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PROBLEMS OF SYSTEMATIZATION AND TREATMENT OF VASCULAR LESIONS OF THE PAROTID GLAND IN CHILDREN AND ADULTS: LITERATURE REVIEW AND CLINICAL OBSERVATIONS

Summary.

The classification of vascular lesions of the parotid gland remains a subject of considerable debate, complicating clinical management. This article addresses these controversies by proposing a systematic categorization into malformations, hyperplasias, and tumors (hemangiomas). We advocate for a diagnostic and therapeutic approach guided by the assessment of GLUT-1 and CD34 immunohistochemical markers to resolve diagnostic uncertainty.

***The objective** is to investigate the issues surrounding the classification and treatment of parotid salivary gland vascular lesions in children and adults, summarize contemporary literature, and analyze our own clinical observations with particular consideration of immunohistochemical markers (GLUT-1, CD34).*

***Materials and methods.** We conducted a review of current literature on the classification, diagnosis, and treatment of parotid gland vascular pathologies. Three clinical cases of patients aged 33 to 57 years with vascular lesions of the parotid salivary gland were analyzed. Diagnostic methods included clinical examination, ultrasonography with Doppler imaging, magnetic resonance imaging, fine-needle aspiration biopsy, and subsequent histological and immunohistochemical studies. The recommended principles of bioethics were adhered to during the research. The research was conducted as part of the scientific research project 'Development of methods for surgical and comprehensive treatment of patients with congenital and acquired pathologies of the maxillofacial region with restoration of functions and taking into account concomitant pathologies', state registration number 0123U100745, completion date – 2023-2028.*

***Results.** The analyzed cases demonstrated different vascular pathologies: capillary hemangioma, a combined hemangioma and pleomorphic adenoma, and vascular hyperplasia. Immunohistochemical analysis with GLUT-1 and CD34 markers confirmed the diagnoses. Treatment involved partial parotidectomy with tumor excision using organ-preserving techniques and intraoperative facial nerve monitoring. All patients experienced an uneventful postoperative recovery.*

***Conclusions.** Vascular lesions of the parotid salivary gland in children are typically hemangiomas with a characteristic clinical course. In adults, rare variants such as epithelioid hemangiomas and vascular hyperplasias are more common. Precise differential diagnosis requires a combination of clinical presentation, imaging modalities, and immunohistochemical profiling. Surgical intervention remains the treatment of choice in complicated or progressive cases.*

***Key words:** Oral Cavity; Salivary Glands; Congenital Malformations; Hyperplasia, Tumors; Pathological Salivary Gland Lesions; Immunohistochemical Markers of Proliferation and Apoptosis; Surgical Interventions on Salivary Glands.*

Introduction

Vascular anomalies of the salivary glands are among the most common benign vascular tumors in childhood and, in many cases, typically following a three-phase course of proliferation, stabilization, and spontaneous involution [1, 2]. Approximately 60% of all vascular lesions are localized in the head and neck region, with up to 10% involving the parotid salivary gland [3]. In adults, hemangiomas of the parotid salivary gland are exceedingly rare, comprising only 0.4-0.6% of all parotid tumors [4]. This localization is clinically significant due to the proximity of the facial nerve, risk of functional impairment, and significant cosmetic defects associated with tumor growth [5].

The objective is to examine the challenges in classifying and treating vascular lesions of the parotid salivary gland in children and adults, review contemporary literature, and analyze our clinical experience with consideration of immunohistochemical markers (GLUT-1, CD34).

Materials and methods.

We conducted a review of current literature on the classification, diagnosis, and management of parotid gland vascular pathologies. Three clinical cases of patients aged 33 to 57 years with vascular lesions of the parotid salivary gland were analyzed. Diagnostic methods included clinical examination, Doppler ultrasonography, magnetic resonance imaging, fine-needle aspiration biopsy, and histological/immunohistochemical studies. The recommended principles of bioethics were adhered to during the research. The research was conducted as part of the scientific research project 'Development of methods for surgical and comprehensive treatment of patients with congenital and acquired pathologies of the maxillofacial region with restoration of functions and taking into account concomitant pathologies', state registration number 0123U100745, completion date – 2023-2028.

