

Pharyngeal Liposarcoma: The importance of MDM2 testing for early diagnosis

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Abstract:

Background: Liposarcomas (LPS) of the upper aerodigestive tract are rare and the diagnosis is often confounded by bland histological features that mimic benign entities such as lipoma or polypoid reactive lesions.

Design: A statewide pathology database was searched for adipocytic tumours of upper aerodigestive tract sites, and reviewed for demographic data, clinical outcomes, histopathological features and MDM2 testing.

Results: A total of 6 adipocytic tumours of the upper aerodigestive tract were found, 5 LPS and one chondroid lipoma. Four of the 5 LPS were originally classified as benign and there was marked histological heterogeneity. Two of the 5 cases showed recurrence. All 5 cases of LPS showed amplification of MDM2 and the chondroid lipoma did not.

Conclusions: Adipocytic tumours of the upper aerodigestive appear to be disproportionately represented by LPS. The morphological heterogeneity and absence of atypia were associated with a high proportion of the tumours being misclassified, emphasizing the critical role of molecular confirmation of well differentiated liposarcoma via MDM2 amplification testing.

Keywords: Lipoma, lipomatous tumor, liposarcoma, giant fibrovascular polyp, pharynx, larynx, esophagus, oral, palate, tongue, glottis

Introduction:

Liposarcomas (LPS) are malignant mesenchymal tumours at least partly comprising adipocytes and are generally classified as locally aggressive tumours found in the upper or lower limbs, retroperitoneum and paratesticular area^{1,2}. The terms atypical lipomatous tumour (ALT) and well differentiated LPS (WDLPS) are synonymous with ALT preferred at sites where surgical resection is likely curative such as extremities, and WDLPS used at sites which are less amenable to surgical resection such as the retroperitoneum, paratesticular area and mediastinum².

Liposarcomas of the pharynx, larynx, and oral cavity are rare. Pharyngeal LPS account for 3.8% of head and neck LPS and 0.12% of LPS from all sites^{3,4,5}. They often mimic benign lipomatous neoplasms, non-lipogenic mesenchymal tumours and reactive lesions²⁷⁽⁶⁾. Adipocytic lesions of the upper aerodigestive tract often present clinically as pedunculated, submucosal, smooth and well-defined masses; hence the painless tumour may go unnoticed. Additionally, these lesions show bland features and resemble benign lipomas or fibrolipomas histologically⁶. Radiological findings are generally non-specific, typically a well – circumscribed submucosal lesion without features that reliably distinguish benign from malignant adipocytic tumours⁷. Therefore, the clinical, radiological and histological features are often non-specific, making diagnosis difficult based on clinical presentation and light microscopy alone.

There are various morphological subtypes of LPS, which also significantly complicates the diagnosis of these tumours. The most common LPS of the head and neck are WDLPS (44.5%), followed by myxoid/round cell LPS (37.3%), pleomorphic LPS (9.5%) and dedifferentiated LPS (DDLPS) (7.3%)³. The histology of WDLPS typically shows mature adipocytes arranged in lobules with thick fibrous septa containing scattered atypical, hyperchromatic stromal cells. Although it can mimic a benign lipoma clinically and histologically, the identification of these atypical cells is key in recognising the diagnosis of WDLPS⁸. Dedifferentiation represents progression to non-lipogenic sarcoma which in most cases is high grade², although low grade dedifferentiation is increasingly recognised^{9,10}. Tumour sampling bias can further confound diagnosis by missing the dedifferentiated, atypical stromal cell or adipocytic components of the tumour.

Molecular techniques are a well-recognised tool used to confirm the diagnosis of LPS. ALT/WDLPS and DDLPS typically show supernumerary ring and giant chromosomes with amplification of the 12q13-15 region which includes the murine double minute-2 (MDM2) gene, as well as CDK4, HMGA2 and DDIT3 genes^{11,12}. Under normal conditions MDM2 binds to p53 (a tumour suppressor protein) and regulates its degradation¹³. Consequently, amplification of MDM2 results in inhibition of the pro-apoptotic function of p53 which in turn leads to cell proliferation^{12,14}. No MDM2 amplification is observed in benign lipomatous lesions. Amplification can be detected using fluorescent in-situ hybridisation (FISH), droplet digital polymerase chain reaction (PCR), single nucleotide polymorphisms (SNP) microarray and immunohistochemistry.

The evaluation of MDM2 testing is fundamental in the LPS diagnostic workup as the sensitivity of FISH MDM2 amplification testing in detecting ALT/WDLPS and DDLPS is approximately 93–95%, with specificity close to 100%¹³.

Although MDM2 immunohistochemistry is also often used, its sensitivity and specificity are inferior to FISH, which is currently considered the gold standard. Despite this, FISH is not without limitations. False positives can occur due to low- level amplification or technical variability¹³, and MDM2 amplification can be detected in other sarcomas, so results must be interpreted within the clinical and histopathological context¹⁵.

