The benign salivary corpuscular bassinet-Myoepithelioma

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ABSTRACT: Myoepithelioma of the salivary gland constitutes a 2.2% and 5.7% respectively of the benign major and minor salivary gland neoplasm. The tumours originate from the transformed myoepithelial or basket cells located amidst the basement membrane and the basal plasma membrane of the acinar cell. The classic variants of the tumour are Spindle cell (65%), Plasmacytoid or Hyaline (20%), Epitheloid and Clear cell. The salivary gland neoplasm may be immune reactive for CK7+/CK20-. Also, the transformed myoepithelial cells elucidate immune -reactive cyto- keratins, dual types of S-100 protein, p63, CD109 with a proportion of vimentin, actin and myosin. An estimated 5%-10% of the myopitheliomas constitute the presence of ducts with one duct per medium to high power field (220-400 X). One miniature cluster of ducts is diagnostic of a myo-epithelial lesion. Myoepithelioma necessitates a demarcation from Pleomorphic adenoma, Adenoid cystic carcinoma, Terminal duct carcinoma and Epi-myoepithelial carcinoma of the intercalated duct origin. Benign disorders such as an abscess, a mucocoele, schwannoma, neurofibroma, leiomyoma, benign fibrous histiocytoma or neoplasm such as extramedullary plasmacytoma, rhabdomyosarcoma, leiomyosarcoma, nodular fasciitis, synovial sarcoma, hamangiopericytoma, solitary fibrous tumour, paraganglioma, pleomorphic adenoma, mucoepidermoid carcinoma, adenocarcinoma, myoepithelial carcinoma may mandate a segregation. Malignant myoepitheliomas or myoepithelial carcinomas emerges de novo or as a consequence of malignant transformation of a benign mixed tumour or a basal cell adenoma. A p-53 mutation induces a malignant conversion indicated by immune reactivity for c- KIT.

Key Words: *Myoepithelloma; Benign salivary; Tumor*

A salivary gland neoplasm identical to a Myoepithelioma was initially scripted in 1943 (1). Subsequently, it was designated variously as the "parotid clear cell adenoma" probably of myoepithelial derivation or "adeno-myopeithelioma". Myoepithelioma of the salivary gland was formally categorized as a salivary neoplasm in 1991 (2,3). Tumors constituted predominantly of myoepithelial cells are designated as myoepitheliomas. Myoepithelioma or the Myoepithelial Adenoma is a benign myopeithelial neoplasm, composed of ectodermal contractile "myoepithelial" cells, which perform as smooth muscle cells (3). Myoepitheliomas constitute a 2.2% and 5.7% respectively of the benign major and minor salivary gland neoplasm, although the condition is allocated a 1% of the comprehensive salivary gland tumors. The parotid gland is frequently involved (40%) the sublingual gland (33%), the submandibular gland (13%) (3), besides the minor salivary glands (21%) (3,4). Despite the parotid gland being a frequent site, myoepithelioma remains an exceptional salivary gland neoplasm.

DISEASE CHARACTERISTICS

Myoepithelioma may emerge in young to middle-aged individuals with a sporadic pediatric occurrence. The development of a tumor occurs within a period of months or years (16). A tumor usually emerges betwixt 9-85 years of age with a median at 44 years or during the fourth decade of life (40-50 years) A gender predominance may be lacking (4-6). The de novo neoplasm may be locally aggressive. The neoplasm may emerge from a pleomorphic adenoma and gradually convert into a malignancy. A demarcation betwixt the two conditions may be necessitated as myoepithelioma may articulate as a mixed tumor and in-spite of a slow progression may turn malignant. A soft tissue myoepithelioma may be discerned as a benign neoplasm situated in the subcutaneous or sub-fascial tissue of the extremities (3,4). The lesion is usually asymptomatic and classically depicts a gradual, painless tumefaction. The tumor development is localized and it does not infiltrate the adjacent structures such as the facial nerve, in contrast to a benign parotid tumefaction (4). The radiologic images and tissue histology with immune histochemistry guide the appropriate diagnosis.

