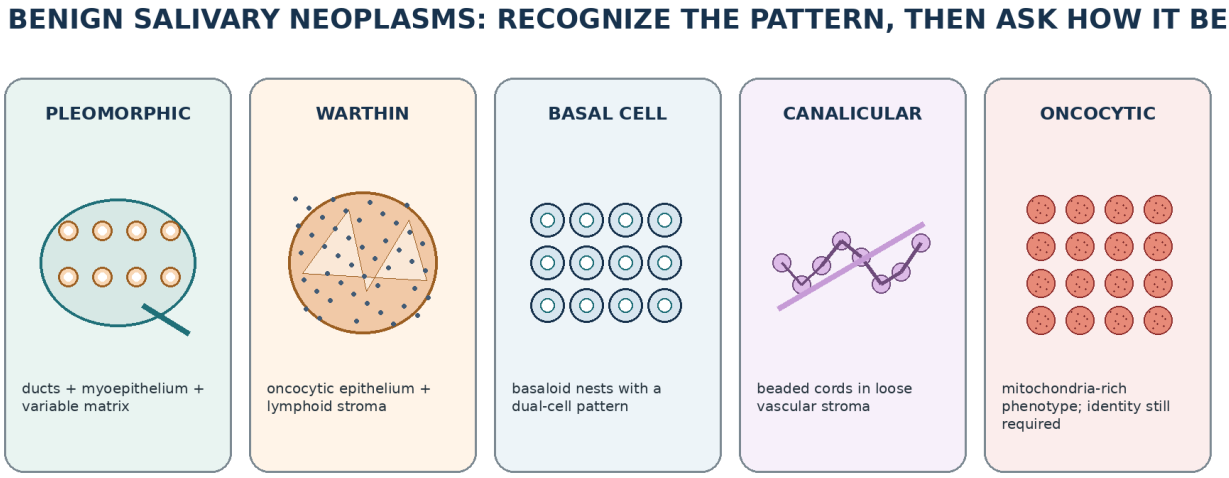
SALIVARY GLAND NEOPLASMS

VOLUME II

Benign and Borderline Neoplasms

Recognition, biologic behavior, operative implications, and the decision to observe or excise



The diagnosis is not the color of the cells. It is the architecture, the interface, and the clinical setting.

Figure 1. Original synthesis. Benign salivary tumors become easier to distinguish when low-power architecture, cell phenotype, and the tumor-normal interface are considered together.

THE CENTRAL QUESTION | Is this diagnosis reliable enough—and is its natural history favorable enough—that the patient can safely avoid or limit surgery?

A first-principles guide for otolaryngology residents • Prepared July 2026

CHAPTER 0

Orientation

Benign is a behavior category, not a promise of diagnostic certainty or easy surgery.

Salivary pathology is difficult because a small set of cellular programs—ductal, basal, myoepithelial, oncocytic, mucous, squamoid, and acinar—can be recombined into many tumors. A resident who starts with a name often memorizes a fragile list. A resident who starts with architecture can predict which names belong in the differential, which ancillary studies matter, and which clinical findings should make a "benign" diagnosis feel unsafe.

This volume follows the sequence used in real practice. It first builds a pattern language, then applies that language to the major benign entities, and finally returns to management: observation, limited parotid surgery, formal nerve dissection, complete gland excision, or escalation when the clinical story and pathology do not agree. Detailed operative technique is reserved for Volume IV, but the operation-changing implications are developed here.

SCOPE | Pleomorphic adenoma and Warthin tumor receive the most space because they dominate clinical practice. Basal cell adenoma, canalicular adenoma, myoepithelioma, oncocytoma, and the newer WHO entities are taught at the depth needed to recognize them, understand the malignant mimics, and choose a defensible plan.

Learning Objectives

- Recognize the major benign salivary neoplasms from their clinical setting and low-power architectural pattern.
- Explain why pleomorphic adenoma recurs after capsular violation and why recurrent disease becomes a qualitatively different surgical problem.
- Identify when a concordant Warthin tumor can reasonably be observed and when surgery is the safer choice.
- Distinguish basal cell adenoma from canalicular adenoma and understand why invasion—not cytologic blandness alone—separates basal cell adenoma from basal cell adenocarcinoma.
- Treat oncocytic change as a phenotype that requires a differential rather than as a final diagnosis.
- Match diagnostic confidence, anatomy, natural history, surgical morbidity, and patient preference to an observation or excision plan.
- Recognize board-relevant warning signs for carcinoma ex pleomorphic adenoma and other malignant mimics.

Contents

CHAPTER 1

A Pattern Language for Benign Salivary Tumors

Use low power to identify the grammar before high power supplies the vocabulary.

OPENING CASE | A 52-year-old patient has a 2.1-cm mobile superficial parotid mass. FNA is reported as "salivary gland neoplasm of uncertain malignant potential, basaloid." The useful question is not which single stain will name the lesion. It is whether the specimen shows a biphasic ductal/basal process, a myoepithelial-rich tumor with matrix, an adenoid cystic pattern, or insufficient architecture to decide.

At low power, most benign salivary neoplasms can be placed into a small number of families. The purpose is not to make the surgeon practice surgical pathology. It is to understand why FNA may be definitive for Warthin tumor, suggestive but imperfect for pleomorphic adenoma, and indeterminate for a basaloid or oncocytic neoplasm. Architecture is often the missing variable in cytology.

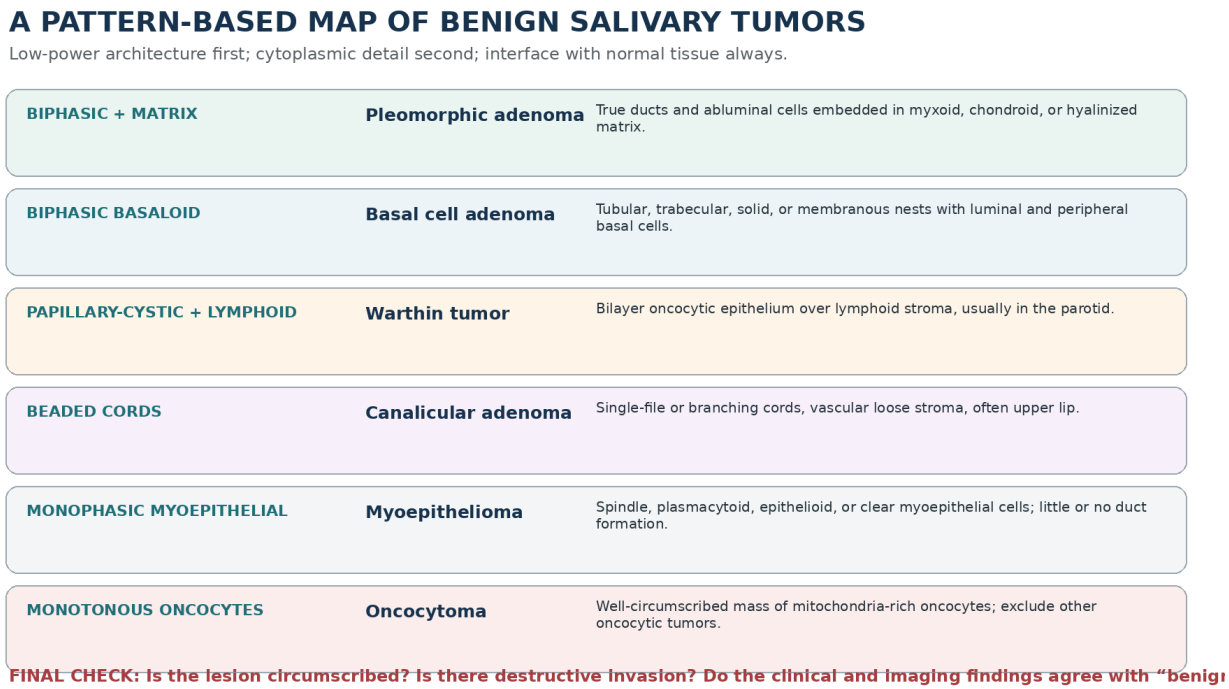


Figure 2. Original synthesis. A pattern map for the major benign epithelial neoplasms. The tumor-normal interface remains essential because several malignant tumors may be cytologically bland.

1.1 Begin with the interface

A circumscribed or encapsulated border supports benign behavior, but it does not end the analysis. Pleomorphic adenoma can have an incomplete capsule, pseudopodia, and satellite nodules without being malignant. Conversely, basal cell adenocarcinoma can resemble basal cell adenoma internally and declare itself only by destructive infiltration. "Bland" describes the cells; "benign" describes the entire lesion and its relationship to surrounding tissue.

FIRST PRINCIPLE | Cytologic blandness cannot substitute for demonstration of a noninvasive interface when the benign-versus-malignant distinction is defined by invasion.

1.2 Luminal and abluminal cells

Normal salivary units contain luminal epithelial cells and abluminal basal or myoepithelial cells. Many classic tumors preserve or exaggerate that dual-cell organization. Pleomorphic adenoma, basal cell adenoma, epithelial-myoepithelial carcinoma, and adenoid cystic carcinoma are therefore easier to understand as

different outputs of a shared biphasic program rather than as unrelated names. The stroma, growth pattern, and interface separate them.

Table 1. The first-pass pathology questions that change the differential.

Question	What to look for	Tumors moved up the differential
Are true ducts present?	Small lumina with an inner epithelial layer; sometimes an outer basal or myoepithelial layer.	Pleomorphic adenoma, basal cell adenoma, ductal adenomas.
Is there matrix?	Myxoid, chondroid, or hyalinized material between cells.	Pleomorphic adenoma; selected myoepithelial tumors.
Is there lymphoid stroma?	Polymorphous lymphocytes, often with germinal centers beneath cystic oncocytic epithelium.	Warthin tumor; lymphadenoma.
Are the cells oncocytic?	Abundant granular eosinophilic cytoplasm from mitochondrial accumulation.	Warthin tumor, oncocytoma, oncocytic hyperplasia—and many mimics.
Are cords beaded?	Branching single- or double-row cords in loose vascular stroma.	Canalicular adenoma.
Is invasion present?	Irregular tongues, entrapment, perineural or vascular invasion, destruction of surrounding tissue.	A carcinoma until proven otherwise.

