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The Wiry Wen-Sclerosing Polycystic Adenoma

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Sclerosing polycystic adenoma manifests as an exceptionally encountered, benign neoplasm emerging within salivary glands. Initially scripted in 1996, tumefaction appears reminiscent of fibrocystic change or sclerosing adenosis of breast. Although attributes are obscure, the condition is currently contemplated to be neoplastic in nature.

Additionally designated as sclerosing polycystic adenosis, sclerosing adenosis, polycystic adenosis or sclerosing polycystic sialadenopathy, sclerosing polycystic adenoma simulates fibrocystic change and sclerosing adenosis of mammary tissue. Tumefaction is constituted of acini and ductal articulations coated with dual epithelial layer irregularly disseminated within a dense, sclerotic stroma.

Sclerosing polycystic adenoma may incriminate individuals between 9 years to 84 years with mean age of disease occurrence at 40 years. A mild female predominance is encountered with female to male proportion of 3:2.

Sclerosing polycystic adenoma predominantly appears within parotid gland (~70%). Occasionally, submandibular gland or minor salivary glands disseminated within oral cavity may be incriminated [1,2].

Of obscure aetiology, sclerosing polycystic adenoma was initially contemplated to be a reactive, non-neoplastic lesion, reminiscent of fibrocystic change of breast. However, contemporary evidence and classification indicates representation of a possible neoplastic process. Tumour reoccurrence occurs in ~30% neoplasms.

Neoplasm may delineate clonal countenance, as discerned with human androgen receptor assay (HUMARA). Targeted next generation sequencing (NGS) demonstrates genomic mutations confined to genes which represent as drivers within diverse human neoplasms [1,2].

Sclerosing polycystic adenoma manifests with genetic alterations within PI3K pathway. Additionally, genetic mutations within PTEN and PIK3CA along with alterations within PIK3R1 may be encountered.

Sclerosing polycystic adenoma manifests as a gradually progressive tumefaction. Neoplasm may be accompanied by pain or altered sensation [1,2].

Upon cytological examination, neoplasm is configured of cohesive sheets and cellular aggregates. Tumour cells appear permeated with moderate to abundant, finely granular, eosinophilic cytoplasm, spherical to elliptical nuclei, uniform nuclear chromatin and indistinct nucleoli. Appropriate tumour diagnosis upon cytological assessment can be challenging and misrepresentation is commonly encountered [2,3].

Grossly, a well demarcated, pale, firm, rubbery nodule appears embedded within normal salivary gland tissue. Besides, tumefaction may be multinodular.

Upon frozen section, a well circumscribed proliferation composed of distended, disseminated ducts of variable magnitude is observed. Neoplastic ducts are layered by hyperplastic epithelium configuring an indistinct, nodular pattern. Foci of apocrine metamorphosis may ensue. Circumscribing stroma is dense [2,3].

Frozen section may designate an interpretation of 'sclerotic fibrous material with benign appearing glandular elements', as appropriate tumour categorization may be challenging [2,3].

Upon microscopy, a well circumscribed neoplasm demonstrating preserved lobular architecture is delineated. Tumefaction is composed of bi-layered ducts and acini irregularly disseminated within a dense, sclerotic stroma.

Neoplastic ducts configure miniature tubules or ectatic cellular spaces permeated with secretory substance intermingled with foamy macrophages. Ductal cells may exemplify a vacuolated, foamy, apocrine or mucous cellular countenance [2,3].

Hyperplastic ductal epithelium enunciates solid, microcystic or cribriform articulations. Miniature eosinophilic globules constituted of basement membrane-like material may impact focal cribriform configurations, indicative of foci of collagenous spherulosis [2,3].

Neoplastic acini are permeated with numerous, coarse, eosinophilic, intracytoplasmic granules which may be highlighted by Periodic acid Schiff's stain [2,3].

The well circumscribed, sharply demarcated lesion appears to be segregated from adjacent or circumscribing, uninvolved glandular parenchyma. An admixture of cystic articulations, miniature ducts and acinar component appears enmeshed within a fibrous and sclerotic stroma. Epithelium layering ducts and cystic structures exhibits variable morphology composed of granular, foamy or apocrine cellular constituents [3,4].

Apocrine cells are imbued with abundant, eosinophilic cytoplasm and display prominent apical snouts. Acinar structures are characteristically permeated with bright, eosinophilic granules [3,4].

Intra-ductal apocrine proliferation demonstrates cyto-architectural features simulating ductal carcinoma *in situ* (DCIS) of breast. Cogent architectural features are comprised of rigid bridges, micro-papillary projections and solid or cribriform tumour pattern.

Cytological manifestations appear as neoplastic cells depicting low to intermediate nuclear grade. Intra-ductal cellular proliferation may be associated with an obvious, infiltrative component of malignant tumour cells [3,4].