HISTOGENESIS

Myopitheliomas are preponderantly benign, though a malignant conversion is possible with tumor relapses and in the absence of treatment (4,6). As an infrequent, benign enlargement, it is surmised to originate from the transformed myoepithelial or basket cells located amidst the basement membrane and the basal plasma membrane of the acinar cell. Multitudinous cellular components such as smooth muscle actin, myosin, and intermediate filaments are implicated in the tumor ancestry. Myopithelial cells possibly contain contractile units which aid the discharge of glandular secretions (6,7). Transformed myoepithelial cells may exist globally in the exocrine glands of tissues such as skin, soft tissue, sweat glands, breast, and lacrimal glands. Bartholin's gland, nasal septum, nasopharynx, larynx, trachea, lung, esophagus, retroperitoneum, and prostate gland, though they are infrequent in the pancreas, soft tissue and the carpal tunnel (6,7).

NAKED EYE INSPECTION

Myoepithelioma may evolve as a component of adenoma polymorphum. the glandular capsule may confine a tumor (3). The tumors delineate a solid, tan or yellow-tan lustrous appearance. Preponderantly, the lesions elucidate a well outlined, encompassed, smooth or lobular, homogenous lump (5).

MICROSCOPY

A spindle cell articulation may be elucidated in an estimated 70% individuals and epitheloid or Plasmacytoid cell predominance in 20%. The neoplasm depicts a glandular, myxoid or reticular configuration with the cellular component configuring nests, cords, and sheets. A myxoid or chondro myxoid stroma with foci of hyalinization may be elucidated. Squamous metaplasia and calcification may be infrequent (4,5). Myoepithelioma cells display an eosinophilia or brightly stained cytoplasm. The eccentrically placed nucleus may be round to oval with well-dispersed chromatin (3). Variegated microscopy of a tumor may cause a dispute with the diagnosis. Frequently identified as an adenoma polymorphum, a myoepithelioma may be configured from proliferating myopeithelial cells with an aggressive development, in contrast to an adenoma polymorphum. The benign, parotid neoplasm may be enveloped with a dense, fibrous tissue capsule, while a tumor may be un-encapsulated when confined to the palate. The mitotic index may be crucial for assessing the prognosis (3). Myoepithelioma may be demarcated from adjunctive salivary gland tumors with the application of immune histochemistry.

VARIANTS OF MYOEPITHELIOMA

The cellular configuration may categorize the myoepitheliomas as (5):

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- **Spindle cell** (displaying a stroma like configuration, constituted of intertwined fascicles)
- **Plasmacytoid** Or **Hyaline** (polygonal cells with eccentric nuclei, dense, non-granular or hyaline, abundant, eosinophilia cytoplasm). The plasmacytoid type on electron microscopy displays copious, uniformly distributed microfilaments extending 50-100 A^0 in diameter. Pure hyaline myoepithelioma is depicted in the minor salivary glands, especially in the palate. Hyaline myoepithelioma may conduct as a benign tumor (5)
- **Epitheloid** (nests or cords of the round to polygonal cells with central nuclei and variable eosinophilia cytoplasm).
- **Clear cell** (polygonal cells with ample, optically clear cytoplasm filled with abundant glycogen with the absence of mucin and fat). Clear cell myoepithelioma elucidates thematic variations in the form of regressive (ancient) adaptations, schwannoma like zones, sebaceous glandular evolution, oncocytic modification and a double clear cellular manifestation (5) the spindle cell variant is the most frequent subtype (65%) followed by the plasmacytoid (20%) variant. Spindle cell and clear cell epithelioma usually arise in the parotid gland (6,7)
- The oncocytic epithelioma variant displays a cytoplasm which has a granular, oxyphilic quality, a minimal collagen stroma, and composition of micro-cysts (5). A variable, secondary myxoid change or lipomatous metaplasia of the stroma may emerge. Electron microscopy may demonstrate collagen crystals. Immune- reactivity to the pair of microfilaments actin and keratin can be visualized (5).

A malignant conversion may configure aspects such as an elevated mitotic rate, an infiltrating growth pattern, a classical cytology and zones of necrosis. Tumors persisting for an extended duration of time with lack of therapeutic intervention may depict malignant conversion (4)

