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MORPHOLOGICAL CHARACTERISTICS OF PURULENT-NECROTIC MAXILLOFACIAL LESIONS OF DIFFERENT ETIOLOGIES

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Abstract. Background. Purulent-necrotic lesions of the maxillofacial region are morphologically heterogeneous and may reflect a combination of suppurative inflammation, vascular injury, tissue ischemia, necrosis, and secondary reparative responses. Histopathological evaluation is particularly important when the process develops in association with polyangiitis, post-COVID conditions, or chronic renal failure, because the dominant pathogenetic mechanisms and diagnostic priorities differ.

Objective. To characterize the pathomorphological features of purulent-necrotic maxillofacial lesions of different etiologies and to systematize the histological variants identified in polyangiitis-associated cases.

Materials and methods. A comparative observational histomorphological study included surgical tissue specimens from 200 patients treated between 2020 and 2024: 60 with polyangiitis-associated lesions, 80 with post-COVID purulent-necrotic complications, and 60 with chronic renal failure.

Results. Macroscopically, the specimens were irregular, rough-surfaced, soft tissue fragments measuring approximately 1.0×0.5 to 3.0×2.5 cm. The common microscopic pattern included epithelial desquamation or loss, neutrophil-predominant purulent infiltration, cellular detritus, stromal edema, collagen fragmentation, vascular congestion and stasis, endothelial injury, fibrinoid change, microthrombosis, muscle-fiber necrosis, and, in prolonged cases, granulation tissue and fibrosis.

Conclusion. Purulent-necrotic maxillofacial lesions demonstrate a combined morphological spectrum of suppurative inflammation, ischemic-necrotic vascular injury, and secondary repair.

Keywords: maxillofacial region, purulent-necrotic process, morphology, histopathology, polyangiitis, post-COVID condition, chronic renal failure, vasculitis, osteonecrosis.

Annotatsiya. Dolzarbligi. Yuz-jag' sohasining yiringli-nekrotik zararlanishlari morfologik jihatdan turlicha bo'lib, yiringli yallig'lanish, qon-tomir shikastlanishi, to'qima ishemiyasi, nekroz va ikkilamchi reparativ reaksiyalarning birgalikda namoyon bo'lishi bilan tavsiflanadi. Patologik jarayon poliangiit, post-COVID holatlari yoki surunkali buyrak yetishmovchiligi fonida rivojlanganda, yetakchi patogenetik mexanizmlar va diagnostik mezonlar farqlanishi sababli gistopatologik tekshiruv alohida ahamiyat kasb etadi.

Tadqiqot maqsadi. Turli etiologiyali yuz-jag' sohasi yiringli-nekrotik zararlanishlarining patomorfologik xususiyatlarini tavsiflash va poliangiit bilan bog'liq holatlarda aniqlangan gistologik variantlarni tizimlashtirish.

Tadqiqot materiallari va usullari. 2020–2024-yillarda davolangan 200 nafar bemordan jarrohlik yo'li bilan olingan to'qima namunalari qiyosiy kuzatuv asosidagi gistomorfologik tadqiqotga kiritildi. Ulardan 60 nafarida poliangiit, 80 nafarida post-COVID holati va 60 nafarida surunkali buyrak yetishmovchiligi fonida yuzaga kelgan yiringli-nekrotik jarayonlar kuzatildi.

Natijalar. Makroskopik tekshiruvda o'lchami taxminan 1,0×0,5 sm dan 3,0×2,5 sm gacha bo'lgan, g'adir-budir va notekis yuzali, yumshoq konsistensiyali to'qima bo'laklari aniqlandi. Umumiy mikroskopik manzara epiteliy deskvamatsiyasi yoki yo'qolishi, neytrofillar ustun bo'lgan yiringli infiltratsiya, hujayraviy detrit, stromal shish, kollagen tolalarining fragmentatsiyasi, qon tomirlarning to'laqonliligi va stazi, endoteliy shikastlanishi, fibrinoid o'zgarishlar, mikrotrombozlar, mushak tolalari nekrozi, jarayon uzoq davom etganda esa granulyatsion to'qima va fibroz bilan tavsiflandi.

