

Swapan Kumar Purkait



# *Essentials of* **ORAL PATHOLOGY**



**4**<sup>th</sup> Edition

*Forewords*  
**RR Paul  
Jay Gopal Ray  
Tamal Kanti Pal**



---

# CONTENTS

---

## 1. Developmental Anomalies of Oral and Paraoral Structures

1

### **Anomalies of Lips and Palate 2**

- ❑ Lip Pits and Fistulas 2    ❑ Double Lip 2    ❑ Frenal Tag 3
- ❑ Hereditary Intestinal Polyposis Syndrome 3    ❑ Oral Melanotic Macule 4
- ❑ Uvula Elongata 5    ❑ Cheilitis Glandularis 5    ❑ Cheilitis Granulomatosa 7

### **Anomalies of the Oral Mucosa 8**

- ❑ Fordyce's Granules 8    ❑ Focal Epithelial Hyperplasia 9    ❑ White Sponge Nevus 10

### **Developmental Defects of the Gingivae 11**

- ❑ Fibromatosis Gingivae 11    ❑ Retrocuspid Papilla 12

### **Developmental Anomalies Involving the Jawbone 12**

- ❑ Agnathia 12    ❑ Micrognathia 12    ❑ Macrognathia 13    ❑ Facial Hemihypertrophy 14
- ❑ Facial Hemiatrophy 15    ❑ Torus in the Jaws 16    ❑ Orofacial Clefts 16
- ❑ Cleft Lip and Cleft Palate 16    ❑ Cleft in Lower Lip and Mandible 21

### **Developmental Anomalies of the Tongue 21**

- ❑ Aglossia 21    ❑ Microglossia 22    ❑ Macroglossia 22    ❑ Ankyloglossia 24
- ❑ Cleft Tongue 24    ❑ Fissured Tongue 25
- ❑ Median Rhomboid Glossitis or Glossal Central Papillary Atrophy 25    ❑ Lingual Varices 27
- ❑ Geographic Tongue or Benign Migratory Glossitis 27    ❑ Hairy Tongue 29
- ❑ Lingual Thyroid Nodule 29    ❑ Thyroglossal Tract Cyst 30

### **Anomalies of Oral Lymphoid Tissue 31**

- ❑ Reactive Lymphoid Aggregates 31    ❑ Lymphoepithelial Cyst 31
- ❑ Angiolymphoid Hyperplasia with Eosinophilia 32

### **Developmental Anomalies Involving Oral Hard Tissues 32**

- ❑ Abnormalities of Teeth 32

### **Disturbance in Size of Teeth 32**

- ❑ Microdontia 32    ❑ Macrodontia 34

### **Disturbance in Number of Teeth 35**

- ❑ Anodontia 35    ❑ Supernumerary Teeth 36

### **Disturbances in Eruption of Teeth 38**

- ❑ Types of Eruption Abnormalities 39    ❑ Premature Eruption 39
- ❑ Delayed Eruption 39    ❑ Impacted Teeth 40

### **Disturbances in the Shape of Teeth 41**

- ❑ Gemination 41    ❑ Fusion 41    ❑ Concrescence 43    ❑ Dilaceration 44
- ❑ Taurodontism 45    ❑ Dens in Dente 46    ❑ Dens Evaginatus 48    ❑ Talon Cusp 48
- ❑ Enamel Pearl 49

### **Disturbance in the Structure of Teeth 50**

- ❑ Disturbance in the Structure of Enamel 50

### **Acquired Disturbances of Enamel 50**

- ❑ Focal Enamel Hypoplasia 50    ❑ Idiopathic Enamel Opacities 50
- ❑ Generalized Enamel Hypoplasia 51

### **Effect of Individual Systemic Conditions on Enamel Hypoplasia 51**

- ❑ Nutritional Deficiency 51    ❑ Congenital Syphilis 51    ❑ Hypocalcemia 52
- ❑ Exanthematous Disease 52    ❑ Birth Injuries and Low Birthweight 52
- ❑ Fluorides and Mottling 52

**Hereditary Disturbance of Enamel Formation 53**

- ❑ Amelogenesis Imperfecta 53    ❑ Syndrome Associated Enamel Defects 55

**Disturbances in Structure of Dentin 56**

- ❑ Dentinogenesis Imperfecta 56
- ❑ Dentinal Abnormality due to Systemic or Environmental Disturbances 60
- ❑ Dentin Dysplasia 60    ❑ Regional Odontodysplasia 62

**Disturbance in Structure of Cementum 63**

- ❑ Hypercementosis 63    ❑ Hypocementosis 64

**2. Benign and Malignant Neoplasms of the Oral Cavity****67****Neoplasm 67**

- ❑ Definition 67    ❑ Local Invasion 67    ❑ Metastasis 68
- ❑ Classification of Oral Neoplasms 68
- ❑ Benign Neoplasms of the Epithelial Tissue Origin 71
- ❑ Malignant Neoplasms of the Epithelial Tissue Origin 77
- ❑ Clinical Staging of Carcinomas of Head and Neck 101
- ❑ Second Primary Tumors in Squamous Cell Carcinoma 102
- ❑ Screening Tests for Squamous Cell Carcinoma 103
- ❑ Special Investigations in Oral Cancer 103    ❑ Prevention of Oral Cancer 104

**Basal Cell Carcinoma 104**

- ❑ Definition 104    ❑ Origin 104    ❑ Etiology 104    ❑ Clinical Features 104
- ❑ Presentation 105    ❑ Histopathology 105    ❑ Differential Diagnosis 106
- ❑ Treatment 106

**Verrucous Carcinoma 106**

- ❑ Definition 106    ❑ Etiology 106    ❑ Clinical Features 106    ❑ Presentation 106
- ❑ Histopathology 107    ❑ Differential Diagnosis 109    ❑ Treatment 109

**Malignant Melanoma 109**

- ❑ Definition 109    ❑ Clinical Features 109    ❑ Clinical Types of Melanoma 109
- ❑ Presentation 110    ❑ Histopathology 111    ❑ Differential Diagnosis 112
- ❑ Treatment 112

**Primary Intra-alveolar Carcinoma 112**

- ❑ Definition 112    ❑ Origin 112

**Neoplasms of Mesenchymal Tissue Origin: Benign Neoplasms of Fibrous****Connective Tissue 112**

- ❑ Fibroma 112    ❑ Desmoplastic Fibroma 114    ❑ Giant Cell Fibroma 115
- ❑ Myofibroma 115    ❑ Peripheral Ossifying Fibroma 116
- ❑ Central Ossifying Fibroma 117

**Giant Cells 120**

- ❑ Peripheral Giant Cell Granuloma 120    ❑ Central Giant Cell Granuloma 123
- ❑ Benign Fibrous Histiocytoma 126    ❑ Myxoma 127    ❑ Nodular Fasciitis 128

**Benign Neoplasm of Adipose Tissue Origin 129**

- ❑ Lipoma 129

**Benign Neoplasm of Vascular Tissue Origin 131**

- ❑ Hemangioma 131

**Benign Neoplasm of Lymphatic Vessels 136**

- ❑ Lymphangioma 136    ❑ Cystic Hygroma 137

**Benign Neoplasm of Bone 138**

- ❑ Osteoma 138    ❑ Osteoid Osteoma/Osteoblastoma 140

**Benign Neoplasm of Cartilage Tissue 141**

- ❑ Chondroma 141    ❑ Benign Chondroblastoma 142

**Benign Neoplasm of Smooth Muscles 143**

- Leiomyoma 143

**Benign Neoplasm of Striated Muscle 114**

- Rhabdomyoma 144 □ Granular Cell Tumor/Granular Cell Myoblastoma 145

**Benign Neoplasms of Neural Tissue 146**

- Neurilemmoma 146 □ Neurofibroma 149
- Melanotic Neuroectodermal Tumor of Infancy 151 □ Traumatic Neuroma 152

