

Sinonasal Angiofibroma

Clinicopathology • Morphology • Molecular basis • Diagnostic strategy

A benign fibrovascular neoplasm with potentially serious local effects

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Learning outcomes

- Distinguish benign histology from locally aggressive behavior in sinonasal angiofibroma.
- Link vascular–stromal architecture to epistaxis, imaging findings and biopsy risk.
- Interpret CTNNB1/ β -catenin and APC/FAP associations without overstating causation.
- Plan a safe clinicopathologic diagnostic pathway and recognize important mimics.

The entity: Vascular tissue and fibrous stroma

Angio-

Numerous vascular channels

And prominent blood supply

-fibroma

Collagen-rich stroma

With bland fibroblastic cells

Clinical behavior

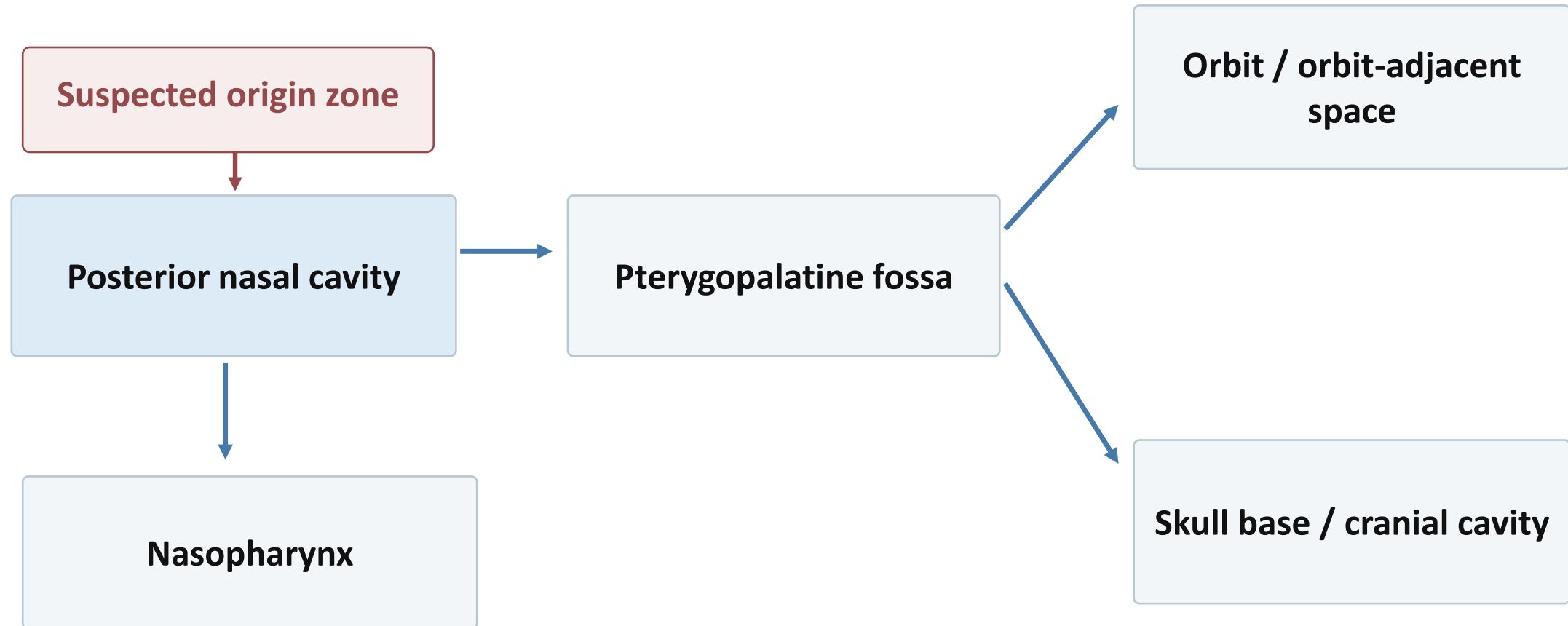
Histologically benign

But often locally aggressive

Why the modern name emphasizes “sinonasal”

- Historically called juvenile nasopharyngeal angiofibroma (JNA).
- Origin is not restricted to the nasopharyngeal mucosal compartment.
- The posterolateral nasal cavity and nearby lateral nasopharyngeal region are implicated.
- WHO 5th-edition terminology places sinonasal tract angiofibroma among sinonasal mesenchymal tumors.

Posterolateral nasal cavity: Topologic relationships



Topologic relationship schematic only — not a scaled anatomical section

The sphenopalatine region as a disease hub

Anatomic neighborhood

Posterolateral nasal wall

Near sphenopalatine foramen

Vascular context

Arterial feeders can supply

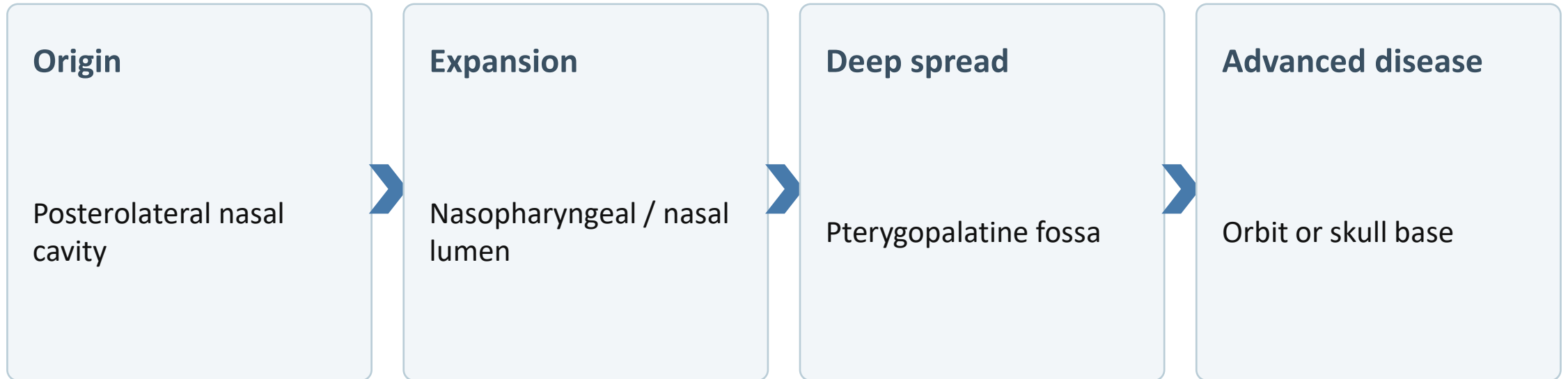
A densely vascular mass

Extension context

Pterygopalatine compartment

Communicates with deeper spaces

How a localized lesion becomes extensive



Extent describes anatomical reach, not malignant histology.

