Chapter 5

Major and Minor Salivary Glands

S. Di Palma · R.H.W. Simpson · A. Skalova · I. Leivo

Contents

5.1 Introduction ........................................ 132
5.1.1 Normal Salivary Glands ......................... 132
5.1.2 Developmental Disorders ....................... 132
5.2 Obstructive Disorders ............................... 132
5.2.1 Mucus Escape Reaction ......................... 132
5.2.2 Chronic Sclerosing Sialadenitis of the Submandibular Gland (Küttner Tumour) ............ 133
5.3 Infections ............................................ 133
5.3.1 Bacteria, Fungi .................................. 133
5.3.2 Viruses ............................................. 133
5.4 Miscellaneous Inflammatory Disorders .......... 133
5.5 Miscellaneous Non-Inflammatory Disorders ...... 133
5.5.1 Necrotising Sialometaplasia (Salivary Gland Infarction) ..................................... 133
5.5.2 Sialadenosis ........................................ 133
5.5.3 Adenomatoid Hyperplasia of Mucoepidermoid Carcinoma .................................... 134
5.5.4 Irradiation Changes ............................... 134
5.5.5 Tissue Changes Following Fine Needle Aspiration ............................................. 134
5.6 Oncocytic Lesions ...................................... 134
5.6.1 Focal and Diffuse Oncocytosis .................. 134
5.6.2 Ductal Oncocytosis ................................ 134
5.6.3 Multifocal Nodular Oncocytic Hyperplasia ......................................................... 135
5.7 Cysts ................................................... 135
5.7.1 Salivary Polycystic Dysgenetic Disease ......... 135
5.7.2 Mucoceles .......................................... 135
5.7.3 Simple Salivary Duct Cysts ..................... 135
5.7.4 Lymphoepithelial Cyst ............................ 135
5.7.5 Cystic Lymphoid Hyperplasia of AIDS ........ 136
5.7.6 Other Cysts ........................................ 137
5.8 Benign Tumours ........................................ 137
5.8.1 Pleomorphic Adenoma ......................... 137
5.8.1.1 Salivary Gland Anlage Tumour
(“Congenital Pleomorphic Adenoma”) ............ 140
5.8.2 Benign Myoepithelioma .......................... 140
5.8.3 Basal Cell Adenoma ............................. 141
5.8.4 Warthin’s Tumour ................................ 142
5.8.5 Oncocytoma ........................................ 143
5.8.6 Canaliculal Adenoma ............................ 143
5.8.7 Sebaceous Adenoma ............................... 144
5.8.8 Sebaceous Lymphadenoma ....................... 144
5.8.9 Ductal Papilloma .................................. 144
5.8.10 Cystadenoma ...................................... 144
5.9 Malignant Epithelial Tumours ...................... 144
5.9.1 Acinic Cell Carcinoma ......................... 144
5.9.2 Mucoepidermoid Carcinoma .................... 146
5.9.3 Adenoid Cystic Carcinoma ....................... 147
5.9.4 Polymorphous Low-Grade Adenocarcinoma ................................. 148
5.9.4.1 Cribriform Adenocarcinoma of the Tongue ............................................. 149
5.9.5 Epithelial-Myoepithelial Carcinoma ............. 150
5.9.6 Hyalinising Clear Cell Carcinoma ................ 151
5.9.7 Basal Cell Adenocarcinoma ...................... 151
5.9.8 Myoepithelial Carcinoma
(Malignant Myoepithelioma) ............................. 152
5.9.9 Salivary Duct Carcinoma ......................... 154
5.9.10 Oncocytic Carcinoma ............................. 155
5.9.11 Malignancy in Pleomorphic Adenoma
Malignant Mixed Tumour ................................ 156
5.9.11.1 Carcinoma (True Malignant Mixed Tumour)
Ex Pleomorphic Adenoma ................................ 156
5.9.11.2 Carcinosarcoma Ex Pleomorphic Adenoma .............................................. 157
5.9.11.3 Metastasising Pleomorphic Adenoma ......................................................... 157
5.9.12 Sebaceous Carcinoma ............................. 158
5.9.13 Lymphoepithelial Carcinoma ..................... 158
5.9.14 Small Cell Carcinoma .............................. 158
5.9.15 Higher Grade Change in Carcinomas ........... 159
5.9.16 Metastatic Malignancies ......................... 159
5.10 Hybrid Carcinoma ..................................... 160
5.11 Endodermal Sinus Tumour .......................... 160
5.12 Sialoblastoma ......................................... 160
5.13 Alterations in Gene Expression
and Molecular Derangements
in Salivary Gland Carcinoma .......................... 160
5.13.1 Predominantly Myoepithelial Malignancies ................................................. 161
5.13.2 Predominantly Epithelial Malignancies ......................................................... 161
5.14 Benign and Malignant Lymphoid Infiltrates ........ 162
5.14.1 Non-Autoimmune Lymphoid Infiltrates ........ 162
5.14.2 Benign Autoimmune Lymphoid Infiltrates ......................................................... 162
5.14.3 Malignant Lymphoma ............................. 163
5.15 Other Tumours ....................................... 163
5.16 Unclassified Tumours ................................ 163
References ................................................... 164
5.1 Introduction

5.1.1 Normal Salivary Glands

The salivary glands include paired major glands (parotid, submandibular and sublingual) and minor glands throughout the upper aerodigestive tract.

