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Pseudo-Histological Lesions in Oral Pathology: Diagnostic Challenges and Histological Mimics

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Abstract: Pseudo-histological lesions in oral pathology represent a diagnostic challenge due to their striking histological resemblance to true neoplastic, cystic, or inflammatory conditions. Although these lesions are benign and lack malignant potential, they often mimic serious pathologies such as carcinomas, sarcomas, lymphomas, and glandular tumors, leading to potential misdiagnosis and inappropriate treatment. Common examples include pseudoepitheliomatous hyperplasia, pseudocysts, pseudorosettes, pseudoglandular structures, and nodular fasciitis. These lesions arise from reactive, inflammatory, traumatic, or degenerative processes rather than neoplastic transformation. Distinguishing between true and pseudo-forms requires careful histopathological analysis, correlation with clinical and radiological findings, and often the application of ancillary techniques such as immunohistochemistry and molecular diagnostics. This review provides an overview of key pseudo-histological entities in the oral cavity, emphasizing the importance of accurate diagnosis to avoid overtreatment and to ensure appropriate patient management.

Index Terms: Pseudo-histological lesions, pseudoepitheliomatous hyperplasia, pseudocyst, pseudorosette, pseudosarcoma, pseudolymphoma.

I. INTRODUCTION

Histopathological examination remains the gold standard for diagnosing oral lesions. However, in oral pathology, certain benign, reactive, or degenerative conditions may closely mimic true neoplastic, cystic, or inflammatory processes both clinically and microscopically. While they do not share the aggressive biological behavior of their malignant counterparts, their deceptive morphological features can lead to diagnostic confusion, misclassification, and occasionally, overtreatment [1,2]. The "pseudo-histological" designation implies histological similarity without shared histogenesis or pathological progression. These pseudo-histological lesions often arise in response to trauma, chronic inflammation, or reactive tissue processes, and although they may mimic carcinomas, sarcomas, lymphomas, or cystic neoplasms under light microscopy, they are fundamentally non-neoplastic and biologically benign.

For example, pseudoepitheliomatous hyperplasia (PEH) may closely resemble well-differentiated squamous cell carcinoma due to irregular epithelial proliferation and keratin pearl formation [3]. Likewise, pseudorosettes, found in neuroectodermal tumors, mimic true rosette architecture but lack a central lumen or neuropil [4]. Other examples include pseudocysts, which lack epithelial lining yet appear radiolucent and cystic radiographically, and pseudolymphomas, which may histologically simulate true lymphomas if not interpreted with clinical correlation [5,6].

A primary challenge lies in the histological overlap with true lesions, especially in limited or superficial biopsies. Factors such as secondary inflammation, necrosis, or artifact can obscure essential diagnostic criteria. Ancillary tools like immunohistochemistry (IHC), molecular testing, and clinical-radiographic correlation are crucial adjuncts to avoid overdiagnosis or under-treatment [2,5]. This review highlights major pseudo-histological lesions encountered in oral pathology.

Classification of Pseudo-Histological Lesions in Oral Pathology

Pseudo-histological lesions can be classified in two ways: by histological mimicry, and etiological context. This multidimensional framework enhances diagnostic accuracy, helping pathologists distinguish deceptive pseudo-patterns from true pathological entities. **Table 1** summarizes the classification based on the mimicked entity and associated etiological factors.

Table 1. Classification of Pseudo-Histological Lesions in Oral Pathology

A.Classification by histological mimicry

Mimicked Entity	Pseudo-Lesion Example
Carcinoma (SCC)	Pseudoepitheliomatous hyperplasia [3]
Sarcoma	Pseudosarcomatous proliferation [7]
Lymphoma	Pseudolymphoma [6]
Cystic Lesion	Pseudocyst (e.g., simple bone cyst) [5]
Glandular Neoplasm	Pseudoglandular SCC variant [8]
Neuroendocrine Tumor	Pseudorosette formations [4]

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B.Classification by Etiological Context

Etiology	Associated Pseudo-Lesions
Infectious	PEH in fungal infections, tuberculosis [3]
Inflammatory	Pseudolymphoma [6]
Traumatic	Pseudocyst (traumatic bone cyst)[5]
Reactive Neoplasms	Nodular fasciitis (pseudosarcomatous fibromatosis)[7]
Degenerative	Pseudoglandular degeneration in SCC [8]

