# CRASH COURSE IN ORAL PATHOLOGY



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**DENTISCOPE 2020** 



# Table of Contents

	/
DEVELOPMENTAL DEFECTS OF THE ORAL & MAXILLOFACIAL REGION	8
DEVELOPMENTAL DEFECTS OF THE LIP & PALATE	8
Cleft lip	8
Cleft palate	8
Cleft lip & palate	8
Submucous palatal cleft	
Lateral facial cleft	9
Oblique facial cleft	9
Median cleft of the upper lip	9
Congenital lip pits	9
Double lip	9
DEVELOPMENTAL DEFECTS OF ORAL MUCOSA	9
Fordyce granules	9
Leukodema	9
White spongy nevus	
DEVELOPMENTAL DEFECTS OF THE TONGUE	10
Macroglossia	
Lingual thyroid nodule	
Fissured tongue	
Black hairy tongue	
Geographic tongue	
DEVELOPMENTAL DEFECTS OF THE JAW BONES	11
Micrognathia	
Macrognathia	
Coronoid hyperplasia	
Condylar hyperplasia	
Condylar Hypoplasia	
Bifid condyle	
Bony exostosis	
Hemi facial hypertrophy	
Hemifacial Atrophy	
Vraniofacial dysostosis (Crouzen Syndrome)	
Mandibulofacial dysostosis ( Treacher-Collins Syndrome)	
Cleido cranial dysostosis	
Eagle syndrome (stylohyoid syndrome)	
DEVELOPMENTAL ALTERATION IN THE NUMBER OF THEETH	12
Andontia	
Hypodoptia	
пуродонна	
Oligodontia	
Oligodontia Hyperdontia [supernumerary teeth]	

Neo natal	12
Microdontia	13
Macrodontia	13
Gemination	13
Fusion	13
Concrescence	13
Accessory cusps	13
Enamel pearls	
Cervical enamel extension	
Taurodontism	
Dilaceration	
Globodontia	
Lobodontia	
Amelogenesis Imperfecta	14
Dentinogenesis Imperfecta	14
Shell teeth	14
Dentin Dysplasia (Rootless teeth)	14
Regional odontodysplasia (Ghost teeth)	14
Turner's hypoplasia	14
Syphilitic Hypoplasia	14
Congenital erythropoietic porphyria	15
Hyper billirubinemia	15
	15
Alveolar osteitis ( Dry Socket)	
Focal sclerosing (Condensing) osteitis	
Acute suppurative osteomyelitis	
Chronic suppurative osteomyelitis	
Chronic focal sclerosing osteomyelitis	
Diffuse scierosing osteomyelitis	
Chronic osteomyelitis with proliferative periostitis (Garre's Osteomyelitis, Periostitis Ossificans)	
Osteoradionecrosis	
Usteochemonecrosis	
METABOLIC BONE CONDITIONS	18
Osteoporisos	20
Hyperparathyroidism	20
Central Giant cell granuloma ( giant cell lesions; giant cell tumor)	20
Osteopetrosis "marble bone disease"	
Osteogenesis imperfecta	21
BENIGN FIBRO OSSEOUS LESIONS	
Periapical cemento-osseus dysplasia (periapical cementoma)	
Florid cemento-osseus dysplasia	
Firbous dysplasia	
Cherubism	
NEOPLASTIC BONE LESIONS	24
Osteoma	
Osteoblastoma & Osteoid Osteoma	
Ossifying (cemento-ossifying) fibroma	



Hemangioma	
Osteosarcoma	
Myeloma	
Langerhans cell histiocytosis	
Ewing Sarcoma	
CYSTS	28
INFLAMMATORY CYST	29
Radicular cyst	
Residual cyst	
Lateral radicular cyst	
Inflammatory collateral cyst/ paradental cyst or mandibular infected buccal cyst	
Dentigerous cyst	
Eruption cyst	
DEVELOPMENTAL CYST(ODONTOGENIC)	31
Odontogenic keratocyst /	
keratotic odontogenic tumor	
Lateral periodontal cyst	
Glandular odontogenic cyst	
NON EPITHELIAL PRIMARY BONE CYSTS	33
Solitary bone cyct	
Simple or traumatic or Hemorrhagic bone cyst	
Aneurysmal bone cyct (ABC)	
Stafne's idopathic cavity	
DEVELOPMENTAL CYSTS	
Epstien's pearls	
Bohn's nodules	
Nasolabial cyst (Nasoalveolar cyst, Klestadt cyst)	
Globulomaxillary cyst	
Median palatal cyct	
Nasopalatine duct cyst(Incisive canal cyst)	
Median Mandibular cyst	
Dermoid cyst	
Thyroglossal duct cyst	
Cervical lymphoepithelial cyst (Branchial cleft cyst)	
Oral lymphoepithelial cyst	
ODONTOGENIC TUMORS	
Ameloblastoma	
Adenomatoid odontogenic tumor (AOT)	
Calcifying epethelial odontogenic tumor (PINDBORG TUMOR) CEOT	
Calcifying odontogenic cyst/ tumor ( Ghost cell cyst)	
Squamous odontogenic tumor	
Ameloblastic fibroma	
Ameloblastic fibro-odontoma	
Odontogenic myxoma	
Cementoplastoma	



LYMPHOID LESIONS	
Lymphoid hyperplasia	
Angiolymphoid hyperplasia with eosinophilia (ALHE)	
Hodgkin's lymphoma	
Non Hodgkins lymphoma	
Granulocytic sarcoma( Extra medullary myeloid tumor)	
VERRUCA PAPILLARY LESIONS	47
Squamous papilloma	
Papillary hyperplasia /palatal papillomatosis	
Condylomata latum	
Condylomata accuminatum	
Focal epithelial hyperplasia (Heck's Disease)	
Pyostomatitis vegetans	
Verruciform xanthoma	
Verrucous carcinoma	
Keratoacanthoma	
Basal cell carcinoma	
Malignant melanoma	
REACTIVE AND NEOPLASTIC SOFT TISSUE LESIONS	51
REACTIVE HYPERPLASTIC FIRROUS TISSUE LESIONS	51
Fibroma	51
Giant cell fibroma	51
Enulis fissuratum	51
Pyogenic granuloma	52
Enulis granulomatosa	52
Peripheral giant cell granuloma (Giant cell epulis)	
Peripheral ossifying fibroma (Ossifying fibroid epulis)	
NEOPLASTIC FIBROUS TISSUE LESIONS	53
Fibrous histiocytoma	
Fibromatosis	
Lipoma	53
NEURAL TISSUE LESIONS	54
Traumatic neuroma ( Amputation neuroma)	
Neurolemoma (Schwannoma)	
Neurofibroma	
Multiple endocrine neoplasia type 2B (MEN Syndromes)	54
Melanotic neuroectodermal tumor of infancy	55
MUSCULAR LESIONS	55
Granular cell tumor	
Congenital epulis (Congenital granular cell lesion)	55
Leiomyoma	55
VASCULAR LESIONS	56
Hemangioma	
Vascular malformation	



