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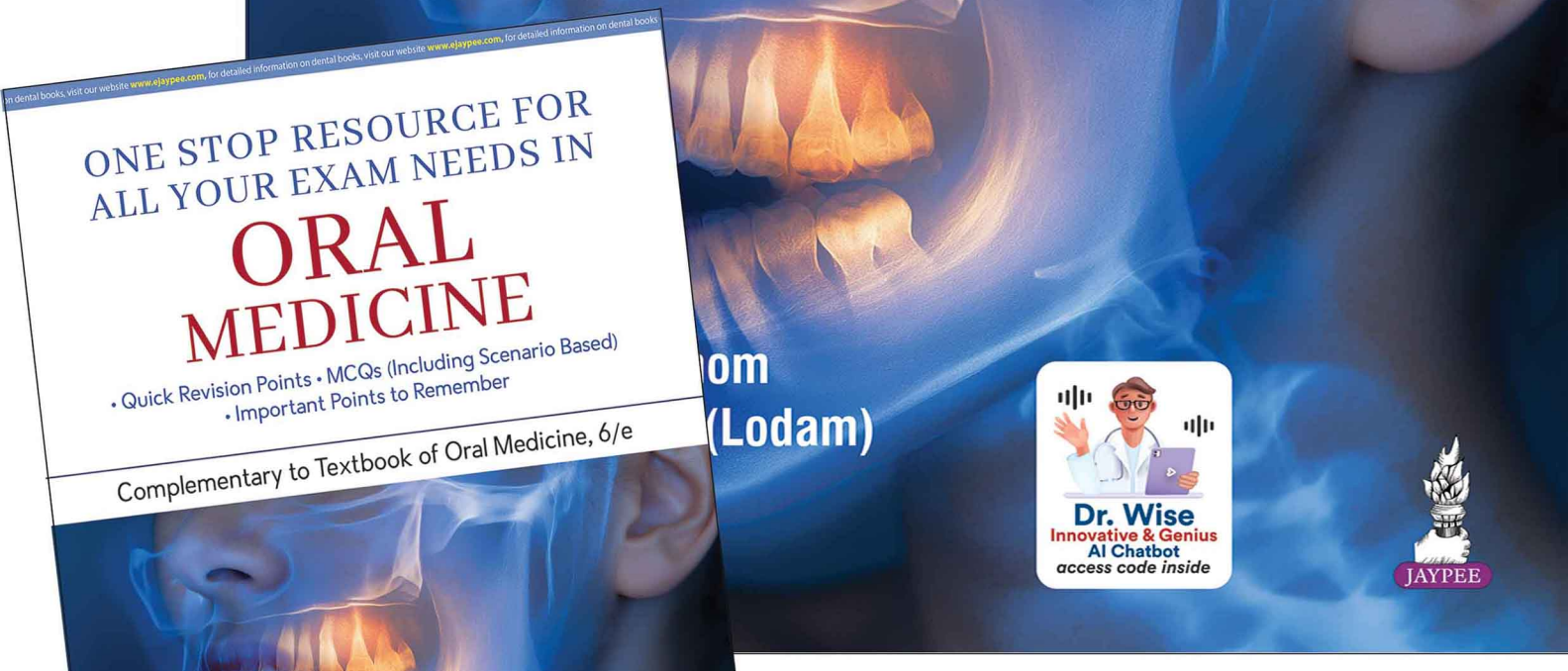

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Contents

Section 1: Basics and Case History 1

Chapter 1: An Introduction to Oral Medicine 3
Anil Ghom, Savita Ghom
- Definitions and History 3
- Goals and Objectives of Oral Medicine 3
- Curriculum of Oral Medicine and Radiology in India 4

Chapter 2: Case History and Diagnosis in Oral Medicine 6
Savita Ghom, Abhijeet Deoghare
- Case History 6
- Components of Dental History Taking 7

Data and History Recording 7
- Identifying Data or Personal Information 7
- Chief or Presenting Complaint 8
- History of Present Illness 9
- Past Dental History 11
- Past Medical History 12
- Drug History 12
- Personal History 12
- Family History 14

Examination 14
- General Examination 14
- Extraoral examination 17
- Intraoral Examination 23
- Examination of Nondental Structures 24
- Examination of Dental Structures 27
- Examination of Swelling 31
- Examination of Ulcer 36
- Examination of Sinus or a Fistula 37
- Examination of Cranial Nerves 38

Establishing the Diagnosis and Treatment Plan 40
- Provisional Diagnosis 40
- Investigations 40
- Final Diagnosis 40
- Making a Treatment Plan and Medical Risk Assessment 41

Chapter 3: Chairside Investigations 43
Anil Ghom, Deeplaxmi Dewangan

Investigations for Dental Hard Tissues and Vascular Structures 44
- Pulp Vitality Tests 44
- Caries Detection Test 46
- Plaque Disclosing Agents 49
- Percussion Test 49
- Mobility Test 50
- Depressibility Test 50
- Tests to Detect Cracked Tooth 50
- Transillumination Test 50

Test for Detection of Potentially Malignant Lesions and Conditions 50
- Diascopy 50
- Vital Staining 51
- Light-Based Detection System 52
- Cytological Techniques 52
- Histological Techniques 54

Investigation for Saliva and Taste Evaluation 55
- Saliva Collection 55
- Tests for Xerostomia 55
- Tests for Detection of Taste Disorders 55
- Test for Oral Malodor 56

Advanced Chairside Tests 57
- Investigation for Allergy 57
- Investigation for Neuromuscular Disorder 57
- Hematological Investigation 57
- Test for Trauma from Occlusion 61
- Oral Fluid Nanosensor Test 61

Section 2: Developmental Disturbances 63

Chapter 4: Developmental Disturbances of Jaws and Cranial Structures 65
Savita Ghom, Siddharth Pundir, Anil Ghom
- Introduction and Definitions 65
- Agnathia 66
- Micrognathia 66
- Macrognathia 67
- Facial Hemihypertrophy 68
- Facial Hemiatrophy 69
- Hemi-maxillofacial Dysplasia 70
- Cleidocranial Dysplasia 70
- Craniofacial Dysostosis 72
- Mandibulofacial Dysostosis 73
- Hyperplasia of Maxillary Tuberosity 75
- Hyperplasia of Coronoid Process 75
- Focal Osteoporotic Bone Marrow Defect 75
- Chondroectodermal Dysplasia 75
- Arhinencephaly 76

- Phlebectasia 77
- Ectodermal Dysplasia 77
- Gardner's Syndrome 79
- Turner's Syndrome 79
- Apert Syndrome 80
- Kallman Syndrome 81
- Johanson-Blizzard Syndrome 82
- Hallermann-Streiff Syndrome 82
- Russell-Silver Syndrome 84
- Distal Arthrogryposis Syndrome 85

Chapter 5: Developmental Disturbances of Teeth.....87
Anil Ghom, Siddharth Pundir, Savita Ghom

Developmental Defects Affecting Size of Teeth 87
- Microdontia 87
- Macrodontia 88

Developmental Defects Affecting Shape of Teeth 89
- Gemination 89
- Twining 89
- Fusion 89
- Concrescence 90
- Talon's Cusp 91
- Cusp of Carabelli 92
- Dilaceration 92
- Dens in Dente 93
- Dens Evaginatus 95
- Shovel-Shaped Incisors 96
- Taurodontism 96
- Supernumerary Roots 97
- Paramolar Tubercle or Bolk Cusp 97
- Enamel Pearls or Droplet, Nodules 97
- Globodontia or Otodental Syndrome 98
- Mulberry Molar 99
- Moon's Molar 99
- Hutchinson's Incisor 99
- Hypercementosis 99

Developmental Defects Affecting Number of Teeth 100
- Anodontia, Hypodontia and Oligodontia 100
- Supernumerary Teeth 101
- Pre-deciduous Dentition 103
- Post-permanent Dentition 104

Developmental Defects Affecting Structure of Teeth 104
- Amelogenesis Imperfecta 104
- Dentinogenesis Imperfecta 107
- Dentin Dysplasia 109
- Regional Odontodysplasia 110

Environmental Alteration Affecting Tooth 111
- Structure of Teeth 111
- Disturbances in Eruption 114

Section 3: Diseases of Teeth and Peridontium 121

Chapter 6: Dental Caries.....123
Anushka Deoghare, Anil Ghom
- Etiology 123
- Pathogenesis 123
- Classification of Dental Caries 125
- Diagnosis of Dental Caries 125
- Types of Caries 128
- Radiographic Differential Diagnosis of Dental Caries 136
- Control of Dental Caries 137

Chapter 7: Pulp and Periapical Infections.....141
Savita Ghom, Anil Ghom

Pulpal Pathologies 142
- Reversible Pulpitis 142
- Irreversible Pulpitis (Acute and Chronic) 143
- Chronic Hyperplastic Pulpitis 143

Periapical Pathologies 144
- Acute Apical Periodontitis 144
- Periapical Abscess 145
- Acute Exacerbation of a Chronic Lesion 149
- Pericoronal Abscess 150
- Periapical Granuloma 151
- Periapical Scar 152

Bone Infections 152
- Osteomyelitis 152
- SAPHO Syndrome 163

Chapter 8: Gingival and Periodontal Diseases.....164
Anil Ghom, Aena Jain (Pundir)
- Classification of periodontal diseases 164

Plaque-Induced Diseases of Gingiva 166
- Gingivitis 166
- Plasma Cell Gingivitis 169
- Gingival Enlargement 170
- Necrotizing Ulcerative Gingivitis 175

Non-plaque-induced Gingival Disease 177
- Hereditary Gingival Fibromatosis or Fibromatosis Gingiva 177
- Gingival Diseases of Bacterial Origin 179
- Gingival Diseases of Viral Origin 179
- Gingival Diseases of Fungal Origin 179
- Gingival Manifestations of Systemic Conditions 179
- Gingival Recession 180
- Traumatic Lesions of Gingiva 181
- Foreign Body Reaction 181
- Gingival Pigmentation 181
- Carcinoma of the Gingiva 183

Periodontal Diseases 183
- Necrotizing Ulcerative Periodontitis 184
- Periodontitis 184

- Periodontal Pockets 188
- Periodontal Abscess 189
- Prepubertal Periodontitis 190
- Tooth Mobility 190
- Papillon-Lefevre Syndrome 191
- Endodontic–Periodontal Lesions 191
- Occlusal Trauma in Periodontal Diseases 192

Radiological Assessment of Periodontal Diseases 192

- Radiographic Features of Healthy Periodontium 192
- Radiological Types of Periodontitis 192

Chapter 9: Facial Space Infections 196
Savita Ghom, Noopur Deshpande (Gupta), Manan Gupta

- Anatomic Considerations in Dentoalveolar Infection 196
- Spread of Infection 196
- Pathophysiology of Infection 197
- Classification of Facial Space Infections 198

Localized Infections 198

- Canine Space 198
- Buccal Space 199
- Infratemporal Space 199
- Submental Space 199
- Submandibular Space 200
- Sublingual Space 200
- Submasseteric Space 201
- Temporal Space 201
- Pterygomandibular Space 202
- Parotid Space 202
- Lateral Pharyngeal Space 202
- Retropharyngeal Space 203
- Management of Facial Space Infections 203

Complications of Facial Space Infections 204

- Cellulitis 204
- Ludwig's Angina 205

Fatal Complications of Oral Infections 207

- Bacterial Meningitis 207
- Brain Abscess 208
- Cavernous Sinus Thrombosis 208
- Odontogenic Infections of Orbit 209
- Mediastinitis 209
- Septicemia and Bacteremia 210
- Necrotizing Fasciitis 210
- Significance of Oral Foci of Infection 211

Chapter 10: Regressive Alterations of Teeth 213
Anil Ghom, Savita Ghom

- Classification 213
- Loss of Tooth Structure 213
- Resorption of Teeth 218
- Regressive Alterations in Dentin 222
- Regressive Alterations of Pulp 222
- Regressive Alterations of Cementum 222
- Discoloration of Teeth 223

Section 4: Cysts and Tumors of Head and Neck 227

Chapter 11: Cysts of Orofacial Region 229
Savita Ghom, Aarti Panchbhai, Anil Ghom

- Definition of Cyst 229
- General Diagnostic Features of Cysts 229
- Theories of Cyst Enlargement 230
- Classification 231
- Dentigerous Cyst 232
- Eruption Cyst 235
- Lateral Periodontal Cyst 236
- Gingival Cyst of Adult 237
- Palatal Cyst of Newborn (Epstein's Pearls, Bohn's Nodules) 238
- Dental Lamina Cyst 238
- Calcifying Odontogenic Cyst 239
- Glandular Odontogenic Cyst 240
- Odontogenic Keratocyst 241
- Orthokeratinized Odontogenic Cyst 244
- Radicular Cyst 244
- Residual Cyst 246
- Inflammatory Collateral Cysts 247
- Surgical Ciliated Cyst 248
- Suppurating Cyst 248
- Healing Cyst 248
- Nasopalatine Duct Cyst 248
- Nasolabial Cyst 250
- Epidermoid Cyst 250
- Dermoid Cyst 251
- Lymphoepithelial Cyst 252
- Thyroglossal Duct Cyst 252
- Anterior Median Lingual Cyst 252
- Oral Cyst with Gastric or Intestinal Epithelium 253
- Cystic Hygroma 253
- Nasopharyngeal Cyst 253
- Thymic Cysts 253
- Parasitic Cyst/Hydatid Cyst 253
- Cysticercosis Cellulosae 253
- Jaw Cyst-Basal Cell Nevus-Bifid Rib Syndrome 254
- Treatment of Cysts 255

Chapter 12: Odontogenic Tumors 256
Savita Ghom, Aarti Panchbhai, Anil Ghom

- Classification 256

Benign Epithelial Odontogenic Tumors 257

- Adenomatoid Odontogenic Tumor 257
- Calcifying Epithelial Odontogenic Tumor 258
- Ameloblastoma 260
- Unicystic Ameloblastoma 265
- Peripheral Ameloblastoma 266
- Adenoid Ameloblastoma 266
- Squamous Odontogenic Tumor 267
- Metastasizing Ameloblastoma 267

Benign Mixed Epithelial and Mesenchymal Odontogenic Tumors 268

- Ameloblastic Fibroma 268
- Odontoma 269
- Primordial Odontogenic Tumor 271
- Dentinogenic Ghost Cell Tumor 272

Benign Mesenchymal Odontogenic Tumors 273

- Odontogenic Myxoma 273
- Odontogenic Fibroma 274
- Benign Cementoblastoma 275
- Cemento-ossifying Fibroma 277

Malignant Odontogenic Tumor 279

- Ameloblastic Carcinoma 279
- Primary Intraosseous Carcinoma, NOS 280
- Sclerosing Odontogenic Carcinoma 281
- Clear Cell Odontogenic Carcinoma 282
- Ghost Cell Odontogenic Carcinoma 282
- Odontogenic Carcinosarcomas 283
- Odontogenic Sarcomas 283

Chapter 13: Benign Tumors of Non-odontogenic Origin.....285
Aarti Panchbhai, Anil Ghom, Jayanti Bishal

- Characteristics of Benign Tumors 286
- Classification of Benign Tumors 286

Epithelial Tumors 286

- Squamous Papilloma 286
- Condyloma Acuminatum 288
- Verruca Vulgaris 288
- Multifocal Epithelial Hyperplasia 289
- Oral Melanoacanthoma 289
- Keratoacanthoma 289
- Benign Melanocytic Nevus 290
- Verruciform Xanthoma 292

Fibroblastic and Myofibroblastic Tumors 292

- Fibroma 292
- Fibrous Hyperplasia 293
- Fibrous Epulis 294
- Nodular Fasciitis 294
- Desmoid Fibromatosis 295
- Solitary Fibrous Tumor 295
- Fibrous Histiocytoma 295
- Giant Cell Fibroma 295
- Myofibroma 295