This study re-examines five upper aerodigestive tract masses that were originally or provisionally reported as benign entities or non-diagnostic, but were ultimately confirmed to be liposarcomas upon retrospective review and/or MDM2 amplification testing. By correlating the clinical presentation, detailed histology and molecular findings, we aim to (i) illustrate the diagnostic pitfalls that lead to delayed or erroneous classification and (ii) emphasise the utility of MDM2 amplification testing in resolving ambiguous adipocytic lesions of the head and neck.

Method:

This retrospective case series searched the Pathology Queensland laboratory information systems from 1999 to 2023 using the following search terms: 'lipoma', 'lipomatous tumour', 'adipocytic tumour', 'liposarcoma', 'fibrovascular polyp', 'pharynx', 'larynx', 'oesophagus', 'oral', 'palate', 'tongue' and 'glottis'. The histopathology reports were reviewed from each search and assessed against the inclusion criteria, which were adipocytic tumours and/or giant fibrovascular polyp with an adipocytic component (or similar descriptive term) for a patient of any age describing a tumour in the pharynx (naso-, oro- laryngo-, hypo-), larynx or oesophagus. Clinical data were reviewed including age at diagnosis, tumour size, sex, initial diagnosis, surgical management, and follow-up outcomes including recurrence and survival. The histopathological slides from the relevant cases were retrieved from archives and reanalysed and characterised by a histopathologist (DL). This study was approved by our institutional review boards (HREC/2025/QMS/124136 and SSA/2026/QHC/124136).

Results:

Six cases met the inclusion criteria, five of which were ultimately diagnosed as liposarcoma and one a chondroid lipoma. The clinical characteristics of the LPS are included in Table 1. Three cases were located in the hypopharynx, one case in the supraglottic pharynx and one case was located in the soft palate/oropharynx. Cases 1-4 (figures 1-4) all clinically presented as polypoid partially obstructing masses within the pharynx. Case 5 presented as a large submucosal mass within the soft palate and anterior oropharynx (figure 5). Radiology was not available in all cases but in case 1 the

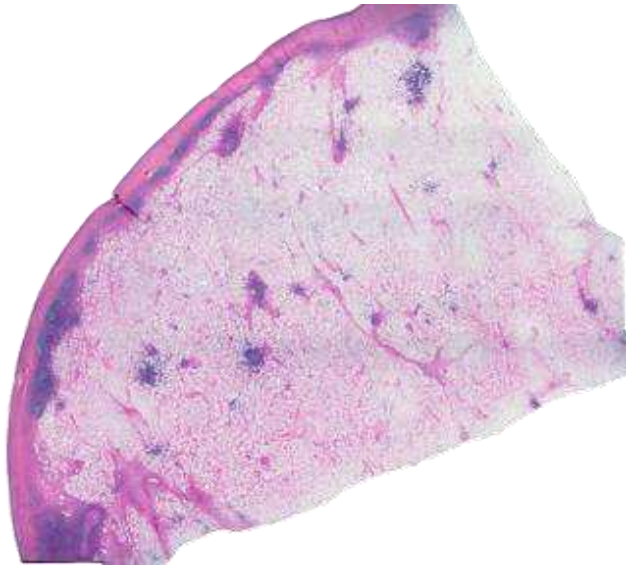
fatty and more solid fibrous zones were demonstrated on computed tomography (CT) scan (Figure 6).

Table 1

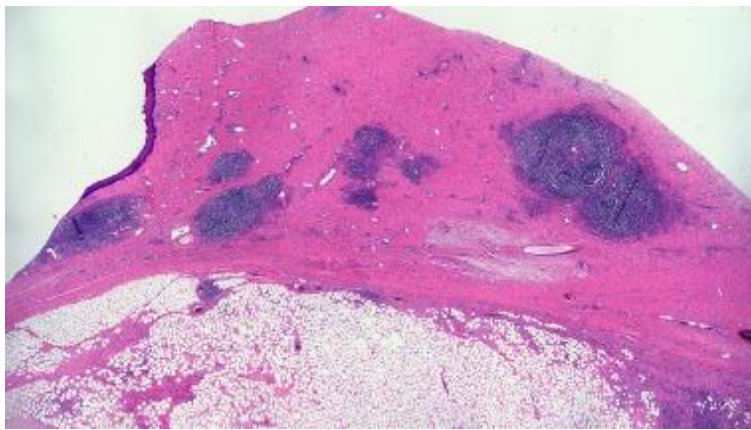
Case number	Site	Age at diagnosis	Sex	Size	Original diagnosis	Behaviour
1	Hypopharynx	63	F	100 mm	Two non-diagnostic biopsies; proceed to excision without histological diagnosis	Completely excised; No recurrence; FU 3 years
2	Pharynx, supraglottic	33	M	65 mm	Spindle cell lipoma	Excised with positive margin, re-excised with no residual and further excision because of nodularity at surgical site, no residual No recurrence at 4 years
3	Posterior hypopharynx	42	M	25 mm	Polypoid reactive lymphoid hyperplasia	Completely excised; no recurrence after 22 months
4	Hypopharynx	53	F	150 mm	Spindle cell lipoma, reassessed as vascular malformation, further reassessed as lipoma	Excised, recurred after 3 years and recurred 5 years after that. Deceased; 6/52 after final excision. COD unclear, multiple comorbidities
5	Soft palate / oropharynx	52	F	30 mm	Lipoma, reassessed spindle cell lipoma	Non diagnostic biopsy with rebiopsy after 4/12, excised with positive margin, recurred after 20 months; no recurrence 3 years later

Figure 1

A.



