
- The Data: Patients presenting with both asthma and CRSwNP, particularly those with severe anosmia, often exhibit elevated type 2 inflammatory markers, indicating a common underlying endotype.
- The Action: Clinicians should routinely assess for anosmia in patients with asthma and CRSwNP, as its presence may guide a more holistic, unified airway disease management strategy, potentially involving biologics targeting type 2 inflammation.

The traditional view of asthma as a lower airway disease and chronic rhinosinusitis with nasal polyps (CRSwNP) as an upper airway condition has led to fragmented patient care. Patients often navigate separate specialists, receiving treatments that address individual symptoms rather than the overarching inflammatory process. This compartmentalization overlooks the anatomical and immunological continuity of the respiratory tract, a concept increasingly recognized as the 'unified airway'.

This unified airway hypothesis posits that inflammatory processes in one part of the respiratory tract can influence, or be influenced by, inflammation elsewhere. For patients with both asthma and CRSwNP, this connection is particularly evident. The presence of nasal polyps, for instance, is a strong predictor of concomitant asthma, and the severity of one condition often correlates with the severity of the other. The challenge lies in identifying a clinical marker that reliably signals this interconnectedness, prompting a more integrated therapeutic approach.

## Anosmia as a Clinical Bridge

Anosmia, the loss of the sense of smell, is a common and debilitating symptom in patients with CRSwNP, but its significance extends beyond the nasal cavity. It is not merely a local symptom of nasal obstruction or inflammation. Instead, anosmia in this context often reflects profound, chronic inflammation within the sinonasal passages, frequently driven by type 2 inflammatory pathways. This same type 2 inflammation is a key driver in a significant proportion of

patients with severe asthma.

Patients with CRSwNP and severe anosmia often exhibit higher levels of eosinophils, IgE, and other type 2 inflammatory biomarkers. When these patients also have asthma, the anosmia can serve as a clinical flag for a systemic type 2 inflammatory burden affecting both upper and lower airways. This shared inflammatory signature suggests that therapies targeting type 2 inflammation, such as biologics, could offer benefits across both conditions, rather than just one.

## The Inflammatory Underpinnings

The epithelial barrier plays a critical role in both asthma and CRSwNP. Dysfunction of the airway epithelium, whether in the nose or the bronchi, can initiate and perpetuate chronic inflammation. This epithelial dysfunction allows allergens, pathogens, and irritants to penetrate more easily, triggering an exaggerated immune response. In many cases, this response is characterized by type 2 inflammation, involving cytokines like IL-4, IL-5, and IL-13.

These cytokines drive eosinophilic inflammation, mucus overproduction, and airway remodeling, contributing to the pathology seen in both conditions. The presence of nasal polyps, which are essentially inflammatory growths rich in eosinophils, further highlights this connection. For a deeper dive into how these epithelial changes contribute to airway disease, consider our previous coverage.

When a patient with asthma also experiences CRSwNP with significant anosmia, it is a strong indicator of this underlying type 2 inflammatory endotype. This recognition moves beyond treating symptoms in isolation and towards addressing the root cause. This unified approach is particularly relevant given the availability of biologics that specifically target these type 2 pathways.

## Implications for Diagnosis and Management

For clinicians, recognizing anosmia as a marker of unified airway disease means a more comprehensive diagnostic workup is warranted. Simply treating nasal symptoms with topical steroids or asthma with inhaled corticosteroids may not be sufficient if a systemic type 2 inflammatory process is driving both. A thorough assessment of smell function, perhaps using standardized olfaction tests, should become a routine part of evaluating patients with either condition, especially when both are present.

The presence of anosmia, particularly when severe and persistent, should prompt consideration of advanced therapies. Biologics, for example, have demonstrated efficacy in both severe eosinophilic asthma and CRSwNP. By targeting specific cytokines or their receptors, these agents can reduce inflammation in both the upper and lower airways, potentially improving not only asthma control and nasal polyp burden but also restoring olfaction. This integrated view aligns with the evolving understanding of severe asthma beyond airway focus.