• The malignant counterpart of myoepithelioma is the malignant myopeithelioma or Myoepithelial Carcinoma (MC) which demarcates from the benign analog by infiltrative, destructive progress. Myoepithelial carcinoma is an estimated 10% of the benign myoepitheliomas (6). The contemporary, malignant variant of Mucinous Malignant Carcinoma displays a histology that is composed predominantly of enormous mucin-containing cells organized in solid sheets, nests, cords, and trabaculae, enveloped in a mucoid stroma. The mucin-containing cells frequently depict a signet ring configuration with ample, clear or vacuolated cytoplasm with indented, compressed nuclei. The existence of intracellular mucin may be corroborated by mucicarmine, alcian blue, and diastase resistant periodic acid Schiff stain (6,7). Admixed and adjunctive cell types may be represented, comprising of epitheloid (polygonal cells with eosinophilia cytoplasm and ovoid nuclei) or plasmacytoid cells (abundant brightly eosinophilia cytoplasm and eccentric nuclei) (6,7). Minimal to moderate cytological atypia may be discerned. Since the histological criterion for distinguishing the benign and malignant mucinous myoepithelioma are not well delineated, it has been recommended that identical parameters may be employed to categorize the malignancy, as are applicable for the prototypical myoepithelioma. Signet ring cells in the salivary gland neoplasm mandate a demarcation from a metastatic adenocarcinoma. Immune staining with cyto-keratins CK 7 and CK 20 may be recommended for the distinction. The reaction of the salivary gland neoplasm is as CK7+/CK20- while the appearance of a CK7-/CK20+ clone is an evidence of the intestinal origin of a mucinous adenocarcinoma (3,4) Mucinous myoepithelial carcinoma requires demarcation from the various primary mucin-rich adenocarcinomas of salivary glands such as the mucinous adenocarcinoma, mucin-rich mucoepidermoid carcinoma, mucin-rich salivary duct carcinoma and mucin (colloid) adenocarcinoma

• Numerous architectural formulations may be delineated by the neoplasm such as the non–myxoid (solid), the myxoid (identical to a pleomorphic adenoma), the reticular (canalicular) and the MIXED (5,6). Myoepithelioma may resemble a pleomorphic adenoma on histology though the glandular- duct differentiation, the chondroid or chondromyxoid foci are practically absent and the myoepithelioma may appear identical to the non-luminal portion of the pleomorphic adenoma. Only 5%-10% of the myopitheliomas may be comprised of ducts with an estimated one duct per medium to high power field (220-400 X) and the appearance of one miniature cluster of ducts is diagnostic of a myoepithelial lesion (6,7). Besides, with cytogenetic similarities, the two conditions may be misinterpreted. Despite comparable outcomes, the demarcation of the Myoepithelioma from the Pleomorphic adenomas is a pre-requisite, in order to detect co-existent malignancies with identical histology. Adenoid cystic carcinoma, terminal duct carcinoma and epi-myoepithelial carcinoma of the intercalated duct origin may require a clarification (6,7). Myoepithelioma, the tumour deriving from the myoepithelial cells, would mandate a segregation from diseases such as an abscess, a mucocele, schwannoma , neurofibroma, leiomyoma, benign fibrous histiocytoma, extramedullary plasmacytoma, rhabdomyosarcoma, leiomyosarcoma, nodular fasciitis , synovial sarcoma, hamangiopericytoma , solitary fibrous tumour, paraganglioma, pleomorphic adenoma, mucoepidermoid carcinoma, adenocarcinoma , myoepithelial carcinoma and adjunctive benign and malignant salivary gland neoplasm (6,7). The cystic myoepithelial tumors may be erroneously interpreted as a parotid cyst. On account of the identical clinical countenance, demarcation of the afore-mentioned lesions is a pre-requisite, from the conventional myoepithelioma. The distinguishing histology is of infrequent mitosis, lack of nuclear and cellular pleomorphism and a non-infiltrative tumor configuration- characteristic of a benign tumor (7,8). Tumour morphology is a reliable indicator of distinction from analogous lesions. Myoepithelioma preponderantly displays the spindle cell, epithelioid cell, clear cell or plasmacytoid composition with concomitant and overlapping patterns existing in the same lesion. The clear cell variant is analogous with a clear cell adenocarcinoma and mucoepidermoid carcinoma and the spindle cell variant with peripheral nerve sheath tumors. The stipulated cytogenetic anomaly is identified at the chromosome 12q. Myoepithelioma reoccurs at an estimated 15%-18% and may reappear with the proportion of 1 in 16 tumours in the duration of 7 years. Malignant transformation may develop in archaic and relapsing lesions (6,7). Numerous instances of malignancy have been demonstrated in the spindle cells and clear cell subtypes, characterized by invasive properties and atypical cytological features. Malignant myoepitheliomas or myoepithelial carcinomas may emerge de novo or as a consequence of the malignant transformation of a benign mixed tumor or a basal cell adenoma. The clear cell variant may be considered as possibly malignant. Amplification of the c KIT receptors and a p-53 mutation may induce a malignant conversion. Carcinoma connotes a mere 10% of the Myoepithelioma (8). The carcinoma displays an aggressive pattern with infiltration, tumor evolution and tissue eradication. Enhanced mitosis, tissue necrosis, cellular pleomorphism, the absence of muscle markers and the presence of myofilaments delineate a monomorphic tumor configuration, in contrast to a benign myoepithelioma (6,7). Myoepitheliomas may elucidate a local reoccurrence (37%), lymph node metastasis (17%), distant metastasis (9%) and tumor related deaths (9%) (5). Dedifferentiated tumors as exemplified in the high-grade adenocarcinoma, undifferentiated carcinoma or the sarcomatoid carcinoma augment the tumor onslaught (7) (Figures 1-8).