1.3 Molecular findings are confirmatory maps, not the starting point

The fifth WHO framework increasingly incorporates recurrent molecular alterations, but morphology remains the entry point. Pleomorphic adenoma commonly has PLAG1- or HMGA2-related alterations. Basal cell adenoma often shows CTNNB1 pathway abnormalities, while the membranous pattern may be associated with CYLD alterations. Molecular testing is most valuable when morphology is limited, unusual, or overlaps a malignant entity; a fusion or mutation should be interpreted in the architectural context rather than used as a free-standing diagnosis.[1,2]

> **BOARDS BRIDGE** | The most testable molecular association in this volume is pleomorphic adenoma with PLAG1/HMGA2. For benign basaloid lesions, remember CTNNB1/β-catenin and the CYLD-associated membranous pattern.

1.4 The clinical triple test still governs

A pathology report must be reconciled with tempo, nerve function, imaging, and site. New facial weakness, fixation, pain along a nerve, destructive bone change, or infiltrative skull-base disease is not explained by a routine benign adenoma. When the clinical map and specimen disagree, the appropriate response is additional tissue, expert pathology review, better imaging, or an oncologic operation—not averaging the findings into reassurance.

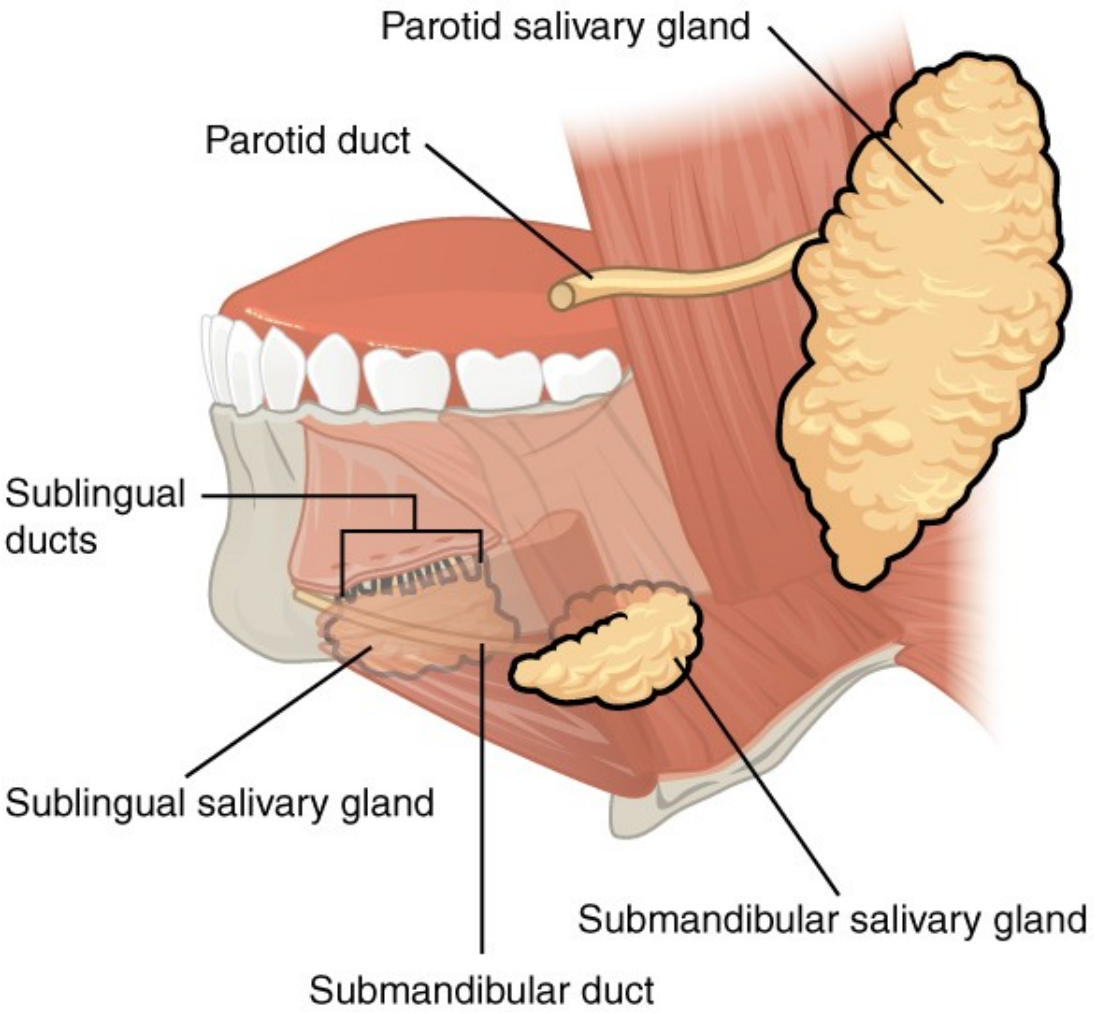


Figure 3. Major salivary glands and ducts. OpenStax College, CC BY 3.0. Site changes both the prior probability of malignancy and the structures placed at risk by surgery.

Chapter Synthesis

- Architecture, cell phenotype, stroma, and interface together define a coherent diagnosis.
- FNA is strongest when the entity has a distinctive cytologic mixture; it is weaker when invasion or complete architecture is required.
- A benign pathology label must explain the clinical behavior and imaging map.
- Molecular findings refine morphology; they do not replace it.

CHAPTER 2

Pleomorphic Adenoma

The commonest benign salivary neoplasm—and the prototype of why "benign" still demands oncologic discipline.

OPENING CASE | A 34-year-old patient has a painless 2.8-cm mobile parotid mass. MRI is sharply circumscribed and very T2 bright; FNA favors pleomorphic adenoma. The patient asks why the surgeon cannot simply shell it out. The answer lies at the microscopic edge, not at the center of the tumor.

2.1 The clinical archetype

Pleomorphic adenoma is usually a slow-growing, painless, firm, mobile mass. It is the most common parotid neoplasm and also occurs in the submandibular gland and minor salivary glands, particularly the palate. A long history supports indolent biology but does not make rapid recent growth irrelevant. Pain, fixation, ulceration, cervical adenopathy, or facial weakness should trigger concern for a malignant alternative or carcinoma ex pleomorphic adenoma.

The name "pleomorphic" refers to morphologic diversity, not nuclear pleomorphism. The lesion combines epithelial and myoepithelial elements with a variable myxoid, chondroid, or hyalinized matrix. One tumor may be predominantly cellular and another markedly myxoid; this variability explains why small samples can look different from the resection and why cytology may be reported as a generic benign neoplasm or SUMP when the classic mixture is underrepresented.

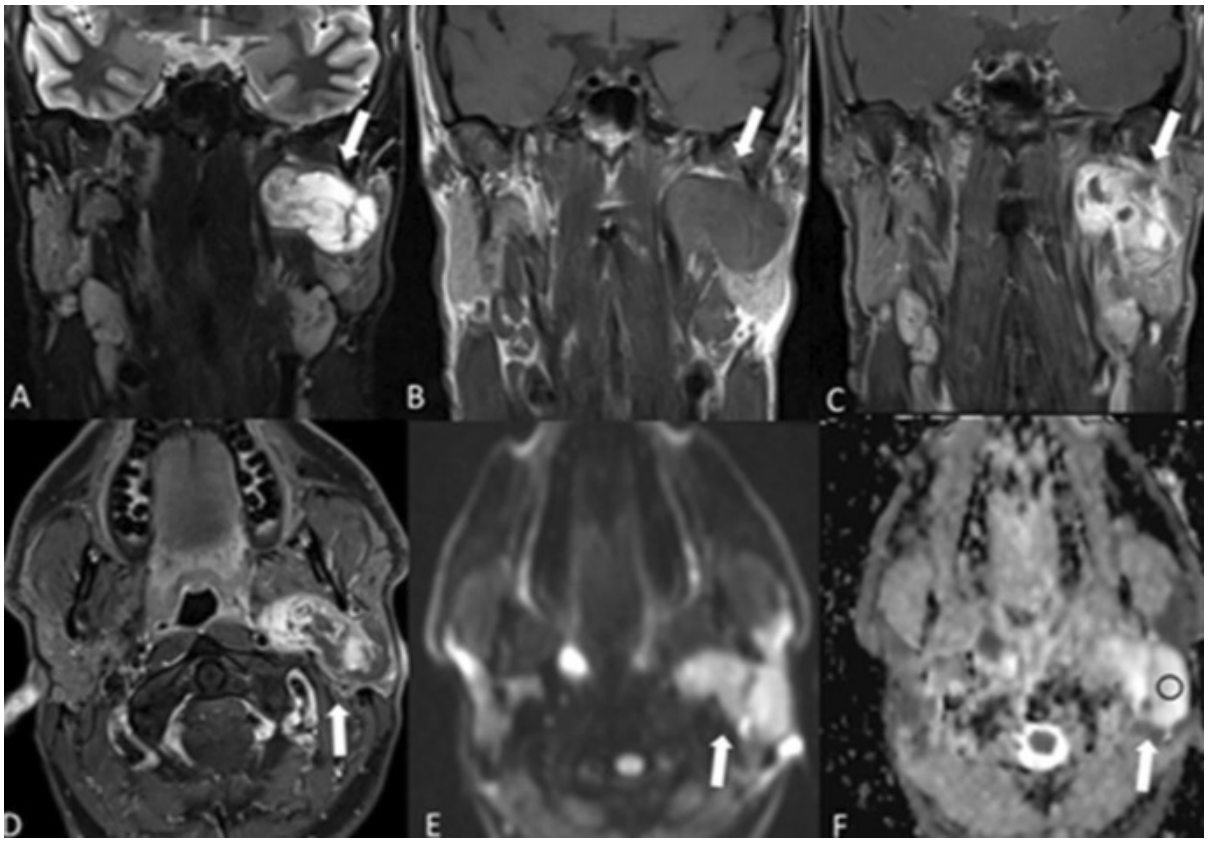


Figure 4. Pleomorphic adenoma spanning superficial and deep parotid compartments with an hourglass configuration. Very high T2 signal and facilitated diffusion are supportive but not pathognomonic. Kim et al., BMC Medical Imaging 2022; CC BY 4.0.