The clinical picture of a patient with severe asthma and CRSwNP, complicated by anosmia, often represents a significant burden of disease. These patients frequently experience impaired quality of life, sleep disturbances, and a reduced ability to detect hazards like smoke or spoiled food, adding to their morbidity. Traditional treatments may offer only partial relief, leaving patients with persistent symptoms and a sense of therapeutic futility.

The current standard of care for CRSwNP often involves intranasal corticosteroids and, for more severe cases, endoscopic sinus surgery. While surgery can provide temporary relief by removing polyps and improving drainage, recurrence is common, especially in patients with underlying type 2 inflammation. Similarly, asthma management relies heavily on

inhaled corticosteroids and bronchodilators, with oral corticosteroids used for exacerbations. But for patients with severe, uncontrolled asthma and concomitant CRSwNP, these therapies often fall short.

## A Shift Towards Integrated Care

The concept of unified airway disease, with anosmia as a key indicator, advocates for a shift towards integrated care models. Pulmonologists, allergists, and otolaryngologists should collaborate more closely, sharing insights and coordinating treatment plans. This multidisciplinary approach ensures that both upper and lower airway pathologies are addressed concurrently, optimizing patient outcomes.

Consider the case of a patient with severe eosinophilic asthma who also presents with extensive nasal polyps and complete anosmia. This patient is likely to benefit from a biologic that targets IL-5 or the IL-4/IL-13 pathway, rather than simply escalating doses of inhaled corticosteroids or undergoing repeated sinus surgeries. The biologic addresses the systemic inflammation driving both conditions, offering a more comprehensive and sustained response. This approach is supported by growing evidence for dupilumab in severe asthma, for example.

The long-term implications of untreated or inadequately treated anosmia extend beyond immediate quality of life. The inability to smell can lead to nutritional deficiencies due to lack of enjoyment of food, social isolation, and even depression. Addressing anosmia, therefore, is not merely a cosmetic concern but a critical component of holistic patient care. The Oxford Handbook of Respiratory Medicine provides a concise reference for managing such complex respiratory conditions.

The challenge remains in consistently identifying this unified airway phenotype in clinical practice. Routine screening for anosmia, perhaps through simple validated questionnaires or smell identification tests, could help flag patients who might benefit from a more integrated assessment and advanced therapeutic strategies. This proactive approach could prevent years of fragmented care and suboptimal outcomes for patients suffering from both asthma and CRSwNP.

The open-label design of many real-world observations is an obvious caveat when considering these associations, as is the inherent difficulty in standardizing smell assessment across diverse clinical settings. Still, the consistent biological rationale for shared inflammatory pathways provides a strong foundation for further investigation into anosmia as a prognostic and therapeutic marker. The next step involves prospective studies designed to evaluate the impact of unified treatment strategies, particularly biologics, on both asthma control and olfactory function in this specific patient population.

> **CLINICAL IMPLICATIONS**
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> The persistent separation of asthma and CRSwNP in clinical practice is an anachronism. Anosmia, far from being a mere nuisance, is a potent clinical signal of a shared type 2 inflammatory endotype that demands a unified approach. GPs and specialists alike must recognize that a patient presenting with both conditions and loss of smell is likely dealing with a systemic inflammatory burden, not two isolated problems.
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> This means moving beyond the reflex to simply refer to separate specialists. Instead, a collaborative assessment for biologics targeting IL-4, IL-5, or IL-13 should be considered earlier. These agents offer the potential to address the underlying inflammation in both the upper and lower airways, improving not only respiratory function but also restoring a critical sense that profoundly impacts quality of life.
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> The industry has developed effective therapies for type 2 inflammation, but their optimal deployment requires

> clinicians to think beyond organ-specific silos. Patients with severe asthma and CRSwNP, especially those with anosmia, are often the most difficult to manage with conventional treatments. Identifying this phenotype early allows for a more targeted and effective therapeutic strategy, potentially reducing the need for repeated surgeries or chronic oral corticosteroid use.
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> The goal is to improve patient outcomes by treating the patient, not just the disease. Anosmia provides a tangible, patient-reported symptom that can guide this more holistic, evidence-based approach to unified airway disease. Ignoring it is to miss a critical piece of the clinical puzzle.

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## REFERENCES

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