Results. The first clinical manifestations of parotid salivary gland vascular lesions typically appear in children

before six months of age. Most present as a painless, soft-elastic swelling in the parotid region, often with bluish or purplish skin discoloration due to superficial vascularization. The active growth phase generally occurs within the first 6-12 months of life, followed by gradual involution occurs in 70-90% of cases [6, 7], a pattern consistent with our clinical observations (Fig. 1).

However, according to Leaute-Labreze et al. (2008), approximately 20% of vascular lesions follow a complicated course that necessitates active pharmacological or surgical intervention [8], a finding supported by our clinical cases (Figs. 2-4).



Fig. 1. Spontaneous regression of an infantile hemangioma in a 5-month-old child.



Fig. 2. Patient M., 9 months old. High-flow malformation of the parotid gland.

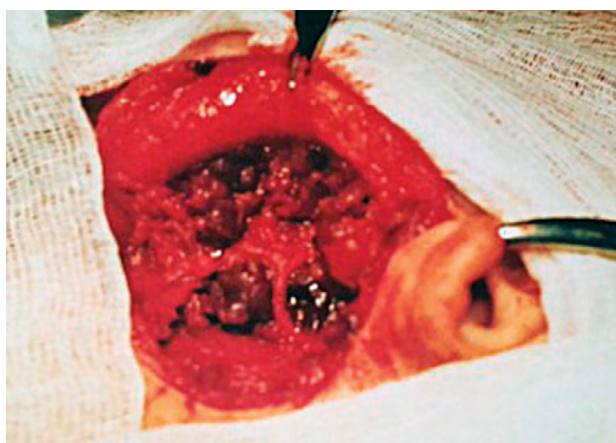


Fig. 3. The same patient. Procedure: superficial parotidectomy with tumor excision.



Fig. 4. The same patient. Completion of the procedure: superficial parotidectomy with excision of the vascular tumor.

Doppler ultrasonography serves as the primary imaging modality, enabling evaluation of vascular architecture, tumor margins, and vascularization density [9]. Contrast-enhanced magnetic resonance imaging (MRI) provides the most detailed assessment of volumetric characteristics, invasion depth, and involvement of adjacent structures [6]. Differential diagnosis should include lymphangiomas, venous malformations, pleomorphic adenomas, and embryonal sarcomas, particularly in cases with atypical growth patterns [10].

Histologically, vascular lesions consist of dense capillary network lined by proliferatively active endothelial cells showing high GLUT-1 expression. This immunohistochemical marker is pathognomonic for infantile hemangiomas and facilitates differentiation from other vascular malformations [11].

The pharmacological management of complicated hemangiomas changed significantly after the introduction of the β -blocker propranolol in 2008. According to Leaute-Labreze et al. (2008), propranolol therapy at a dose of 2-3 mg/kg/day led to a clinically significant reduction in tumor volume within the first 2-4 weeks in 88-92% of patients [8]. Subsequent studies have confirmed the efficacy and safety of prolonged treatment for up to 6-12 months. For patients with contraindications or an inadequate response to β -blockers, therapeutic alternatives include systemic corticosteroids, Pulsed Dye Laser (PDL) treatment, or surgical intervention [12-15].

Surgical management of parotid gland vascular lesions is typically deferred until after the proliferative phase, except, in cases requiring earlier intervention for ulceration, hemorrhage, facial nerve compression, or facial deformity. Organ-preserving techniques with intraoperative neuromonitoring are preferred to minimize the risk of facial nerve injury [16].