PSEUDO-HISTOLOGICAL LESIONS IN ORAL PATHOLOGY:

Pseudoepitheliomatous Hyperplasia (PEH) vs. Squamous Cell Carcinoma (SCC)
Pseudoepitheliomatous hyperplasia (PEH) is a benign epithelial proliferation triggered by chronic inflammation, trauma, or infection. In the oral cavity, it is commonly associated with chronic candidiasis, deep fungal infections, or inflammatory gingival hyperplasia. Histologically, PEH exhibits epithelial thickening with irregular elongation of rete ridges, often forming jagged, downward projections with sharply pointed bases. The epithelial-stromal interface tends to be less well-defined, and the proliferating epithelial tongues may anastomose, entrapping stromal components. Hypergranulosis and ortho- or parakeratosis are common, and keratin pearl formation—seen as concentric layers of keratinocytes around a central focus—is frequently observed. Although mitotic figures may be present, they are neither numerous nor atypical. The basal cell layer remains orderly, and there is an absence of cytologic atypia.

In contrast, oral squamous cell carcinoma (SCC) shows architectural disruption, dysplastic basal cells, nuclear pleomorphism, increased mitotic activity, individual cell keratinization, and stromal invasion. Histopathological features that favor SCC include nuclear atypia, atypical mitoses, necrotic keratinocytes, and deep epithelial invasion into connective tissue. Differentiating PEH from SCC is often challenging, especially in small or superficial biopsies due to overlapping features. Therefore, a thorough clinical evaluation, review of multiple histologic sections, and identification of any underlying disease process are essential. In some cases, additional or deeper biopsies may be required. Accurate distinction is crucial, particularly in mucosal sites where SCC carries a poor prognosis with early local invasion and potential lymph node metastasis.[3,9]

Pseudocyst vs. True Cyst

In the oral cavity, lesions may appear cystic on clinical and radiographic examination, but histological evaluation is essential to distinguish between true cysts and pseudocysts. A pseudocyst, such as a traumatic bone cyst (simple bone cyst), is not a true cyst because it lacks an epithelial lining. It typically arises following trauma, ischemia, or disruption of local vasculature, leading to intraosseous cavity formation. Microscopically, the cavity is empty or may contain serosanguinous fluid, and its walls are composed of fibrous connective tissue, often with evidence of hemosiderin-laden macrophages, granulation tissue, and scattered inflammatory cells. No epithelial component is observed.

In contrast, true odontogenic cysts, such as radicular cysts and dentigerous cysts, are characterized by a well-defined epithelial lining, typically of non-keratinized stratified squamous epithelium. The epithelial lining often shows proliferative activity, with arcading patterns or hyperplasia, especially in inflamed cysts. The supporting fibrous wall may show a chronic inflammatory infiltrate, cholesterol clefts, multinucleated giant cells, and occasionally Rushton bodies—linear or curved calcified structures of unknown significance. These histological features confirm the diagnosis of a true cyst.

Although both entities may have a similar radiographic appearance—often presenting as well-defined radiolucencies—histopathological analysis remains the gold standard for diagnosis, relying primarily on the presence or absence of an epithelial lining to differentiate between pseudocystic and true cystic lesions. [5,10]

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Pseudo Horn Cyst vs. True Horn Cyst

Pseudo horn cysts are compact keratin-filled invaginations into hyperplastic squamous epithelium without a true cystic epithelial lining. These structures can be seen in reactive epithelial lesions or inflamed mucosa. True horn cysts, on the other hand, are completely enclosed keratin-filled cystic structures with a distinct basal epithelial border, often found in benign epithelial tumors such as seborrheic keratosis or verrucous hyperplasia. Differentiation is important, as true horn cysts may indicate a neoplastic process requiring excision.[11]

Pseudo Capsule vs. True Capsule

Pseudo capsules are fibrous zones formed by tissue compression or inflammation, commonly seen around mucoceles or organized hematomas. [12] They consist of loosely arranged collagen and chronic inflammatory cells. True capsules, in contrast, are collagenous envelopes that completely encapsulate benign neoplasms such as pleomorphic adenomas, facilitating surgical excision. True capsules have well-demarcated fibrous borders, while pseudo capsules often appear irregular and lack clear separation from surrounding tissue.

