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MORPHOLOGICAL FEATURES OF ANTROCHOANAL POLYPS

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Abstract

Aim. To determine the morphological features of antrochoanal polyps.
Materials and methods. The study was conducted using surgical material – removed antrochoanal polyps from 18 patients, 3 of whom had giant polyps extending into the oropharynx. From the obtained material, we prepared specimens stained using histological, histochemical, and immunohistochemical methods.
Results. Our studies confirm that antrochoanal polyps originate from the mucous membrane of the posterior wall of the maxillary sinus. Their maxillary, nasal and nasopharyngeal parts are covered with pseudostratified ciliated epithelium, where basal cell hyperplasia, desquamation of ciliated cells, and goblet cell transformation are observed, leading to a disrupted mucociliary transport on the surface. As a result of indirect squamous metaplasia, a stratified squamous epithelium is formed in the oropharyngeal part. Dysregenerative processes occurring in their stroma also play an important role in the pathogenesis of antrochoanal polyps. Diffuse leukocyte-plasmacyte cellular infiltrates, including B cells, eosinophils, and mast cells, are present in the stroma. Due to the proliferation of elastic fibers and adhesion of collagen fibers, fibrotic-cirrhotic changes occur, compressing arterial and venous microvessels in perivascular areas. In response to reduced blood flow in some vessels, compensatory blood flow increases in others. In the stroma of giant antrochoanal polyps that grow into the oropharynx new blood vessels form. Their angiogenic potential is realized through the secretion of vascular endothelial growth factor, which stimulates vasculogenesis and neoangiogenesis.
Conclusions. Antrochoanal polyps are covered with ciliated epithelium with goblet cell changes, but in the oropharyngeal part it is covered with stratified squamous epithelium. Edema, lymphostasis, diffuse leukoplasmacytic infiltration and fibro-cirrhotic changes with circulatory disorders are observed in their stroma. During the development of polyps their angiogenic potential is gradually formed which realized through the secretion of VEGF and stimulates the processes of vasculogenesis and neoangiogenesis.

Keywords: **antrochoanal polyp, mucous membrane, morphology**

INTRODUCTION

Antrochoanal polyps (ACPs), first described by Killian, account for 4-6% of all nasal polyps and share some common features as well as differences with other polyp types. The inflammatory spectrum of ACPs is centered around neutrophilic inflammation of a T1-dominant endotype [1, 2]. This represents a distinct pathophysiological mechanism of inflammatory remodeling of the mucosa [3]. In most cases, ACPs originate from the mucous membrane of the maxillary sinus; exit through the natural or accessory ostium into the middle nasal meatus, and then grow toward the choana and nasopharynx [4, 5]. There are also other primary localizations of choanal polyps, such as the sphenoid sinus (sphenochoanal polyp, which represents a rare form). As the name suggests, such a polyp originates in the sphenoid sinus and extends downward to the choana into the nasopharynx [6, 7]. There is evidence of differences in clinical and immunopathological characteristics in children and adults with ACPs [8, 9]. Clinically, ACPs manifest as nasal congestion or complete obstruction of the nasal cavity, headache, a sensation of a foreign body, rhinorrhea, and snoring, while larger polyps can also cause other symptoms such as difficulty swallowing [10]. The «gold standard» for their diagnosis is nasal endoscopy and computed tomography of the paranasal sinuses [11]. This type of nasal polyp is practically unresponsive to topical corticosteroids, making surgical treatment the only option [10, 12]. Microscopically, ACPs are described as remodeling of edematous mucosal tissue with fibrin deposition and the formation of cysts in the epithelium and stroma [13]. Overall, the morphology of ACPs has not been extensively studied. Currently, there are

only a few articles about the epithelium and stroma of similar polyps [14, 15, 16]. Despite the fact that modern diagnosis and treatment of ACPs are well-established, no fundamental morphological studies that would expand our understanding of their pathogenesis exist to date.

AIM

To identify the morphological features of antrochoanal polyps.