Tumors of Cartilage Tissue Origin 296

- Chondroma 296
- Chondroblastoma 297
- Chondromyxoid Fibroma 297

Tumors of Adipose Tissue Origin 297

- Lipoma 297

Bone Tumors 299

- Osteoma 299
- Osteoblastoma 300
- Osteoid Osteoma 301
- Osteochondroma 302
- Torus Palatinus 302
- Torus Mandibularis 303
- Exostosis 303
- Enostosis 304

Tumors of Vascular Tissue Origin 305

- Hemangioma 305
- Sturge-Weber Syndrome 309
- Lymphangioma 310
- Arteriovenous Malformation/Fistula/Shunt 311
- Glomus Tumor 312
- Hemangiopericytoma 312
- Juvenile Nasopharyngeal Angiofibroma 313

Tumors of Neural Tissue Origin 313

- Neurofibroma or Neurofibromatosis 313
- Schwannoma 316
- Traumatic Neuroma 317
- Melanotic Neuroectodermal Tumor of Infancy 318

Tumors of Muscle Origin 318

- Leiomyoma 318
- Granular Cell Tumor 319

Giant Cell Neoplasm 320

- Central Giant Cell Tumor 320
- Peripheral Giant Cell Granuloma 321

Other Benign Tumors of Orofacial Region 322

- Teratoma 322
- Inflammatory Hyperplasia 323
- Myofibroma 323
- Oral Focal Mucinosis 323
- Myxofibroma 323
- Myxoma 323

Chapter 14: Malignant Tumors of Non-odontogenic Origin.....326
Savita Ghom, Aarti Panchbhai, Anil Ghom

- Classification of Non-odontogenic Malignancies 326

Malignant Tumors of Epithelial Tissues 327

- Basal Cell Carcinoma 327
- Malignant Melanoma 328
- Verrucous Carcinoma 330

Malignant Tumors of Connective Tissues 332

- Fibrosarcoma 332
- Liposarcoma 333

Malignant Tumors of Cartilage Tissue 334

- Chondrosarcoma 334

Malignant Tumors of Bone Tissue 336

- Osteosarcoma 336
- Ewing's Sarcoma 338

Malignant Tumors of Vascular Tissues 339

- Malignant Hemangioendothelioma 339

Malignant Tumors of Neural Tissues 339

- Neuroblastoma 339

Malignant Tumors of Muscle Tissues 340

- Leiomyosarcoma 340
- Rhabdomyosarcoma 341

Tumors of Lymphoid Tissues 341

- Hodgkin's Lymphoma 341
- Non-Hodgkin's Lymphoma 343

- Burkitt's Lymphoma 344
- Leukemia 346

Tumors of Plasma Cells 350

- Multiple Myeloma 350

Chapter 15: Oral Carcinoma....353
Anil Ghom, Savita Ghom

- Etiology and Risk Factors 353
- Oral Carcinogenesis 356
- Grading and Staging of Oral Carcinoma 357
- Epidemiology of OSCC in Indian Subcontinent 357
- Clinical Features 358
- Diagnosis of Oral Squamous Cell Carcinoma 363
- Differential Diagnosis 363
- Management 364
- Metastatic Carcinoma 364
- Treatment Modalities of Oral Cancer 365

Section 5: Infectious Diseases of Oral Cavity 371

Chapter 16: Bacterial Infections....373
Anil Ghom, Aena Jain (Pundir)

Granulomatous Infections 373

- Syphilis 373
- Nonvenereal Treponematoses 377
- Tuberculosis 379
- Leprosy (Hansen Disease) 381
- Actinomycosis 384
- Rhinoscleroma 386
- Granuloma Inguinale 386
- Lymphogranuloma Venereum 387

Specific Gingival and Oropharyngeal Infections 387

- Gonorrhea 387
- Streptococcal Tonsillitis and Pharyngitis 388
- Noma 389
- Scarlet Fever 390
- Diphtheria 390
- Impetigo 391
- Erysipelas 391

Lymph Node Associated Infections 392

- Cat Scratch Disease 392
- Tularemia 393
- Tetanus 393

Chapter 17: Viral Infections....395
Anil Ghom, Savita Ghom, Vivek Lath

- Classification 395
- Infections Caused by Viruses 396
- Herpes Virus-induced lesions 396
- Paramyxoviridae 408
- Rubella (German Measles) 409
- Coxsackievirus Infection (Enteroviruses) 410
- Poxviridae 411
- Chikungunya 411
- Coronaviruses 412

Chapter 18: Fungal and Parasitic Infections 413
Savita Ghom, Sanvil Ghom

Fungal Infections of Orofacial Region 413

- Classification 413
- Superficial Mycoses 413
- Aspergillosis 413
- Histoplasmosis 415
- Blastomycosis 416
- Mucormycosis 418
- Cryptococcosis 419
- Geotrichosis 419
- Paracoccidioidomycosis 420
- Rhinosporidiosis 420
- Coccidioidomycosis 421
- Sporotrichosis 421
- COVID-19 Associated Oral Mycoses 422
- Medical management of oral fungal infections 422

Parasitic Infections Affecting Orofacial Region 422

- Leishmaniasis 423
- Trichinosis 423
- Myiasis 423
- Lagochilascariasis 424
- Entamoeba Gingivalis Infection 425
- Trichomonas Tenax Infection 425
- Gongylonema Pulchrum Infection 425
- Hydatid Disease of the Oral Cavity 425
- Cysticercosis 425
- Dirofilariasis 425
- Filarial Infection of the Oral Cavity 425

Chapter 19: Acquired Immunodeficiency Syndrome and Oral Lesions....426
Anil Ghom, Milini Ghom

- Definition 426
- Prevalence and Epidemiology 426
- Transmission of HIV Infections 427
- Pathogenesis of AIDS 427
- Staging of HIV/AIDS 429
- Oral Manifestations of HIV 429
- Diagnostic Test for AIDS 437
- Management of AIDS 438
- Prophylaxis for occupational exposure 440
- Prevention of HIV Transmission 441

Section 6: Diseases of Oral Structures 443

Chapter 20: Diseases of Lip....445
Anil Ghom, Manjari Gupta (Gandhi)

- Classification of Lip Disorders 445

Developmental Disorders of Lip 445

- Congenital Lip Pits 446
- Commissural Lip Pits 446
- Double Lip 446
- Cleft Lip and Cleft Palate 447

Cheilitis 451
- Glandular Cheilitis 451
- Granulomatous Cheilitis or Orofacial Granulomatosis 451
- Angular Cheilitis 452
- Eczematous Cheilitis 453
- Contact Cheilitis 453
- Actinic Cheilitis 454
- Exfoliative Cheilitis 455
- Plasma Cell Cheilitis 456
- Drug-induced Cheilitis 456

Other Disorders of Lips 457
- Carcinoma of Lip 457
- Chapped Lips 458
- Actinic Elastosis 458
- Lip Ulcers Due to Persistent Caliber Artery 458

Chapter 21: Diseases of Tongue.....459
Anil Ghom, Manjari Gupta (Gandhi)
- Examination of Tongue 460
- Classification of Tongue Disorders 460

Congenital and Developmental Disorders 460
- Aglossia and Microglossia 460
- Macroglossia 460
- Ankyloglossia 463
- Cleft Tongue 463
- Ankyloglossum Superius Syndrome 464
- Lingual Varices 464
- Lingual Thyroid Nodule 464
- Variations in Tongue Movement 465
- Tongue Thrusting 465
- Patent Thyroglossal Duct Cyst 465
- Reactive Lymphoid Aggregate 466
- Lingual Cyst 466

Disorders of Lingual Mucosa 466
- Fissured Tongue 466
- Median Rhomboid Glossitis 467
- Benign Migratory Glossitis 468
- Hairy Tongue 469
- Crenated Tongue 470
- Foliate Papillitis 470
- Leukokeratosis Nicotina Glossi 470
- Pigmentation of Tongue 471

Papillary Disorders of the Tongue 471
- Depapillation 471

Neurological Disorders 473
- Glossodynia 473
- Dyskinesia 474
- Paralysis 475
- Oropharyngeal Dysphagia 475
- Dysgeusia and Hypogeusia 475
- Sleep Apnea 476

Premalignant Lesions and Conditions 476
- Leukoplakia 476

Neoplastic Conditions of Tongue 477
- Squamous Cell Carcinoma 477

Chapter 22: Traumatic Injuries of Orofacial Region...480
Anil Ghom, Nupoor Deshpande (Gupta), Manan Gupta

Orofacial Soft Tissue Injuries 480
- Traumatic Erythematous Macule or Hematoma 480
- Ecchymosis and Purpuric Macule (Early Stage) 481
- Cervicofacial Emphysema 481
- Factitious Injury 482

Traumatic Injuries to the Hard Tissue 482
- Traumatic Injury to Teeth/Dental Trauma 482
- Fracture of Teeth 486
- Traumatic Injury to Facial Bones 489
- Iatrogenic injury in Head and Neck Region 501
- Complications of Fracture Healing 501
- Dry Socket 502

Chapter 23: Soft Tissue Calcifications.....503
Anil Ghom, Savita Ghom, Ram Tiwari
- Classification of Soft Tissue Calcification 503

Dystrophic Calcification 503
- General Dystrophic Calcification of the Oral Region 504
- Calcified Lymph Nodes 504
- Tonsillolithiasis 504
- Cysticercosis 505
- Calcified Carotid Artery 506

Idiopathic Calcification 507
- Sialoliths 507
- Phleboliths 507
- Rhinolith 507

Metastatic Calcification 508
- Ossification of the Stylohyoid Ligament 508
- Osteoma Cutis 509
- Myositis Ossificans 509

Chapter 24: Disorders of Maxillary Sinus.....512
Savita Ghom, Raj Diwan
- Examination and Investigations of Maxillary Sinus 512
- Classification of Maxillary Sinus Disorders 514
- Developmental Disorders 514

Inflammatory Disorders 514
- Mucositis 514
- Maxillary Sinusitis 514

Cysts of Maxillary Sinus 518
- Non-odontogenic Cysts 518
- Odontogenic Cysts 520

Tumors of Maxillary Sinus 521
- Antral Polyp 521
- Antral Papilloma 522
- Osteoma 522
- Ameloblastoma 522
- Squamous Cell Carcinoma 523
- Invasion of the Maxillary Sinus by Local Malignant Disease 524
- Metastatic Carcinoma of the Maxillary Sinus 525

Traumatic Injuries to the Paranasal Sinuses 525
- Root in Antrum 525
- Foreign Bodies 525
- Sinus Contusion 526
- Blow-out Fracture 526
- Isolated Fracture 526
- Zygomatic Complex Fracture 526
- Fractured Tuberosity 526
- Oroantral Fistula 526
- **Calcifications in Maxillary Sinus 529**
- Antroliths 529

Chapter 25: Disorders of Temporomandibular Joint....530
Anil Ghom, Savita Ghom, Rituraj Kesri
- Anatomy of TMJ 530
- Classification of TMJ Disorders 530

Developmental Disorders of the TMJ 533
- Hypoplasia of Condyle 533
- Agenesis of the Condyle 534
- Hyperplasia of the Condyle 535
- Double Condyle or Bifid Condyle 536
- Coronoid Hyperplasia 537

Degenerative Joint Diseases 537
- Osteoarthritis 537

Inflammatory Disorders of the Joint 540
- Rheumatoid Arthritis 540
- Juvenile Rheumatoid Arthritis 542
- Psoriatic Arthritis 542
- Infective Arthritis 543
- Ankylosing Spondylitis 544

Traumatic Disorders of TMJ 545
- Condylar Fracture 545
- Ankylosis 547
- Dislocation 550
- Subluxation (Hypermobility) 552
- Internal Derangement or Disc Displacement 552
- Adhesions 556

Metabolic Disorders 556
- Gout 556

Neoplastic Disorders 557
- Benign Tumors 557
- Malignant Tumors 557

Miscellaneous Disorders 558
- Synovial Chondromatosis 558
- Drug-induced Disorders (Steroids) 559
- TMJ Dysfunction Syndrome or Myofascial Pain Dysfunction Syndrome 559

Chapter 26: Disorders of Salivary Gland....564
Anil Ghom, Praveen Lambade, Dipti Lambade
- Clinical Anatomy of Salivary Glands 564
- Saliva 566
- Examination of Salivary Glands 567
- Diagnostic Tests of the Salivary Glands 568
- Classification of Salivary Gland Disorders 569

Developmental Disorders of Salivary Gland 570
- Aberrancy/Ectopic Salivary Gland 570
- Aplasia and Hypoplasia 570
- Hyperplasia of Salivary Gland 571
- Atresia 571
- Accessory Duct 571
- Diverticuli 571
- Congenital Fistula 571
- Developmental Salivary Gland Defect 572

Functional Disorders of Salivary Gland 572
- Sialorrhea (Ptyalism) 572
- Xerostomia 573

Obstructive Disorders 575
- Sialolithiasis (Salivary Gland Stone or Salivary Gland Calculus) 575
- Mucus Plugs 577
- Strictures and Stenoses 578
- Other Obstructive Conditions 579

Cysts of Salivary Gland 579
- Mucocele 579
- Ranula 580
- Salivary Duct Cyst 581

Asymptomatic Enlargement of the Salivary Gland 582
- Sialosis (Sialadenosis) 582
- Allergic Sialadenitis 582

Infectious Conditions of Salivary Glands 583
- Viral Infections 583
- Bacterial Infections 584

Autoimmune Disorders 586
- Sjögren's Syndrome 586
- Mikulicz's Disease or Benign Lymphoepithelial Lesion 589
- Uveoparotid Fever 590
- Recurrent Nonspecific Parotitis 591

Non-neoplastic Conditions 591
- Necrotizing Sialometaplasia 591
- Nodular Oncocytic Hyperplasia 592
- Lymphoepithelial Sialadenitis 592

Salivary Gland Tumors 593
- Pathophysiology of Salivary Gland Tumors 593
- Clinical Staging of Salivary Gland Tumors 593
- Benign Tumors 593
- Malignant Tumors 600

Chapter 27: Fibro-osseous and Bone Diseases of Maxillofacial Region....605
Anil Ghom, Savita Ghom

Fibro-osseous Conditions 605
- Classification of Fibro-osseous Lesions 605
- Fibrous Dysplasia 606
- Paget's Disease 610
- Segmental Odontomaxillary Dysplasia 613
- Periapical Cemento-osseous Dysplasia 613
- Focal Cemento-osseous Dysplasia 615
- Florid Osseous Dysplasia 615

- Familial Florid Cemento-osseous Dysplasia 616
- Familial Gigantiform Cementoma 616
- Juvenile Ossifying Fibroma 616
- Cherubism 618
- Central Giant Cell Granuloma 620
- Peripheral Ossifying Fibroma 622
- Simple Bone Cavity 623
- Aneurysmal Bone Cyst 625

Diseases of Bone Manifested in Jaw 625
- Osteoporosis 625
- Infantile Cortical Hyperostosis 627
- Osteopetrosis 628
- Osteogenesis Imperfecta 630
- Pierre-Robin Syndrome 631
- Marfan's Syndrome 632
- Down's Syndrome 633
- Achondroplasia 634
- Skeletal Fluorosis (Fluoride Toxicity) 635
- Generalized Cortical Hyperostosis 636
- Massive Osteolysis 636

Section 7: Mucocutaneous Diseases 639

Chapter 28: Red and White Lesions of Oral Mucosa....641
Anil Ghom, Deeplaxmi Dewangan

Normal Variations of Oral Mucosa 641
- Leukoedema 641
- Fordyce's Granules 643
- Linea Alba 643

Oral Genodermatoses 643
- White Sponge Nevus 643
- Hereditary Benign Intraepithelial Dyskeratosis 644
- Pachyonychia Congenita 645
- Porokeratosis 646
- Keratosis Follicularis 646
- Warty Dyskeratoma 647
- Pseudoxanthoma Elasticum 648
- Hyalinosis Cutis et Mucosa Oris 648

