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Schwannoma-Like Pleomorphic Adenoma: A Rare Diagnostic Pitfall in Salivary Gland Pathology. A Critical Review of The Literature with A Practical Diagnostic Approach

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Abstract | **Original Research Article**

Schwannoma-like pleomorphic adenoma (SLPA) is an uncommon variant of pleomorphic adenoma (PA) in which modified myoepithelial cells grow as spindle cell sheets with nuclear palisading, closely resembling the Antoni A areas of a schwannoma. The pattern can mislead the pathologist on cytology, on small biopsies, and sometimes on resection specimens. To gather the published cases, appraise the diagnostic criteria used to separate SLPA from true schwannoma and other spindle cell lesions of the salivary region, and propose a pragmatic diagnostic approach. Narrative review with structured case extraction. We identified reports through targeted searches and citation tracking, and included cases in which a PA showed extensive schwannoma-like areas. Schwannomas with glandular elements were treated as a differential diagnosis, not as cases. We identified eight reports describing 11 cases (1977-2025). Individual data were extractable for 10 cases: median age 54 years (range 39-90), eight women, eight parotid tumors, one submandibular and one hard palate lesion; the size, when reported, was 1.5-3.5 cm. All cases showed palisaded spindle cell areas; the ductal epithelial component could be as small as about 5% of the tumor. Cytokeratin and S-100 protein were consistently expressed. In the four recent reports that documented p63, spindle cells were positive, but the extent varied from focal to diffuse. S-100 protein, SOX10, and CD10 did not discriminate SLPA from schwannoma. We found no reports of recurrence or malignant transformation, although follow-up was inconsistently documented. SLPA is probably under-recognised. A panel including cytokeratin, p63, and a neural marker, supported by generous sampling for ductal or myxochondroid foci, resolves most cases. The role of PLAG1 remains to be tested in this specific setting. Standardised reporting criteria and molecular data are needed.

Keywords: Pleomorphic adenoma; schwannoma; salivary gland neoplasms; myoepithelial cells; p63; PLAG1.

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INTRODUCTION

Pleomorphic adenoma is the most frequent salivary gland neoplasm and accounts for about two-thirds of benign salivary tumors [1,23]. The parotid gland is the usual site, but minor glands can also be involved [1]. Its reputation rests on morphological variety: ductal epithelium is intermingled with modified myoepithelial cells that may be polygonal, plasmacytoid, clear, spindle-shaped, or embedded in a myxoid, chondroid, or hyalinised stroma [1,23]. This plasticity is the source of most of the diagnostic traps associated with the tumor.

One such trap is the schwannoma-like pattern. Spindle-shaped modified myoepithelial cells commonly form interlacing fascicles that suggest neural or smooth muscle differentiation. Occasionally their nuclei line up in palisades, which is usually a focal finding inside an otherwise conventional tumor [7,23]. When this pattern occupies most of the lesion, and the epithelial component is scanty, the tumor looks like a schwannoma, and the term schwannoma-like pleomorphic adenoma (or, in older papers, schwannoma-like mixed tumor) is applied [1,2,3]. The first descriptions of PAs resembling mesenchymal tumors date from 1977 [6].

The distinction matters for practical reasons. Schwannomas can occur within the parotid, usually as facial nerve tumors, and their management raises specific questions about nerve preservation [14,15]. Pleomorphic adenomas, on the other hand, are prone to recurrence

after incomplete or enucleation surgery, and a small proportion give rise to carcinoma ex pleomorphic adenoma, particularly after repeated recurrences [1,21,22,25]. A tumor labelled as schwannoma on a limited sample may therefore be approached with the wrong surgical plan and the wrong follow-up.

The available literature on SLPA consists of isolated case reports and small summaries, written under different names and with different immunohistochemical panels. No synthesis has examined how consistent the proposed diagnostic criteria really are. The aims of this review are (i) to consolidate the reported cases, (ii) to appraise the diagnostic features and markers, in particular their reproducibility across reports, and (iii) to propose a practical approach for diagnosis and clinical management.

METHODS

Design and reporting

This is a narrative review with structured case extraction. Because the evidence base is composed entirely of case reports, a quantitative synthesis was neither feasible nor appropriate. The reporting follows the spirit of the PRISMA 2020 statement for the items that apply to a narrative synthesis.

Eligibility criteria

We included reports in which a salivary gland tumor was diagnosed as PA with extensive schwannoma-like (Antoni A-like, palisaded spindle cell) areas. Terms such as "schwannoma-like mixed tumor" and "pleomorphic adenoma with nuclear palisading of modified myoepithelial cells" were accepted as equivalent. Benign glandular schwannoma, schwannoma with epithelial elements, and intraglandular schwannoma were excluded as cases but were reviewed as differential diagnoses [11-13]. Articles in any language were considered if an English abstract or full text was accessible.

