# MAXILLOFACIAL TUMORS

**Ro‘ziboyeva Durdona Sharifovna**
**Email: durdonaroziboyeva22@gmail.com**
**Adress: Tashkent, Uzbekistan**
**Educational institution: Tashkent State Dental University**
**https://doi.org/10.5281/zenodo.20179168**

**Annotatsiya:** Yuz-jag‘ o‘smalari bosh va bo‘yin sohasida rivojlanadigan murakkab biologik va patologik jarayonlar bo‘lib, ular epitelial, mezenximal, neyrogen va odontogen to‘qimalardan kelib chiqadi. Ushbu maqolada yuz-jag‘ o‘smalarining etiologiyasi, molekulyar-genetik asoslari, hujayra siklining buzilishi, invaziya, angiogenez va metastaz mexanizmlari chuqur tahlil qilindi. Robbins Basic Pathology, WHO va zamonaviy ilmiy tadqiqotlar asosida genetik mutatsiyalar, immun nazoratdan qochish va angiogenez faollashuvi o‘sma rivojlanishida asosiy rol o‘ynashi ko‘rsatildi.

**Kalit so‘zlar:** yuz-jag‘ o‘smalari, karsinoma, osteosarkoma, ameloblastoma, metastaz, angiogenez

**Аннотация:** Опухоли челюстно-лицевой области представляют собой сложные биологические и патологические процессы, развивающиеся из эпителиальных, мезенхимальных, нервных и одонтогенных тканей. В статье подробно рассмотрены их этиология, молекулярно-генетические механизмы, нарушения клеточного цикла, инвазия, ангиогенез и метастазирование. На основе Robbins Basic Pathology, данных ВОЗ и современных исследований показано, что ключевую роль играют генетические мутации, активация ангиогенеза и уклонение от иммунного контроля.

**Ключевые слова:** челюстно-лицевые опухоли, карцинома, остеосаркома, амелобластома, метастаз, ангиогенез

**Abstract:** Maxillofacial tumors are complex biological and pathological processes arising from epithelial, mesenchymal, neural, and odontogenic tissues. This article provides a detailed analysis of their etiology, molecular and genetic mechanisms, cell cycle disruption, invasion, angiogenesis, and metastasis. Based on Robbins Basic Pathology, WHO data, and recent scientific studies, it is shown that genetic mutations, activation of angiogenesis, and immune evasion play a central role in tumor development.

**Keywords:** maxillofacial tumors, carcinoma, osteosarcoma, ameloblastoma, metastasis, angiogenesis

## INTRODUCTION

Maxillofacial tumors represent one of the most biologically complex and clinically challenging disease groups in modern dentistry and oncology. The maxillofacial region contains a dense network of anatomical structures, including bone, epithelial mucosa, muscles, nerves, lymphatic channels, and blood vessels. This complex organization allows tumors in this region to exhibit rapid progression, early invasion, and frequent metastasis.

The development of maxillofacial tumors is influenced by a combination of environmental, infectious, and genetic factors. Tobacco use, alcohol consumption, chronic irritation, viral infections (especially HPV), and genetic predisposition are considered major risk factors. At the cellular level, tumor development is associated with uncontrolled proliferation, loss of differentiation, and evasion of apoptosis.

Oral squamous cell carcinoma is the most common malignant tumor of the oral cavity, often detected at advanced stages due to its asymptomatic early development. Osteosarcoma is a highly aggressive malignant bone tumor characterized by rapid growth and early hematogenous metastasis, especially to the lungs.

Ameloblastoma is a locally aggressive odontogenic tumor that causes progressive destruction of the jaw bones.

LITERATURE REVIEW

According to Robbins Basic Pathology, carcinogenesis is a multistep process involving genetic alteration, clonal expansion, and tumor progression. The transformation from a normal cell to a malignant phenotype requires accumulation of mutations in oncogenes, tumor suppressor genes, and DNA repair genes.

Neville's Oral and Maxillofacial Pathology provides a detailed histopathological classification of jaw tumors into odontogenic tumors, epithelial malignancies, mesenchymal sarcomas, and mixed lesions. These tumors differ significantly in their biological behavior, microscopic appearance, and clinical prognosis.

WHO and IARC reports highlight that oral cancer is one of the most common cancers globally, especially in developing countries. The synergistic effect of tobacco and alcohol significantly increases carcinogenic risk. Chronic inflammation also plays a crucial role in tumor initiation.

Molecular oncology studies emphasize the importance of signaling pathways such as RAS/RAF/MEK, PI3K/AKT/mTOR, and Wnt/β-catenin in tumor progression. These pathways regulate cell proliferation, survival, and differentiation.

