MedMentor EDU

ENT Study Notes — Otology Series

TUMOURS OF THE EXTERNAL EAR

Pinna · External Auditory Canal · Temporal Bone

Optimized for MBBS · NEET-PG · INI-CET · Viva Voce

Reference Textbooks:

Dhingra & Dhingra | K. K. Ramalingam | Logan Turner

SECTION 1 | Introduction — Tumours of the External Ear

1.1 Definition

Tumours of the external ear encompass a broad spectrum of neoplastic lesions — benign, premalignant, and malignant — arising from any component of the external ear, including the pinna (auricle), the external auditory canal (EAC), and related soft tissue and bony structures.

1.2 Epidemiology

- Tumours of the pinna and EAC are uncommon but clinically significant due to proximity to critical neurovascular structures.
- Squamous cell carcinoma (SCC) is the most common malignancy of the external ear.
- Basal cell carcinoma (BCC) is the most common malignancy of the pinna specifically.
- Male predominance for most malignant tumours (sun exposure, occupational factors).
- Peak incidence: 6th–7th decade of life.
- EAC carcinoma accounts for approximately 1 in 5,000 to 1 in 15,000 ENT referrals.

1.3 Relevant Surgical Anatomy

Blood Supply of External Ear

- Posterior auricular artery (from external carotid artery) — posterior and medial pinna.
- Superficial temporal artery (from external carotid artery) — anterior and lateral pinna.
- Deep auricular artery (from maxillary artery) — EAC and tympanic membrane.

Lymphatic Drainage — Clinically Critical

EXAM FAVOURITE — Lymphatic Drainage of External Ear:

• Pinna (anterior surface + tragus) ? Preauricular (parotid) nodes

• Pinna (posterior surface) ? Mastoid (retroauricular) nodes

• Lobule ? Upper deep cervical nodes

• External auditory canal ? Parotid / Infraauricular nodes + Upper deep cervical nodes

? Lymph node involvement = key prognostic factor in EAC carcinoma

Embryological Remnants — Clinical Relevance

- Preauricular sinus — persistence of ectodermal pit near ascending helix; may become site of recurrent infection or tumour.
- Accessory auricle — persistence of auricular hillocks; occasionally confused with soft tissue lesion.
- Fissures of Santorini — cartilage defects in the anterior wall of EAC; allow tumour spread to parotid.
- Foramen of Huschke — bony defect in the anterior bony EAC in some adults; route for tumour extension to temporomandibular joint (TMJ) and infratemporal fossa.

[DIAGRAM: External Ear — Anatomy, Blood Supply, Lymphatic Drainage & Key Spread Routes]

Insert labeled diagram here

SECTION 2 | Classification of Tumours of the External Ear

CLASSIFICATION OVERVIEW

1. Benign Tumours

2. Premalignant Lesions

3. Malignant Tumours — Primary

4. Secondary / Metastatic Tumours

2.1 Benign Tumours

A. Epithelial Origin

- Papilloma — most common benign tumour of EAC; squamous papilloma caused by HPV.
- Appears as a warty, pedunculated or sessile growth in the cartilaginous EAC.
- Can be single or multiple (papillomatosis).
- Sebaceous cyst (epidermal inclusion cyst) — most common benign cyst of pinna.
- Contains cheesy keratinous material; arises from blocked sebaceous duct.
- Dermoid cyst — contains skin appendages (hair follicles, sebaceous glands); occurs along fusion lines.
- Syringoma — benign eccrine sweat gland tumour; may occur on pinna.
- Trichoepithelioma — hamartoma of hair follicle origin.
- Cylindroma — benign adnexal tumour (turban tumour); rarely involves EAC.

B. Mesenchymal / Bony / Cartilaginous Origin

- Osteoma — solitary, pedunculated bony growth; arises near tympanosquamous or tympanomastoid suture line.
- Exostosis — multiple, broad-based, bilateral bony swellings in the EAC; associated with cold water swimming.
- Most common benign bony tumour of EAC.