2.2 Pathology that predicts recurrence

The center of a pleomorphic adenoma is often reassuring. The edge is where management is determined. The capsule may be thin, incomplete, or absent in focal areas. Pseudopodia may protrude through or along the capsule, and satellite nodules may sit in adjacent salivary tissue or fat. These features do not represent carcinoma, but they make simple enucleation unreliable. Myxoid tumors tend to have thinner or less complete capsules, and larger tumors are more likely to harbor satellite nodules.[3]

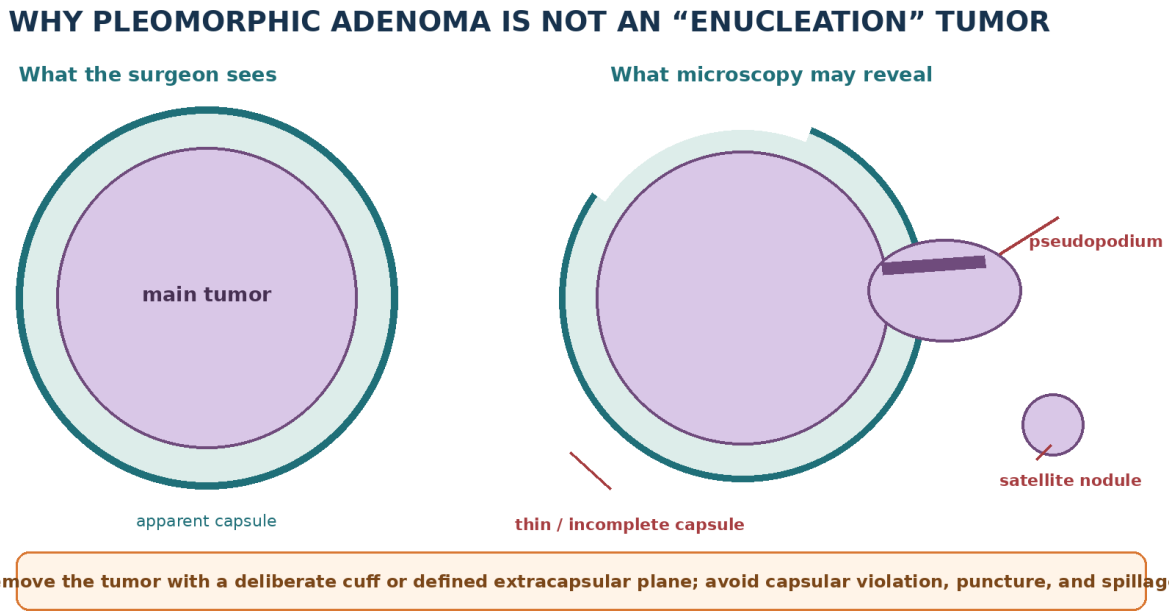


Figure 5. Original synthesis. Pleomorphic adenoma can have an apparently discrete gross border while microscopic extensions project beyond the visible shell.

OPERATING-ROOM RELEVANCE | The aim is not an arbitrary wide oncologic margin. It is complete removal in a controlled extracapsular or facial-nerve-dissection plane without tumor puncture, capsular rupture, or piecemeal extraction.

2.3 Cytology and molecular biology

Classic FNA shows epithelial or myoepithelial cells in cohesive groups and singly dispersed cells within fibrillary myxochondroid matrix. The relative proportions vary. A matrix-rich aspirate may be paucicellular; a cellular lesion may resemble basal cell adenoma, adenoid cystic carcinoma, or another myoepithelial neoplasm. The clinical consequence is that a “favor pleomorphic adenoma” result should be treated as strong but not absolute evidence, especially when imaging or symptoms are discordant.

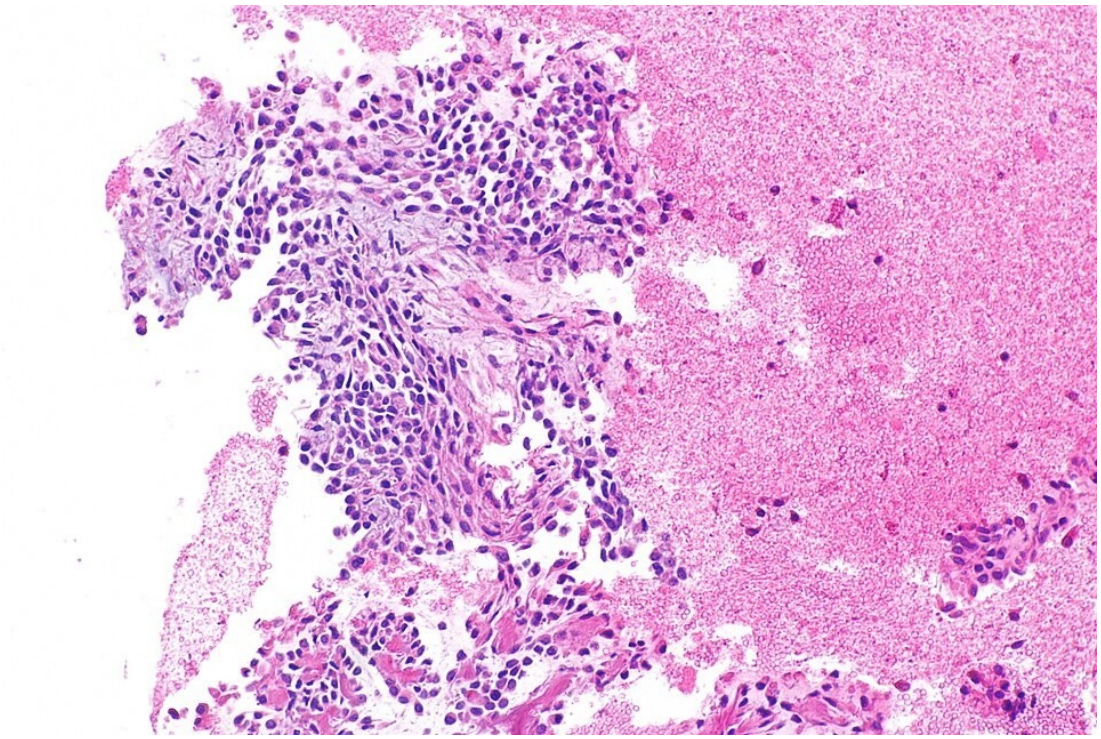


Figure 6. Cell-block appearance of pleomorphic adenoma with epithelial/myoepithelial cells and stromal material. Librepath, "Pleomorphic adenoma - cell block - intermediate magnification," CC BY-SA 3.0.

PLAG1 rearrangement or overexpression is the dominant molecular pathway; HMGA2 alterations account for another subset. These findings can support diagnosis in unusual morphology and help establish that a carcinoma arose from pleomorphic adenoma, but routine straightforward cases do not require molecular testing.[1]

2.4 Selecting the operation

The extent of surgery is a relationship problem: tumor size and location, expected facial-nerve relationship, mobility, diagnostic confidence, and surgeon experience. Historical enucleation is unacceptable because it follows the capsule itself and leaves microscopic extensions behind. Modern options include extracapsular dissection, partial superficial parotidectomy, superficial parotidectomy, and total parotidectomy for selected deep-lobe or extensive lesions. The terminology varies across institutions, so the operative principle matters more than the label.

Table 2. Operation families for primary parotid pleomorphic adenoma. Selection—not a universal winner—drives outcomes.

Approach	Best-fit situation	Strength	Main concern
Extracapsular dissection	Small, mobile, superficial, confidently benign tumor separated from major nerve branches; experienced surgeon.	Less normal gland dissection; lower transient weakness and Frey risk in selected series.	Selection bias; narrow working margin; conversion needed if the nerve or tumor interface is unfavorable.
Partial superficial parotidectomy	Localized superficial lesion with planned identification of relevant nerve branches.	Controlled nerve anatomy with less gland removal than formal superficial parotidectomy.	Definitions vary; still requires disciplined capsular handling.
Superficial parotidectomy	Larger or less mobile superficial tumor, uncertain relationship to branches, institutional standard.	Broad exposure and established nerve dissection.	More normal-gland dissection, transient weakness, contour change, Frey syndrome.
Total parotidectomy with nerve preservation	Deep-lobe, multifocal/recurrent, or extensive lesion when complete gland access is required.	Access to deep compartment and multiple nodules.	Higher facial-nerve manipulation and morbidity; not routine for a small superficial primary PA.

Recent meta-analyses suggest similar recurrence rates for carefully selected small pleomorphic adenomas treated with extracapsular dissection versus superficial parotidectomy, with less transient facial weakness and Frey syndrome after the more limited approach. These data are mostly observational and depend heavily on selection, definitions, follow-up duration, and surgeon expertise. They support individualized conservative surgery—not indiscriminate shelling out of every mass.[4,5]

2.5 Pleomorphic adenoma outside the parotid

Submandibular pleomorphic adenoma is usually treated by complete gland excision because the tumor is embedded within a compact gland and limited tumor-only excision offers little advantage. Palatal and other minor-gland tumors require complete excision with an intact specimen and an appropriate soft-tissue margin; the apparent absence of a capsule in minor-gland lesions makes a simple mucosal “peel” inadequate. Bone remodeling can occur from pressure; frank destructive invasion should prompt reconsideration of the diagnosis.

2.6 What to tell the patient

- The tumor is benign, but it does not reliably separate from the surrounding gland at a microscopic level.
- Definitive treatment is usually surgery because growth makes later surgery harder and long-standing tumors carry a small risk of malignant transformation.
- Facial-nerve risk depends on tumor location and the amount of nerve dissection required, not simply on the word “benign.”
- A ruptured capsule does not guarantee recurrence, but it increases concern and lengthens the surveillance horizon.
- Recurrence may occur many years later; the patient should retain the pathology and operative history.