Anatomic planes help explain local extension

Medial / posterior

Nasal airway and choanal space

→ obstruction

Lateral

Pterygopalatine and adjacent

Infratemporal compartments

Superior

Skull-base and orbital proximity

→ serious complications

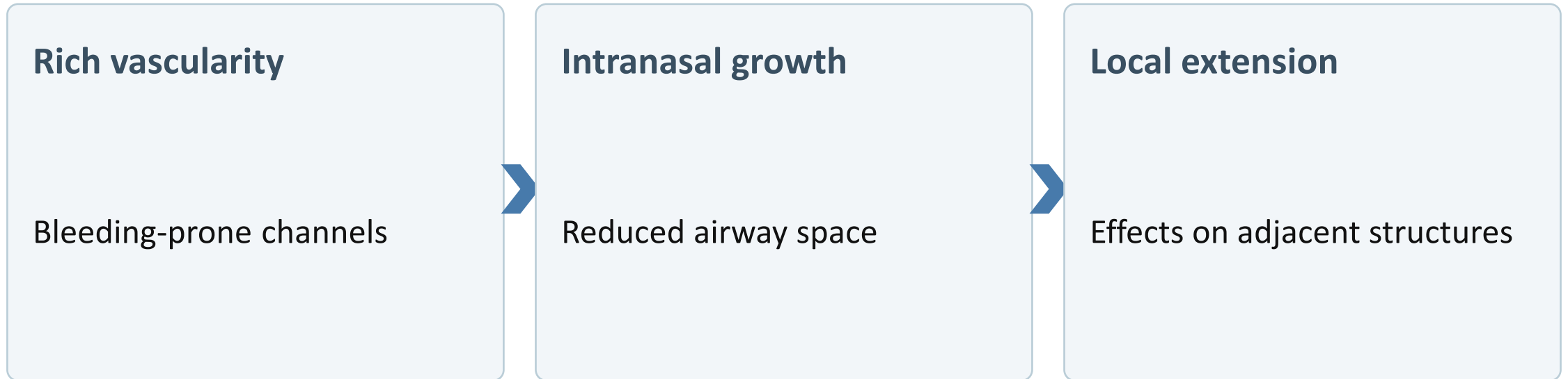
Benign is a histologic classification—not a safety guarantee

- The tumor is composed of bland vascular and stromal elements.
- No malignant cytologic features are required for substantial morbidity.
- Expansile growth and extension may compromise neighboring structures.
- Hemorrhage can be a major procedural and clinical hazard.

Clinical setting: The characteristic patient

- The strongest epidemiologic clue is an adolescent male.
- Occurrence outside this population is unusual, but not impossible.
- Age and sex contribute to pre-test probability; They do not prove a diagnosis.
- Consider the lesion particularly when a posterior nasal mass is also highly vascular.

Why symptoms arise



One lesion produces both hemorrhagic and obstructive symptom patterns.

Epistaxis: The key presenting clue

- Epistaxis is prominent in the Robbins description of sinonasal angiofibroma.
- Fragile and numerous vascular channels provide a pathologic explanation.
- Recurrent or marked bleeding should heighten concern before tissue sampling.
- Blood-loss consequences must be evaluated clinically when episodes are significant.

Nasal obstruction: Direct mechanical effect

- Expansion within the nasal cavity or choana narrows the breathing passage.
- Obstruction may be unilateral initially because of the mass location.
- A vascular mass causing obstruction requires a different workup from an ordinary polyp.
- Persistent obstruction plus epistaxis is more informative than either symptom alone.

Features suggesting disease beyond the nasal lumen

Orbit-adjacent

Proptosis or orbital symptoms

May indicate extension

Cranial / neural

Neurologic findings can signal

Advanced skull-base
involvement

Facial compartments

Facial contour or palatal
changes

Can arise from mass effect

Clinical problem: A posterior nasal mass

A 15-year-old boy presents with repeated brisk epistaxis and progressive unilateral nasal obstruction. Examination suggests a reddish posterior nasal lesion.

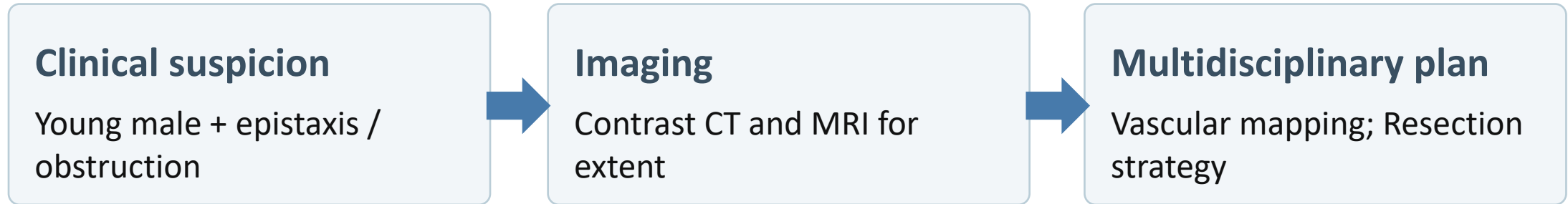
Questions to answer

- Which diagnosis should immediately enter the differential?
- Which commonly used office procedure could cause severe hemorrhage?

The key safety rule: Avoid an unplanned biopsy

- Sinonasal angiofibroma is highly vascular and can bleed profusely.
- Suspected cases are generally characterized using cross-sectional imaging first.
- Biopsy is not routine when clinical and imaging findings are characteristic.
- If diagnostic tissue is necessary, the approach requires specialist planning and bleeding control.

Safe initial diagnostic sequence



Avoid an unplanned office biopsy of a highly vascular suspected angiofibroma.

Nasal endoscopy: Evaluate without traumatizing

- An endoscopic examination may reveal a vascular-appearing posterior nasal or nasopharyngeal mass.
- Inspection can establish laterality, accessible surface appearance and obstruction.
- Surface appearance alone cannot determine the full extent or histology.
- Avoid forceful instrumentation or routine tissue sampling of a suspected vascular lesion.

Imaging answers distinct clinical questions

CT

Bone remodeling / erosion

And compartment anatomy

MRI

Soft-tissue extent

And intracranial relations

Angiography

Arterial supply

And embolization planning

Contrast-enhanced CT: Establishing lesion geography

- Avid contrast enhancement supports the presence of a hypervascular mass.
- CT maps nasal, nasopharyngeal and paranasal sinus involvement.
- It can show altered bony contours and widened deep facial spaces.
- Interpret a characteristic location and enhancement pattern together—not in isolation.