Invasive apocrine adenocarcinoma of salivary gland is comprised of irregular glands and cords of neoplastic cells enmeshed within a desmoplastic stroma. Neoplastic component appears to abut normal salivary gland architecture [3,4].

Tumour cells are pervaded with abundant, granular, eosinophilic cytoplasm, enlarged pleomorphic nuclei and coarse nuclear chromatin with prominent nucleoli. Proliferating, intra-ductal apocrine cells along with or devoid of cellular or nuclear atypia and neoplastic cells constituting invasive adenocarcinoma of salivary duct origin may be diffusely immune reactive to nuclear androgen receptor [3,4].

Generally, perineural or lymphovascular tumour infiltration is absent. Occasionally, disseminated foci of mature adipose tissue or extensive stromal lipomatous may be encountered. Besides, tumour associated lymphoid proliferation (TALP) can be exemplified [3,4].

Upon ultrastructural examination, neoplastic cells demonstrate abundant cytoplasm incorporated with electron dense granules of variable magnitude, consistent with zymogen granules [3,4].

Neoplastic epithelial cells appear immune reactive to cytokeratin as AE1/AE3 or CAM 5.2. Around 80% cells appear immune reactive to progesterone receptors (PR). Myoepithelial cells appear immune reactive to smooth muscle actin (SMA), p63 and calponin. Cellular component of intra-ductal lesions delineating rigid bars and cribriform architecture appears immune reactive to myoepithelial markers [4,5].

Acinar cells are pervaded with coarse eosinophilic cytoplasm and appear immune reactive to gross cystic disease fluid protein-15 (GCDFP-15). Acinar cells can be highlighted with Periodic acid Schiff's (PAS) stain with diastase resistance.

Neoplastic epithelial cells appear immune non reactive to carcinoembryonic antigen (CEA) and HER2/neu [4,5].

Sclerosing polycystic adenoma of salivary gland requires segregation from neoplasm such as chronic sclerosing sialadenitis, polycystic (dysgenetic) disease, pleomorphic adenoma, mucoepidermoid carcinoma, acinic cell carcinoma or intra-ductal carcinoma as low grade salivary duct carcinoma or low grade cribriform cystadenocarcinoma. Sclerosing polycystic adenoma can be appropriately categorized upon histological assessment of surgical tissue sampling or core tissue biopsy [4,5].

Ultrasonography depicts a well circumscribed, hypoechoic tumefaction comprised of micro-cysts.

T2 weighted magnetic resonance imaging (MRI) exhibits miniature, cystic areas demonstrating enhanced signal intensity [4,5].

Sclerosing polycystic adenoma can be appropriately managed with conservative therapy. Comprehensive or localized surgical eradication of the neoplasm appears optimal. Subsequently, extensive tumour monitoring is recommended [4,5].

Localized tumour reoccurrence emerges in up to 30% neoplasms. However, multiple tumour reoccurrences appearing within an extended duration may ensue within neoplasms delineating an invasive, carcinomatous component [4,5].

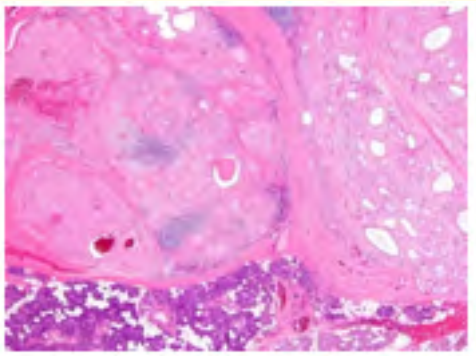

Figure 1: *Sclerosing polycystic adenoma demonstrating an admixture of ducts and cystic structures layered by foamy, granular and apocrine cells. Circumscribing stroma is abundant, fibrous and sclerotic [6].*

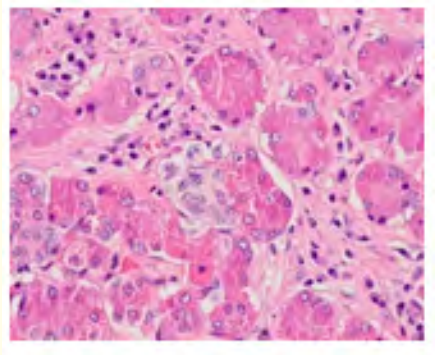

Figure 2: *Sclerosing polycystic adenoma delineating an intermingling of ducts and cystic articulations lined by granular, foamy and apocrine cells. Encompassing stroma is abundant, foamy and sclerotic [7].*

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6. Image 1 Courtesy: Webpathology.com.

7. Image 2 Courtesy: Pathology outlines.

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