BOARD TRAP | Pleomorphic adenoma is not “mixed” because it contains mesenchymal tissue. The chondromyxoid-appearing matrix is produced by neoplastic epithelial/myoepithelial cells.

Chapter Synthesis

- Pleomorphic adenoma is defined by epithelial/myoepithelial differentiation plus variable matrix.
- Thin or incomplete capsule, pseudopodia, satellite nodules, positive margins, and spillage explain recurrence risk.
- Enucleation is not an acceptable operation; conservative parotid surgery is appropriate only when it preserves a deliberate tumor-free plane.
- Rapid change in a long-standing mass requires evaluation for carcinoma ex pleomorphic adenoma.

CHAPTER 3

Warthin Tumor

A benign tumor in which diagnostic confidence can legitimately change the indication for surgery.

OPENING CASE | A 71-year-old smoker has a 1.8-cm inferior-pole parotid lesion found on PET/CT during lung-cancer surveillance. It is intensely FDG avid, partly cystic, and diffusion restricting. FNA shows oncocytes, lymphocytes, and granular debris. The imaging looks alarming only if Warthin tumor is not recognized as an exception.

3.1 A distinctive clinicopathologic package

Warthin tumor is almost restricted to the parotid and periparotid lymphoid tissue, often in the tail. It typically affects older adults and has a strong association with tobacco exposure. Bilateral and multifocal tumors are much more common than in other salivary neoplasms. A new lesion after prior surgery often represents another focus rather than biologic recurrence.

Histologically, papillary and cystic spaces are lined by a bilayer of oncocytic epithelium over a lymphoid stroma that may contain germinal centers. The classic FNA triad is cohesive oncocytes, numerous small lymphocytes, and a dirty granular or proteinaceous background. When all three are present in the correct clinical setting, cytology is highly accurate. A meta-analysis of 17 studies and 1,710 cases estimated sensitivity near 94% and specificity near 98%.[7]

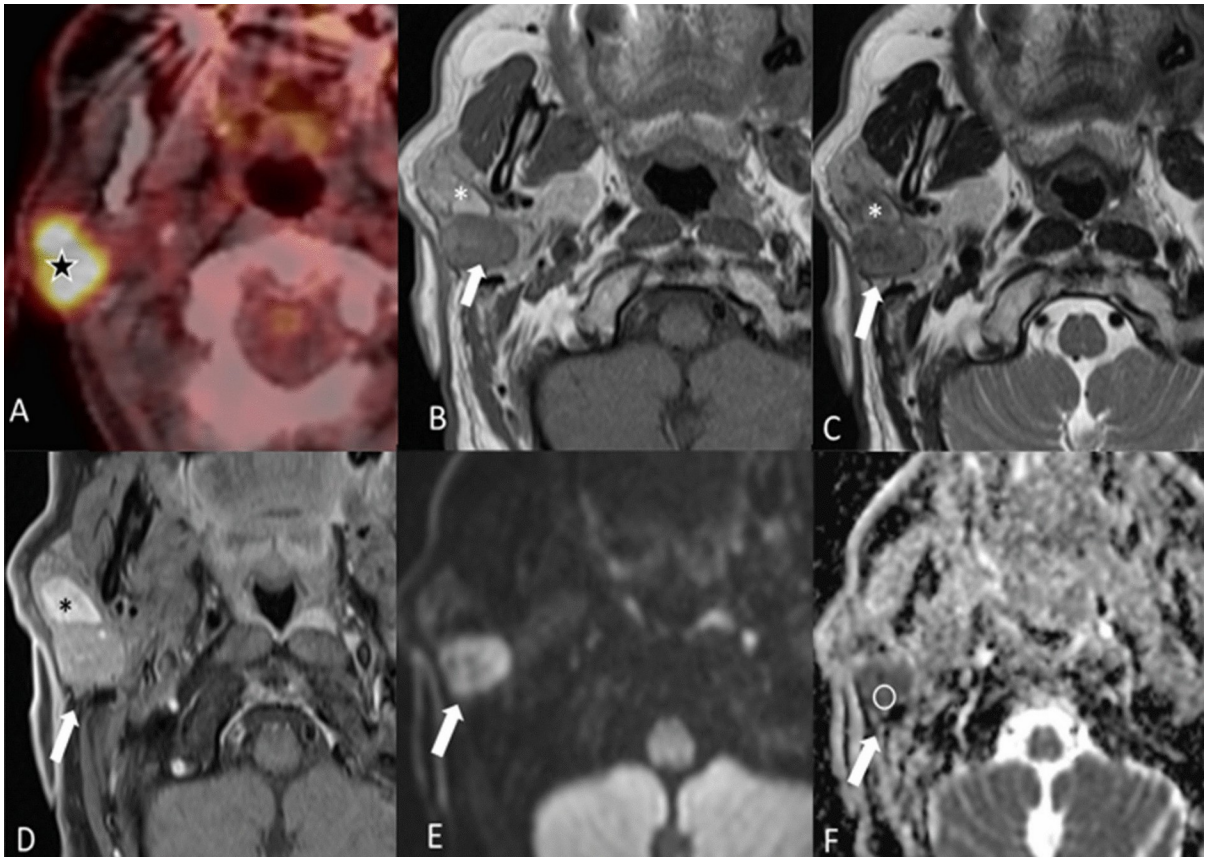


Figure 7. Warthin tumor may be intensely FDG avid and may restrict diffusion. PET avidity and low ADC are not synonymous with malignancy. Kim et al., BMC Medical Imaging 2022; CC BY 4.0.

3.2 Why the FNA can become misleading

The cystic component may yield mostly debris or macrophages. A lymphocyte-poor aspirate can resemble oncocytoma; an oncocyte-poor aspirate can resemble a lymph node. Infarction, inflammation, or squamous

metaplasia—sometimes after FNA—can create atypical squamoid cells and necrotic debris that mimic mucoepidermoid carcinoma or squamous carcinoma. Discordant pain, fixation, skin change, facial weakness, or a non-parotid site should therefore not be explained away by a partial Warthin pattern.

FIRST PRINCIPLE | Observation is safe only when the diagnosis is safe. "Possible Warthin tumor" is not the same management category as a concordant clinical-imaging-cytology diagnosis.

3.3 Active surveillance versus surgery

Unlike pleomorphic adenoma, a reliably diagnosed asymptomatic Warthin tumor does not require removal to prevent a meaningful malignant-transformation risk. Many patients are older or medically complex, and parotid surgery carries real facial-nerve, sensory, contour, salivary, and Frey morbidity. Active surveillance has therefore become a standard option when the triple test is concordant and the patient accepts follow-up.[6]

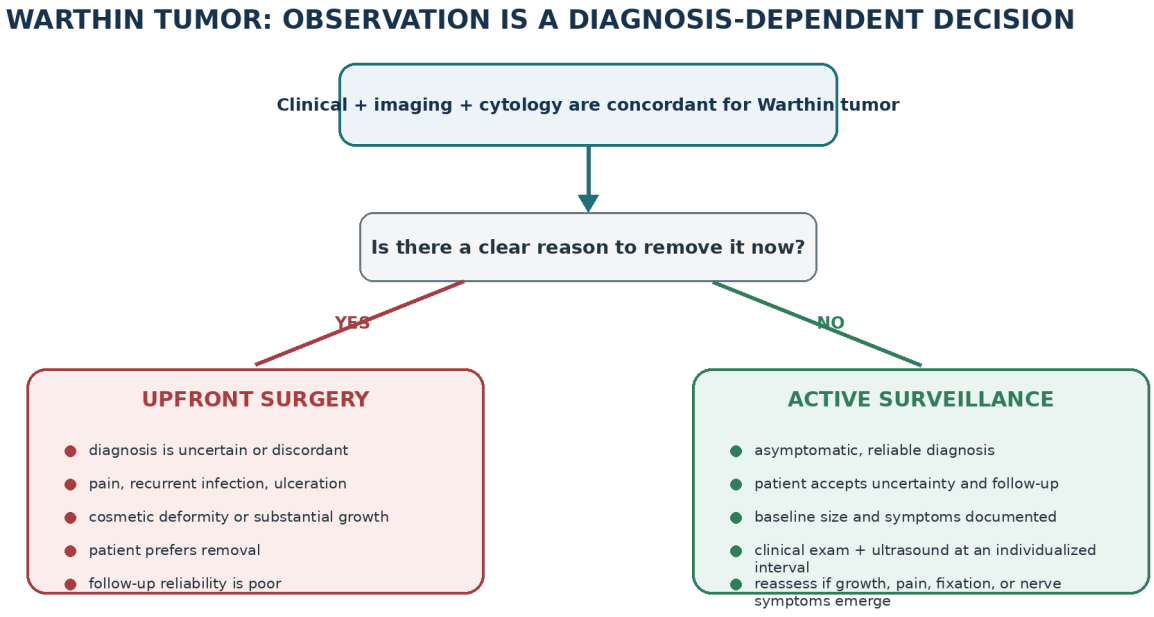


Figure 8. Original synthesis. The decision to observe a Warthin tumor is contingent on diagnostic concordance, symptoms, growth, patient preference, and reliable follow-up.

Table 3. A practical Warthin tumor decision balance.

Favor observation	Favor surgery
Classic parotid-tail location with concordant imaging and adequate FNA; asymptomatic; limited cosmetic effect.	Uncertain or discordant diagnosis; solid atypical component; progressive pain, ulceration, recurrent infection, fixation, nerve symptoms.
Older or comorbid patient for whom parotidectomy morbidity is disproportionate to benefit.	Substantial growth, large lesion, deformity, or compression symptoms.
Bilateral or multifocal disease where repeated surgery would remove excessive gland and manipulate both facial nerves.	Patient preference for definitive removal after informed discussion.
Patient can return for examination and ultrasound when indicated.	Follow-up is unreliable or the patient cannot tolerate residual uncertainty.

