

Does Lymphatic Obstruction Play a Role in the Pathophysiology Of Antrochoanal Polyps?

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Introduction

The close relationship between choanal polyps and the maxillary sinus was first reported by Killian in 1906. Because of their rare incidence (3% to 6% of all nasal polyps), antrochoanal polyps (ACPs) have not been extensively studied. It has been suggested that ACPs are associated with inflammatory or allergic antral disease. Mills (1) stated that choanal polyps arise from acinous mucous glands that are blocked and ruptured during the healing of bacterial sinusitis, thus being the extension of a mucocele. An “epithelium rupture theory,” or “new gland formation theory,” has also been proposed, which includes the presence of cysts as the precursors of ACP formation (2).



Hosemann et al (3) demonstrated with histochemical detection of 5'-nucleotidase that both the nasal and maxillary sinus mucosae showed distinct superficial and deep longitudinal lymphatic capillary networks interconnected through vertically arranged lymphatic vessels. The purpose of our study was to determine whether lymphatic obstruction, either primary or secondary to chronic sinus infection or allergy, plays a leading role in the formation and further growth of ACPs.

Materials and Methods

Patients and Polyps

The study included 25 cases of ACPs and 25 cases of chronic maxillary sinusitis. The antral part of the polyp was cystic in only 5% of patients, and polypoid in 95%. In ACPs cases, the transitional area between the sinus mucosa and the polyp pedicle was the target area of examination. The specimens of mucosa from patients with chronic maxillary sinusitis (“chronic sinusitis biopsy” or CSB group), approximately 1 cm², were collected during functional endoscopic sinus surgery procedures.

Polyp Removal and Preparation

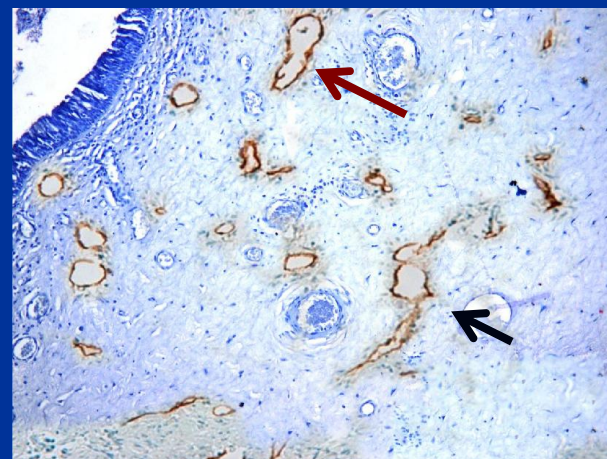
The ACPs were strictly unilateral, and in most patients, there was a single polyp. In 3 patients, 2 polyps could be traced coming out of the maxillary sinus, and 6 patients had ipsilateral ethmoid polyps. Endoscopic removal of the ACP by middle meatal antrostomy was typically performed under general anesthesia. The pedicle of the polyp was identified and incised, along with the surrounding 1 cm² of the sinus mucosa, by means of 30° and 70° endoscopes. The location of the pedicle attachment was determined during surgery.

Histopathologic Study

Two 5-μm-thick serial sections were cut from each specimen. One was stained by hematoxylin and eosin to confirm the histopathologic diagnosis. The other section was mounted on positively charged slides for immunohistochemical staining based on identification of lymph vessel endothelial hyaluronic acid receptor-1 (LYVE-1) in the endothelial cells of the lymphatic capillaries; this was the first lymph-specific hyaluronic acid receptor to be characterized and is a uniquely powerful marker for lymph vessels themselves. The number of lymphatic vessels and the extent of their dilatation were estimated individually; markedly positive, if there were more than 3 individual lymph vessels in the low-power field; moderately positive, if there were 2 or 3 individual lymph vessels in the low-power field; or mildly positive, if there was 1 individual lymph vessel in the low-power field.

