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An immunohistological study of nasal and paranasal mucosa of patients with relapsing chronic sinusitis

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SUMMARY

Mucosal specimens from patients with chronic sinusitis have been immunohistologically investigated. Secretory IgA was found decreased in type 1 (relapsing type sinusitis) whereas C_3 was increased. Lysozyme was decreased or deplated in longstanding cases (type 2). Fibronectin was found both in type 1 and type 2 chronic sinusitis. It was concluded that depletion of IgA and lysozyme with activation of C_3 and fibronectin interferes with the successful treatment of sinusitis.

INTRODUCTION

Inflammatory responses essentially protect and repair the body for the purpose of maintaining homeostasis under adverse environmental influences. If an inflammatory response only incompletely restores the injured tissue to its original state or, if tissue repair is not accomplished and less resistant to reinvasion of microorganisms, the events may progress to a state of chronic inflammation. The purpose of this study is to detect immunohistologically diverse factors participating sinusitis. Of these, we have studied IgA (including secretory pieces), lysozyme, C_3 and fibronectin.

MATERIAL AND METHODS

Mucosal specimens for immunohistological examinations were obtained from 23 patients with chronic sinusitis, of whom, 6 patients suffered from frequent relapse and severe symptoms with longstanding course and the other 17 patients one or two relapses and mild symptoms with long intervals. Histologically, mucosa of the former (6 patients) revealed lesions of thick fibrosis, a few dilated cystic mucous glands, engorgement and dense or sparce inflammatory cells including eosinophils (type I, relapsing type).

Those of the latter with curable course revealed well-preserved glands, edematous or loose stroma and generally moderate number of inflammatory cell infil-

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tration (type 2). The antisera were anti-IgA (with secretory pieces), anti-C3, anti-lysozyme and antifibronectin (DAKO Co.). They were demonstrated by the peroxidase-antiperoxidase method (PAP method) using 10% formalin fixed and paraffin embedded sections. Hematoxylin counterstaining exhibited good contrast.

RESULTS

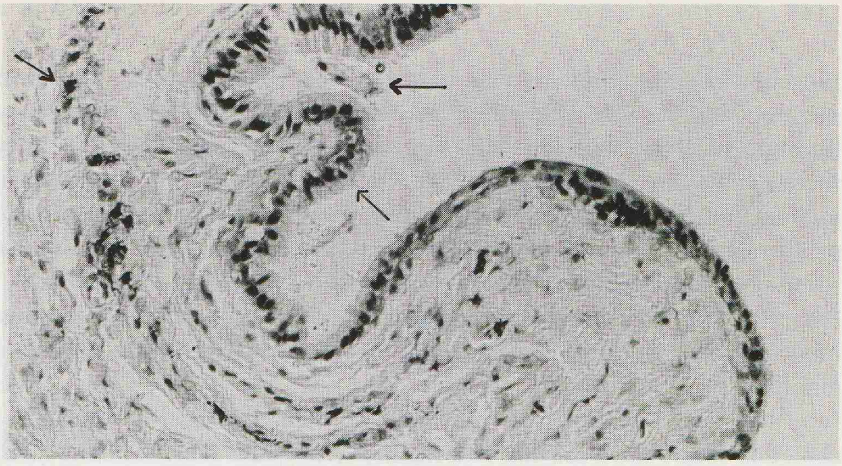
Comparison of the mucosa from type 1 and type 2 for immunohistological differences has shown the following findings. Secretory IgA and IgA containing plasma cells decrease or are depleted focally or generally in type 1 chronic sinusitis (Figure 1a).

The C3 component of the complement was localized in the surface of the vascular endothelium, the vascular lumen, muscle cells of the vascular wall and the apical portions of the surface mucous epithelium and mucous glands, as well as in the macrophages, neutrophils and eosinophils. In type 1 chronic sinusitis, the C3 deposited markedly at these sites. Decreased secretion or depletion of lysozyme was observed in the mucous glands focally or generally, which were thought to be the sequences of glandular epithelial disruption (Figure 1b).

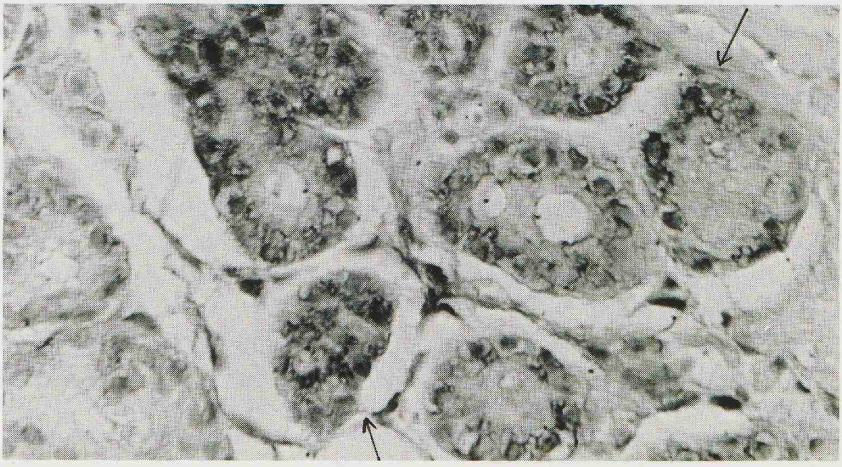
Fibronectin was detected in the matrix expecially in the perivascular resion in active inflammatory lesions of type 1 as welle as type 2 (Figure 1c).