3.4 Surveillance is active, not passive

Document the baseline examination, facial function, symptoms, imaging dimensions, and diagnostic evidence. A common practical strategy is clinical examination and ultrasound at approximately six months to establish

behavior, followed by individualized intervals if stable; there is no universal evidence-based schedule. Re-biopsy or surgery is warranted when the lesion develops a solid discordant component, accelerates, becomes painful or fixed, or produces nerve dysfunction.

3.5 Surgical principles

When surgery is chosen, the extent should match tumor position and multiplicity. An inferior superficial lesion may be amenable to extracapsular dissection or partial superficial parotidectomy in experienced hands. Multiple lesions may require a broader gland-preserving plan rather than chasing each focus through scar. Total parotidectomy is not a reflex response to bilaterality or multifocality; the competing objective is preservation of facial function and salivary tissue.

> **BOARD TRAP** | Warthin tumor is a classic cause of a false-positive oncologic PET impression. It can be intensely FDG avid despite benign behavior.

Chapter Synthesis

- Warthin tumor is a parotid-centered, oncocytic, papillary-cystic tumor with lymphoid stroma.
- Concordant FNA is highly accurate, but infarction, metaplasia, and incomplete sampling create important traps.
- Active surveillance is reasonable for a reliable, asymptomatic diagnosis; diagnostic uncertainty is itself an indication for surgery.
- Bilateral or metachronous lesions usually represent multifocal tumor biology rather than aggressive recurrence.

CHAPTER 4

Basaloid Adenomas

Basal cell adenoma and canalicular adenoma share historical terminology but differ in site, architecture, and malignant differential.

OPENING CASE | A 68-year-old woman has a 1.5-cm painless upper-lip nodule. A small biopsy shows cords of bland basaloid cells in a loose vascular stroma. Calling it "basal cell adenoma" because the cells are basaloid would miss the strongest clinical clue: canalicular adenoma has a striking predilection for the upper lip.

4.1 Basal cell adenoma

Basal cell adenoma usually arises in the parotid of an older adult. It is a well-circumscribed or encapsulated biphasic tumor composed of luminal ductal cells and peripheral basal cells. Tubular, trabecular, solid, and membranous patterns may coexist. Peripheral palisading and a jigsaw-puzzle arrangement can be prominent. Unlike pleomorphic adenoma, a chondromyxoid stromal component is absent or minimal.

The major diagnostic problems are pleomorphic adenoma, adenoid cystic carcinoma, and basal cell adenocarcinoma. β-catenin nuclear staining or CTNNB1 alteration supports many basal cell adenomas. The membranous subtype is associated with CYLD pathway alterations and may be multifocal, particularly in the setting of CYLD cutaneous syndrome. Cribriform architecture alone does not make adenoid cystic carcinoma; the overall interface, stroma, cytology, and perineural behavior matter.[1,8]

Table 4. The distinction between basal cell adenoma and adenocarcinoma is fundamentally architectural.

Feature	Basal cell adenoma	Basal cell adenocarcinoma
Interface	Circumscribed or encapsulated.	Infiltrative growth into gland, fat, muscle, or adjacent tissue.
Cells	Often bland basaloid cells; dual-cell organization.	May remain deceptively bland; atypia is variable.
Mitoses / Ki-67	Usually low.	May be increased, but proliferation does not replace evidence of invasion.
Perineural / vascular invasion	Absent.	Supports carcinoma when present.
Practical implication	Conservative complete excision appropriate.	Oncologic resection and malignant salivary pathway.

BOARDS BRIDGE | The benign-versus-malignant distinction in a basaloid salivary tumor may depend on invasion. A bland FNA can therefore correctly identify a basaloid neoplasm yet remain unable to exclude carcinoma.

4.2 Canalicular adenoma

Canalicular adenoma is predominantly a minor-salivary-gland tumor of older adults, especially women, and the upper lip is the signature site. The lesion is typically a small, slowly growing, painless submucosal nodule. Histology shows branching and anastomosing cords of columnar or cuboidal cells with a beaded appearance in loose, vascular, often myxoid stroma. The tumor is luminal rather than truly biphasic and lacks the continuous peripheral myoepithelial layer expected in basal cell adenoma.

Multifocality is a recognized feature. In a 67-case clinicopathologic series, most lesions involved the upper lip or buccal mucosa, the mean age was about 70 years, and approximately 13% were multifocal.[9] Complete local excision is usually curative. A later nearby nodule may represent an additional focus rather than a consequence of inadequate wide margins.

Table 5. Basal cell adenoma and canalicular adenoma are not interchangeable "monomorphic adenomas."

Feature	Basal cell adenoma	Canalicular adenoma
Typical site	Parotid.	Upper lip; then buccal mucosa and palate.
Architecture	Solid, tubular, trabecular, membranous; peripheral palisading.	Beaded single/double cords and canal-like spaces in loose vascular stroma.
Cell layers	Biphasic luminal and basal/abluminal pattern.	Predominantly luminal; no continuous myoepithelial rim.
Multifocal clue	Membranous/CYLD-associated cases.	Recognized in the oral cavity, especially upper lip.
Main malignant mimic	Basal cell adenocarcinoma; adenoid cystic carcinoma.	Polymorphous adenocarcinoma and other minor-gland tumors.

4.3 Management

A parotid basal cell adenoma is managed like another confidently benign, localized parotid neoplasm: complete removal with a controlled tumor-free plane and an operation matched to its relationship with the facial nerve. A canalicular adenoma is usually treated by local oral excision. Positive or indeterminate margins in a fragmented minor-gland specimen should prompt pathology review and correlation with the surgical bed rather than automatic radical resection; true infiltrative growth should instead trigger reconsideration of the diagnosis.

Chapter Synthesis

- Basal cell adenoma is usually parotid and biphasic; canalicular adenoma is usually upper lip and luminal with beaded cords.
- Basal cell adenocarcinoma may be cytologically bland; invasion is the decisive feature.
- Membranous basal cell adenoma raises the possibility of CYLD pathway disease and multifocality.
- Canalicular adenoma may be multifocal, but simple complete local excision is generally adequate.

CHAPTER 5

Myoepithelioma

A monophasic tumor that reveals how versatile—and diagnostically treacherous—myoepithelial cells can be.

OPENING CASE | A 45-year-old patient has a well-circumscribed parotid mass. Core biopsy shows plasmacytoid cells positive for S100 and p63, with no convincing duct formation. The diagnosis cannot be made from a single myoepithelial marker; the lesion must be shown to be a coherent, noninvasive myoepithelial neoplasm.

5.1 What makes a myoepithelioma?

Myoepithelioma is a benign neoplasm composed almost entirely of myoepithelial cells. It overlaps conceptually with pleomorphic adenoma, but significant duct formation and abundant chondromyxoid matrix are absent. The practical threshold is not perfectly absolute: limited ducts may be present, and terminology can vary at the boundary. The diagnosis is most secure when morphology, immunophenotype, and a circumscribed interface agree.

The cells may be spindle, plasmacytoid, epithelioid, clear, or mixed. Plasmacytoid myoepithelioma is especially associated with minor salivary sites, including the palate. Immunohistochemistry is a panel problem rather than a single-stain problem: cytokeratins establish epithelial differentiation, while S100, SOX10, p63/p40, calponin, smooth-muscle actin, and GFAP may be variably expressed. No marker is perfectly sensitive or specific.

5.2 The malignant boundary

Myoepithelial carcinoma can be deceptively bland. The essential question is whether the lesion infiltrates surrounding gland, fat, muscle, bone, or nerves. Marked atypia, necrosis, mitotic activity, and high proliferation increase concern, but benign-versus-malignant classification ultimately depends on the interface. A small biopsy may therefore identify a myoepithelial neoplasm without safely proving benign behavior.

Table 6. The interface is again the decisive variable.

Finding	Supports benign myoepithelioma	Raises concern for carcinoma
Border	Circumscribed, encapsulated, pushing.	Irregular destructive infiltration or entrapment of normal structures.
Cytology	Uniform; low mitotic activity; no necrosis.	Atypia, brisk mitoses, necrosis—although these may be absent.
Clinical behavior	Slow, painless, mobile.	Rapid growth, fixation, pain, nerve dysfunction.
Sampling result	Coherent morphology on adequate core/resection.	Limited FNA/core unable to evaluate interface; discordant imaging.

RESIDENT TAKEAWAY | A report of “myoepithelial neoplasm” should trigger a question: has benign behavior actually been demonstrated, or has only lineage been established?

5.3 Treatment

Complete surgical excision is standard. The operation depends on site: a localized superficial parotid lesion may receive a conservative parotid operation; submandibular disease generally requires gland excision; minor-gland disease requires local excision with an intact specimen. Recurrence is uncommon after complete removal but is more consequential when the initial specimen was fragmented or the interface was not adequately evaluated.

BOARD TRAP | Myoepithelioma is not diagnosed by S100 positivity alone. S100 and SOX10 are shared by several salivary tumors and non-salivary lesions; morphology and a panel are required.

Chapter Synthesis

- Myoepithelioma is predominantly monophasic and lacks the robust duct-plus-matrix pattern of pleomorphic adenoma.
- Myoepithelial cells have multiple morphologies and variable immunophenotypes.
- A limited specimen may establish myoepithelial lineage without establishing benign behavior.
- Complete intact excision permits both treatment and definitive interface assessment.

CHAPTER 6

Oncocytic Lesions

Oncocytic cytoplasm is a metabolic phenotype shared by hyperplasia, benign tumors, carcinomas, and metastases.

OPENING CASE | A 63-year-old patient has a 2.4-cm solid parotid mass. FNA shows sheets of bland oncocytes but few lymphocytes. The correct next thought is not “oncocytoma.” It is “oncocytic lesion”: Warthin tumor, oncocytoma, nodular oncocytic hyperplasia, oncocytic change in another salivary tumor, or metastatic disease remain possible.