Non-keratotic White Lesions 648
- Chronic Cheek or Lip Biting 648
- Burns 649
- Radiation Mucositis 651
- Uremic Stomatitis 652
- Candidiasis 652
- Traumatic Keratosis 658

Oral Potentially Malignant Disorders 658
- Leukoplakia 658
- Erythroplakia 666
- Carcinoma in Situ 668
- Actinic Keratosis 669
- Lichen Planus 669
- Erosive Lichen Planus 674
- Lichenoid Reaction 676
- Graft-versus-Host Disease (GVHD) 677
- Oral Submucous Fibrosis (OSMF) 678
- Dyskeratosis Congenita 684
- Lupus Erythematosus 685

Oral Lesions Associated with Use of Tobacco 687
- Stomatitis Nicotina 687
- Snuff Dipper Lesion 688
- Cigarette Smoker's Lip Lesion 689

Chapter 29: Oral Pigmentations.....690
Savita Ghom, Anil Ghom
- Classification of Oral Pigments 690
- Pathogenesis of Oral Pigmentation 691

Endogenous Pigmentations 692
- Melanoplakia 692
- Ephelis (Freckles) 693
- Melanotic Macule 693
- Smoker's Melanosis 694
- Blue Nevus 695
- Pigmented Nevus 695
- Melanoma 696
- Melanotic Neuroectodermal Tumor of Infancy 698
- Drug-induced Pigmentation 698
- Addison's Disease 698
- Peutz-Jegher's Syndrome 700
- Neurofibromatosis 700
- Albright's Syndrome 701
- Varix and Varices 701

Exogenous Pigmentations 702
- Accidental Pigmentation 702
- Iatrogenic Pigmentation 702

Pigmentation due to Drugs and Metals 703
- Bismuthism 703
- Plumbism or Lead Poisoning 703
- Mercurialism 704
- Argyria or Silver Poisoning 705
- Arsenism 705
- Auric Stomatitis or Gold Poisoning 706

Hypopigmentation in Oral Cavity 706
- Albinism 706
- Vitiligo 707

Chapter 30: Vesiculobullous and Ulcerative Lesions of Oral Cavity.....708
Anil Ghom, Nitin Vaidya, Vidya Vaidya
- Classification 708

Autoimmune Vesiculobullous Disorders 710
- Pemphigus 710
- Bullous Pemphigoid 716
- Benign Mucous Membrane Pemphigoid 717
- Familial Benign Chronic Pemphigus 719
- Dermatitis Herpetiformis (Duhring–Brocq Disease) 719
- Linear IgA Disease 720
- Epidermolysis Bullosa 720
- Angina Bullosa Hemorrhagica 722

Immune Complex/Hypersensitivity Reactions 722
- Drug Allergy 722
- Contact Allergy 724

- Angioedema 725
- Erythema Multiforme 725
- Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis 728
- Plasma Cell Stomatitis 730

Recurrent Immune Mediated Conditions 731
- Aphthous Stomatitis 731
- Behçet's Disease 735

Ulcers Associated with Trauma 735
- Traumatic Ulcer 735
- Anesthetic Necrosis 736
- Oral Trauma from Sexual Practice 737

Section 8: Systemic and Metabolic Conditions 739

Chapter 31: Cardiovascular Diseases 741
Anil Ghom, Savita Ghom, Milini Ghom
- General Examination of CVS 741
- Angina Pectoris 742
- Myocardial Infarction 744
- Rheumatic Heart Disease and Fever 745
- Hypertension 746
- Infective Bacterial Endocarditis 748
- Congestive Cardiac Failure 749
- Congenital Heart Disease 749
- General Considerations for Dental Management of Cardiac Patients 750

Chapter 32: Respiratory Diseases 751
Anil Ghom, Savita Ghom, Sanvil Ghom
- Bronchial Asthma 751
- Chronic Obstructive Pulmonary Disease 753
- Upper Respiratory Tract Infection 754
- Allergic Rhino-conjunctivitis 755
- Otitis Media 755
- Pneumonia 756
- Cystic Fibrosis 756
- Pulmonary Embolism 756

Chapter 33: Endocrine Diseases 758
Anil Ghom, Savita Ghom, Pinakinarayan Moitra
- Hypothalamus-pituitary-adrenal (HPA) Axis 758

Diseases of Pituitary Gland 758
- Hyperpituitarism 758
- Hypopituitarism or Pituitary Dwarfism 760
- Progeria 761

Diseases of Thyroid Gland 762
- Hyperthyroidism 762
- Hypothyroidism 763

Diseases of Parathyroid Gland 764
- Hyperparathyroidism 764
- Hypoparathyroidism 767
- Pseudohypoparathyroidism 768

Diseases of Pancreatic Gland 769
- Diabetes Mellitus 769
- Diabetic Insipidus 773

Diseases of Adrenal Gland 774
- Addison's Disease 774
- Adrenogenital Syndrome 774
- Cushing's Syndrome 775
- Adrenal Insufficiency 776
- Conn' Syndrome 777

Diseases of Gonads 777
- Hypergonadism 777
- Hypogonadism 777
- Pregnancy 777
- Menopause 778

Chapter 34: Renal Diseases 780
Anil Ghom, Savita Ghom, Sanvil Ghom
- Renal Failure 780
- Renal Osteodystrophy 781
- Kidney Transplantation 782

Chapter 35: Gastrointestinal Diseases 785
Anil Ghom, Savita Ghom, Sanvil Ghom
- Hepatitis 785
- Inflammatory Bowel Disease 787
- Jaundice 788
- Peptic Ulceration 789
- Gastroesophageal Reflux Disease 790
- Hiatal Hernia 790
- Liver Cirrhosis 790
- Gastrointestinal Syndromes 791

Chapter 36: Blood Disorders 792
Anil Ghom, Milini Ghom, Rituraj Kesri

Diseases of Red Blood Cells 792
- Anemia 792
- Polycythemia 808

White Blood Cell Disorders 810
- Quantitative Disorders 810
- Qualitative Disorders of WBC 813

Platelet Disorders 814
- Thrombocytopenic Purpura 814
- Von Willebrand's Disease 817
- Aldrich Syndrome 818
- Familial Thrombasthenia 819
- Onyalai 819
- Thrombocytosis or Thrombocythemia 819

Hemorrhagic Disorders 820
- Hemophilia 820
- Factor V Deficiency or Parahemophilia 821
- Hypofibrinogenemia 822
- Macroglobulinemia 822
- Hereditary Hemorrhagic Telangiectasia 823
- Rare Hemorrhagic Disorders 823

Chapter 37: Collagen and Granulomatous Disorders 825
Anil Ghom, Savita Ghom, Pankaj Agrawal

Collagen Disorders 825
- Scleroderma or Systemic Sclerosis 825
- Kawasaki Disease 827

Granulomatous Disorders 828
- Wegener's Granulomatosis or Granulomatosis with Polyangiitis 828
- Sarcoidosis 829
- Midline Lethal Granuloma 830
- Chronic Granulomatous Disease 831

Chapter 38: Neurological and Muscular Disorders 832
Anil Ghom, Vidya Vaidya
- Auriculotemporal Syndrome 832
- Bell's Palsy 833
- Motor System Disease 834
- Multiple Sclerosis 835
- Alzheimer Disease 836
- Cerebral Palsy 836
- Epilepsy 837
- Parkinson's Disease 838
- Orofacial Dyskinesia 839
- Muscular Dystrophy 840
- Myotonias 840
- Hemifacial Spasm 841
- Paramyotonia 841
- Myasthenia Gravis 842
- Dermatomyositis 842
- Moebius Syndrome 843
- Meige's Syndrome (Oromandibular Dystonia) 845

Chapter 39: Dermatological Diseases 847
Savita Ghom, Nitin Vaidya
- Pityriasis Rosea 847
- Incontinentia Pigmenti 847
- Acanthosis Nigricans 848
- Ehlers-Danlos Syndrome 849
- Tuberous Sclerosis 849
- Seborrheic Keratosis 850
- Sebaceous Hyperplasia 850
- Actinic Lentigo 851
- Melasma 851
- Xeroderma Pigmentosum 851
- Clouston Syndrome 851

Chapter 40: Nutritional and Metabolic Disorders ... 853
Anil Ghom, Arkaprava Banerjee, Vaibhav Sharma
Vitamins 853
- B-complex Vitamins 853
- Vitamin C 861
- Choline 863
- Inositol 863
- Vitamin A (Retinol) 863
- Vitamin D 865
- Vitamin E (Tocopherol) 868
- Vitamin K (Phylloquinone) 868

Metabolic Disorders 869
- Disturbances in Protein Metabolism 869
- Porphyria 871
- Disturbances in Lipid Metabolism 872
- Disturbances in Carbohydrate Metabolism 876
- Disturbances in Mineral Metabolism 877
- Miscellaneous Disorders 880

Section 9: Miscellaneous Conditions 883

Chapter 41: Orofacial Pain 885
Savita Ghom, Ajit Mishra
- Definition 885
- Types and Nature of Pain 886
- Classification of Orofacial Pain 887
- Factors Affecting Pain 887
- Measurement of Pain Intensity 888
- Types of Pain 888
- Pains of Dental Origin 889
- Musculoskeletal Pain 890
- Temporomandibular Joint Pain 891
- Atypical Facial Pain 891
- Neurogenic Pain 892
- Therapeutic Modalities for Pain Control 898

Chapter 42: Medical Emergencies in Dental Practice 904
Anil Ghom, Praveen Lambade
- Drug Allergy 904
- Syncope 905
- Shock 906
- Basic Life Support 907

Chapter 43: Psychological Disorders 909
Savita Ghom, Milini Ghom
- Background 909
- Etiology and Pathogenesis 909
- Classification of Psychosomatic Oral Disorders 910
- Common Oral Diseases Associated with Psychological Factors 911
- Mental Health Disorders and their Oral Manifestations 912
- Medically Unexplained Oral Symptoms 912
- Management Principles 913
- Management of Common Oral Manifestations of Psychosomatic Disorders 913
- Future Research Direction to Improve Oral Health in Patients with Psychosomatic Disorders 913

Chapter 44: Geriatric Dentistry 915
Shalu Rai
- Classification 915
- Age Changes Affecting the Structures in Oral Cavity 915
- Diseases Common Among Geriatric Patients 916
- Goals of Oral Physicians 917
- Medical Conditions in Older Adults 917
- Other Dental Treatment Considerations 919
- Conclusion 921

Chapter 45: Forensic Dentistry.....922
Anil Ghom, Savita Ghom
- Definition 922
- Scope of Forensic Odontology 922
- Identification 924
- Palatal Rugae Pattern (Rugoscopy) 926
- Age Assessment in Forensic Dentistry 926
- Bite Marks 927
- Cheiloscopy 929
- Classification of Skin Wounds 931
- Various Laws Applicable in Dental Practice 931

Index **933**

7

CHAPTER

Pulp and Periapical Infections

Savita Ghom, Anil Ghom

Chapter Outline

Pulpal Pathologies
- ❖ Reversible Pulpitis
- ❖ Irreversible Pulpitis (Acute and Chronic)
- ❖ Chronic Hyperplastic Pulpitis

Periapical Pathologies
- ❖ Acute Apical Periodontitis
- ❖ Periapical Abscess
- ❖ Acute Exacerbation of a Chronic Lesion
- ❖ Pericoronal Abscess
- ❖ Periapical Granuloma
- ❖ Periapical Scar

Bone Infections
- ❖ Osteomyelitis
- ❖ SAPHO Syndrome

Dental caries when left untreated serves as the primary gateway for oral bacteria to penetrate the dental pulp. If pulp-dentin complex defensive mechanisms fails to wall off the advancing infection, the bacterial biofilm advances toward the pulp chamber triggering a localized inflammatory response known as *pulpitis*.

In its early stages, the inflammation may be classified as *reversible pulpitis* where the pulp remains vital and can heal if the irritant is removed. If inflammation persists, the intrapulpal pressure rises leading to *irreversible pulpitis*, which is characterized by severe tissue damage and eventual liquefaction necrosis of pulp having predominantly anaerobic polymicrobial biofilm.

The migration of this beyond the apical foramen triggers a periapical inflammatory response resulting in symptomatic apical periodontitis and further to periapical abscesses, granulomas, or radicular cysts. In severe cases, the infection may disseminate through the alveolar bone into adjacent soft tissues, potentially leading to cellulitis or osteomyelitis.

Understanding the sequelae of dental caries from a localized carious lesion to a spreading periapical infection is fundamental for effective diagnosis and treatment planning.

The sequalae of dental caries is given in **Figure 7.1**.

- *Pulpitis*—it is due to irritating inflammatory products. Various factor like host resistance, number and virulence of bacteria, amount of antigenic material, and degree of tooth function and extent of other trauma also play a vital role.
- *Local inflammatory response*—the inflammatory products escape from pulp canal and initiate local inflammatory response. The pulp is non-vital in such cases.
- *Periapical lesion*—if infection reaches the periapical tissue then in some cases host defense effectively combats and localizes the resultant inflammation in circumscribe area.
- *Acute periapical abscess*—teeth with contaminated gangrenous pulp have large number of bacteria passing in the root canal which may be sufficient to overwhelm the defense of periapical tissues resulting in an acute periapical abscess.
- *Chronic alveolar abscess*—resultant infection may be partially controlled by body defense, by surgically induced draining or by antibiotic therapy, thus results in the development of chronic periapical periodontitis or chronic alveolar abscess.
- *Radicular cyst*—odontogenic epithelial rests of Malassez (present in PDL and periapical granuloma) proliferate and cyst may develop in response. If adequate root canal treatment is done, these will be ultimate disappearance of granuloma and complete resolution of radiolucency to normal bone.
- *Periapical scar*—if granuloma has been subjected to repeated exacerbation due to periodic contact with the irritation from root canal it may become fibrosed. If root canal treatment is done successfully, remaining inflammation and granulomatous tissue will resolve leaving only fibrosed areas behind. Such entities are referred to periapical scar.