Vascular malformations, hyperplasias, and hemangiomas of the parotid gland present particular diagnostic and therapeutic challenges. The pathogenesis of vascular anomalies of the parotid gland in adults require further investigation. Intraglandular localization complicates differential diagnosis, necessitating distinction from pleomorphic adenoma, lymphangioma, Warthin tumor, and angiolymphoid dysplasia

based on clinical presentation, histological architecture, and the extent of surgical intervention.

We present a description of three clinical cases of vascular neoplasms of the parotid gland in adults.

Case 1. Patient K., 33 years old, was admitted to the Department of Maxillofacial Surgery at the Vinnytsia

Regional Clinical Hospital named after M. I. Pirogov in January 2025 with complaints of a mass in the right parotid region (Fig. 5). The lesion had appeared approximately six months earlier, demonstrating slow enlargement interspersed with three distinct episodes of rapid growth followed by spontaneous regression.



Fig. 5. Patient K., 33 years old. Diagnosis: Vascular lesion of the right parotid gland.

Clinical findings: Examination revealed a 4 × 5 cm, ovoid, soft-elastic formation with limited mobility beneath the tragus. The mass exhibited slight enlargement when the head was tilted downward.

MRI findings: The imaging study described the lesion as suggestive of either a *pleomorphic adenoma* or a *Warthin tumor (adenolymphoma)*.

Fine-needle aspiration biopsy: The procedure yielded 0.2-0.3 ml of blood.

Surgical intervention: A *partial parotidectomy with tumor excision* was performed. The obtained

specimens were submitted for histopathological and immunohistochemical evaluation.

Histological report (No. 6556-6558): The benign lesion consisted of clusters of tightly arranged, small capillary-type vessels. These vessels displayed congested and optically empty lumina, were lined by a single-to-double layered endothelium, and were separated by thin fibrous strands. The analysis also identified moderate cellular inflammatory infiltration and ulceration of the lesion.

Pathohistological conclusion: *Capillary hemangioma* (Figs. 6-9).

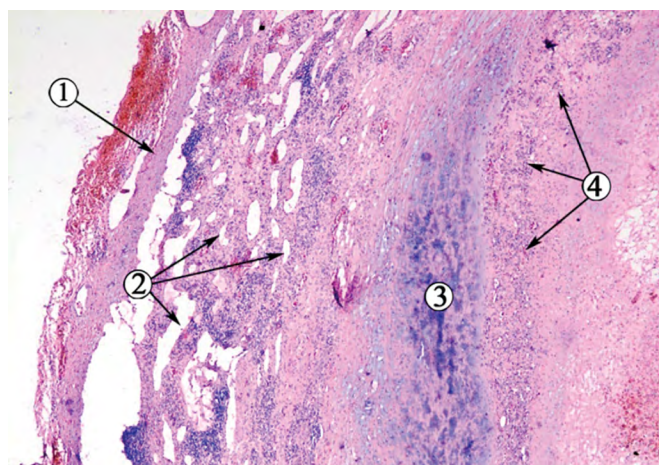


Fig. 6. Histopathology of the right parotid gland lesion from Patient K., 33 years. Hemangioma of the parotid salivary gland with an outer fibrous capsule (1), a zone of predominantly cavernous-type vessels (2), an area with chondroid structure (3), and a region of predominantly capillary-type vessels (4). HE stain, 40×.

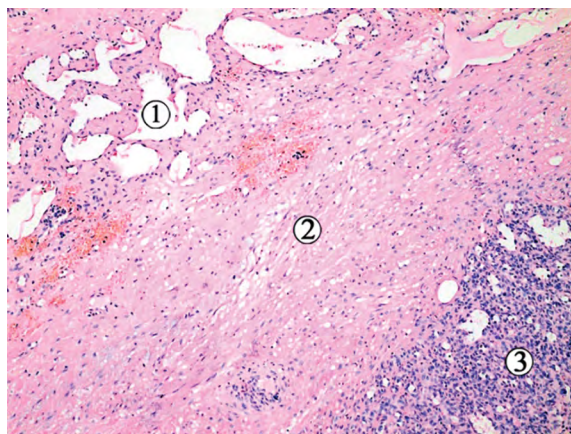


Fig. 7. Histopathology of the right parotid gland lesion from the same patient. Hemangioma of the parotid salivary gland with an area predominantly of cavernous-type vessels (1), separated by a layer of dense fibrous tissue (2) from an area of predominantly capillary-type vessels (3). HE stain, 100×.