**Neoplasm of the Mixed Tissue 153**

- Teratoma 153

**Malignant Neoplasms of Mesenchymal Tissue 154**

- Fibrosarcoma 154 □ Malignant Fibrous Histiocytoma 156 □ Liposarcoma 157
- Hemangioendothelioma 158 □ Hemangiopericytoma 159 □ Kaposi's Sarcoma 160
- Ewing's Sarcoma/Ewing's Tumor 162 □ Staging System for Bone Cancer  
Including Ewing Tumor: By American Joint Committee on Cancer 164
- Stages 164 □ Chondrosarcoma 165 □ Osteosarcoma 169
- Lymphomas 173 □ Multiple Myeloma 183 □ Solitary Plasmacytoma 187
- Leiomyosarcoma 187 □ Rhabdomyosarcoma 188 □ Neurogenic Sarcoma 189
- Metastatic Tumors of the Jaws 190

**3. Oral Precancerous Lesions and Conditions****197**

- Precancerous Lesion 197 □ Precancerous Condition 197 □ Leukoplakia 197
- Tobacco Pouch Keratosis 208 □ Oral Hairy Leukoplakia 208 □ Leukoedema 209
- Carcinoma in Situ 210 □ Erythroplakia 211 □ Stomatitis Nicotina 213
- Oral Submucous Fibrosis 214 □ Sideropenic Dysphagia 220 □ Lichen Planus 220
- Lichenoid Reactions 227

**4. Diseases of the Salivary Glands****229**

- Classification of Salivary Gland Diseases 230
- Developmental Anomalies of the Salivary Gland 230
- Reactive Lesions of the Salivary Gland 232 □ Infective Lesions 237
- Immune-mediated Disease 238 □ Miscellaneous Disorders of Salivary Gland 242
- Neoplasm of the Salivary Glands 244 □ Malignant Salivary Gland Neoplasms 254
- Histopathology 256 □ TNM Classification of Carcinomas of the Salivary Glands 264

**5. Odontogenic Neoplasms****268**

- Definition 268 □ Formation of Dental Papilla 268
- Dental Follicle 269 □ Formation of Root 269 □ Neoplasms of Debatable Origin 269

**Ameloblastoma 269**

- Definition 269 □ Etiology 270 □ Histogenesis of Ameloblastoma 270
- Clinical Features □ Clinical Presentation 270 □ Radiological Features 272
- Differential Diagnosis 272 □ Macroscopic Features 273
- Histopathological Features 273 □ Plexiform Ameloblastoma 273
- Follicular Ameloblastoma 273 □ Other Histological Patterns of Ameloblastoma 274

**Unicystic Ameloblastoma 277**

- Origin 277 □ Clinical Presentation 277 □ Radiographic Finding 277
- Histopathology 277 □ Differential Diagnosis 279

**Adenomatoid Odontogenic Tumor 279**

- Definition 279 □ Origin 279 □ Clinical Features 279 □ Clinical Presentation 280
- Radiological Features 280 □ Macroscopic Features 281 □ Differential Diagnosis 281
- Histopathological Features 281

**Calcifying Epithelial Odontogenic Tumor/Pindborg's Tumor 282**

- ❑ Origin 283    ❑ Clinical Features 283    ❑ Clinical Presentation 283
- ❑ Radiological Features 284    ❑ Differential Diagnosis 284
- ❑ Histopathological Features 284

**Dentinogenic Ghost-cell Tumor 285****Squamous Odontogenic Tumor/Benign Epithelial Odontogenic Tumor 286**

- ❑ Definition 286    ❑ Origin 286    ❑ Clinical Features 286    ❑ Presentation 286
- ❑ Radiographic Features 286    ❑ Histopathology 287    ❑ Differential Diagnosis 288

**Ameloblastic Fibroma 288**

- ❑ Clinical Features 288    ❑ Clinical Presentation 288    ❑ Radiological Features 288
- ❑ Histopathological Features 288

**Ameloblastic Fibro-odontome 289**

- ❑ Clinical Features 289    ❑ Clinical Presentation 289    ❑ Radiological Appearance 290
- ❑ Histopathology 290    ❑ Treatment 290

**Odontomes 290**

- ❑ Definition 290    ❑ Types 290    ❑ Clinical Features 290    ❑ Clinical Presentation 290
- ❑ Radiological Features 291    ❑ Differential Diagnosis 291    ❑ Histopathology 293
- ❑ Treatment 293

**Odontogenic Fibroma 293**

- ❑ Definition 293

**Peripheral Odontogenic Fibroma 293**

- ❑ Definition 293    ❑ Origin 293    ❑ Clinical Features 293    ❑ Histopathology 293
- ❑ Differential Diagnosis 293    ❑ Treatment 293

**Central Odontogenic Fibroma 294**

- ❑ Definition 294    ❑ Clinical Features 294    ❑ Radiographic Features 294
- ❑ Histological Presentation 294    ❑ Differential Diagnosis 295    ❑ Treatment 295

**Odontogenic Myxoma 295**

- ❑ Definition 295    ❑ Origin 295    ❑ Clinical Features 295    ❑ Presentation 295
- ❑ Radiographic Features 295    ❑ Macroscopic Appearance 296
- ❑ Histological Presentation 296    ❑ Differential Diagnosis 296    ❑ Treatment 296

**Periapical Cemental Dysplasia 296**

- ❑ Clinical Features 296    ❑ Clinical Presentations 296    ❑ Radiological Features 296
- ❑ Histopathology 297    ❑ Differential Diagnosis 297    ❑ Treatment 297

**Central Cementifying Fibroma 297****Familial Gigantiform Cementoma 297**

- ❑ Definition 297    ❑ Origin 298    ❑ Clinical Features 298    ❑ Radiographic Features 298
- ❑ Histological Presentation 298    ❑ Differential Diagnosis 298    ❑ Treatment 299

**Cementoblastoma 299**

- ❑ Clinical Features 299    ❑ Origin 299    ❑ Clinical Presentation 299
- ❑ Radiological Features 299    ❑ Macroscopy 300    ❑ Histopathology 300
- ❑ Differential Diagnosis 300    ❑ Treatment 300

**Malignant Odontogenic Neoplasms 300**

- ❑ Malignant Ameloblastoma 300    ❑ Ameloblastic Carcinoma 300
- ❑ Odontogenic Carcinoma 301    ❑ Clinical Features 301    ❑ Radiographic Features 301
- ❑ Histopathology 302    ❑ Treatment 302    ❑ Odontogenic Sarcomas 302
- ❑ Clear Cell Odontogenic Carcinoma 302    ❑ Clinical Features 302
- ❑ Clinical Presentation 302    ❑ Radiological Finding 302    ❑ Histological Presentation 302
- ❑ Treatment 302    ❑ Primary Intra-alveolar Carcinoma 302



## 6. Cysts of the Oral Region

306

- Definition 306   □ Classification of Cysts 306

### Odontogenic Cysts 308

- Odontogenic Keratocyst/Primordial Cyst 308   □ Dentigerous Cyst 318
- Radicular Cyst 324   □ Eruption Cyst 331   □ Lateral Periodontal Cyst 332
- Dental Lamina Cyst (Gingival Cyst) of the Newborn 333   □ Gingival Cysts of the Adult 334
- Sialo-Odontogenic Cysts/Glandular Odontogenic Cyst 335
- Botryoid Odontogenic Cysts 336
- Calcifying Epithelial Odontogenic Cyst/Gorlin Cyst 337   □ Paradental Cyst 340

### Nonodontogenic Cysts 340

- Globulomaxillary Cyst 340   □ Nasolabial Cyst/Nasoalveolar Cyst 341
- Nasopalatine Duct Cyst 342   □ Solitary Bone Cyst 344   □ Stafne's Bone Cyst 345
- Treatment 346   □ Aneurysmal Bone Cyst 346   □ Cyst of the Salivary Gland 347
- Ranula 350   □ Dermoid Cyst 351   □ Surgical Ciliated Cyst of Maxilla 352