Information sources and search

The search terms were combinations of "schwannoma-like pleomorphic adenoma", "schwannoma-like mixed tumor", "pleomorphic adenoma palisading", "pleomorphic adenoma mimicking schwannoma" and "salivary gland schwannoma", with additional queries on PLAG1 and p63 in salivary and soft tissue tumor. The index reports and their reference lists were then screened (citation tracking). Searches were carried out in October 2026.

Data extraction

For each case we recorded age, sex, site, size, duration, imaging and cytology findings when available, the proportion of schwannoma-like areas, the epithelial component, the immunohistochemical profile, the final diagnosis and the follow-up. Where only an abstract was accessible (one report, ref. 5), no individual data were extracted and the case is counted in the total but not in the descriptive statistics. Descriptive statistics are ours, computed from the published values.

Appraisal

A formal risk-of-bias tool was not applied to single case reports. Instead, each report was appraised for the clarity of its diagnostic reasoning: whether the immunohistochemical panel was sufficient to exclude schwannoma, whether the epithelial component was documented, and whether follow-up was stated.

RESULTS

Volume and nature of the evidence

Eight reports were retained: Merino and LiVolsi (two cases, 1977) [6], Takeda and Shimono (two cases, 1999) [7], Kajor *et al.,*(2006) [8], Tille *et al.,*(2011) [4], Lombardi *et al.,*(2013/2014) [1], Chandra *et al.,*(2017) [3], Roth *et al.,*(two cases, 2020) [2] and Gautam *et al.,*(2025) [5], for 11 cases in total. The two previous compilations counted seven [3] and ten [2] cases, respectively, which is consistent with our count once the later reports are added. All are single-centre case reports or very small series. The reporting rate has been roughly one report every few years, and a good part of the literature predates routine use of p63.

Clinical and imaging features

Table 1. Reported cases of schwannoma-like pleomorphic adenoma with extractable data

REPORT	AGE (Y)	SEX	SITE	SIZE (CM)
Merino and LiVolsi, case 1 [6]	74	F	Parotid	2.5
Merino and LiVolsi, case 2 [6]	39	F	Parotid	1.5
Takeda and Shimono, case 1 [7]	62	M	Parotid	NR
Takeda and Shimono, case 2 [7]	48	F	Hard palate	NR
Kajor *et al.,*[8]	75	F	Parotid	3.0
Tille *et al.,*[4]	47	F	Parotid	3.5
Lombardi *et al.,*[1]	44	F	Parotid (deep lobe)	1.5
Chandra *et al.,*[3]	60	M	Parotid (superficial lobe)	NR
Roth et al., case 1 [2]	90	F	Submandibular	NR
Roth et al., case 2 [2]	40	F	Parotid	NR

NR, not reported in the sources accessible to us. The case of Gautam *et al.,*[5] is counted in the total (n = 11) but not tabulated.

The median age was 54 years (range 39-90), and eight of ten patients were women. Eight tumors arose in the parotid, one in the submandibular gland (reported by Roth *et al.,* as the first example in this location [2]) and one in the hard palate. Where size was given, the lesions measured between 1.5 and 3.5 cm. The patient in the report by Chandra *et al.,* had noticed the swelling for about 12 years, with recent growth [3]. The small numbers do not allow any claim about true sex or age predilection.

Imaging was rarely informative. In the case of Lombardi et al., MRI showed a well-defined deep-lobe mass with parapharyngeal extension and was read as compatible with PA [1]. In the case of Chandra et al., CT showed a hyperdense, circumscribed lesion of the superficial lobe, and the pre-operative working diagnosis was a benign salivary or soft tissue tumor [3]. We found no imaging sign that was specific for SLPA.

Cytology and small biopsies

Fine-needle aspiration is the usual first step for a salivary mass, and it is generally reliable for PA. The schwannoma-like pattern is a recognised weakness. Several authors warn that aspirates and incisional biopsies of SLPA can be taken for schwannoma or other spindle cell lesions [1,3]. The mirror situation also exists: schwannomas of the parotid have been aspirated and interpreted as PA [9,10], and a schwannoma containing epithelial elements has mimicked PA on cytology [11]. In the two cases of Roth et al., the cytological differential included schwannoma, cellular PA and myoepithelioma [2]. In terms of the Milan System, such aspirates, which show spindle cells without the typical fibrillary myxoid matrix, probably fall in the non-diagnostic, atypia of undetermined significance or salivary gland neoplasm of uncertain malignant potential categories, depending on cellularity and the observer [17]. This point has not been studied formally for SLPA.