Pseudoglandular Spaces vs. True Glandular Structures

In histopathology, distinguishing between pseudoglandular spaces and true glandular structures is crucial for accurate diagnosis, particularly in oral squamous cell carcinoma (SCC) and salivary gland neoplasms. Pseudoglandular spaces arise due to acantholysis, necrosis, or pseudoluminal degeneration of squamous epithelium, creating cavities that mimic true glandular formations. These spaces are typically angular or irregular, lack ductal differentiation, and do not contain mucin. They often contain fibrin, necrotic debris, or serum-like fluid but are negative for mucin stains such as mucicarmine or PAS. These features are characteristic of adenoid (pseudoglandular) variants of SCC, which may be misdiagnosed as glandular tumors on routine H&E staining.
In contrast, true glandular structures originate from ductal or acinar epithelium and are composed of well-organized, round to oval luminal formations. They are lined by cuboidal or columnar epithelial cells and characteristically contain mucin, which stains positively with mucicarmine, Alcian blue, or PAS. These features are seen in mucoepidermoid carcinoma, adenocarcinoma, or adenosquamous carcinoma. Unlike the diffuse pseudoglandular patterns seen in SCC, adenosquamous carcinomas show focal glandular differentiation, and mucoepidermoid carcinomas exhibit abundant mucinous material and true glandular morphology, especially in low to intermediate-grade tumors.
The presence or absence of true mucin, type of lining epithelium, and overall architectural pattern are key to differentiating these lesions. Immunohistochemistry and special stains can aid diagnosis, but careful histopathological evaluation remains the cornerstone. Misinterpreting pseudoglandular SCC as a glandular malignancy can lead to inappropriate management, highlighting the importance of this distinction.[8]

Pesudo rosettes Vs. True rosettes

Pseudorosettes are typically associated with neoplasms of neuroectodermal origin and are defined by the radial disposition of neoplastic cells around a central blood vessel. Unlike true rosettes, the central structure in pseudorosettes is not formed by tumor tissue, but is a native, non-neoplastic element, most often a blood vessel, hence the prefix “pseudo.” These are considered secondary architectural features, often resulting from reactive or degenerative changes in the tumor environment. Pseudorosettes are commonly encountered in a range of tumors, including ependymoma, medulloblastoma, central neurocytoma, and oral neuroectodermal tumors, where their presence can serve as an important diagnostic clue, particularly in poorly differentiated or high-grade malignancies. [13]

A noteworthy histopathologic variant within this category is the Dendritic Cell Neurofibroma with Pseudorosettes (DCNP), a rare, benign intraoral neoplasm. Histologically, DCNP presents with a biphasic cell population forming rosette-like structures: smaller central cells are surrounded by larger, pale-staining dendritic cells arranged in a spoke-wheel fashion. Immunohistochemically, these tumors consistently express S100 protein and CD57, supporting their neural differentiation. Despite their rosette-like appearance, DCNPs are benign and require careful histopathologic evaluation to avoid misinterpretation as aggressive neoplasms. Accurate identification is crucial to prevent overtreatment and ensure appropriate conservative management.[14]

In contrast, true rosettes are composed of tumor cells that form a symmetrical, concentric arrangement around a central lumen or neuropil-rich core, both of which are derived from the tumor itself. These represent a primary architectural feature and suggest a higher level of organization and tumor differentiation. Prominent examples include the Homer Wright rosette, where the center contains fibrillary material composed of primitive neuronal processes (neuropil), and the Flexner-Wintersteiner rosette, which features a clear, empty-appearing lumen. True rosettes are classical features in malignancies such as neuroblastoma, retinoblastoma, and embryonal tumors with abundant neuropil (ETANTR). Though rare in oral pathology, their recognition has significant implications for diagnosis, tumor classification, and prognosis.[13]