## 7. Regressive Alterations of Teeth

355

### Attrition of Teeth 355

- Definition 355   □ Types of Attrition 355   □ Causes of Pathological Attrition 356
- Clinical Features of Attrition 356   □ Treatment 357

### Abrasion of Teeth 357

- Definition 357   □ Etiology and Pathogenesis 357   □ Causes of Abrasion 357
- Treatment 359

### Tooth Abfraction 359

- Definition   □ Forces Causing Abfraction 359   □ Clinical Features 359

### Erosion of Teeth 359

- Definition 359   □ Etiologic Factors for Erosion 360   □ Clinical Features of Erosion 360
- Treatment 361

### Resorption of Teeth 361

- Definition 361   □ Types of Root Resorption of Teeth 361

### Pulp Calcification 365

- Definition 365   □ Pathogenesis of Pulp Calcification 365   □ Types 366
- Types of Pulp Stones 366   □ Clinical Symptoms 366   □ Diagnosis 366
- Clinical Significance of Pulp Calcifications 367

### Hypercementosis 367

- Definition 367   □ Etiology of Hypercementosis 367   □ Clinical Features 367
- Radiographic Features 367   □ Microscopy 367   □ Clinical Significance 367

### Age Changes in Teeth 368

- Changes in Enamel 368   □ Changes in Dentin 368   □ Changes in Cementum 368
- Changes in Pulp 368

### Cementicles 368

- Pathogenesis 368

## 8. Bacterial, Viral and Fungal Infections

370

### Specific Bacterial Infections 370

- Tuberculosis 370   □ Syphilis 373   □ Gonorrhea 378   □ Actinomycosis 379
- Streptococcal Infections 381   □ Diphtheria 382   □ Sarcoidosis 383
- Leprosy/Hansen Disease 384   □ Tetanus 385   □ Midline Lethal Granuloma 387
- Wegener's Granulomatosis 387   □ Noma 387   □ Pyogenic Granuloma 388

### Viral Infections 390

- Acquired Immunodeficiency Syndrome 390   □ Herpes Virus Infections 398
- Herpes Simplex Virus Type-I Infections 398   □ Herpes Simplex Virus Type-II Infections 401

- Herpes Zoster 401   □ Cytomegalovirus Infection 404
- Epstein-Barr Virus Infections 405   □ Human Papillomavirus Infection 405
- Paramyxovirus Infection 406   □ Coxsackie Virus Infections 407

### **Fungal Infection 413**

- Candidiasis 413   □ Deep Fungal Infections 417   □ Oral Myiasis 420

## **9. Dental Caries**

426

- Definition of Dental Caries 426   □ Epidemiology of Dental Caries 426
- Pathophysiology of Dental Caries 427   □ Contributing Factors in Dental Caries 434
- Clinical Aspects of Dental Caries 437   □ Histopathological Aspect of Dental Caries 441
- Protective Responses of Dentin and Pulp Against Caries 444   □ Caries Activity Tests 444
- Methods of Caries Prevention 446   □ Caries Vaccine 446

## **10. Disease of Dentin-pulp Complex and Periapical Tissues**

450

### **Pulpal Diseases 450**

- Introduction 450   □ Dentin-pulp Complex 450   □ Etiology of Pulpal Diseases 451
- Classification of the Pulpal Diseases 451   □ Diagnosis of Pulpal Diseases 457

### **Diseases of the Periapical Tissues 458**

- Primary Acute Apical Periodontitis 458   □ Periapical Granuloma 458
- Acute Exacerbation of Chronic Periapical Granuloma/Abscess 460
- Periapical Abscess 460   □ Osteomyelitis 463   □ Endodontic-Periodontic Lesions 474

## **11. Spread of the Oral Infection**

477

- Important Factors for Odontogenic Infections 477

### **Space Infections 477**

- Space Infections Related to Maxilla 478   □ Space Infections Related to Mandible 479

### **Cellulitis 483**

- Definition 483   □ Pathogenesis 483   □ Clinical Features 483
- Histopathologic Features 485   □ Treatment 485

### **Ludwig's Angina 485**

- Definition 485   □ Causative Microorganisms 485   □ Pathogenesis of Ludwig's Angina 485
- Clinical Features 485   □ Diagnosis 486

### **Cavernous Sinus Thrombosis 486**

- Definition 486   □ Routes of Spread of Infections 486   □ Clinical Features 487
- Treatment 487

### **Maxillary Sinusitis 487**

- Definition 487   □ Etiology 487   □ Clinical Features 487   □ Radiological Features 487
- Histological Features 488   □ Treatment 488

### **Focal Infection 488**

- Definition 488   □ Mechanism of Focal Infection 488
- Common Consequences of "Focal Infections" from the Orofacial Region 488

## **12. Physical and Chemical Injuries of the Oral Cavity**

491

### **Physical Injuries 491**

- Fractures of Teeth 491   □ Root Fracture 491   □ Cemental Tear 491
- Bruxism 491   □ Ankylosis of Teeth 493   □ Submerged Teeth 493
- Toothbrush Injury 494   □ Toothpick Injury 494   □ Linea Alba 494
- Traumatic Atrophic Glossitis 494   □ Traumatic Ulcer 495   □ Factitious Injuries 495
- Denture Related Injuries or Lesions 496   □ Electrical Burns in the Mouth 497
- Thermal Burns in Mouth 497   □ Radiation Injuries 498   □ Osteoradionecrosis 502
- Laser Radiation 503

**Chemical Injuries 503**

- ❑ Congenital Porphyria 503    ❑ Biliary Atresia 503    ❑ Erythroblastosis Fetalis 504
- ❑ Fluorosis 504    ❑ Oral Manifestations of Various Metal Poisoning 504
- ❑ Oral Manifestations of Cytotoxic Drug Therapy 504
- ❑ Oral Manifestations of Tetracycline Staining 505    ❑ Angioneurotic Edema 506
- ❑ Chemical Burns 506    ❑ Chemical Burns due to Other Medicaments 506

**13. Biopsy and Healing of Oral Wounds****509****Biopsy 509**

- ❑ Definition 509    ❑ Indications of Biopsy 509    ❑ Contraindications of Biopsy 510
- ❑ Types of Biopsy 510    ❑ Procedure of Tissue Biopsy 514    ❑ Labelling of Specimen 515
- ❑ Biopsy Report 515

**Healing of Oral Wounds 516**

- ❑ Healing of Biopsy Wound 518    ❑ Healing of Gingivectomy Wound 518
- ❑ Healing of the Extraction Wound 519    ❑ Healing of the Fractured Jawbone 521
- ❑ Healing around Osteointegrated Implants 523

**14. Oral Aspects of Metabolic Disorders****526****Disturbances in Mineral Metabolism 526**

- ❑ Calcium 526    ❑ Phosphorus 528    ❑ Iron 528    ❑ Zinc 529

**Disturbance in Vitamin Metabolism 529**

- ❑ Vitamin D 529    ❑ Vitamin A 531    ❑ Vitamin E 531    ❑ Vitamin B Complex 531
- ❑ Vitamin C 532    ❑ Vitamin K 533

**Disturbances in Protein Metabolism 533**

- ❑ Amyloidosis 533    ❑ Porphyria 534

**Disturbances in Carbohydrate Metabolism 535**

- ❑ Hurler's Syndrome 535

**Disturbances in Lipid Metabolism 535**

- ❑ Hand-Schuller-Christian Disease 535    ❑ Eosinophilic Granuloma 536
- ❑ Letterer-Siwe Disease 536    ❑ Gaucher's Disease 537    ❑ Niemann-Pick Disease 537