Bone on CT: Remodeling versus destructive reach

Displacement

Progressive mass effect can

Bow neighboring bony walls

Remodeling

Expansion changes the shape

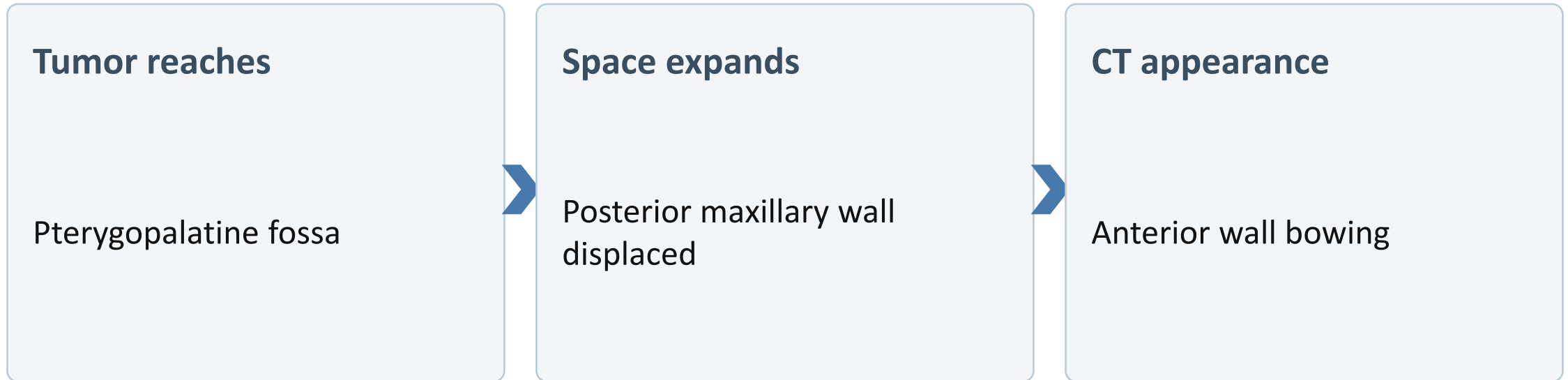
Of adjacent structures

Advanced extent

Skull-base erosion may occur

Even though tumor is benign

Holman–Miller sign: A cause–effect sequence



Supportive imaging sign, not a standalone histologic diagnosis.

MRI clarifies soft-tissue extension

- MRI is helpful for defining the interface with orbit and skull base.
- It identifies disease extending toward the cranial cavity or soft-tissue compartments.
- Contrast enhancement and vascular flow phenomena provide clues to tumor vascularity.
- MRI is valuable in assessing residual or recurrent tissue after treatment.

Flow voids: The imaging correlate of moving blood

Histologic premise

Abundant vascular channels

Carry flowing blood

MR observation

High-flow channels may appear

As internal signal voids

Interpretation

A vascular clue that complements

Location and enhancement

CT versus MRI: Complementary roles

Feature	CT emphasizes	MRI emphasizes
Anatomic overview	Bone and sinonasal boundaries	Soft-tissue compartments
Tumor vascularity	Contrast enhancement	Flow voids and enhancement
Deep extension	Pterygopalatine bony changes	Orbit / intracranial relationships
After treatment	Remodeling and anatomy	Residual or recurrent enhancing tissue

Angiography: Not simply another diagnostic picture

- Catheter angiography delineates feeding arterial branches and vascular anastomoses.
- The arterial map informs hemorrhage-reduction strategies.
- Embolization can be performed when appropriate to the vascular anatomy.
- Potential hazardous anastomoses make specialist technique and patient selection essential.

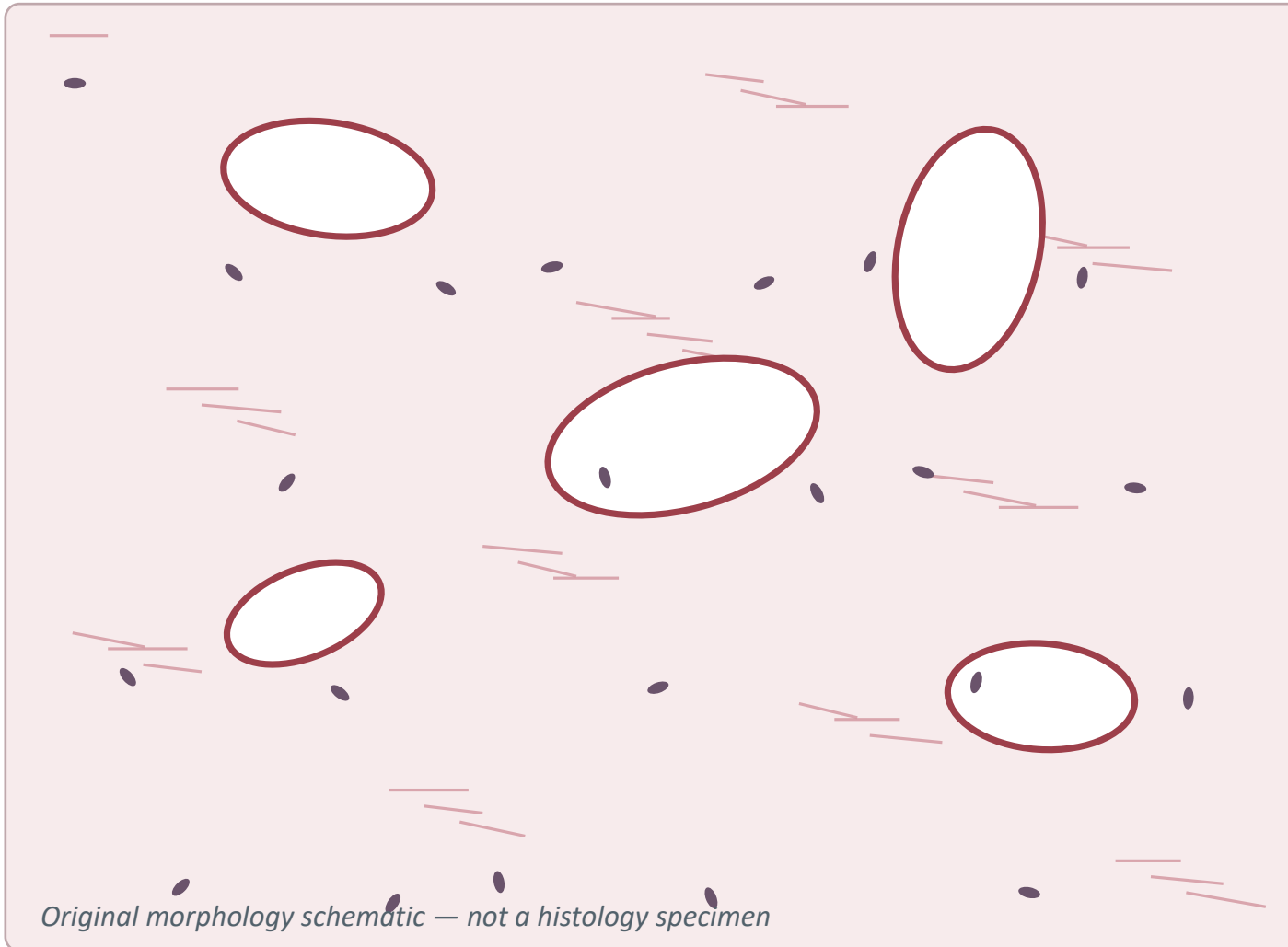
Before surgery, imaging should answer four questions

- What is the full extent of the mass and its deep-space involvement?
- Does it abut or extend through the skull base or toward the orbit?
- Where are major arterial feeders and potentially dangerous connections?
- What boundaries will influence the feasibility and completeness of excision?