MATERIALS AND METHODS

The study used surgical material – ACPs removed under general endotracheal anesthesia from 18 patients. A shaver-assisted polypectomy of the nasal cavity and nasopharynx was performed, along with endoscopic maxillotomy to remove the base of the polyp. In 3 cases (16.7%), the polyps were giant and extended into the oropharynx. In these cases, the polyp body was first removed using forceps via an oral approach, followed by surgery as described above. All patients had an uncomplicated postoperative course. A follow-up examination was conducted 4 weeks later, and in none of the cases were remnants or recurrences of ACPs detected.

The obtained material was prepared according to standard methods using histological and histochemical staining: hematoxylin-eosin, picrofuchsin by Van Gieson's method, fuchsin-picrofuchsin, and PAS reaction with alcian blue and additional staining by Bergmann's method. For immunohistochemical studies, monoclonal antibodies were applied to differentiation clusters CD20, CD31 and VEGF (by «Dakocytomation»). The stained specimens were examined under an «Olympus BX-41» light microscope using objectives ×10, ×20, ×40. Microphotographs were taken with an «Olympus C 4040» camera using «Olympus DP-Soft».

RESULTS

In all cases, ACPs were unilateral and originated from the mucous membrane of the posterior wall of the maxillary sinus. The largest ACP measured 16 cm, extended into the oropharynx and protruded into the oral cavity (Fig. 1).

Figure 1. Pharyngoscopy: ACP in the oral cavity.

CT scans demonstrate that the base of the polyp originated from the mucosa of the posterior wall of the left maxillary sinus. The ACP extended through the natural opening, expanding the fontanelle and partially obstructing the posterior sections of the nasal cavity, descending into the nasopharynx and oropharynx. Pneumatization of the left maxillary sinus remained largely unchanged, except for polypoid changes in the mucosa of the posterior wall (the base of the polyp).

Morphological studies of the surgical material revealed that the maxillary, nasal, and nasopharyngeal parts of ACPs are covered with pseudostratified ciliated epithelium. However, unlike normal tissue, due to basal cell hyperplasia, the ciliated epithelial cells were positioned at varying distances from the basal membrane. Desquamation of ciliated cells was also observed, with destructive processes including loss of cilia, cytoplasmic vacuolization, and karyolysis. As a result of the desquamation of some ciliated cells, the epithelium became stratified, and in the underlying connective tissue of the propria of the mucosa, edema, moderate inflammatory infiltration, and signs of fibrosis were observed.

Some ACPs are covered with epithelium dominated by goblet cells, indicating goblet cell transformation. Their cytoplasm contains varying sizes of alcian-positive granules filled with mucous secretions. The cytoplasm of both short and long intercalated cells is vacuolated, making their

contours indistinct. The basal membrane of the epithelium is edematous and has a tortuous course. Numerous diffuse leuko-plasmacytic infiltrates that stain red are located in the underlying connective tissue (Fig. 2).

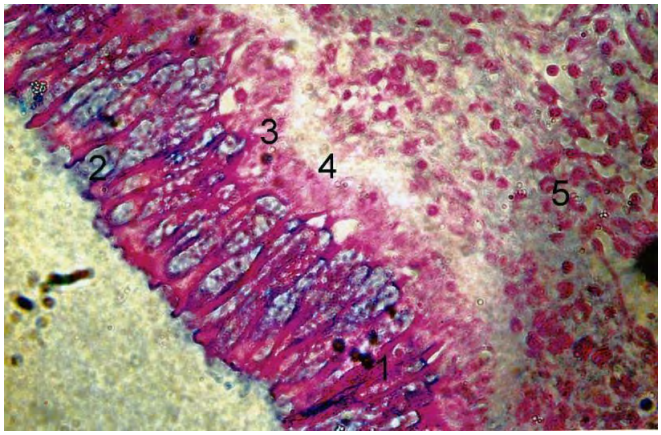

Figure 2. Epithelium of ACP with goblet-cell transformation: 1. ciliated cells; 2. goblet cells with alcian-positive granules; 3. short and long intercalated cells; 4. edematous basal membrane; 5. diffuse leuko-plasmacytic infiltrates in connective tissue. Stained using PAS-alcian blue with additional staining by Bergman's method. Magnification 20×10.