IMMUNE HISTOCHEMISTRY

The transformed myoepithelial cells elucidate immune reactive cytokeratins, dual types of S-100 protein, p63, CD109 and a certain amount of vimentin, actin, and myosin. Numerous myoepithelial cells lack the reactivity to smooth muscle actin. Immunohistochemical examination usually assists in the determination of a tumor which expounds immune-reactivity to S-100, cytokeratins (chiefly 7 and 14), p63, Glial Fibrillary Acidic Protein (GFAP), Calponin and Muscle-specific molecules (actin, myosin) may be established (5). No reactivity is delineated with intracytoplasmic immunoglobulins, in contrast to a plasmacytoma. Nodular fasciitis, leiomyomas, and leiomyosarcomas are usually non-reactive to the cytokeratins and S-100 immune markers Specific muscle actin HHF35 and CD57 (Leu

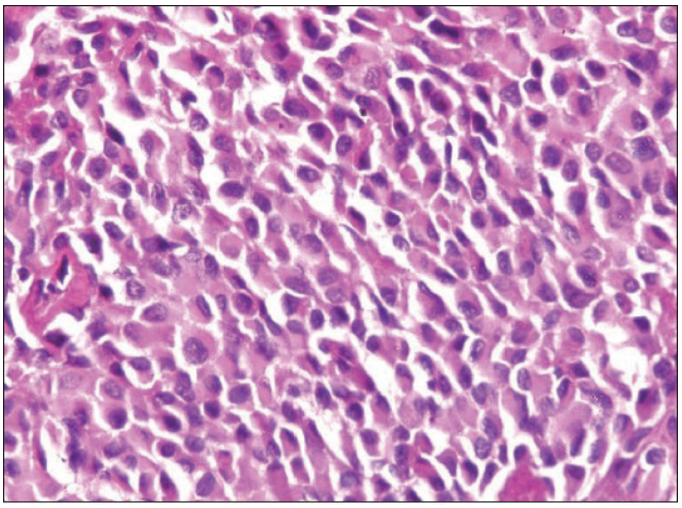

Figure 1) *Plasmacytoid myopeithelioma (19)*

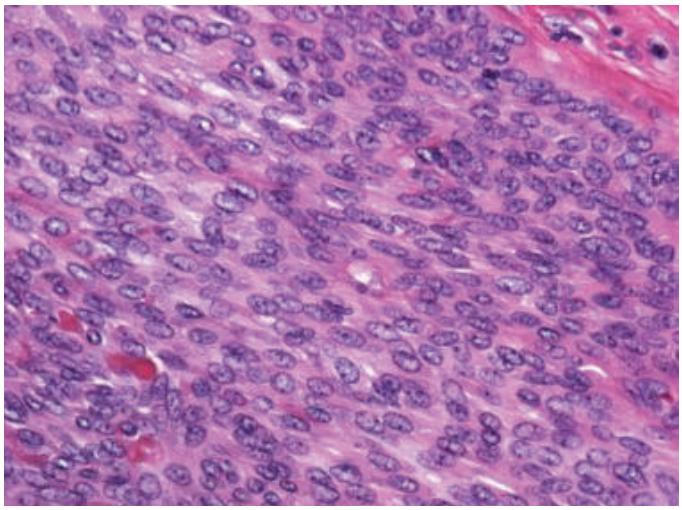

Figure 2) *Spindle cell myopeithelioma (20)*

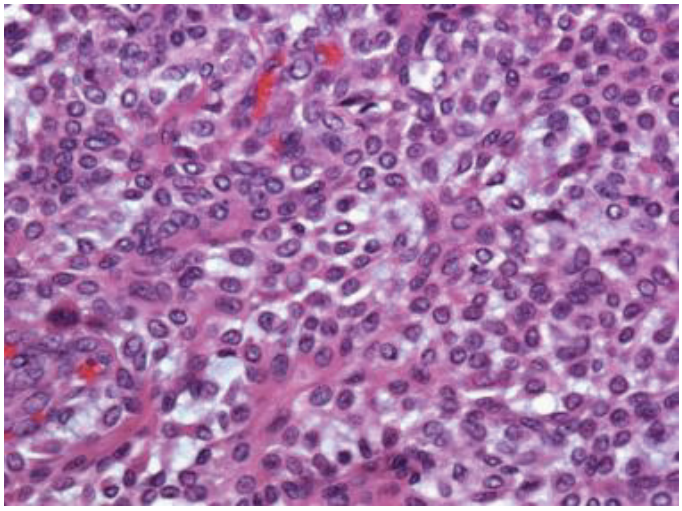

Figure 3)*Epitheloid myoepithelioma (20)*

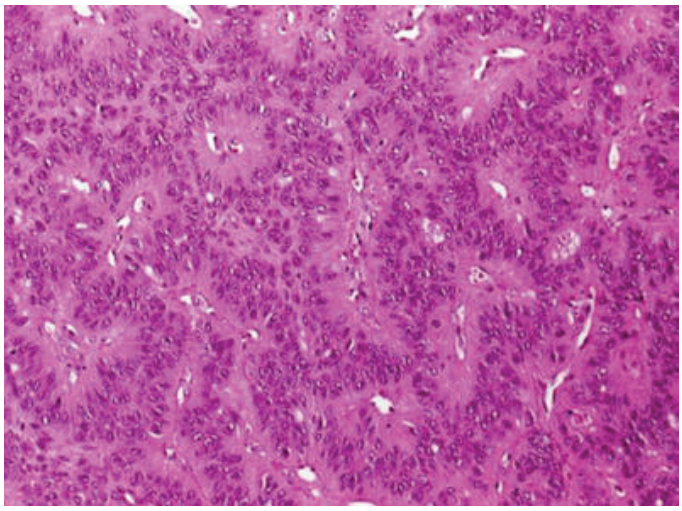

Figure 4) *Myoepithelioma-organoid pattern (21)*

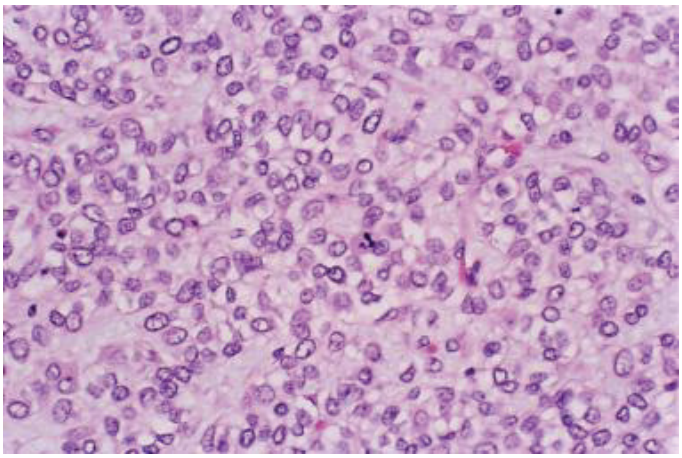

Figure 5) *Clear cell myopithelioma (20)*

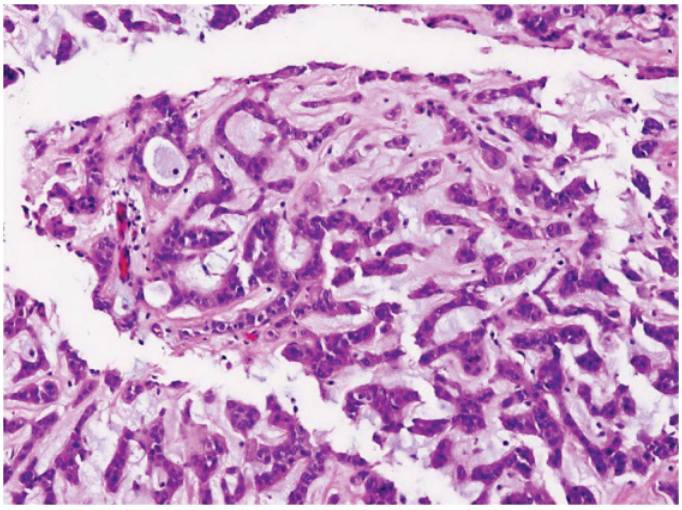

Figure 6) *Malignant myoepithelioma with cellular pleomorphism and invasion (22)*