Oncocytes are epithelial cells packed with mitochondria, producing abundant granular eosinophilic cytoplasm. Aging and chronic cellular stress can produce oncocytic metaplasia in normal ducts, and many unrelated salivary tumors can become predominantly oncocytic. The fifth WHO framework no longer treats “oncocytic carcinoma” as a simple catch-all independent diagnosis because many historical cases can be assigned to a specific carcinoma with oncocytic change.[1,10]

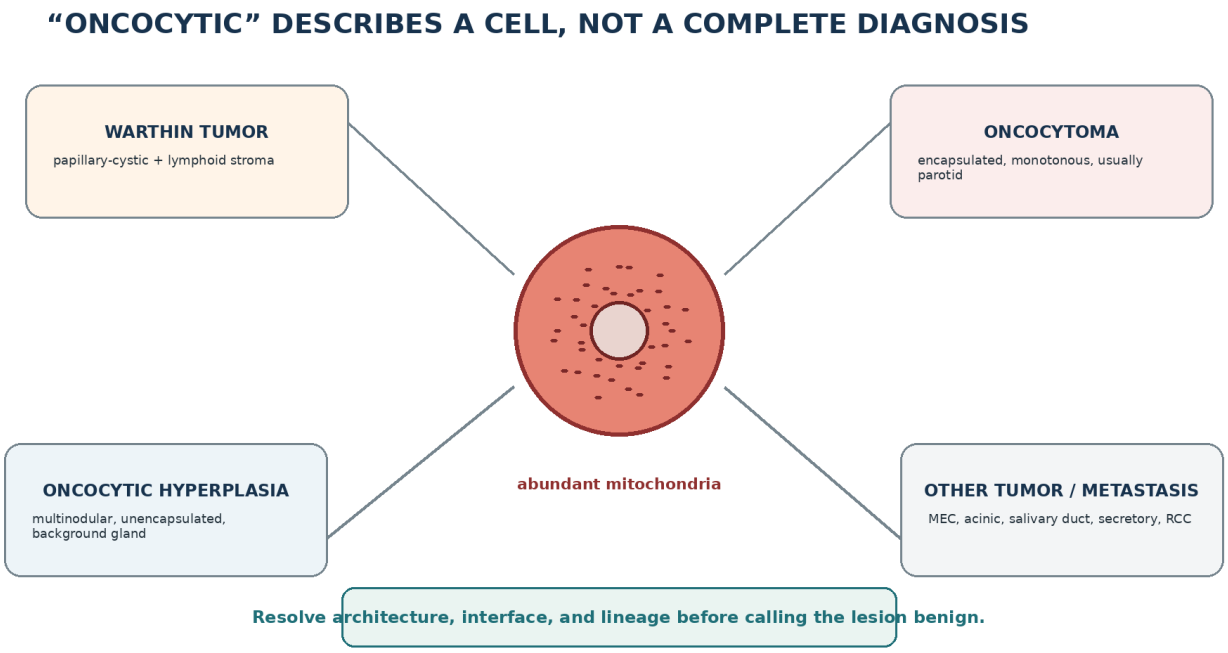


Figure 9. Original synthesis. Oncocytic morphology must be resolved into a process by integrating architecture, background, interface, lineage, and clinical context.

6.1 Nodular oncocytic hyperplasia versus oncocytoma

Nodular oncocytic hyperplasia is a non-neoplastic or hyperplastic process composed of multiple unencapsulated oncocytic nodules intermingled with salivary tissue. Oncocytoma is a solitary, well-circumscribed or encapsulated neoplasm of monotonous oncocytes arranged in nests, sheets, trabeculae, or small tubules. Cytology may not reliably distinguish them because both yield bland oncocytes; unifocal encapsulation and the relationship to background gland are histologic and imaging clues.

Oncocytoma is rare and most often arises in the parotid. Complete excision is generally curative. Bilateral or multifocal oncocytomas should prompt consideration of Birt-Hogg-Dubé syndrome in the appropriate personal or family context, although this association is uncommon and the evidence base is limited.[11]

6.2 The differential of an oncocytic aspirate

Table 7. "Oncocytic lesion" is a differential diagnosis, not diagnostic failure.

Entity	Clue that moves it up	Clue against a simple oncocytoma
Warthin tumor	Parotid tail; cystic lesion; lymphocytes and granular debris; tobacco exposure.	Oncocytes without the lymphoid/cystic context may be incomplete sampling.
Oncocytoma	Solitary circumscribed solid mass; monotonous oncocytes; no lymphoid background.	FNA cannot prove encapsulation or exclude all oncocytic carcinomas.
Nodular oncocytic hyperplasia	Multinodular gland; background oncocytic change.	A single encapsulated mass favors oncocytoma.
Acinic cell carcinoma	SOX10/DOG1-positive acinar differentiation; cytoplasmic granules/vacuoles; infiltrative lesion.	Can look bland and oncocytic on FNA.
Mucoepidermoid carcinoma	Mucous or intermediate cells, mucin, squamoid population; MAML2 in many cases.	Metaplastic Warthin tumor may mimic it.
Salivary duct carcinoma	High-grade apocrine features, AR/GATA3/HER2 context.	Usually overtly atypical, but oncocytic variants exist.
Metastatic renal cell carcinoma	Clear/oncocytic cells, rich vasculature, renal history; PAX8/CAIX profile.	Parotid is a possible metastatic site; history matters.

6.3 Management consequence

A small, superficial, confidently benign oncocytoma can be treated with a gland-preserving parotid operation. A generic oncocytic FNA in a solid enlarging mass should not automatically lead to observation. Core biopsy, expert review, cross-sectional imaging, or excision may be necessary because the clinically important distinction often depends on architecture and invasion. The goal is to avoid both an unnecessary radical operation and an inadequately planned first operation for a malignant oncocytic mimic.

BOARD TRAP | Warthin tumor and oncocytoma are both oncocytic. Lymphoid stroma and papillary-cystic architecture make Warthin tumor; encapsulated monotonous oncocytes without lymphoid stroma make oncocytoma.

Chapter Synthesis

- Oncocytes are defined by mitochondrial abundance, not by a single tumor identity.
- Oncocytic hyperplasia is multinodular and unencapsulated; oncocytoma is usually a solitary encapsulated neoplasm.
- Warthin tumor, several salivary carcinomas, and metastatic renal cell carcinoma belong in the oncocytic differential.
- A limited oncocytic sample may justify a category diagnosis and additional architecture rather than false specificity.

CHAPTER 7

Other Benign Epithelial Neoplasms

Know the defining pattern, the favored site, and the malignant mimic—without giving every rare entity equal cognitive weight.

The fifth WHO classification recognizes a broader benign epithelial category than older resident texts. Most of these tumors are rare. The correct educational strategy is tiered: master pleomorphic adenoma and Warthin tumor; recognize basal cell adenoma, canalicular adenoma, myoepithelioma, and oncocytoma; and retain a compact profile of the remaining entities so that an unfamiliar pathology report is interpretable.[1,2]

Cystadenoma

Typical site / patient	Defining pattern	Clinical management	Board-level trap
Parotid or minor salivary glands; Adults	Unicystic or multicystic epithelial lesion, sometimes papillary; lacks Warthin-type lymphoid stroma.	Complete excision.	Mucous or oncocytic lining may mimic low-grade mucoepidermoid carcinoma or other cystic tumors.

Ductal papillomas

Typical site / patient	Defining pattern	Clinical management	Board-level trap
Minor glands are common; Adults	Intraductal papilloma is confined to a dilated duct; inverted ductal papilloma grows endophytically; terminology depends on architecture.	Local complete excision.	Do not confuse with sialadenoma papilliferum or papillary carcinoma based on a small surface fragment.

Sialadenoma papilliferum

Typical site / patient	Defining pattern	Clinical management	Board-level trap
Palate and other oral minor-gland sites; Middle-aged/older adults	Biphasic lesion with an exophytic squamous papilloma connected to an underlying salivary duct proliferation.	Local excision; ensure full lesion is sampled.	The surface component may dominate a superficial biopsy, obscuring the glandular lesion.

Intercalated duct adenoma / hyperplasia

Typical site / patient	Defining pattern	Clinical management	Board-level trap
Parotid predominance; Adults	Small bland intercalated-type ducts; may occur near other salivary neoplasms.	Complete excision when mass-forming; pathology correlation.	Can mimic basal cell adenoma, epithelial-myoepithelial carcinoma, or a precursor-like change.

Striated duct adenoma

Typical site / patient	Defining pattern	Clinical management	Board-level trap
Usually parotid; Adults	Well-circumscribed lesion of striated-duct-type columnar cells with eosinophilic cytoplasm and characteristic basal striations.	Complete excision.	New WHO entity; distinguish from oncocytic and apocrine lesions by architecture and phenotype.

Sclerosing polycystic adenoma

Typical site / patient	Defining pattern	Clinical management	Board-level trap
Parotid most often; can involve other glands; Broad age range	Dense sclerosis with cystically dilated ducts, acinar/ductal proliferation, apocrine change, and sometimes intraductal atypia.	Complete excision and thoughtful follow-up.	Formerly called “sclerosing polycystic adenosis”; PI3K-pathway clonality supports neoplastic classification, and rare carcinoma can arise within it.[12]

Keratocystoma

Typical site / patient	Defining pattern	Clinical management	Board-level trap
Very rare, major glands; Adults	Multicystic squamous-lined lesion with keratin production.	Complete excision; expert pathology review.	Exclude cystic squamous carcinoma or metastasis; rarity makes overdiagnosis a risk.

Lymphadenoma

Typical site / patient	Defining pattern	Clinical management	Board-level trap
Parotid; Adults	Epithelial nests or cysts with prominent lymphoid stroma; sebaceous and nonsebaceous forms.	Complete excision.	Distinguish from Warthin tumor, lymphoepithelial lesions, and metastatic disease in intraparotid nodes.

Sebaceous adenoma

Typical site / patient	Defining pattern	Clinical management	Board-level trap
Parotid or minor glands; Adults	Circumscribed sebaceous differentiation without the lymphoid architecture of sebaceous lymphadenoma.	Complete excision.	Confirm true sebaceous differentiation and exclude cutaneous or metastatic origin.