B

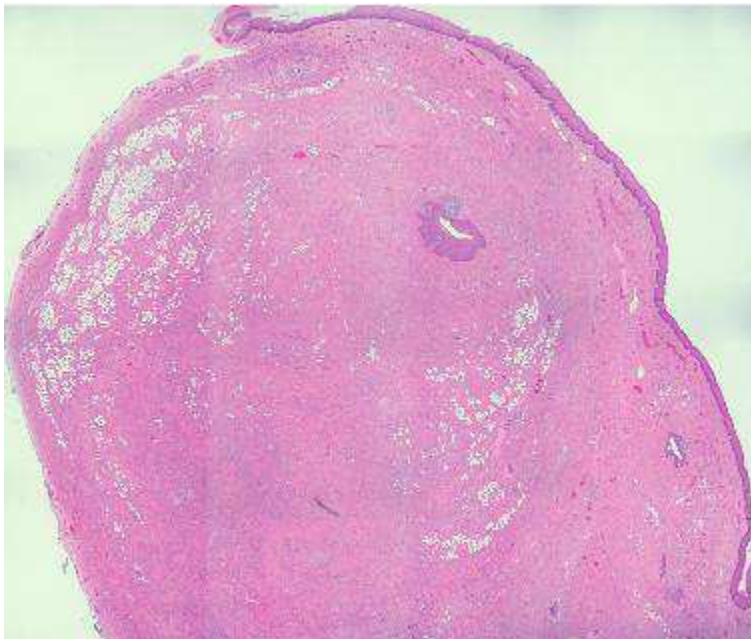


C

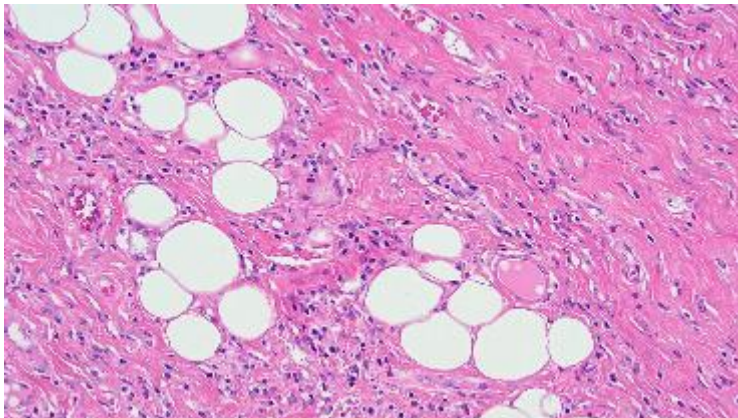
Case 1: A. Polypoid adipocytic lesion with lymphoid aggregates (H&E 20X). B. Sharp demarcation of fatty and fibrous components (H&E 20X). C. Atypical stromal cells with irregular hyperchromatic nuclei (H&E 200X).