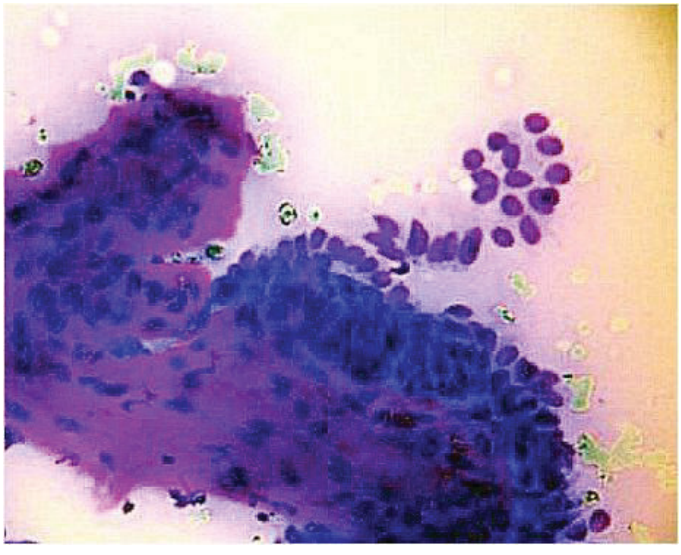

Figure 7) *Myoepithelioma with minimal anisonucleosis -aspiration cytology (23)*

7) may be discerned (8). An elucidation of S 100 protein may be moderate when confined to the cytoplasm and intense with the nuclear staining (4). A dubious lesion requires a suitable panel of immune-reactive antibodies to initiate appropriate therapy and follow up.

RADIOGRAPHIC ASSAY

A conventional diffusion and magnetic resonance imaging (MRI) may be inculcated within the contrast MRI of salivary glands. Advanced techniques of diffusion imaging of salivary glands commonly incorporate diffusion tensor imaging, diffusion kurtosis imaging and intra-voxel incoherent motion MRI. The classical and advanced diffusion imaging methodologies may competently discern the benign and malignant salivary gland tumors (9).

A Magnetic Resonance Imaging (MRI) may be a preferred technique to investigate the salivary gland tumors as a competent imaging may be crucial for an adequate pre-operative localization and classification of salivary gland tumors. A post-contrast MRI may be appropriate for assessing the specific location and extent of salivary gland tumors. A multi parametric assay of diffusion-weighted and dynamic contrast-enhanced MRI may be employed to

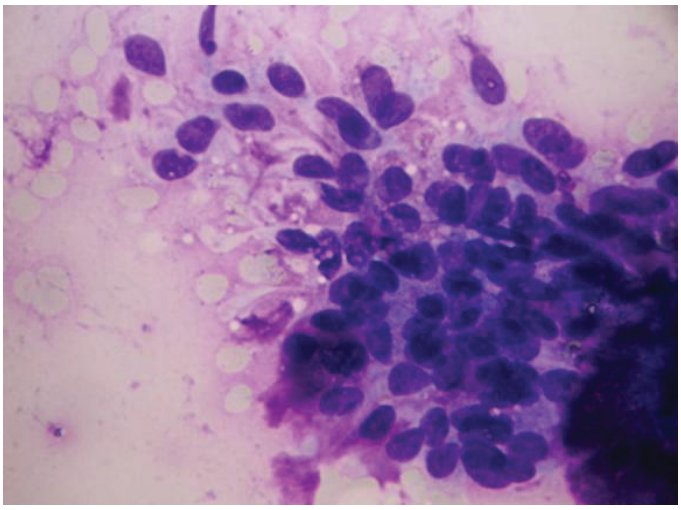

Figure 8) *Malignant myoepithelioma with loss of cohesion-aspiration cytology (23)*

categorize benign and malignant salivary glands tumors (10). Loco-regional and perineural extension, a lymph node and distant metastasis assessment, suitable for staging the malignant salivary gland may be achieved with the procedure. Imaging may be beneficial for discerning the tumor relapse, a competent follow up of individuals subsequent to therapy, anticipating the malignant transformation of benign tumors and efficiently diagnosing salivary gland tumors from identical inflammatory and autoimmune disorders (10).