Lymphangioma       56         Angiosarcomas       56         Kaposi's sarcoma       56         ORAL SQUAMOUS CELL CARCINOMA       58         Causes of SCC       58         Causes of SCC       58         Clinical presentation of SCC       58         Metastasis of SCC       58         Grading of tumors       59         Staging of tumors [TNM system ]       60         Treatment of SCC       61         Reactive salivary gland lesions       62         MUCOUS EXTRAVASATION PHENOMENONA       62         MUCOUS EXTRAVASATION PHENOMENONA       62         MUCOUS EXTRAVASATION PHENOMENONA       63         MAXILLARY RETENTION CYST       63         SALIVARY GLAND DEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       67         Monconyphic adenoma       67         Mucoepidermoid carcinoma       70         Actinic cell carcinoma       70         Actinic cell carcinoma       70         Actinic cell carcinoma       71         Carcinoma ex-pleomorphic adenoma       71         Carcinoma ex-pleomorphic adenoma       71         Carcinoma ex-pleomorphic ade		
Angiosarcoma       56         Kaposi's sarcoma       56         ORAL SQUAMOUS CELL CARCINOMA.       58         Causes of SCC       58         Clinical presentation of SCC       58         Metastasis of SCC       58         Grading of tumors       59         Staging of tumors [TNM system ]       60         Treatment of SCC       61         Reactive salivary gland lesions       62         MUCOUS EXTRAVASATION PHENOMENONA       62         Mucous retention cyst [ obstructive sialadenitis]       63         NALILARY RETENTION CYST       63         SALIVARY GLAND DOBSTRUCTION - SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Matury statumationa       67         Oncocytit tumors       68         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       70         Actinic cell carcinoma       70         MaligNANT MIXED SALIVARY GALND TUMORS       71         Carcinoma       71         Carcinoma ex-pleomorphic adenoma       71      <	Lymphangioma	
Kaposi's sarcoma       56         ORAL SQUAMOUS CELL CARCINOMA       58         Causes of SCC       58         Clinical presentation of SCC       58         Metastasis of SCC       58         Grading of tumors       59         Staging of tumors [TNM system ]       60         Treatment of SCC       61         Reactive salivary gland lesions       62         MUCOUS EXTRAVASATION PHENOMENONA       62         MUCOUS EXTRAVASATION PHENOMENONA       62         MUCOUS EXTRAVASATION PHENOMENONA       63         MAXILLARY RETENTION CYST       63         SALIVARY GLAND OBSTRUCTION - SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         MALIGNANT SALIVARY GALND TUMORS       68         MALIGNANT SALIVARY GALND TUMORS       70         Actinic cell carcinoma       69         Mucospidermoid carcinoma       69         Mucospidermoid carcinoma       70         Actinic cell carcinoma       71         Carcinoma ex-pleomorphic adenoma       71         Clarcinoma ex-pleomorphic adenoma	Angiosarcomas	
ORAL SQUAMOUS CELL CARCINOMA.       58         Causes of SCC       58         Clinical presentation of SCC       58         Metastasis of SCC       58         Grading of tumors       58         Staging of tumors [TNM system ].       60         Treatment of SCC       61         Reactive salivary gland lesions       62         MUCOUS EXTRAVASATION PHENOMENONA.       62         MUCOUS EXTRAVASATION PHENOMENONA.       62         MUCOUS EXTRAVASATION PHENOMENONA.       63         MAXILLARY RETENTION CYST       63         SALIVARY GLAND OSTRUCTION - SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Mucoepidermoid carcinoma       67         Oncocytic tumors       68         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       70         Acinic cell carcinoma       70         Acinic cell carcinoma       71         Clar cell tumor       71         Clar cell tumor       71         Clar cell tumor       71         Clar	Kaposi's sarcoma	
Causes of SCC       58         Clinical presentation of SCC       58         Metastasis of SCC.       58         Grading of tumors       59         Staging of tumors       60         Treatment of SCC       61         Reactive salivary gland lesions       62         MUCOUS EXTRAVASATION PHENOMENONA       62         Mucous retention cyst [ obstructive sialadenitis]       63         MAXILLARY RETENTION CYST       63         SALIVARY GLAND OBSTRUCTION – SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Warthin's tumor       66         MaligNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       70         Acinic cell carcinoma       70         Acinic cell carcinoma       71         Carcinoma ex-pleomorphic adenoma       71         Carcinoma ex-pleomorphic adenoma       71         Polymorphous low grade adenocarcinoma       71         Polymorphous low grade adenocarcinoma       71         Polymorphous low grade adenocarcinoma       71         References </td <td>ORAL SQUAMOUS CELL CARCINOMA</td> <td>58</td>	ORAL SQUAMOUS CELL CARCINOMA	58
Clinical presentation of SCC       58         Metastasis of SCC       58         Grading of tumors       59         Staging of tumors [TNM system]       60         Treatment of SCC       61         Reactive salivary gland lesions       62         MUCOUS EXTRAVASATION PHENOMENONA       62         Mucous retention cyst [ obstructive sialadenitis]       63         MAILLARY RETENTION CYST       63         SALIVARY GLAND OBSTRUCTION – SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Warthin's tumor       66         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       69         Adenoid Cystic carcinoma       70         Acinc cell carcinoma       71         Carcinoma ex-pleomorphic adenoma       71         Carcinoma ex-pleomorphic adenoma       71         Polymorphous low grade adenocarcinoma       71         Polymorphous low grade adenocarcinoma       71         Polymorphous low grade adenocarcinoma       71         References       73         Di	Causes of SCC	
Metastasis of SCC       58         Grading of tumors       59         Staging of tumors [TNM system ]       60         Treatment of SCC       61         Reactive salivary gland lesions       62         MUCOUS EXTRAVASATION PHENOMENONA       62         Mucous retention cyst [ obstructive sialadenitis]       63         MAXILLARY RETENTION CYST       63         SALIVARY GLAND DOBSTRUCTION - SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Warthin's tumor       66         Mocoepidermoid carcinoma       67         Oncocytic tumors       68         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       70         Acinic cell carcinoma       70         MALIGNANT MIXED SALIVARY GALND TUMORS       71         Carcinoma ex-pleomorphic adenoma       71 <t< td=""><td>Clinical presentation of SCC</td><td></td></t<>	Clinical presentation of SCC	
Grading of tumors       59         Staging of tumors [TNM system ]       60         Treatment of SCC       61         Reactive salivary gland lesions       62         Mucous EXTRAVASATION PHENOMENONA       62         Mucous retention cyst [ obstructive sialadenitis]       63         MAXILLARY RETENTION CYST       63         SALIVARY GLAND OBSTRUCTION – SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Maxillary retrinon cyst [ obstructions]       68         MALIGNANT SALIVARY GALND TUMORS       68         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       69         Adenoid Cystic carcinoma       70         MALIGNANT MIXED SALIVARY GALND TUMORS       71         Carcinoma ex-pleomorphic adenoma       71         Clear cell carcinoma       71         Squamous cell carcinoma       71         Carcinoma ex-pleomorphic adenoma       71         Carcinoma ex-pleomorphic adenoma       71         Carcinoma ex-pleomorphic adenoma       71         Carcinoma ex-pleomorphic adenocarcinoma<	Metastasis of SCC	
Staging of tumors [ TNM system ]       60         Treatment of SCC       61         Reactive salivary gland lesions       62         MUCOUS EXTRAVASATION PHENOMENONA       62         Mucous retention cyst [ obstructive sialadenitis]       63         MAXILLARY RETENTION CYST       63         SALIVARY GLAND OBSTRUCTION – SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Warthin's tumor       66         MALIGNANT SALIVARY GALND TUMORS       68         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       60         Macine cell carcinoma       70         MALIGNANT SALIVARY GALND TUMORS       71         Carcinoma       70         MALIGNANT SALIVARY GALND TUMORS       71         Carcinoma       70         MALIGNANT MIXED SALIVARY GALND TUMORS       71         Carcinoma ex-pleomorphic adenoma       70         MALIGNANT MIXED SALIVARY GALND TUMORS       71         Carcinoma       71         Clear cell tumor       71         Squamous cell carcinoma <td>Grading of tumors</td> <td></td>	Grading of tumors	
Treatment of SCC       61         Reactive salivary gland lesions       62         MUCOUS EXTRAVASATION PHENOMENONA       62         Mucous retention cyst [ obstructive sialadenitis]       63         MAXILLARY RETENTION CYST       63         SALIVARY GLAND OBSTRUCTION – SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Warthin's tumor       66         Monomorphic adenoma       67         Oncocytic tumors       68         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       69         Adenoid Cystic carcinoma       70         Acinic cell carcinoma       71         Carcinoma ex-pleomorphic adenoma       71         Clear cell tumor       71         Squamous cell carcinoma       71         References       73         Disclaimer       74	Staging of tumors [ TNM system ]	
Reactive salivary gland lesions.       62         MUCOUS EXTRAVASATION PHENOMENONA       62         Mucous retention cyst [ obstructive sialadenitis]       63         MAXILLARY RETENTION CYST.       63         SALIVARY GLAND OBSTRUCTION - SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Warthin's tumor       66         Monomorphic adenoma       67         Oncocytic tumors       68         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       70         Actinic cell carcinoma       70         Macine cell carcinoma       71         Carcinoma ex-pleomorphic adenoma       71	Treatment of SCC	61
MUCOUS EXTRAVASATION PHENOMENONA.       62         Mucous retention cyst [ obstructive sialadenitis].       63         MAXILLARY RETENTION CYST.       63         SALIVARY GLAND OBSTRUCTION - SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Warthin's tumor       66         MALIGNANT SALIVARY GALND TUMORS       68         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       69         Adenoid Cystic carcinoma       70         Acinic cell carcinoma       71         Carcinoma ex-pleomorphic adenoma       71         Ciear cell tumor       71         Squamous cell carcinoma       71         Squamous cell carcinoma       71         Squamous cell carcinoma       71         Squamous cell carcinoma       71         References       73         Disclaimer       74	Reactive salivary gland lesions	62
Mucous retention cyst [ obstructive sialadenitis]       63         MAXILLARY RETENTION CYST       63         SALIVARY GLAND OBSTRUCTION – SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Warthin's tumor       66         Monomorphic adenoma       67         Oncocytic tumors       68         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       69         Adenoid Cystic carcinoma       70         Acinic cell carcinoma       71         Clear cell tumor       71         Squamous cell carcinoma       71         No of SALIVARY GLAND TUMORS       72         References       73         Disclaimer.       74	MUCOUS EXTRAVASATION PHENOMENONA	
MAXILLARY RETENTION CYST.       63         SALIVARY GLAND OBSTRUCTION - SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Warthin's tumor.       66         Monomorphic adenoma       66         Mucoepidermoid carcinoma       67         Oncocytic tumors       68         MALIGNANT SALIVARY GALND TUMORS.       69         Mucoepidermoid carcinoma       70         Acinic cell carcinoma       70         Carcinoma ex-pleomorphic adenoma       71         Clear cell tumor       71         Squamous cell carcinoma       71         Polymorphous low grade adenocarcinoma       71         TNM OF SALIVARY GLAND TUMORS       72         References       73         Disclaimer.       74	Mucous retention cyst [ obstructive sialadenitis]	
SALIVARY GLAND OBSTRUCTION - SIALOLITH       63         NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Warthin's tumor       66         Monomorphic adenoma       67         Oncocytic tumors       68         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       70         Acinic cell carcinoma       70         Acinic cell carcinoma       71         Carcinoma ex-pleomorphic adenoma       71         Squamous cell carcinoma       71         Polymorphous low grade adenocarcinoma       71         NMLIGNANT MIXED SALIVARY GALND TUMORS       72         References       73         Disclaimer       74	MAXILLARY RETENTION CYST	
NECROTIZING SIALOMETAPLASIA       65         SALIVARY GLAND NEOPLASTIC DISEASES       66         BENIGN SALIVARY GALND TUMORS       66         Pleomorphic adenoma       66         Warthin's tumor       66         Monomorphic adenoma       67         Oncocytic tumors       68         MALIGNANT SALIVARY GALND TUMORS       69         Mucoepidermoid carcinoma       69         Adenoid Cystic carcinoma       70         Acinic cell carcinoma       70         MALIGNANT MIXED SALIVARY GALND TUMORS       71         Carcinoma ex-pleomorphic adenoma       71         Clear cell tumor       71         Squamous cell carcinoma       71         Polymorphous low grade adenocarcinoma       71         TIM OF SALIVARY GLAND TUMORS       72         References       73         Disclaimer       74	SALIVARY GLAND OBSTRUCTION – SIALOLITH	
SALIVARY GLAND NEOPLASTIC DISEASES66BENIGN SALIVARY GALND TUMORS66Pleomorphic adenoma66Warthin's tumor66Monomorphic adenoma67Oncocytic tumors68MALIGNANT SALIVARY GALND TUMORS69Mucoepidermoid carcinoma69Adenoid Cystic carcinoma70Acinic cell carcinoma70MALIGNANT MIXED SALIVARY GALND TUMORS71Carcinoma ex-pleomorphic adenoma71Clear cell tumor71Squamous cell carcinoma71Polymorphous low grade adenocarcinoma71TNM OF SALIVARY GLAND TUMORS72References73Disclaimer74	NECROTIZING SIALOMETAPLASIA	
BENIGN SALIVARY GALND TUMORS66Pleomorphic adenoma66Warthin's tumor66Monomorphic adenoma67Oncocytic tumors68MALIGNANT SALIVARY GALND TUMORS69Mucoepidermoid carcinoma69Adenoid Cystic carcinoma70Acinic cell carcinoma70MALIGNANT MIXED SALIVARY GALND TUMORS71Clear cell tumor71Clear cell tumor71Squamous cell carcinoma71Polymorphous low grade adenocarcinoma71TNM OF SALIVARY GLAND TUMORS72References73Disclaimer74	SALIVARY GLAND NEOPLASTIC DISEASES	66
Pleomorphic adenoma66Warthin's tumor66Monomorphic adenoma67Oncocytic tumors68MALIGNANT SALIVARY GALND TUMORS69Mucoepidermoid carcinoma69Adenoid Cystic carcinoma70Acinic cell carcinoma70MALIGNANT MIXED SALIVARY GALND TUMORS71Carcinoma ex-pleomorphic adenoma71Clear cell tumor71Squamous cell carcinoma71Polymorphous low grade adenocarcinoma71TNM OF SALIVARY GLAND TUMORS72References73Disclaimer74	BENIGN SALIVARY GALND TUMORS	66
Warthin's tumor66Monomorphic adenoma67Oncocytic tumors68MALIGNANT SALIVARY GALND TUMORS69Mucoepidermoid carcinoma69Adenoid Cystic carcinoma70Acinic cell carcinoma70MALIGNANT MIXED SALIVARY GALND TUMORS71Carcinoma ex-pleomorphic adenoma71Clear cell tumor71Squamous cell carcinoma71Polymorphous low grade adenocarcinoma71TNM OF SALIVARY GLAND TUMORS72References73Disclaimer74	Pleomorphic adenoma	
Monomorphic adenoma67Oncocytic tumors68MALIGNANT SALIVARY GALND TUMORS69Mucoepidermoid carcinoma69Adenoid Cystic carcinoma70Acinic cell carcinoma70MALIGNANT MIXED SALIVARY GALND TUMORS71Carcinoma ex-pleomorphic adenoma71Clear cell tumor71Squamous cell carcinoma71Polymorphous low grade adenocarcinoma71TNM OF SALIVARY GLAND TUMORS72References73Disclaimer74		66
Oncocytic tumors68MALIGNANT SALIVARY GALND TUMORS69Mucoepidermoid carcinoma69Adenoid Cystic carcinoma70Acinic cell carcinoma70MALIGNANT MIXED SALIVARY GALND TUMORS71Carcinoma ex-pleomorphic adenoma71Clear cell tumor71Squamous cell carcinoma71Polymorphous low grade adenocarcinoma71TNM OF SALIVARY GLAND TUMORS72References73Disclaimer74	Warthin's tumor	
MALIGNANT SALIVARY GALND TUMORS69Mucoepidermoid carcinoma69Adenoid Cystic carcinoma70Acinic cell carcinoma70MALIGNANT MIXED SALIVARY GALND TUMORS71Carcinoma ex-pleomorphic adenoma71Clear cell tumor71Squamous cell carcinoma71Polymorphous low grade adenocarcinoma71TNM OF SALIVARY GLAND TUMORS72References73Disclaimer74	Warthin's tumor Monomorphic adenoma	
Mucoepidermoid carcinoma69Adenoid Cystic carcinoma70Acinic cell carcinoma70MALIGNANT MIXED SALIVARY GALND TUMORS71Carcinoma ex-pleomorphic adenoma71Clear cell tumor71Squamous cell carcinoma71Polymorphous low grade adenocarcinoma71TNM OF SALIVARY GLAND TUMORS72References73Disclaimer74	Warthin's tumor Monomorphic adenoma Oncocytic tumors	
Adenoid Cystic carcinoma70Acinic cell carcinoma70MALIGNANT MIXED SALIVARY GALND TUMORS71Carcinoma ex-pleomorphic adenoma71Clear cell tumor71Squamous cell carcinoma71Polymorphous low grade adenocarcinoma71TNM OF SALIVARY GLAND TUMORS72References73Disclaimer74	Monomorphic adenoma Oncocytic tumors MALIGNANT SALIVARY GALND TUMORS	
Acinic cell carcinoma 70   MALIGNANT MIXED SALIVARY GALND TUMORS 71   Carcinoma ex-pleomorphic adenoma 71   Clear cell tumor 71   Squamous cell carcinoma 71   Polymorphous low grade adenocarcinoma 71   TNM OF SALIVARY GLAND TUMORS 72   References 73   Disclaimer 74	Monomorphic adenoma Oncocytic tumors MALIGNANT SALIVARY GALND TUMORS Mucoepidermoid carcinoma	
MALIGNANT MIXED SALIVARY GALND TUMORS71Carcinoma ex-pleomorphic adenoma71Clear cell tumor71Squamous cell carcinoma71Polymorphous low grade adenocarcinoma71TNM OF SALIVARY GLAND TUMORS72References73Disclaimer74	Marthin's tumor Monomorphic adenoma Oncocytic tumors MALIGNANT SALIVARY GALND TUMORS Mucoepidermoid carcinoma Adenoid Cystic carcinoma	
Carcinoma ex-pleomorphic adenoma	Monomorphic adenoma Oncocytic tumors <b>MALIGNANT SALIVARY GALND TUMORS</b> Mucoepidermoid carcinoma Adenoid Cystic carcinoma Acinic cell carcinoma	
Clear cell tumor       71         Squamous cell carcinoma       71         Polymorphous low grade adenocarcinoma       71         TNM OF SALIVARY GLAND TUMORS       72         References       73         Disclaimer       74	Monomorphic adenoma Oncocytic tumors MALIGNANT SALIVARY GALND TUMORS Mucoepidermoid carcinoma Adenoid Cystic carcinoma Acinic cell carcinoma MALIGNANT MIXED SALIVARY GALND TUMORS	
Squamous cell carcinoma       71         Polymorphous low grade adenocarcinoma       71         TNM OF SALIVARY GLAND TUMORS       72         References       73         Disclaimer       74	Monomorphic adenoma Oncocytic tumors MALIGNANT SALIVARY GALND TUMORS Mucoepidermoid carcinoma Adenoid Cystic carcinoma Acinic cell carcinoma MALIGNANT MIXED SALIVARY GALND TUMORS Carcinoma ex-pleomorphic adenoma	
Polymorphous low grade adenocarcinoma	Monomorphic adenoma Oncocytic tumors MALIGNANT SALIVARY GALND TUMORS Mucoepidermoid carcinoma Adenoid Cystic carcinoma Acinic cell carcinoma Acinic cell carcinoma Carcinoma ex-pleomorphic adenoma Clear cell tumor	67 68 69 69 70 70 70 70 71 71
TNM OF SALIVARY GLAND TUMORS	Monomorphic adenoma Oncocytic tumors MALIGNANT SALIVARY GALND TUMORS Mucoepidermoid carcinoma Adenoid Cystic carcinoma Acinic cell carcinoma MALIGNANT MIXED SALIVARY GALND TUMORS Carcinoma ex-pleomorphic adenoma Clear cell tumor Squamous cell carcinoma	67 68 69 69 70 70 70 70 71 71 71 71
References	Monomorphic adenoma Oncocytic tumors MALIGNANT SALIVARY GALND TUMORS Mucoepidermoid carcinoma Adenoid Cystic carcinoma Acinic cell carcinoma Acinic cell carcinoma Carcinoma ex-pleomorphic adenoma Clear cell tumor Squamous cell carcinoma Polymorphous low grade adenocarcinoma	67 68 69 69 70 70 70 71 71 71 71 71
Disclaimer74	Monomorphic adenoma Oncocytic tumors MALIGNANT SALIVARY GALND TUMORS Mucoepidermoid carcinoma Adenoid Cystic carcinoma Acinic cell carcinoma Acinic cell carcinoma Carcinoma ex-pleomorphic adenoma Clear cell tumor Squamous cell carcinoma Polymorphous low grade adenocarcinoma	67 68 69 69 70 70 71 71 71 71 71 71 71 71
	Monomorphic adenoma	67 68 69 69 70 70 71 71 71 71 71 71 71 71 71 71 71 71



# EXTRA / INTRA ORAL EXAMINATION

#### Examination is done initially and at every recall.

#### Extra oral examination: BE SYSTEMATIC TO MAKE SURE YOU DON'T MISS ANY PART

- 1- Face [ note skin color, facial swellings or asymmetries ]
- 2- **TMJ** [note any deviation on mouth opening, clicking, popping tenderness in the muscles of mastication]
- 3- Palpate **lymph nodes** [ sub mandibular, sublingual, pre and post auricular, cervical, supra clavicular lymph nodes ]
- 4- Palpate the **thyroid gland** [ place both hands on the trachea and ask the pt to swallow the thyroid gland moves upward ]
- 5- Lips exmaination [ note the vermilion border, lip color and then Bi-digitally palpate the tissue around the lips , avert the lips and examine the internal surface and frenal attachments]

#### Intraoral exmaination:

- 1- **Buccal vestibule** [ note the color + palpate the vestibules and check stenson's duct opening for any inflammation or blockage]
- 2- Gingiva [ note color, consistency and texture]
- 3- Examine the **hard palate** [ note any tori present or any swelling of the minor salivary glands ]
- 4- Exmaine soft palate + tonsils [ ask the pt to say AHHH ]
- 5- **Tongue** [ wrap the tip of the tongue with gauze and pull it from side to side to observe the lateral borders of the tongue]
- 6- Floor of the mouth [ palpate bi manually + exmaine wharton's duct fro any signs of inalmmation or blockage]
- 7- Exmaine occlusion

## 2. Which one of the techniques listed below would be appropriate for palpating the buccal and labial mucosa?

a. Digital

#### <mark>b. Bidigital</mark>

- c. Bilateral
- d. Bimanual
- e. Unilateral

1. All of the following structures would be noted when examining the oropharynx except one. Which one is the EXCEPTION?

- a. Tonsils
- <mark>b. Adenoids</mark>
- c. Tonsillar crypt
- d. Anterior/posterior pillars
- e. Posterior pharyngeal wall

#### https://quizlet.com/216119896/the-intraoral-and-extraoral-exam-dentalcare-flash-cards/





Tongue and floor of the mouth are the most common sites for oral cancer





# DESCRIBING LESIONS

#### When describing any lesion you need to mention:

- 1- radiodensity [ radio opaque, radiolucent or both]
- 2- site [ where is it exactly in the maxilla or the mandible ]
- 3- size [ either measured in Cm or according to it's boundaries in 2 dimensions ( lesion extending from this area to this area ) ]
- 4- shape [unilocular / multilocular , oval / round/ kidney shaped/ irregular shaped]
- 5- outline [well / poorly defined , smooth / ragged/ moth eaten/ corticated margin]
- 6- effect of lesion on adjacent teeth [ resorption, PDL widening, Lamina dura obliteration, displacement , delayed eruption ]
- 7- effect of lesion on adjacent bone [ bone expansion, resorption, displacement of structure like the inferior dental canal]
- 8- how long the lesion has been present