Macroscopic appearance: Integrate, do not overdiagnose

- Angiofibroma is often described as a vascular, firm or lobulated soft-tissue mass.
- Visible vessel-rich areas correspond to the “angio-” component.
- Fibrous texture corresponds to the collagenized stromal component.
- Gross appearance is nonspecific: Definitive tissue interpretation uses microscopy and context.

The biphasic histologic pattern



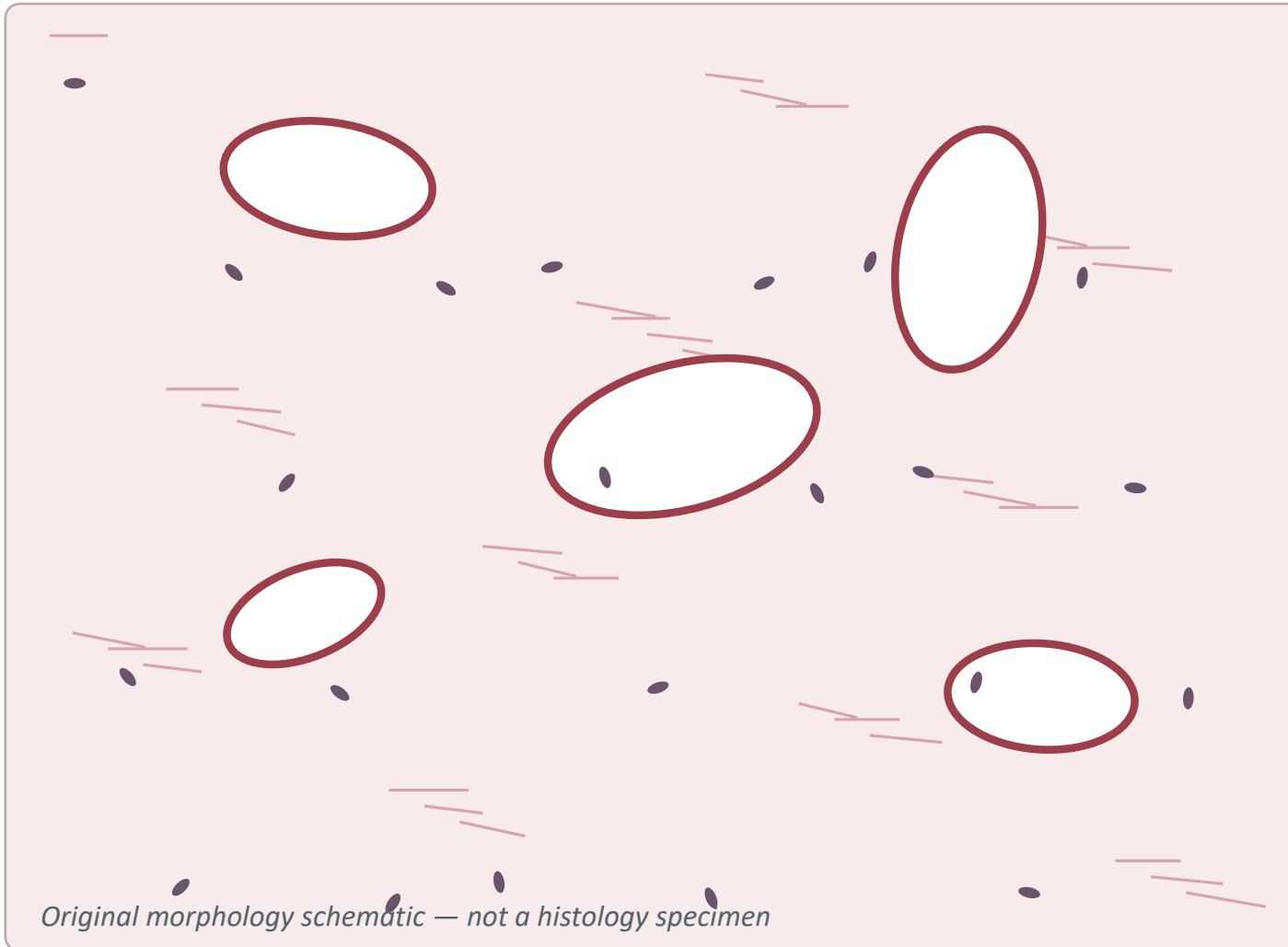
What to recognize

Numerous endothelial-lined vascular spaces

Fibrous, collagen-rich supporting tissue

Scattered bland stromal fibroblasts

Vascular channels: Varying size and shape



What to recognize

Vascular profiles vary in caliber and contour

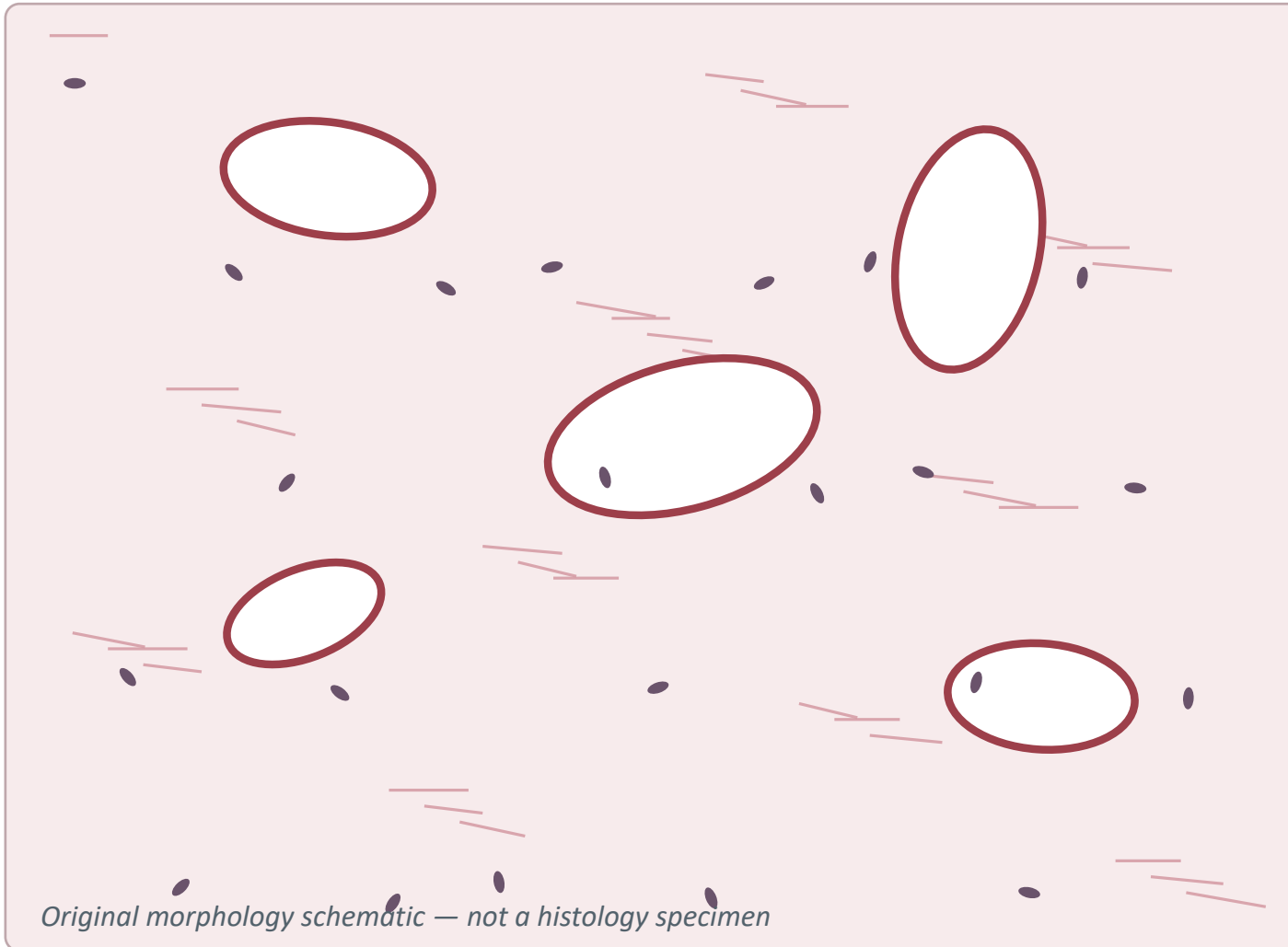