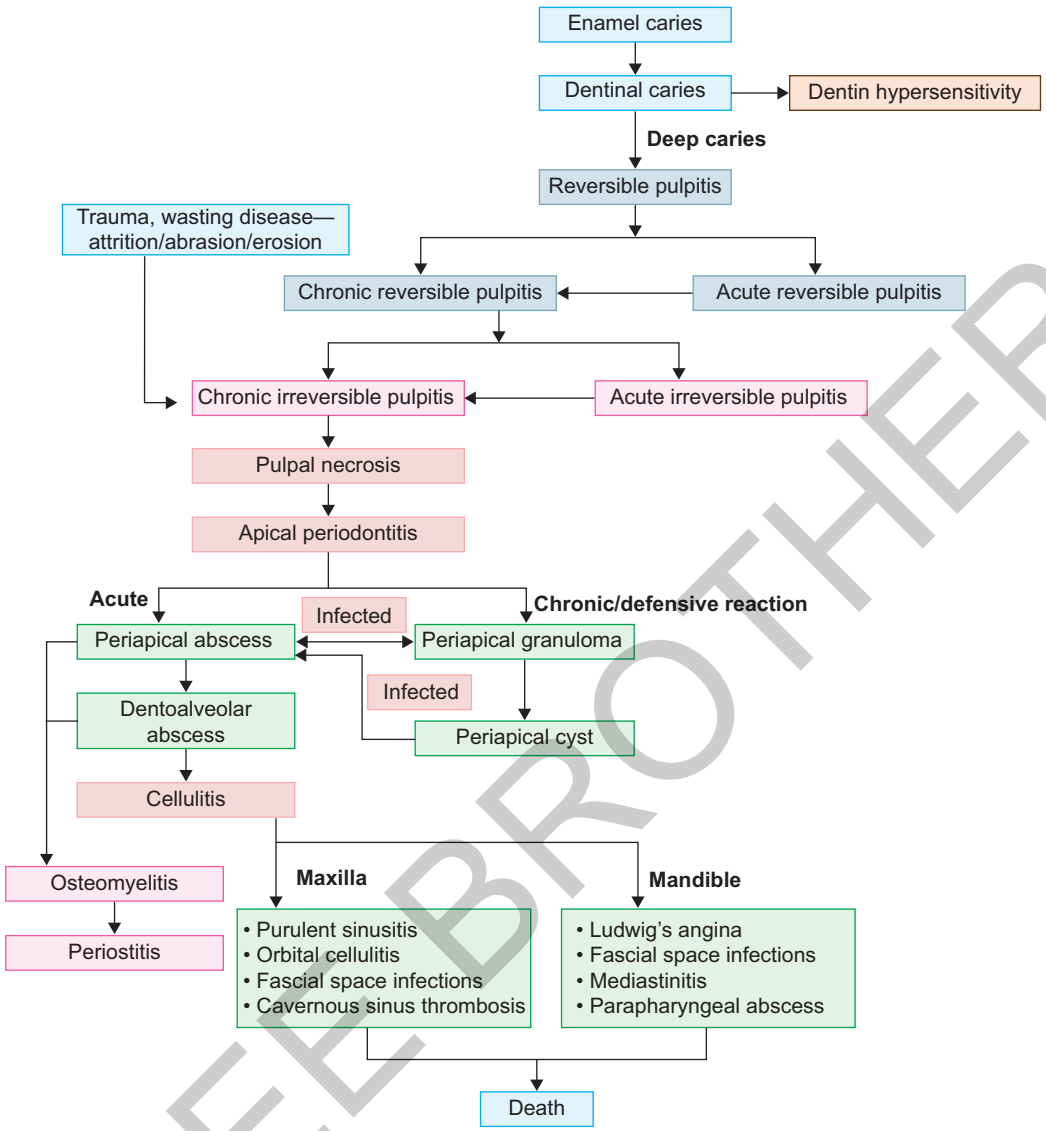


Fig. 7.1: Sequelae of dental caries involving pulp.

- *Osteomyelitis*—if the infection is not controlled there re chances of infection that may turn into osteomyelitis.

Radiographic Considerations

- *Lamina dura*—there are variations in lamina dura from person to person and region to region. Lamina dura is not continuous in case of periapical pathology.
- *Amount of bone destruction*—30–60% bone destruction required for radiographically detectable changes. Radiographic changes cannot be detected without involvement of junctional and cortical bone.
- *Root resorption*—external root resorption is caused by orthodontic movement, inflammation or infection of bone and benign and malignant tumor.

PULPAL PATHOLOGIES

REVERSIBLE PULPITIS

Reversible pulpitis is a mild-to-moderate inflammatory condition of the pulp caused by noxious stimuli, where the pulp is capable of returning to a healthy state if the stimulus is removed.

Etiology

- *Trauma*—accidental trauma, occlusal attrition, or cracked tooth syndrome.
- *Thermal/chemical shock*—thermal conduction through deep metallic restorations without a liner or acidic etching of dentin.
- *Caries*—early bacterial invasion of dentin.

Clinical Features

- *Location:* Localized to the affected tooth.
- *Symptoms:* Sharp, fleeting pain triggered by cold, sweet, or sour stimuli.
- *Signs:* Pain subsides immediately (within seconds) after removal of the stimulus. The tooth is not tender to percussion.

Radiographic Features

- *Normal periapical area*—periodontal ligament space and lamina dura appear intact.
- *Proximity*—caries or restorations may be seen close to the pulp but without frank exposure.

Diagnosis

- *Clinical diagnosis*—quick response to cold test with immediate relief.
- *Radiological diagnosis*—no periapical changes.
- *Laboratory diagnosis*—hyperemia and edema of the pulp; no signs of necrosis.

Management is conservative management by removal of the irritant (e.g., caries removal, sedative filling with zinc oxide eugenol). Pulp capping can be done and when the pulp heals, a permanent restoration is placed.

IRREVERSIBLE PULPITIS (ACUTE AND CHRONIC)

A persistent inflammatory condition where the pulp is damaged beyond the point of recovery, eventually leading to necrosis.

Acute Irreversible Pulpitis

It is characterized by severe, spontaneous, and throbbing pain that lingers significantly after the removal of a stimulus, often intensifying when the patient lies down due to increased intrapulpal pressure.

Etiology

- *Bacterial invasion*—progression of deep caries into the pulp.
- *Persistent irritation*—untreated reversible pulpitis.

Clinical Features

- *Symptoms:* Spontaneous, severe, throbbing pain. Pain is often worse when lying down due to increased intrapulpal pressure.
- *Signs:* Pain is prolonged (minutes to hours) after removal of stimulus. In late stages, heat may exacerbate pain, while cold may actually relieve it.

Radiographic Features

- *Caries-pulp exposure*—deep carious lesion or large restoration involving the pulp chamber.
- *PDL*—the periodontal ligament space may appear slightly widened in very late stages.

Diagnosis

- *Clinical diagnosis*—history of spontaneous pain and lingering response to thermal tests.
- *Radiological diagnosis*—deep caries approaching or involving the pulp.

Management—is primarily emergency endodontic management with complete pulpectomy (root canal treatment) or extraction if the tooth beyond restorable. NSAIDs are used for systemic pain relief.

Chronic Irreversible Pulpitis

Chronic irreversible pulpitis is a long-standing, low-grade inflammation of the pulp that is often asymptomatic or characterized by a dull, intermittent ache, and may manifest as a "pulp polyp" (chronic hyperplastic pulpitis) in young, highly vascular teeth.

Etiology

- *Slowly progressing caries*—allows the pulp to mount a defensive response.
- *Chronic irritation*—persistent low-grade stimulus in a young, highly vascular pulp.

Clinical Features

- *Signs:* Often asymptomatic or mild "dull ache."
- *Chronic hyperplastic pulpitis (pulp polyp):* Discussed later.

Radiographic Features

- *Large cavity*—significant destruction of the crown.
- *Periapical*—may show early signs of thickening of the PDL.

Diagnosis

- *Clinical diagnosis*—visual identification of a pulp polyp or a deep, painless cavity that bleeds easily upon probing.
- *Laboratory diagnosis*—presence of granulation tissue, chronic inflammatory cells (lymphocytes, plasma cells).

Management—is removal of the diseased pulp tissue followed by root canal treatment. Pulpotomy may be considered in specific pediatric cases with immature apices.

CHRONIC HYPERPLASTIC PULPITIS

It is also called as *'pulp polyp'* or *'pulpitis aperta'*. It is essentially an excessive, exuberant proliferation of chronically inflamed dental pulp tissue.

Etiology

- *Slow carious exposure*—slow progressive carious exposure of the pulp is the cause.

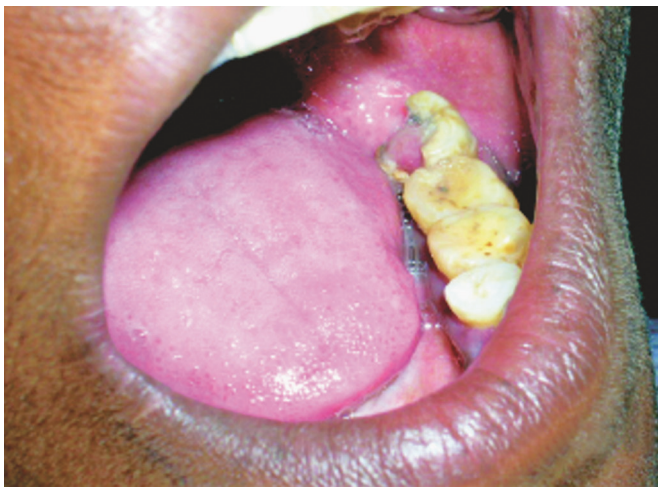


Fig. 7.2: Chronic hyperplastic pulpits presenting as reddish mass in mandibular third molar region.
(*Courtesy:* Dr Tapasya Karamore)

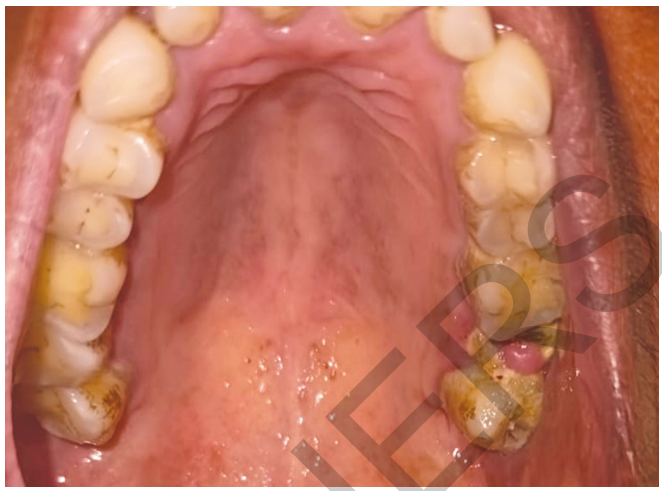


Fig. 7.3: Chronic hyperplastic pulpitis seen in maxillary second molar region.

- *Chronic low grade infection*—for the development of a hyperplastic pulp, a large open cavity, a young resistant pulp and a chronic low grade stimulus is necessary.
- *Mechanical irritation*—mechanical irritation from chewing and bacterial infection often provides the stimuli.

Clinical Features

- *Site*—teeth most commonly involved are deciduous molars and first permanent molars as they have an excellent blood supply because of a large root opening, and this coupled with high tissue resistance and reactivity in young person's accounts for unusual proliferative properties of the pulp tissue.
- *Age*—it is seen only in teeth of children and young adults.
- *Symptoms*—it is asymptomatic and there may be feeling of pressure when masticator forces are applied.
- *Appearance*—polypoid tissue appears as a fleshy, reddish pulpal mass filling most of the pulp chamber **(Figs. 7.2 and 7.3)** or cavity or even extending beyond the confines of the tooth.
- *Signs*—sometimes, mass if large enough interferes with comfortable closure of teeth. Polypoid tissue is less sensitive than normal pulp tissue and more sensitive than the gingival tissue. This tissue bleeds easily because of rich network of blood vessels.
- *Thermal testing*—the tooth may respond feebly or not at all to the thermal test.

Radiography—will show a large open cavity with direct access to the pulp chamber.

Diagnosis

Clinical diagnosis—fleshy pulpal mass in pulp chamber with negative thermal test will aid in diagnosis.

Differential Diagnosis

- *Proliferating gingivitis*—one should raise and trace the stalk of the tissue back to its origin, i.e., the pulp chamber.

Management

- *Pulp extirpation*—elimination of polypoid tissue, followed by extirpation of the pulp. After removing the hyperplastic tissue, bleeding can be controlled by pressure.
- *Extraction*—extraction of tooth can also be done.

PERIAPICAL PATHOLOGIES

ACUTE APICAL PERIODONTITIS

When the inflammatory degradation products from infected pulp penetrate apically to periodontal ligament in sufficient amount, inflammation is initiated. In this condition there is no frank abscess formation present. This will help to differentiated it with acute periapical abscess.

Etiology

- *Trauma*—it may occur due to occlusal trauma caused by abnormal occlusal contact or by recently inserted restoration beyond the occlusal plane.
- *Wedging*—wedging of foreign object between the tooth such as toothpicks, food or a sliver of rubber dam left by teeth can cause inflammation.

Clinical Features

- *Location*—inflammation is restricted to periodontal ligament.
- *Symptoms*—pain is of throbbing type.
- *Signs*—tooth is non-sensitive to hot, cold, sweet or sour food. Due to apical edema, tooth is elevated in the socket. It is very tender to pressure and percussion.

Radiographic Features

PDL space widening—widening of periodontal ligament space is caused by edema **(Fig. 7.4)**.

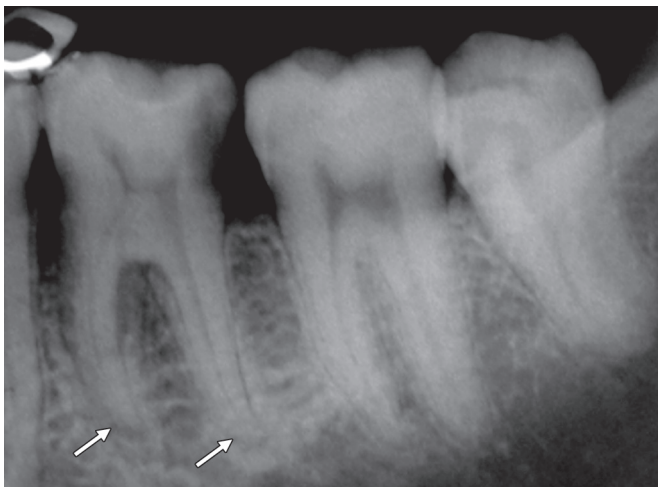


Fig. 7.4: Acute apical periodontitis seen with first mandibular molar.
(*Courtesy:* Dr Ashok L)

Diagnosis

- *Clinical diagnosis*—tender tooth without abscess formation and secondary to trauma will give clue to diagnosis
- *Radiological diagnosis*—periodontal ligament (PDL) space widening is present.
- *Laboratory diagnosis*—an inflammatory reaction occurs in the apical periodontal ligament. The blood vessels are dilated. Polymorphonuclear leukocytes are present and an accumulation of serous exudate distends the periodontal ligament and extrudes the tooth slightly.

Management

Conservative management—determine the cause and relieving the symptoms. When the acute phase is subsided, the tooth is treated by conservative means. NSAIDS are used for relief of pain and can be given as intracanal management. Trephination through bone may be required.

PERIAPICAL ABSCESS

An abscess is a localized collection of pus, surrounded by an area of inflamed tissue in which hyperemia and infiltration of leukocytes is marked. Abscess can be caused by trauma, chemical or mechanical irritation. Due to trauma, there is bacterial invasion of dead pulp.

Types

- *Acute or symptomatic*—it is localized collection of pus producing symptoms of severe pain in the tooth.
- *Chronic or asymptomatic*—it is a long standing, low grade infection of the periradicular tissues. Symptoms of chronic infection are less severe.

Pathogenesis

- *Microorganism*—*Streptococci viridans* and staphylococci are frequently associated with abscess formation. They produce the enzyme called coagulase which causes fibrin deposition and thus helps in walling off the lesion. Coagulase also promotes virulence by inhibiting phagocytosis.
- *Bacterial invasion*—bacterial invasion of pulp canal takes place. Canal contains large number of virulent bacteria that rapidly spread to periapical tissues, causing acute periodontitis, tender tooth and alveolar swelling.
- *Necrosis of periapical tissue*—when inflammatory response may extend into adjacent periapical alveolar bone, it will initiate necrosis of periapical tissue.
- *Abscess formation*—necroses tissue coalesces and enlarges, compressing the surrounding fibrous connective tissue. Thus an abscess is generated, which is a collection of pus surrounded by a wall of fibrous connective tissue.
- *Progress of abscess*—enlarging dentoalveolar abscess contains purulent material that is under pressure due to the production of pus. The purulent material travels along path of least resistance, until it reaches the surface, where due to limitation of periosteal layer, it temporally forms subperiosteal abscess. Eventually, it erodes through the periosteum and penetrates the soft tissue, again, following the path of least resistance. Path of least resistance is determined by the location of breakthrough in the bone and the anatomy of muscles and fascia plane in the area.
- *Sinus formation*—well circumscribed lesion may form sinus due to:
  - Inability of the body to completely contain or localized the causative organisms.
  - Increase in number of causative organisms.
  - Lowering of patient's general resistance.
  - Trauma or surgical intervention.

Pathogenesis is summarized in **Figure 7.5**.