Figure 2

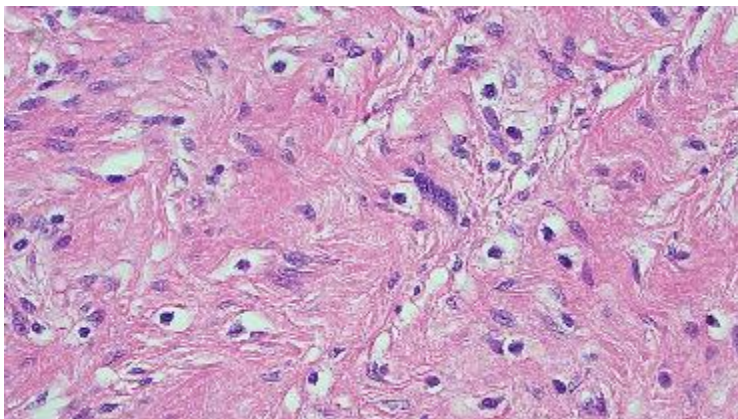
A



B



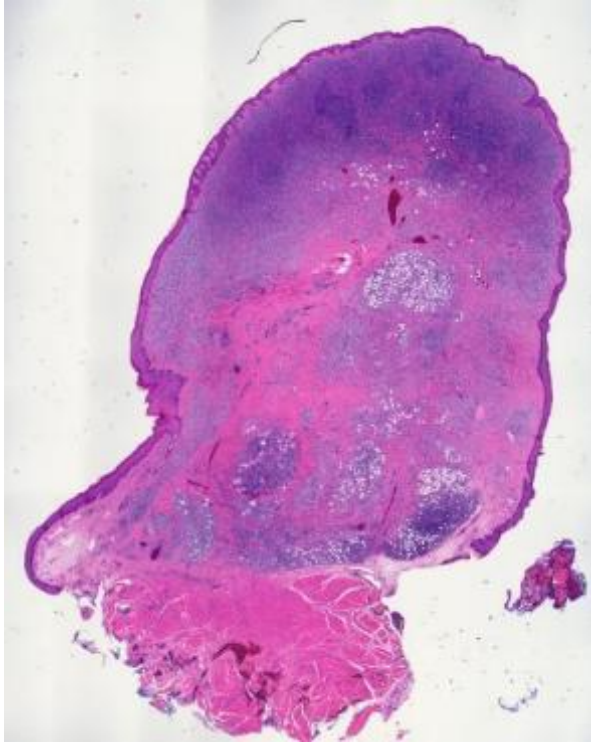
C



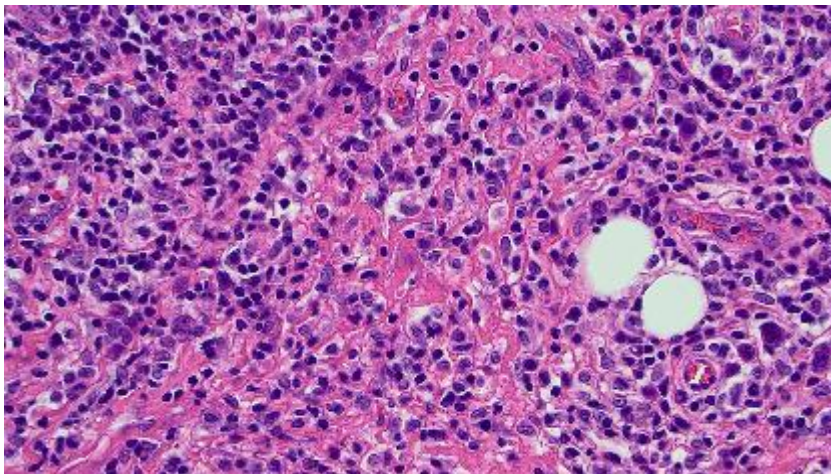
Case 2. A. Submucosal polypoid predominantly fibrous lesion with clusters of adipocytes. H&E 20X B. Spindle cells are mostly banal, set in collagenous stroma with clusters of adipocytes. H&E 100X. C. Occasional enlarged hyperchromatic nuclei are present in the fibrous areas. H&E 400X

Figure 3

A



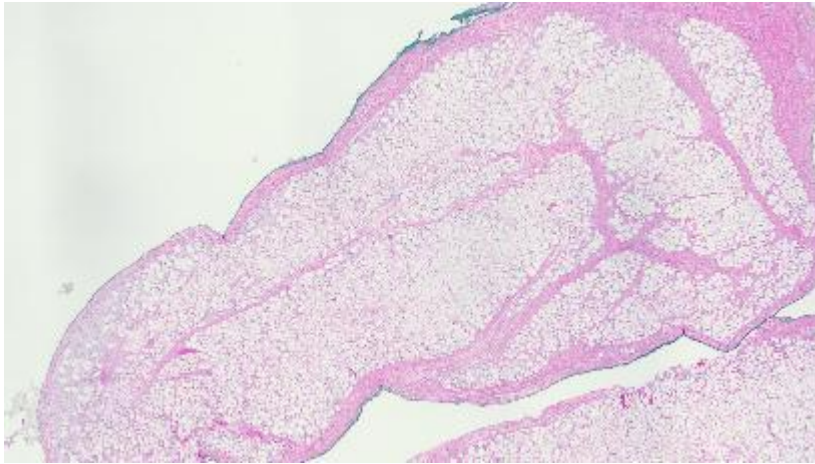
B



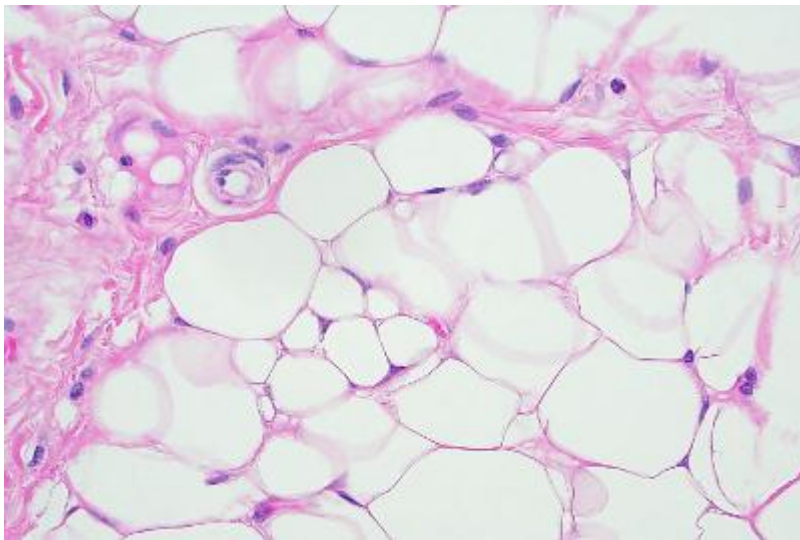
Case 3. A. Submucosal polypoid lesions with predominantly fibrous stroma, clusters of adipocytes and marked inflammation. H&E 20X. B. Stromal atypia difficult to discern in amongst the inflammatory infiltrate. H&E 400X.