Pseudo-Alveolar Structures vs. True Alveoli

Pseudo-alveolar structures are tumor-related architectural patterns, commonly identified in alveolar rhabdomyosarcoma (ARMS) of the head and neck region. These structures are formed by nests of neoplastic cells that demonstrate central discohesion, creating hollow-appearing spaces that resemble alveoli. These spaces are delineated by fibrous septa, but crucially, they lack a true epithelial lining. This pattern gives a deceptive alveolar appearance, hence the term "pseudo-alveolar." Microscopically, these structures are not functional units but reflect tumor growth dynamics. Immunohistochemically, markers such as MyoD1 and desmin are used to

confirm the myogenic differentiation of ARMS, assisting in its distinction from other small round cell tumors. [2,15,16] On the other hand, true alveoli, such as those in pulmonary tissue or salivary gland acini, are functional epithelial structures composed of well-organized secretory cells surrounding a central lumen. These are lined by polarized epithelial cells with defined secretory roles and exhibit basal-apical orientation and cytoplasmic granularity based on the secretory product.

Pseudo-Lymphoma vs. True Lymphoma

Pseudo-lymphoma (PL) refers to a benign lymphoid hyperplasia that mimics lymphoma both clinically and histologically. It is often associated with chronic oral mucosal inflammation, where reactive B-cell clusters resemble lymphoid follicles but lack the architectural and cellular features of true neoplasia. These pseudo-follicular patterns typically present with preserved tissue architecture, polyclonal lymphoid populations, and increased mitotic activity consistent with a reactive process.

In contrast, true lymphomas such as MALT lymphoma or follicular lymphoma—display well-organized germinal centers with surrounding mantle zones and show monoclonal proliferation of lymphoid cells. Histologically, lymphomas are characterized by a monomorphic infiltrate of immature lymphoid cells, often infiltrating deep into the connective tissue stroma. Additional features suggesting lymphoma include nuclear atypia, increased nuclear-to-cytoplasmic ratio, nuclear crowding, and architectural effacement.

Importantly, clinical and histological features alone are insufficient to definitively differentiate pseudo-lymphoma from true lymphoma. A combined approach using clinical history, detailed histopathology, and immunohistochemical analysis is crucial. Immunophenotyping can distinguish lymphocytes by surface markers (e.g., light chain restriction, BCL2, CD20, CD3), enabling assessment of clonality. While PL typically shows a mixed population of mature inflammatory cells, including follicular aggregates with reactive germinal centers, lymphomas exhibit clonal expansion and altered immunoprofiles. Accurate differentiation is critical, as pseudo-lymphomas are benign and often self-limiting, whereas true lymphomas require prompt oncologic management.[9]

Pseudosarcoma (Nodular Fasciitis) vs. True Sarcoma

Nodular fasciitis is a benign, pseudosarcomatous lesion that may present in the oral cavity, commonly as a rapidly enlarging, painless mass. Histologically, it is characterized by a proliferation of spindle-shaped fibroblasts and myofibroblasts within a myxoid to collagenous stroma, often exhibiting a tissue culture-like pattern. Although mitotic figures can be seen, they are typically non-atypical, and nuclear pleomorphism is minimal. The lesion's rapid growth and cellularity can mimic soft tissue sarcomas, leading to potential diagnostic pitfalls.

In contrast, true sarcomas, such as fibrosarcoma or leiomyosarcoma, demonstrate definitive features of malignancy, including cellular atypia, high mitotic index with atypical mitoses, necrosis, and infiltrative margins. Immunohistochemistry and histopathology are essential tools for distinguishing between the two. Importantly, the identification of USP6 gene rearrangements in nodular fasciitis through molecular techniques has become a useful adjunct in confirming its benign nature and avoiding overtreatment.

Diagnosing pseudosarcomatous lesions remains challenging due to histological overlap with malignant neoplasms. While lesions like proliferative fasciitis were once considered reactive to physical trauma, current understanding emphasizes the importance of recognizing specific cytomorphological features to prevent misdiagnosis. Misinterpretation may lead to unnecessary and aggressive treatment of benign conditions.

Therefore, thorough histopathological evaluation remains the cornerstone of diagnosis, supported by ancillary studies to guide appropriate clinical management.