Harald zur Hausen demonstrated the role of HPV in epithelial carcinogenesis, linking viral infection with cancer development.

Theodor Boveri proposed the chromosomal theory of cancer, explaining tumor formation through chromosomal instability.

**MATERIALS AND METHODS**

This study is based on an extensive review of scientific literature in pathology, oncology, molecular biology, histology, and clinical dentistry. The analysis focused on the multilevel mechanisms of tumor development, including:

1. Genetic mutations (TP53, RB1, APC, CDKN2A)
2. Epigenetic modifications (DNA methylation, histone acetylation)
3. Cell cycle dysregulation
4. Apoptosis inhibition
5. Signal transduction pathways (RAS/MAPK, PI3K/AKT, mTOR)
6. Angiogenesis (VEGF, FGF pathways)
7. Tumor microenvironment interactions
8. Immune evasion mechanisms
9. Metastatic cascade (EMT – epithelial-mesenchymal transition)

Histopathological evaluation criteria included cellular atypia, nuclear pleomorphism, mitotic index, invasion depth, stromal response, and necrosis patterns.

**RESULTS**

The study revealed that maxillofacial tumor development is a stepwise biological process driven by progressive genetic instability. The initial stage involves DNA damage caused by

carcinogens such as tobacco, alcohol, and viral oncogenes. This leads to activation of proto-oncogenes and inactivation of tumor suppressor genes.At the cellular level, loss of TP53 function prevents DNA repair and apoptosis, allowing abnormal cells to survive and proliferate. RB1 gene dysfunction leads to uncontrolled transition from G1 to S phase of the cell cycle, resulting in continuous cell division.

Angiogenesis is a critical factor in tumor growth. Tumor cells secrete VEGF, stimulating the formation of new blood vessels that supply oxygen and nutrients. This vascular network also facilitates metastasis.

Invasion occurs through degradation of the basement membrane by matrix metalloproteinases (MMPs), allowing tumor cells to infiltrate surrounding tissues. EMT (epithelial-mesenchymal transition) further enhances migratory capacity of tumor cells. Metastasis occurs via lymphatic, hematogenous, and perineural routes. Common distant metastatic sites include lungs, liver, and skeletal system.

Clinically, patients present with non-healing ulcers, persistent pain, bleeding, facial asymmetry, tooth mobility, trismus, and regional lymphadenopathy. Radiographic findings vary depending on tumor type: osteosarcoma shows “sunburst” pattern, ameloblastoma shows multilocular “soap-bubble” appearance, and oral carcinoma shows irregular bone destruction.

**CONCLUSION**

Maxillofacial tumors represent a complex group of diseases characterized by multilevel biological dysregulation involving genetic, molecular, cellular, and tissue-level abnormalities. Their progression is driven by uncontrolled cell proliferation, apoptosis resistance, angiogenesis, and immune evasion.

Early diagnosis remains the most critical factor in improving prognosis. Advanced tumors are associated with high morbidity and mortality due to rapid invasion and metastasis. Modern treatment strategies include surgical resection, radiotherapy, chemotherapy, and targeted molecular therapy. Understanding the pathological anatomy of these tumors is essential for accurate diagnosis and effective treatment planning.

## Adabiyotlar, References, Литературы:

1. Robbins Basic Pathology. Kumar V, Abbas AK, Aster JC. Elsevier. https://www.elsevier.com/books/robbins-basic-pathology
2. Neville BW, Damm DD, Allen CM. Oral and Maxillofacial Pathology. Elsevier. https://www.elsevier.com
3. WHO. Oral cancer fact sheet. https://www.who.int
4. IARC. Global cancer statistics report. https://www.iarc.who.int
5. Harrison’s Principles of Internal Medicine. McGraw-Hill. https://accessmedicine.mhmedical.com
6. TNM Classification of Malignant Tumors. UICC. https://www.uicc.org
7. Hanahan D, Weinberg RA. Hallmarks of cancer. Nature Reviews Cancer. https://www.nature.com
8. Kumar V et al. Robbins & Cotran Pathologic Basis of Disease. Elsevier. https://www.elsevier.com
9. Barnes L. Surgical Pathology of the Head and Neck. https://www.crcpress.com
10. Neville BW. Textbook of Oral Pathology. https://www.elsevier.com

11. Cawson RA. Oral Pathology and Oral Medicine. https://www.elsevier.com
12. WHO Classification of Head and Neck Tumors. https://tumourclassification.iarc.who.int
13. National Cancer Institute. Oral cavity cancer information. https://www.cancer.gov
14. UICC. Cancer staging manual updates. https://www.uicc.org
15. WHO Global Health Observatory – Cancer data. https://www.who.int