# DEVELOPMENTAL DEFECTS OF THE ORAL & MAXILLOFACIAL REGION

## **DEVELOPMENTAL DEFECTS OF THE LIP & PALATE**

- Central face develops  $\rightarrow$  end of 4<sup>th</sup> week of intrauterine life
- Upper lip develops  $\rightarrow$  6- 7<sup>th</sup> week of intrauterine life
- Primary palate  $\rightarrow$  forms by the fusion of the medial nasal processes
- Secondary palate  $\rightarrow$  formed from the maxillary processes at week 6 of intrauterine life
- Fusion of the palatal shelves begins anteriorly and proceeds posteriorly and end at week 12

Cleft lip: wedge shaped defect caused by the failure of fusion between the medial nasal process and the maxillary process

Cleft palate : oro nasal communication caused by the failure of fusion





## Cleft lip & palate:



between the 2 lateral portions of the palate





#### Causes of cleft lip and palate: [CL & CP]

- 1- Maternal alcohol and smoking
- 2- Maternal consumption of corticosteroids / anticonvulsants
- 3- Folic acid / riboflavin deficiency
- 4- Excess vitamin A
- 5- Relative ischemia to the areas and infections
  - CL +/- CP is more common in males but isolated CP is more common in females •
  - Most CL is **unilateral** and on the **left side** •

## Submucous palatal cleft :

- the mucosa is intact the cleft is in the muscles of the soft palate
- There is a notch in the bone along the posterior border of the hard palate





• Cleft uvula

## Lateral facial cleft :

- lack of fusion b/w maxillary and mandibular processes
- Extends from commissures to the ears → Macrosomia [ baby is larger than normal]

## **Oblique facial cleft:**

- Failure of fusion of the lateral nasal process and the maxillary process
- Extends from the upper lip to the eye
- Always associated with cleft palate

Median cleft of the upper lip: Failure of fusion of the medial nasal processes

## Congenital lip pits:

- A. Paramedian : might be associated with vander Woude Syndrome
- B. Commissural : might be cause by the failure of fusion b/w maxillary and mandibular processes [ might be a blind fistula or a dilated ectopic salivary gland]

**Double lip** : associated with non toxic thyroid enlargement + edema of the upper eyelid (*Ascher syndrome*).

# **DEVELOPMENTAL DEFECTS OF ORAL MUCOSA**

Fordyce granules: normal variation – bilateral small yellow maculo popular structures on the buccal mucosa – basically are ectopic sebaceous glands

Leukodema : Asymptomatic , bilateral diffuse translucent grayish white filmy apperance on the buccal mucosa – <u>disappear when the check is</u> <u>stretched</u> – no Tx needed









White spongy nevus : whitish thickening of the buccal mucosa – no Tx needed

# **DEVELOPMENTAL DEFECTS OF THE TONGUE**

## Macroglossia (Abnormal Large tongue):

- A. Congenital Macroglossia: in Down's syndrome, Hemangioma & Lymphangioma.
- **B.** Acquired Macroglossia: in Edentulism, Amyloidosis, Acromegaly & myesthenia gravis.

## Macroglossia leads to Noisy breathing, Drooling & difficulty in eating.

**Clinically:** Tongue has crenated lateral borders, patient will have open bite & mandibular prognathism.

## Lingual thyroid nodule:

- thyroid remnant in the region of the thyroid gland origin.
- smooth, sessile mass on mid-posterior dorsum of the tongue in the region of foramen caecum.
- Causes Dysphagia, Dysphonia, Dyspnoea & hypothyroidism.
- Diagnosed by iodine isotopes or Tecnetium -99m with CT or MRI.
- No treatment, periodic follow up

Fissured tongue: geographic tongue may cause fissured tongue – no Tx required just brush the tongue

## Black hairy tongue:

- caused by accumulation of keratin on the **filliform papillae** on the dorsum of the tongue + over growth of pigment-producing bacteria or fungi.
- Associated with Antibiotic therapy, poor oral hygeine, use of oxidizing mouth washes, overgrowth of bacteria or fungi.
- Patients may complain of **gagging** sensation or a bad taste in the mouth.













## Geographic tongue (Benign Migratory Glossitis):

- Multiple large, red, atrophic patches on the tongue with white, slightly raised borders on the dorsum of the tongue.
- resolve in days to weeks & papillae regenerate.
- The red areas are devoid of filiform papillae, whereas white areas show hypertrophy of papillae.
- Recurrent issue and the lesion appears to migrate from area to area.
- can be confused with more serious form of glossitis & even premalignant or malignant lesions.



# **DEVELOPMENTAL DEFECTS OF THE JAW BONES**

CONDITION	CHARACTERISTIC FEATURE [S] / NOTES
MICROGNATHIA	Small jaw Associated with Pierre robin sequence [Cleft palate + micrognathia + glossoptosis] Causes posterior displacement of the tongue and airway obstruction
MACROGNATHIA	Large jaw Associated with [ fibrous dysplasia, acromegaly , paget's disease]
CORONOID HYPERPLASIA	Results in limitation in mandibular movement Unilateral – caused by osteoma / osteosarcoma Bilateral – cause by endocrine influence during puberty
CONDYLAR HYPERPLASIA	Excessive growth of one condyle due to local circulatory problems, endocrine disturbances , trauma
CONDYLAR HYPOPLASIA	<b>Congenital :</b> associated with <b>Mandibulofacial dysostosis</b> & <b>Hemifacial Macrosomia</b> . <b>Acquired:</b> due to disturbances of growth center of the condyle due to trauma, radiation or <b>Rheumatoid arthritis</b>
BIFID CONDYLE	Double-headed mandibular condyle Anteroposterior bifid condyle : due to trauma in childhood. Mediolateral bifid condyle : due to abnormal muscle attachment.
BONY EXOSTOSIS	Torus platainus: bony protuberance in the midline of the vault of the hard palate Might have many shapes [ flat, nodular, lobular, spindle ]
	<b>Torus Mandibularis:</b> bony protuberance on the lingual aspect of the mandible <b>above the mylohyoid line in the region of the premolars</b> . [ mostly bilateral]
HEMI FACIAL HYPERTROPHY	unilateral enlargements of the face due to <b>increased neurovascular</b> <b>supply of the affected side</b> of the face [ asymmetry of the face with malocclusion & deviation of the towards the unaffected side]

HEMIFACIAL ATROPHY	Atrophic changes affecting one side of the face. The mouth & nose are deviated toward the defective side. The overlying skin often exhibit dark pigmentation
VRANIOFACIAL DYSOSTOSIS (CROUZEN SYNDROME)	Premature closing of the cranial sutures. Brachycephaly (short head) Scaphocephaly (Boat-shaped head) Trigonocephaly (Triangle-shaped head) Proptosis Maxilla is underdeveloped resulting in a mid-face hypoplasia.
MANDIBULOFACIAL DYSOSTOSIS ( TREACHER-COLLINS SYNDROME)	Hypoplastic zygoma +narrow face with depressed cheek & downward slanting of palpebral fissures. Underdeveloped mandible with retruded chin
CLEIDO CRANIAL DYSOSTOSIS	Patient has the ability to <b>appose the shoulders to near the midline of the chest</b> . Frontal & occipital bossing with enlarged head. primary dentition retained into adulthood+ Supernumerary teeth
EAGLE SYNDROME (STYLOHYOID SYNDROME)	Slender bony projection that originate from inferior aspect of temporal bone vague facial pain, especially during swallowing. Dysphagia, dysphonia, otalgia, headache, dizziness & Transient ischemic attack

# DEVELOPMENTAL ALTERATION IN THE NUMBER OF THEETH

ANDONTIA	TOTAL LACK OF TOOTH DEVELOPMENT ASSOCIATED WITH ECTODERMAL DYSPLASIA [ SKIN, HAIR, NAIL OR SWEAT GLANDS FAIL TO DEVELOP]
HYPODONTIA	Lack of development of one or more teeth From most to least commonly missing teeth : Third molar, second premolar & then lateral incisors. Usually seen in <b>Ehlers -Danlos, Down, Turner's syndrome</b>
OLIGODONTIA	Lack of development of <b>6 or more teeth.</b>
HYPERDONTIA [SUPERNUMERARY TEETH]	Mostly in the maxilla Most common site is maxillary <b>incisor area between centrals ( Mesiodens),</b> followed by accessory <b>fourth molar &amp; often called (Distomolar)</b> A supernumerary tooth situated lingually or buccaly to the molar teeth is called ( Paramolar). usually seen in <b>Cleido Cranial dysplasia &amp; Gardner's syndrome</b>
NATAL TEETH	Erupted decidous tooth at birth - mostly the mandibular incisors.
NEO NATAL	Erupted decidous tooth that appears in the first 30 days of life - mostly the mandibular incisors.



MICRODONTIA	Teeth are smaller than normal – assocciated with <b>hyodontia [ more in F]</b> <b>True generalized microdontia:</b> seen in Down's syndrome & Pituitary dwarfism. <b>Relative generalized microdontia:</b> when jaws are larger than normal, but teeth are of normal size <b>Isolated microdontia :</b> mainly involve <b>maxillary laterals ( Peg -shaped lateral) &amp; third</b> <b>molar.</b>
MACRODONTIA	Teeth are larger than normal – associated with hyperdontia [ more in M] True generalized macrodontia: in Pituitary Gigantism
GEMINATION	Extra wide crowns due to <b>development of two crowns from one tooth germ</b> More in the maxilla Single root canal
FUSION	union of two adjacent tooth germs by dentin during development. More in the mandible Separate canals Incisors & canines are most commonly affected by gemination & fusion If normal tooth count $\rightarrow$ the condition is gemination If tooth count is missing one $\rightarrow$ the condition is fusion
CONCRESCENCE	Union of roots of two or more normal teeth by cementum alone Most in posterior maxillary region
ACCESSORY CUSPS	<ul> <li>Talon cusps: on lingual aspect of maxillary incisors.</li> <li>Cusps of Carabelli: on palatal surface of the mesiolingual cusp of maxillary permanent molars.</li> <li>Dens evaginatus: A cusp -like of enamel located in the central groove or lingual ridge of the buccal cusp of permanent premolar or molar teeth</li> <li>Dens Invaginatus ( Dens in Dente) : Deep surface invagination of the crown that is lined by enamel.</li> </ul>
ENAMEL PEARLS	Localized bulge of enamel Mostly in molars
CERVICAL ENAMEL EXTENSION	buccal surface of the root, overlying the bifurcation. Most in mandibular molars
TAURODONTISM	molar with elongated crown & apically placed furcation of the roots, resulting in an enlarged rectangular coronal pulp chamber. Associated with : Ectodermal dysplasia, Klinefelter's syndrome , Down's syndrome
DILACERATION	Sharp bend or angulation of the root - results from <b>trauma during tooth development</b> (before 4 years of age). <b>Mostly affect maxillary anterior teeth which prevent its normal eruption.</b>
GLOBODONTIA	Gigantic globe-shaped teeth . Associated with hearing loss Diagnostic features of Otodental syndrome.
LOBODONTIA	Cuspid & premolars have fang-like cusps.

Dentiscope 2020 Page 13 of 74

AMELOGENESIS IMPERFECTA	<pre>hereditary defects of the enamel formation has 14 differnet subtypes can be : Hypoplastic= inadequate deposition of enamel matrix. Hypocalcified = defect in the maturation Hypomaturation = Enamel matrix is normal, but no significant mineralization occurs.</pre>
DENTINOGENESIS IMPERFECTA	Hypomineralized dentin - Normal appearance of enamel , but is weekly attached & tends to chip away from the dentin easily. Oblitrated pulp chamber & stunted roots. crowns of molars are bulbous with short roots. Tooth is uniformly brownish or purplish & abnormally translucent Type I: It occurs in patients affected with osteogenesis imperfecta Type II: called ( Hereditary Opalescent Dentin). Type III: Called Brandy wine type.
SHELL TEETH	Varient of dentinogenesis imperfecta mostly in deciduous teeth.
DENTIN DYSPLASIA (ROOTLESS TEETH)	Type I (Radicular Dentin Dysplasia) : Permanent molars have characteristic W - shaped roots with pulp obliteration.
	<b>Type II (Coronal Dentin Dysplasia):</b> Oblitrated pulp chamber& canals, Roots are normal in shape , <b>Pulp stones</b>
REGIONAL ODONTODYSPLASIA (GHOST TEETH)	Teeth show marked decrease in radiodensity (Ghost teeth). Enamel & Dentin are very thin & indistinct. Pulp chambers are extremely large with occasional pulp stones
TURNER'S HYPOPLASIA	seen in permanent teeth secondary to periapical inflammatory diseases or traumatic injury of the deciduous teeth. Resulting in focal areas of white, yellow or brown discoloration Mostly seen in permanent premolars & maxillary centrals
SYPHILITIC HYPOPLASIA	Congenital syphilis → Hutchinson's Incisors [ a screw-driver shape & constricted incisal edge with hypoplastic notch.] + Mulberry Molars [ disorganized surface anatomy that resembles a mulberry.]

Dentiscope 2020 Page 14 of 74



CONGENITAL ERYTHROPOIETIC PORPHYRIA	Teeth shows marked <b>red-brown discoloration that exhibits red flour</b> <b>exposed to UV light.</b>	escence when
HYPER BILLIRUBINEMIA	sharp dividing line separating green portion (formed during hyper billirubinemia) from normal colored portion (formed after normal level of billirubin is restored).	

## **INFLAMMATORY BONE CONDITIONS**

## DIVIDED BROADLY INTO : OSTEITIS , OSTEOMYELITIS AND PERIOSTITIS

DISEASE	Characteristic features
ALVEOLAR OSTEITIS ( DRY SOCKET)	<ul> <li>Postoperative pain in and around extraction site, which increases in severity 1-3 days after extraction</li> <li>Caused by either failure of a blood clot to form in the socket, or premature loss or disintegration of the clot</li> </ul>
	Failure of a clot to form may be due to:
	1. Excessive extraction trauma
	2. Limited local blood supply
	3. Excessive use of Local anestnesia and excessive imgation of the alveolus
	5 Smoking
	6. Oral Contraceptives
	Risk factors:
	1. Previous experience of Alveolar Osteitis
	2. Deeply impacted mandibular third molar
	3. Poor oral hygiene
	4. Active or recent history of acute ulcerative gingivitis or pericoronitis
	associated with tooth to be extracted.
	5. Immuno-compromised individuals
	In cases where an adequate blood clot forms it might :
	1- Get washed away by excessive mouth rinsing
	2- disintegrate prematurely due to fibrinolysis of the clot most likely as a
	result of infection
	<ul> <li>highest incidence of dry socket follows the extraction of impacted lower third molars</li> </ul>
	Dry socket content:
	• Food dehris salive and hacteria collect in the empty socket the hone of
	which becomes infected and necrotic.



• Healing is extremely slow

**Clinical Features:** 

- Severe pain developing a few days after the extraction.
- foul tasting and smelling decomposing food debris which can be washed away to reveal the denuded bone lining the cavity.