- Chondroma — cartilaginous tumour of pinna; rare.
- Fibroma — fibrous tissue tumour.
- Lipoma — fatty tissue tumour.
- Neurofibroma — nerve sheath tumour; may be part of NF1 (von Recklinghausen disease).
- Hemangioma — vascular tumour; capillary or cavernous; may involve pinna or EAC.

C. Glandular / Adnexal Origin

- Ceruminous gland adenoma — benign tumour from ceruminous (apocrine) glands of the cartilaginous EAC; presents as smooth subepithelial mass.
- Pleomorphic adenoma — rare benign mixed tumour from EAC glands.

D. Pigmented Lesions

- Nevus (melanocytic nevus) — may occur on pinna; requires monitoring for malignant change.

E. Other Benign Lesions

- Keloid — excessive scar tissue after ear piercing; more common in dark-skinned individuals.
- Keratosis obturans — accumulation of desquamated keratin in EAC; causes widening of canal and severe pain.
- EAC papillomatosis — multiple papillomas in EAC; HPV-related.

2.2 Premalignant Lesions

KEY — Premalignant Lesions (High-Yield for NEET-PG):

- Actinic keratosis ? Most common premalignant lesion of the pinna (sun-exposed area)
- Bowen disease (SCC in situ) ? Erythematous scaly plaque; 5–10% progress to invasive SCC
- Cutaneous horn ? Hard keratinous projection; may overlie SCC or keratoacanthoma
- Keratoacanthoma ? Mimics SCC; rapidly growing keratin-filled crater; may self-resolve

- Actinic (solar) keratosis — rough scaly patch from UV-damaged skin; SCC precursor.
- Bowen disease — full-thickness epidermal atypia (SCC in situ); does not breach basement membrane.
- Cutaneous horn — keratinous projection; underlying pathology may be benign, premalignant, or malignant.
- Keratoacanthoma — rapid growth with central keratin plug; usually regresses; microscopically resembles well-diff SCC.

2.3 Malignant Tumours — Primary

A. Squamous Cell Carcinoma (SCC)

- Most common malignancy of the EAC and overall external ear.
- Can arise from pinna, EAC, or both.
- Aggressive — invades bone, parotid, TMJ, skull base, and intracranially.

B. Basal Cell Carcinoma (BCC)

- Most common malignancy of the pinna (skin of auricle).
- Locally invasive but rarely metastasizes.
- Types: nodular (most common), morphoeic/sclerosing (most aggressive), superficial.

C. Melanoma

- Arises from melanocytes of pinna skin.
- Rare but aggressive; early lymph node metastasis.
- EAC melanoma is extremely rare.

D. Other Malignant Tumours

- Verrucous carcinoma — low-grade, exophytic variant of SCC; locally aggressive, rarely metastasizes; "cauliflower" appearance.
- Ceruminous gland carcinoma — malignant tumour from ceruminous glands; subtypes include adenocarcinoma and adenoid cystic carcinoma.
- Adenoid cystic carcinoma — from EAC glands; noted for perineural invasion and late distant metastases.
- Basosquamous carcinoma — mixed BCC and SCC features; more aggressive than BCC alone.
- Merkel cell carcinoma — rare neuroendocrine carcinoma; highly aggressive; associated with Merkel cell polyomavirus (MCPyV).
- Adenocarcinoma — from EAC glandular tissue; rare.

2.4 Secondary and Metastatic Tumours

- Parotid malignancy extension — direct spread into EAC via fissures of Santorini.
- Metastasis to temporal bone — from breast, lung, kidney, stomach, and prostate (haematogenous).
- Skull base extension from nasopharyngeal carcinoma — perineural or direct spread.

2.5 Classification Summary Tables

Table 1: Classification Overview

Category	Examples	Key Feature
Benign	Osteoma, Exostosis, Papilloma, Sebaceous cyst, Ceruminous adenoma, Keloid	No invasion; no metastasi
Premalignant	Actinic keratosis, Bowen disease, Keratoacanthoma, Cutaneous horn	Risk of malignant transfor
Malignant — Primary	SCC, BCC, Melanoma, Adenoid cystic ca, Ceruminous gland ca, Merkel cell ca	Invasion ± metastasis
Secondary / Metastatic	Parotid extension, Haematogenous metastasis	Late stage; poor prognosis