Clinical Features

Acute Periapical Abscess

- *Symptoms*—pain is severe and of throbbing type. Periapical abscess may confine to osseous structures and during the early period of abscess formation, may cause excruciating pain without observable swelling. The patient may appear pale, irritable and weak from pain, loss of sleep as well as from absorption of septic products. Patient may have slight fever (99–100°F).
- *Signs*—patients experience sensitivity or pressure in the affected area. Ice relives the pain and heat intensifies it. Aspiration yields yellowish pus.
- *Positive tenderness*—the tooth becomes more painful, appears elongated and mobile. In acute periapical infection, tooth is sensitive to percussion and movement.

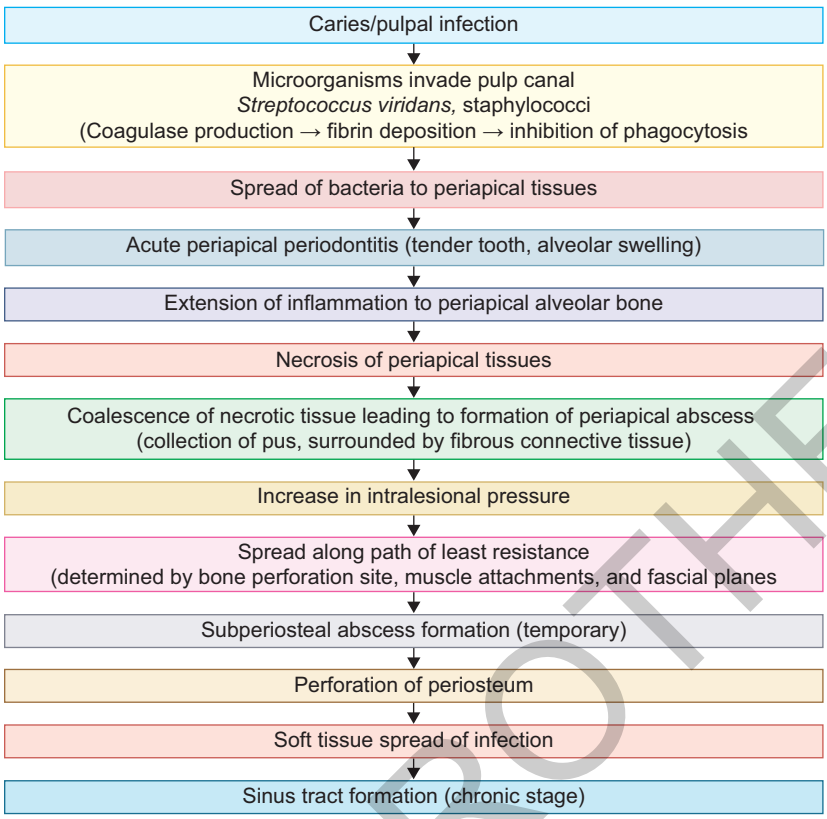
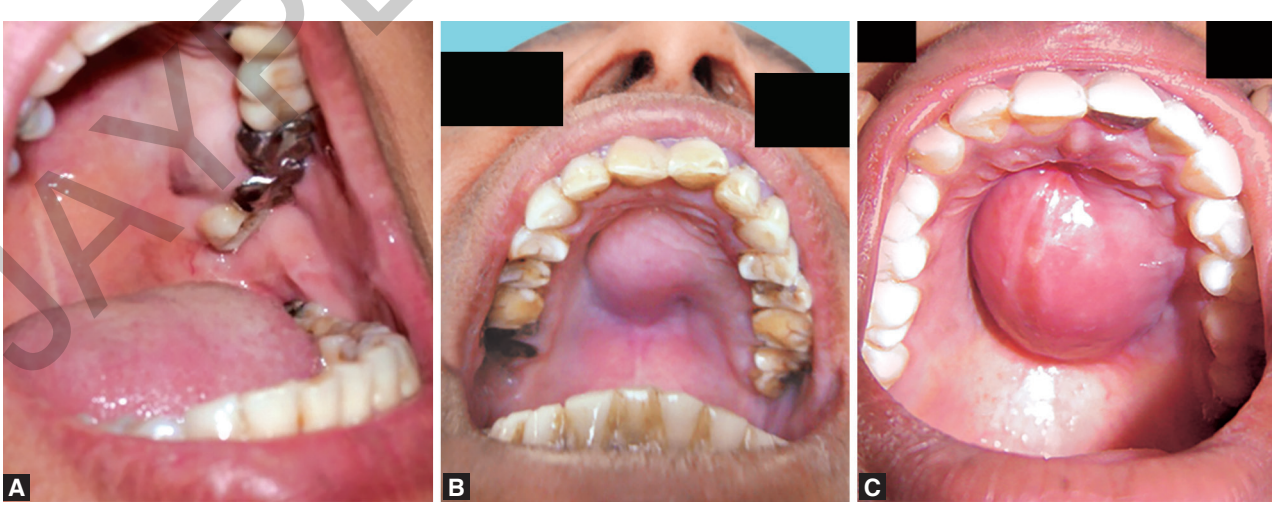


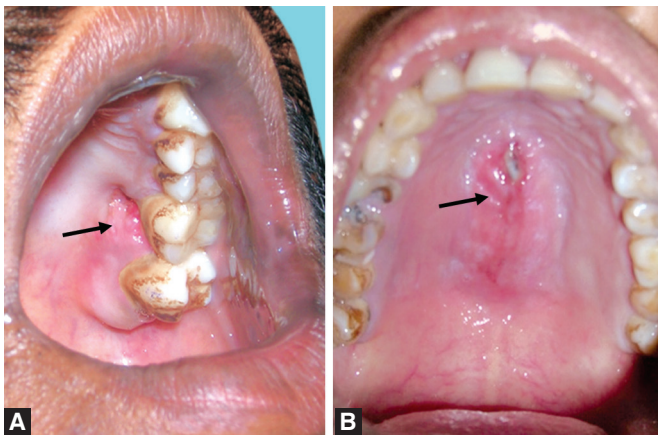
Fig. 7.5: Pathogenesis of periapical abscess.

- *Lymph node involvement*—there is also painful lymphadenopathy.
- *Swelling*—is usually seen in tissues adjacent to the affected tooth **(Figs. 7.6A to C)**. The tissues at the surface of swelling appear to be taut and inflamed. The surface of tissue become distended from the pressure of underlying pus and finally ruptures due to pressure and lack of resistance caused by continued liquefaction.
- *Maxillary involvement*—when the maxillary anterior teeth are involved, swelling of upper lip may extend to one or both eyelids. When the maxillary teeth are involved, the cheek may swell to an immense size, distorting the patient's face. When the maxillary posterior teeth are involved, there is possibility of maxillary sinus involvement.
- *Mandibular involvement*—when the mandibular posterior teeth are involved, swelling of the cheek may extend to ear.



Figs. 7.6A to C: (A) Periapical abscess showing swelling on palatal surface; (B and C) Swelling involving palatal surface and extending into mid-palatal region.

(*Courtesy:* Dr Bhaskar Patle)



Figs. 7.7A and B: (A) Intraoral sinus formation seen in case of chronic periapical infection in maxilla; (B) With ulceration.

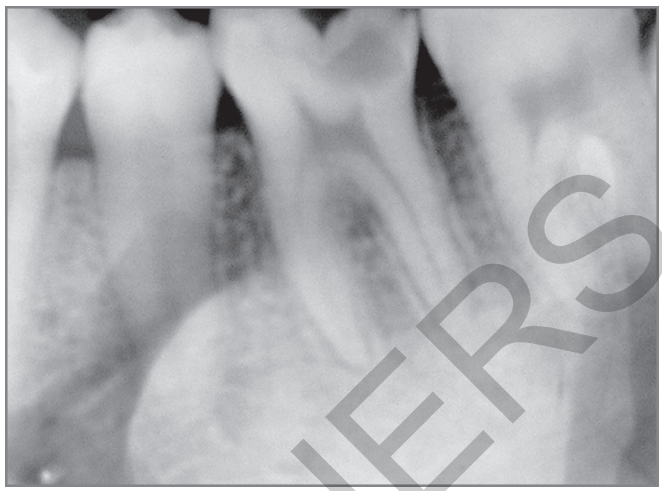


Fig. 7.9: PDL space widening seen in acute periapical infection.

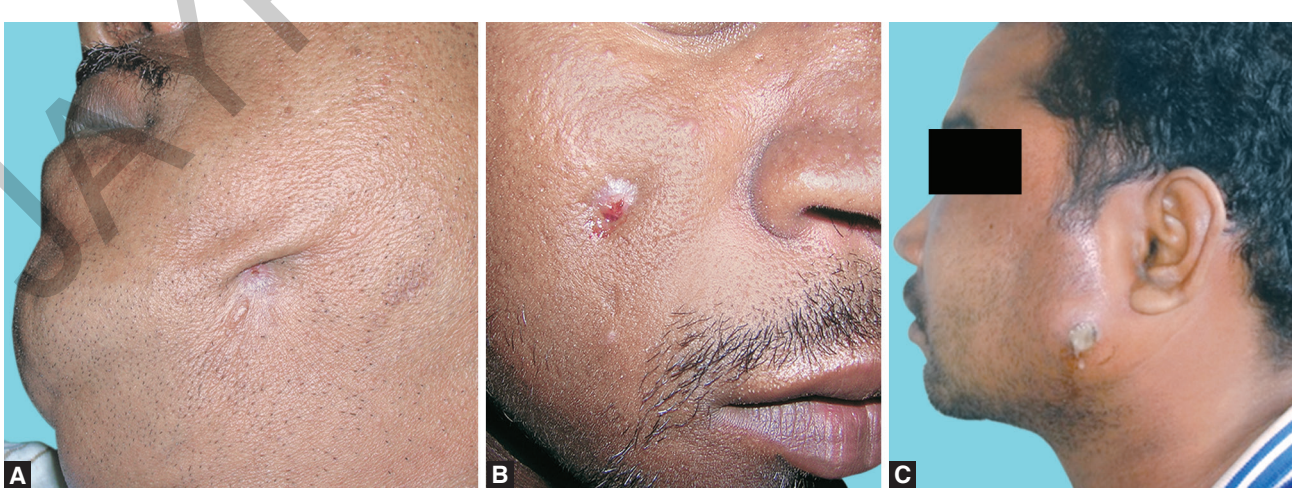
Chronic Periapical Abscess

- *History*—pain that started as dull ache and progressed to severe throbbing type. Sudden decrease in pain signals the formation of sinus.
- *Signs*—vitality test is negative with involved tooth.
- *Sinus formation*—sinus opening appears as a small ulcer **(Figs. 7.7A and B)**, rough and may bleed easily. Occasionally, after temporary emphysema, sinus heals and form slightly raised pale papule. As the pus accumulates, another sinus formation may take place eventually.
- *Location of draining fistulas or sinus*—these are also commonly associated with chronic alveolar abscess. Majority of sinus or fistula opens on labial and buccal aspect of alveolus, as apices of both maxillary and mandibular teeth are located nearer to the buccal than the lingual cortical plate. In maxilla, roots of lateral incisors and molars are close to palatal cortical plate, so sinus can appear there. Most root tips lie below the mylohyoid muscle, so pus drains into the submandibular space.
- *Parulis*—at the opening of sinus, there is mass of inflamed granulation tissue.
- *Cutaneous sinus*—in some cases, dental abscess may drain into overlying skins and drain via cutaneous sinus **(Figs. 7.8A to C)**.
- *Lymph nodes*—the patients will demonstrate lymphadenopathy as well.
- *Turner hypoplasia*—it is present in case when infection from deciduous teeth spread to the successor teeth resulting in hypoplasia of tooth.

Radiographic Features

Acute Periapical Abscess

- *Periodontal ligament space widening*—swelling of space force the tooth slightly away from its socket, creating widening of space **(Fig. 7.9)**.
- *Unsharpness at the tooth apex*—after some period, the first change seen is usually of slight unsharpness of some of the trabeculae at the tooth apex.
- *Cortex destruction of developing follicle*—if acute alveolar abscess of deciduous teeth occurs, the possibility of



Figs. 7.8A to C: (A and B) Cutaneous sinus showing extraoral opening; (C) Periapical abscess with extraoral draining sinus.

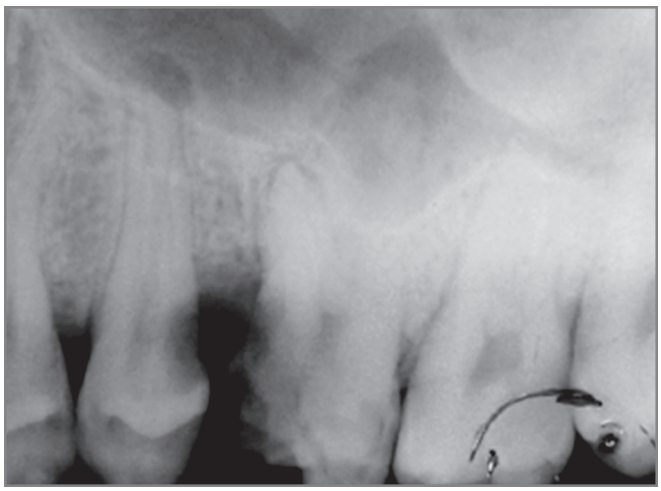


Fig. 7.10: Chronic periapical infection in upper first molar showing loss of lamina dura.
(*Courtesy:* Dr Ashok L)

damage to the permanent successor can occur which is evident by rarefaction produced by destruction of the cortex of the follicle.

Chronic Periapical Abscess

- *Lamina dura*—there is loss of thickness and density of the apical portion of lamina dura of the affected tooth **(Fig. 7.10)**.
- *Periapical rarefaction*—widespread area of diffuse demineralization of the periapical bone, of affected tooth becomes apparent **(Fig. 7.11)**.
- *Margins*—margins vary from well defined with possible hyperostotic borders to poorly defined, in chronic cases. In some areas, margins fade gradually and imperceptibly into the normal bone, while in some there is abrupt demarcation between the normal bone and the radiolucent area.

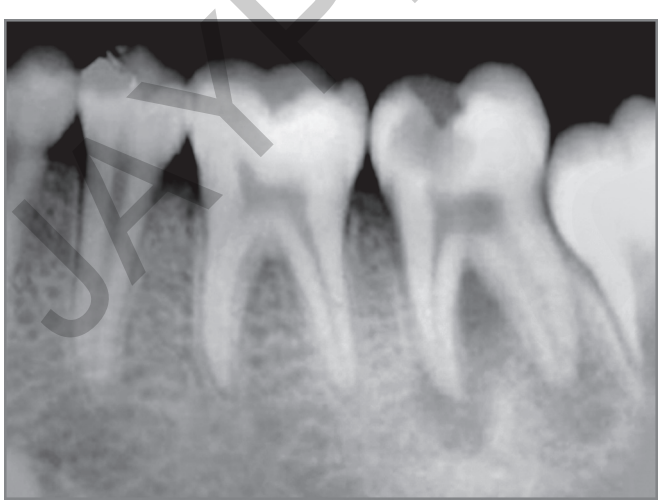


Fig. 7.11: Periapical rarefaction seen in chronic periapical infection.

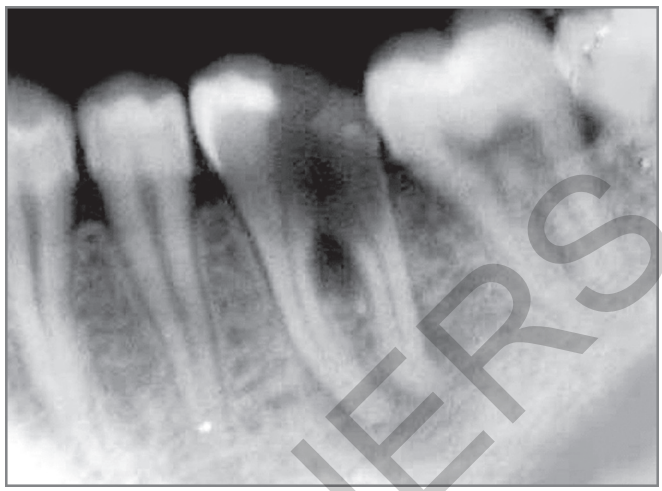


Fig. 7.12: Loss of bone density seen in periapex of the tooth due to chronic periapical infection.