#### Prevention:

- Avoid excessive trauma + Confirm the presence of blood clot after exo
- Encourage patient to stop or limit smoking in the immediate post op period.
- o Advice patient to avoid vigorous mouth rinsing for the first 24 hrs post exo
- Preop administration of antibacterial mouthwash

#### Management

A dry socket will heal with time - Local therapy therefore aims at keeping the area clean allowing connective tissue to fill in defect.

wound irrigation and intra alveolar dressing (antibacterial, topical anaesthetic or combination)

FOCAL SCLEROSING (CONDENSING) OSTEITIS	ASYMPTOMATIC RESULTS FROM LONG TERM LOW GRADE IRRITATION INCREASED RADIO OPACITY AT THE APEX OF THE TOOTH TREATMENT: AFFECTED TOOTH SHOULD BE TREATED OR EXTRACTED BIOPSY TO RULE OUT METASTATIC MALIGNANCY.	
ACUTE SUPPURATIVE OSTEOMYELITIS	Caused by nearby infection [ extraction , PA infection etc through the jaw Mandible is more commonly affected than maxilla [ because of it's poor blood supply]	



ACUTE SUPPURATIVE OSTEOMYELITIS	<ul> <li>Caused by nearby infection [ extraction , PA infection etc] → infection spreading through the jaw</li> <li>Mandible is more commonly affected than maxilla [ because of it's poor blood supply]</li> <li>necrotic bone (a sequestrum) which is bathed in pus becomes separated from the surrounding vital bone</li> <li>After 10-14 days sufficient bone resorption may have occurred to produce <i>irregular</i>, <i>moth-eaten areas of radiolucency</i>.</li> </ul>
	Treatment
	<ol> <li>Bacterial sampling &amp; culture → Vigorous antibiotic treatment.</li> <li>Drainage + Debridement</li> <li>Remove source of infection if possible.</li> <li>Sequestrectomy</li> <li>Hyperbaric oxygen</li> </ol>



CHRONIC SUPPURATIVE OSTEOMYELITIS	Inadequately treated acute osteomyelitis Chronic suppuration and discharge of pus through one or more intraoral or extraoral sinuses. Radiolucency with focal areas of opacity [Moth eaten appearance] Treatment: Sequestrectomy / Decortications if necessary
CHRONIC FOCAL SCLEROSING OSTEOMYELITIS	diffuse sclerosing lesions of the mandible due to spread from low-grade infection/inflammation such as a periapical granuloma or periodontal diseases . <b>Treatment:</b> Elimination of the source of inflammation (exo or endo). ✓
DIFFUSE SCLEROSING OSTEOMYELITIS	Asymptomatic , sometimes vague pain & foul smell could be experienced <b>Cotton wool appearance</b> Treatment: eliminate source of infection but sclerotic areas remain radiographically
	Cotton wool appearance DD: ✓ Paget's disease ✓ Osteopetrosis ✓ cementoma ✓ Late stage of FD
CHRONIC OSTEOMYELITIS WITH PROLIFERATIVE PERIOSTITIS (GARRE'S OSTEOMYELITIS, PERIOSTITIS OSSIFICANS)	<ul> <li>mandible in children and young adults</li> <li>Bony hard swelling on the outer surface of the mandible.</li> <li>Overlying mucosa and skin normal</li> <li>Radiographically : Concentric layers (Onion skin appearance)</li> <li>Treatment: eliminate source of infection</li> </ul>
Osteoradionecrosis	Infection may spread rapidly through the irradiated bone, resulting in extensive osteomyelitis / necrosis of the bone + sloughing of the overlying oral and facial soft tissues <b>Treatment</b> Removal of necrotic bone Hyperbaric treatment



Osteochemonecrosis	Associated with bisphosphonate administration for the treatment of osteoporosis and osteopenia, Paget's disease and Multiple myeloma • Painless exposed bone	
	Treatment	
	Prevention of infection is paramount	
	Surgery Increases risk of further necrosis	
	<ul> <li>Hyperbaric treatment- Not effective</li> </ul>	
	Palliative treatment	
	<ul> <li>Identification of patients at risk</li> </ul>	
	<ul> <li>Avoid extractions</li> </ul>	

# **METABOLIC BONE CONDITIONS**

PAGET'S DISEASE	ABNORMAL AND UNCONTROLLED INCREASE IN THE
OSTEITIS	OSTEOCLASTIC AND OSTEOBLASTIC ACTIVITY OF THE BONE
DEFORMANS]	CELLS OF OLDER ADULTS RESULTING IN :
-	> LARGER BUT WEAKER BONES.
	> EXTENSIVE PAIN
	INCREASE TENDENCY TO DEVELOP MALIGNANT BONE
	NEOPLASM
	IUMBER VERTEBRAF, PELVIS, SKULL AND FEMUR ARE THE
	MOST COMMONIX AFFECTED BONES
	[ ONLY A SMALL DERCENTAGE AFFECTS THE JAWS]
	- WEIGHT BEARING BONES OFTEN SHOW BOWING DEFORMITY, RESULTING IN
	WHAT IS DESCRIBED AS <u>SIMIAN ( MONKEY- LIKE) STANCE</u> .
	– PROGRESSIVE INCREASE IN HEAD CIRCUMFERENCE → RESULT IN A " LION-
	LIKE" FACIAL DEFORMITY ( LEONATOSIS OSSEA)
	<ul> <li>PATIENTS WEARING FULL DENTURES MAY</li> </ul>
	COMPLAIN OF DIFFICULTY WEARING THEM AND
	REQUIRE FREQUENT CHANGES + WIDENING OF
	ALVEOLAR RIDGE + FLATTENED PALATAL VAULT

INCREASED SPACING AND LOOSENING OF TEETH



1.86



RADIOGRAPHICALLY :

EARLY STAGE : DECREASE BONE DENSITY AND ALTERED TRABECULAR PATTERN , PARTICULARLY IN SKULL ( OSTEOPOROSIS CIRCUMSCRIPTA) LATE STAGE : COTTON WOOL APPEARANCE TEETH HAVE ROOT RESORPTION + HYPERCEMENTOSIS

 BONE SCINTIGRAPHY: IF MANDIBLE IS AFFECTED: MARKED UPTAKE FROM CONDYLE TO CONDYLE (BLACK BEARD OR LINCOLIN'S SIGN)





- ELEVATED LEVELS OF <u>SERUM ALKALINE PHOSPHATAS</u>E AND URINARY HYDROXYL PROLINE.
- ✓ NORMAL SERUM CALCIUM AND PHOSPHOROUS LEVELS.

A CHARACTERISTIC MICROSCOPIC FEATURE : REVERSAL LINES WHICH REPRESENTS ALTERNATING RESORPTIVE AND FORMATIVE PHASE OF THE BONE, THIS RESULT IN (JIGSAW PUZZLES) OR (MOSAIC APPEARANCE) OF THE BONE.



#### MANAGEMENT :

- **BONE PAIN :TX ASPIRIN OR ANOTHER ANALGESICS.**
- NEUROLOGIC COMPLICATIONS SUCH AS DEAFNESS OR VISUAL DISTURBANCES MAY RESULT FROM BONY ENCROACHMENT ON CRANIAL NERVES PASSING THROUGH SKULL FORAMINA.
- USE OF PARATHYROID HORMONE ANTAGONIST SUCH AS CALCITONIN AND BISPHOSPHONATES CAN REDUCE BONE TURN OVER
   DIFFICULT EXTRACTION OF GROSSLY HYPERCEMENTOSED TEETH, EDENTULOUS
   PATIENTS REQUIRES NEW DENTURES PERIODICALLY.

DEVELOPMENT OF OSTEOSARCOMA IS A RECOGNIZED COMPLICATION ESPECIALLY IN PELVIS AND LONG BONES.



OSTEOPORISOS	↓ density of bone & ↓ quantity of bone. defective quality not quantity of bone - defective quantity of bone in severe cases. Mostly postmenopausal women Radiograph: increased radiolucency of bone, the cortex is thinned, and there are more marrow spaces with thin trabeculae. mandible reduced to a thin fragile strip of boneImage: Comparison of the second secon
HYPERPARATHYROI DISM	<ul> <li>raised serum calcium, PTH and ALP, but low serum</li> <li>Phosphorous <ul> <li>Subperiosteal resorption of bone of fingers.</li> <li>In severe cases (Oseitis fibrosa cystica): multilocular radiolucent cyst-like areas are seen.</li> <li>Large destructive radiolucency may be present which is indicative of giant cell tumor of hyperparathyroidism (Brown tumor).</li> </ul> </li> <li>Microscopically : Extensive hemosiderine deposits cause the lesion to appear as a brown tumor.</li> </ul>
CENTRAL GIANT CELL GRANULOMA ( GIANT CELL LESIONS; GIANT CELL TUMOR)	Mainly anterior part of the mandible + crosses the midline Asymptomatic [ chance radiographical lesion] Unilocular RL can be mistaken as PA granuloma Multilocular RL can be mistaken as ameloblastoma Microscopically : multinucleated giant cells + foci of osteid + RBC extravasation TX: curettage [ high chance of recurrence ] Aggressive lesions : Corticosteroids, calcitonin & IFN-α2a DD of GC lesions :

OSTEOPETROSIS "MARBLE BONE DISEASE"	<ul> <li>Marked increase in bone density due to failure in osteoch function</li> <li>Infentile osteopetrosis</li> <li>Begins in infancy → breathing &amp; hearing difficulties, due oversized facial and mastoid bones, followed by function defects in ocular &amp; trigeminal nerves as they compressed sclerosis of foramina of base of skull.</li> <li>Replacement of bone marrow with dense bone which lead to pancytopenia → pts die because of bone marrow depletion</li> <li>Adult osteopetrosis = asymptomatic</li> <li>Sinuses are reduced in size + cranial plate is thickened</li> <li>Microscopically : bone is dense &amp; sclerotic, with most of marrow spaces replaced with bone</li> </ul>	ast to al by
OSTEOGENESIS IMPERFECTA	inability of the bone matrix to fully mineralize multiple broken bones blue sclera of the eyes dentinogenesis imperfecta short deformied extremities + All patients have spinal scoliosis. Microscopically : increased number of osteoblasts, osteo decreased mineral content	lasts and osteocytes with



## **BENIGN FIBRO OSSEOUS LESIONS**

## - FIBROUS TISSUE REPLACING BONE

DISEASE	Characteristic features
PERIAPICAL CEMENTO- OSSEUS DYSPLASIA ( PERIAPICAL CEMENTOMA)	Multiple lesions affecting the <b>lower anterior region Mostly in black females</b> Asymptomatic + teeth are vital No tx needed
FLORID CEMENTO-OSSEUS DYSPLASIA	Black women Involves all 4 quadrants of the jaws Dentulous and edentulous areas are involved Treatment : Asymptomatic : periodic recall with maintenance of good oral hygiene Symptomatic: infection can lead to chronic osteomyelitis → give ABX [ but they are ineffective ] best is to do Saucerization of dead bone and cementum
FIRBOUS DYSPLASIA	Mostly are monostotic affecting one bone only Polyostotic affecting 3/4 of entire skeleton is called <u>Jaffe type</u> . Polyosotic FD also have multiple area of cutaneous pigmentation + hyperfunction of one or more endocrine glands . [McCune Albright syndrome] Mandibular lesions are truly monostotic, while maxillary lesions often involve adjacent bones, such as zygoma, sphenoid and occiput. [Craniofacial FD] Diagnostic radiographic feature is a fine "ground glass"
	Microscopically : Irregularly shaped woven bone trabeculae - not connected with each other. Treatment : Cosmetic correction after skeletal bone maturation
CHERUBISM	Bilateral expansion of the posterior mandible [ chubby facial appearance ] Maxillary involvement causes → " eye upturned to heaven" appearance.

Radiographically : *Multilocular expansile radiolucencies* 

Miscroscopically :

Multi nucleated giant cells Characteristic feature = Eosinophilic cuffing surrounding small blood vessels.(characteristic)

Treatment = Curettage Radiation therapy is contraindicated because of the risk of development of post irradiation sarcoma.





## **NEOPLASTIC BONE LESIONS**

DISEASE	CHARACTERISTIC FEATURES
OSTEOMA	Osteomas are usually solitary Multiple osteomas of the jaws occur as a feature of Gardner syndrome Gardner's syndrome = Polyposis coli + multiple jaw osteomas + Epidermal/sebaceous cysts + Multiple impacted supernumerary
OSTEOBLASTOMA & OSTEOID OSTEOMA	Osteoid Osteoma: Contain tumor nidus with a concentration of peripheral nerves → significant pain " relieved by Aspirin".
Ossifying (cemento- ossifying) fibroma	Resembles fibrous dysplasia but it very well demarcated Occurs in premolar molar region fibrous dysplasia is <u>homogeneously radio-opaque with a ground-glass</u> <u>appearance</u> and <u>poorly defined margins</u> Ossifying Fibroma appears as a unilocular <u>mixed radiolucent and</u> <u>radio-opaque</u> lesion with <u>well-defined borders</u> . Ossifying fibroma has calcified bodies
HEMANGIOMA	Radiograph : honey comb appearance         Aspiration will reveal fresh blood         Most hemangiomas of bone are of the cavernous type         Image: Comparison of the cavernous type         Image: Compariso
OSTEOSARCOMA	Commonest primary malignant tumor of bone but is relatively rare in the jaws. Osteosarcoma incidence is increased in : Paget's disease, retinoblastoma, radiation therapy



	<ul> <li>bony hard swellings of the buccal &amp; lingual cortices, with or without pain &amp; often associated with separation of teeth &amp; parasthesia in mental nerve area.</li> <li>Some appear as soft tissue epulides termed as " Juxtacortical OS"</li> <li>Radiographically : <ul> <li>Symmetrical widening of PDL space in adjacent teeth.</li> <li>spiking root resorption</li> <li>" a sun burst pattern"</li> <li>Triangular elevation of periosteum (Codman's triangle).</li> </ul> </li> <li>Microscopically : abnormal osteoid formation by malignant osteoblasts <ul> <li>TX: Preoperative chemotherapy, followed by post-operative chemotherapy.</li> </ul> </li> </ul>
MYELOMA	composed of plasma cells and involves multiple bone [multiple myeloma] – causes high levels of immunoglobin [IgG] paraproteins or 'M' components appear in serum Bence Jones Proteins in urine amyloid deposition in tongue causing macroglossia characteristic radiographic appearance = sharply demarcated, round
	or oval osteolytic lesions [ nunched-out annearance ]
	Micrscopically : Monotonus sheets of myeloma cells - resemble mature plasma cells
	Tx: chemotherapy + Bone marrow
	transplant
LANGERHANS CELL HISTIOCYTOSIS	Hand-Schuller-Christian syndrome multifocal eosinophilic granulomas involving the craniofacial bones, orbit, and posterior pituitary skull defects exophthalmos diabetes insipidus
	The disseminated form of Langhans cell histiocytosis is <b>Letterer-Siwe</b> <b>disease</b> . [in infants and children under 2 years of age and has a high mortality]

	Multiple eosinophilic granuloma [ a type of langerhanz cell histiocytosis] → teeth appear <u>floating in air.</u> <u>Microscopically : Birbeck granules</u>	
	Tx: Intra lesional corticosteroids (localized). Chemotherapy (dissiminated)	
EWING SARCOMA	Slight to moderate fever, leucocytosis & increase ESR." Misdiagnosed with ostomyelitis". pain with rapid swelling + loosening of teeth + parasthesia radiograph : The involved bone appears "moth- eaten" + The periostium often has a la to as an "onion-skin" reaction. Microscopically : neuroectodermal cells <i>Tx:</i> Combined Surgery, radiotherapy & muti drug chemotherapy. Prognosis depend on anatomic location of the lesion. "pelvic lesions has poorest progno	with the second seco

- The most common primary tumors reported as metastasizing to the jaws are carcinomas of the breast, bronchus, prostate, thyroid and kidney
- Most metastatic tumors are osteolytic but carcinomas of prostate & breast, may be osteoblastic and appear radiographically as an area of radiopacity rather than radiolucency
- common sites for metastases to the oral mucosa are the **gingiva or alveolar mucosa, followed by the tongue**.
- **Common feature of metastatic tumors = anaesthesia of the lip** due to involvement of the inferior **dental** nerve," numb-chin syndrome".





