Invited Review

Preoperative cytopathological investigatory aids in the diagnosis of salivary gland lesions

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Abstract

Salivary gland lesions are a group of heterogeneous lesions inclusive of non-neoplastic and neoplastic lesions. History, clinical examination and preoperative investigations attempt to minimise the challenges faced in diagnosing these diverse lesions. Preoperative investigations include imaging and cytopathology. The advent of onsite evaluation methods to ensure sample adequacy and newer reporting systems that assign risk of malignancy has improved the sensitivity and specificity of cytopathology. The scope of this review is limited to the preoperative cytopathological investigations and the diagnostic challenges met in reporting salivary gland tumours.

Keywords: Challenges in FNAC, digital cytology, fine needle aspiration cytology, Milan's reporting system of cytopathology, salivary gland cytopathology

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Submitted: 19-May-2024, **Revised:** 25-May-2024, **Accepted:** 01-Jun-2024, **Published:** 11-Jul-2024

INTRODUCTION

Salivary gland pathology comprises of group of heterogenous lesions that encompasses non-neoplastic and neoplastic lesions. Salivary gland tumours (SGT) contribute to around 3%–6% of head and neck tumours worldwide.[1] There is a profound overlapping of clinical and cytological features of lesions arising from this region, thereby limiting the application of cytology in definitive diagnosis. The diversity in histopathology is owing to the arrangement of the cells and their extracellular material and the altered process of differentiation.[2] Some of the reasons that may add complexity to the difficulty in diagnosis could be an oversimplification of grading systems, subjective bias in diagnosis and no specific staging guidelines for intra-oral minor salivary glands. Despite molecular advances, the WHO classifies salivary gland pathologies based on histomorphology.[3,4] History, clinical examination and preoperative investigations attempt to minimise the challenges faced in diagnosing these diverse groups of lesions. Preoperative investigations include imaging and cytopathology. The scope of this review is limited to the preoperative cytopathological investigations and the diagnostic challenges met in reporting SGT.

Preoperative investigatory methods

History, clinical examination and choice of imaging (CT, PET-CT, MRI and neck ultrasound) followed by ultrasound-guided fine needle aspiration cytology (USG

Access this article online

Quick Response Code:

Website: https://journals.lww.com/JPAT/

DOI: 10.4103/jomfp.jomfp_132_24

How to cite this article: Vidyalakshmi S, Shanmugasamy K. Preoperative cytopathological investigatory aids in the diagnosis of salivary gland lesions. J Oral Maxillofac Pathol 2024;28:172-7.

FNAC) molecular testing will provide a preliminary diagnosis of SGT.[1]

Imaging

Preoperative imaging (pre-contrast MRI) provides enormous information on the localization and extension of SGT. MRI aids in differentiating a healing area and foci of recurrence during postoperative follow-up. MRI with contrast should be considered for lesions that hold a clinical suspicion of malignancy. Similarly, post-contrast CT and PET-CT aid in evaluating cortical invasions into the skull base and distant metastasis.[1,5,6]

American Society of Oncology guidelines for preoperative cytological diagnostic methods

American Society of Oncology strongly recommends FNAB/core needle biopsy (CNB) to distinguish malignant from nonmalignant salivary gland lesions. CNB is preferred over FNAB for deeper lesions and to handle sample inadequacies. The risk of malignancy (ROM) should be reported by pathologists using a risk stratification scheme for the FNAB with greater preference for high-grade features. Ancillary testing such as immunohistochemistry (IHC) and molecular studies should be performed in FNAB/CNB to reinforce the diagnosis and the risk of malignancy if present.[7]

Fine needle aspiration cytology

FNAC has established its role as an effective preoperative diagnostic tool for salivary gland lesions. FNAC when performed by a trained clinician/pathologist gives a high specificity of 97% and a low sensitivity of 80%. The clinical usefulness of FNAC is impacted by the adequacy of the sample, the experience of the pathologist, imaging and clinical examination.[8] Gudmundsson, *et al.* (2016)[9] give FNAC a diagnostic accuracy of 100% and 98% for Warthin's tumour (WT) and pleomorphic adenoma (PA). The sensitivity of detecting malignant tumours is much less when compared to benign tumours. The absence of significant atypia in the smears may lead to underdiagnosis of the lesion. Possibility of injury to the facial nerve might occur if the procedure is practiced without any ultrasound guidance[10]