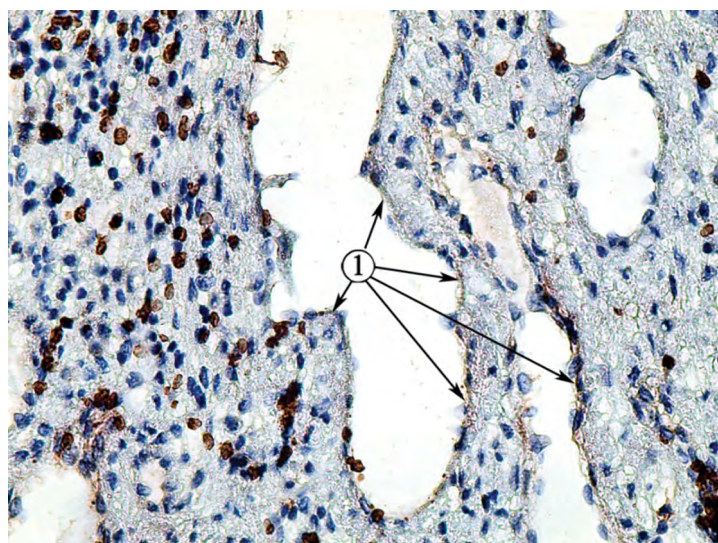


Fig. 8. Histopathology of the right parotid gland lesion from the same patient. Weakly positive membranous expression of the GLUT1 marker in the vascular endothelium of the tumor (1). HE stain, 100×..

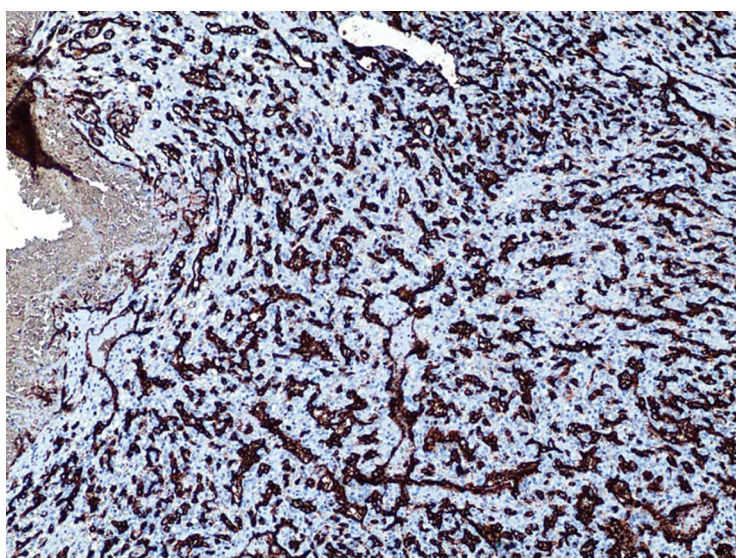


Fig. 9. Histopathology of the right parotid gland lesion from the same patient. Strong positive membranous–cytoplasmic expression of the CD34 marker in the vascular endothelium of the tumor. HE ×100.

Postoperative course: The wound healed by primary intention without complications.

Clinical case 2.

Patient Ch., 57 years old, presented to the Department of Maxillofacial Surgery at the Vinnytsia Regional Clinical Hospital named after M. I. Pirogov in June 2025 with a slowly enlarging mass anterior to the right tragus.

Clinical findings: Examination revealed a 2 cm, ovoid, soft-elastic, and minimally mobile lesion in the parotid region anterior to the tragus. The tumor size increased slightly upon head tilting.

