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Successful prevention of recurrent nasal polyposis by means of systemic low-dose IFN- α 2a

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To the Editor:

Chronic nasal polyposis is a severe, recurrent clinical condition. Although systemic steroids have been successful in preventing relapse after endonasal sinus surgery, they are—at least when administered as long-term treatment—accompanied by serious side effects.

Histopathologic findings in nasal polyposis are characterized mainly by an eosinophilic infiltrate in the nasal sinusoidal tissue.¹ Several factors account for the recruitment of eosinophils into the polyp epithelium; among them, the most important is IL-5.¹ In an in vitro study, it was shown that IFN- α selectively downregulates IL-5 synthesis in human CD4+ cells.² In this pilot study involving 4 patients, we demonstrate for the first time that recurrence of thus-far recalcitrant nasal polyposis can be successfully prevented during treatment with low-dose IFN- α .

Four middle-age patients who had severe nasal polyposis with at least 1 recurrence after surgical removal, resulting in at least 2 operations, were included in the study (Table I). In most cases, recurrence had occurred within a very short time period (4 weeks to 3 months)

after each course of sinus surgery. After informed consent was obtained, IFN- α prophylaxis was initiated within 4 to 6 weeks after the most recent surgical removal of nasal polyps. IFN- α 2a (Roferon) in a dose of 3×10^6 IU was subcutaneously injected 3 times per week. No other systemic treatment for symptoms of nasal polyposis, such as corticosteroids, antihistamines, and other immunomodulatory medication, was allowed during the treatment with IFN- α , except intranasal steroids for relief of acute symptoms of rhinitis. Other medications used during the treatment time included the antipyretic agent paracetamol against potential IFN-associated flulike side effects (see below), lovastatin (Mevinacor) in patient 1, and a 10-day course of penicillin V for treatment of streptococcal tonsillitis in patient 4. Laboratory parameters, such as complete and differential blood count, serum IgE, and blood chemistry, including liver function test results, were determined before entry into the study and subsequently every 4 weeks. Nasal endoscopy was carried out before treatment and every 3 months thereafter to determine therapeutic efficacy.

Apart from flulike symptoms in the first few days, no specific side effects of IFN- α , such as arthralgia, nausea and vomiting, leukopenia, and elevation of transaminases, were noted. All laboratory parameters remained normal, except for 1 slight temporary elevation of creatine kinase, which most likely resulted from an intramuscular injection of Diclofenac by a general practitioner. IgE values were initially slightly elevated in 2 patients and did not decline throughout the study.

In all 4 patients, no recurrence of nasal polyposis was observed at 3 and 6 months after the initiation of IFN- α -therapy. In a patient who had been treated for more than 1 year (patient 1), discontinuation of IFN- α prophylaxis was attempted after 6 months. On follow-up 1 month later, he presented with 2 tiny new polyps, which had not developed any further at the time of this writing (month 7 postinterruption) after he resumed IFN- α prophylaxis and tapered to 3×10^6 IU 2 times per week at month 4 postinterruption.

Inflammatory reactions associated with eosinophilia are characterized by a variety of pathophysiologic reactions; increased diapedesis of T_H2 lymphocytes leads to an increased production of IL-4, IL-5, and IL-13, not only causing immediate recruitment and production of eosinophils as a typical consequence of IL-5 but also leading to an increased release of chemotactic factors, such as eotaxin, RANTES, and MCP-4, predominantly by endothelia.³ In nasal polyposis especially, an increased induction of IL-5 is held responsible for the dramatically enhanced recruitment of eosinophils and subsequently for the increased reactive proliferation of sinusoidal epithelium.¹

In accord with previous reports about the response of other eosinophil-associated disorders to IFN- α ,^{4 5} this study has demonstrated for the first time that IFN- α can prevent recurrence of nasal polyposis. Further studies analyzing these effects with regard to altered expression levels of proinflammatory cytokines before and after treatment with IFN- α are underway.

Table I. Summary of Clinical Data

Patient no.	Age (y)	Sex	No. of recurrences before IFN- α therapy	No. of operations	Duration of IFN- α therapy after last operation (mo)
1	57	Male	2	3	13
2	45	Female	1	2	7
3	49	Male	3	4	6
4	49	Male	1	2	3

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References

TOP

1. Kramer MF, Rasp G. Nasal polyposis: eosinophils and interleukin-5. *Allergy* 1999;54:669-80.

MEDLINE

CROSSREF

2. Schandené L, DelPrete GF, Cogan E, Stordeur P, Crosiaux A, Kennes B et al. Recombinant interferon alpha selectively inhibits the production of IL-5 by human CD4+ cells. *J Clin Invest* 1997;2:309-15.

3. Barnes PJ. Therapeutic strategies for allergic diseases. *Nature* 1999;402 (Supp):B31-B38.

MEDLINE

4. Butterfield JH, Gleich GJ. Interferon-alpha treatment of six patients with the idiopathic hypereosinophilic syndrome. *Ann Intern Med* 1994;121: 648-53.

MEDLINE

5. Tatsis E, Schnabel A, Gross WL. Interferon-alpha treatment of four patients with the Churg-Strauss syndrome. *Ann Intern Med* 1998;129: 370-4.

MEDLINE