Ultrasound Guided FNAC

USG FNAC holds greater sensitivity for the identification of malignant SGT. Accuracy in the placement of the needle and depiction of the tumour is possible with USG FNAC. USG FNAC has a sensitivity of 68.9%and specificity of 92.5% and diagnostic accuracy of 84.3% and predictive value of 83% for malignant SGT.[9]

Reporting systems

FNAC provides an intermediate accuracy rate of 60%–75% in the SGTs owing to inherent limitations in the nature of the tumours.[11] International Academy of Cytology and American Society of Cytopathology together in 2015 developed a risk assessment and classification system called the Milan System of Reporting Salivary Gland Cytopathology (MSRSGCP) to standardize and improve the diagnostic accuracy and better understanding of clinicians and pathologists.[10]

MSRSGCP is a tiered reporting system built to bring uniformity in reporting. It has 6 tiers of categories of diagnosis with the stratification of ROM and management algorithms for the surgeons.[12] The risk stratification and categorization aimed to face challenges such as the decision of the extent of surgery, whether to conserve facial nerve, better communication among clinicians, pathologists and surgeons, and cytohistological correlation [Table 1].[13–15]

Sample adequacy Minimum of 60 lesional cells is the adequacy criterion set by MSRSGC.

Many authors studied the application of MSRSGC prospectively and retrospectively in their institutions and came out with differing ROMs and some of the categories (SUMP and AUS) presented with diagnostic challenges. Furthermore, reproducibility studies will attempt to refine the categories and their ROM and the diagnostic challenges.[11,14,16–19]

Digital FNAC

Digital cytology

Digital cytopathology (DC) is useful in conducting quality checks, expert opinions educational, research and documentation purposes.[20] Depth of focus and lack of resolution could be the limitations encountered with DC. Multiplane scanning in virtual microscopy allows visualization of cellular material that is obscured in high-grade lesions.[21]

Telecytology

Telecytology (TC) is the reporting of digitalized cytologic slides in the form of images or videos from any remote distance. TC ensures seamless workflow and diagnostic accuracy with MSRSGC.[11,22] Static TC mandates an experienced cytopathologist, unlike dynamic TC.[23]

Diagnostic challenges in fine needle aspiration cytology

FNAC aids in differentiating non-neoplastic lesions from neoplastic lesions and also provide details on the sub-type of the lesion. Benign and malignant lesions can be differentiated, exceptional being the low-grade tumours where the atypia is not appreciated.[24]

Table 1: Milan system of reporting salivary gland cytopathology (MSRSGCP)[10,12-15]

Risk stratification Category	Risk of malignancy assigned	Decision
Non Diagnostic	25%	Correlate clinical features and Imaging details or Repeat FNAC
Non Neoplastic	10%	Clinical follow up, Radiological correlation
Atypia of undetermined significance (AUS)	20%	Repeat FNAC/Surgery
Neoplasm		
I. Benign	<5%	Surgery/Clinical follow up
II. Salivary gland Neoplasm of uncertain malignant potential (SUMP)	35%	Surgery
Suspicious of malignancy	60%	Surgery
Malignant	90%	Surgery

Basaloid lesions

Pleomorphic adenoma

PA is the most common (77,5%) of all SGTs comprising 93.93% of all benign SGTs.[24–26] Smears of PA can be identified based on the typical magenta fibrillar chondromyxoid stroma and the biphasic pattern of cells. The proportion of the cellular and the stromal components poses diagnostic challenges. The abundance of a fibrillar homogenous stroma with basaloid cells should include Adenoid cystic carcinoma (AdCC) in the differential diagnosis. Squamous metaplasia and mucoid material should be differentiated from muco epidermoid Carcinoma (MEC). The presence of stellate, spindle and plasmacytoid cells in the smears should rule out plasma cell tumours and malignant lymphoma.[27–31]

Adenoid cystic carcinoma

AdCC presents a characteristic acellular globular matrix surrounded by basaloid round cells with scanty cytoplasm. The nuclei show coarse granular chromatin. The solid variants resemble PA, basal cell adenoma (BCA), and polymorphous adenocarcinoma (PAC). In PA, cells will have a moderate cytoplasm and fine chromatin, unlike AdCC.[30,32,33]

PLAG1 IHC is more sensitive than FISH. PA tests positive for PLAG1 in IHC, where the pattern cannot be identified in smears. PAC shows positivity for PLAG1, which can be ruled out based on the biphasic presentation of PA and clinical presentation of PAC.

AdCC shows increased expression of *Myb*. The ductal cells and myoepithelial cells of AdCC show patchy and strong immunoreactivity for *Myb*.