In the oropharyngeal part of ACPs indirect squamous metaplasia is observed, with gradual replacement of ciliated epithelium by stratified squamous epithelium. This type of epithelium consists of basal, parabasal, and spinous cells that undergo sequential differentiation. Unlike differentiated ciliated epithelial cells, which exhibit vertical anisomorphism (with nuclei oriented perpendicularly to the basal membrane), the nuclei of spinous cells are oriented parallel to the membrane, showing horizontal anisomorphism (Fig. 3).

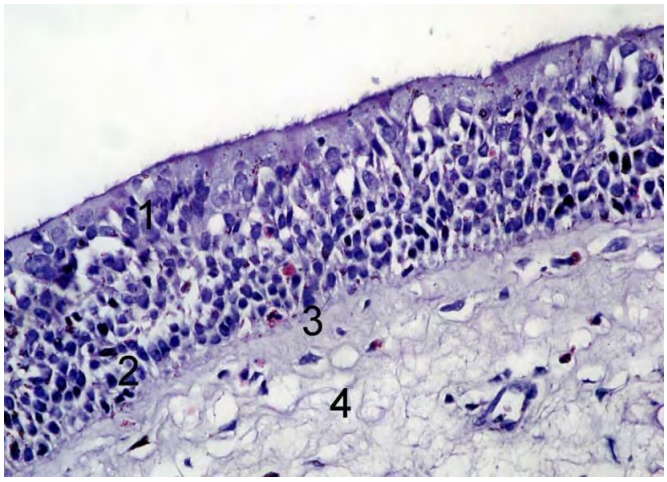

Figure 3. Area of ACP with squamous metaplasia of the epithelium: 1. spinous cells; 2. basal cell hyperplasia; 3. basal membrane; 4. edematous connective tissue. Stained with hematoxylin-eosin. Magnification 20×10.

In the stroma of ACPs, edema, lymphostasis, inflammatory cellular infiltration, fibrotic-cirrhotic changes, and even processes of vasculogenesis and neoangiogenesis are observed.

Initially, numerous perivascular cellular infiltrates form, and later, leukocyte-plasmacytic infiltration of the stroma becomes more pronounced and diffuse. The inflammatory infiltrates include eosinophils with segmented nuclei and red cytoplasm containing eosinophilic granules. Other cellular elements contain an eccentrically located nucleus and basophilic cytoplasm, characteristic of plasmocytes. Nearby, one can see lymphocyte-like cells with a round nucleus and a small rim of cytoplasm, as well as occasional mast cells, which are large cells with basophilic cytoplasm containing numerous granules.

In the perivascular connective tissue of the stroma, a large number of CD20-positive B cells with varying degrees of expression are identified. The examination of micropreparations stained with picrofuchsin by Van Gieson's method indicates that fibroblast proliferation occurs in the stroma of ACPs. These cells have elongated cytoplasm and contain spindle-shaped nuclei. Nearby, within the formed collagen fibers, fibrocytes with flattened cytoplasm and more elongated nuclei are located. In some areas of the stroma, elastic fibers form alongside the proliferation of collagen fibers. This is confirmed by

our own research using a combined staining method with fuchselin by Hart's method and additional staining with picrofuchsin by Van Gieson. The proliferation of elastic fibers and the adhesion of collagen fibers lead to the formation of scar tissue in perivascular areas, compressing the arterial and venous microvessels. Adaptive remodeling of the vascular wall occurs in response to the reduction of blood flow in arteries. It is manifested by hyperplasia of their smooth muscle layer. As a result of hyperplasia of longitudinally oriented smooth muscle cells, valve-like structures form in the lumen of the arteries. Such adaptive remodeling of the vascular wall suggests that compensatory blood flow increases in some vessels in response to reduced blood flow in others.

Immunohistochemical studies using the specific endothelial marker CD31 confirm that circulatory disturbances occur in the stroma of ACPs. Two types of adaptive changes are observed in the walls of arterial vessels. The first type characterizes a reduction in blood flow and is manifested by the narrowing of lumens. The endothelium in such vessels has a corrugated appearance and a high degree of CD31 expression. The second type of remodeling, on the contrary, is characterized by increased blood flow, manifested by dilation of the vessel lumens. The endothelium lining the inner surface of these arterial vessels also exhibits a high degree of CD31 expression and a more orderly structure (Fig. 4).