**Disturbances in Hormone Metabolism 537**

- ❑ Hypopituitarism 537    ❑ Pituitary Insufficiency in Adults 537
- ❑ Diabetes Insipidus 538    ❑ Hyperpituitarism 538    ❑ Pituitary Gigantism 538
- ❑ Acromegaly 539    ❑ Hypothyroidism 539    ❑ Cretinism 540    ❑ Myxedema 540
- ❑ Hyperthyroidism 541    ❑ Hyperparathyroidism 541    ❑ Hypoparathyroidism 543
- ❑ Adrenal Hormones 544    ❑ Waterhouse-Friderichsen Syndrome 545
- ❑ Chronic Adrenocortical Insufficiency 545    ❑ Pancreatic Hormone 547
- ❑ Hypoglycemia 549    ❑ Progeria 549    ❑ Imbalance of Sex Hormones 549

**15. Diseases of Bone****552**

- ❑ Basic Structure and Function of Bone 552
- ❑ Paget's Disease of Bone 553    ❑ Fibrous Dysplasia of Bone 558
- ❑ Cherubism/Familial Fibrous Dysplasia or Disseminated Juvenile Fibrous Dysplasia 563
- ❑ Osteogenesis Imperfecta 566    ❑ Cleidocranial Dysplasia 568
- ❑ Osteopetrosis/Marble Bone Disease/Albers-Schönberg Disease 570
- ❑ Marfan Syndrome 571    ❑ Down Syndrome 572    ❑ Infantile Cortical Hyperostosis 573
- ❑ Mandibulofacial Dysostosis 574    ❑ Achondroplasia 574    ❑ Massive Osteolysis 575

**16. Diseases of Temporomandibular Joint****578**

- ❑ Developmental Disorders 578    ❑ Traumatic Disorders 578
- ❑ Inflammatory Disorders 581



## 17. Oral Aspects of Hematological Disorders 586

- ❑ Pernicious Anemia 586    ❑ Iron Deficiency Anemia 588    ❑ Aplastic Anemia 589
- ❑ Hemolytic Anemia 589    ❑ Thalassemias 590    ❑ Sickle Cell Anemia 592
- ❑ Erythroblastosis Fetalis 596    ❑ Polycythemia Vera 593    ❑ Leukemias 594
- ❑ Agranulocytosis 596    ❑ Cyclic Neutropenia 597    ❑ Purpura 598
- ❑ Hemophilia or Royal Disease 600

## 18. Periodontal Diseases 606

- ❑ Epidemiology 606    ❑ Pathogenesis of Periodontal Disease 610
- ❑ Clinical Features of Periodontitis 612    ❑ Gingival Hyperplasia 614
- ❑ Desquamative Gingivitis 617
- ❑ Acute Necrotizing Ulcerative Gingivitis/Vincent's Disease/Trench Mouth 617
- ❑ Lateral Periodontal Abscess 619    ❑ Pericoronitis 620    ❑ Staining of Teeth 620

## 19. Oral Aspects of Dermatological Disorders 623

- ❑ Hereditary Ectodermal Dysplasia 623    ❑ Psoriasis 625    ❑ Pityriasis Rosea 626
- ❑ Histopathology 626    ❑ Incontinentia Pigmenti 626    ❑ Erythema Multiforme 627
- ❑ Dermatitis Herpetiformis 630    ❑ Keratosis Follicularis 630    ❑ Acanthosis Nigricans 631
- ❑ Dyskeratosis Congenita 631    ❑ White Sponge Nevus 632    ❑ Polymyositis 633
- ❑ Autoimmunity 633    ❑ Pemphigus 635    ❑ Pemphigoid 641    ❑ Epidermolysis Bullosa 644
- ❑ Lupus Erythematosus 646    ❑ Scleroderma 651    ❑ Ehlers–Danlos Syndrome 654

## 20. Diseases of the Nerves and Muscles 658

### Diseases of the Nerves 658

- ❑ Trigeminal Neuralgia 658    ❑ Sphenopalatine Neuralgia 660
- ❑ Glossodynia and Glossopyrosis 661    ❑ Auriculotemporal Syndrome 661
- ❑ Glossopharyngeal Neuralgia 662    ❑ Bell's Palsy 662    ❑ Causalgia 663
- ❑ Eagle's Syndrome 664

### Disease of the Muscles 664

- ❑ Generalized Familial Muscular Dystrophy 664    ❑ Myasthenia Gravis 665
- ❑ Myositis Ossificans 665

## 21. Oral Manifestations of Generalized Diseases 668

- ❑ Vitamin Deficiencies 668    ❑ Important Causes of Lymphadenopathy 668
- ❑ Blood Dyscrasias 669    ❑ Metabolic Disorders 670    ❑ Heavy Metal Poisoning 670
- ❑ Endocrine Disturbances 671    ❑ Granulomatous Diseases 672
- ❑ Dermatological Diseases 672    ❑ Bone Diseases 673
- ❑ Acute Lethal Type Infectious Diseases 673    ❑ Helminthic Diseases 673
- ❑ Renal Diseases 674    ❑ Neural Diseases 674    ❑ Sexually Transmitted Diseases 674
- ❑ Cardiovascular Diseases 675    ❑ Genetic Disorders 675    ❑ Allergic Conditions 675
- ❑ General Manifestations of Oral Diseases 675

## 22. Syndromes Related to Oral Diseases 677

- ❑ Definition of Syndrome 677

## 23. Important Classifications of Oral Diseases 688

- ❑ White Lesions of the Oral Cavity 688    ❑ Red-Blue Lesions of the Oral Cavity 688
- ❑ Pigmented Lesions of the Oral Cavity 689    ❑ Classification of Vesiculobullous Diseases 689
- ❑ Classification of Ulcerative Conditions 690    ❑ Classification of Discoloration of Tooth 690
- ❑ Classification of Giant Cell Lesions 690

- ❑ Classification of Verrucal-Papillary Lesions of Oral Cavity 691
- ❑ Classification of Fibro-osseous Lesions 691    ❑ Classification of Vascular Tissue Diseases 691
- ❑ Classification of Diseases of the Hemopoietic Tissues and Lymphoreticular System 691
- ❑ Classification of Stomatitis 691
- ❑ Classification of Severe Infections of the Orofacial Tissues 692
- ❑ Classification of Chronic Orofacial Pain 692    ❑ Classification of Diseases of Tongue 692
- ❑ Classification of Gingival Enlargements 693    ❑ Classification of Taste Disorders 693
- ❑ Classification of Neck Swellings 693
- ❑ Classification of Yellow Conditions of Oral Mucosa 694
- ❑ Anatomic Radiolucencies of Jaw Bones 694
- ❑ Radiolucent Lesions of the Periapical Region 694
- ❑ Classification of Pericoronal Radiolucent Lesions 694
- ❑ Classification of Interradicular Radiolucent Lesions 694
- ❑ Classification of Multilocular Radiolucent Lesions of the Jaws 695
- ❑ Mixed Radiolucent-Radiopaque Lesions Associated with Teeth 695
- ❑ Mixed Radiolucent-Radiopaque Lesions not Necessarily Associated with Teeth 695
- ❑ Multiple Separate Radiopaque Lesions of the Jaws 695
- ❑ Generalized Radiopacities of the Jaws 695
- ❑ Classification of Causes of Trismus 696
- ❑ Classification of Hamartomatous Lesions of Oral and Maxillofacial Region 696
- ❑ Classification of Oral Granular Cell Lesions Including Odontogenic and Nonodontogenic Tumors 696
- ❑ Classification of Granulomatous Diseases 697

# Benign and Malignant Neoplasms of the Oral Cavity

## NEOPLASM (TUMOR)

### ■ DEFINITION

A neoplasm is an abnormal mass of tissue, the growth of which exceeds and is incoordinated with that of the surrounding normal tissues and persists in the same excessive manner after cessation of the stimuli that evoked the change (Rupert Willis 1950).