Surgical intervention: The patient underwent *partial parotidectomy with tumor excision*. The obtained specimens were submitted for histopathological and immunohistochemical evaluation.

Histopathological conclusion: *Hemangioma in combination with pleomorphic adenoma of the right parotid salivary gland* (Figs. 10-12).

Postoperative course: The wound healed by primary intention without complications..

Clinical case 3. Patient P., 50 years old, presented to the Department of Head and Neck Tumors at the Podillia Regional Oncology Center with a gradually enlarging mass anterior to the right tragus. The patient's initial symptoms had appeared nine years prior.

Clinical findings: Examination identified a soft, painless, non-fluctuating mass with well-defined margins measuring 3 × 4 cm in the specified location.

Surgical intervention: *Parotidectomy with tumor excision* was performed. The specimens were submitted to histopathological and immunohistochemical analysis.

Histopathological conclusion: *Vascular anomaly (hyperplasia) of the right parotid salivary gland* (Figs. 13-15).

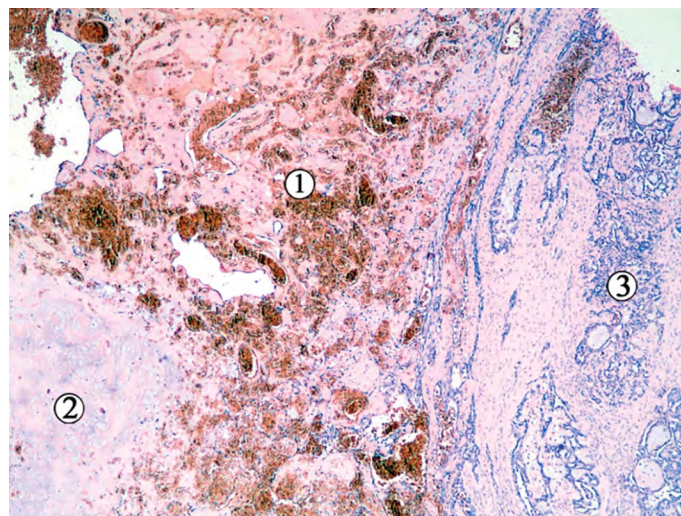


Fig. 10. Patient Ch., 57 years old. Hemangioma (1) in combination with pleomorphic adenoma of the parotid salivary gland. Mesenchymal component (2) and epithelial component (3). HE stain, 40×.

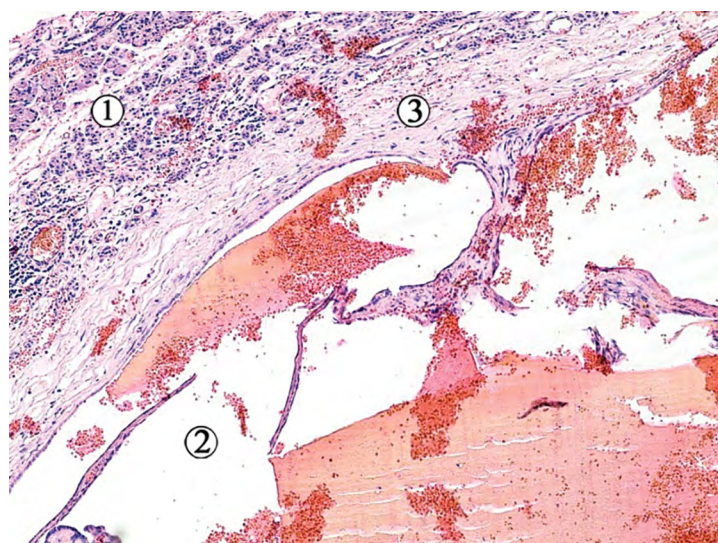


Fig. 11. The same patient. Hemangioma (1) in combination with pleomorphic adenoma of the parotid salivary gland. Preserved salivary gland parenchyma (1), separated from the hemangioma (2) by a fibrous septum (3). HE stain, 100×.