Results

In this series of 25 ACPs, the antral part of the polyp was cystic in only 8% of cases (2 polyps) and polypoid in 92% (23 polyps). Twelve polyps (48%) originated from the inferolateral wall, 7 (28%) from the anterior wall, and 4 (16%) arose from the lateral and posterior walls; the remaining 2 cases (8%) in which there

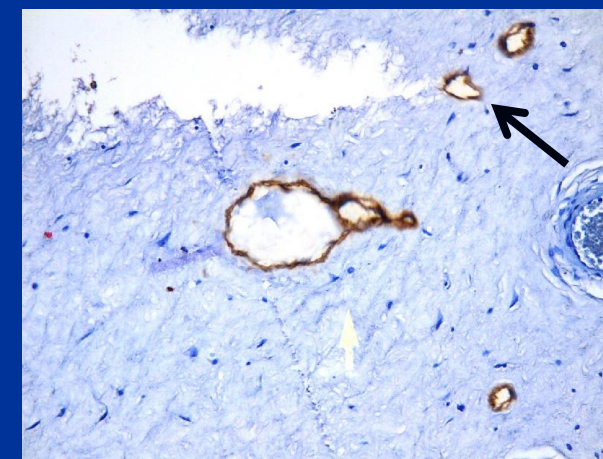


were cystic intramaxillary parts, the exact origin could not be determined.

Of the 25 ACP specimens, the density of the lymphatic vessels was marked in 22 (88%) and moderate in 3 (12%). There were 3 times as many superficial lymphatic vessels (red arrows) as deep vessels (black arrows) detected in each examined section. In the control group (25 CSB specimens), the density of the lymphatic vessels was marked in 4 specimens (16%), moderate in 5 (20%), and mild in 16 (64%), and there were twice as many deep lymphatic vessels as superficial ones. The orientation of both systems was the same as in normal tissue; however, the vertical interconnecting vessels were fewer than in normal tissue.

Discussion

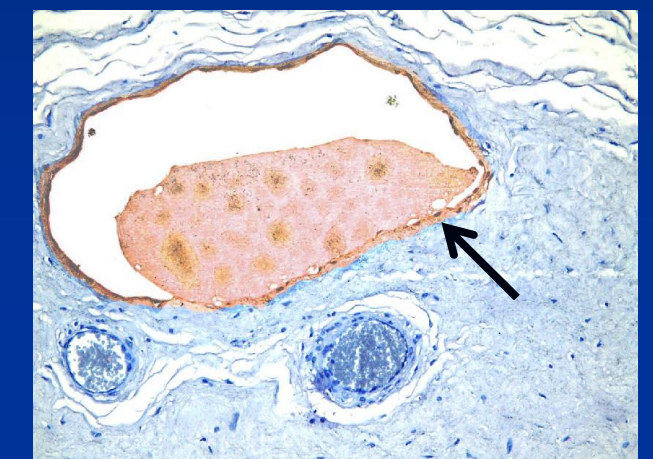
There is convincing evidence that most, if not all, ACPs develop from intramural cysts of the maxillary sinus. This peculiar origin is reflected in the presence of cysts in the antral portion of the polyp. Hosemann et al (4) observed postoperative mucosal swelling in the maxillary sinus after extensive middle meatal antrostomy.



They speculated that the inflammation represents a secondary lymphatic edema caused by drainage of lymph vessels that is obstructed during major maxillary fenestration. Kim et al (5) described the distributional and quantitative changes in the lymphatic vessels of normal sinus mucosa, edematous sinus mucosa, and polyps by using immunohistochemistry and Western blotting with D2-40 antibody. They concluded that the absence of lymphangiogenesis caused mucosal edema and the consequent formation of polyps.

Our investigation into the origin of ACPs resulted in two important findings: 1) the pedicled origin of the ACPs (23 cases; 92%) and the absence of an intramaxillary cyst; and 2) lymphatic vessels in ACPs of intrasinus mucosal origin that were of high density and marked enlargement in comparison with those of CSB specimens. These two findings refute “the

blocked acinus” theory and support lymphatic obstruction as the cause of ACPs.



Conclusions

This study resulted in two main findings. The first was the absence of intramaxillary cysts in the ACPs in 23 cases (92%). The second was the markedly high density of lymphatic vessels in the transitional area between the sinus mucosa and the pedicle of the ACPs, in comparison with the density in the control group. These two findings refute the “blocked acinus theory” and indicate that lymphatic obstruction, whether primary or secondary to chronic sinus infection, might play a leading role in the formation and further growth of ACPs.

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