- *Advanced cases*—after some period, the trabeculae which are rarefied are destroyed and the dark area become darker and larger, as more of the surrounding bone is taken into the diseased area **(Fig. 7.12)**.
- *Adjacent tooth*—in some cases, radiolucency may involve adjacent tooth and there may be loss of lamina dura of that tooth.
- *Side of root*—in some cases, osteitis can occur at the side of root rather than the apex. The reasons for this are that the infection may spread from adjacent tooth or perforation of the root (while filling the canal) or from an aberrant canal opening.
- *Maxillary upper posterior teeth*—in case of maxillary upper posterior teeth (as they are near to maxillary sinus), the radiographic appearance shows bone destruction at the apex of the tooth, along with loss of lamina dura and with destruction of a portion of the antral floor.
- *Root*—roots of the affected teeth may show resorption.

Diagnosis

- *Clinical diagnosis*—positive tenderness, carious tooth, sinus formation, non-vital teeth will give clue to the diagnosis.
- *Radiological diagnosis*—PDL space widening, loss of lamina dura and periapical rarefaction will diagnosed this condition.
- *Laboratory diagnosis*—central region of necrosis contain dense accumulation of polymorphonuclear leukocytes, surrounded by inflamed connective tissue wall of varying thickness.

Differential Diagnosis

- *Periodontal abscess*—it is an accumulation of pus along the root surface of a tooth. It is associated with periodontal pocket with mild pain. It is usually associated with vital, rather than pulp less teeth.

- *Periapical osteofibrosis*—it is associated with vital tooth. There may be persistence of lamina dura in the periapical osteofibrosis, even in the presence of well marked bone destruction.
- *Foramina*—in it, lamina dura is intact. If foramens are exactly superimposed on the roots, then another radiograph should be taken at different angulation.
- *Inferior dental canal*—it is frequently superimposed on the lower molars and if the canal is dark and apex is small, it is difficult to trace the lamina dura. In this case, magnifying glass should be used to trace the continuity of lamina dura.
- *A large normal marrow space*—it can be confused if superimposed on the apex of the root. Again, by using magnifying glass, lamina dura can be traced.

Management

- *Establish drainage immediately, if possible*—it may be done by opening the pulp chamber and passing file through the canal into the periapical region. Trephination opening through mucosa and bore to the abscess at apex. Through and through drain is placed in the abscess and irrigated with 1:1 mix of 3% H_2O_2 and normal saline solution.
- *Antibiotics*—penicillin (ampicillin) IM 500 mg every 6 hours orally or IV 250–500 mg every 6 hours.
- *Endodontic treatment*—in 24–48 hours, it can be determined if the tooth can be treated endodontically or extraction is necessary. If there is need of retention of offending tooth, tooth should be treated endodontically.
- *Warm saline mouth rinse* often aid in localizing the infection and maintaining adequate drainage, before endodontic treatment or extraction.

ACUTE EXACERBATION OF A CHRONIC LESION

It is also called as *'phoenix abscess.'* It is an acute inflammatory reaction superimposed on an existing chronic lesion, such as on cyst or granuloma.

Etiology

The peri-radicular area may react to noxious stimuli form a diseased pulp with chronic peri-radicular disease. At times, because of an influx of necrotic product from a diseased pulp or because of bacteria and there toxins, this apparently dormant lesion may react and cause an acute inflammatory response.

Clinical Features

- *History*—the patient has history of traumatic accident that turned the tooth dark after a period of time or of postoperative pain in a tooth that had subsided until the present episode of pain.

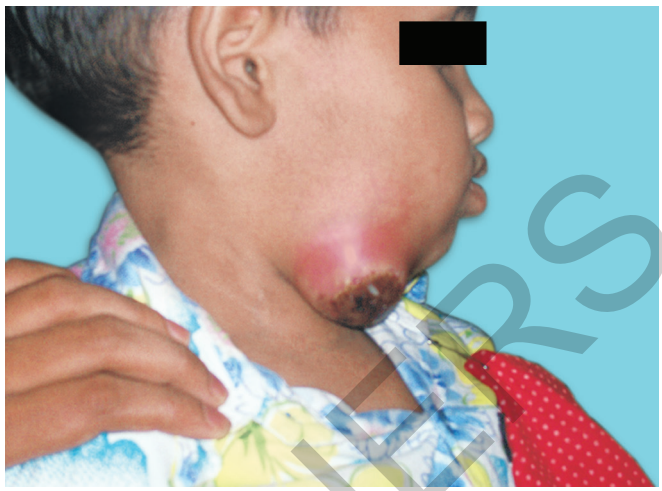


Fig. 7.13: Local associated cellulitis occur in patient with phoenix abscess.

- *Signs and symptoms*—patient complains of intense pain, local swelling and possibly associated cellulitis **(Fig. 7.13)**. Tooth may be tender to touch. Lack of response to vitality test points to diagnose necrotic pulp.
- *Surface*—mucosa over the radicular area may be sensitive to palpation and may appear red and swollen.

Radiological features of this condition are same as that of chronic periapical infection **(Fig. 7.14)**.

Diagnosis

- *Clinical and radiological diagnosis*—positive tenderness with radiological finding of chronic periapical infection will diagnose this condition.
- *Laboratory diagnosis*—area of liquefaction necrosis with disintegrating polymorphonuclear neutrophils and cellular debris. These are surrounded by infiltration of macrophages and some lymphocytes.

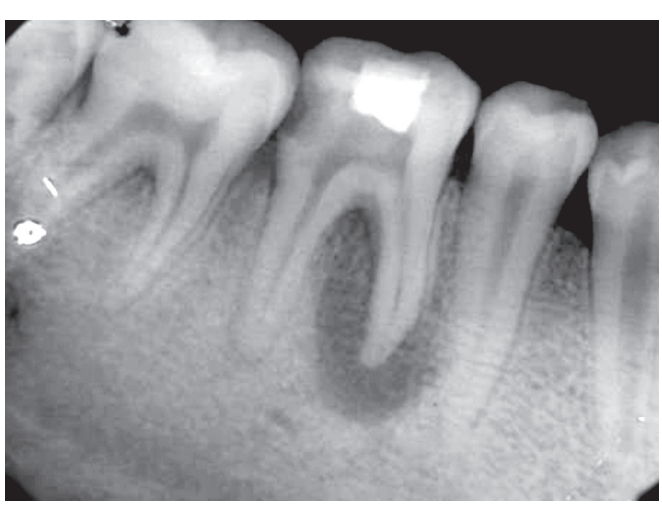


Fig. 7.14: Phoenix abscess showing same appearance as that of chronic periapical abscess.

Differential Diagnosis

Acute alveolar abscess—vitality test; it will react to electrical pulp test and application of cold, as compared to acute exacerbation of chronic infection.

Management is by drainage either via the root canal or by incision if there is localized swelling. Antibiotics and anti-inflammatory drugs should be given to the patient.

PERICORONAL ABSCESS

It is also called as '*pericoronitis*'. It is the infection of soft tissues surrounding the crown of a partially erupted tooth.

Clinical Features

- *Location*—the most common type is found around the mandibular 3rd molar.
- *Symptoms*—pain, malaise is present. Pain may radiate to throat, ear or floor of mouth.
- *Sequelae of pericoronitis*—it may result in cellulitis and muscular trismus. There is also regional lymphadenopathy, submaxillary and pharyngeal abscess.
- *Signs*—operculum may get traumatized by opposing teeth during mastication **(Fig. 7.15)**. Edema, visible in both submandibular area and peritonsillar region. There is extreme tenderness on palpation of the abscess.
- *Pericoronal infection of infancy*—pericoronal infection of infancy is often associated with the supra-dental tissue, involving the superior portion of the follicle and the overlying mucoperiosteum, which may become inflamed. It ultimately develops into small fluctuant abscess. When this fluctuance is digitally ascertained, incision and drainage should be carried out, followed by warm saline rinses at frequent intervals.
- *Complication*—the involvement may become localized in the form of a pericoronal abscess. It may spread posteriorly into the oropharyngeal area and medially, to the base of the tongue. Peritonsillar abscess, cellulitis and Ludwig's angina are common.

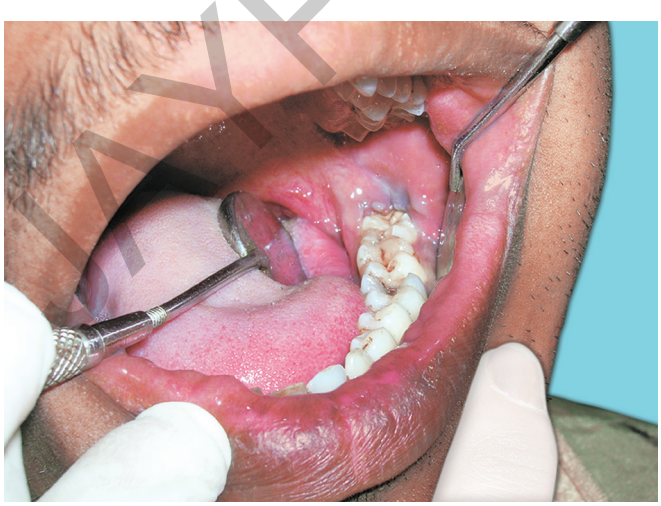


Fig. 7.15: Pericoronitis affecting partially erupted mandibular third molar with inflammation of operculum.

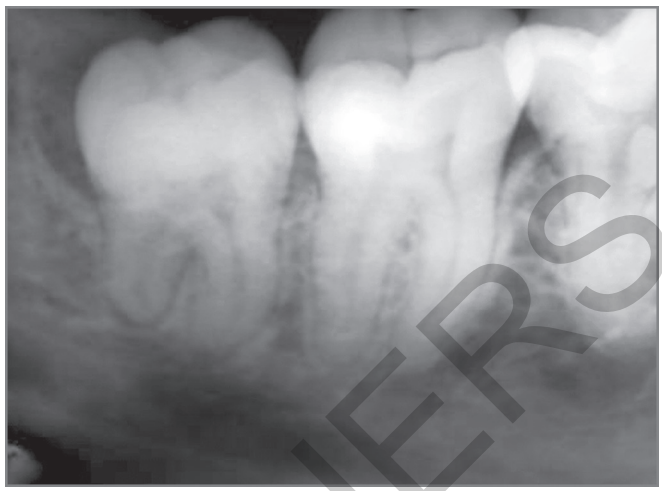


Fig. 7.16: Semilunar-shaped distal bone loss seen in association with mandibular third molar suggestive of pericoronitis.

Radiographic Features

- *Appearance*—defect in bone on mesial or distal side which appears as the step like distortion of crypt wall distal to the crown **(Fig. 7.16)**. In cases of lower third molar, there is circumferential bone resorption around the tooth. Mesially tipped impaction display semilunar shaped bone resorption mesial to the crown and in cases of distally placed impaction, it is distal to the crown.
- *Sclerosing osteitis*—follicular crypt may show sclerosing osteitis. Generalized thickening of the wall of crypt indicates low-grade infection.

Diagnosis

- *Clinical diagnosis*—inflammation of pericoronal tissue around 3rd molar with pain and trismus will give clue to the diagnosis.
- *Radiological diagnosis*—there is semilunar shaped distal bone loss.

Management

- *Antibiotics*—immediately, antibiotics should be started. Most commonly use antibiotics are amoxicillin with clavulanic acid 625 mg twice daily. In pericoronitis due to ulceromembranous gingivitis, metronidazole 400 g three times daily for 7 days is given.
- *Drainage*—careful probing should be done around the 3rd molar, which permits entry into the expanded follicle and allows evacuation of pus and other septic material.
- *Extraction*—when the symptoms become sub-acute, the impacted 3rd molar should be extracted.
- *Operculectomy*—sometimes when the retention of 3rd molar is necessary, the inflamed tissue surrounding the occlusal portion of the tooth should be excided.

- *Extraction of maxillary 3rd molar*—maxillary 3rd molar can be a contributing factor to the pericoronal infection of the mandibular 3rd molar. In such cases, especially when the mandibular 3rd molar is fully erupted in proper place, the maxillary 3rd molar should be extracted prior to the retention of the mandibular 3rd molar, considering the recurrent nature of the inflammatory episode.

PERIAPICAL GRANULOMA

It is the most common type of pathologic radiolucency encountered in dentistry. It is a growth of granulation tissue continuous with the periodontal ligament resulting from the death of the pulp and diffusion of bacteria and bacterial toxins from the root canals into the surrounding periradicular tissues through the apical and lateral foramina.

It is the result of a successful attempt by the periapical tissues to neutralize and confine the irritating toxic product that is escaping from the root canal. But continuous discharge into the periapical tissues induces a vascular inflammatory response.

Etiopathogenesis

- *Prolonged irritation*—it occurs as a response to intense and prolonged irritation from infected root canals producing extension of chronic apical periodontitis beyond the periodontal ligament.
- *Release of inflammatory mediators*—insult from diseased pulp represents broad spectrum of inflammatory mediators like prostaglandins, kinin and endotoxins. Elevated level of IgG in pulpoperiapical lesion.
- *Replacement of bone by granulation tissue*—the expanding inflammation and increased vascular pressure result in abscess formation and resorption of the bone in the affected area, which in period of time is replaced by granulation tissue.

Clinical Features

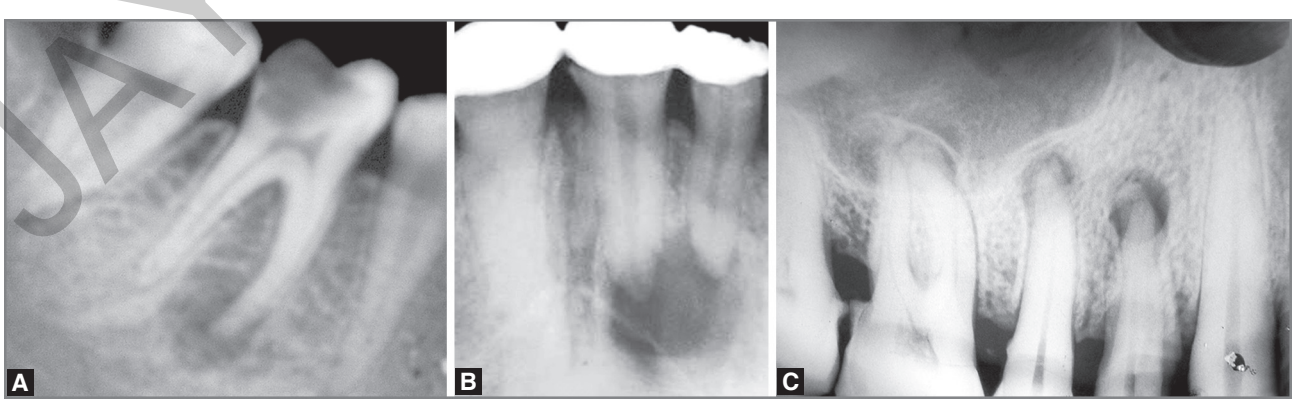
- *Signs and symptoms*—mild pain can be occasionally experienced while biting or chewing on solid foods. Tooth may be darker in color, because of the blood pigments that diffuse into the dentinal tubules. There is, seldom, swelling or expansion of the overlying cortical bone. Tooth may feel to be slightly elongated in the socket.
- *Vitality test*—the tooth is non-vital as it does not respond to thermal and electric pulp test.
- *Sensitivity*—sensitivity occurs due to hyperemia, edema and inflammation of the apical periodontal ligament.