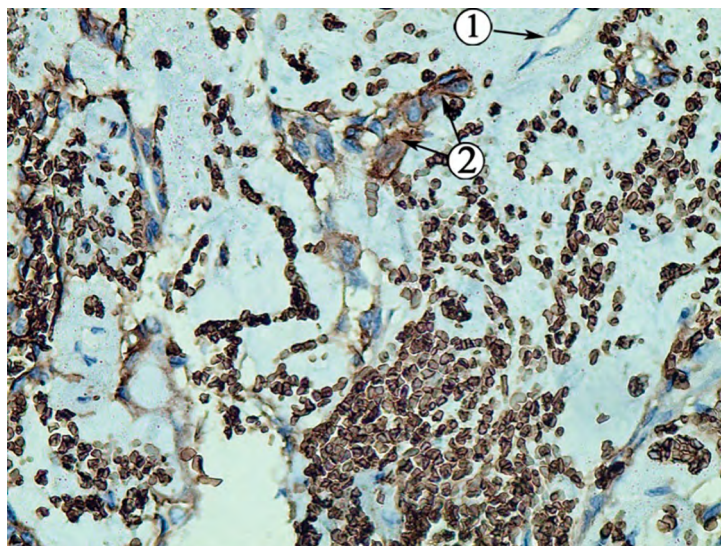


Fig. 12. The same patient. Hemangioma (1) in combination with pleomorphic adenoma of the parotid salivary gland. Absence of membranous GLUT1 expression in the vascular endothelium of the tumor stroma (1); positive GLUT1 expression (2) in the endothelium of tumor-associated vessels. HE stain, 400 \times .

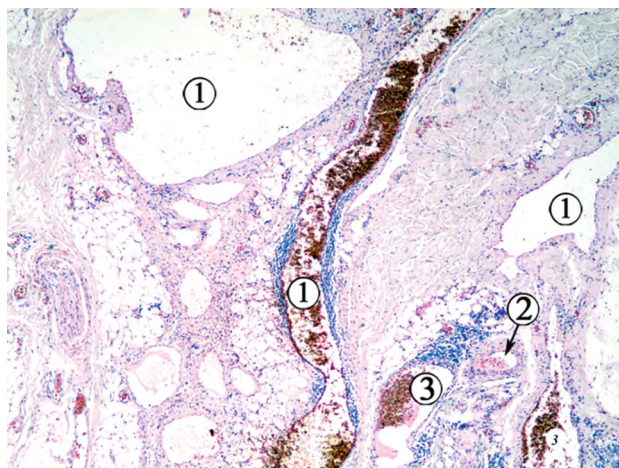


Fig. 13. Histopathology of the right parotid gland lesion from Patient P., 50 years. Vascular hyperplasia of the parotid salivary gland. Predominantly venous vessels (1) of varying calibers; vascular bundle: arteriole (2), accompanying venules (3). HE stain, 40 \times .

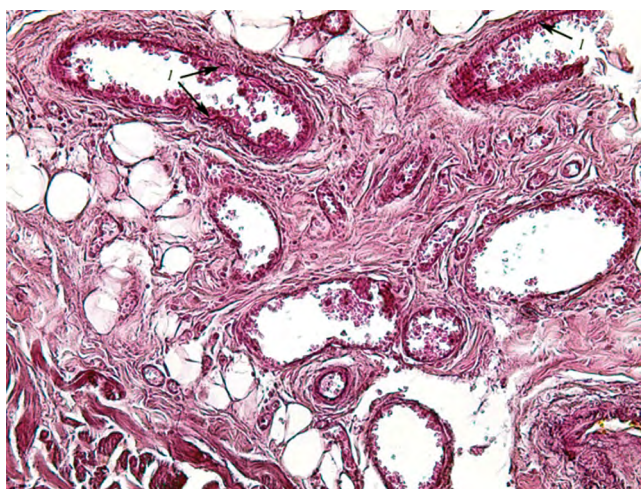


Fig. 14. Histopathology of the right parotid gland lesion from the same patient. Blood vessels with a discontinuous internal elastic membrane (1) in their wall. Weigert's resorcin-fuchsin stain, 200 \times .