Table 2: Osteoma vs Exostosis — High-Yield Comparison

Feature	Osteoma vs Exostosis
Feature	Osteoma | Exostosis
Number	Solitary | Multiple
Laterality	Unilateral | Bilateral
Surface	Pedunculated | Broad-based (sessile)

Location	Near bony-cartilaginous junction / suture lines | Deep bony EAC
Association	No specific cause | Cold water swimming (surfer's ear)
Histology	Mature lamellar bone | Cancellous / lamellar bone
Treatment	Excision if symptomatic | Canalplasty if symptomatic
MCQ distinction	More medial

SECTION 3 | Etiology and Risk Factors

3.1 Risk Factors for SCC

- Chronic ultraviolet (UV) sun exposure — most important risk factor for pinna SCC.
- Chronic suppurative otitis media (CSOM) — longstanding ear infection; major risk for EAC SCC.
- Radiation exposure — prior radiotherapy to head and neck region.
- Chronic irritation and inflammation of EAC.
- Advanced age — SCC incidence rises sharply after 60 years.
- Immunosuppression — organ transplant recipients, HIV patients.
- Occupational UV exposure — farmers, outdoor workers.
- Previous actinic keratosis or Bowen disease.
- Arsenic exposure.

3.2 Risk Factors for BCC

- Chronic sun (UV) exposure — most important; BCC primarily arises on sun-exposed pinna skin.
- Fair skin, red/blonde hair, blue eyes (phototype I and II).
- Albinism.
- Gorlin-Goltz syndrome (basal cell nevus syndrome) — multiple BCCs from childhood; autosomal dominant; PTCH1 mutation.
- Ionizing radiation exposure.
- Immunosuppression.
- Xeroderma pigmentosum — defective DNA repair.

3.3 Risk Factors for Melanoma

- UV sun exposure and sunburn history.
- Family history of melanoma.
- Pre-existing dysplastic nevus or large congenital nevus.
- Fair skin, multiple nevi.
- BRAF mutation (somatic) — 40–60% of melanomas.
- CDKN2A, CDK4 mutations (familial).

3.4 Risk Factors for Ceruminous Gland Carcinoma

- No specific known cause.
- May arise from pre-existing ceruminous adenoma (benign precursor).
- Rare; may mimic benign lesions on clinical examination.

SUMMARY: Most Important Risk Factors

- SCC of pinna ? Chronic UV / sun exposure + Chronic CSOM (for EAC)
- BCC of pinna ? Chronic UV / sun exposure
- Osteoma ? No specific cause
- Exostosis ? Cold water swimming (surfer's ear)
- Keloid ? Ear piercing; more common in Black / dark-skinned individuals
- Keratosis obturans ? Chronic sinusitis and bronchiectasis (associated)

SECTION 4 | Pathology and Histopathology

4.1 Squamous Cell Carcinoma (SCC)

Gross Pathology

- Ulcerative or exophytic growth in the EAC or pinna.
- Indurated, hard edges with central necrosis or crusting.
- Bleeding on touch is common.

Histopathology — SCC

- Malignant squamous cells with intercellular bridges.
- Keratin pearls (concentric whorls of keratinized cells) — pathognomonic of well-differentiated SCC.
- Individual cell keratinization.
- Pleomorphism, nuclear hyperchromasia, abnormal mitoses.
- Grades: well-differentiated (keratin pearls present) ? poorly differentiated (no pearls, anaplastic).
- Perineural invasion and bone invasion indicate aggressive disease.

? IMPORTANT: Keratin pearls = most characteristic histopathological feature of well-differentiated SCC. HIGH-YIELD MCQ.

[DIAGRAM: Histopathology Slide — SCC: Keratin Pearls, Intercellular Bridges, Perineural Invasion]

Insert labeled diagram here

4.2 Basal Cell Carcinoma (BCC)

Gross Pathology

- Pearly, translucent nodule with central ulcer ("rodent ulcer") and rolled-in edges — classic.
- Telangiectatic vessels over the surface.
- Morphoeic BCC — flat, scar-like; ill-defined; most aggressive variant.