Figure 4

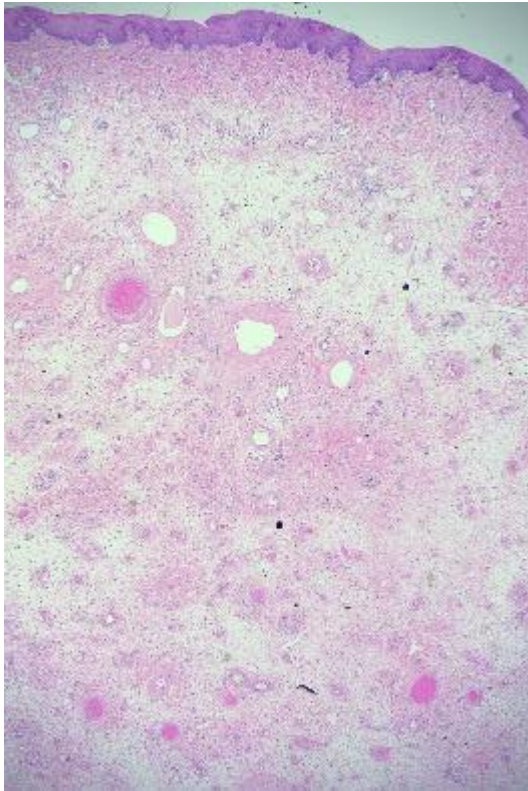
A



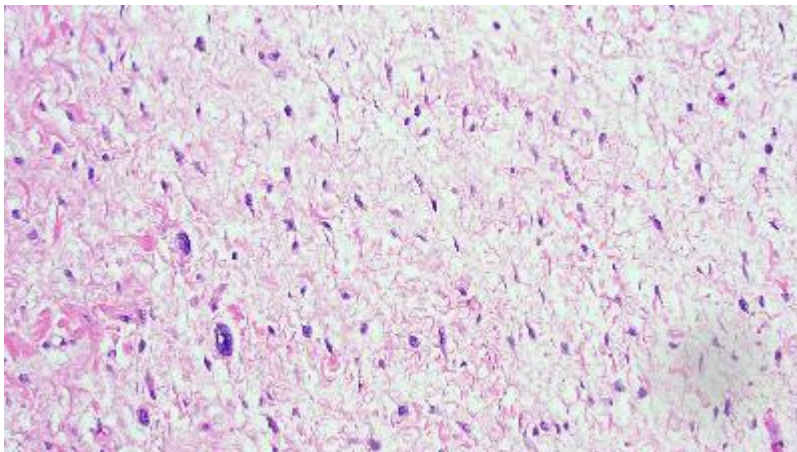
B



C



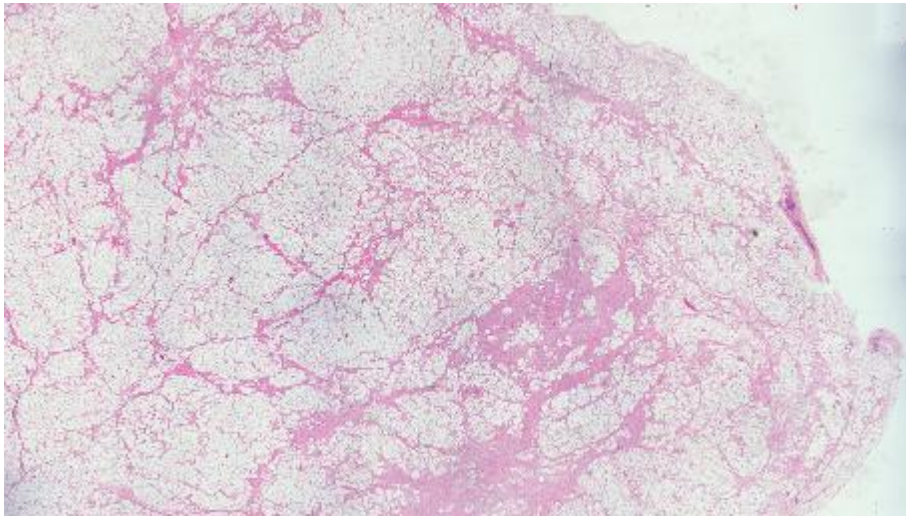
D



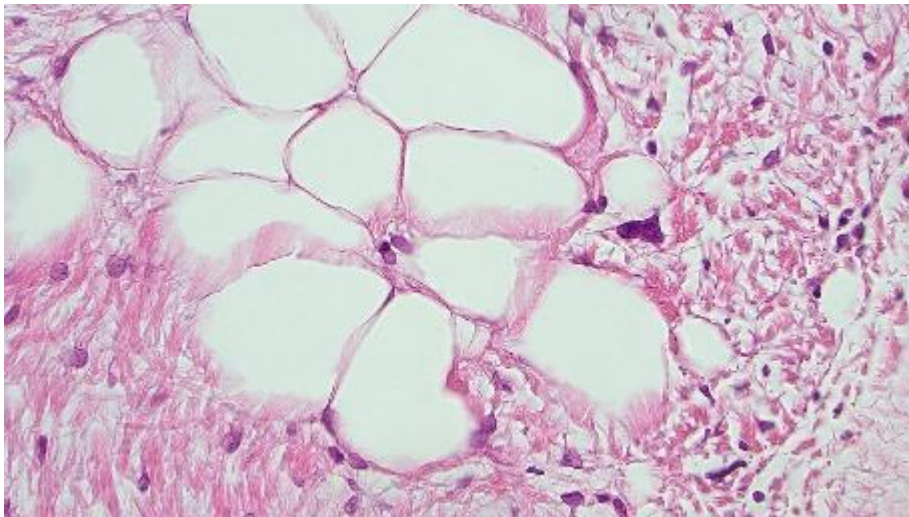
Case 4. A. Lobulated fatty component with thick fibrous septa. H&E 20X. B. Variably sized adipocytes and a multivacuolated lipoblast with a centrally indented nucleus. H&E 200X. C Submucosal myxoid zone with prominent thick-walled blood vessels. H&E 20X. D. Myxoid zone containing spindle cells with enlarged and hyperchromatic nuclei. H&E 200X.

Figure 5

A



B



Case 5. A. Fatty tumour with thick and thin fibrous septa, H&E 20X. B. Rare enlarged hyperchromatic nuclei are present in the fibrous areas. H&E 400X.

Figure 6

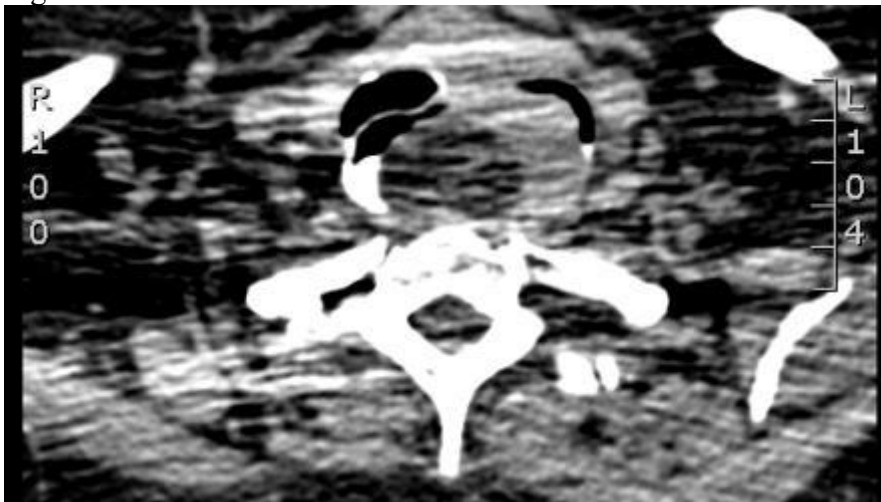


Figure 6. CT image of hypopharyngeal LPS (case 1) with radiolucent zone indicating fat and radiopaque zones demonstrating fibrous tissue.

Histological characteristics (Table 2) varied considerably between cases, reinforcing the morphological heterogeneity of these tumours. However, all cases in our series had significant non-lipogenic components with prominent fibrous septa or fibro-inflammatory stroma; in three cases at least 50% of the tumour comprised fibrous stroma. Lipoblasts were absent (n=3) or rare (n=2) in all cases. Hyperchromatic stromal cells were inconsistently distributed with none found in one case, rare atypical cells seen in 3 cases and