Xulosa. Yuz-jag' sohasining yiringli-nekrotik zararlanishlari yiringli yallig'lanish, ishemik-nekrotik qon-tomir shikastlanishi va ikkilamchi reparatsiyaning qo'shma morfologik spektri bilan tavsiflanadi.

Kalit so'zlar: yuz-jag' sohasi, yiringli-nekrotik jarayon, morfologiya, gistopatologiya, poliangiit, post-COVID holati, surunkali buyrak yetishmovchiligi, vaskulit, osteonekroz.

Аннотация. Актуальность. Гнойно-некротические поражения челюстно-лицевой области морфологически неоднородны и характеризуются сочетанием гнойного воспаления, сосудистого повреждения, ишемии тканей, некроза и вторичных репаративных реакций. Гистопатологическое исследование приобретает особое значение при развитии процесса на фоне полиангиита, постковидных состояний или хронической почечной недостаточности, поскольку ведущие патогенетические механизмы и диагностические приоритеты в этих случаях различаются.

Цель исследования. Охарактеризовать патоморфологические особенности гнойно-некротических поражений челюстно-лицевой области различной этиологии и систематизировать гистологические варианты, выявленные при полиангиит-ассоциированных состояниях.

Материалы и методы исследования. В сравнительное наблюдательное гистоморфологическое исследование включены операционные образцы тканей 200 пациентов, проходивших лечение в 2020–2024 гг.: 60 пациентов с полиангиит-ассоциированными поражениями, 80 — с постковидными гнойно-некротическими осложнениями и 60 — с хронической почечной недостаточностью.

Результаты. Макроскопически определялись фрагменты мягких тканей размером приблизительно от 1,0×0,5 до 3,0×2,5 см, с неровной бугристой поверхностью и мягкой консистенцией. Общая микроскопическая картина включала десквамацию или утрату эпителия, гнойную инфильтрацию с преобладанием нейтрофилов, клеточный детрит, отёк стромы, фрагментацию коллагеновых волокон, полнокровие и стаз сосудов, повреждение эндотелия, фибриноидные изменения, микротромбозы, некроз мышечных волокон, а при длительном течении — формирование грануляционной ткани и фиброза.

Заключение. Гнойно-некротические поражения челюстно-лицевой области характеризуются сочетанным морфологическим спектром гнойного воспаления, ишемически-некротического сосудистого повреждения и вторичной репарации.

Ключевые слова: челюстно-лицевая область, гнойно-некротический процесс, морфология, gistopatologiya, полиангиит, постковидное состояние, хроническая почечная недостаточность, васкулит, остеонекроз.

Relevance. Purulent-necrotic processes of the maxillofacial region are among the most severe conditions in maxillofacial surgery because interconnected fascial spaces, rich vascular networks, and proximity to the orbit, skull base, and cranial nerves facilitate rapid progression. The clinical appearance may underestimate deep tissue destruction, whereas histopathological examination directly demonstrates the balance between acute suppurative inflammation, vascular-mediated ischemia, tissue necrosis, and reparative responses. This information is

particularly important when the clinical phenotype is modified by systemic vasculitis, post-infectious vascular injury, immunosuppression, or chronic organ failure [1, 3].

In granulomatosis with polyangiitis and related ANCA-associated vasculitides, the classical pathological spectrum includes necrotizing inflammation, small-vessel vasculitis, fibrinoid necrosis, leukocytoclasia, thrombosis, and granulomatous reactions. In the oral and maxillofacial region, these features may coexist with secondary bacterial infection and extensive tissue destruction, complicating differential diagnosis. Recognition of a vasculitic component is clinically relevant because purely antimicrobial or surgical approaches cannot address the underlying immune-mediated vascular injury.

Post-COVID purulent-necrotic lesions of the maxillofacial region may also show marked necrosis, vascular thrombosis, and angioinvasive fungal infection, especially in patients with diabetes, corticosteroid exposure, or other immunocompromising conditions [4, 8]. Chronic renal failure, in turn, is associated with secondary immunodeficiency, chronic inflammation, endothelial dysfunction, impaired neutrophil activity, and reduced regenerative potential [9, 10]. Because these pathogenetic pathways can produce overlapping morphological signs, a structured histological assessment is necessary to identify the dominant injury pattern and to guide further microbiological, immunological, and radiological investigations.