Pseudo-Inclusions vs. True Inclusions

Nuclear inclusions, observable under light microscopy, can be categorized as true inclusions or pseudoinclusions, each with distinct origins and diagnostic implications. True inclusions are intracellular accumulations of viral particles, cytoplasmic substances (e.g., surfactant, immunoglobulins, glycogen), or biotin and are seen in specific disease contexts such as viral infections (e.g., HSV, CMV) or pulmonary adenocarcinoma, often requiring special stains like PAS or immunoperoxidase for confirmation.[17] In contrast, pseudoinclusions are cytoplasmic invaginations into the nucleus, bounded by the nuclear membrane, and are commonly observed in conditions like papillary thyroid carcinoma, meningioma, and follicular dendritic cell tumors. The melanoma cells have large nuclei, often with prominent nucleoli, and show pseudoinclusions due to nuclear membrane irregularity. These may mimic inclusions but do not represent true intracellular deposits. Additionally, pseudoinclusions, which are artefactual vacuoles mimicking pseudoinclusions or clear nuclei, can lead to misdiagnosis, especially in thyroid pathology. In the oral cavity, pseudo-inclusions may refer to extracellular materials like keratin, calcium, or mucin, typically seen in degenerative or reactive lesions such as pseudoepitheliomatous hyperplasia. Distinguishing these various forms through histopathological evaluation, supplemented by immunohistochemistry and molecular diagnostics, is crucial to avoid misinterpretation and ensure accurate diagnosis.[18, 19]
To synthesize the diagnostic distinctions between pseudo-histological lesions and their true pathological counterparts, a comparative table has been constructed (Table 2). This summary outlines the essential histological, immunohistochemical, and clinical criteria that aid in differentiating these mimics, thereby minimizing the risk of misdiagnosis and inappropriate management.

Table 2. Comparison Between Pseudo-Histological Lesions and Their True Counterparts

Pseudo-Lesion	True Counterpart	Key Distinguishing Features
Pseudoepitheliomatous Hyperplasia (PEH)	Squamous Cell Carcinoma (SCC)	PEH: No cytologic atypia, orderly basal layer, minimal mitoses; SCC: Nuclear pleomorphism, atypical mitoses, stromal invasion [3]
Pseudocyst	True Cyst	Pseudocyst: No epithelial lining; True cyst: Lined by epithelium [5]
Pseudoglandular SCC	Glandular Tumors (e.g., MEC)	Pseudoglands: Irregular spaces, no mucin, no ductal lining; True glands: Organized acini, mucin-positive staining [8]
Pseudorosettes	True Rosettes	Pseudorosettes: Cells around blood vessels (non-neoplastic center); True rosettes: Tumor cells around lumen or neuropil-rich core [13]
Pseudo-Alveolar Structures	True Alveoli	Pseudo-alveoli: Central cell discohesion, no epithelial lining; True alveoli: Functional epithelial cells with lumen [15]
Pseudolymphoma	Lymphoma	Pseudolymphoma: Polyclonal, preserved architecture; Lymphoma: Monoclonal, effacement of architecture, atypia [6]
Pseudosarcoma (Nodular Fasciitis)	True Sarcoma	Pseudosarcoma: Spindle cells, no atypia, USP6 rearrangement; True sarcoma: Pleomorphism, atypical mitoses, infiltrative growth [7,14]
Pseudoinclusions	True Nuclear Inclusions	Pseudoinclusions: Cytoplasmic invagination, common in melanoma; True inclusions: Accumulated material (e.g., viral, proteinaceous) within nucleus [17, 18]

CONCLUSION

Pseudo-histological lesions represent a distinct subset of benign conditions in oral pathology that closely simulate more serious diseases both clinically and microscopically. Their resemblance to carcinomas, sarcomas, lymphomas, or cystic lesions demands careful histopathological scrutiny, especially in small or inflamed biopsies. Misinterpretation can lead to overtreatment or delay in managing actual malignancies. Key features such as lack of atypia, organized architecture, or true epithelial/glandular elements must be evaluated in conjunction with the clinical context. Ancillary diagnostic modalities can assist in challenging cases, but recognition of classical pseudo-patterns remains fundamental. An informed approach to these mimics is vital to ensuring accurate diagnosis and appropriate patient care.

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