frequent in one case. High-grade dedifferentiation, necrosis or mitotic activity were not seen in any case. Immunohistochemistry (IHC) findings were nonspecific. The adipocytes showed positive staining for S100 protein and the fibrous stroma was positive for CD34.

Table 2

CASE	HYPERCHROMATIC CELLS	LIPOBLASTS	HIGH-GRADE DEDIFFERENTIATION	LOW-GRADE DEDIFFERENTIATION / CELLULAR NON-LIPOGENIC	INFLAMMATION	FIBROUS COMPONENT
1	Rare	No	No	Yes	Moderate	20%
2	Rare	No	No	Yes	Mild	70%
3	No	No	No	Yes	Severe	80%
4	Frequent	Rare	No	Yes	Mild	50%
5	Rare	Rare	No	No	None	10%

In 4 of the 5 cases, the tumours were initially classified as benign; three definitively and one was favoured to be benign pending further investigation. Cases 2,4 and 5 were originally classified as a lipoma or spindle cell lipoma due to the predominance of mature adipose tissue and the absence of overt cellular atypia⁶. Case 3 harboured a prominent inflammatory component with a dense infiltrate of lymphocytes and plasma cells which obscured much of the fibrous and adipocytic background. This case was provisionally diagnosed as reactive lymphoid hyperplasia. Case 1 did not receive a definitive initial histological diagnosis at the time of presentation due to superficial sampling which included squamous mucosa and superficial subepithelial stroma. Subsequent formal excision was performed and a final diagnosis was rendered on the excision specimen.