Histopathology — BCC

- Nests, cords, or islands of basaloid cells with peripheral palisading (picket-fence arrangement).
- Retraction artefact between tumour islands and stroma — classic feature.
- Mucinous stroma.
- No keratin pearls (unlike SCC).
- Low mitotic activity.

? IMPORTANT: Peripheral palisading of basaloid cells = hallmark of BCC. Retraction artefact from stroma = classic finding.

4.3 Melanoma

Gross Pathology

- Asymmetric pigmented lesion with irregular border, color variation, and diameter >6 mm (ABCDE rule).
- May be amelanotic — pigment-free; easily missed.

Histopathology — Melanoma

- Malignant melanocytes at dermo-epidermal junction spreading radially (radial growth phase) then vertically (vertical growth phase — poor prognosis).
- Pagetoid spread — single malignant melanocytes scattered throughout epidermis.
- Nuclear pleomorphism with prominent nucleoli.
- Breslow thickness — measured from granular cell layer to deepest tumour cell; most important prognostic factor.
- Clark level — anatomical depth of invasion (I–V).

4.4 Adenoid Cystic Carcinoma

- Arises from ceruminous (apocrine-like) glands of EAC.
- Histology: classic cribriform (Swiss-cheese) pattern — tumour cells surrounding cylindrical mucoid spaces.
- Also shows tubular and solid patterns.
- Perineural invasion is a hallmark and explains skip lesions and late recurrences.
- Distant metastases (lung) can occur decades after apparent cure — very important clinically.

? IMPORTANT: Cribriform (Swiss-cheese) pattern + perineural invasion = hallmark of adenoid cystic carcinoma. VERY COMMON MCQ.

4.5 Ceruminous Gland Carcinoma

- Rare malignant tumour of modified apocrine glands in the cartilaginous EAC.
- Subtypes: ceruminous adenocarcinoma, ceruminous adenoid cystic carcinoma, ceruminous mucoepidermoid carcinoma.
- Histology: glandular structures with nuclear atypia; decapitation secretion (apocrine snouts) may be seen.

4.6 Verrucous Carcinoma

- Low-grade variant of SCC; exophytic, cauliflower-like, highly keratinized.
- Histology: broad pushing (non-infiltrating) base of well-differentiated squamous cells — no keratin pearls.
- Minimal cytological atypia — may be mistaken for benign lesion on superficial biopsy.
- Locally destructive but rarely metastasizes.
- IMPORTANT: Superficial biopsy often insufficient — full-depth biopsy needed to demonstrate pushing base.

4.7 Merkel Cell Carcinoma

- Rare, aggressive neuroendocrine carcinoma of the skin.
- Histology: small round blue cells with scanty cytoplasm; paranuclear dot-like CK20 positivity on IHC (pathognomonic).
- IHC: CK20+ (perinuclear dot), synaptophysin+, chromogranin+, CD56+; CK7 negative.
- Associated with Merkel cell polyomavirus (MCPyV) and immunosuppression.

4.8 Key Histopathology Comparison Table

Feature	SCC	BCC	Adenoid Cyst

Origin	Squamous epithelium	Basal layer of epidermis	Ceruminous/EA
Key histology	Keratin pearls, intercellular bridges	Peripheral palisading, retraction artefact	Cribriform / Sw
Perineural invasion	Present (less characteristic)	Rare	HALLMARK –
Metastasis	Regional nodes (common)	Rare	Lung, liver (lat
Prognosis	Stage-dependent	Generally good	Prolonged cour

SECTION 5 | Pathways of Spread

Understanding spread is critical for surgical planning and staging. EAC carcinoma has multiple routes of extension.

PRIMARY TUMOUR (Pinna / EAC)

?

Local Soft Tissue Spread ? Cartilage / Skin

?

Bone Erosion ? Temporal Bone / Mastoid / Tegmen

?

Fissures of Santorini / Foramen of Huschke ? Parotid / TMJ / Infratemporal Fossa

?

Perineural Spread ? Skip Lesions / Facial Nerve / Greater Auricular Nerve

?

Anterior ? Internal Carotid Artery / Skull Base

?

Superior ? Middle Cranial Fossa / Dura / Brain

?