BOARDS STRATEGY | For a rare benign entity, retain four anchors: favored site, defining architecture, malignant mimic, and usual treatment. Molecular minutiae are secondary unless they define the entity.

Chapter Synthesis

- The WHO benign category has expanded, but rare entities should be learned by defining pattern rather than equal-length lists.
- Sclerosing polycystic adenoma is now regarded as neoplastic and can contain intraductal atypia.
- Papillary, cystic, sebaceous, and squamous features often create overlap with low-grade carcinoma or metastasis.
- Complete intact excision and expert pathology review solve most rare benign-neoplasm problems.

CHAPTER 8

Benign Mesenchymal Lesions and Non-neoplastic Mimics

A salivary-region mass may arise beside, within, or through a gland without being a salivary epithelial tumor.

8.1 Why localization still matters

The parotid contains lymph nodes, nerves, vessels, fat, and ductal structures. The submandibular triangle contains level Ib nodes, the gland, facial vessels, the marginal mandibular nerve, and the floor-of-mouth compartment. A “parotid mass” may therefore be nodal metastasis, lymphoma, schwannoma, vascular malformation, lipoma, inflammatory disease, or a first branchial cleft anomaly. The differential should be driven by compartment and behavior before the pathology vocabulary begins.

8.2 Sialolipoma and lipomatous lesions

Sialolipoma is a salivary-specific lipomatous tumor containing mature adipose tissue and entrapped or proliferative salivary elements. Imaging may show a fat-dominant lesion, but the glandular component can make the mass heterogeneous. Complete excision is usually curative. The important differential includes ordinary lipoma, infiltrating lipomatosis, lipoadenoma, and fat-containing malignant or syndromic processes.

8.3 Neural and vascular lesions

Facial-nerve schwannoma may mimic a deep parotid tumor. Imaging clues include a mass centered along the expected nerve course or stylomastoid foramen, but many intraparotid nerve tumors remain difficult to diagnose preoperatively. A normal preoperative facial examination does not exclude schwannoma. Entering surgery without counseling about a nerve-origin lesion can convert a diagnostic surprise into an avoidable consent problem.

Vascular malformations or hemangiomas may be compressible, bluish, pulsatile, or flow-dependent and may enlarge with Valsalva or dependency. Doppler ultrasound and MRI define flow and extent. Blind FNA or an unplanned open biopsy is inappropriate when a vascular lesion is plausible.

8.4 Inflammatory and cystic mimics

Table 8. Common non-neoplastic or non-salivary mimics.

Mimic	Clues	Why it matters
Chronic sialadenitis / obstructive disease	Meal-related pain, stones or duct dilation, heterogeneous atrophic gland.	A firm gland can mimic neoplasm; treat the obstructive/inflammatory process rather than performing tumor surgery.
Abscess or infected cyst	Pain, erythema, fever, rim enhancement, inflammatory aspirate.	Persistent solid component after treatment requires reassessment.
First branchial cleft anomaly	Recurrent peri-auricular/parotid infection, tract toward external auditory canal, childhood or young adult history.	Facial-nerve relationship can be complex; incision and drainage create scar.
Lymph node	Typical nodal morphology or nodal chain; skin cancer history.	Intraparotid nodes harbor metastatic cutaneous SCC and melanoma.
Lymphoma	Multiple nodes, systemic context, homogeneous lesions; flow cytometry helpful.	Open parotidectomy is not the first-line diagnostic strategy.
Ranula / floor-of-mouth cyst	Sublingual-space origin, plunging component through mylohyoid defect.	Not a submandibular-gland tumor; operation and recurrence logic differ.

CLINICAL BRIDGE | A diagnosis that does not explain the compartment is incomplete. Before accepting

"salivary neoplasm," ask whether the lesion is truly intraglandular and whether a nodal, neural, vascular, or inflammatory process fits better.

Chapter Synthesis

- The parotid and submandibular regions are anatomic compartments, not histologic diagnoses.
- Facial-nerve schwannoma and vascular lesions are high-impact preoperative mimics.
- Intraparotid lymph nodes require a deliberate skin-cancer and lymphoma differential.
- Imaging should identify the structure of origin before tissue sampling is planned.

CHAPTER 9

A Management Framework for Benign Disease

Integrate diagnostic confidence, natural history, anatomy, morbidity, and patient preference.

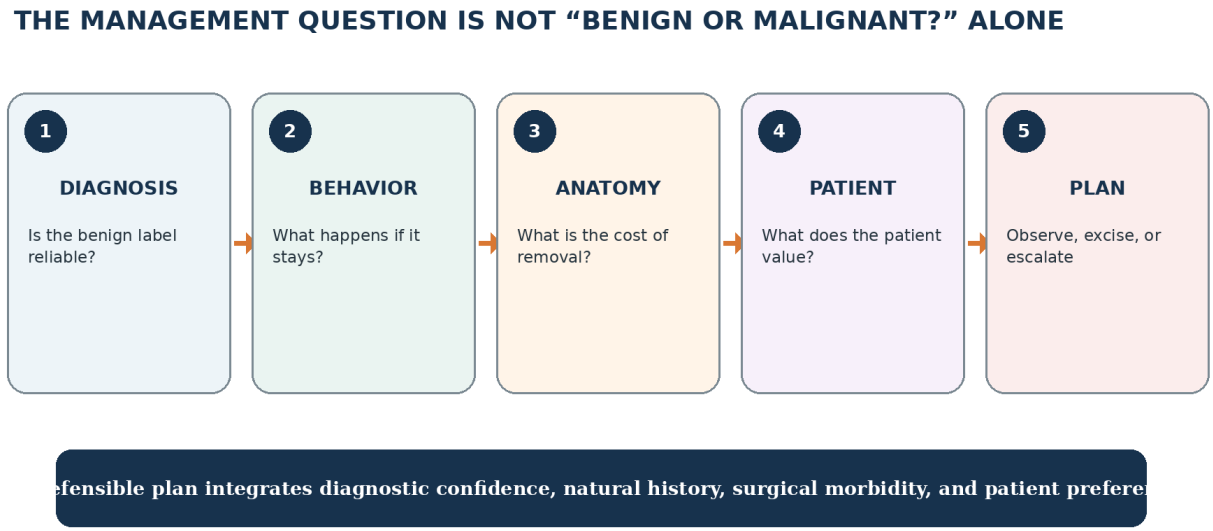


Figure 10. Original synthesis. Benign-tumor management is a multi-variable decision rather than a pathology label converted automatically into surgery.

9.1 The four management states

A salivary-region mass generally enters one of four management states. First, the diagnosis is uncertain and more information is needed. Second, the diagnosis is reliably benign and observation is reasonable. Third, the diagnosis is benign but surgery is favored because of growth, symptoms, future transformation risk, cosmetic effect, or patient preference. Fourth, the lesion is biologically or clinically discordant and should be managed through a malignant or indeterminate-neoplasm pathway.

Table 9. The four practical management states.

State	Typical example	Next step
Need more certainty	Basaloid SUMP, generic oncocytic lesion, cyst fluid only from a solid-cystic mass.	Repeat targeted FNA, core biopsy, MRI, expert review, or diagnostic excision depending on anatomy.
Observation reasonable	Concordant asymptomatic Warthin tumor in an older/comorbid patient.	Document baseline; planned clinical and imaging follow-up; define triggers for re-biopsy or surgery.
Benign but remove	Pleomorphic adenoma; enlarging symptomatic benign tumor; patient prefers definitive treatment.	Plan complete intact excision with morbidity matched to anatomy.
Escalate	Facial weakness, fixation, infiltrative MRI, destructive bone change, rapid growth, suspicious node.	Oncologic imaging, adequate tissue, neck assessment, reconstructive/nerve planning.

9.2 Observation requires a surveillance contract

Observation should specify what is being observed, why the diagnosis is trusted, how progression will be detected, and which changes end observation. Record size in reproducible dimensions, symptoms, facial

function, skin and mucosal findings, nodal examination, and the imaging/tissue evidence. A vague instruction to "follow up as needed" is not active surveillance.

9.3 Operation selection is not tumor-name selection

A small superficial Warthin tumor and a small superficial pleomorphic adenoma may both be removed with a limited operation, but the reasons differ. In pleomorphic adenoma, the dominant priority is controlled intact removal despite microscopic extensions. In Warthin tumor, the dominant issue may be whether surgery is necessary at all. A deep-lobe benign tumor may require extensive nerve dissection because of anatomy rather than biologic aggression.

Table 10. Why the same pathologic diagnosis can lead to different operations.

Variable	How it changes the operation
Superficial vs deep lobe	Deep disease increases facial-nerve manipulation and parapharyngeal exposure; "benign" does not make the access trivial.
Size and mobility	Large or fixed lesions narrow the safe extracapsular plane and raise diagnostic concern.
Facial-nerve relationship	Expected branch proximity may favor formal identification rather than blind limited dissection.
Histology / recurrence biology	Pleomorphic adenoma requires special attention to capsule and satellite nodules; Warthin tumor can be observed.
Prior surgery	Scar obscures planes and increases nerve injury; recurrent PA may be multinodular.
Patient factors	Age, comorbidities, occupation, baseline facial function, tolerance of uncertainty, and cosmetic priorities matter.
Surgeon and institutional expertise	A limited operation is not "less technical"; it depends on correct selection and the ability to convert safely.

9.4 Consent should be anatomy-specific

- Temporary and permanent facial weakness, including which divisions are most at risk for the tumor location.
- Greater auricular numbness, contour change, scar, and ear-lobule symptoms.
- Frey syndrome, salivary leak, sialocele, infection, hematoma, and seroma.
- Need to extend the operation if the diagnosis or nerve relationship differs from expectations.
- Possibility of nerve sacrifice and reconstruction when malignancy or inseparable gross invasion is encountered—when this is a realistic preoperative concern.
- Need for final pathology to revise the treatment plan.

OPERATING-ROOM RELEVANCE | "Benign parotidectomy" is not an adequate consent description. The operation should be described by the predicted nerve dissection, gland volume removed, diagnostic contingencies, and expected functional/cosmetic tradeoffs.