DISCUSSION

Secretory IgA is the predominant immunoglobulin defending surface mucous membrane against microorganisms. Plasma cells in the submucosa contained IgA predominantly and secretory IgA was detected in the surface mucous epithelium and mucous glands. Lysozyme is a very important bactericidal substance secreted from mucous epithelium (Hill et al., 1974), macrophages and polymorphnuclear leukocytes (Glynn et al., 1962; 1965), which are found in the cytoplasm of the surface epithelium and mucous glands. Decreased secretory IgA and lysozyme resulting from the disruption of the glandular epithelium imply lowered defence against inflammatory agents (Tomasi, 1972) and arouse vicious cycle. This partly explains the reasons why some patients with chronic sinusitis have a unfavourable relapsing course. Simultaneous formation of squamous metaplasia in mucous membrane was thought to increase defence against inflammation too. Existence of C3 in tissue implies an antigen-antibody reaction uninvolved IgA and activation of the Hageman factor, kinin, plasminogen or fibrinogen which are active mediators involved in inflammation. C3 is broken down into chemotactic substances for neutrophils and monocytes. On the other hand, activation of C3 was through an alternative pathway triggered by IgA and bacteria. In this case, invasion of inflammatory agents such as bacteria or other antigens may activate and accumulate complement in tissues.



a



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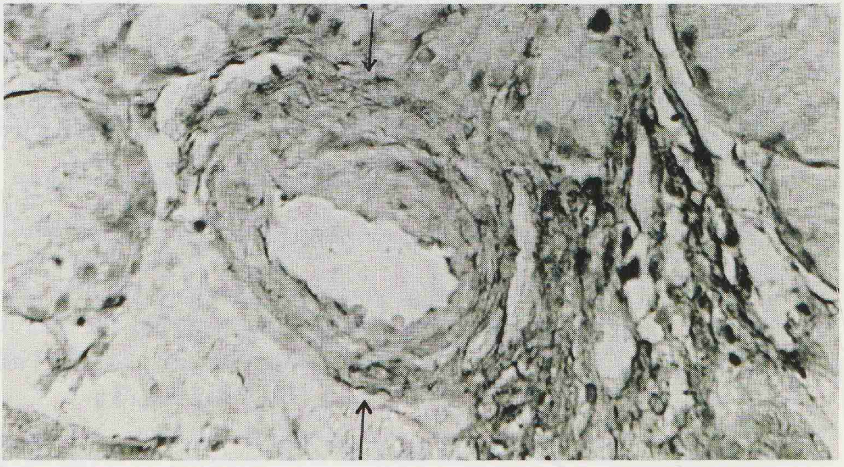

c

Figure 1. PAP immunohistological staining. Positivity is shown as blackened areas.
a. (110 ×) Decreased IgA secretion (arrows) and squamous metaplasia;
b. (200 ×) Lysozyme: decreased lysozyme secretion;
c. Fibronectin: perivascular depositis (arrows).

Fibronectin, a glycoprotein found in the connective tissue matrix and the basement membranes of blood vessels, circulates in the plasma and associates with fibrin upon activation of the clotting system (Mosher and Furcht, 1981). Hypothetically, fibronectin stimulates phagocytosis and proliferation of vascular endothelium and on the other hand, prolongs inflammation and interferes tissue repair. In this study, no significant differences of fibronectin deposits between type 1 and type 2 chronic sinusitis.

Bacteriological examination has revealed that Haemophilus influenzae was the most frequent organisms obtained from the nasal discharge of the patients with longstanding type 1 chronic sinusitis.

In conclusion, it was inferred from this immunohistological examination that depletion of IgA and lysozyme with significant increase of activation of C3 interfered with the curability of sinusitis and fibronectin had no special participation.

RÉSUMÉ

Des spécimens de mucus de patients souffrent de sinusite chronique ont été soumis à un examen immuno-histologique.

Dans le type 1 (type de sinusite récidivante) la sécrétion de IgA se trouvait être diminuée, tandis que celle de C3 était augmentée. Dans les cas de sinusite de longue durée (type 2) le présence de lysozyme était réduite ou très basse. La présence de fibronectine a été constatée aussi bien dans le type 1 de sinusite chronique que dans le type 2.

La conclusion était que le succès du traitement de sinusites est entravé par la secrétion réduite de IgA et de lysozyme avec une activité accrue de C3 et de fibronectine.

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