Radiographic Features

- *Lamina dura*—periapical area is radiolucent with loss of lamina dura **(Fig. 7.17A)**.
- *Size*—radiolucency is less than 1.5 cm in diameter **(Fig. 7.17B)**.
- *Margins*—there may or may not be hyperostotic borders. It may or may not have well-defined borders.
- *Teeth*—involved tooth may show a deep restoration, extensive caries, fracture or a narrow pulp canal with non-vital pulp.
- *Multiple periapical granuloma*—in some cases, multiple periapical granuloma may be present **(Fig. 7.17C)**.

Diagnosis

- *Clinical diagnosis*—it is not possible to make clinical diagnosis on the basis of signs and symptoms.
- *Radiological diagnosis*—well defined radiolucency less than 1.5 cm will go in favor of periapical granuloma.
- *Histopathological diagnosis*—shows proliferating endothelial cells capillaries, young fibroblasts minimum amount of collagen and occasionally, nests of odontogenic epithelium, Russell's bodies, foam cells and cholesterol clefts.



Figs. 7.17A to C: (A) Periapical granuloma presented as radiolucency in the periapex of tooth; (B) As radiolucency in relation with two teeth; (C) Multiple periapical granulomas seen at the apices of the teeth.

Differential Diagnosis

- *Osteolytic stage of cementoma*—in case dental granuloma, tooth is non-vital.
- *Radicular cyst*—it is described in differential diagnosis of radicular cyst.
- *Surgical defect or periapical scar*—tooth shows root canals filling.

Management

- *Root canal therapy*—root canal therapy is treatment of choice.
- *Extraction of teeth*—if the tooth is unrestorable then extraction of the involved tooth should be carried out.
- *Curettage*—curettage of apical tissue can be carried out.
- *Causes of failure of treatment*—lesion may heal due to inadequate endodontic, vertical foreign material, associated periodontal disease, and cysts formation.

PERIAPICAL SCAR

It is a possible end point of healing. It is composed of dense fibrous tissue and is situated at the periapex of pulp less tooth, in which usually, the roots canal have been successfully filled.

Formation of Scar

- *Irritant substance*—confined in the periapical area, which leads to accumulation of chronic inflammatory cells.
- *Granuloma formation*—young fibroblasts, endothelial cells and capillaries proliferate, which lead to granuloma formation.
- *Scar formation*—after endodontic treatment, the granuloma resolves, but in some cases, granulation tissue gets slowly organized with the production of more and more collagen fibers, which in turn leads to scar formation.

Clinical and Radiological Features

- *Clinical features*—it is more common in anterior region of maxilla. Tooth is non-vital and the patient is asymptomatic.
- *Radiolucency*—well circumscribed radiolucency that is more or less round and is smaller than granuloma and cyst **(Fig. 7.18).** Scar is constant in size throughout the period.

Diagnosis

- *Radiological diagnosis*—radiolucency at the apex of endodontically treated tooth.
- *Histopathological diagnosis*—shows spindle shaped fibroblast scattered throughout the dense collagen bundles, which show advanced degree of hyalinization.

Management—is not necessary.

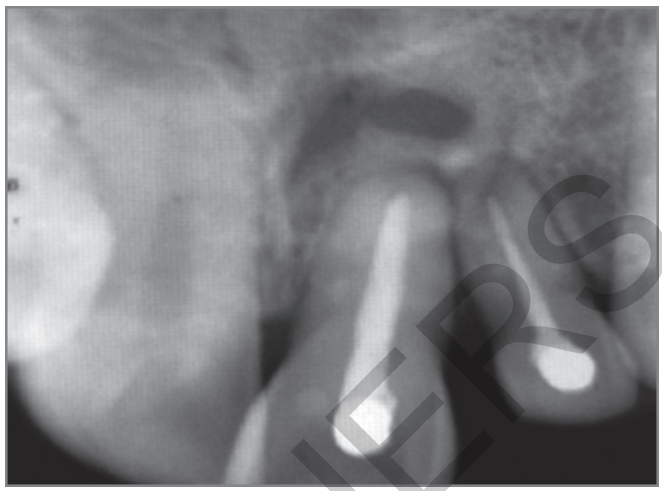


Fig. 7.18: Periapical scar seen as radiolucency at the apex of central incisor.
(*Courtesy:* Dr RN Mody)

BONE INFECTIONS

OSTEOMYELITIS

It is the inflammation of the bone marrow that produces clinically apparent pus and secondarily affects the calcified components. It is infection of the bone that involves all the three components viz. periosteum, cortex, and marrow.

It may be defined as an inflammatory condition of the bone that begins as an infection of medullary cavity and the haversian system and extends to involve the periosteum of the affected area.

Predisposing Factors

Predisposing factors significantly influence the onset and severity of osteomyelitis, apart from the virulence of the causative microorganisms. These factors mainly act by reducing host resistance or compromising jaw vascularity, thereby facilitating infection **(Table 7.1)**.

Etiology

- *Odontogenic infections* which can be periapical or periodontal infection, pericoronal infection and infection from infected dental cyst.
- *Compound fractures of the jaws*—generally, these fractures are compound through the tooth socket into the mouth and rarely, to the skin.
- *Traumatic injury*—local traumatic injury of the gingiva leads to periostitis, in patients with low resistance to infection and later to osteomyelitis.
- *Middle ear infection and respiratory infection*—via hematogenous route, either from middle ear infection or from infection of the upper respiratory tract.

TABLE 7.1: Predisposing factors for osteomyelitis.

Category	*Conditions*
Conditions affecting host resistance	Diabetes mellitus, tuberculosis, severe anemia, leukemia, agranulocytosis, acute illnesses such as influenza, scarlet fever, typhoid, and exanthematous fever, sickle cell anemia, malnutrition, and chronic alcoholism will affect host resistance. This will predispose for osteomyelitis occurrence.
Condition affecting jaw vascularity	Metastasis from remote area of infection such as another bony site, skin and kidneys, radiation, osteoporosis, osteopetrosis, fibrous dysplasia, bone malignancy, and peripheral vascular disease will affect jaw vascularity.

- *Furunculosis of chin*—furunculosis of chin, i.e., spread through lymphatic channel via infected lymph nodes.
- *Peritonsillar abscess*—peritonsillar abscess has also been reported to cause osteomyelitis of the ramus of mandible.

Pathogenesis

Osteomyelitis of the jaws commonly arises from odontogenic infections, where bacteria spread from periapical or periodontal lesions into the medullary bone, especially in areas of compromised blood supply. The infection causes intense marrow inflammation, vascular thrombosis, and ischemia, leading to bone necrosis and sequestration. The most common causative organisms are Staphylococcus aureus, Streptococcus species, and anaerobic bacteria associated with dental infections **(Table 7.2)**.

TABLE 7.2: Key events in pathogenesis of osteomyelitis.

Stage	*Key events*
Predisposing factor	Compromised blood supply is critical for establishment of osteomyelitis
Entry of microorganisms	Virulent bacteria enter medullary cavity via odontogenic infections, fractures, periostitis, hematogenous and lymphatic routes
Inflammatory reaction	Intense inflammation occurs within bone marrow; pain is a prominent feature
Initial localization	Infection is initially localized by pyogenic membrane or abscess wall like most of the odontogenic infections
Breakdown of barrier	Pyogenic membrane disorganized by virulent bacteria or chronic movement of unreduced fracture
Mechanical trauma	Repeated trauma causes bone ischemia and drives bacteria deeper into underlying tissues
Pus accumulation	Increased intramedullary pressure causes breakdown of barrier which leads to vascular compression and collapse, venous stasis, and ischemia
Elevation of periosteum	Pus spreads through Haversian and Volkmann canals and accumulates beneath the periosteum, elevating it from the cortex, thereby further reducing the blood supply
Necrosis of bone	Reduced blood supply results in gradual necrosis of bone
Penetration of periosteum	Continued pus accumulation causes periosteum perforation leading to mucosal or cutaneous fistula discharging the purulent pus
Chronic phase following therapy	Therapy induces host resistance becoming chronic process. Granulation tissue and neovascularization form resulting in lysis of bone causing separation of necrotic bone from viable bone
Involucrum formation	Small necrotic bone fragments may undergo complete lysis, while larger fragments become separated from newly formed bone by granulation tissue. The dead bone surrounded by viable new bone is termed involucrum
Sequestra formation	Small avascular fragments of necrotic bone are called sequestra which harbor microorganisms and act as a persistent source of infection. If not removed, they maintain chronic infection of surrounding granulation tissue. Continued sequestration weakens the bone and may lead to pathological fracture
Cloacae formation	An involucrum may contain 1 or 2 surface openings called cloacae, which are channels extending deep into areas of bone destruction around a sequestrum. Pus drains from the depth of bone to the surface through these openings, indicating the presence of dead bone or a foreign body
Systemic spread	Microorganisms precipitate thrombi formation by virtue of their destructive lysosomal packages. Coagulum provides the host medium for further pathogenic proliferation and isolating barrier from the host immune response which allows systemic spread of infection
Advanced necrosis of bone	Thrombosis and vascular compression worsen necrosis; compression of neurovascular bundles can result in osteomyelitis mediated anesthesia which can be complicated by pre-existing predisposing factors
Remodeling	After removal of sequestra and infection resolution, jaw undergoes complete remodeling

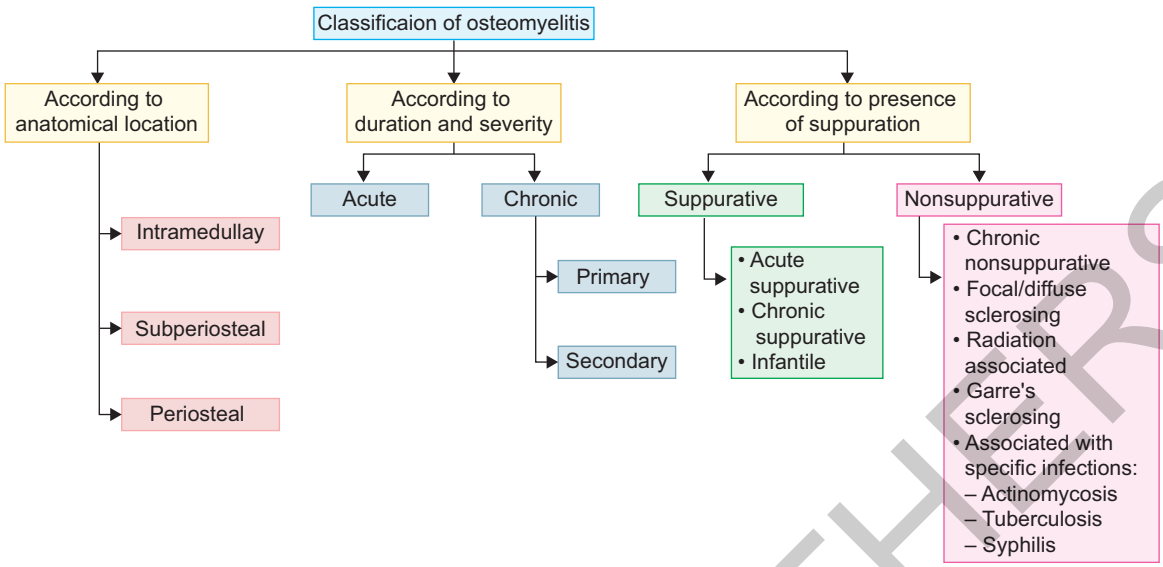


Fig. 7.19: Classification of osteomyelitis.

Microbiology of Osteomyelitis

Microorganism associated are *Staphylococcus aureus, Staphylococcus albus,* hemolytic streptococci, gram-negative organisms like *Klebsiella*, *Pseudomonas*, *Protease*, *E. coli*, anaerobic microorganisms like pepto-streptococci, bacteroides and fusobacteria. Some specific forms like *Mycobacterium tuberculosis, Treponema palladium* and *A. israeli.* Following parameters should be used for recognition of pure anaerobic or mixed anaerobic infection:

- Presence of foul smelling exudate.
- Sloughing of necrotic tissues or gas in the necrotic tissue.
- Gram stain revealing multiple organisms of different morphological character.
- Presence of sequestra.

Classification

Classification of osteomyelitis is based on its anatomical location within bone, duration and severity and presence of suppuration **(Fig. 7.19)**. Acute osteomyelitis occurs when initial infection is due to microorganisms while chronic is further divided into primary and secondary. In primary osteomyelitis microorganism virulence is low and host resistance is high and there is no preceding acute symptoms. Chronic osteomyelitis is secondary to untreated acute osteomyelitis.

Clinical Staging of Osteomyelitis

Clinical course follows the pathogenic stages of the condition and is depicted in **Table 7.3**.

Occurrence

- *Sex*—it is more common in men, than women.
- *Sites*—it occurs primarily in mandible in premolar area because the cortical plate of the bone in mandible is

TABLE 7.3: Clinical staging of osteomyelitis.

Stage	*Description*
Initial stage	Spontaneous pain (localized)
Acute stage (suppurative stage)	In this stage, there is severe pain, soreness and looseness of the involved teeth
Early acute stage	In reference to the involved tooth, progressive sensitivity of the adjacent teeth to percussion and pain in the involved side of jaw
Late acute stage	Paresthesia or anesthesia of the lip region supplied by the mental nerve. Other systemic symptoms can occur
Osteonecrotic stage	Diminished spontaneous pain, abscess formation and pus discharge **(Figs. 7.20 to 7.22)**
Sequestrum stage	Lack of symptoms, sequestrum formation visible on the radiograph

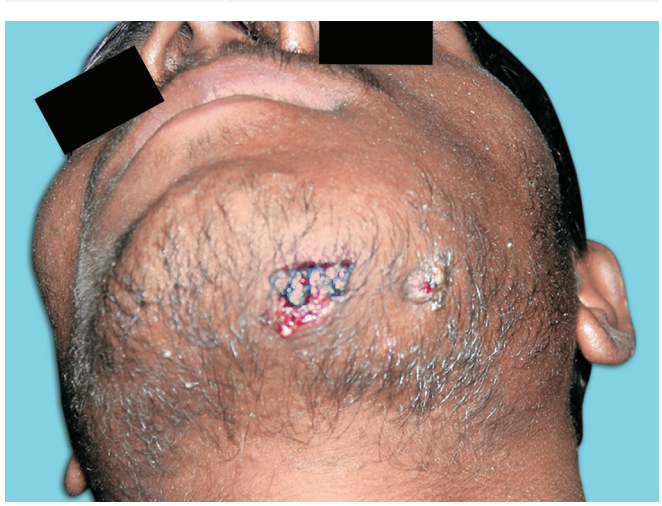


Fig. 7.20: Osteomyelitis in osteonecrotic stage showing extraoral discharging sinus.

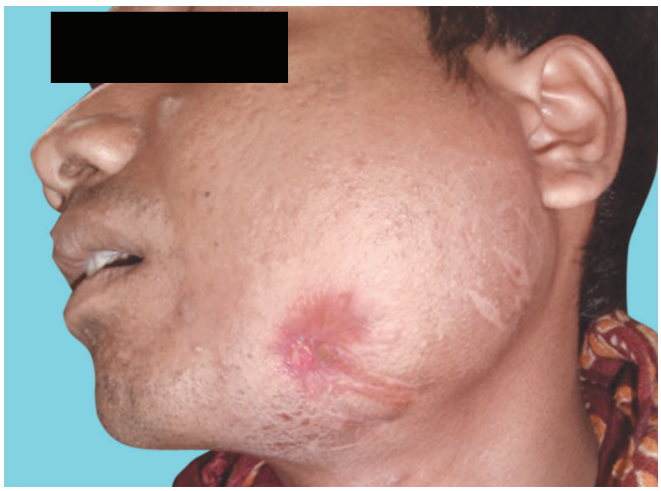


Fig. 7.21: Extraoral sinus seen in osteomyelitis patient.