Posterior ? Sigmoid Sinus / Posterior Cranial Fossa

?

Lymphatic ? Parotid / Deep Cervical Nodes

?

Haematogenous ? Lung / Liver (especially Adenoid Cystic Ca)

5.1 Local Soft Tissue Spread

- Tumour spreads along the skin of the EAC and pinna initially.
- Invasion of perichondrium and cartilage follows.
- Soft tissue of the auricle may be entirely replaced in advanced tumours.

5.2 Bone Erosion

- EAC tumours invade the bony EAC early due to thin periosteum.
- Mastoid and tegmen invasion leads to intracranial spread.
- Cochlea and vestibule involvement causes sensorineural hearing loss.

5.3 Anterior Spread — Fissures of Santorini & Foramen of Huschke

EXAM FAVOURITE: Anterior Spread Routes

- Fissures of Santorini (cartilaginous EAC) ? Parotid gland spread
- Foramen of Huschke (bony EAC defect) ? TMJ / infratemporal fossa spread
- Both routes are clinically important for explaining seemingly unexpected parotid or TMJ involvement

5.4 Perineural Spread

- Especially characteristic of adenoid cystic carcinoma.
- Allows tumour to travel along nerve sheaths — explains skip lesions and wide surgical margins needed.
- Facial nerve involvement ? facial palsy (poor prognostic sign).
- Greater auricular nerve involvement ? spread to upper neck.

5.5 Lymphatic Spread

- Primary echelon nodes: preauricular (parotid), retroauricular (mastoid), and upper deep cervical nodes.
- Nodal involvement significantly worsens prognosis.
- EAC carcinoma: ~20% present with lymph node metastasis at diagnosis.

5.6 Intracranial Extension

- Via tegmen tympani ? middle cranial fossa and temporal lobe.
- Via sigmoid sinus ? posterior cranial fossa.
- Dural involvement ? meningitis, brain abscess, CSF leak.

- Cranial nerve involvement: V, VII, IX, X, XI, XII depending on extent.

[DIAGRAM: Pathways of Spread — EAC Carcinoma: Bone erosion, Perineural spread, Anterior/Posterior/Superior ex

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SECTION 6 | Clinical Features

6.1 Symptoms

Early / General Symptoms

- Otalgia (ear pain) — most common presenting symptom; may be severe and nocturnal.
- Otorrhoea — ear discharge; may be purulent or bloody.
- Bloody otorrhoea — very important sign; should always raise suspicion of malignancy in an adult.
- Hearing loss (conductive) — due to canal obstruction or ossicular involvement.
- Itching and persistent crusting in the EAC.
- Persistent granulation tissue in EAC that does not heal despite treatment — STRONG WARNING SIGN.

WARNING SIGNS — Suspect Malignancy When:

• Persistent granulation tissue in EAC unresponsive to treatment

• Bloody or blood-stained otorrhoea in adults without obvious trauma

• Otalgia disproportionate to clinical findings

• Rapidly growing lesion on pinna or in EAC

• Palpable pre/retroauricular lymph node with ear symptoms

? Any non-healing lesion of pinna or EAC >4 weeks = biopsy mandatory

Advanced Disease Symptoms

- Facial palsy (LMN) — involvement of facial nerve in the temporal bone; ominous sign (T4 disease).
- Trismus — invasion of temporomandibular joint or pterygoid muscles.
- Neck mass — lymph node metastasis (preauricular, upper cervical nodes).
- Headache, vomiting, diplopia — intracranial extension.
- Dysphagia, hoarseness — lower cranial nerve involvement (IX, X, XII).
- Sensorineural hearing loss — cochlear involvement.
- Weight loss and cachexia — advanced malignancy.

6.2 Signs on Examination

Pinna

- Ulcerated, indurated lesion with hard rolled edges — SCC.
- Pearly nodule with telangiectasia and central ulcer — BCC.
- Pigmented, asymmetric lesion — melanoma.
- Verrucous, cauliflower-like growth — verrucous carcinoma.