Basal cell adenoma

Cytosmears of BA comprising 1%–3% of the SGTs, show very less matrix components and small round cells arranged in varying patterns. Smear is highly cellular with cohesive basaloid epithelial cells arranged in a solid, tubular pattern. Individual cells show scant cytoplasm with monomorphic bland nuclei, surrounded by variable amounts of dense eosinophilic acellular stromal material. The small round basaloid cells might pose diagnostic difficulty because they resemble PA, AdCC and PAC. The presence of myoepithelial cells, chondromyxoid stroma and lack of acellular round globular matrix favours the diagnosis of PA and AdCC. PAC can be distinguished histopathologically with variable degrees of patterns.[34–36]

Ancillary technique

Lymphocyte enhancer-binding factor 1 (LEF-1), a downstream mediator of the WNT/b-catenin signaling pathway, is overexpressed in the nuclei of BA but is typically negative in AdCC. However, LEF-1 expression can also be seen in other benign (PA) and malignant (BCAC) neoplasms.

Oncocytic neoplasms

Warthin's tumour

The cytological smears of WT show two types of cells in a dirty background. Cohesive oncocytes in sheets interspersed with abundant reactive lymphoid cells in lymphoglandular bodies and macrophages.[37,38] The absence of cellular components and the presence of metaplastic cells in the smears may be challenging in the cytological diagnosis of WT.[38] Individual oncocytic cells show abundant eosinophilic granular cytoplasm with uniform bland nuclei and basaloid cells show scant cytoplasm with bland nuclei. Surrounding areas show matured lymphocytes. Oncocytoma, oncocytic-rich acinic cell carcinoma (ACC) and lymphoepithelial lesions should be considered in differential diagnosis.[37]

Oncocytoma

Oncocytoma represents less than 1% of the SGTs and shows large cells with abundant cytoplasm and intense eosinophilia arranged in varied patterns. The cytopathological diagnosis is challenging because it resembles non-neoplastic lesions such as oncocytic hyperplasia, WT, oncocytic carcinoma. The large cells of oncocytes can be confused with ACC.[39] Individual tumour cells show abundant eosinophilic granular cytoplasm with bland uniform nuclei. Oncocytic-rich needs to be

considered in the differential diagnosis of oncocytoma along with WT. All oncocytic neoplasms show positive phosphotungstic acid-hematoxylin and basal cells are positive for p63 in oncocytoma.

Oncocytic rich acinic cell carcinoma

The highly cellular smear shows acinar cells arranged in sheets admixed with oncocytic cells. Acinar cells show abundant granular cytoplasm with multiple vacuoles with uniform low-grade nuclei. Few clear cells are seen in the background admixed with inflammatory cells.

SOX10 positivity helps differentiate ACC from Oncocytoma and WT in cytology. Oncocytoma and WT are SOX10-negative tumours. DOG 1 is positive in Oncocytic-rich ACC. P63 is negative in ACC and positive in MEC thus helping to differentiate it from oncocytic MEC and other oncocytic neoplasms.[37]

Acinic cell carcinoma

ACC comprises 17% of the malignant SGTs. Well-differentiated ACC is diagnosed in the presence of large polygonal acinar cells with abundant cytoplasm and round monomorphic nucleus. Tumour pose difficulty in diagnosis where the acinic cells resemble the acinar cells of normal salivary glands. Cystic changes may resemble MEC, however, the absence of intermediate cells and epidermoid cells favours the diagnosis of ACC. Acinic cells display a greater morphological similarity to oncocytes and can be differentiated using a PAS stain. The presence of lymphoid cells may again include WT in the differential diagnosis. Plasma cells, mast cells and macrophages in addition to large oncocytes and lymphoid cells favours the diagnosis of WT. When the acinar cells are less differentiated it might be difficult to differentiate them from high-grade tumours and mandates a tissue biopsy.[40–42]

Lymphoepithelial lesions

1) WT (cyst adenolymphoma, papillary cystadenoma lymphomatosum).
2) Noncancerous lymphoepithelial lesions, such as adenolymphoma, lymphoepithelioma, Mikulicz's disease and syndrome and Sjogren's disease.
4) A harmless lymphoepithelial cyst.
5) Malignant lymphoepidermal lesion (lymphoepithelial carcinoma).[38]

Other malignant tumours

Salivary duct carcinoma

The highly cellular smear shows atypical epithelial cells arranged in papillary, tubular and cribriform patterns admixed with areas of necrosis. Individual tumour cells show atypia with moderate eosinophilic cytoplasm nuclei showing pleomorphism with prominent nucleoli. AR, GCDFP15, CK7, 34βE12, CEA, AE1/AE3, EMA and GATA3 shows positivity in salivary duct carcinoma.