Certain adaptive changes are also observed in the arterioles, indicating circulatory disturbances in the more superficial layers of the stroma. In some arterioles, perivascular sclerosis and hyalinization are associated with reduced blood flow. In contrast, increased blood flow is observed in other arterioles, accompanied by hyperplasia of their smooth muscle layer. Thus, compensatory increases in blood flow occur in some arterioles in response to reduced blood flow in others.

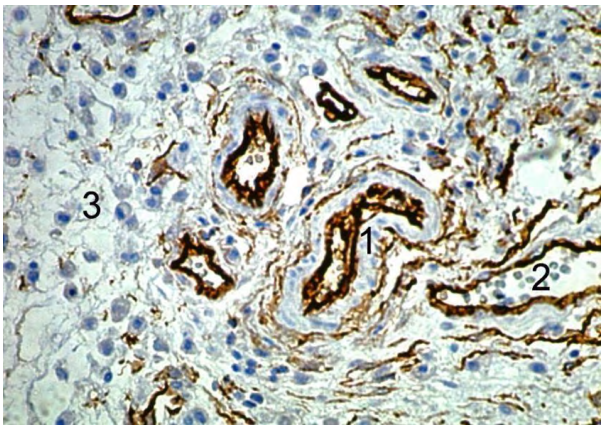

Figure 4. Expression of CD31 in the stroma of ACP: 1. expression in the endothelium of narrowed arteries; 2. expression in the endothelium of dilated arteries; 3. connective tissue. Immunohistochemical method, additional staining with Mayer's hematoxylin. Magnification 20×10.

The conducted immunohistochemical studies demonstrate that in the stroma of giant ACPs that grow into the oropharynx new blood vessels form. Initially, bud-like clusters of VEGF-positive proliferating endothelial cells appear in the connective tissue beneath the epithelium. These clusters subsequently give rise to capillaries. Simultaneously, arterioles and venules form in the deeper layers of the stroma (Fig. 5).

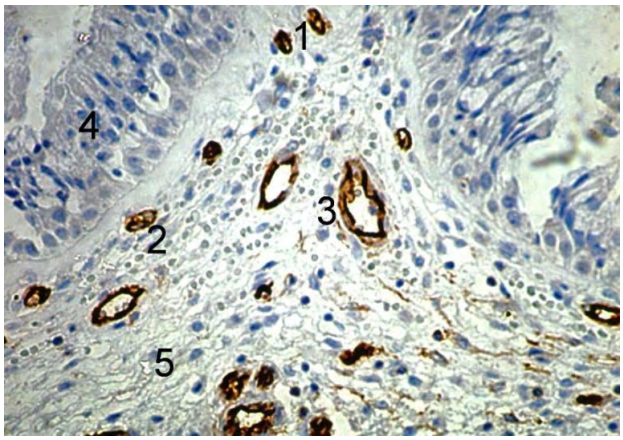

Figure 5. Expression of VEGF in the stroma of ACP: 1. clusters of VEGF-positive endothelial cells; 2. expression in the endothelium of capillaries; 3. expression in the endothelium of arterioles and venules; 4. epithelium; 5. connective tissue. Immunohistochemical method, additional staining with Mayer's hematoxylin. Magnification 20×10.

DISCUSSION

Our study confirms existing research [1, 4, 5] indicating that ACPs represent a unique remodeling of the mucosa, with their base located on the posterior wall of the maxillary sinus near the natural opening.

Ciliary structural impairment is an important pathological phenomenon in ACPs [16]. There is an opinion that only intranasal portion of ACP is covered with a ciliated respiratory epithelium similar to the normal mucosa of the sino-nasal tract and the choanal portion shows squamous metaplasia [17]. This is confirmed by our research, that showed that the maxillary, nasal and nasopharyngeal parts of ACPs are covered with pseudostratified ciliated epithelium, where basal cell hyperplasia, desquamation of ciliated cells and goblet-cell transformation are observed. Such changes lead to disruption of mucociliary transport on their surface. In the oropharyngeal part indirect squamous metaplasia results in the formation of stratified squamous epithelium.