Characteristically, the magnetic resonance imaging (MRI) analysis of a tumor delineates a circumscribed iso-intense or hyper-intense knot reciprocally on T-1 or T-2 weighted MRI (6). Malignant lesions appear within the minor salivary gland commonly in the oral cavity and may be frequent, in contrast to the benign tumors. A frequent malignant tumor may be the adenoid cystic carcinoma and mucoepidermoid carcinoma and pleomorphic adenoma may be a frequent benign tumor (11). Minor salivary glands may also be afflicted by inflammatory an immune-mediated disorders such as the Sjogren's syndrome, immunoglobulin G4 related disease, necrotizing sialometaplasia, and subacute necrotizing sialadenitis. A contrast MRI with Computerized Tomography (CT) may ably localize and assess the extent of the malignant, benign, inflammatory and immune-mediated disorders of the minor salivary glands (11). The mean Fractional Anisotropy (FA) and Mean Diffusivity (MD) of malignant salivary gland tumors may be significantly altered from the benign tumors. A combined value of FA and MD may be employed to distinguish malignant from benign tumors with an area under the curve (AUC) of 0.974 and an accuracy of 86% (12). Similarly, a significant divergence between the FA and MD of malignant tumors and pleomorphic adenoma may be discerned. A concurrent FA and MD for identifying a malignancy from a pleomorphic adenoma may depict an AUC of 0.993 and a precision of 93%. A diffusion-weighted imaging may be applied as a non-invasive methodology for categorizing and distinguishing benign from malignant salivary gland neoplasm (12). Also, the standardized prostate imaging, reporting, and data system (PI-RADS) may be considered as a dependable, non-invasive imaging procedure applicable for characterizing parotid lesions and predicting a malignant conversion (23). A proportionate precision of 92% and 90%, a sensitivity of 79% and 65% and a specificity of 94% and 96% may be achieved with inter-observers of PI-RADS (13). A conventional magnetic resonance imaging (MRI) may be mandated in order to assess the type of surgery (a parotid resection) with a concomitant reconstruction of salivary gland recommended for the tumors. Conventional and advanced magnetic imaging such as the diffusion and perfusion MRI may be opted for assaying the extent of salivary gland cancer and loco-regional reoccurrence (14). An 18 fluorodeoxyglucose positron emission computerized tomography (FDG PET CT) scan may discern the appearance of distant metastasis and a concurrent or alternate malignancy. An ultrasound with a conventional Computerized Tomography (CT) scan may be adequate for detecting complications secondary to surgical intervention of the salivary glands such as a sialocoele, fluid collection hematoma or a pseudo-aneurysm. A sequential, advanced MRI or diffusion imaging modalities may ascertain and quantify the radiation with coexistent radioactive iodine-induced sialadenitis (14).

PROGNOSTIC GUIDE

An extensive monitoring of the individual may be required in order to assess a probable tumor reoccurrence. The disease outcome may be dictated by the competent assessment of the disorder and depends upon the extent of malignancy in the lesion (15). The 5-year survival rate of a malignant myopeithelioma may vary from 25%-65% with a decline to 10%-35% at 15 years, irrespective of employed therapies (4).

THERAPEUTIC INTERVENTION

Surgical excision is the preferred therapy. Comprehensive surgical excision requires a mandatory follow up in order to assess the reoccurrence of a tumor (15). The specified neoplasm does not delineate multitudinous reoccurrences. Radiotherapy may be a pre-requisite where a surgical eradication is not feasible (4). As the neoplasm is relatively infrequent, the objective of chemo-radiation is inadequately defined (15,16). The therapeutic protocol varies according to the category of the neoplasm. Generally, adenoid cystic carcinoma, epi-myoepithelial carcinoma, and mucoepidermoid carcinoma necessitate a comprehensive surgical excision with a post-operative intensity modulation radiotherapy with or without chemotherapy (16,17). The standard treatment for mucoepidermoid carcinoma and adjunctive benign tumors is a definitive surgical excision with a margin of healthy, uninvolved tissue.