Mucoepidermoid carcinoma

MEC comprises 10%–15% of malignant SGTs. High-grade MEC can be diagnosed with minimal effort in FNAC in the presence of atypical squamous cells, small intermediate cells and large foamy mucous cells with eccentrically placed nuclei in a dirty mucoid background. Low-grade MEC is difficult to differentiate from retention cysts, WT in the presence of lymphoid cells, and squamous cell carcinoma in the presence of atypical squamous cells. Careful screening for intermediate cells avoids the diagnostic dilemma.[43,44]

Polymorphous adenocarcinoma

Smears of PAC are difficult to differentiate from PA and AdCC. However, the presence of large cytoplasm and pale vacuolated nuclei and the greater propensity for occurrence in the intra-oral minor salivary glands help in the diagnosis of AdCC and PAC, respectively.[32,39,45]

Core needle biopsy

CNB employs ultrasound thereby intact tissue cores can be retrieved from the specimen. CNB can diagnose both low grade and high-grade tumours and also assist in better tumour typing than FNAC**.** Minor complications such as transient facial injury, hemorrhage and fistulas can occur with CNB.[33,40]

Ancillary tests to improve the diagnostic accuracy

Systematic application of ancillary tests such as immunohistochemical (IHC) stains and Fluorescence *in situ* hybridization (FISH) can be potential diagnostic tools to improve precise diagnosis of FNAC. The chromosomal rearrangements in SGTs are found to be characteristic and recently recognized as a diagnostic marker that provides better information on the nature of the tumour.[41]

Molecular diagnostics

Tissue susceptibility, genetic and epigenetic predispositions, increased rate of formation and greater degree of penetrance of the fusion oncogene will be a precise diagnostic and prognostic aid. Transcription factors *PLAG1* and *HMGA2* and *MAML2* and *TORC1* are found to be involved in Notch and cAMP signalling pathways. *PLAG1* is PA gene 1. *HMGA2*, *MAML2* and *TORC1* are not specific for SGTs they are expressed in embryonic tissues, salivary glands, lung carcinomas and benign glandular tumours.[42]

More than 50% of PA shows chromosomal rearrangement in 8q12 and fusions in the *PLAG1* gene. 15% of PA presents with chromosomal rearrangement in 12q13–15 loci and fusions in the *HMGA2* gene. Detection of these two genes is unique to PA and can be diagnosed in RT-PCR and FISH.

However, 40%–80% of MEC shows fusions in CRTC1-MAML2 gene and chromosomal rearrangement in t(11;19)(q21;p13) region. 80% of AdCC shows fusions and activation in the *Myb* gene and translocations in the 6q22-23 region.

10% of AdCC shows fusions in the *MybL1* gene and translocations in the 8q13 region. However, 5%–10% of AdCC shows mutations in NOTCH1.[46,47]

Recommendations

Multiple smears from multiple sites after confirming the site for aspiration should be done and assessed to avoid heterogeneity and sampling inadequacy. Sampling adequacy can be ensured by the rapid onsite evaluation technique (ROSE technique) by the pathologist. The smears should be screened for the presence of cellularity (spindle, atypical squamous cells, oncocytes, acinar cells and lymphoid cells), matrix (afibrillar, fibrillar and chondromyxoid) and cystic components. Special stains such as Giemsa and Papanicolaou aid in differentiating the stroma and the cells in case of dilemma. Ancillary testing should be employed to confirm the suspicion. Imaging and clinical information like site of the lesion helps in differentiating lesions with cytologically overlapping features. Frozen sections can be considered in case of misleading diagnosis.

CONCLUSION

Preoperative evaluation is crucial for treatment planning. Effective clinical examination, history and imaging and other diagnostic investigations along with ancillary tests and adapting MSRSGCP will limit the differential diagnosis in case of rare and complex SGTs. A redefined classification for salivary gland pathology that includes imaging, cytology, and molecular characteristics of the lesions would further bring down the complexity of the diagnostic challenges of this category.

Key messages

Salivary gland cytopathology provides adequate information to the clinician and surgeon, provided the procedure is performed adapting the guidelines. FNAC of salivary glands differentiates between non-neoplastic from neoplastic lesions. The cells and their patterns and matrix help in identifying the tumour and its subtypes.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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