Thin or incomplete muscular vessel walls

Fragile channels help explain brisk epistaxis

Endothelial lining is deceptively simple

- Vessel lumina are lined by a single layer of endothelial cells.
- Vascular spaces may be irregular, angulated or variably dilated.
- The lesion's diagnostic pattern depends on the vessel–stroma relationship.
- Endothelial cellular atypia is not the defining abnormality in this benign neoplasm.

Vessel walls and bleeding propensity



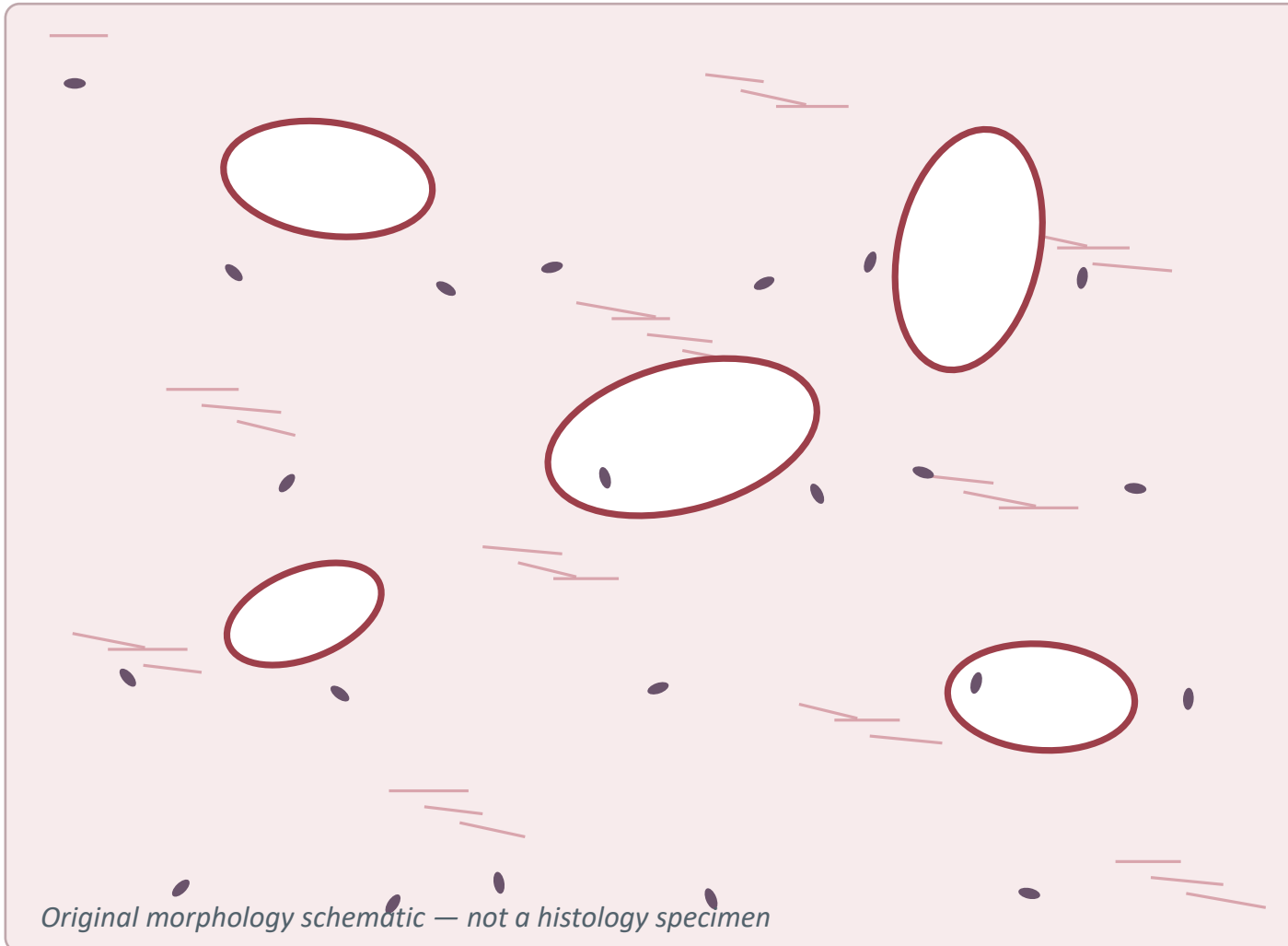
What to recognize

Vascular profiles vary in caliber and contour

Thin or incomplete muscular vessel walls

Fragile channels help explain brisk epistaxis

Collagen-rich stroma and bland fibroblasts



What to recognize

Pink field depicts collagen
(conceptually)