External Auditory Canal

- Otoscopy: narrowed canal with granulation, polypoid mass, or ulcerated lesion.
- Bleeding on probing.
- Tenderness on tragal pressure.
- Widened canal with keratin plug — keratosis obturans (benign).
- Hard sessile or pedunculated bony projection — osteoma or exostosis.

Lymph Nodes

- Preauricular nodes — enlarged, hard, fixed in SCC with nodal spread.
- Retroauricular (mastoid) nodes.
- Upper deep cervical nodes.

Neurological

- Facial nerve testing — Bell palsy pattern (LMN type) indicates advanced EAC tumour.
- Cranial nerves V–XII examination in extensive disease.

SECTION 7 | Differential Diagnosis

7.1 Important Differentials

Condition	Key Differentiating Features	Investigation to Co
Malignant otitis externa	Diabetic/immunocompromised patient; Pseudomonas; skull base osteomyelitis; osteolysis on CT; no mass lesion	Biopsy (to exclude SC scan

Chronic otitis externa	Diffuse canal inflammation; no discrete mass; responds to topical antibiotics; no bone erosion	Clinical; biopsy if nor
Keratosis obturans	Keratin accumulation; widened bony canal; severe pain; no ulceration; bilateral; associated sinusitis	Microscopy, CT
EAC cholesteatoma	White pearly keratin; bone erosion on CT; no mucosal bleeding; not ulcerated	CT temporal bone; bi
Granulation tissue (benign)	Chronic infection; responds to treatment; soft; no bone erosion	Biopsy if non-resolvi
Keratoacanthoma vs SCC	KA: rapid growth, central keratin plug, may self-involute; SCC: progressive, ulcerated, no self-regression	Full-depth biopsy + h

7.2 Benign vs Malignant Lesion — Clinical Comparison

Benign Lesion	Malignant Lesion
Growth rate	Slow / slow to moderate | Rapid, progressive
Edges	Regular, well-defined | Irregular, indurated, rolled edges
Ulceration	Absent (usually) | Present; may have central necrosis

Bleeding	Absent / minor | Spontaneous or on touch
Bone erosion	Absent | Present in advanced cases
Lymphadenopathy	Absent | May be present
Nerve involvement	Absent | Facial palsy in EAC carcinoma
Response to antibiotics	Possible (if inflammatory) | None

7.3 Causes of Bloody Otorrhoea — Differential

CAUSES OF BLOODY OTORRHOEA — EXAM LIST:

- Carcinoma of EAC / middle ear (must exclude in adults)
- Trauma — temporal bone fracture; foreign body
- Granulation tissue from CSOM
- Glomus tumour (pulsatile, red mass behind TM)
- Acute otitis media with TM perforation (early; serosanguinous)
- Malignant otitis externa (advanced)
- EAC myringitis bullosa (viral; bullae rupture)

? Painless bloody otorrhoea in adult without recent trauma = malignancy until proven otherwise

SECTION 8 | Investigations

8.1 Clinical Investigations

Otoscopy and Microscopic Ear Examination

- Mandatory first step.
- Use operating microscope for magnified view of EAC lesion.
- Document: size, site, surface, bleeding tendency, relationship to TM.

Biopsy

- Gold standard for diagnosis — tissue diagnosis essential before any treatment.
- Technique: punch biopsy or cup forceps biopsy from ulcer edge (avoid necrotic centre).
- Must include full-depth tissue for verrucous carcinoma to demonstrate pushing base.
- Frozen section — used intraoperatively to assess surgical margins.

? IMPORTANT: Never treat a suspected EAC malignancy without histopathological confirmation.

FNAC of Neck Nodes

- Fine needle aspiration cytology of suspicious lymph nodes.
- Confirms nodal metastasis — guides staging and management.

Audiological Assessment

- Pure tone audiometry (PTA) — assess type and degree of hearing loss.
- Conductive HL — EAC occlusion or ossicular involvement.
- Sensorineural HL — cochlear or internal auditory canal invasion.

8.2 Imaging

High-Resolution CT (HRCT) Temporal Bone

- Imaging modality of choice for bony involvement.
- Evaluates: bone erosion, EAC destruction, mastoid involvement, ossicular chain, tegmen, sigmoid sinus plate.
- Staging: identifies intracranial extension, TMJ, infratemporal fossa, carotid canal invasion.