Aim of the study. To characterize the macroscopic and microscopic features of purulent-necrotic maxillofacial lesions associated with polyangiitis, post-COVID conditions, and chronic renal failure, and to classify the principal histomorphological variants identified in polyangiitis-associated cases.

Materials and methods. A comparative observational histomorphological study was conducted using surgical tissue specimens obtained from 200 patients treated in the maxillofacial surgery department between 2020 and 2024. The mean age of the study cohort was 57.09 ± 4.6 years. According to the underlying condition, the patients were divided into three groups: group I included 60 patients with polyangiitis-associated purulent-necrotic lesions, group II comprised 80 patients with post-COVID purulent-necrotic complications, and group III consisted of 60 patients with chronic renal failure.

The following histological domains were assessed systematically: integrity and differentiation of the covering epithelium; type and density of inflammatory infiltration; degree of interstitial edema and stromal destruction; cellular detritus and tissue necrosis; vascular congestion, stasis, plasmatic impregnation, fibrinoid swelling or necrosis, endothelial injury, luminal narrowing, obliteration, and thrombosis; muscle-fiber dystrophy and necrosis; granulomatous reactions; and reparative changes represented by granulation tissue, fibroblast proliferation, collagen formation, fibrosis, sclerosis, and hyalinosis. Microbial colonies were recorded when identifiable in routine sections, but definitive etiological identification required microbiological or special-stain confirmation.

For the 60 polyangiitis-associated cases, the dominant histopathological pattern was used to classify lesions into six clinicomorphological variants: necrotic-polyangiitic, purulent-necrotic vasculitic, ischemic-necrotic, granulomatous-necrotic, chronic inflammatory-fibrotic, and mixed. Quantitative data are presented as absolute numbers and percentages. Because the source dataset contained complete morphotype frequencies only for the polyangiitis group, no inferential comparison of specific histological variants among all three etiological groups was performed.

Results and discussion. The etiological distribution of the 200 examined patients is shown in Table 1. Post-COVID complications accounted for the largest proportion of cases (40.0%), whereas polyangiitis-associated lesions and lesions developing in chronic renal failure each represented 30.0% of the cohort.

Table 1. Distribution of patients according to the underlying condition

No.	Underlying condition	Number of patients	%
1	Polyangiitis-associated purulent-necrotic lesions	60	30.0
2	Post-COVID purulent-necrotic complications	80	40.0
3	Purulent-necrotic lesions associated with chronic renal failure	60	30.0
	Total	200	100.0

Macroscopic characteristics. The surgical material usually consisted of irregular tissue fragments measuring approximately 1.0×0.5 cm to 3.0×2.5 cm. The surface was rough and uneven, and the specimens had a predominantly soft consistency. These macroscopic features reflected tissue disintegration, edema, inflammatory exudation, and necrosis. However, macroscopic examination alone did not permit reliable differentiation between vasculitic, infectious, ischemic, and reparative components.

General microscopic characteristics of affected soft tissues. The covering epithelium was frequently absent or preserved only as necrotic fragments. Where present, it demonstrated focal desquamation, thinning, dystrophic changes, and loss of normal stratification. Nuclear alterations included pyknosis, karyorrhexis, and karyolysis. In some specimens, the epithelial surface was replaced by fibrin-rich purulent exudate and amorphous detritus. In more chronic areas, reactive epithelial thickening, acanthosis, and papillomatosis were observed.

The connective-tissue stroma demonstrated pronounced edema, loosening and fragmentation of collagen fibers, and focal or diffuse matrix lysis. Necrotic regions contained amorphous eosinophilic material mixed with cellular debris. A demarcation zone composed of neutrophils, macrophages, lymphocytes, and plasma cells formed at the periphery of necrotic foci. The inflammatory infiltrate was predominantly purulent, with numerous neutrophils, including degenerative forms. Macrophage activation and phagocytosis were evident, and bacterial colonies of coccoid or bacillary morphology could be seen in some routine sections.

Microvascular injury was one of the most consistent components of the pathological picture. Vessels showed congestion, stasis, aggregation of formed blood elements, plasmatic impregnation of the wall, fibrinoid swelling, and, in severe cases, fibrinoid necrosis. Endothelial cells were swollen, focally desquamated, or destroyed. Small vessels and venules often contained thrombotic masses at different stages of organization. Luminal narrowing and partial or complete obliteration aggravated tissue hypoxia and promoted progression of necrosis.