# CYSTS

## Cysts can be :

- Epithelial lined (True cysts)
- Non –epithelial lined (pseudocysts)

Cysts		
Inflammatory	Developmental	
1- Radicular : a consequence of PA	Odontogenic	Non odontogenic
pathology [apical, lateral, residual]	1- Dentigerous	Nasopalatine duct
2- Paradental cyst	2- Eruption	cyst
	3- OKC	Nasolabial cyst
	4- Lateral Periodontal	Median Palatal
	5- Gingival	
	6- Glandular	

CYST ORIGIN	CYSTS
RESTS OF MALASSEZ	PA cyst
	Residual cyst
REDUCED ENAMEL EPITHELIUM – REE	Dentigerous cyst
	Eruption cyst
DENTAL LAMINA	ОКС
	Lateral periodontal cyst
	Gingival cyst of adult
	Dental lamina cyst of newborn
	Glandular cyst
UNCLASSIFIED	Paradental



# **INFLAMMATORY CYST**

CONDITION	CHARACTERISTIC FEATURES
RADICULAR CYST	Most common cyst         Always associated with apex of non vital tooth         Consequence of PA pathology         Small cyts → asymptomatic         Large cyst → bony expansion + discharge through a sinus         Eggshell crackling on palpation         Sometimes can perforate the cortex and produce a bluish submucosal swelling         RG= round or oval radiolucency at the apex that is continuous with the LD + peripheral radio opaque margin         ** if the margin is very radio opaque = indicates chronicity of the lesion         Tx= enucleation
RESIDUAL CYST	Radicular cyst that remained in the jaw following extraction of the involved tooth
LATERAL RADICULAR CYST	RARE - Due to extension of inflammation from the pulp into the periodontium by the lateral canals Commonly misdiagnosed with lateral periodontal cyst [ lateral periodontal cyst is associated with a vital tooth , lateral radicular cyst is associated with non vital tooth ]



**Q: explain the hypertonicity of the cyst content in radicular cysts ?** because it contains degenerating epithelia, inflammatory cells, tissue components and serum proteins





# **DEVELOPMENTAL CYST(ODONTOGENIC)**



	<ul> <li>Odontogenic myxoma</li> <li>Non-odontogenic lesions eg. central giant cell granuloma</li> <li>Tx: Surgical enucleation or in severe cases surgical resection.</li> </ul>
LATERAL PERIODONTAL CYST	Mainly in canine premolar region <b>Vital tooth</b> polycystic <b>"Bortryoid odontogenic cyst"</b> can be seen which represents simultaneous cyst changes in multiple adjacent rests of dental lamina. <b>Tx: enucleation</b>
GLANDULAR ODONTOGENIC CYST	Variable number of small glandular structures or microcysts within the lining epith goblet-like mucous secreting cells

### **Q: mention reasons of OKC growth and expansion in a specific manner?**

- 1. Hydrostatic forces
- 2. Keratocyst contents are hypertonic
- 3. Active epithelial growth
- 4. Production of bone resorbing factors
- 5. Accumulation of mural squames

#### Q: what causes the high recurrence rate of OKC?

- Thinness of the cyst wall & its low tensile strength & rupture → retention of fragments of torn lining.
- 2. Presence of daughter cysts in cyst wall.
- 3. Focal separation of epith. Lining from underlying C.T make surgical removal very difficult.

#### Gorlin goltz syndrome :

- 1- Multiple OKC of jaws.
- 2- Multiple Basal cell carcinoma of skin.
- 3- Bifid ribs & vertebral deformities.
- 4- Calcification of Falx cerebri.
- 5- Palmer & planter dyskeratosis.
- 6- Frontal bossing.
- 7- Hypertelorism.
- 8- Ovarian fibromas.





## NON EPITHELIAL PRIMARY BONE CYSTS

CONDITION	CHARACTERISTIC FEATURES
SOLITARY BONE CYCT SIMPLE OR TRAUMATIC OR HEMORRHAGIC BONE CYST	in premolar & molar regions of mandible. Rough bony-walled cavity devoid of any detectable soft tissue lining. The cavity is <b>empty</b> or with little clear or blood stained fluid. Rapid healing follow surgical exploration & cyst resolve spontaneously with time. RG = Scalloping around & between roots of standing teeth
ANEURYSMAL BONE CYCT (ABC)	RG = Scalloping around & between roots of standing teeth Rare, arise in posterior part of the body or angle of the mandible RG = Multilocular radiolucency with a characteristic ballooned-out appearance due to gross cortical expansion. MS = • Numerous, non-endothelial lined blood-filled spaces • Multinucleated giant cells & evidence of old & recent hemorrhage Can be produced by Fibrous Dysplasia or Central Giant cell granuloma Or trau
STAFNE'S IDOPATHIC CAVITY	Round or oval well-demarcated radiolucency between premolar region & angle of jaw just below inferior dental canal. Sialography is useful in identification RG: Saucer-shaped depression or concavity of varying depth on lingual aspect of the mandible.





# DEVELOPMENTAL CYSTS

CONDITION	CHARACTERISTIC FEATURES
EPSTIEN'S PEARLS	As the palatal shelves meet & fuse in the midline to form secondary palate, small epithelia may become entrapped leading to a cyst <b>anterior part of median palatal raphe</b>
BOHN'S NODULES	Arise from epithelial remnants of the minor salivary gland of palate → cysts present scattered over hard palate & near soft palate cluster of 206 cysts are observed Keratin-filled cyst Tx: No treatment is required [ self Healing within several weeks as the covering epith degenerates, the cyst rupture onto mucosal surface & eliminate their keratin contents]
NASOLABIAL CYST (NASOALVEOLAR CYST, KLESTADT CYST)	IN THE UPPER LIP LATERAL TO THE MIDLINE. CONSIDERED AS "FISSURAL" CYST ARISE FROM EPITHELIAL REMNANT ENTRAPPED ALONG THE LINE OF FUSION OF MAXILLARY, MEDIAL & LATERAL NASAL PROCESSES. OR IT IS THOUGH TO DEVELOP FROM MISPLACED EPITHELIA OF THE NASOLACRIMAL DUCT BECAUSE OF THEIR SIMILAR LOCATION & HISTOLOGY. CLINICALLY: SWELLING OF THE UPPER LIP LATERAL TO THE MIDLINE, RESULTING IN ELEVATION OF THE ALA OF THE NOSE. NO RADIOGRAPHIC PICTURE IS SEEN FOR THIS CYST, SINCE IT IS IN THE SOFT TISSUE. MS = RESPIRATORY EPITHELIUM [PSEUDOSTRATIFIED COLOMINAR CILIATED EPITHELIUM WITH GOBLET CELLS] TX: SURGICAL EXCISION OR MARSUPIALIZATION
GLOBULOMAXILLARY CYST	USED TO BE CONSIDERED A FISSURAL CYST BUT NOW CONSIDERED OF ODONTOGENIC ORIGIN BETWEEN MAXILLARY LATERAL INCISOR &CUSPID TEETH [ CANINE] WELL-CIRCUMSCRIBED UNILOCULAR RADIOLUCENCY AS AN INVERTED PEAR BETWEEN & APICAL TO THE TEETH

MEDIAN PALATAL CYCT	CAUSED BY EPITHELIA ENTRAPPED ALONG THE LINE OF FUSION OF THE LATERAL PALATINE SHELVES OF MAXILLA. DIFFICULT TO DISTINGUISH FROM NASOPALATINE DUCT CYST, SINCE MOST MEDIAN PALATAL CYSTS MAY REPRESENTS POSTERIORLY POSITIONED NASO-PALATINE DUCT CYST.
	ASYMPTOMATIC - FLUCTUANT SWELLING OF THE MIDLINE OF THE HARD PALATE RG= WELL- CIRCUMSCRIBED RADIOLUCENCY IN THE MIDLINE OF THE HARD PALATE.
	SURGICAL REMOVAL
NASOPALATINE DUCT CYST(INCISIVE CANAL CYST)	MOST COMMON NON-ODONTOGENIC CYST OF ORAL CAVITY CAUSED BY CYSTIC DEGENERATION OF THE EPITHELIAL REMNANT OF THE NASOPALATINE DUCT SWELLING OF THE ANTERIOR PALATE RG= WELL-CIRCUMSCRIBED RADIOLUCENCY (ROUND OR OVAL) IN OR NEAR THE MIDLINE OF THE ANTERIOR MAXILLA BETWEEN & APICAL TO THE CENTRAL INCISORS. ** DIFFICULT TO DISTINGUISH FROM A LARGE INCISIVE FORAMEN [ NORMAL SIZE OF INCISIVE FORAMEN IS MAX 6 MM ] A RADIOLUCENCY THAT IS 6MM OR SMALLER IN THIS AREA $\rightarrow$ NORMAL FORAMEN, UNLESS OTHER CLINICAL SIGNS & SYMPTOMS ARE PRESENT.
	NASOPALATINE DUCT CYST MAY DEVELOP IN SOFT TISSUE OF INCISIVE PAPILLA WITHOUT BONY INVOLVEMENT [CYST OF INCISIVE PAPILLA] → BLUISH DISCOLORATION AS A RESULT OF FLUID CONTENTS IN THE CYST LUMEN. MS= DEPENDS ON THE VERTICAL POSITION OF THE CYST : CYST IN THE SUPERIOR ASPECT OF THE CANAL NEAR NASAL CAVITY → RESPIRATORY EPITH CYSTS IN THE INFERIOR PORTION NEAR THE ORAL → SQUAMOUS EPITH CYST WALL ALSO CONTAINS NEUROVASCULAR BUNDLE TX: SURGICAL ENUCLEATION + BIOPSY


# MEDIAN USED TO BE CONSIDERED A FISSURAL CYST BUT NOW MANDIBULAR CYST CONSIDERED OF ODONTOGENIC ORIGIN RG = MIDLINE RADIOLUCENCY BETWEEN & APICAL TO THE MANDIBULAR CENTRAL INCISORS WITH CORTICAL EXPANSION



DERMOID CYST	Occur in the midline of mouth region & represents the <b>minimal manifestation of</b> <b>Tratoma/Dermoid cyst/Epidermoid cyst spectrum.</b> Tx: surgical removal
THYROGLOSSAL DUCT CYST	Caused by epithelial remnant of the thyroglossal duct [ as the thyroid gland descends from it's origin in the tongue to it's final position] Develops in the midline & may occur anywhere from the foramen cecum area of the tongue to the substernal notch - In most cases the cyst develop below hyoid bone. Painless, fluctuant movable swelling - moves vertically during swallowing or protrusion of the tongue. MS= Thyroid tissue may occur in cyst wall. Tx: Siistrunk procedure Q: what represents the remnant of the thyroid gland on the tongue? The foramen cecum
CERVICAL LYMPHOEPITHELIAL CYST (BRANCHIAL CLEFT CYST)	Caused by remnant of the 2 <sup>nd</sup> branchial clefts during 4 <sup>th</sup> week of gestation. Or from cystic changes in parotid gland epith. That become entrapped in the upper cervical lymph node during embryo life. Soft fluctuant mass located in the upper lateral neck along the anterior border of the sternocleidomastoid muscle. MS= Cyst wall = lymphoid tissue with germinal center formation Tx: surgical removal

Dentiscope 2020 Page 36 of 74



ORAL LYMPHOEPITHELIAL CYST	MICROSCOPICALLY SIMILAR TO CERVICAL LYMPHO EPITHELIAL CYST, BUT MUCH SMALLER IN SIZE WHITE OR YELLOW & OFTEN CONTAINS <u>CREAMY</u> <u>OR CHEESY KERATINACEOUS MATERIALS</u> IN THE LUMEN. ASYMPTOMATIC & AFFECT THE FLOOR OF THE MOUTH MS = OF LYMPHOID TISSUE IN THE CYST WALL WITH TX: SURGICAL REMOVAL	GERMINAL CENTER FORMATION.
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#### To differentiate the median palatal cyst from other cystic lesion of the maxilla:

- 1. Symmetrical along the midline.
- 2. Ovoid or circular.
- 3. Located posterior to palatine papilla.
- 4. Not associated with non-vital teeth.
- 5. No microscopic evidence of large NV bundles, hyaline cartilage or minor salivary glands in the wall of the cyst.



## **ODONTOGENIC TUMORS**

CONDITION	CHARACTERISTIC FEATURES				
ΔΜΕΙΟΒΙΔΣΤΟΜΔ	Most common Odontogenic epithelial tumor				
ANILLODLASTONIA	Arise from remnants of dental lamina, reduced enamel epithelium , rest of Malassez,				
	basal cell layer overlying surface epithelium				
	locally invasive, but does not metastasize.				
	Three types of Ameloblastoma:				
	A. Common (Follicular, Polycystic) Ameloblastoma. Posterior mandible and ascending ramus				
	Most common type				
	Characteristic feature: "Eggshell crackling" because				
	ameloblastoma can expand the bony cortices, but due				
	to their slow growth the periosteum can produce a thin				
	shell of bone .				
	RG= Multilocular radiolucency or soap bubble				
	appearance				
	MS = Epithelial processes composed of well-organized				
	single layer of Ameloblast-like cells with "Reversed				
	polarity" which surround loosely arranged polyhedral				
	or angular cells resembling stellate reticulum.				
	1 1. Berlin 199				
	Histologic patterns are:				
	1. Follicular Pattern:				
	Most prevalent, resembling the <u>earlier stages of</u>				
	tooth development.				
	Palisaded ameloblast-like cells with reversed polarity +				
	centrally a stellate reticulum-like cells + microcysts.				
	2. Plexiform Pattern:				
	Epithelium in "fishnet" or mesh arrangement.				
	at the second of				
	3.Acanthomatous Pattern:				
	Central epithelial cells transform into squamous cells				
	that produce keratin within individual cells or in the				
	form of keratin pearls.				
	4. Granular cell Pattern:				
	sheets of large eosinophilic granular cells.				

Done By : Sima Habrawi Edit By : Haif AlQahtani Dentiscope 2020 Page 38 of 74



#### 5. Basal cell Pattern:

- darkly stained cells with little evidence of palisading at periphery.
- They have mistaken for basal cell carcinoma.

#### 6. Desmoblastic Pattern:

- The epithelial component is widely separated by fibrous tissue that is dense & scar-like.
- has a mixed radiolucent/radiopaque radiographic appearance that resembles Fibro-osseus lesions
- It is more difficult to treat, because it penetrates the surrounding bone trabeculae & remains undetected.