Dysregenerative processes occurring in their stroma play a significant role in the genesis of ACPs [15]. Based on our research, it was found that edema, lymphostasis, diffuse leukoplasmacytic cellular infiltration and fibro-cirrhotic changes with circulatory disorders are observed in their stroma. The obtained results of the study are in line with findings reported in the literature [14, 18, 19], indicating that inflammatory changes are the main pathophysiological processes in the pathogenesis of ACPs while allergy plays only a minor role.

Angiogenesis probably plays a role in the pathogenesis of ACPs. However, their angiogenic potential is much lower than in nasal polyps [20]. Our research confirms, that in the stroma of giant ACPs that grow into the oropharynx VEGF-positive proliferating endothelial cells are determined and new blood vessels form. Thus, the angiogenic potential of ACP is gradually formed during their development and is realized through the secretion of VEGF, which stimulates the processes of vasculogenesis and neoangiogenesis. Since surgery is not always successful, further studies of ACP morphology will facilitate the development of pathogenetic anti-recurrence treatments.

CONCLUSIONS

Antrochoanal polyps exhibit morphological heterogeneity of the epithelial lining: the maxillary, nasal, and nasopharyngeal portions are covered with pseudostratified ciliated epithelium with goblet-cell transformation, whereas the oropharyngeal region is lined with stratified squamous epithelium due to indirect squamous metaplasia. The stromal component demonstrates marked edema, lymphostasis, diffuse leukoplasmacytic infiltration, and fibro-cirrhotic alterations, all accompanied by microcirculatory disturbances. Vascular remodeling, including compensatory changes in arterial and venular structures, reflects adaptive responses to impaired blood flow.

The gradual development of the angiogenic potential of polyps, mediated by the secretion of vascular endothelial growth factor (VEGF), constitutes a significant element of their pathogenesis. This mechanism underlies the processes of vasculogenesis and neoangiogenesis in the stroma, particularly evident in giant polyps extending into the oropharynx. The findings provide novel insights into the morphological features of antrochoanal polyps and may inform the development of pathogenetically targeted therapeutic approaches.

Perspectives for further research. Given the limited efficacy of topical corticosteroids in the treatment of antrochoanal polyps (ACPs), further in-depth morphological investigations are warranted to elucidate the cellular and molecular mechanisms underlying their resistance to conservative therapy. Special attention should be paid to the role of epithelial remodeling, stromal fibrosis, and angiogenic signaling pathways, particularly the VEGF-mediated cascade, in the progression and recurrence of ACPs.

Future studies should aim to identify potential histopathological or immunohistochemical markers predictive of recurrence risk and responsiveness to adjuvant pharmacological interventions. This may facilitate the development of targeted pathogenetic therapies that would complement surgical treatment and reduce the likelihood of relapse, thereby improving patient outcomes and optimizing long-term disease management strategies.

COMPLIANCE WITH ETHICAL REQUIREMENTS

The research was performed according to the Rules of Humane Treatment of Patients in accordance with the requirements of the Tokyo Declaration of the World Medical Association, the International Recommendations of the Helsinki Declaration on Human Rights and the Council of Europe Convention on Human Rights and Biomedicine. The study protocol was approved by the Ethics Committee of Poltava State Medical University (No. 234 from 23.01.2025).

In line with confidentiality regulations, all data was collected anonymously and processed in accordance with applicable data protection standards. All information was utilized solely within the context of this study and was provided in an aggregated form for results analysis.