Small dark ovals represent
bland stromal nuclei

Diagnosis requires real
specimen evaluation

What the microscope should separate

Vascular element

Endothelial-lined lumina

With diverse caliber

Stromal element

Collagenous matrix with

Small bland fibroblasts

Overall pattern

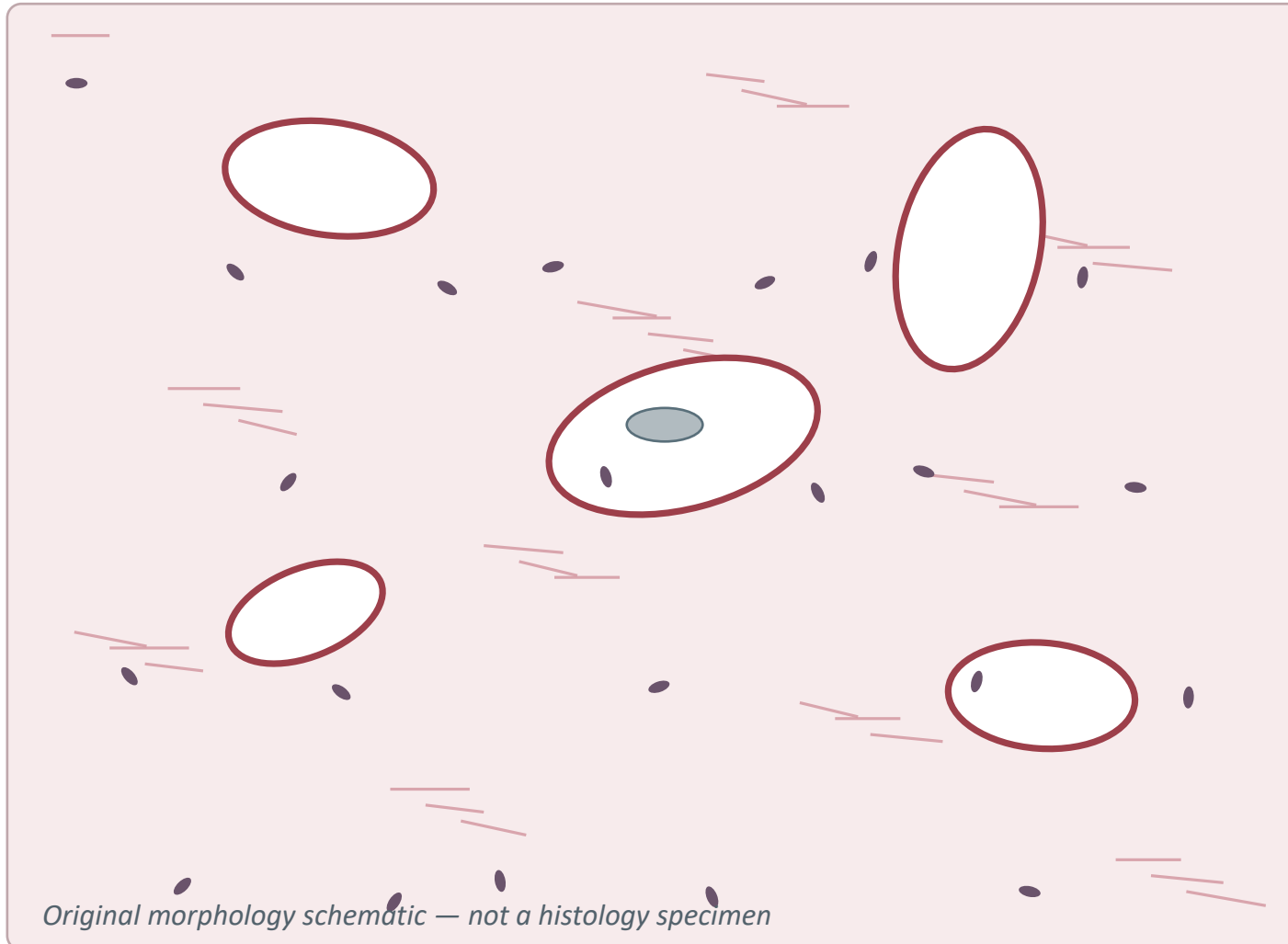
Mixed fibrovascular
architecture

Without a malignant
phenotype

Interpretation pitfall: Architecture outranks one shape

- A staghorn-like or branching vessel contour is not unique to angiofibroma.
- Collagenization can vary between cases and microscopic fields.
- Limited sampling may fail to show the overall vascular–stromal architecture.
- Age, site, imaging and histology must be integrated when morphology overlaps mimics.

Prior embolization can modify histologic vessels



What to recognize

Gray intraluminal material is schematic embolus

Actual treated tissue may show vascular changes

Interpret treatment effects with procedure history

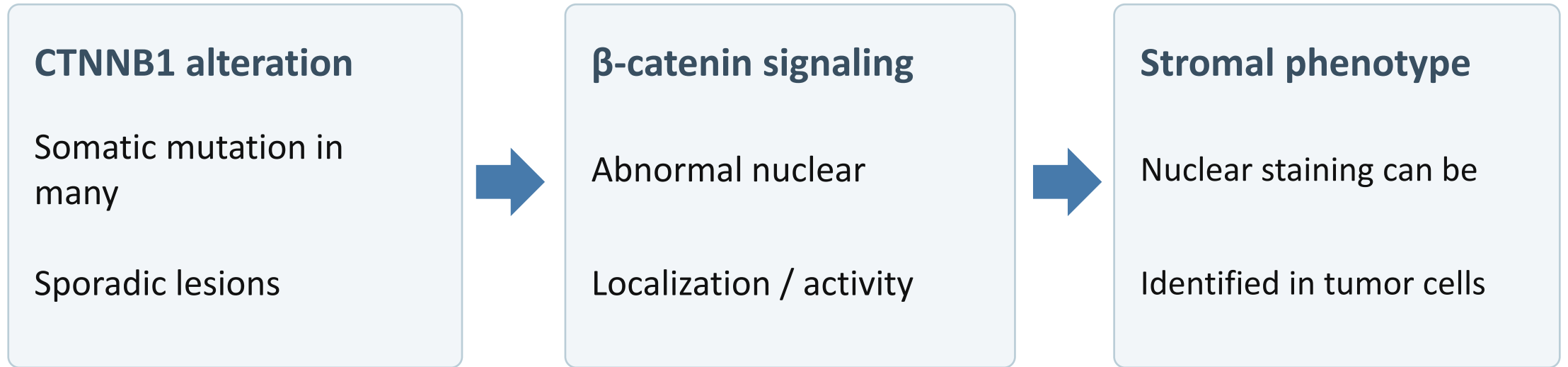
Handling a vascular excision specimen

- Confirm procedure history, especially whether preoperative embolization occurred.
- Assess the fibrovascular lesion on multiple representative sections.
- Correlate cautery, hemorrhage and treatment-related material with operative context.
- Communicate uncertainty rather than forcing a diagnosis from a fragmented small sample.