Important characteristics of a neoplasm:

- Abnormal growth—due to multiplication of cells that cannot be controlled
- Ceaseless—growth of a neoplasm never ends
- Purposeless—it is an unnecessary mass of cells
- Uncoordinated—growth pattern of tissue/cells is far in excess than that of the surrounding normal tissue
- Cessation of stimuli—a neoplasm continues to grow even after the stimulus or the initiating factor is removed.

Neoplasms can occur from virtually any tissue anywhere in the body and the oral cavity is an important location where a large variety of neoplasms often develop with diverse pathogenicity.

The modern classification of oral neoplasms is based primarily on the structural basis or in other words, several neoplastic conditions are put into different categories on the basis of their tissue of origin.

Depending on the pathologic state, the oral neoplasms can be divided into two broad categories or groups, namely:

1. Benign neoplasm
2. Malignant neoplasm

In the following section, we will see how a benign neoplasm may differ clinicopathologically from its malignant counterpart (Table 2.1).

Generally, the benign tumor is designated by attaching the suffix “oma” to the cell type from which it arises. For example, a benign tumor arising from the fibrous tissue is called a “fibroma” while a benign cartilaginous tumor is called a “chondroma”. A benign epithelial tumor arising from the gland is known as “adenoma”.

A malignant tumor arising from the epithelial tissue is called “carcinoma” and a malignant tumor arising from the connective tissue is known as “sarcoma”. Recent literatures have documented about another malignancy, which is called “carcinosarcoma” and it is characterized by simultaneous malignant transformation of both the epithelial and mesenchymal components of the tissue.

### ■ LOCAL INVASION

When a tumor penetrates into the adjoining tissues due to its increased rate of growth, it is known as invasion. Most of the malignant tumors as well as few benign tumors show this behavior. Invasion is an important pathological

**Table 2.1:** Difference between benign and malignant neoplasms.

	Features	Benign	Malignant
On the basis of clinical features	Size of the tumor	Usually small	Usually large
	Rate of growth	Slow	Very fast
	Pain	Absent	Mostly painful
	Hemorrhage	Not usual	Very common
	Ulceration	Absent	Present
	Paresthesia	Does not occur	Commonly occurs
	Induration	Absent	It is often present
	Symptoms	Asymptomatic	Always symptomatic
	Superadded infection	Usually absent	Commonly present
	Necrotic areas	Usually absent	Commonly present
	Metastasis	Usually Absent	Very common
On the basis of histopathologic features	Cell multiplication rate	Slow	Very fast
	Cell maturation	Good	Cells are often immature
	Cell uniformity	Uniform	Irregular size and shape
	Cell morphology	Not changed	Normal cell morphology is lost
	Cell function	Restored	Mostly lost
	Stroma	Almost normal	Exhibits invasion
	Tissue architecture	Intact	Mostly lost or altered
	Capsule	(Resembles normal tissue)	Absent
Prognosis		Good	Mostly poor

change in any malignant neoplasm, which determines the future course of the neoplasm as well as the prognosis.

### ■ METASTASIS

Metastasis can be defined as the distant spread of tumor cells anywhere in the body away from its primary location. This is an important characteristic of the malignant tumor. The tumor which occurs initially is called the primary tumor; while the newly formed tumor developing as a result of metastasis at a distant site is called the metastatic or secondary tumor.

During metastasis, the tumor cells spread either via the lymphatic channels or the blood vessels, besides this, in some cases, the metastatic cells can spread via the nerve sheath or even through other natural tissue spaces. With some exceptions, the carcinomas generally metastasize via lymphatic channels while the sarcomas metastasize via blood vessels.

### ■ CLASSIFICATION OF ORAL NEOPLASMS (TUMORS)

In the oral cavity, several types of neoplasms often develop and these entire varieties of neoplastic lesions are broadly divided into two categories:

1. Odontogenic neoplasm and
2. Nonodontogenic neoplasms.

**Odontogenic neoplasms:** These are a group of neoplastic conditions either benign or malignant, which develop from the dental formative tissues or their remnants.

**Nonodontogenic neoplasms:** These are the neoplastic lesions, which arise from virtually any tissue in the oral cavity excepting from those arising from the dental formative organs.

The nonodontogenic neoplasms can develop from several tissues like skin or mucous membrane, fibrous connective

**Table 2.2:** Neoplasms of epithelial tissue origin.

<b>Benign neoplasms</b>	<b>Malignant neoplasms</b>
Papilloma	Basal cell carcinoma
Keratoacanthoma	Squamous cell carcinoma
Pigmented cellular nevus	Verrucous carcinoma
Papillary hyperplasia	<ul style="list-style-type: none"> <li>• Adenoid squamous cell carcinoma</li> <li>• Adenosquamous cell carcinoma</li> <li>• Malignant melanoma</li> <li>• Spindle cell carcinoma</li> <li>• Primary intra-alveolar carcinoma</li> <li>• Multicentric oral carcinoma</li> </ul>

tissue, blood vessels, muscles, bone, cartilage, neural tissue and lymphoid tissue, etc. It is important to remember that unlike the odontogenic neoplasms which can arise only in the oral cavity or its surrounding areas, the nonodontogenic neoplasms are not always confined to the oral region, rather they can develop in other parts of the body as well.

### Classification of Oral Nonodontogenic Neoplasms

The neoplasms of epithelial tissue origin and neoplasms of mesenchymal tissue origin are given in Tables 2.2 and 2.3, respectively.

**Table 2.3:** Neoplasms of mesenchymal tissue origin.

<b>Benign neoplasms</b>	<b>Malignant neoplasms</b>
<i>Neoplasms of fibrous connective tissue</i>	<i>Neoplasms of fibrous connective tissue</i>
Fibroma	Fibrosarcoma
Fibromatosis	Malignant fibrous
Desmoplastic fibroma	Histiocytoma
Pyogenic granuloma	
Fibroepithelial polyp	
Giant cell fibroma	
Peripheral ossifying fibroma	
Central ossifying fibroma	
Peripheral giant cell granuloma	
Central giant cell granuloma	
Benign fibrous histiocytoma	
Nodular fasciitis	
Myxoma	
<i>Neoplasms of adipose tissue</i>	<i>Neoplasms of adipose tissue</i>
Lipoma	Liposarcoma
Angiolipoma	
<i>Neoplasms of vascular tissue</i>	<i>Neoplasms of vascular tissue</i>
Hemangioma	Hemangiopericytoma
Lymphangioma	Hemangioendothelioma
Juvenile angiofibroma	Angiosarcoma
Hereditary hemorrhagic telangiectasia	Kimura's disease
Glomus tumor	

Contd...

Contd...

<b>Benign neoplasms</b>	<b>Malignant neoplasms</b>
<i>Neoplasms of osseous tissue</i>	<i>Neoplasms of osseous tissue</i>
Osteoma	Osteosarcoma
Osteomatosis	Parosteal osteosarcoma
Osteoid osteoma	Ewing's sarcoma
Osteoblastoma	
Osteoclastoma	
Torus palatinus	
Torus mandibularis	
<i>Neoplasms of cartilaginous tissue</i>	<i>Neoplasms of cartilaginous tissue</i>
Chondroma	Chondrosarcoma
Chondroblastoma	Mesenchymal
Chondromyxoid fibroma	Chondrosarcoma
<i>Neoplasms of neural tissue</i>	<i>Neoplasms of neural tissue</i>
Neurolemmoma	Neurosarcoma
Neurofibroma	Olfactory neuroblastoma
Neurofibromatosis	
Multiple endocrine neoplasia syndrome	
Melanotic neuroectodermal tumor of infancy	
Neuroblastoma	
Ganglioneuroma	
Traumatic neuroma	
Plexiform neuroma	
<i>Neoplasms of smooth muscle tissue</i>	<i>Neoplasms of smooth muscle tissue</i>
Leiomyoma	Leiomyosarcoma
Angiomyoma	Angiomyosarcoma
<i>Neoplasms of striated muscle tissue</i>	<i>Neoplasms of striated muscle tissue</i>
Rhabdomyoma	Rhabdomyosarcoma
Granular cell myoblastoma	
Congenital epulis of newborn	
<i>Neoplasms of lymphoid tissue</i>	<i>Neoplasms of lymphoid tissue</i>
No benign neoplasm	Hodgkin's lymphoma
	Non-Hodgkin's lymphoma
	Burkitt's lymphoma
	Mycosis fungoides
	Leukemias
	Multiple myeloma
	Plasmacytoma
<i>Neoplasms of mixed tissue</i>	<i>Neoplasms of mixed tissue</i>
Teratoma	
<i>Neoplasms of salivary gland tissue</i>	<i>Neoplasms of salivary gland tissue</i>
See the chapter of Salivary Gland Neoplasm (Chapter 4)	



## BENIGN NEOPLASMS OF THE EPITHELIAL TISSUE ORIGIN

### Papilloma

#### Definition

Papilloma is a common benign neoplasm of the oral cavity, arising from the epithelial tissue. It is characterized by an exophytic papillary growth with a typical “cauliflower like” appearance.