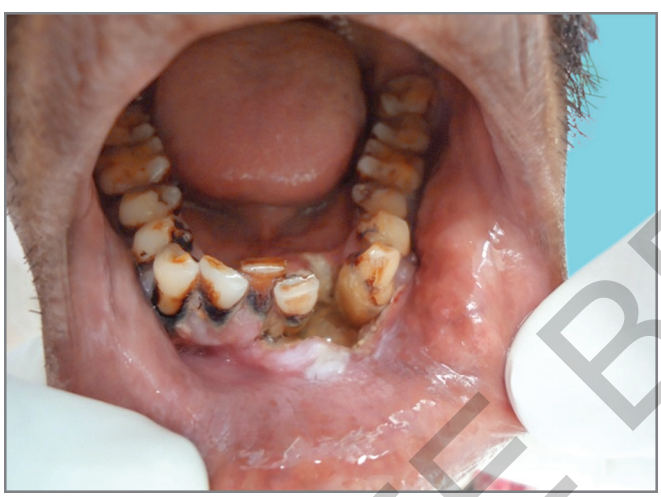


Fig. 7.22: Intraoral ulceration seen in osteomyelitis.

dense. It takes longer time for sinus formation and release of pits and hence, the infection gets directs into spongiosa and spreads. Additionally, removal of posterior mandibular teeth is attended by more damage to the bone and mandible is less vascular than maxilla. Thin cortical plates and relative paucity of medullary tissue in the maxilla precludes confinement of infection within the bone and permits dissipation of edema and pus into the soft tissues and paranasal sinuses.

- *Infantile osteomyelitis* is more common in maxilla than mandible, as it spreads by hematogenous route and maxilla has more blood supply than mandible.

Clinical Features

Acute

- *Initial signs and symptoms*—it has rapid onset and course. Patient complaint of severe pain, paresthesia or anesthesia of the mental nerve. At this stage, the process is truly intramedullary, therefore swelling is absent, teeth are not mobile and fistulae are not present.
- *Late signs and symptoms*—there is deep intense pain, anorexia, malaise, fever, and regional lymphadenopathy. Patient also complain of soreness of involved teeth which become loose within 10–14 days. There is also fetid oral odor. Pus exudates around the gingival sulcus or through mucosal and cutaneous fistula. There is firm cellulitis of cheek and abscess formation with localized warmth and tenderness on palpation. The patient feels toxic and dehydrated.

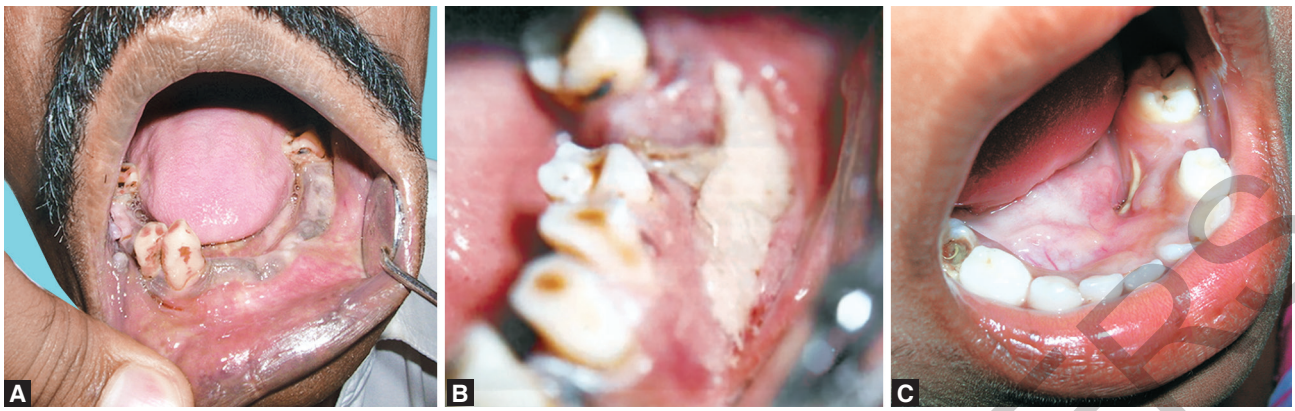
Chronic

- *Onset*—it has insidious onset with slight pain, slow increase in jaw size and a gradual development of sequestra without fistula.
- *Signs and symptoms*—it is painless unless there is an acute or sub-acute exacerbation. Local tenderness and swelling develop over the bone in the area of abscess **(Fig. 7.23A)**.
- *Necrotic bone*—in some cases, necrotic bone may be visible inside the oral cavity **(Figs. 7.23B and C)**.
- *Sinus*—intraorally and extraorally sinus develops **(Fig. 7.24)** intermittently and drains small amount of pus and then gradually heals. Sinus extends from medullary bone, through cortical plate, to mucous membrane or skin. Sinus may be at a considerable distance from the offending infection.
- *Lymph nodes*—regional lymphadenopathy is present.

Radiographic Features

Acute

- *Radiodensity*—about 10 days after acute infection, the density of trabeculae will be decreased, with blurred and fuzzy. For the radiographs to reveal any changes, there must be a loss of from 30 to 60% in the calcium content.
- *Trabecular pattern*—the earliest radiographic change is that trabeculae in the involved area are thin, of poor density and slightly unsharp or blurred. The trabeculae soon loose their continuity as well as the little density present. Individual trabeculae become fuzzy and indistinct.
- *Multiple radiolucency*—subsequently, multiple radiolucency appear which become apparent on radiograph. These are enlarged trabeculae spaces caused by foci of necrosis and frank bone destruction.
- *Saucer-shaped destruction*—in some cases, there is a saucer shaped area of destruction with irregular margins and containing teeth, with variable amount of supporting bone.
- *Periosteal reaction*—it can either stimulate bone resorption or bone formation. Inflammatory exudate can lift the periosteum and stimulates bone formation. Radiologically, it appears as a thin faint, radiopaque line, adjacent to and almost parallel or slightly convex to the surface of the bone.
- *Lamina dura*—there is loss of continuity of lamina dura, which is seen in more than one tooth.
- *Technetium bone scan followed by gallium citrate scan*—it will help to confirm the diagnosis. With inflammatory



Figs. 7.23A to C: (A) Swelling seen in alveolar bone in case of osteomyelitis of mandible (*Courtesy:* Dr Chandrasekhar Bande); (B and C) Necrotic bone seen in osteomyelitis.

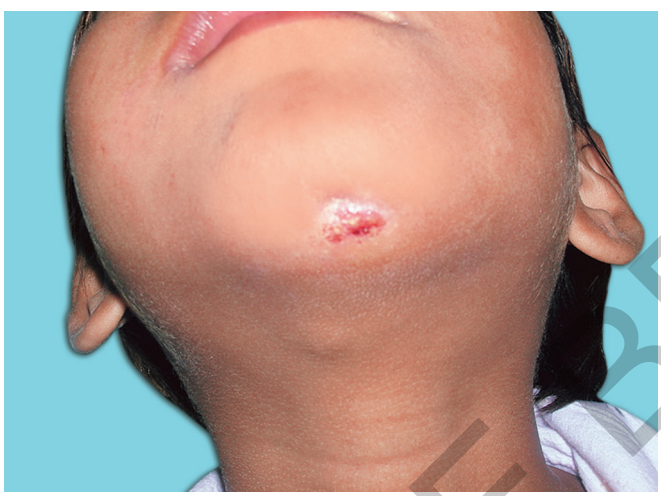


Fig. 7.24: Extraoral discharging sinus seen in osteomyelitis. (*Courtesy:* Dr Chandrasekhar Bande)

lesion, a positive result on the Tc99 scan indicates increased bone activity and a positive result on gallium scan in the some location indicates an inflammatory reaction.

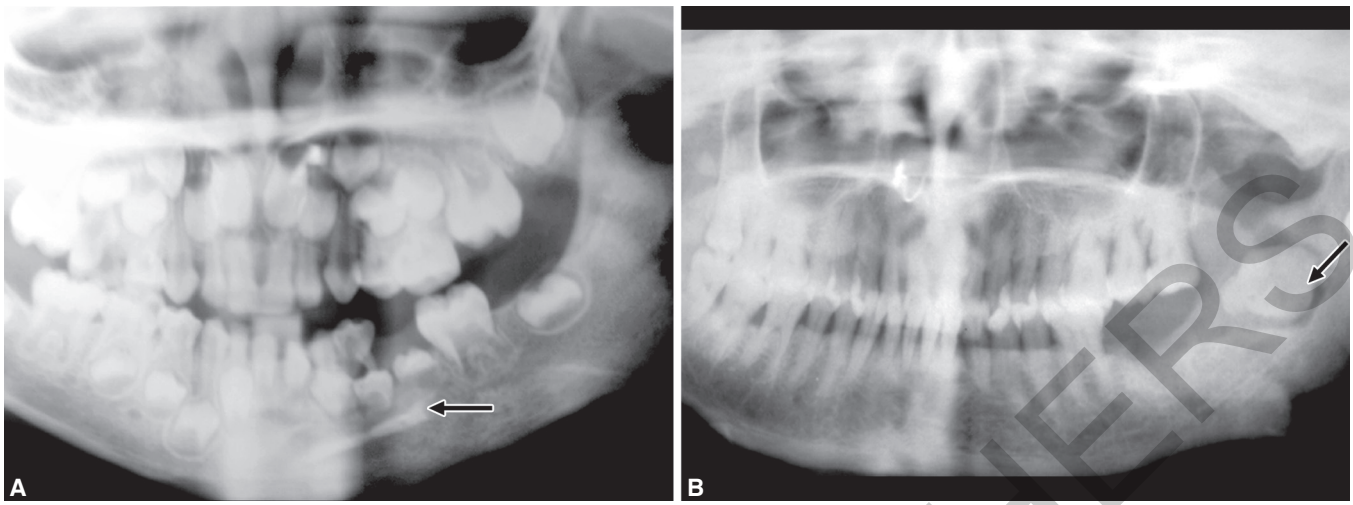
- *Osteomyelitis in children*—it shows paucity or absence of trabeculae in the tooth bearing area. In some cases, there may be loss of density on a part or whole of the cortical layer of one or more tooth follicles. Follicular cortex may become fragmented or lost over the variable area. Following destruction of the wall of the tooth follicle, it is common for the teeth to show evidence of moving.

Chronic

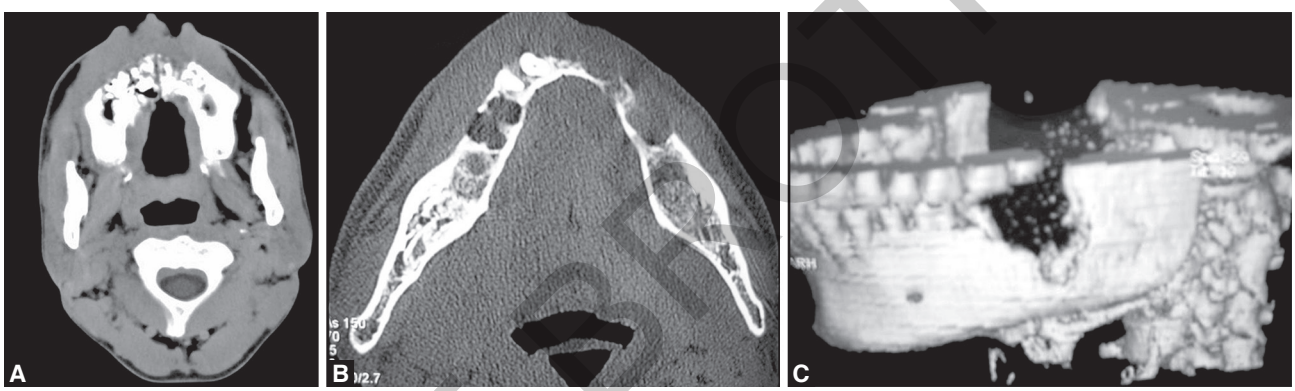
- *Radiodensity*—single or multiple radiolucency of variable sizes are seen.
- *Margins*—irregular outline and poorly defined borders.
- *'Moth eaten appearances'* is seen as the radiolucent areas enlarge and become irregular in outline and get separated by islands of normal appearing bone. This is due to the enlargement of medullary spaces and widening of Volkmann's canals, secondary to lysis of bone and replacement with granulation tissue.
- *Sequestra*—segments of necrotic bone become detached; irregular calcified areas separate from the remaining bone and become distinguishable as sequestra. Sequestra are more dense and better defined **(Figs. 7.25A and B)** due to following reasons:
  - Sclerosis that was induced before the bone became necrotic.
  - Dead bone has affinity for calcium. Hence, it absorbs calcium.
  - Inflammatory reaction is the probable stimulation for demineralization of the vital bone surrounding the sequestra. This enhances the contrast.
- *Teeth*—the roots of the teeth may undergo external resorption and the lamina dura may become less apparent as it blends with surrounding granular sclerotic bone.
- *Fistula tract*—fistula tracts may appear on the radiograph as radiolucent bands transversing the body of the jaw and penetrating the cortical plates.
- *Joint involvement*—in patients with extensive chronic osteomyelitis, the disease may spread to mandibular condyle and joint, resulting in septic arthritis.
- *Computed tomography*—computed tomography is more useful in revealing the internal structure and sequestra more readily than conventional radiography **(Figs. 7.26A to C)**.
- *Pathological fracture*—in some cases osteomyelitis can lead to pathological fracture **(Fig. 7.27)**.

Diagnosis

- *Radiological diagnosis*—loss of lamina dura, saucer shaped destruction, sequestrum formation and moth eaten appearance will give clue to the diagnosis.
- *Histopathological diagnosis*—the medullary spaces are filled with inflammatory exudate that may or may not progress to the actual formation of pus. The inflammatory



Figs. 7.25A and B: Sequestration seen as radiopaque structure in the mandibular bone.



Figs. 7.26A to C: (A and B) Computed tomography showing destruction of mandibular cortex; (C) Three dimensional CT of osteomyelitis.

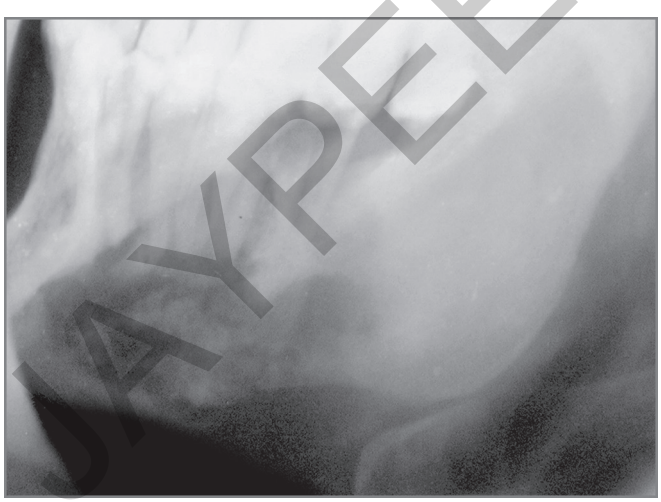


Fig. 7.27: Osteomyelitis with pathological fracture of mandible.

cells are chiefly neutrophilic polymorphonuclear leukocytes, but may show occasional lymphocytes and plasma cells.

- *Investigation to be carried out in osteomyelitis*—investigation like Gram staining, culture and sensitivity, WBC count and complete hemogram, blood sugar, Mantoux test, radiographs, scintigraphy and computerized tomography.

Differential Diagnosis

- *Paget's disease*—it affects multiple bones and the complete involvement of individual bone.
- *Eosinophilic granuloma*—margins are better than osteomyelitis and have no evidence of bone sclerosis.

Differences between osteomyelitis and carcinoma are summarized in **Table 7.4**.

Management

The goal of definitive therapy is to attenuate and eradicate the proliferating pathogenic microorganisms and to support healing. This is accomplished by removing pathogenic supporative debris, providing regional stability and disrupting pathophysiology barriers while re-establishing vascular permeability to the infected area.

- *Incision and drainage*—when early diagnosis is made, drainage of the fluctuant areas should be carried out

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