Histology

The tumors were circumscribed and mostly encapsulated. The defining feature in every report was a sheet-like proliferation of spindle cells with elongated, sometimes hyperchromatic nuclei arranged in palisades, with Antoni A-like architecture [1,3,6,7]. The proportion of schwannoma-like tissue was explicitly quantified in only one report: about 95% of the lesion, with a minor (about 5%) tubular epithelial component and a Ki-67 index of about 5% [1]. In the report of Chandra et al., glandular structures with duct-like differentiation were found in a background dominated by Antoni A-like tissue [3]. Cytological atypia, necrosis and infiltrative growth, the features that raise the possibility of malignancy, were absent in the reports that describe the tumor in detail [1,3].

Immunohistochemistry

Table 2: Immunohistochemical findings in schwannoma-like areas and their diagnostic value

MARKER	FINDINGS IN SLPA	DIAGNOSTIC VALUE
Pan-cytokeratin	Positive in spindle and epithelial cells, stronger in glands [1]	Supports epithelial lineage; essential to exclude schwannoma
S-100 protein	Positive [1,3]	None; shared with schwannoma [3]
SOX10	Positive in the case tested [3]	None; does not separate SLPA from schwannoma [3]
p63	Focal in one case [1]; strong or diffuse in others [2,3,5]	High when positive; negative or focal staining does not exclude SLPA
CD10	Focal [1,3]	Limited [3]
Smooth muscle actin	Focal or weak [1]	Helps to discuss myoepithelioma and leiomyoma
Ki-67	About 5% in one case [1]	Low index supports benignity

The central observation is that the neural markers are of no help. S-100 protein and SOX10 are expressed by myoepithelial cells and by Schwann cells alike, and CD10 is only focal [1,3]. What separates SLPA from schwannoma is therefore the demonstration of epithelial lineage in the palisaded cells. Cytokeratin staining of the spindle cell areas is the argument most often invoked [1]. p63 is the second pillar: in the cases of Roth et al., diffuse nuclear p63 positivity in the schwannoma-like areas was used to exclude both benign glandular schwannoma and intrasalivary schwannoma [2], and the same reasoning is used in the most recent report [5]. The literature should not be read too optimistically, however. p63 was only focal in the case of Lombardi *et al.,*[1], and p63 is not entirely absent from soft tissue tumors [16]. The extent of staining therefore varies between reports, and this variation has not been explained. It may reflect real biological heterogeneity, differences in antibodies and thresholds, or sampling.

PLAG1 deserves a separate comment. Rearrangement of PLAG1 is the most common genetic event in PA, and PLAG1 immunohistochemistry is a sensitive marker for the tumor [18]. Chandra *et al.,* proposed that PLAG1 might play a role in SLPA similar to that of p63 [3]. This is still a hypothesis. We found no report that tested PLAG1 in a confirmed SLPA, and the specificity of the marker against soft tissue myoepithelial tumor is debated [19]. Novel PLAG1 fusion partners continue to be described in PAs with unusual

morphology [20], which makes a systematic molecular study of SLPA feasible and desirable.

Differential diagnosis

Table 3: Practical differential diagnosis of a salivary spindle cell lesion with palisading

ENTITY	DISTINGUISHING FEATURES
Schwannoma (intraparotid or facial nerve)	No epithelial elements; cytokeratin and p63 negative in tumor cells; capsule continuous with nerve [14,15]
Benign glandular schwannoma	Entrapped or metaplastic glands in an otherwise pure schwannoma; spindle cells lack cytokeratin and p63 [2,12,13]
Cellular PA or myoepithelioma	Cytokeratin and p63 positive; no true Antoni A pattern; the distinction from SLPA is partly semantic [2]
Leiomyoma and other myogenic tumor	Desmin and actin positive; cytokeratin negative [1]
Carcinoma ex pleomorphic adenoma	Atypia, mitoses, necrosis, invasion; examine thoroughly [1,25]

The boundary between SLPA and a myoepithelial-rich cellular PA is more a matter of pattern than of biology. If spindle cell tumors with palisading are all products of the same myoepithelial plasticity, the label "schwannoma-like" is descriptive and the clinical consequence lies elsewhere: it is important mostly because it prevents the wrong diagnosis.