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AUTHOR CONTRIBUTIONS

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REFERENCES

1. Fokkens, W. J., Lund, V. J., Hopkins, C., Hellings, P. W., Kern, R., Reitsma, S., Toppila-Salmi, S., Bernal-Sprekelsen, M., Mullol, J., Alobid, I., Terezinha Anselmo-Lima, W., Bachert, C., Baroody, F., von Buchwald, C., Cervin, A., Cohen, N., Constantinidis, J., De Gabory, L., Desrosiers, M., Diamant, Z., … Zwetsloot, C. P. (2020). European Position Paper on Rhinosinusitis and Nasal Polyps Rhinology, 58(Suppl S29), 1-464. https://doi.org/10.4193/Rhin20.600.
2. Bousquet, J., Anto, J. M., Bachert, C., Haahtela, T., Zuberbier, T., Czarlewski, W., Bedbrook, A., Bosnic-Anticevich, S., Walter Canonica, G., Cardona, V., Costa, E., Cruz, A. A., Erhola, M., Fokkens, W. J., Fonseca, J. A., Illario, M., Ivancevich, J. C., Jutel, M., Klimek, L., Kuna, P., … Zidarn, M. (2021). ARIA digital anamorphosis: Digital transformation of health and care in airway diseases from research to practice. Allergy, 76(1), 168-190. https://doi.org/10.1111/all.14422.
3. Garaycochea, O., Van Strahlen, C. R., Alobid, I., & Mullol, J. (2023). Pheno-Endotyping Antrochoanal Nasal Polyposis. Current allergy and asthma reports, 23(3), 165-180. https://doi.org/10.1007/s11882-023-01066-1.
4. Alharbi, S. M., Alshehri, A. M., & Erwe, I. H. (2023). Huge Antrochoanal Polyp: A Case Report. Cureus, 15(9), e45592. https://doi.org/10.7759/cureus.45592.
5. Maldonado, M., Martínez, A., Alobid, I., & Mullol, J. (2004). The antrochoanal polyp. Rhinology, 42(4), 178-182.
6. Jadia, S., Goyal, R., & Biswas, R. (2010). Nasal mass mimicking antrochoanal polyp. BMJ case reports, 2010, bcr12.2009.2578. https://doi.org/10.1136/bcr.12.2009.2578.
7. Bayazid, L., & Josephson, G. D. (2023). Sphenochoanal polyps. BMJ case reports, 16(5), e251182. https://doi.org/10.1136/bcr-2022-251182.
8. Tu, Y., Jiang, T., Wu, Y., Luo, J., Sun, S., Liu, C., Yu, P., Chen, A., Ji, H., Wan, Y., Yu, L., & Shi, L. (2024). Difference of Antrochoanal Polyps Between Children and Adults in the Chinese Population. The Laryngoscope, 134(5), 2093-2099. https://doi.org/10.1002/lary.31154.
9. Tywoniuk, K., Haber, K., & Mierzwiński, J. (2022). Sphenochoanal polyps in children – a systematic review (1995–2021). Brazilian journal of otorhinolaryngology, 88 Suppl 5(Suppl 5), S179–S187. https://doi.org/10.1016/j.bjorl.2022.02.006.
10. Frosini, P., Picarella, G., & De Campora, E. (2009). Antrochoanal polyp: analysis of 200 cases. Acta otorhinolaryngologica Italica: organo ufficiale della Societa italiana di otorinolaringologia e chirurgia cervico-facciale, 29(1), 21-26.
11. Hasiuk, P., Korobeinikova, Y., Vorobets, A., Korobeinikov, L., Dzetsiukh, T., Rosolovska, S., & Gurando, V. (2021). Comparative assessment of the state of bone resorption in patients with chronic generalized periodontitis according to orthopantomogram and cone-beam computed tomography. Polski merkuriusz lekarski: organ Polskiego Towarzystwa Lekarskiego, 49(292), 286-289.
12. Jagadeeswaran, V. U., Vallur, S., & Shivanand, J. S. (2022). Management of Angiomatous Antrochoanal Polyp: Our Experience. Indian journal of otolaryngology and head and neck surgery: official publication of the Association of Otolaryngologists of India, 74(Suppl 2), 1082-1087. https://doi.org/10.1007/s12070-020-02148-6.
13. Keshri, N., Bansal, A., Popli, G., Venkatesh, A., & Goel, S. (2017). Antrochoanal polyp arising from benign pseudocyst of maxillary antrum. Journal of the Indian Society of Pedodontics and Preventive Dentistry, 35(3), 275-278. https://doi.org/10.4103/JISPPD.JISPPD_153_16.
14. Jin, P., Zi, X., Charn, T. C., Liu, J., Yan, Y., Shi, L., Zhang, H., & Wang, D. (2018). Histopathological features of antrochoanal polyps in Chinese patients. Rhinology, 56(4), 378-385. https://doi.org/10.4193/Rhin18.057..
15. Bezshapochnyĭ, S. B., Gasiuk, I.uA., & Balinskiĭ, V. A. (2014). Vestnik otorinolaringologii, (3), 9-11.
16. Zi, X., Peng, Y., Zang, Y., Chen, S., Li, M., Yu, K., Liang, X., Jin, P., Wang, D., & Shi, L. (2023). An Integrated Analysis Reveals Ciliary Abnormalities in Antrochoanal Polyps. Journal of inflammation research, 16, 605-615. https://doi.org/10.2147/JIR.S398371.
17. Meir, W., Bourla, R., Huszar, M., & Zloczower, E. (2021). Antrochoanal Polyp: Updated Clinical Approach, Histology Characteristics, Diagnosis and Treatment. IntechOpen. doi: 10.5772/intechopen.96329
18. Fiorentino, V., Martini, M., Galletti, C., Pizzimenti, C., Franchina, M., Ieni, A., Fadda, G., Galletti, B., &