All five cases demonstrated amplification of the MDM2 gene region by fluorescence in situ hybridisation (FISH). In four cases (cases 1,2 ,4, 5), MDM2 amplification testing was performed retrospectively, as the possibility of liposarcoma was not initially considered due to the benign-appearing histology. Case 3 was provisionally diagnosed as benign pending MDM2 testing, with the definitive diagnosis rendered once the MDM2 result was finalised. This molecular result was decisive (Figure 7), both in confirming the diagnosis of liposarcoma and in prompting reclassification of cases that were initially diagnosed as benign entities.

Figure 7

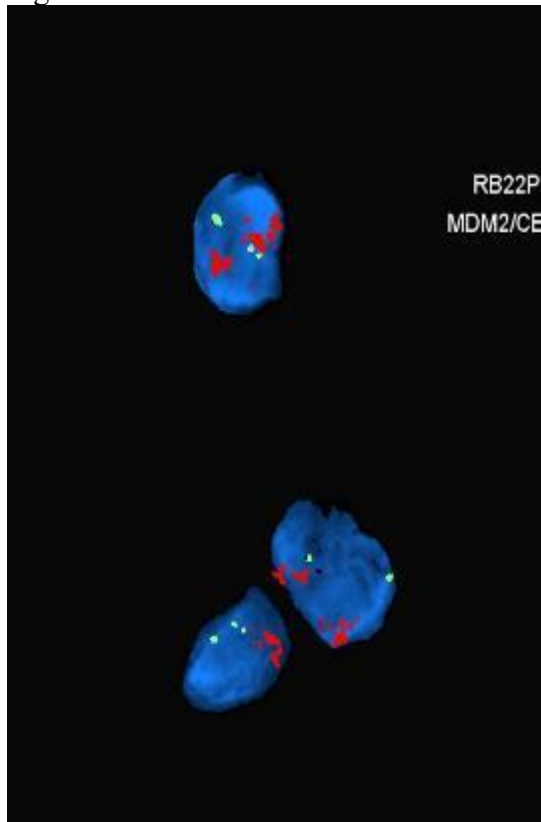


Figure 7. MDM2 red /CEP green FISH case 3.

(These findings align with prior studies demonstrating that lesions historically termed giant fibrovascular polyp or lipoma are frequently re-designated as liposarcoma when MDM2 amplification is demonstrated. Collectively, this underscores the essential role of molecular testing in resolving histologically ambiguous cases and highlights the diagnostic challenges posed by adipocytic tumours in the upper aerodigestive tract.)

Discussion

Adipocytic tumours of the upper aerodigestive tract represent a rare and diagnostically challenging group of neoplasms¹⁶. While lipomas are commonly encountered soft tissue tumours of the extremities and trunk, their presence in the upper aerodigestive tract is exceedingly uncommon. This series supports the growing consensus that adipocytic lesions in this region are far more likely to represent liposarcomas than benign lipomas or fat-containing fibrovascular polyps^{6,18}. Four of the five cases in this study arose in the hypopharynx, with the fifth involving the oropharynx and soft palate—locations where true lipomas are rare⁵.

One of the most critical insights drawn from this cohort is that histological appearances may be deceptively bland, with few or no atypical cells often seen in pharyngeal LPS. Several tumours, notably Cases 2, 4, and 5, exhibited histologically bland cytology

composed of mature adipocytes and fibrous septa with inconspicuous or absent atypical, hyperchromatic cells¹⁷. Despite the paucity or complete lack of these atypical cells, these lesions were ultimately confirmed as LPS following molecular testing on the basis of a tumour of adipocytic lineage with MDM2 gene amplification. This finding emphasizes that even in the absence of overt atypia, routine molecular testing (particularly FISH or droplet digital PCR for MDM2) is essential to differentiate liposarcoma from lipoma in the pharyngeal region^{6,16}.

Case 3 highlights a further diagnostic challenge when an inflammatory component may obscure the nature of the lesion including the presence of cellular atypia¹⁸. The original interim diagnosis was rendered as 'suggestive of reactive lymphoid hyperplasia'. However, the presence of a fatty component warranted further investigation, including submitting tissue for MDM2 amplification testing. Inflammatory WDLPS is a subtype of LPS and can be mistaken for an inflammatory process, including pseudo tumours. Recognition of an adipocytic component is vital and the presence of atypical stromal cells, while not identified in this case, is important. Adipocytic cells in the submucosa in these regions are unusual¹⁹ and their presence in a mass-forming lesion should warrant further investigation with MDM2 amplification testing.

This series also demonstrated significant non-lipogenic areas in four of the five liposarcoma cases. These were characterised by fat-free fibrous zones comprising bland spindle cells in a collagenous stroma, with one case showing a prominent inflammatory component. Fritchie et al described similar non-lipogenic and fibro-inflammatory zones in their series of upper aerodigestive tract LPS²⁰. The possibility of low grade dedifferentiation needs consideration when there is a non-lipogenic component that is not highly atypical¹⁹. Graham et al present evidence that the absence of an adipocytic component is not sufficient for dedifferentiation and that true low grade dedifferentiation requires mitotic activity (≥ 5 mitoses/10 high power field)²¹. Non-lipogenic zones in liposarcomas with mitoses less than 5 per high power field should be considered cellular WDLPS, as they appear to behave in similar fashion to WDLPS rather thanDDLPS which has a poor prognosis^{10,22}. Non-lipogenic zones can be misinterpreted as fibrous scar tissue or reactive stroma, especially when seen on superficial biopsies and in unusual sites. The mitotic index was low in all our cases (<5 mitoses/10 HPF) and none showed high grade sarcoma, therefore definitive evidence of dedifferentiation was not present. Importantly, low-grade dedifferentiation does not show the high-grade, pleomorphic morphology traditionally associated with dedifferentiation and can also be mistaken for tumours of a different lineage²⁰, making it even more critical to combine histology with MDM2 amplification testing^{21,22}.