MRI Temporal Bone

- Superior to CT for soft tissue extent.
- Best modality for: perineural invasion, dural involvement, intracranial extension, parotid involvement.
- T1 + gadolinium — perineural spread shows as enhancement along nerve sheaths.
- T2 — fluid-filled spaces, labyrinthine enhancement.

PET-CT (FDG-PET)

- Used for: nodal staging, distant metastasis detection, recurrence monitoring.
- Especially useful in ceruminous gland carcinomas and advanced SCC.

8.3 Metastatic Workup

- Chest X-ray / CT chest — lung metastases (common in adenoid cystic carcinoma).
- Liver function tests, abdominal ultrasound — liver metastases.
- Bone scan — skeletal metastases (skull base, vertebra).

8.4 Imaging Findings Summary

Modality	Best For	Key Findings in EAC Carcinoma
HRCT Temporal Bone	Bone erosion, EAC destruction, staging	Irregular bone erosion, loss of EAC w opacification, tegmen destruction
MRI Temporal Bone	Soft tissue, perineural, dural spread	T1+Gd enhancement along nerves; du parotid mass; intracranial disease
PET-CT	Nodal/distant metastasis, recurrence	Hypermetabolic nodes/distant sites; su treatment

[DIAGRAM: Radiology Figures — HRCT: Bone Erosion in EAC SCC | MRI: Perineural Spread & Dural Involvem

Insert labeled diagram here

SECTION 9 | Staging of External Ear Tumours

9.1 Pittsburgh Staging System for EAC Carcinoma

The Pittsburgh staging system is the most widely used for carcinoma of the EAC and temporal bone.

PITTSBURGH STAGING — EAC CARCINOMA:

T1 — Tumour limited to EAC; no bone erosion; no soft tissue involvement

T2 — Tumour with limited bone erosion of EAC (not full thickness)

OR tumour with limited (<0.5 cm) soft tissue involvement

T3 — Tumour erodes full thickness of bony EAC; limited soft tissue (<0.5 cm)

OR tumour involving middle ear / mastoid

T4 — Tumour erodes cochlea, petrous apex, medial tympanic wall, carotid canal,

jugular foramen, dura; OR extensive (>0.5 cm) soft tissue involvement

OR facial nerve involvement

N0 — No regional node metastasis

N1 — Single ipsilateral node involvement

Stages: T1N0=I | T2N0=II | T3N0 or T1-2N1=III | T4N0 or any N1=IV

? MCQ PEARL: Facial nerve palsy = T4 disease in Pittsburgh staging. Worst prognostic indicator.

9.2 TNM Staging — Pinna Tumours

Pinna tumours are staged according to AJCC TNM classification for cutaneous malignancies.

- T1 — Tumour ?2 cm; no high-risk features.
- T2 — Tumour >2 cm but ?4 cm; or any size with 1 high-risk feature.
- T3 — Tumour >4 cm; or involvement of maxilla, mandible, orbit, temporal bone; or any with perineural invasion of named nerves.
- T4 — Tumour with cortical bone invasion, base of skull, foraminal invasion, extensive CNS invasion.

HIGH-RISK FEATURES for Pinna SCC (AJCC):

• Thickness >2 mm or Clark level ?IV

• Perineural invasion

• Poorly differentiated or undifferentiated

• Location: ear or hair-bearing lip

9.3 Melanoma Staging (Breslow Thickness)

Stage	Breslow Thickness	Significance
T1a	< 0.8 mm; no ulceration	Best prognosis; >95% 5-year survival
T1b	< 0.8 mm with ulceration or 0.8–1.0 mm	Intermediate

T2	1.0–2.0 mm	Moderate risk
T3	2.0–4.0 mm	High risk
T4	> 4.0 mm	Very high risk

SECTION 10 | Treatment

10.1 Surgical Treatment — Pinna Tumours

Local Excision

- Excision with adequate surgical margins (5–10 mm) — treatment of choice for small pinna SCC and BCC.
- Mohs micrographic surgery — staged excision with intraoperative margin control; highest cure rate for BCC and SCC; ideal for critical anatomical locations (near EAC, tragus, antihelix).