#### [These histological variants do not affect tumor behavior]

B. Unicystic Ameloblastoma: large unilocular cyst commonly associated with the crown of an impacted tooth [mostly with a severely displaced mandibular third molar]

**RG** =unilocular radiolucency with well-demarcation & even corticated

#### Three histological variants:

- 1. Luminal Unicystic Ameloblastoma: fibrous C.T capsule surrounding a large fluid-filled lumen + cytoplasmic vacuolization.
- 2. Intraluminal Unicystic Ameloblastoma:One or more nodules of ameloblastoma project from cyst lining to the lumen mostly of plexiform type
- 3. Intramural Unicystic Ameloblastoma: The fibrous cyst wall is infiltrated by typical *follicular or plexiform* ameloblastoma

C. Peripheral Ameloblastoma [least common] Rare, Limited to the soft tissue of the posterior gingiva Resemble Pyogenic granuloma or fibroma

RG = Only superficial saucerization of the cortical plate.

Tx of all Ameloblatsomas : Local excision to Block resection















Dentiscope 2020 Page 39 of 74



ADENOMATOID ODONTOGENIC TUMOR (AOT)	ORIGINATES FROM REDUCED ENAMEL EPITHELIUM RG= UNILOCULAR RADIOLUCENCY WITH WELL-CORTICATED BORDERS THAT SURROUND THE CROWN OF IMPACTED TOOTH (CUSPID), LIKE DENTIGEROUS CYST & THE DIFFERENCE BETWEEN THEM ARE: • RADIOLUCENCY EXTENDS APICALLY BEYOND CEJ. • THE PRESENCE OF FLECKS OF RADIOPACITIES (SNOWFLAKE). MSE • EPITHELIA IN DUCTAL LIKE PATTERNS [MICROCYSTS RESEMBLING DUCT CUT IN CROSS SECTION (THEY ARE NOT DUCTS & ARE NEVER SEEN CUT LONGITUDINALLY) LINED BY CELLS SIMILAR TO AMELOBLASTS. • SPHERICAL CALCIFICATION TX: ENUCLEATION • Calcified • Ca	
CALCIFYING EPETHELIAL ODONTOGENIC TUMOR (PINDBORG TUMOR) CEOT	Coriginats from dental lamina A/O REE could be mistaken for a poorly differentiated carcinoma. Affects posterior body of the mandible MS= squamous & clear cells that spherical calcification amyloid staining & hyaline deposits lack of stromal inflammatory reaction Concentric spherical calcifications ( Liese-gang ring calcifications) ** It differs from ameloblastoma by: • of epithelial cells do not resemble ameloblasts. • contains <u>spherical diffuse calcification</u> RG= uni or multi locular radiolucency with <u>scalloped</u> margins with flecks of calcified structure around crown of impacted mand. molar Differential Diagnosis: • Dentigerous cyst • Adenomatoid odontogenic tumor • Ameloblastic Fibroodontoma	



Calcifying odontogenic cyst/ tumor ( GHOST CELL CYST)	<pre>contains "ghost cells" &amp; "Spherical calcification" It affects mostly areas anterior to the first molar RG= Unilocular radiolucency containing flecks of indistinct radiopacities + Associated with unerupted tooth (mostly canine)</pre>
	MS= outer layer of palisaded cells & an inner layer of stellate reticulum Enlarged eosinophilic keratinized , epithelial cells without visible nuclei "ghost cells" within the stellate reticulum + multiple calcifications Tx: enucleation for cystic lesion More aggressive tx for solid lesion
SQUAMOUS ODONTOGENIC TUMOR	anterior to the molars of either jaw Triangular Unilocular radiolucency - close to the roots of erupted teeth. TX: curettage and extraction of involved tooth Local curettage & exo of involved tooth. usually misdiagnosed as ameloblastoma, resulting in unnecessary radical surgery.
AMELOBLASTIC FIBROMA	TRUE BIPHASIC TUMOR May represent the early developing stage of Ameloblastoma Unilocular or multilocular radiolucency with impacted tooth in mandibular or maxillary molar area
	MS= odontogenic epithelia resembelling dental lamina & the Cap & Bell stages of early odontogenesis. Zones of hyalinization are often surrounding the epithelial component of the lesion." Juxtaepithelial"
	TX: enucleation [lesion is well-encapsulated & easily separated from the surrounding]         Recurrence is high due to inadequate initial removal of what are frequently multilocular lesions





	reversa during Central TX ; enu	I lines which is indicate extensive remodeling growth of the lesion. region has MNGC	
ODONTOMA	Most co Hamart pulp & (comple	ommon odontogenic tumor comatous lesion found over unerupted teeth, c cementum in either recognizable teeth shape ( ex).	containing enamel, dentin, (Compond) or as a solid mass
	А.	Compound odontoma: <u>anterior maxilla</u> - over of unerupted teeth or between the roots of ere Contains radio opaque structures that resemb teeth	the crowns upted ones. ole miniature
	В.	<b>Complex odontoma:</b> <u>posterior mandible</u> – ove impacted teeth – appears as solid radio opaque mass	r
	TX: enu	cleation	



## LYMPHOID LESIONS

CONDITION	CHARACTERISTIC FEATURES
LYMPHOID HYPERPLASIA	starry sky appearance enlarged germinal center with mitosis & macrophages
Angiolymphoid hyperplasia with eosinophilia (ALHE)	Subcutaneous nodules - Rare inside the oral cavity ,         mostly in the head & neck region         Aggregates of lymphocytes and eosinophils <u>+ Blood eosinophilia</u> DD:         • Minor Salivary gland lesions         • Lipoma or Schwannoma         • Eosinophilic Granuloma         TX: excision or intralesional steroid injections
HODGKIN'S LYMPHOMA	<ul> <li>Affect bone or soft tissue [ rarely affects oral cavity]</li> <li><u>Painless enlargement of lymph nodes</u></li> <li>Within oral cavity causes Unilateral tonsillar</li> <li>enlargement</li> <li>Ann Arbor Staging System: <ul> <li>Stage I : single lymph node region or single extranodal site.</li> <li>Stage II : 2 or more lymph nodes region on the same side of diaphragm or localized involvement of an extra nodal site and one or more lymph node.</li> <li>Stage III : lymph node regions of both sides of the diaphragm, + localized involvement of an extra nodal organ or site IIIE or spleen IIIS or both IIISE</li> <li>Stage IV : Diffuse or disseminated involvement of one or more distant extra nodal organ</li> </ul> </li> <li>Sub classification: <ul> <li>A: Without Symptoms</li> <li>B: With systemic symptoms</li> </ul> </li> </ul>



	MS = Reed Sternberg cells [ large cells & bilobed nucleus] Classic HL comprises 4 entities: (Likes - Bulter Histo. Classification) • Lymphocytic -rich = most favorable diagnosis. • Nodular Sclerosis = most common form • Mixed cellularity • Lymphocytic depletion =least favorable prognosis. TX: • External Radiotherapy • Chemotherapy
NON HODGKINS LYMPHOMA	<ul> <li>Histiocytic type (least favorable prognosis)</li> <li>Most common extra nodal site is GIT</li> <li>Head &amp; neck is the second most common site (Waldeyer's Ring)</li> <li>Histological Types involving head &amp; neck region: <ul> <li>A. Large B cell Lymphoma (Burkitt's</li> <li>Lymphoma) → mostly involves BONE</li> <li>MS= <u>Starry Sky appearance to the tumor</u></li> <li>children and adolescents</li> </ul> </li> </ul>
	<ul> <li>B. T-cell &amp; NK Lymphoma (Midline Lethal Granuloma)</li> <li>C. Extra nodal Marginal Zone Lymphoma (Unique; arise in LN present in salivary glands + other mucosal sites in the body) <ul> <li>Predisposing factors:</li> <li>Hashimato's thyroiditis</li> <li>Sjogren Syndrome</li> <li>H. P gastritis</li> <li>MS = Centrocyte-like cells</li> </ul> </li> <li>TX: chemo and radiotherapy</li> </ul>



#### GRANULOCYTIC SARCOMA( EXTRA MEDULLARY MYELOID TUMOR)

Auer rods: crystalline, rod-like , intracytoplasmic acidophilic bodies



#### Burkitt's lymphma has three forms:

- A. Endemic : In Africa related to malaria and EBV
- B. Sporadic: In north America & Europe
- **C.** Associated with immuno deficiency
- ✓ Q: Reed Stenberg cells are seen in → Hodgkin's lymphoma
- ✓ Q: unilateral tonsillar enlargement is seen in → hodgkin's lymphoma
- ✓ **Q:** Auer bodies are seen in  $\rightarrow$  Granulocytic sarcoma
- ✓ Q: Burkitt's lymphoma mostly affects → bone [remember B, B]
- ✓ Q: starry sky appearance is seen in → Burkitt's lymphoma
- ✓ Q: centrocyte like cells are seen in → extra nodal marginal zone lymphoma



## VERRUCA PAPILLARY LESIONS



CONDITION	CHARACTERISTIC FEATURES
Squamous	Most common of all oral lesions
papilloma	Associated with HPV ( non- oncogenic types 2,6,11 &37)
	On lip or hard palate
	cauliflower- like surface alteration
	MS=
	Koilocytic cells = virally altered cells [pyknotic nuclei,
	surrounded by an edematous or clear zone ]
	Differential Diagnosis:
	✓ Verruciform xanthoma
	✓ Condylomata accuminatum
	Tx: Surgical excision or Laser ablation
Papillary	Exclusive in hard palate , always associated with
hyperplasia	removable prosthesis.
/nalatal	Caused by low grade chronic trauma with fungal
nanillomatosis	infection under ill fitting dentures + poor OH
papillolliatosis	Clinically = cobble-stone appearance [ papillary
	projections]
	MS= Pseudoepitheliomatous hyperplasia-( mimic
	SCC) ,but with no evidence of dysplasia.
	Differential Diagnosis:
	✓ Nicotina stomatitis
	✓ Darrier's disease **
	✓ Squamous papilloma
	Tx:
	Surgical excision or cryosurgery, microablation or laser ablation
Condylomata latum	expression of secondary syphilis Exophytic, friable, papillary lesions within oral cavity Potentially infectious ( abundant T pallidum)

Condylomata accuminatum	Caused by HPV 6 & 11 MS= Koilocytes [ virally altered cells- pyknotic nuclei, surrounded by an edematous or clear zone ] TX: Surgical excision or laser ablation	o 2007 Logical Images, inc.
Focal epithelial hyperplasia ( HECK'S DISEASE)	Caused by low-grade irritation, vitamin deficiency with HPV 13 & 32 infection. Nodular soft tissue masses all over mucosal surfaces [same color as adjacent mucosa] MS = Prominent fusion of epithelial ridges. TX: No particular tx - Spontaneous regression may be seen	
Pyostomatitis vegetans	<ul> <li>Pustular form of mucocutaneous disease, mostly in association with inflammatory bowel diseases.</li> <li>Numerous tiny yellow pustules all over the mouth + small vegetating papillary projections</li> <li>MS= Pseudoepitheliomatous hyperplasia</li> <li>PMNS &amp; eosinophils infiltrate ( consistent findings)</li> <li>TX: Control of inflammatory Bowel diseases</li> <li>Topical steroids, antibiotics, multivitamins &amp; nutritional supplements.</li> </ul>	
Verruciform xanthoma	Appears on skin and genitalia as well MS= Invigilated crypts alternate with papillary extension + Numerous foa xanthoma cells TX: conservative excision	m or
		7



Verrucous carcinoma	Associated with the use of tobacco Buccal mucosa (most common) then gingiva ( especially mandibular) Early lesion: As verrucous hyperplasia (white, indurated with irregular borders). Late lesions: Exophytic with white-gray shaggy surface. MS= Papillary frond covered with highly keratinized epithelium + bulbous epithelium mass extend into the submucosa with blunt, pushing margins TX: Surgical excision + radiotherapy [excellent prognosis because it is highly differentiated + no metastesis]	
Keratoacanthoma	<ul> <li>Squamo-proliferative lesion in sun-exposed skin &amp; sometimes at mucocutaneous junction <ul> <li>On skin: It originates within pilosebacious apparatus</li> <li>Fully developed lesion: contain a core of keratin surrounded by a concentric collar of raised skin or mucosa.</li> <li>If lesions not removed, spontaneous regression occur and the central keratin mass is exfoliated</li> <li>MS= Central keratin plug with overhanging lip of epith.</li> <li>Pseudoepitheliomatous hyperplasia - Mimic SCC</li> <li>Differential Diagnosis : <ul> <li>Molluscum contagiosum</li> <li>Solar keratosis</li> <li>Verruca vulgaris</li> </ul> </li> </ul></li></ul>	rognosis)
Basal cell carcinoma	" pearly nodule" Ms= Nests of hyperchromatic but uniform basaloid cells + cleft-like retraction spaces	
Malignant melanoma	Usually appear as <b>black or brown patches</b> Mucosal melanoma are common in <b>India, Japan &amp;</b> Africa. Amelanotic melanoma appear red Palate & upper alveolar ridge. It grows in predictable manner: ✓ Radial or horizontal growth phase (pre invasiv	re or in situ stage)

Dentiscope 2020 Page 49 of 74



$\checkmark$	Vertical growth phase (Invasive stage)	
Types:		
1-	Superficial spreading melanoma ( R gro cutaneous melanoma.	wth phase), most common
2-	Nodular melanoma ( V growth phase), 1	/3 develop in head & neck region
3-	Lentigo maligna melanoma ( R growth p freckles.	phase), develop from Hutchinson's
4-	Acral Lentiginous melanoma ( R	
	growth phase), most common <b>inblacks</b> + most common form of <u>oral</u>	Melanoma
ABCDE > Asy grov > Bor > Colo > Dian > Evo	<b>system :</b> mmetry ( because of uncontrolled wth pattern) der irregularity ( often with notching). or variation meter ( greater than 6 mm) lving ( lesion changes overtime)	Invasion into Lymphocytes (
MS=		States and a set
<ul> <li>Nec clea</li> <li>Nec spin inte</li> <li>Tx: Lesion s probabl</li> </ul>	oplastic melanocytes surrounded by ar halos oplastic melanocytes = round to ndle-shaped that are speckled or ensely pigmented with melanin. should be excised , but median survival ly not longer than 2 years.	Ervasive turnor celis Reference

- Q: HPV is associated with ? squamous papilloma , condylomata accuminatum , focal epithelial hyperplasia [ heck's disease]
- > Q:Associated with inflammatory bowel disease? Pyostomatitis vegetans
- > Q: associated with polio sebaceous apparatus? Keratoacanthoma



## REACTIVE AND NEOPLASTIC SOFT TISSUE LESIONS

REAC	TIVE HYPERPLASTIC FIBROUS TISSUE LESIONS
CONDITION	Characteristic features
Fibroma	<ul> <li>Also called (Irritation fibroma, Traumatic fibroma, Fibrous Hyperplasia)</li> <li>hyperplasia of fibrous CT in response to local irritation</li> <li>mostly on the buccal mucosa along the bite line or on sites that get frequently traumatized</li> <li>smooth surface pink nodule [ same color as surrounding tissues]</li> <li>MS= <ul> <li>atrophy of the rete ridges</li> <li>Collagen bundles arranged in radiating, circular or haphazard fashion</li> </ul> </li> <li>Tx: surgical excision</li> </ul>
Giant cell fibroma	Mostly in the mandibular gingiva MS = - large, stellate fibroblasts [may contain several nuclei] - rete ridges that appear narrow & elongated Tx: surgical excision
Epulis fissuratum	Also called Denture Epulis, Inflammatory Fibrous hyperplasia Caused by irritation from the flange of an ill- fitting denture [mostly facial aspect in anterior region] folds of hyperplastic tissue in the alveolar vestibule similar lesion = Fibro-epithelial polyp or leaf-like denture fibroma on the hard palate beneath a maxillary denture -The edge of the lesion is often serrated & resembles a leaf. MS = Pseudo-epitheliomatous hyperplasia hyperplasia of the rete ridges. Osteoid or chondroid tissues is observed (osseous & chondromatous metaplasia). Tx: Surgical removal with relining or remodeling the ill-fitted dentures