This lesion constitutes about 2% of all oral neoplasms and it is believed by many investigators that they are caused by human papilloma virus (HPV).

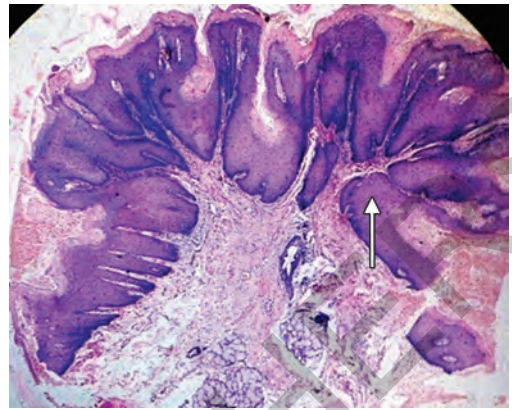
HPV virus subtypes 6 and 11 frequently detected from neoplastic tissues of papilloma.

#### Clinical Features

- **Age:** Any age but mostly third, fourth and fifth decade
- **Sex:** Both sexes are equally affected
- **Site:** Tongue, lips, buccal mucosa, gingiva, hard and soft plate, etc.

#### Clinical Presentation (Figs. 2.1 and 2.2)

- Clinically, papilloma appears as a slow growing, exophytic, soft, usually pedunculated, painless, nodular growth often with a typical “cauliflower-like” appearance.
- Papillomas often characteristically have numerous finger-like projections on their surface, which can be either blunt or pointed.



**Fig. 2.2:** Histopathology of papilloma (low power).

- Because of these projections, the papilloma often appears as an ovoid swelling with a rough, corrugated surface.
- The size of the lesion is usually small and that varies from few millimeters to about one centimeter in diameter.
- The base of the lesion can be either pedunculated or sessile (broad based) but papilloma is mostly a well-circumscribed growth.
- The lesion is mostly white in color and is firm in consistency as the surface is highly keratinized.
- On rare occasions, papillomas may grow in an inwardly direction (inverted type) instead of growing in the usual exophytic manner. Such lesions are mostly



**Figs. 2.1A**

(A) Papilloma on the palate; (B) Papillomatosis of tongue.



**Fig. 2.3:** Keratoacanthoma of lower lip.

seen in the lateral nasal wall, paranasal sinuses and in the maxillary antrum, etc. Moreover, they have great tendency for local destruction and malignant transformation.

- Multiple papillomas may sometimes coalesce together and form a large lesion in the oral cavity and the condition is commonly known as “papillomatosis” (Figs. 2.3 and 2.4).
- Papillomatosis of oral mucosa may sometimes occur in association with skin disorders, e.g. focal dermal hypoplasia syndrome, nevus unius lateris, Cowden syndrome and acanthosis nigricans, etc.

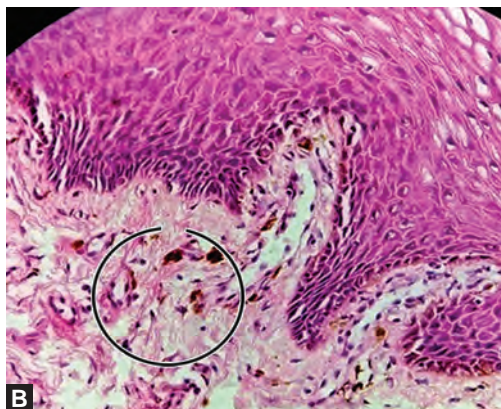
### Histopathological Features

Microscopically, papillomas present the following features:

- Proliferating keratinized stratified squamous epithelium in the form of multiple finger-like projections.
- Every single finger-like projection has a fibro-vascular connective tissue core in the center, which contains few inflammatory cells.
- The covering squamous epithelium shows hyperkeratosis and acanthosis. Thickening of the keratin is seen in lesions which are clinically whiter.
- In the spinous cell layer “koilocytes” are sometimes seen, these are virus-altered epithelial clear cells with small dark (pyknotic) nuclei surrounded by clear halos.
- There can be little cellular atypia in some papillomas, however, the dysplastic changes in the epithelium is rarely found.
- Papilloma is not a premalignant lesion and malignant transformation in preexisting oral papillomas has not been documented.

### Differential Diagnosis

- Verruca vulgaris
- Focal dermal hyperplasia
- Verruciform xanthoma
- Verrucous carcinoma
- Condyloma acuminatum.



**Figs. 2.4A and B:** (A) Nevus on the lip; (B) Nevus cells.

**Treatment**

Conservative surgical excision of the lesion including the base is the common treatment. Recurrence is rare.

**Keratoacanthoma (Self-healing Cancer)****Definition**

Keratoacanthoma is a benign endophytic epithelial tissue neoplasm with profound clinical and histological resemblance to well-differentiated squamous cell carcinoma (SCC). It commonly occurs in the sun-exposed skin of the face and it usually appears as a circumscribed keratin filled crater.

**Origin**

Keratoacanthoma of the skin surfaces probably develops from the hair follicles above the sebaceous glands.

On the mucosal surfaces these lesions are extremely rare but if they occur at all, they probably develop from the superficial epithelium of the sebaceous ducts.

**Causes**

- Chronic sun exposure
- HPV infection (especially types—9, 11, 13, 16, 18, 25, 33, 37 and d5)
- Immunosuppression
- *Heredity*: Chromosomal aberrations such as gains on 8q, 1p, and 9q with deletions on 3p, 9p, 19p, and 19q.
- Trauma.

**Clinical Features**

- *Age*: Middle-aged adults are frequently affected between the age group of 50 years and 70 years
- *Sex*: Male to female ratio in this tumor is about 2:1
- *Site*: Keratoacanthoma chiefly develops over the sun-exposed skin surface of the lips (both upper and lower lips) near the outer edge of vermilion border. Besides this, the lesion can

also occur on the cheeks, nose, eyelids and ear. Intraoral lesions of keratoacanthoma are rare, although few have been reported in the palate and gingiva.

**Presentation (Fig. 2.3)**

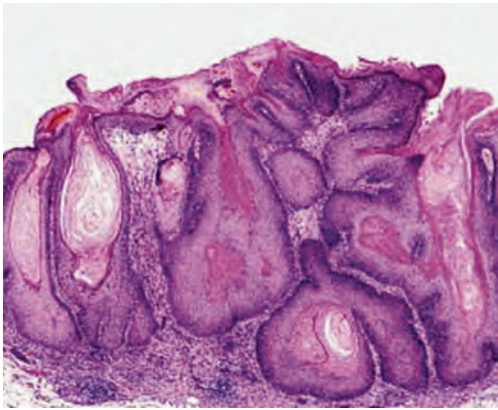
- Keratoacanthoma initially begins as a small, red macule that soon turns into a well-circumscribed, elevated and umbilicated, crater-like lesion with a central depression.
- The lesion is firm, painless and sessile in nature; and can be single or multiple in number.
- The fully developed lesion of keratoacanthoma clinically presents a well-circumscribed, elevated nodule, which has a sharply delineated, rolled margin and a central keratotic core.
- Clinically, the outer surface of the lesion shows normal skin color or slight erythema, while the central keratin plug appears yellow, brown or black with an irregular crusted appearance.
- The disease is often painful and sometimes it may have an associated lymphadenopathy.