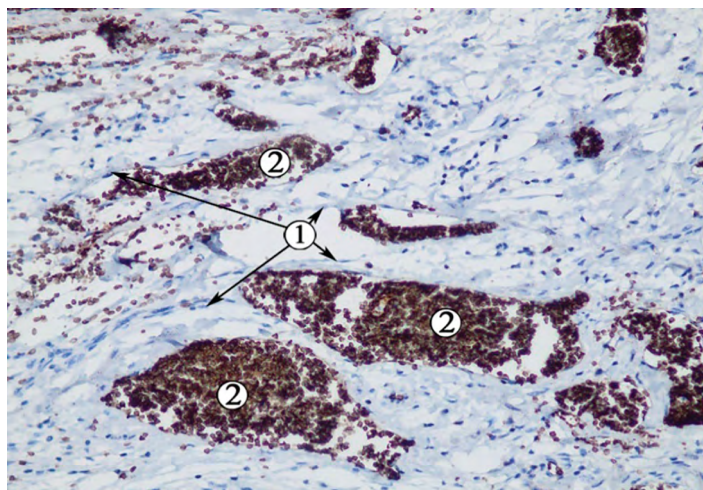


Fig. 15. Histopathology of the right parotid gland lesion from the same patient. Vascular (venous) hyperplasia of the parotid salivary gland. Absence of GLUT1 expression in the vascular endothelium of the tumor (1), with positive expression in erythrocytes within the vascular lumen (2). 200x.

Discussion

According to the current recommendations of the International Society for the Study of Vascular Anomalies (ISSVA, 1996), vascular pathologies are classified into vascular tumors and malformations. Hemangiomas represent the most common vascular tumor in childhood, with an incidence of 4-5% [17]. In adults, hemangiomas account for only 0.4-0.6% of cases [4], with isolated instances occurring in the parotid gland.

The development of hemangiomas in adulthood may involve of endothelial cells alongside epithelioid cells featuring eosinophilic cytoplasm. Histologically, epithelioid hemangiomas are categorized into three subtypes: (1) conventional epithelioid hemangioma, (2) cellular epithelioid hemangioma, and (3) angiolymphoid hyperplasia with eosinophilia.

Angiolymphoid hyperplasia was first described by Wells and Whimster in 1969 [18], a finding consistent with our observation of two such cases [19]. Similar cases have been documented by Meningaud (2007) [20].

A consistent feature noted across studies is the involvement of the parotid gland by these vascular tumors. Periodic inflammatory episodes with alternating enlargement and reduction of the lesion may serve as a pathognomonic sign. In some instances, a well-defined capsule is evident both clinically and histologically. These tumors are often peripherally located within the gland, facilitating their excision within healthy tissue margins.

The pathogenesis of vascular dysplasias and tumors affecting the parotid glands remains unclear and warrants further investigation. Both localized and diffuse forms involving the entire gland have been described.

During embryonic and fetal development, vascular-neural and glandular tissues form at different stages, with salivary gland development occurring later. A similar pattern occurs

phylogenetically, as the parotid gland emerges exclusively in mammals [21, 22]. This developmental and phylogenetic disparity likely accounts for the distinct separation of vascular pathology from the glandular tissue [23-25].

When establishing the pathohistological diagnosis of vascular developmental anomalies in the parotid gland, the proposal by Roginski et al. (2011) should be considered, which classifies some childhood hemangiomas as vascular hyperplasias rather than tumors. This concept likely extends to adulthood, where hyperplastic lesions, rather than true tumors (hemangiomas), may develop within the parotid gland.

Consequently, we propose a classification of vascular pathologies in the head and neck region, specifically those involving the parotid gland, into three distinct entities: tumors (hemangiomas), vascular hyperplasias, and malformations.