Crash Course in Oral	Pathology
Pyogenic granuloma	<ul> <li>Tissue response to local irritation or trauma</li> <li>Smooth or lobulated mass that is usually pedunculated + ulcerated surface</li> <li>More common in maxillary gingiva -Anterior areas - Facial aspects</li> <li>painless, although it bleed easily because of extreme vascularity.</li> <li>MS=         <ul> <li>Highly vascular proliferation</li> <li>Numerous endothelium-lined channels that are engorged with r.b.c.</li> <li>Sometimes these vessels are organized in lobular aggregates (lobular capillary hemangioma)</li> <li>PMNs are more prevalent near the ulcerated surfaces while plasma cells &amp; lymphocytes are more in deeper areas</li> </ul> </li> </ul>
	<ul> <li>Tx: surgical excision</li> <li>Pyogenic granulomas of the gingiva frequently develop in pregnant women [<i>Pregnancy tumor or granuloma gravidarum</i>] - during the first trimester &amp; their incidence increases up through the 7th. Month of pregnancy</li> <li>➢ Related to the increasing levels of <u>estrogen &amp; progesterone</u> as the pregnancy progresses – mostly resolves after delivery , if it doesn't → surgical excision [ lesion is removed during pregnancy in case it interferes</li> </ul>
Epulis granulomatosa	Hyperplastic growths of granulation tissue that sometimes arise in healing sockets in response to bony sequestra
Peripheral giant cell granuloma (GIANT CELL EPULIS)	<ul> <li>Does not represent a true neoplasm but a reactive lesion caused by local irritation or trauma.</li> <li>soft tissue counterpart of Central giant cell granuloma.</li> <li>Resemble pyogenic granuloma but it is more bluish-purple compared with bright red of typical pyogenic granuloma</li> <li>Cupping resorption of the underlying alveolar bone</li> <li>MS=</li> <li>Multinucleated giant cells + Abundant hemorrhage + hemosiderin deposits</li> </ul>

TX: excision to the underlying bone



Peripheral ossifying fibroma (OSSIFYING FIBROID EPULIS)	<ul> <li>does not represent a soft tissue counterpart of the central ossifying fibroma.</li> <li>incisor-cuspid region</li> <li>Exclusive on gingiva</li> <li>MS = Fibrous proliferation + mineralized products [may consist of bone, cementum-like material, or dystrophic calcification ]</li> </ul>	
	or dystrophic calcification ]	63 80

TX: excision to the underlying bone

	NEOPLASTIC FIBROUS TISSUE LESIO	NS
CONDITION	Characteristic features	
FIBROUS HISTIOCYTOMA	MS = Tumor cells are arranged in <i>storiform</i> pattern histiocyte-like cells xanthoma cells Tx: surgical excision	
FIBROMATOSIS	MS= spindle-shaped cells arranged in <b>streaming</b> <b>fascicles</b> <b>Tx:</b> wide local excision – has high recurrence rate	
LIPOMA	Some buccal cases are herniation of the buccal fat pad, which occur after surgical removal of third molar MS = Well circumscribed - Lobular arrangement of <b>mature fat cells</b> that differ from the surrounding normal fat Types : fibro lipoma – angiolipoma – spindle cell lipoma pleomorphic lipoma – intra muscular (infiltrating lipom Tx : conservative local excision	a – myxoid lipoma – na)



NEURAL TISSUE LESIONS				
CONDITION TRAUMATIC NEUROMA ( AMPUTATION NEUROMA)	<ul> <li>Characteristic features</li> <li>proliferation of neural tissue after transaction or other damage to a nerve bundle</li> <li>Smooth-surfaced, non ulcerated nodules mostly in the mental foramen region</li> <li>MS = Haphazard proliferation of mature, myelinated nerve bundles</li> <li>Tx: surgical excision with a small portion of the associated nerve</li> </ul>			
NEUROLEMOMA (SCHWANNOMA)	<ul> <li>2 microscopic patterns:</li> <li>1. Antoni A: streaming facicles of spindle – shaped Schwann cells around central acellular areas known as Verocay bodies. [Chinese lantern apperance]</li> <li>2. Antoni B: random arrangement of spindle cells</li> <li>TX: surgical excision</li> </ul>			
NEUROFIBROMA	MS = spindle-shaped cells with wavy nuclei + mast cells TX: excision			
Multiple endocrine neoplasia type 2B (MEN SYNDROMES)	<ul> <li>[Bilateral neuromas of the commissural mucosa]</li> <li>characteristic facial appearance: narrow face, thick lip with averted upper eyelid.</li> <li>Those pts at risk to develop Pheochromocytoma &amp; medullary carcinoma of the thyroid gland</li> <li>MS = hyperplasia of nerve bundles</li> <li>TX: Prophylactic removal of thyroid gland.</li> </ul>			

Dentiscope 2020 Page 54 of 74



Melanotic neuroectodermal tumor of infancy	AAAA	develops during the first year of life & it is of neural crest origin. Maxillary anterior region rapidly expanding mass that is frequently blue or black <i>"sun ray " radiographic appearance that may be mistaken for osteosarcoma</i> .	

MS = bi	ohasic po	pulation of o	cells ( er	ithelial 8	& neurob	lastic cells	) + melanin
	phasic po	paration of t		nunchar e	x neuros		, incluinin

	MUSCULAR LESIONS	
CONDITION GRANULAR CELL TUMOR	Characteristic features   > Not derived from muscles [derived from Schwann cells or neuroendocrinne cells]   > Mostly on the dorsum of the tongue     MS =   Large , polygonal cells with pale eosinophilic cytoplasm   Pseudoepitheliomatous hyperplasia   TX= conservative local excision	
CONGENITAL EPULIS (CONGENITAL GRANULAR CELL LESION)	<ul> <li>Mostly on the maxilla - lateral to the midline in the area of developing lateral incisor &amp; canine</li> <li>MS = Large , rounded cells with pale eosinophilic cytoplasm</li> <li>Pseudoepitheliomatous hyperplasia</li> </ul>	
LEIOMYOMA	<ul> <li>Benign tumor of smooth muscle cells</li> <li>Oral leiomyomas are either solid or vascular</li> <li>MS = interlacing bundle of smooth muscle cells</li> </ul>	



	VASCULAR LESIONS
CONDITION	Characteristic features
HEMANGIOMA	<ul> <li>Present at birth as pale macule with threadlike telangeictasias</li> <li>half of all hemangiomas will show complete resolution by 5 years of age</li> <li>MS = plump endothelial cells</li> <li>TX: systemic corticosteroids or Interferon-α</li> </ul>
VASCULAR MALFORMATION	<ul> <li>Portwine stains are relatively common capillary malformations on face along the distribution of trigeminal nerve. [Tx =Flashlamp-pulsed dye laser]</li> <li>grow with the patient</li> <li>MS = do not show endothelial cell proliferation &amp; the channels resemble the vessels of origin</li> </ul>
STURGE WEBER ANGIOMATOSIS	<ul> <li>Portwine stains or Nevus Flammeus</li> <li>Epilepsy</li> <li>TX: neurosurgery + laser therapy</li> </ul>
LYMPHANGIOMA	<ul> <li>Cavernous lymphangioma are more <i>frequent in anterior 2/3 of the tongue</i></li> <li>"frog eggs appearance"</li> <li>Tx: surgical excision</li> </ul>
ANGIOSARCOMAS	<ul> <li>Resemble a simple bruise which may lead to delay in diagnosis</li> <li>Mostly on scalp and forehead</li> </ul>
KAPOSI'S SARCOMA	<ul> <li>Caused by HIV</li> <li>Types:         <ol> <li>Classic – affects Slavic, Jewish &amp; Italian men, multiple bluish-purple macules on skin of lower extremities.</li> <li>Endemic (African)</li> <li>latrogenic immunosuppressant-associated – organ transplant pts</li> <li>AIDS-related.</li> </ol> </li> <li>Kaposi's sarcoma evolves through 3 stages:         <ol> <li>Patch (macular)</li> <li>Plaque.</li> <li>Nodular.</li> </ol> </li> </ul>



- ✓ Q: xanthoma cells arranged in storiform pattern are seen in? fibrous histocytoma
- ✓ Q: Antoni A & Antoni B are variants of ? neurolemmoma [ shcwanoma ]
- ✓ Q: Pheochromocytoma & medullary carcinoma of the thyroid gland are seen in? multiple endocrine neoplasia type 2B
- ✓ Q: frog egg appearance is seen in ? cavernous lymphangioma
- ✓ Q: Pseudoepithelamatous hyperplasia is seen in?
  - 1. papillary hyperplasia [ palatal papillomatosis ]
  - 2. pyostomatitis vegetans
  - 3. keratoacanthoma
  - 4. necrotizing sialometaplasia
  - 5. epulis fissuratum
  - 6. Granular cell tumor
  - 7. Congentinal epulis



## ORAL SQUAMOUS CELL CARCINOMA

#### The most common feature of SCC is ulceration then swelling and pain

## Causes of SCC

- 1- Smoking [Main carcinogens in tobacco are N-nitrosamines Pipe and cigar smoking have been linked with carcinoma of the lip , reverse smoking is associated particularly with cancer of the palate]
- 2- Alcohol [ usually people smoke and drink at the same time ethanol can act as a solvent helping harmful chemicals in tobacco to get inside the cells ]
  - **\*\*** smoking and alcohol act synergistically to increase the relative risk to 15 times
- 3- Sunlight [ causes SCC of the lower lip mostly seen in outdoor workers ]
- 4- **Diet and nutrition** [Deficiencies of **iron** and of the antioxidant vitamins A, C, and E increase the risk for oral cancer + anemia like in **Plummer-Vinson or Patterson-Kelly syndrome**]
- 5- Dental factors [ ill fitting dentures, poor oral hygiene ]
- 6- Oncogenic viruses [ HSV, HPV 16 & 18, EBV]
- 7- Immunosuppression like HIV
- 8- Chronic candida infections [ chronic hyperplastic candidiasis]

## Clinical presentation of SCC

Clinical features raise suspicion of malignancy:

- 1- persistent ulceration
- 2- Induration
- 3- fixation to underlying tissue + bone destruction

### Metastasis of SCC

- ✓ Carcinoma of lower lip → via superior jugular vein & digastric nodes.
- ✓ Oropharyngeal carcinoma → via jugulo-digastric or retropharyngeal nodes.

HPV oncogene E6  $\rightarrow$  inactivates tumor suppressor gene [P53]

HPV oncogene E7 → inactivate retinoblastoma Gene [ Rb]

enlarged regional nodes do not necessarily indicate metastatic spread they might be only nonspecific changes of **reactive hyperplasia** 

Oral SCC might start as asymptomatic erythroplakia, ulceration, white patches  $\rightarrow$  in later stages might become painful causing bone destruction, loosening, displacement of teeth and altered nerve sensations



✓ **Distant metastasis** to the Lungs & liver.

## Grading of tumors

Well-differentiated tumors " low grade or grade 1":

- Squamous Epithelium + masses of prickle cells surrounded by basal cells
- Intercellular bridges + Keratin pearls

Nuclear and cellular pleomorphism is not prominent and there are relatively few mitotic figures.



#### Moderately differentiated tumors" Grade 2 or moderate grade":

less keratinization and more nuclear and cellular pleomorphism and mitotic activity, but are still readily identified as squamous in type

#### Poorly differentiated tumors " high grade or grade 3"

Keratinization is absent and the cells show **prominent nuclear** and **cellular pleomorphism** and **abundant**, often bizarre, mitoses.

- Most oral squamous cell carcinomas are extremely locally destructive.
- SCC has Broad front of invasion (better prognosis), in others separate islands of carcinoma or even individual malignant cells may be seen well in advance of the main growth.
- Bone invasion occurs as a result of local spread:
  - A. Edentulous pt  $\rightarrow$  route of entry through crest of the ridge
  - B. Dentate pt  $\rightarrow$  route of entry through PDL

#### Signs of dysplasia :

- 1- Loss of polarity of basal cells
- 2- Drop shaped rete ridges
- 3- Keratin pearls / dyskeratosis
- 4- Cellular and nuclear polymorphism
- 5- Atypical mitotic figures

Mild dysplasia = only the lower 1/3 of epithelium

Moderate = middle 3<sup>rd</sup> of epithelium

Severe = 2/3 of the epithelium

Carcinoma in situ = full thickness of epithelium shows cellular changes but BM is still intact







**Q:** The further back in the mouth the tumor, then the worse the prognosis, why? Because they tend not to be diagnosed at an early stage + the rich lymphatic drainage around the base of the tongue may will aid in metastatic spread.

**Q: Carcinomas in females have a better prognosis than carcinomas in males, why?** Because they tend to be diagnosed and treated at an earlier stage.

Q: **Age affects prognosis, partly, why?** With increasing age the patient becomes less well able to withstand extensive surgery or radiotherapy + there will be reduction in cell mediated immune response

## Staging of tumors [TNM system]

T [ size of the pr	imary lesion ]		
Т0	No evidence of primary site		
Tis	Carcinoma in situ		
T1	2cm or less in greatest diameter		
T2	More than 2cm but less than 4 cm		
Т3	More than 4 cm		
T4 a	Tumor invade cortical bone or extrinsic tongue muscles, or maxillary sinus or skin of face.		
	(Resect able)		
T4b	Tumor involve masticator space, pterygoid plate, or skull base and/or encases internal		
	carotid artery (unresectable)		
N [ extent of me	etastasis in the regional lymph nodes ]		
Nx	Nodes could not be or were not assessed		
NO	No regional LN metastasis		
N1	Metastasis in single ipsilateral node 3 cm or less in greatest dimension		
N2A	Metastasis in single ipsilateral node 3 cm ,but not greater than 6 cm in greatest diameter		
N2B	Metastasis in multiple ipsilateral node, none more than 6 cm in greatest diameter		
N2C	Metastasis in bilateral or contralateral nodes, none more than 6 cm in greatest diameter		
N3	Metastasis in a node more than 6 cm in greatest diameter		
M [ presence or	M [ presence or absence of distant metastasis]		
Mx	Distant metastasis was not assessed		
M0	No evidence of distant metastasis		
M1	Distant metastasis is present		

Stage		TNM classification	5 year prognosis
1	<b>T1</b> N0M0		72 %
Ш	<b>T2</b> N0M0		58%
Ш	<b>T3</b> N0M0, or T1, T2, or T3, <b>N1</b> M0		45%
IV A	<b>T4a</b> N0 or N1M0, or T1,T2,T3 or T4 <b>N2</b> M0		22%
IV B	Any T <b>N3</b> M0 or <b>T4b</b> any NM0		
IV C	Any T Any N & <b>M1</b> lesion		



## Treatment of SCC

- Lip Carcinoma: Wedge resection
- Intraoral Carcinoma: Early stage: Surgery &/or Radiotherapy
- Cervical lymph nodes involvement: Radical Neck dissection or selective neck dissection.
- > Chemotherapeutic agents: Platinum containing compounds " cisplatin", 5 FU.
- > Neoadjuvent chemotherapy could be used initially to shrink the tumor prior to additional therapy.
- > Targeted therapy: Cetuximab & Panitumomab (monoclonal Abs)



- > Mucocele: Can be mucus extravasation phenomenon or mucus retention cyst
- Ranula : mucus extravasation phenomenon and mucus retention cyst that occurs specifically in the floor of the mouth

**MUCOUS EXTRAVASATION PHENOMENONA:** Trauma will cause severance of the salivary duct but the acinar cells will continue to secrete saliva into the severed duct → mucous pools into the CT forming a mucocele

- Most common site for mucous extravasation cyst = the lower lip
- Initially the mucoceles are well circumscribed but with repeated trauma they become nodular and firm

#### MS =

- > Free mucin in connective tissue with no epithelial lining
- > The base of the mucocele will reveal feeder duct.