**Stages of Development of Keratoacanthoma**

- *Growth phase*: The lesion initiates as a small lump or a bud like growth on the sun-exposed skin surface of the face, it grows rapidly and achieves the maximum size (1–2 cm in diameter) over a period of about 4–8 weeks.
- *Stationary phase*: After the initial growth, the disease remains static for an indefinite period of up to 4–8 weeks and then it starts to regress spontaneously.
- *Involution phase*: Within the next 6–12 months time, the lesion regresses completely leaving only a small depressed scar.

*Muir-Torre syndrome*: Gastrointestinal carcinoma, keratoacanthoma, and sebaceous neoplasm.





**Fig. 2.5:** Photomicrograph of keratoacanthoma.

### Histopathology (Fig. 2.5)

- Keratoacanthoma clinically and histologically appears very similar to well-differentiated SCC and because of this, it is often known as “self-healing” cancer (Box 2.1).
- The cells appear mature and often there is individual cell keratinization and even keratin pearl formation in the tumor.
- The lesion consists of a thick hyperkeratinized covering epithelium with a central zone of keratin or parakeratin plugging.
- Pseudoepitheliomatous hyperplasia may be observed in some cases.
- Pathognomonic nonmalignant feature of this neoplasm can be identified at the margin, where the lesion shows a crater-like area, plugged with keratin and is surrounded by hyperplastic normal epithelium.
- This abrupt transition of the normal surrounding epithelium at the margin

#### Box 2.1

The distinction between keratoacanthoma and squamous cell carcinoma.

- The epithelium in this neoplasm exhibits a pseudocarcinomatous rather than a true carcinomatous growth pattern
- Dyskeratosis is always absent in keratoacanthoma
- The epithelium is composed of well-differentiated spinous cells with abundant cytoplasm, minimal nuclear pleomorphism, infrequent mitotic figures and no abnormal mitosis

of the crater-like area is an important diagnostic clue for keratoacanthoma.

- Although, keratoacanthomas are benign and self-limiting conditions, serial sectioning is always required of the available tissue sample for confirmation of the diagnosis.
- Moreover, careful long-term follow-up evaluations are necessary since the neoplasm is often confused with SCC.

### Differential Diagnosis

- Basal cell carcinoma
- Squamous cell carcinoma.

### Treatment

Surgical excision is the treatment of choice for keratoacanthoma, usually before the lesion reaches its maximum size of 2–2.5 cm diameter. Waiting for spontaneous regression of the lesion is not advisable for the following reasons:

- Confusion with SCC
- The scar developing after spontaneous regression is depressed and cosmetically unacceptable
- Surgical treatment always provides good tissue specimen for confirmation of the diagnosis.

## Pigmented Cellular Nevus

### Definition of Nevus

The term “nevus” has got several meanings; in *Latin* nevus means birth marks, however, the common lay term used for nevus is “mole”. A nevus can be defined as a congenital, developmental, tumor-like malformation of the skin or mucous membrane; which are often present at birth or they can be seen any time after birth (Box 2.2).

Oral nevi are much rarer than their cutaneous counterparts with a prevalence of 0.1% among the general population; Women are affected twice as common as men and most cases are seen in the third and fourth decades. In the mouth these are commonly seen in the hard palate, buccal and labial mucosa.

**Box 2.2** Different types of nevi.

- Intradermal (intramucosal) nevus
- Junctional nevus
- Compound nevus
- Blue nevus
- Halo nevus (halo mole)

Nevus is composed of “nevus cells” which are neuroectodermal in origin, these cells, except for their tendency to form cell nests and their less prominent dendritic processes, are nothing but melanocytes or their precursors. After their formation, nevus cells migrate through the peripheral nerves and finally reach to the basal layer of the skin or mucous membrane (See Figs. 2.4A and B).

The function of nevus cells is to produce melanin, this pigment, after being synthesized within the nevus cells is passed on to the adjacent keratinocytes of the oral mucous membrane.

**Intradermal (Intramucosal) Nevus**

The term intradermal nevus and intramucosal nevus are synonymous, the former occurs on the skin surfaces while the later occurs over the mucous membrane.

**Clinical Features**

- Intradermal nevus is a very common lesion of the skin (comprise more than 50% of all types of nevi) and it usually occurs in children
- This lesion is often referred to as common “mole”
- Intradermal nevus clinically appears as a raised or flat area on the skin surface, with a typical tan or dark brown color
- The mucosal lesions clinically appear as asymptomatic, slightly elevated papules or flat macules with a pigmented surface
- The color of these nevi varies from brown to black
- The intraoral lesions are often slow growing and their size is usually less than 1 cm in diameter.

**Histopathology**

- Microscopically intramucosal nevus reveals clusters or nests of nevus cells which are confined within the connective tissue.
- The cells may appear as epithelioid cells or lymphocyte like cells; however few cells may be even spindle-shaped.
- Multiple multinucleated giant cells may be found in some cases.
- Intramucosal nevus often characteristically presents a narrow zone of connective tissue devoid of nevus cells, which separates the zone of nevus cells from the overlying epithelium.
- The amount of melanin produced by these nevus cells varies; some cells are heavily pigmented whereas other cells are almost nonpigmented.

**Treatment**

Intramucosal nevi usually do not require any treatment. However, when these lesions are subjected to persistent trauma during mastication, surgical excision should be preferred. Once excised, they usually do not recur.

**Junctional Nevus****Clinical Features**

- Junctional nevus is usually a less common variety as compared to the intradermal nevus.
- It appears as an asymptomatic, brown or black macule, affecting both skin as well as the oral mucosal surfaces.
- Histologically, junctional nevus reveals focal areas of proliferating nevus cells or in some cases clusters of cells at the basement membrane zone of the epithelium.
- The cluster of nevus cells is often specifically present at the apex of the epithelial rete-pegs.

### **Treatment**

Junctional nevus should be excised surgically. Postsurgical recurrence is uncommon.

### **Compound Nevus**

The compound nevus characteristically presents the combined features of intradermal nevus and the junctional nevus.

### **Clinical Features**

- This lesion occurs far more commonly in skin as compared to the oral mucosa
- Intraorally they appear as pigmented papules or macules over the hard palate or the gingiva.

### **Histopathology**

Microscopically, compound nevus reveals the presence of nevus cells, which are distributed both in the basal layer of the epithelium as well as in the adjacent superficial connective tissue.

### **Treatment**

Surgical excision is the treatment of choice for compound nevus.

### **Blue Nevus**

Blue nevus is a relatively common pigmented lesion of the oral cavity.

- Two major types are recognized—(1) the common blue nevus and (2) the cellular blue nevus.
- The common blue nevus is the second most frequent melanocytic nevus encountered in the mouth.

### **Clinical Features**

- Clinically, it often appears either as a dome-shaped, dark blue papule or as a flat pigmented macule over the skin or the oral mucous membrane.
- Intraoral blue nevi are commonly seen on the mucosal surfaces of the hard palate.

### **Histopathology**

- Instead of being round or epithelioid in shape, the cells of blue nevus are usually elongated, bipolar and spindle-shaped.
- Sometimes, fusiform dendritic cells can also be present in these lesions.
- The spindle-shaped cells are mostly oriented parallel to the overlying epithelium and are not arranged in clusters.
- Few pigmented macrophages may be present among these dendritic nevus cells and they are known as “melanophages”.

### **Treatment**

Blue nevus often clinically resembles a melanoma and therefore surgical excision of the lesion with subsequent histopathological evaluation blue nevi do not have much tendency to undergo malignant transformation.