Historically many polypoid fibrofatty tumours of the pharynx and oesophagus were termed giant fibrovascular polyp (GFP) and were presumed to be benign reactive proliferations^{6,23,24}. These lesions were composed of mature adipose tissue and fibrous stroma and were diagnosed without molecular or immunohistochemical testing. Recent studies—including the current series—have challenged the benign interpretation of these entities²⁰. Many lesions previously labelled as GFPs harbor subtle histological features of liposarcoma, such as enlarged, hyperchromatic stromal cells within fibrous septa and non-lipogenic spindle cell components. Many of these

lesions have been subsequently found to harbour MDM2 amplification. They may also behave in a malignant fashion with multiple recurrences and the potential to dedifferentiate and metastasise. Therefore, many lesions originally diagnosed as GFP likely represent liposarcoma^{6,16}.

Consistent with these observations, our series, showed that two of the five liposarcoma cases which were initially diagnosed as benign behaved in a locally aggressive fashion and developed recurrence. Although no patients developed metastatic disease, one patient died during follow up, though the cause of death could not be determined. These findings emphasise the clinical challenge of managing liposarcomas in the pharynx and upper aerodigestive tract, where complete surgical clearance is often limited by the presence of critical anatomical structures, thereby predisposing to local recurrence.

Three cases in this series were originally diagnosed as lipoma or spindle cell lipoma (Cases 2, 4, and 5). There are numerous descriptions of pharyngeal lipomas in the literature, however, on critical review, the histological descriptions of these tumours were extremely limited or absent^{25-29, 31-35}. Many of the published images were of such poor quality that diagnosis could not be confidently assumed^{32-34,36}, and one contained an image displaying nuclear atypia that would be compatible with LPS but MDM2 amplification testing was not performed³⁸. Some cases described giant cells or multivacuolated adipocytes but did not include images of them^{36,38}. Most cases had no or limited follow up (6 months or less)^{25-28,31,32,34,36,39-41}. Importantly, it is evident that most cases described in the last twelve years (when FISH testing for MDM2 amplification has been available) lacked MDM2 amplification FISH or molecular testing^{25, 26, 29-37,39,42}, although one case showed negative MDM2 immunohistochemistry³⁶.

Interestingly, our search of our local pathology service database did not indicate any pharyngeal lipomatous or spindle cell lipomas that were not subsequently redesignated as liposarcoma with histological review and/or molecular testing over a 24 year period. Mijares group has also reported that liposarcomas are disproportionately represented at this site with 12 of 22 adipocytic tumours representing LPS⁴⁴. The only lipoma in the region which was not subsequently overturned was an unusual chondroid lipoma of the epiglottis, in which MDM2 amplification was not identified and ultimately required external review by a soft tissue subspecialty pathologist with extensive immunohistochemical and molecular work up. These findings reinforce the central thesis of this report: that adipocytic tumours of the upper aerodigestive tract should not be assumed benign based on histology alone. Given their morphologic variability, overlap with historical GFPs, and risk of recurrence, routine MDM2 amplification testing is essential to secure accurate diagnosis and prevent mismanagement.

Conclusion

In summary, this series adds to the growing body of evidence that most adipocytic tumours in the upper aerodigestive tract region represent liposarcoma, despite

mimicking lipomas clinically and histologically. Misdiagnosis is common, both due to bland histological features and sampling bias. The use of objective molecular tools such as MDM2 amplification FISH testing is indispensable in establishing a definitive diagnosis and ensuring appropriate management.

Declarations

Funding: This study was not supported by funding.

Conflict of interest: Duncan Lambie receives consultancy fees from Melanoma and Skin Cancer Trials Ltd.

Alyona Dziouba, Winta Gebreysus and Rachel Wu declare that they have no conflict of interest.

Ethical approval: This is a retrospective study. A waiver of consent was granted by the institutional ethics review board (Metro South Health – Queensland Health).

The study was approved by our institutional review boards (HREC/2025/QMS/124136 and SSA/2026/QHC/124136).

Consent to participate: This study has obtained IRB approval from Metro South Health HREC and the need for informed consent was waived.

Consent for publication: For this type of study consent for publication was not required.

Availability of data and materials: The datasets generated are not publicly available due to the risk of compromising individual privacy and the conditions of the data custodian. De-identified data may be made available to qualified researchers upon reasonable request, subject to approval by the relevant data custodian and completion of appropriate data access agreements. Requests for data access should be directed to Chief Executive Pathology Queensland, and any reuse of the data will be permitted only under these approved conditions.

Code availability: No custom code was used or generated for this study.

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