Salivary gland changes (in long standing case) shows:

- A. Ductal dilation
- B. Chronic inflammation
- C. Acinar degeneration
- D. Increased fibrosis

#### DD of mucous retention cyst:

- A. Mucoepidermoid carcinoma
- B. Cavernous hemangeoma
- C. Blisters seen in some **bullous** and desqumative disease.
- D. Soft tissue neoplasms (neurofibroma & lipoma)

#### Treatment:

Surgical excision with **the feeder gland**.





> Post surgical parasthesia might occur

#### Mucous retention cyst [ obstructive sialadenitis]

obstruction of a salivary duct resulting in an **epithelial lining cavity containing mucus** 

- The mucus retention cyst could occur in the major salivary gland, when they do occur they are multiple [ poly cystic disease of the parotid gland]
- > Most common site for mucous retention cyst  $\rightarrow$  floor of the mouth

#### MS =

- > The cyst has compressed ductal epithelial linning
- > Cyst lumen contains mucin or occasionally a sialolith

#### DD of mucous retention cyst:

- A. Salivary gland neoplasm
- B. Mucus extravasation phenomenon
- C. Benign CT neoplasm
- D. Dermoid cyst

**Treatment :** excision with caution of rupturing the cystic sac [ damage to the adjacent gland may result in a mucocele formation]

**MAXILLARY RETENTION CYST** : Due to blockage of antral sero-mucus gland  $\rightarrow$  ductal epithelium lined cystic structure filled with mucin.

- Asymptomatic, appear as hemispheric, homogenous well- defined radiopacity
- > No treatment just observation

#### SALIVARY GLAND OBSTRUCTION – SIALOLITH:

- > Usually associated with the submandibular gland Wharton's duct
- causes intermittent swelling and pain often at meal times [ when there is increased demand for saliva]

Tx: surgical removal of the stone with or without the gland

## Gout is the only systemic disease known to cause salivary calculi and these are composed of <u>uric acid</u>

#### Predisposing factors for sialolith formation:

- increase in water hardness
- smoking
- xerostomia

How a sialolith forms: mucin proteins and epithelial cells will form a nidus for the calcium salts to precipitate over  $\rightarrow$ calcifications will continue and form concentric layers as the sialolith increases in size

Dentiscope 2020 Page 63 of 74









Q: why is it more common to get sialoliths in the sub mandibular gland?

- 1. Saliva more alkaline
- 2. Higher concentration of calcium and phosphate in the saliva
- 3. Higher mucus content
- 4. Longer duct
- 5. Anti-gravity flow

Q: how can calculi/ sialolith be detected?

- 1- Plain occlusal films [ for intra ductal stones]
- 2- Ct scan
- 3- Ultrasound
- 4- Sialography [descriptions]:
  - A. Tree in winter  $\rightarrow$  normal parotid gland
  - B. Bush in winter  $\rightarrow$  normal sub mandibular gland
  - C. Snowstorm / cherry blossom  $\rightarrow$  sjogren's syndrome
  - D. Sausage link appearance [ dots , blobs]  $\rightarrow$  sialodochitis
- 5- Radio isotope imaging
- 6- MRI [gold standard]
- 7- **Diagnostic Sialendoscopy:** Allows complete exploration of the ductal system, direct visualization of duct pathology- but has risk of perforating the duct and can lead to duct stenosis

#### Q: how can sialolith be treated ?

- A. No treatment  $\rightarrow$  just give ABX and anti inflammatories and hope for the stone to pass
- B. Stone excision → [lithotripsy, interventional sialondoscopy, simple excision]
- C. Gland excision  $\rightarrow$

**Q: when can you remove a sialolith transorally?** If you can palpate it through the mouth, if you can visualize it on an occlusal radiograph , if it is no more than 2 cm from the punctum

**Q: when do you need to remove the gland?** If transoral approach fails, intraglandular stones , very posterior stones

**Q: how does the epithelium in the gland react to the sialolith?** The epithelium shows **squamous and mucus cell metaplasia +** changes to stratified squamous epithelium with goblet cells

Most of SMG calculi are radioopaque

Most of parotid calculi are radiolucent and multiple





#### **NECROTIZING SIALOMETAPLASIA:**

- > Caused by ischemia due to LA vasoconstriction mostly in the palate
- Iarge area of epithelium + underlying connective tissue and minor salivary glands become necrotic while the ducts undergo squamous metaplasia [ within the ulcer you'll see gray granular lobules which represents the necrotic minor salivary glands]
- MS = pseudoepitheliamatous hyperplasia
- self limiting [ no tx needed ] heals in 6-8 weeks





## SALIVARY GLAND NEOPLASTIC DISEASES

Tumor	Origin
Pleomorphic adenomas	intercalated duct cells and myoepithelial cells
Oncocytic tumors	striated duct cells
Acinous cell tumors	acinar cells
Mucoepidermoid tumors and squamous cell	excretory duct cells
carcinomas	

	BENIGN SALIVARY GALND TUMORS
CONDITION	Characteristic features
PLEOMORPHIC	Most common salivary gland neoplasm
ADENOMA	Mostly in the parotid gland [superficial lobe, most in tail of gland ] followed by
	minor salivary glands in the palate
	MS =
	Epithelial (E) and stromal (S) components.
	Epithelial Components:
	Tubular and cord-like or solid sheet arrangements
	Stromal [ mesenchymal ] components:
	Origin: myoepithelial cells
	Loose chondromyxoid stroma + cartilage and Osseous
	metaplasia
	pseudo-encapsulated [tumor islands may be found
	within the fibrous capsule which is <b>continuous</b> with
	main tumor mass & likely to contribute to recurrence
	(tumor pseudopodes) ]
	transformation
	A Careful excision [narotidectomy with VII]
	nreservation]
	B. Submandibular gland excision
	C. Wide local excision of minor SG
	CAUTION: With each recurrence there is an increased possibility of malignant
	transformation.
	Radioresistant, radiotherapy is contraindicated **
WARTHIN'S	Also called Adenolymphoma; benign papillary cystadenoma lymphomatosum
TUMOR	Second most common benign tumor of the parotid gland [Exclusive in parotid gland
	mainly the tail]
	Bilateral in 10% of the cases
	May contain mucoid brown fluid in FNA
	Positive correlation with cigarette smoking & EBV.



	<ul> <li>Dough to cystic mass in the inferior pole of parotid gland adjacent to posterior angle of mandible.</li> <li>MS =         <ul> <li>A. Cystic spaces</li> <li>B. papillary fronds which demonstrate 2 layers of oncocytic epithelial cells.(luminal &amp; basal cells)</li> <li>C. lymphoid tissue with germinal center</li> </ul> </li> <li>Doccasionally undergoes squamous metaplasia (may mistakenly diagnose SCC on FNA)</li> <li>Both lymphoid and oncocytic epithelial elements must be present to diagnose Warthin's</li> <li>Doccytes selectively incorporate technetium Tc 99m and appear as hot spots on a radionucleotide scan.</li> <li>Tx: surgical excision</li> </ul>
MONOMORPHIC ADENOMA	Similar to Pleomorphic Adenoma except <b>no mesenchymal</b> stromal component [ epithelial component] <ul> <li>Occurs in minor salivary glands (upper lip)</li> <li>12% bilateral</li> <li>Types:</li> </ul>
	A. Basal Cell Adenoma : mostly in the parotid , but upper lip is the most common intraoral site uniform basaloid epithelial cells with a monomorphous pattern



	<ul> <li>B. Canicular Adenoma : exclusive in the upper lip - Bilayer s branch &amp; anastamose</li> <li>A. Myoepithelioma Adenoma : just myoepithelial cells Most of them arise within MSG, parotid gland, SMG plasmacytoid or spindle cells</li> </ul>	trands of basaloid cells that
ONCOCYTIC TUMORS	<ul> <li>Include Oncocytoma &amp; oxyphilic adenoma</li> <li>Mainly parotid gland</li> <li>MS=</li> <li>Polyhydral cells with granular eosinophilic cytoplasm &amp; centrally placed vesicular nucleous</li> <li>Tx = SF parotidectomy</li> </ul>	



	MALIGNANT SALIVARY GALND TUMORS
CONDITION	Characteristic features
MUCOEPIDERMOID CARCINOMA	<ul> <li>Most common salivary <u>malignancy</u></li> <li>Mostly parotid followed by submandibular &amp; minor glands</li> <li>MS =         <ul> <li>Epidermoid epithelial cells</li> <li>Intermediate epithelial cells</li> <li>Mucous secreting epithelial cells</li> </ul> </li> </ul>
	LOW GRADE: numerous mucus secreting cells + intermediate cells and <u>few epidermoid cells with minimal cellular</u> <u>atypia.</u>
	INTERMEDIATE GRADE: mucus cells but not as numerous as in low grade
	HIGH GRADE: Clusters of proliferating <u>epidermoid cells</u> + few mucous cells. Mistaken for SCC Q: how can you differnetuate b/w mucoepideromoid carcinoma and SCC? By mucin staining
	<b>TX:</b> Stage I & II $\rightarrow$ Wide local excision Stage III & IV $\rightarrow$ Radical excision +/- neck dissection +/- postoperative radiation



ADENOID CYSTIC CARCINOMA	<ul> <li>&gt; second most common malignancy overall and the first most malignancy of the submandibular gland</li> <li>&gt; characterized by neurotropism [ perineural invasion] → leading to recurrences at the skull base after surgical and radiation treatment</li> </ul>
	<ul> <li>Threehistologic patterns: [patterns may coexist in the same tumor]</li> <li>The cribiform pattern [swiss cheese pattern] - best prognosis.</li> </ul>
	<ul> <li>➤ Tubular pattern - intermediate prognosis</li> <li>➤ The solid pattern - poorer prognosis.</li> <li>Tx: complete local excision [Tendency for perineural invasion → facial nerve has to be sacrificed] + post op radio therapy</li> </ul>
ACINIC CELL CARCINOMA	<ul> <li>low-grade behavior and has the best survival rate of any salivary malignancy</li> <li>Parotid gland</li> <li>Origin: Intercalated duct &amp; reserve cells</li> <li>Second most common parotid and pediatric malignancy</li> <li>MS= Stained by PAS.</li> <li>Cells heavily stained [ called blue dot tumor]</li> <li>Bilateral in 3% of patients, making acinic cell carcinoma the second-most common neoplasm, after Warthin's tumor, to exhibit bilateral presentation.</li> </ul>
	Tx: local excision +/- post op radio therapy



M	ALIGNANT MIXED SALIVARY GALND TUMORS
CONDITION CARCINOMA EX- PLEOMORPHIC ADENOMA	<ul> <li>Characteristic features</li> <li>Carcinoma developing in the epithelial component of preexisting pleomorphic adenoma</li> <li>A typical clinical history includes a longstanding salivary mass that begins to rapidly enlarge</li> <li>TX : radical excision + neck dissection + post op radio therapy</li> <li>Includes Clear cell carcinoma &amp; Epimyoepithelial carcinoma</li> <li>Mosthy in minor salivary glands</li> </ul>
SQUAMOUS CELL CARCINOMA	<ul> <li>Clear cells</li> <li>Clear cells</li> <li>TX: local excision</li> <li>major salivary glands [ submandibular glnads]</li> <li>Obstructive sialadenitis is predisposing factor.</li> <li>Well to moderately well-differentiated with no evidence of mucin production.</li> </ul>
	<ul> <li>You must RULE OUT:</li> <li>High-grade mucoepidermoid carcinoma</li> <li>Metastatic SCCA to intraglandular nodes</li> <li>Direct extension of SCCA</li> </ul>
POLYMORPHOUS LOW GRADE ADENOCARCINOMA	<ul> <li>Exclusive in minor salivary glands - second most common malignancy of Minor salivary glands</li> <li>low grade maliganany with low rate of reccurance</li> <li>Origin: Reserve cells in most proximal portion of salivary duct.</li> <li>MS = Myoepithelial differentiated cells</li> <li>Tx: excision</li> </ul>

- > Carcino-sarcoma : True malignant mixed tumor [carcinomatous and sarcomatous components ]
- > Metastatic mixed tumor : Metastatic deposits of otherwise typical pleomorphic adenoma
### **TNM OF SALIVARY GLAND TUMORS**

T [ size of the primary lesion ]		
ТО	No evidence of primary tumor	
T1	Tumor 2 cm or less in greatest dimension without extra-parenchymal extension	
T2	Tumor > 2 cm but not > 4 cm in greatest dimension without extra-parenchymal extension	
Т3	Tumor > 4 cm and/or tumor having extra-parenchymal extension	
T4 a	Tumor invades skin, mandible, ear canal and/ or facial nerve	
T4b	Tumor invades skull base and/or pterygoid plates and /or encase carotid artery	
N [ extent of metastasis in the regional lymph nodes ]		
Nx	Nodes could not be or were not assessed	
NO	No regional LN metastasis	
N1	Metastasis in single ipsilateral node 3 cm or less in greatest dimension	
N2A	Metastasis in single ipsilateral node 3 cm ,but not greater than 6 cm in greatest diameter	
N2B	Metastasis in multiple ipsilateral node, none more than 6 cm in greatest diameter	
N2C	Metastasis in bilateral or contralateral nodes, none more than 6 cm in greatest diameter	
N3	Metastasis in a node more than 6 cm in greatest diameter	
M [ presence or absence of distant metastasis]		
Mx	Distant metastasis was not or could not be assessed	
M0	No evidence of distant metastasis	
M1	Distant metastasis is present	

Stage		TNM classification
1	T1N0M0	
Ш	<b>T2</b> N0M0	
Ш	<b>T3</b> N0M0, or T1, T2, or T3, <b>N1</b> M0	
IV A	T4aN0 or N1M0, or T1,T2,T3 or T4 N2M0	
IV B	Any T <b>N3</b> M0 or <b>T4b</b> any NM0	
IV C	Any T Any N & <b>M1</b> lesion	

Salivary Gland Neoplasia Another classification [ ABCDs]:

- > Architecture
- Biphasic
- > Cytology
- > Differential



## References

- Brad W. Neville, Douglas D. Damm, Carl M. Allen and Angela C. Chi, . Oral and Maxillofacial Pathology, 4th ed.
- Regezi, J. A., Sciubba, J. J., & Jordan, R. C. K. (2003). Oral pathology: Clinical pathologic correlations. St. Louis, Mo: Saunders.



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