Chapter Synthesis

- Observation, surgery, and escalation are distinct management states with different documentation requirements.
- A benign diagnosis does not by itself determine the extent of surgery.
- Conservative surgery depends on selection, controlled planes, and a credible conversion plan.
- Consent should reflect anatomy and uncertainty, not only the most likely pathology.

CHAPTER 10

Recurrence and Malignant Transformation

When benign biology meets time, scar, multifocality, or a changing clinical phenotype.

10.1 Recurrent pleomorphic adenoma is different disease

Primary pleomorphic adenoma is usually a single, mobile, well-defined mass in an undisturbed facial-nerve plane. Recurrent pleomorphic adenoma may be multinodular, spread through prior operative planes, and wrapped around nerve branches. Nodules may be clinically occult and visible only on high-resolution MRI. The goals shift from removing one mass to mapping all disease while deciding how much nerve risk is acceptable.

Obtain the original operative report and pathology whenever possible. Tumor rupture, positive margins, enucleation, and the interval to recurrence all inform the model. MRI should cover the entire parotid bed and relevant deep spaces. Ultrasound can identify superficial nodules but is insufficient as the only map for complex recurrent disease.

Table 11. Why recurrence changes both anatomy and counseling.

Primary PA	Recurrent PA
Usually one dominant lesion.	Often multifocal or multinodular.
Native tissue planes.	Scarred planes; facial nerve displaced, tethered, or encased.
Conservative operation often feasible.	Revision parotidectomy may require broader dissection and nerve-reconstruction planning.
Low recurrence after intact complete removal.	Risk of additional recurrence remains prolonged even after revision.
Routine preoperative counseling.	Discuss permanent weakness, nerve grafting, incomplete microscopic clearance, and selected adjuvant-radiation considerations.

10.2 Carcinoma ex pleomorphic adenoma

Carcinoma ex pleomorphic adenoma is a malignant epithelial tumor arising within a preexisting pleomorphic adenoma. The classic clinical story is a long-standing mass that changes: accelerated growth, pain, fixation, skin involvement, facial weakness, or nodal disease. Some patients have no known prior mass, so pathology may supply the history by identifying residual pleomorphic adenoma or a PLAG1/HMGA2-related background.

RED FLAGS FOR TRANSFORMATION | Rapid recent enlargement; new pain; facial weakness; fixation; skin or mucosal ulceration; cervical nodes; infiltrative imaging; necrosis or destructive extension.

The malignant component may be salivary duct carcinoma, myoepithelial carcinoma, adenocarcinoma not otherwise specified, or another phenotype. Prognosis depends strongly on the extent of invasion beyond the capsule, grade, stage, margins, and nodal disease. Once transformation is suspected, the workup and operation should be planned as a salivary carcinoma—not as a benign mass with an unexpectedly larger incision.

10.3 Other recurrent benign entities

A new Warthin tumor in the same or opposite parotid often represents multifocal or metachronous disease rather than implantation recurrence. Canalicular adenoma can also be multifocal. For these tumors, the question is whether each new focus requires treatment. Symptoms, diagnostic concordance, cumulative gland loss, and bilateral facial-nerve risk may favor observation or targeted excision rather than progressively wider surgery.

10.4 The role of radiation in recurrent benign PA

Radiation is not used for a routine primary pleomorphic adenoma. In selected patients with multiply recurrent, grossly residual, unresectable, or nerve-threatening pleomorphic adenoma, postoperative or definitive radiation may be discussed in a multidisciplinary setting. The evidence is retrospective and must be balanced against long-term toxicity in a benign disease. This is a case-specific strategy, not a boards-level default.

> **BOARD TRAP** | A long duration does not protect against carcinoma ex pleomorphic adenoma. The testable clue is a change in behavior within a previously indolent mass.

Chapter Synthesis

- Recurrent pleomorphic adenoma is often multinodular and surgically more hazardous than the primary tumor.
- MRI mapping and retrieval of the original operative/pathology record are core preoperative steps.
- A rapidly changing long-standing mass should be treated as possible carcinoma ex pleomorphic adenoma.
- Warthin and canalicular multifocality should not automatically trigger progressively radical surgery.

CHAPTER 11

Integrated Clinical Cases and Board-Relevant Traps

Use the framework: localize, establish diagnostic confidence, identify behavior, then choose the least morbid safe plan.

Case 1. The classic superficial pleomorphic adenoma

A 39-year-old patient has a 2.3-cm mobile, painless superficial parotid mass. Ultrasound shows a circumscribed solid lesion; FNA favors pleomorphic adenoma. Facial function is normal. The appropriate plan is definitive surgery with intact removal in a controlled plane. Depending on branch relationship and expertise, extracapsular dissection, partial superficial parotidectomy, or superficial parotidectomy may be defensible. Enucleation is not.

ANSWER KEY | The pathology predicts the technical priority: preserve the capsule and account for microscopic extensions. The operation is selected by anatomy, not by a universal rule that every pleomorphic adenoma requires the same volume of gland removal.

Case 2. The PET-avid tail lesion

A 74-year-old smoker has a 1.6-cm FDG-avid parotid-tail lesion discovered incidentally. Ultrasound shows a partly cystic, well-defined lesion; FNA shows the classic Warthin triad. The patient is asymptomatic and anticoagulated for atrial fibrillation. Active surveillance is reasonable after documenting concordance and a follow-up plan. PET avidity alone is not an indication for parotidectomy.

Case 3. “Benign” cytology with facial weakness

A 58-year-old patient has a parotid mass and slowly progressive marginal mandibular weakness. FNA is interpreted as pleomorphic adenoma. MRI shows irregular enhancement extending toward the stylomastoid foramen. The result is discordant. The weakness and nerve-pathway imaging dominate; obtain expert pathology review and adequate tissue, stage as appropriate, and plan an oncologic nerve strategy.

ANSWER KEY | The triple test is not majority voting. A benign aspirate cannot cancel objective nerve dysfunction.

Case 4. The upper-lip nodule

A 70-year-old woman has a 1-cm painless upper-lip submucosal nodule. Excision shows beaded cords in loose vascular stroma. Canalicular adenoma is favored. No parotid imaging or neck treatment is needed. If a second nearby lesion appears years later, multifocality is plausible; reassess rather than assuming malignant recurrence.

Case 5. Basaloid SUMP

A 66-year-old patient has a 2.5-cm parotid lesion. FNA shows a basaloid neoplasm classified as SUMP. Imaging is well circumscribed, but the specimen cannot distinguish basal cell adenoma from adenoid cystic carcinoma or basal cell adenocarcinoma. A definitive surgical plan should preserve the ability to extend the operation if invasion or malignancy is found, and frozen section should be asked focused questions rather than expected to solve every basaloid differential.

Case 6. Long-standing mass with sudden growth

A patient reports a parotid lump present for 15 years that has doubled in size over three months and is now painful. This is carcinoma ex pleomorphic adenoma until adequately evaluated. Obtain MRI with attention to nerve pathways, nodal and chest staging as appropriate, and tissue sufficient to identify the malignant component. Do not schedule a routine “benign superficial parotidectomy” without nerve, neck, and reconstruction contingencies.

Rapid Board Review

Table 12. High-yield recall.

Prompt	Best answer
Most common benign salivary neoplasm	Pleomorphic adenoma.
Benign tumor classically bilateral/multifocal and tobacco-associated	Warthin tumor.
Upper-lip tumor in older woman with beaded cords	Canalicular adenoma.
Why PA recurs after enucleation	Incomplete/thin capsule, pseudopodia, satellite nodules, positive margin, rupture/spillage.
Classic Warthin FNA triad	Oncocytes + lymphocytes + granular/proteinaceous debris.
How to distinguish BCA from BCAC	Destructive invasion; cytologic blandness does not exclude carcinoma.
Meaning of oncocytic cytoplasm	Mitochondrial accumulation; requires a differential, not a single diagnosis.
Long-standing PA with rapid growth/pain/weakness	Suspect carcinoma ex pleomorphic adenoma.
When Warthin observation is reasonable	Concordant diagnosis, asymptomatic patient, acceptable follow-up, informed preference.
Most important response to clinic-pathology discordance	Resolve the discordance; do not average it away.

CHAPTER 12

One-Page Consolidation

The smallest durable mental model for benign salivary neoplasms.

Table 13. Core benign neoplasm map.

Entity	Clinical anchor	Pathologic anchor	Management anchor
Pleomorphic adenoma	Slow, painless; parotid most common; palate/minor glands also.	Ductal + myoepithelial cells in variable matrix; incomplete capsule, pseudopodia, satellites.	Surgery; intact controlled removal; no enucleation; long-term recurrence awareness.
Warthin tumor	Older adult, tobacco exposure, parotid tail, bilateral/multifocal.	Papillary-cystic bilayer oncocytes + lymphoid stroma.	Observe when diagnosis is concordant and asymptomatic; surgery for uncertainty, symptoms, deformity, preference.
Basal cell adenoma	Usually parotid, older adult.	Biphasic basaloid tubular/trabecular/solid/membranous patterns.	Complete excision; invasion means adenocarcinoma.
Canalicular adenoma	Older woman, upper lip.	Beaded cords in loose vascular stroma; luminal phenotype.	Local excision; multifocality can occur.
Myoepithelioma	Parotid or minor glands.	Predominantly myoepithelial, little/no ducts or PA matrix.	Complete intact excision; interface required to prove benign behavior.
Oncocytoma	Rare, usually parotid, solid circumscribed mass.	Encapsulated monotonous oncocytes.	Complete excision; exclude Warthin, hyperplasia, carcinoma, metastasis.
Sclerosing polycystic adenoma	Rare, parotid often.	Sclerosis, cysts, acinar/ductal proliferation, apocrine change, possible intraductal atypia.	Complete excision and individualized follow-up.

FINAL RESIDENT TAKEAWAY | A benign salivary mass is managed safely when four statements are simultaneously true: the diagnosis is coherent, the clinical behavior fits, the anatomy is mapped, and the plan accounts for both natural history and treatment morbidity.

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