Changes in Inflammation Markers Associated with Systemic Prednisone Therapy in Chronic Rhinosinusitis

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Abstract

We sought to assess the impact of prednisone therapy on nasal and IgE in plasma, eosinophils in tissue, and systemic expression of periostin and exotoxin. Techniques we contrasted the tissue levels of periostin, exotoxin, IgE, and eosinophils in the nasal and systemic airways between group 1 of patients who received only nasal steroids prior to FESS and group 2 of patients who received oral steroids such as prednisone. Results patients receiving prednisone one week before and after surgery had periostin, exotoxin, and IgE levels in their plasma fall in a statistically meaningful way. Prednisone is a systemic steroid that significantly lowers the plasma levels of periostin, exotoxin, and IgE. Additionally, we noted reduced levels of exotoxin in the stromal, periostin in the epithelium, and eosinophils in the tissues. More research is required to identify the inflammatory markers linked to chronic rhino sinusitis. More tailored therapy might be possible if it were possible to find medications that reduce inflammatory parameters.

Keywords: Rhino sinusitis • Inflammation • FESS • Prednisone • Steroid

Introduction

Chronic Rhino Sinusitis (CRS) is a widespread, complicated condition with an unknown underlying cause. It is a major health issue because it is chronic and recurrent. Several interrelated; interacting variables are involved in the aetiology of CRS. The condition recurs because of the complex etiology, which makes selecting the best treatment challenging. At least two symptoms, one of which should be nasal obstruction, obstruction, or discharge (anterior/posterior nasal drip), characterise it. Other signs include facial pain or pressure, as well as a decrease or loss of smell. Persistent rhino sinusitis is a disease of mucosal inflammation of the nose and paranasal sinuses characterised by persistent symptoms lasting more than 12 weeks, according to the European Position Paper on Rhinosinusitis (EPOS) 2020. We can assess the existence of nasal polyps, mucopurulent discharge, and/or mucosal edema during a nasal endoscopy, particularly in the middle nasal meatus. Mucosal inflammation, infiltrating inflammatory cell profiles (eosinophils or neutrophils, differentiated T cell patterns), and tissue remodelling may be useful in identifying disease entities even when the origin of CRS is yet unknown. The well-known indicator of a poor prognosis, tissue eosinophilia, is linked to a high risk of illness recurrence, chronic postoperative discomfort, and inadequate healing.

Description

The primary reasons preventing normal healing in CRS are the modifications brought on by tissue remodeling, including squamous metaplasia, BM thickening, stromal edoema and fibrosis, goblet cell hyperplasia, and sub epithelial gland hyperplasia. More research is required to identify the various inflammatory indicators connected to chronic rhino sinusitis since the association between histologic modifications and inflammatory markers in CRS is still unclear. Such diagnostic exponents can be defined, allowing us to offer greater insight into potential future treatments. A novel marker called periostin has a unique expression in chronic rhino sinusitis. A protein known as periostin (POSTN) is primarily secreted by connective tissues rich in collagen, including the periosteum, periodontal ligaments, tendons, heart valves, and skin. It is a protein found in the extracellular matrix that interacts with the extracellular matrix and contributes to pathologic remodelling alterations, especially in eosinophilic inflammation. The integrin molecules (v1, v3, and v5) that periostin interacts with on cell surfaces deliver signals for tissue development and remodelling. Periostin, a byproduct of IL-4 or IL-13, has been discovered to activate Th2-type cytokines during immunological responses. It has been established that periostin has a role in allergic inflammation and eosinophil mediated inflammation by accelerating eosinophil recruitment and activation. On the other hand, eosinophil activation and infiltration during NP formation are significantly influenced by RANTES and eotaxin-2 (the CC family of chemokines). It is still unclear how eosinophils are specifically drawn to nasal polyps, and it needs to be further explored whether periostin can affect the tissue eosinophilia of ENP by altering the release of eotaxin-2 and RANTES. The thicker basement membrane and serum of asthmatic patients, particularly those with eosinophilic airway inflammation and atopy, contain periostin. Periostin is linked to asthma, allergic rhinitis, and CRS and increases the production of mucus and type 2 inflammatory factors. Additionally, it can be found in serum, nasal polyps, and sinonasal mucosa. It strongly correlates with the severity of inflammatory alterations in eosinophilia CRSwNP (CRS with Nasal Polyps). Patients with asthma who receive systemic or inhaled corticosteroid therapy see a reduction in serum periostin levels. In clinical settings, measuring periostin serum levels may be a useful tool for keeping track of therapy outcomes. The use of isoforms specific for chronic rhino sinusitis and the standardisation of assay procedures may make it possible to determine periostin's clinical utility as a CRS marker objectively.

Conclusion

On the other hand, exotoxin, a cytokine produced by the epithelium and a member of the C-C family of chemokinesis, can cause the chemotaxis of eosinophils, basophils, and TH2 lymphocytes. Compared to other cytokines, exotoxin is a more focused stimulator of the selective migration of inflammatory cells. According to numerous studies, exotoxin CCR3 is crucial for the buildup of eosinophils in tissues and, subsequently, for the escalation of the inflammatory response. Exotoxin is regarded as one of the primary mediators of the development of eosinophilic infiltrates and inflammation due to its multifaceted influence on eosinophils. Peripheral and tissue eosinophilia are caused by an increase in mature eosinophils in the blood caused by the interaction of exotoxin and interleukin-5.

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