This classification provides a framework for precise diagnosis, thereby warranting continued observation to elucidate the clinical behavior and optimal management of these distinct parotid gland lesions.

Conclusions

1. Vascular lesions of the parotid gland in adult patients are most frequently represented by epithelioid hemangiomas, which constitute angiolymphoid hyperplasia with eosinophilia.

2. In most cases of epithelioid hemangiomas of the parotid gland, tumor enucleation with preservation of the gland can be performed.

3. The peripheral localization of vascular anomalies in the parotid gland may be explained by the peculiarities of phylogenetic and ontogenetic development of the vascular network, as well as by the later integration of salivary gland tissue.

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ПРОБЛЕМИ СИСТЕМАТИЗАЦІЇ ТА ЛІКУВАННЯ ПАТОЛОГІЧНИХ УТВОРЕНЬ З КРОВОНОСНИХ СУДИН БІЛЯ ВУШНОЇ ЗАЛОЗИ У ДІТЕЙ ТА ДОРΟΣЛИХ: ОГЛЯД ЛІТЕРАТУРИ ТА КЛІНІЧНІ СПОСТЕРЕЖЕННЯ

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Резюме.

У статті розглянуті спірні питання систематизації та лікування пацієнтів із патологічними утвореннями кровоносних судин біля вушної залози. Пропонується доцільним поділяти усі судинні патологічні утворення на мальформації, гіперплазії та пухлини (гемангіоми). Вибір тактики лікування та діагностики слід ґрунтувати з урахуванням активності маркерів Glut1 та CD34.

Мета. Вивчити проблеми систематизації та лікування судинних уражень привушної слинної залози у дітей і дорослих, узагальнити сучасні літературні дані та проаналізувати власні клінічні спостереження з урахуванням імуногістохімічних маркерів (GLUT-1, CD34).

Матеріали та методи. Проведено огляд сучасних публікацій щодо класифікації, діагностики та лікування судинних патологій привушної залози. Проаналізовано три клінічні випадки пацієнтів віком від 33 до 57 років з судинними ураженнями привушної слинної залози. Використано клінічне обстеження, ультразвукове дослідження з доплерографією, магнітно-резонансну томографію, тонкогальмову аспіраційну біопсію, а також гістологічні та імуногістохімічні методи. При проведенні досліджень зберігалися рекомендовані принципи біоетики. Дослідження виконані у межах науково-дослідної роботи «Розробка методів хірургічного та комплексного лікування хворих з вродженою та набутою патологією щелепно-лицевої ділянки з відновленням функцій та з урахуванням супутньої патології», номер державної реєстрації 0123U100745, термін виконання – 2023-2028 рр.

Результати. У досліджених випадках виявлено різні форми судинних патологій: капілярна гемангіома, гемангіома у поєднанні з плеїоморфною аденомою та судинна гіперплазія. Імуногістохімічні дослідження підтвердили діагностику за допомогою маркерів GLUT-1 та CD34. Лікувальна тактика включала часткову резекцію привушної залози з видаленням пухлин, із застосуванням органозберігаючих підходів та інтраопераційного контролю цілісності лицевого нерву. Післяопераційний перебіг був без ускладнень.

Висновки. Судинні ураження привушної слинної залози у дітей найчастіше представлені гемангіомами з типовим перебігом, тоді як у дорослих переважають рідкісні варіанти – епітеліоїдні гемангіоми та судинні гіперплазії. Для правильної диференційної діагностики необхідно поєднувати клінічні дані, інструментальні методи візуалізації та імуногістохімічні маркери. Хірургічне лікування є методом вибору при ускладнених або прогресуючих формах патології.

Ключові слова: ротова порожнина; слинні залози; вроджені вади розвитку; гіперплазія; пухлини; патологічні ураження слинних залоз; імуногістохімічні маркери проліферації та апоптозу; хірургічні втручання на слинних залозах.

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