CONCLUSIONS

Myoepithelioma as an exceptional salivary gland neoplasm frequently implicates the parotid gland (40%) and the minor salivary glands (21%). The neoplasm depicts a glandular, myxoid, non myxoid, reticular or mixed configuration with cellular nests, cords, and sheets. Variants such as spindle cell, plasmacytoid or hyaline, epitheloid and clear cell categories are delineated (18). Immune-reactive cyto-keratins, dual types of S-100 protein, p63, CD109, vimentin, myosin, Glial Fibrillary Acidic Protein (GFAP), calponin specific muscle actin HHF35 and CD57 (Leu 7) may be enunciated. Conventional and advanced magnetic imaging such as the diffusion and perfusion MRI may be opted for assaying the extent of salivary gland carcinoma and loco-regional reoccurrence. Comprehensive surgical excision with a pre-requisite follow up in order to assess the tumor relapse may be the preferred mode of therapy.

REFERENCES

1. WH Sheldon. So called mixed tumours of the salivary gland. Arch Pathol Lab Med. 1943;35:1-20.
2. Mark Weitzel, Jason EC, Harvey Spector. Myoepithelioma of the parotid gland: a case report and review of literature and classic histopathology. Case Rep Otolaryngol. 2017;1:4.
3. Ludmila HK, Andrzej Wiktor, Lukasz Warmbier. Myoepithelioma of the parotid gland: Diagnosis and Surgical Treatment Difficulties: a case study. Dent Med Probl. 2016;53:282-90.
4. Estafania AB, Cristobal Sepulveda, Liberto Figueroa. Myoepithelioma of the tongue clinicopathological and IHC features. J Oral Maxillofac Surg 2018;1:26-29.
5. Rosai, Ackerman. Surgical pathology. Expert Consult 2011;830.
6. Shaodong Yang, Ming Zeng, Jinagli Zhang, et al. Mucinous myoepithelial carcinoma of the parotid gland: Report of a new histological variant. Int J Clin Exp Pathol. 2017;10:2224-30.
7. George Kyrgias, Maria, Aggeliki Kalogeridi, et al. Malignant myoepithelioma of the parotid gland: Literature review on the occasion of our report of a case. Int J Clin Med. 2012;3:49-54.
8. Zhu S, Schuerch C, Hunt J. Review and updates of immunohistochemistry in selected salivary gland and neck tumours. Arch Pathol Lab Med. 2015;139:55-56.
9. Abdel Razek AAK, Suresh Mukherji. Routine and advanced diffusion imaging modules of the salivary glands. Neuro Imag Clin N Am. 2018;28:245-54.
10. Abdel Razek AAK, Suresh Mukherji. State of the art imaging of salivary gland tumours. Neuro Imagining J Clin N Am. 2018;28:303-17.
11. Abdel Razek AAK, Suresh Mukherji. Imaging of minor salivary glands. Neuro Imaging Clin N Am. 2018;28:295-302.
12. Abdel Razek AAK. Characterization of salivary gland tumours with diffusion tensor imaging. Dentomaxillofac Radiol. 2018;47: 0343.
13. Abdel Razek AAK. Pilot study of ultrasound parotid imaging reporting data system (PIRADS): Inter-observer agreement. Eur J Radiol. 2015;84:2533-8.

14. Abdel Razek AAK , Suresh Mukherji. Imaging of post treatment salivary gland tumours. Neuro Imag Clin N Am. 2018;28:199-208.

15. Gnepp DR. Mucinous myopithelioma, a recently described new myoepithelioma variant. Head and Neck Pathol. 2013;7:S85-89.

16. GB da Cunha, TC Camurugy, TC Ribeiro, et al. Myoepithelioma of the nasal septum: a rare case of extra salivary involvement. Case Rep Otolaryngol. 2017:4.

17. HO Kamburoglu, AU Kayikcioglu, C Himmetoglu. A case of myopeithelioma mimicking a parotid cysts. Ear Nose And Throat J. 2015;94:37-39.

18. H Iguchi, K Yamada, H Yamane, et al. Epitheloid myoepithelioma of the accessory parotid gland: Pathological and magnetic resonance imaging findings. Case Rep Otolaryngol. 2014;7:310-15.

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20. Image 2, 3 and 5 Courtesy: eMedicine Medscape.

21. Image 4 Courtesy: Basic Medical Key.

22. Image 6 Courtesy: Sarcoma Images.

23. Image 7 and 8 Courtesy: Euro-cytology.