Muscle tissue showed loss of cross-striation, homogenization of sarcoplasm, fragmentation, and necrosis of muscle fibers. Intermuscular spaces were markedly edematous and infiltrated by leukocytes. In prolonged or chronic lesions, reparative changes included capillary-rich granulation tissue, fibroblast proliferation, focal collagen deposition, fibrosis, sclerosis, and hyalinosis. Thus, the overall histological pattern represented a dynamic sequence in which acute suppurative destruction, ischemic vascular injury, and repair could coexist within the same specimen.

Table 2. Key histomorphological features and their diagnostic interpretation

Tissue component	Principal morphological findings	Diagnostic interpretation
Covering epithelium	Desquamation, thinning, dystrophy, necrosis, pyknosis, karyorrhexis, karyolysis; reactive acanthosis in chronic areas	Severity and duration of surface injury; distinction between acute destruction and chronic reactive change
Inflammatory infiltrate	Neutrophil predominance, degenerative neutrophils, macrophages, lymphocytes, plasma cells; demarcation around necrosis	Active suppurative inflammation with a variable chronic immune component
Connective-tissue stroma	Edema, collagen loosening and fragmentation, matrix lysis, amorphous eosinophilic necrotic material	Tissue destruction, hypoxia, and progression of necrosis
Microvasculature	Congestion, stasis, endothelial damage, fibrinoid swelling or necrosis, luminal narrowing, obliteration, microthrombosis	Ischemic-vascular component; supports vasculitic or angioinvasive mechanisms when correlated clinically
Muscle tissue	Loss of cross-striation, sarcoplasmic homogenization, fragmentation, necrosis, intermuscular edema	Extension of the process into deep fascial and masticatory compartments
Reparative component	Granulation tissue, neovascularization, fibroblast proliferation, collagen deposition, fibrosis, sclerosis, hyalinosis	Prolonged disease course, partial organization, and reduced tissue plasticity

Histomorphological variants in polyangiitis-associated lesions. The polyangiitis group demonstrated the most characteristic combination of vascular and inflammatory changes. The pathological process involved capillaries, arterioles, muscular arteries, venules, and, less frequently, larger veins. Destructive-productive panangiitis and thrombovasculitis affected all layers of the vascular wall. The intensity ranged from mucoid swelling to segmental or circumferential fibrinoid necrosis. Dense perivascular infiltrates and leukocytoclasia were accompanied by endothelial destruction, luminal narrowing, partial obliteration, stasis, and microthrombosis.

In some specimens, proliferative changes were expressed as intra- and perivascular granulomatous formations containing epithelioid cells, lymphocytes, plasma cells, neutrophils, eosinophils, fibroblasts, and multinucleated giant cells of Langhans or foreign-body type. Granulomas frequently coexisted with necrosis. In chronic or treated lesions, vascular walls became thickened because of sclerosis and hyalinosis, while endothelial and intimal proliferation caused recalibration and narrowing of the lumen.

Based on the dominant microscopic pattern, six clinicomorphological variants were distinguished (Table 3). The necrotic-polyangiitic and purulent-necrotic vasculitic variants predominated and together accounted for 32 of 60 cases (53.3%). This finding indicates that severe vascular injury was commonly accompanied by secondary purulent inflammation rather than occurring as an isolated process.

Table 3. Clinicomorphological variants of polyangiitis-associated purulent-necrotic lesions (n=60)

No.	Morphological variant	Principal diagnostic features	n (%)
1	Necrotic-polyangiitic	Fibrinoid necrosis of vascular walls, leukocytoclasia, dense perivascular infiltration	18 (30.0%)
2	Purulent-necrotic vasculitic	Neutrophil-rich infiltration, vascular-wall destruction, microabscess formation	14 (23.3%)
3	Ischemic-necrotic	Marked luminal narrowing, thrombosis, tissue ischemia, coagulative or mixed necrosis	10 (16.7%)
4	Granulomatous-necrotic	Epithelioid cells, multinucleated giant cells, necrotic granulomatous foci	8 (13.3%)
5	Chronic inflammatory-fibrotic	Lymphoplasmacytic infiltration, fibrosis, sclerosis, vascular-wall remodeling	6 (10.0%)
6	Mixed	Combination of vasculitis, necrosis, suppuration, and reparative changes	4 (6.7%)
	Total		60 (100%)

Note: Classification was based on the dominant histopathological pattern in each specimen.