β -catenin is part of the tumor's molecular story

- Most sporadic sinonasal angiofibromas harbor CTNNB1 mutations.
- CTNNB1 encodes β -catenin, a protein involved in cellular signaling regulation.
- Abnormal nuclear β -catenin localization provides a tissue-level correlate.
- Association with a molecular alteration does not equate to an independently sufficient diagnostic test.

From CTNNB1 to nuclear β -catenin



Genotype $\rightarrow$ pathway implication $\rightarrow$ tissue-level immunophenotype

Nuclear β -catenin staining: What it can support

- WHO review describes nuclear β -catenin localization in nearly all cases.
- The relevant positive cells are the neoplastic stromal component.
- The pattern supports altered β -catenin regulation when paired with morphology.
- It is not pathognomonic: Other sinonasal tumors can share nuclear staining.

Sporadic CTNNB1 versus syndromic APC context

Sporadic sinonasal angiofibroma

Commonly associated with somatic

CTNNB1 mutations

β -catenin pathway activation

Syndromic context (FAP)

Germline APC predisposition

Can converge on altered

β -catenin regulation

Different upstream genetic events can affect the same regulatory pathway.

Familial adenomatous polyposis: The association

- An angiofibroma can occur in the context of familial adenomatous polyposis (FAP).
- In the hereditary setting, germline APC mutation is the stated driver context.
- APC perturbation also converges on β -catenin regulation.
- The association should prompt appropriate consideration of personal or family history.

Androgen receptor: Relevant but not an explanation by itself

- WHO review notes androgen receptor immunoreactivity in stromal cells is common.
- Clinical predilection for adolescent males is consistent with biological interest in hormonal pathways.
- Receptor expression is not proof that androgen signaling solely causes the neoplasm.
- Do not replace an integrated diagnosis with a single immunostain.

What molecular findings actually mean

Feature	Observation	Interpretive limit
CTNNB1	Mutated in majority of sporadic cases	Not a universal standalone diagnostic criterion
β-catenin IHC	Nuclear localization is common	Pattern overlaps other mesenchymal tumors
Germline APC	FAP-associated pathogenesis	Association ≠ all cases hereditary
Androgen receptor	Stromal immunoreactivity reported	Expression ≠ established sole cause

Differential diagnosis begins before pathology

- Posterior nasal symptoms occur in both benign and malignant diseases.
- Epistaxis, enhancement, demographics and epicenter change the differential.
- Do not biopsy a presumed benign inflammatory polyp before recognizing suspicious vascularity.
- Context determines the safest route to definitive characterization.

Angiofibroma versus inflammatory nasal polyp

Clue	Angiofibroma	Inflammatory polyp
Typical context	Adolescent male / recurrent bleeding	Inflammatory nasal symptoms
Tissue structure	Vascular channels + fibrous stroma	Edematous inflamed mucosal stroma
Vascularity	Marked; Biopsy hazard	Usually far less conspicuous
Imaging priority	Map deep extension and feeders	Assess extent and sinus disease

Angiofibroma versus sinonasal papilloma

Dimension	Angiofibroma	Papilloma
Cell of origin	Fibrovascular mesenchymal lesion	Schneiderian epithelial neoplasm
Age/setting	Predominantly adolescent males	Commonly adults
Microscopic hallmark	Thin-walled vessels in collagen	Epithelial proliferation / inversion
Molecular context	CTNNB1 / β -catenin	EGFR mutation in many inverted cases

Angiofibroma versus glomangiopericytoma

Diagnostic element	Angiofibroma	Glomangiopericytoma
Dominant pattern	Numerous vessels + fibrous stroma	Perivascular myoid spindle-cell tumor
Stromal character	Bland fibroblasts; Collagenized	Cellular perivascular myoid growth
β -catenin IHC	Nuclear expression common	Nuclear expression may also occur
Clinical integration	Young male; Classic site	Often older patients; Other sinonasal sites

Other vascular-appearing lesions to consider

Vascular malformation

Vessel-rich lesion without

The classic neoplastic stroma

Hemangioma and analogues

Distinct vessel architecture

And clinical setting

Other mesenchymal tumors

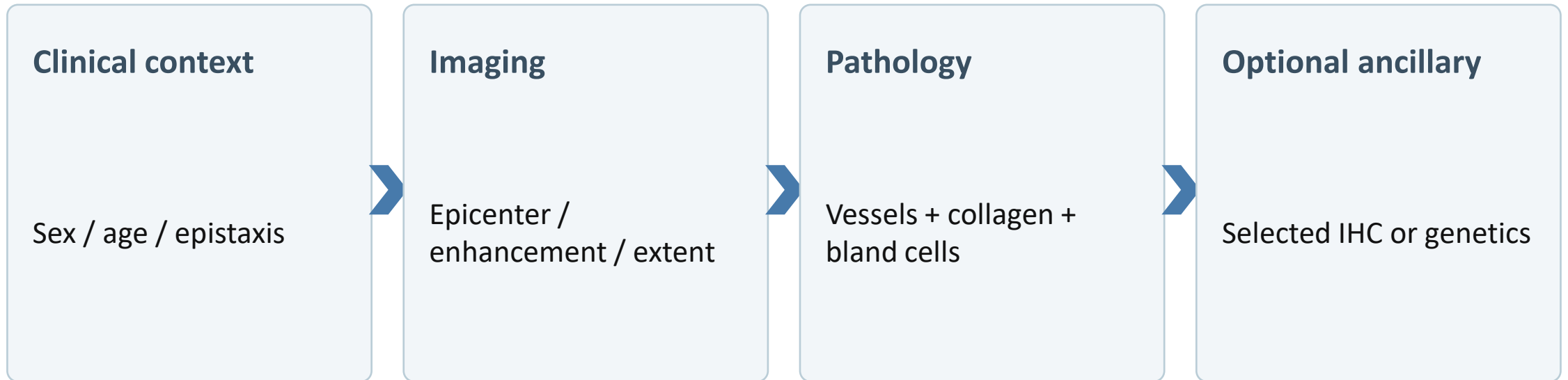
Perivascular or stromal lesions

May mimic selected fields

When to broaden the differential to malignancy

Assessment	Angiofibroma pattern	Concern for alternative
Presentation	Classic demographic and vascular pattern	Atypical age/site or aggressive symptoms
Imaging	Characteristic hyperenhancing epicenter	Infiltrative noncharacteristic pattern
Microscopy	Bland endothelial and stromal cells	Atypia, mitoses, necrosis or epithelial cancer
Decision	Integrated clinicoradiologic diagnosis	Targeted investigation by specialist team

Diagnostic reasoning is multimodal



Each domain supports interpretation; None replaces the others.