The cellular component comprises serous and mucous acinar and ductal epithelial cells, myoepithelial cells and connective tissue components (e.g. fat, fibrous tissue, nerves and blood vessels). The parotid glands consist of predominantly serous acini, the submandibular glands of mixed, serous and mucous acini, while the sublingual glands contain mainly mucous acini. Minor salivary glands also have mixed serous and mucous acini in varying proportions.

Of particular interest are the myoepithelial cells. They are a normal constituent of the major and minor salivary glands, and are believed to have contractile properties that assist in the secretion of saliva. Similar cells are also found in the breast, tracheo-bronchial and sweat glands. They are plentiful in the salivary acini and intercalated ducts, but much less so in the larger excretory ducts of the major glands. Microscopic examination shows that myoepithelial cells are thin and spindle-shaped and situated between the basement membrane and epithelial cells, and ultrastructurally they are seen to possess a number of cytoplasmic processes that extend between and over the acinar and ductal lining cells. They display features of both smooth muscle and epithelium, such as numerous microfilaments with focal densities in the cytoplasmic processes, and desmosomes that attach the myoepithelial to the epithelial cells [62]. Similarly, immunohistochemistry shows that myoepithelial cells stain strongly with alpha smooth muscle actin (αSMA), calponin, smooth muscle myosin heavy chain (SMMHC) [164], h-caldesmon [74], S-100 protein [114] as well as with some cytokeratins (e.g. subtype 14). Maspin, p63 [8, 166] and CD 10 [143, 183] have recently been described as markers of breast myoepithelial cells, and may have a role in identifying their salivary equivalents [62]. Similarly, immunohistochemistry shows that myoepithelial cells stain strongly with alpha smooth muscle actin (αSMA), calponin, smooth muscle myosin heavy chain (SMMHC) [164], h-caldesmon [74], S-100 protein [114] as well as with some cytokeratins (e.g. subtype 14). Maspin, p63 [8, 166] and CD 10 [143, 183] have recently been described as markers of breast myoepithelial cells, and may have a role in identifying their salivary equivalents [62]. Similarly, immunohistochemistry shows that myoepithelial cells stain strongly with alpha smooth muscle actin (αSMA), calponin, smooth muscle myosin heavy chain (SMMHC) [164], h-caldesmon [74], S-100 protein [114] as well as with some cytokeratins (e.g. subtype 14). Maspin, p63 [8, 166] and CD 10 [143, 183] have recently been described as markers of breast myoepithelial cells, and may have a role in identifying their salivary equivalents [62].

Serial sectioning has shown an average of 20 lymph nodes within each parotid [67], and they may be affected by inflammatory processes and neoplasms, both primary and metastatic. Their presence may hamper histologic evaluation of parotid gland lesions [6].

5.1.2 Developmental Disorders

Agenesis, aplasia, hypoplasia and atresia of the main ducts are all extremely rare. In contrast, intra-parotid nodal heterotopias are very common [129], and epithelial tumours may arise from them [175]. Extranodal heterotopia is rare, and can be subdivided into high (involvement of the ear, pituitary, mandible, etc.) or low forms (lower neck, thyroid).

Accessory parotid glands comprising salivary tissue separate from the main gland, adjacent to Stenson’s duct, are found in 20% of people.

5.2 Obstructive Disorders

5.2.1 Mucus Escape Reaction

This forms an extravasation mucocele, which is defined as the pooling of mucus in the connective tissue in a cavity not lined with epithelium. Most patients are under 30 years of age, and the minor glands are most often affected. The incidence by site is lower lip 65%, palate 4%, buccal mucosa 10%, and (in the major glands) parotid 0.6%, submandibular 1.2% and sublingual 1.1%. The pathogenesis is traumatic severance of a duct, leading to mucus pooling. It presents in the lip as a raised, often blue, dome shaped swelling of the mucosa, usually 2–10 mm in diameter, but it is generally larger in the sublingual gland in the floor of the mouth where it is known as a ranula. Microscopy shows a well-defined mucin-filled cavity lacking an epithelial lining, but lined with granulation tissue and macrophages (Fig. 5.1).