Behaviour, treatment and follow-up

All reported tumors were treated surgically, and the recommended approach is complete excision with preservation of the facial nerve, as for conventional PA [3]. The recurrence rate of PA depends on surgical technique, and facial nerve paresis is a recognised postoperative risk [21]. Recurrent PA of the parotid has its own management and prognostic implications [22]. In the reports we could access, follow-up was documented inconsistently, and we found no description of recurrence or of malignant transformation in SLPA. The evidence is too thin to conclude that the behaviour is the same as that of conventional PA, but there is no signal to suggest otherwise.

Proposed diagnostic approach

The approach below is a synthesis of the reports reviewed. It is not validated and should be tested prospectively.

1. Consider the possibility. A well-circumscribed salivary lesion with a spindle cell cytological or biopsy picture should prompt the question of SLPA, not only of schwannoma, especially if the lesion lies outside the expected course of the facial nerve.
2. Cytology. Report spindle cell aspirates cautiously, following the Milan System, and avoid a definitive diagnosis of schwannoma on aspirate alone [2,9,17]. Cell blocks make immunohistochemistry possible.
3. Sampling. Submit the whole tumor in the case of a resection and look for ductal structures, myxoid or chondroid foci and hyalinised stroma. The epithelial component may represent only a few per cent of the lesion [1].
4. Immunohistochemical panel. At minimum: pan-cytokeratin, p63, S-100 protein or SOX10, and an actin or desmin marker. Interpret S-100 protein and SOX10 as non-discriminating. Pair cytokeratin with p63 [1,2,3].
5. Optional extension. PLAG1 immunohistochemistry, or PLAG1/HMGA2 FISH in equivocal cases, if available, and with the explicit caveat that this application is unvalidated [3,18].
6. Reporting. State the approximate proportion of schwannoma-like areas, the presence of an epithelial component, the markers used, and the absence of atypia or invasion. Recommend management as for PA.

DISCUSSION

Principal findings

This review shows that SLPA is a genuine but rare entity, supported by about a dozen cases published over almost five decades, and that its diagnosis depends on a small number of markers that are used in a fairly consistent way across reports: cytokeratin and p63 in the palisaded cells, with neural markers deemed non-informative.

Critical appraisal of the evidence

Several weaknesses deserve emphasis. First, the evidence is anecdotal; no series exists, and the denominator is unknown, so any statement about frequency is speculative. Second, terminology has been unstable, which may have hidden cases from searches. Third, there is no agreed threshold for the proportion of schwannoma-like tissue that justifies the label. Lombardi *et al.,* described 95% [1], whereas conventional PAs frequently have focal palisading [7]. A threshold such as "predominant" (more than half of the lesion) might be proposed for reporting purposes, but this is an expert convention, not an evidence-based cut-off. Fourth, p63 positivity ranged from focal to diffuse [1,2,3], and a rule requiring diffuse staining would reclassify at least one published case. Fifth, no case has been studied at the molecular level, so it is not known whether SLPA is genetically identical to conventional PA. The possibility that the older cases were interpreted without modern markers should also be kept in mind.

Pathogenetic considerations

The most economical explanation is that SLPA represents an extreme form of modified myoepithelial cell plasticity. Myoepithelial cells in PA are known to take on diverse shapes and to express both epithelial and mesenchymal markers, including S-100 protein, which explains the cross-reactivity with neural tumors [1,3,7]. This interpretation fits the immunophenotype and the shared encapsulated growth, but it has not been tested by molecular methods.

Clinical implications

The practical message is simple. An epithelial marker should be requested whenever a salivary spindle cell tumor is thought to be a schwannoma, and the diagnosis of schwannoma should not be made on cytology alone. An intraoperative or pre-operative misdiagnosis may change the surgical strategy and the counselling about recurrence.

Limitations of this review

The formal database search must be documented and updated, as indicated in the Methods. Reports in languages other than English may have been missed, one report was available only as an abstract, and the case series is too small for inferential statistics. The synthesis may also be influenced by publication bias, since only unusual or well-resolved cases are published.

Research priorities

A multicentre retrospective series of spindle cell PAs, with centralised review, a harmonised panel (cytokeratin, p63, SOX10, PLAG1) and PLAG1/HMGA2 testing, would answer most open questions. Registries of salivary pathology and cytology archives (Milan categories) are suitable sources.

CONCLUSION

Schwannoma-like pleomorphic adenoma is rare, benign in all published reports, and diagnostically treacherous. Recognition depends on suspecting it, on sampling the tumor thoroughly, and on demonstrating epithelial lineage in the spindle cells with cytokeratin and p63, since S-100 protein and SOX10 do not discriminate. Standard criteria and molecular studies are still lacking, and larger series are the next step.

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