Locally aggressive growth is the chief paradox

- The underlying cytology is benign rather than frankly malignant.
- Growth can extend into complex facial spaces and the cranial cavity.
- Risk therefore depends strongly on anatomical extent and proximity to critical structures.
- “Benign” must never be interpreted as negligible surgical or clinical risk.

Major complications follow location, not atypia

Blood supply

Severe bleeding during symptoms

Or instrumentation

Orbital relationship

Extension may threaten

Orbital structures

Intracranial reach

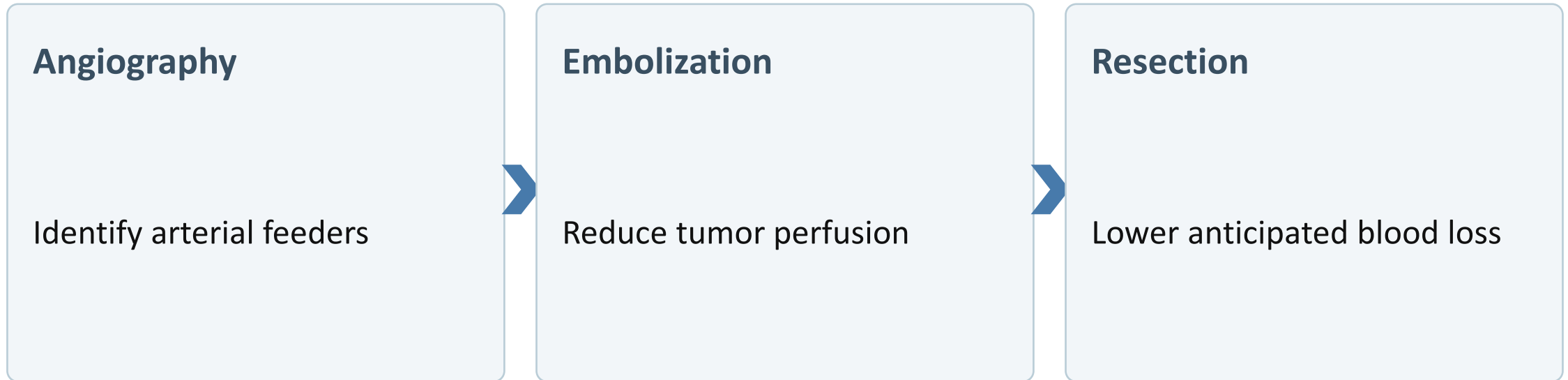
Skull-base extension

Raises treatment complexity

Surgical excision is the principal treatment

- Surgery as the treatment of choice.
- The operative route depends on size, site, vascular supply and deep extension.
- Complete removal may be difficult when disease involves complex skull-base anatomy.
- High-quality preoperative anatomical mapping is therefore part of treatment planning.

Why embolization precedes selected operations



Preoperative embolization is often useful, but the vascular map determines feasibility and risk.

Embolization is an intervention with its own risks

- Dangerous extracranial–intracranial arterial connections require recognition.
- Nontarget embolization may injure ophthalmic or cerebral circulation.
- Preoperative endovascular treatment must be performed by an experienced team.
- Embolization modifies surgical risk; It does not substitute for definitive assessment of extent.

What increases the chance of persistence or recurrence?

- Anatomically inaccessible or deeply extended tumor may be difficult to clear.
- Residual tumor after excision can later be detected as persistent or recurrent disease.
- Imaging surveillance is particularly relevant in patients with complex or advanced lesions.
- Recurrence estimates should be interpreted in relation to tumor stage and treatment series.

How to read published outcome percentages

- Recurrence rates can approach 20% in some settings.
- The same brief textbook section cites a 9% fatal-case figure without a defined contemporary denominator here.
- Do not communicate either number as a universal individual prognosis or modern treatment benchmark.
- Current counseling requires disease extent, treatment details and population-specific outcome data.

Post-treatment assessment: Persistent disease is not invisible

- Contrast-enhanced MRI can help detect and characterize residual or recurrent tumor.
- Interpret enhancing tissue in relation to operative anatomy and previous imaging.
- Follow-up strategy is individualized rather than based on a single universal interval.
- New epistaxis or progressive obstruction after therapy warrants reassessment.

Case exercise: Protect the patient first

An adolescent male has repeated spontaneous epistaxis. CT shows an enhancing posterior nasal mass and enlargement of the pterygopalatine region. A clinic considers endoscopic biopsy.

Questions to answer

- What features suggest sinonasal angiofibroma?
- What immediate change to the diagnostic plan is appropriate?

Case answer: Imaging before tissue

- Demographics + recurrent epistaxis + hypervascular posterior mass point toward angiofibroma.
- Pterygopalatine involvement further supports the characteristic anatomic pattern.
- Avoid an unplanned biopsy because of hemorrhage risk.
- Arrange specialist cross-sectional mapping and appropriate vascular/surgical assessment.

Case exercise: Interpret a vascular–stromal lesion

Resection tissue shows bland spindle fibroblasts in dense collagen. Numerous irregular, thin-walled channels have a single endothelial lining. Nuclear β -catenin is seen in stromal nuclei.

Questions to answer

- Which components establish the morphologic pattern?
- What is the correct interpretation—and limitation—of β -catenin staining?

Case answer: Architecture plus contextual molecular support

- The key morphologic feature is a mixture of vascular channels and collagenized bland stroma.
- Nuclear β -catenin is compatible with the CTNNB1/APC-associated biological context.
- β -catenin expression alone is not uniquely diagnostic of angiofibroma.
- Site, demographic context, imaging and overall morphology complete the diagnosis.

Synthesis: Clinicopathologic correlations

Finding	Underlying explanation
Recurrent epistaxis	Abundant, vulnerable vascular channels
Obstructive symptoms	Space-occupying posterior sinonasal mass
Avid enhancement / flow voids	Rich blood supply / flowing vascular channels
Firm fibrous lesion	Dense collagenous stroma
Local aggressiveness	Expansion into deep adjacent compartments
Postoperative recurrence	Residual tissue in anatomically complex sites

Final diagnostic take-home points

- An adolescent male with recurrent epistaxis and a posterior hypervascular nasal mass warrants consideration of angiofibroma.
- Do not undertake an unplanned biopsy; Map vascularity and extent first.
- Histology is fibrovascular and cytologically benign, yet local extension can be serious.
- CTNNB1 / β -catenin abnormalities and occasional APC/FAP association inform biological interpretation.

Selected references for further reading

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