Stage-related morphological changes. The severity and composition of the lesions varied with disease duration. Acute and subacute specimens were dominated by destructive-productive changes, neutrophil-rich infiltration, severe edema, vascular-wall injury, fibrinoid necrosis, stasis, and fresh thrombosis. In prolonged cases, the inflammatory infiltrate became more mixed, with increasing numbers of lymphocytes, plasma cells, macrophages, and fibroblasts. Granulation tissue and organization of thrombi were followed by collagen deposition, fibrosis, vascular sclerosis, and hyalinosis.

Table 4. Predominant histological features according to the phase of the pathological process

Phase	Predominant findings	Pathomorphological significance
Acute/destructive	Epithelial loss, neutrophil-rich exudate, edema, cellular detritus, vascular congestion, fresh thrombosis, extensive necrosis	Active suppurative and ischemic injury with a high risk of rapid anatomical spread
Subacute/destructive-productive	Mixed inflammatory infiltrate, endothelial and intimal proliferation, organization of thrombi, early granulation tissue	Transition from active destruction to partial organization; persistent vascular compromise
Chronic/reparative	Lymphoplasmacytic infiltration, fibrosis, sclerosis, hyalinosis, luminal narrowing or obliteration, reactive epithelial hyperplasia	Chronicity, reduced perfusion, impaired tissue remodeling, and risk of persistent functional defects

Etiology-oriented interpretation. In polyangiitis-associated lesions, the combination of fibrinoid vascular necrosis, leukocytoclasia, perivascular infiltration, thrombosis, and granulomatous-necrotic changes strongly supported an immune-mediated vascular component [1–3]. Nevertheless, the frequent purulent-necrotic variant demonstrated that secondary infection may substantially modify the classical vasculitic picture. Therefore, histology should not be interpreted in isolation from ANCA status, systemic manifestations, imaging, and microbiological findings.

In post-COVID lesions, extensive necrosis and thrombosis require particular attention to the possibility of angioinvasive fungal disease. Mucormycosis and aspergillosis may produce vascular invasion, thrombosis, ischemia, and necrosis of soft tissue and bone [4, 8]. However, no fungal-specific quantitative analysis or special-stain results were available in the present source dataset. Consequently, routine hematoxylin-and-eosin findings should be supplemented, when clinically indicated, by periodic acid–Schiff or Grocott methenamine silver staining, direct microscopy, culture, and molecular or microbiological methods.

In chronic renal failure, uremic immune dysfunction, chronic inflammation, endothelial abnormalities, impaired neutrophil function, and reduced tissue repair may intensify purulent inflammation and delay organization of necrotic foci [9, 10]. The coexistence of fibrosis, vascular remodeling, and persistent inflammation in prolonged lesions is compatible with impaired regenerative capacity, although specific quantitative subgroup comparisons require further investigation.

Conclusion. Purulent-necrotic lesions of the maxillofacial region were characterized by a complex combination of epithelial destruction, neutrophil-predominant suppurative inflammation, stromal edema and lysis, vascular injury, thrombosis, tissue ischemia, and necrosis. In prolonged cases, these changes were accompanied by granulation tissue, fibrosis, sclerosis, and hyalinosis.

Among polyangiitis-associated lesions, necrotic-polyangiitic and purulent-necrotic vasculitic variants predominated and together constituted 53.3% of cases. Fibrinoid necrosis of vascular walls, leukocytoclasia, perivascular inflammation, thrombosis, and granulomatous reactions were the principal morphological indicators of a vasculitic component.

Routine histology is essential for determining the dominant mechanism of tissue injury but should be supplemented by microbiological, fungal-specific, immunological, and radiological methods whenever infection, angioinvasion, or systemic vasculitis is suspected. Etiology-oriented interpretation of morphological findings can improve diagnostic accuracy, guide the extent of surgical treatment, and support timely multidisciplinary management.

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