### **Halo Mole (Halo Nevus)**

It is a peculiar nevus on the skin, which characteristically has a white ring around it; (also known as Sutton nevus or leukoderma acquisitum centrifugum) (Fig. 2.6).

It develops like any other nevus of the skin with the typical nevus cell being present in it, but later on, an autoimmune reaction develops against the nevus to destroy it, and there is activation of cytotoxic T lymphocytes, which start destroying the nevus cells. The white ring is



**Fig. 2.6:** Halo nevus on the facial skin.



**Box 2.3**

Symptoms of malignant transformation in a preexisting nevus.

- Sudden faster rate of growth
- Size becoming much more than 5 mm in diameter
- Asymmetric growth pattern
- Irregular border of the lesion
- Sudden change of color (becoming darker in color).
- Development of surface ulceration and pain (sometimes)

*(Histopathological confirmation is always required)*

created due to the elimination of the pigmented cells at the periphery of the nevus (Box 2.3).

## ■ MALIGNANT NEOPLASMS OF THE EPITHELIAL TISSUE ORIGIN

### Squamous Cell Carcinoma

#### Definition

Squamous cell carcinoma is the most common malignant epithelial tissue neoplasm of the oral cavity, which is derived from the stratified squamous epithelium. Since oral SCCs constitute bulk of the oral malignancies (above 90%) it is thus commonly referred to as oral cancer (although there are several other malignancies of the oral cavity besides SCC).

#### Epidemiology

- SCC is also termed as epidermoid carcinoma and it is by far the most common malignant neoplasm of the oral cavity.
- The incidence of oral SCC varies in different countries and also in different population groups. It represents the eleventh most common cancer in males and the sixteenth most common in females.
- On an average, oral SCCs represent about 3% of all cancers in males and about 2% of all cancers in females.
- This disease is responsible for 2% of all annual deaths in males and 1% of all annual deaths in females.
- The incidence of oral cancer varies in different countries depending upon the frequency of tobacco usage and other related habits throughout the world.
- The general trend indicates that the incidence of oral SCC increases alarmingly in the societies, where extensive tobacco use begins in the early life and is continued for a longer period.
- The incidence of oral SCC increases with age and most of the cases occur usually after the age of 40 years.
- Although, oral SCCs can arise from virtually any intraoral site, but they develop more frequently from the lower lip, lateral borders of the tongue, buccal mucosa and floor of the mouth, etc.
- According to this ICD classification, oral cancers are numerically categorized in the following manner:
  - Lip cancer-ICD No. 140
  - Tongue cancer-ICD No. 141
  - Cancer of the gingiva and alveolar mucosa-ICD No. 143
  - Cancer of the floor of the mouth-ICD No. 144
  - Cancer of other parts of the mouth-ICD No. 145.
- The annual age-adjusted incidence rates of oral cancer per 100,000 (one lakh) population varies from continents to continents, from countries to countries and also from places to places within the same country.
- In Europe, it varies from 2.0, in UK to 9.4, in France, in USA it varies from 4.4, in Columbia to 13.4 in Canada.
- In Asia, the annual incidence rates of oral cancer per 100,000 population vary from 1.6 in Japan to as high as 13.5 in India.
- In Sri Lanka, the oral cancers constitute about 40% of all malignancies.
- In the Manipuri districts in India, the annual incidence rate of oral cancer is about 21.4 per 100,000 population (Wahi 1968).
- Survey among textile mill workers in Ahmedabad (Gujarat), India, indicates that

the average incidence rate of oral cancer among individuals above 35 years of age is 25 per 100,000 population (Malaowalla et al.).

- Recent trends besides few exceptions indicate that the incidence and mortality rates of oral cancer are declining and it can be due to the reduced exposure to various etiological agents.

### ***Etiology of Oral Cancer***

A large number of etiological factors have been implicated in the development of oral cancer, specifically the oral SCCs, these include the following:

- Tobacco smoking
  - Cigarettes
  - Beedies
  - Pipes
  - Cigars
  - Reverse smoking
- Use of smokeless tobacco
  - Snuff dipping
  - Tobacco sachets (*Gutkha*)
  - Tobacco chewing
  - Tobacco as a toothpaste
- Consumption of alcohol
  - Drinking spirits
  - Drinking wines
  - Drinking beers
  - Tobacco and alcohol synergism (smoking and chewing tobacco with drinking of alcohol)
- Diet and nutrition
  - Vitamin A, B-complex and C deficiency
  - Nutritional deficiency with alcoholism
- Dental factors
  - Chronic irritation from broken teeth
  - Ill-fitting or broken prosthesis
- Radiations
  - Actinic radiation, X-ray radiation
- Viral infections
  - Herpes simplex virus (HSV)
  - Human papilloma virus (HPV)
  - Human immunodeficiency virus (HIV)
  - Epstein-Barr virus (EBV)
- Immunosuppression
  - AIDS
  - Organ transplants
- Chronic infections
  - Candidiasis
  - Syphilis
- Occupational hazards
  - Woolen textile workers
- Genetic factors
  - Oncogenes
  - Tumor-suppressor genes
- Preexisting oral diseases
  - Lichen planus
  - Plummer-Vinson syndrome
  - Oral submucous fibrosis
  - Leukoplakia
  - Discoid lupus erythematosus.

### ***Role of Tobacco in Oral Cancer***

- One person dies in every 10 seconds in the world due to the use of tobacco.
- Epidemiological and experimental studies categorically indicate that tobacco plays an important role in the development oral cancer.
- Tobacco is used in various smoking forms such as cigarettes, cigars, pipes and *beedies*, etc.
- It can also be used in smokeless forms like paan-beetel quid, snuff, tobacco sachets (*khaini*), *zarda* and other popular forms.

### ***Factors Influencing the Carcinogenic Effect of Tobacco***

- Frequency of smoking—number of cigarette or Pipe used per day (smoking 40 or more cigarettes per day increases 10–20 times more cancer risk).
- Duration of smoking—habit continued for how many years.
- Age of beginning of the habit—smoking from a younger age has more risk.
- Composition of tobacco—variation in tar, nicotine and nitrosamine content, e.g. “beedi” (country made cigarette) is made up of crude form of tobacco containing more harmful chemicals and it has no filter.

# Essentials of Oral Pathology

## Salient Features

- All chapters are adequately covered, revised and updated within a concise volume
- Inclusion of large numbers of clinical and radiographic photographs and several photomicrographs of very important cases is the hallmark feature of this edition
- Simple and lucid language for easy understanding
- Concise and comprehensive presentation of each topic will be helpful for both undergraduate and postgraduate students
- Latest classifications of oral diseases, recent grading and staging system of tumors have been added
- Several diagrams, tables and flowcharts have been provided to help students understand the subject better.

**Swapan Kumar Purkait** MDS is Professor and Postgraduate Teacher, Department of Oral and Maxillofacial Pathology, Buddha Institute of Dental Sciences and Hospital, Patna, Bihar, India. He completed his BDS (1990) and MDS (1996) from University of Calcutta, Kolkata, West Bengal, India. Considered as an eminent teacher, he has been continuously associated with teaching oral pathology, oral histology and morphology. He has published several articles on important topics in Oral Pathology in national and international journals. He has been the examiner for undergraduate and postgraduate examinations at various universities. He has great likings towards literature and music, especially the Hindustani Classical Music and he has received a *Sangeet Visharad* degree in *Tabla playing*.

Available at all medical bookstores  
or buy online at [www.jaypeebrothers.com](http://www.jaypeebrothers.com)



**JAYPEE BROTHERS**  
**Medical Publishers (P) Ltd.**  
[www.jaypeebrothers.com](http://www.jaypeebrothers.com)

Join us on [facebook.com/JaypeeMedicalPublishers](https://www.facebook.com/JaypeeMedicalPublishers)

Shelving Recommendation  
**DENTISTRY**

ISBN 978-93-5270-570-2