Tuccari, G. (2024). The Morphology of Nasal Polyps in Different Age Groups: Histopathological Features. Journal of personalized medicine, 14(4), 414. https://doi.org/10.3390/jpm14040414.
19. Chen, C. L., Wang, Y. T., Yao, Y., Pan, L., Guo, B., Zhu, K. Z., Ma, J., Wang, N., Li, X. L., Deng, Y. K., & Liu, Z. (2021). Inflammatory Endotypes and Tissue Remodeling Features in Antrochoanal Polyps. Allergy, asthma & immunology research, 13(6), 863-881. https://doi.org/10.4168/aair.2021.13.6.863.
20. Hirshoren, N., Neuman, T., Gross, M., & Eliashar, R. (2011). Angiogenesis in chronic rhinosinusitis with nasal polyps and in antrochoanal polyps. Inflammation research: official journal of the European Histamine Research Society, 60(4), 321-327. https://doi.org/10.1007/s00011-010-0271-8.

Резюме

МОРФОЛОГІЧНІ ОСОБЛИВОСТІ АНТРОХОАНАЛЬНИХ ПОЛІПІВ

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Мета. Встановити морфологічні особливості антрохоанальних поліпів.
Матеріали та методи. Для дослідження використали операційний матеріал – видалені антрохоанальні поліпи у 18 пацієнтів, 3 з яких були гігантськими і проростали у ротоглотку. Із отриманого матеріалу виготовляли препарати, які фарбували гістологічними, гістохімічними та імуногістохімічними методами.
Результати. Наші дослідження підтверджують, що антрохоанальні поліпи походять із слизової оболонки задньої стінки вехньощелепного синуса. Їх максилярна, назальна і носоглоткова частини покриті багаторядним миготливим епітелієм, де спостерігається базальноклітинна гіперплазія, десквамація миготливих клітин та келихоподібна трансформація, в результаті чого на поверхні виникає порушення мукоциліарного транспорту. Внаслідок непрямої плоскоклітинної метаплазії в ротоглотковій частині формується багатошаровий плоский епітелій. Дисрегенераторні процеси, які відбуваються у їх стромі, також відіграють важливу роль у патогенезі антрохоанальних поліпів. У стромі формуються дифузні лейкоплазмоцитарні клітинні інфільтрати, до складу яких входять В-клітини, еозинофіли, а також лаброцити. Внаслідок розростання еластичних, а також склеювання колагенових волокон виникають фіброзно-циротичні зміни, які у периваскулярних ділянках здавлюють артеріальні і венозні мікросудини. У відповідь на редукцію кровотоку в одних судинах в інших виникає компенсаторне його підвищення. У стромі гігантських антрохоанальних поліпів, що проростають у ротоглотку, формуються нові кровоносні судини. Цей їх ангіогенний потенціал реалізується через секрецію VEGF, який стимулює процеси васкулогенезу і неоангіогенезу.
Висновки. Антрохоанальні поліпи вкриті келихоподібно зміненим миготливим епітелієм, а в ротоглотковій частині – багатошаровим плоским. У їх стромі спостерігається набряк, лімфостаз, дифузна лейкоплазмоцитарна інфільтрація та фіброзно-циротичні зміни з порушенням кровообігу. В ході розвитку поліпів поступово формується їх ангіогенний потенціал, який реалізується через секрецію VEGF, що стимулює процеси васкулогенезу та неоангіогенезу.

Ключові слова: **антрохоанальний поліп, слизова оболонка, морфологія**

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