Wide Local Excision

- Margins of 1–2 cm for SCC; 3–5 mm for BCC in non-Mohs settings.
- May require cartilage resection and auricular reconstruction.

Reconstruction

- Primary closure — small defects.
- Skin grafts — split-thickness or full-thickness.
- Local flaps — advancement, rotation, transposition flaps.
- Postauricular flap — for helical and antihelical defects.
- Prosthetic ear — for total auriculectomy.

10.2 Surgical Treatment — EAC Tumours

Sleeve Resection

- Removal of EAC skin only (with soft tissue sleeve).
- Indicated for T1 tumours confined to EAC skin without bone erosion.

Lateral Temporal Bone Resection (LTBR)

- Resects: EAC + tympanic membrane + ossicular chain + lateral tympanic cavity.
- Preserves: cochlea, inner ear, carotid artery, jugular bulb, facial nerve.
- Indications: T1–T2 EAC carcinoma (limited bone involvement).

Radical Temporal Bone Resection (RTBR)

- Resects all structures of LTBR + middle ear + mastoid.
- May include facial nerve sacrifice, parotidectomy, neck dissection.
- Indications: T3 EAC carcinoma with middle ear/mastoid involvement.

En Bloc Temporal Bone Resection

- Total removal of the temporal bone as a single specimen.
- Includes petrous apex, internal auditory canal.
- High morbidity; indicated for T4 disease with carotid or extensive skull base involvement.

Parotidectomy

- Superficial parotidectomy — for parotid nodal involvement or anterior EAC tumour.
- Total parotidectomy — if deep lobe involved.

Neck Dissection

- Selective neck dissection (levels II–IV) — for clinically positive nodes.
- May be elective in T3–T4 disease due to high occult node rate (20–30%).

Facial Nerve Management

FACIAL NERVE DECISION IN EAC CARCINOMA:

• If facial nerve functioning preoperatively AND no gross invasion ? Preserve + adjuvant RT

• If facial palsy preoperatively (T4) OR direct invasion at surgery ? Sacrifice + cable graft (great auricular or sura nerve)

• Post-sacrifice reconstruction: gold weight for eyelid closure, tarsorrhaphy, nerve cable graft or reanimation

? Sacrifice of functioning facial nerve without direct invasion is controversial

10.3 Types of Temporal Bone Resection — Summary Table

Procedure	Structures Removed	Indication	Facial Nerv
Sleeve resection	EAC skin + soft tissue	T1 (skin only)	Preserved
Lateral TBR	EAC + TM + ossicles + lateral tympanic cavity	T1–T2	Preserved
Radical TBR	LTBR + middle ear + mastoid	T3	Preserved if

En Bloc TBR	Entire temporal bone	T4 (extensive)	Usually sacr

10.4 Radiotherapy

Indications

- Adjuvant (postoperative) radiotherapy — for all T3–T4 disease; positive or close surgical margins; perineural invasion; nodal involvement.
- Palliative radiotherapy — unresectable disease; symptom control.
- Primary radiotherapy — small BCC or SCC in patients unfit for surgery.

Techniques

- External beam radiotherapy (EBRT): conventional or IMRT (intensity modulated).
- Dose: typically 60–66 Gy in adjuvant setting.

10.5 Chemotherapy

- Cisplatin-based concurrent chemoradiation — for unresectable disease or advanced N+ disease.
- Cetuximab (anti-EGFR) — for platinum-refractory or EGFR-overexpressing SCC.
- Immunotherapy (PD-L1 inhibitors: pembrolizumab, nivolumab) — for recurrent/metastatic cutaneous SCC.
- Dacarbazine, temozolomide, ipilimumab + nivolumab — for advanced melanoma.

10.6 Treatment by Tumour Type

Tumour Type	Primary Treatment	Adjuvant / Notes
BCC (small)	Wide local excision or Mohs surgery	RT if margins positive; topical imi superficial BCC
SCC (T1–T2)	Lateral temporal bone resection + wide excision of pinna	Adjuvant RT if close margins / per
SCC (T3–T4)	Radical/En bloc temporal bone resection + parotidectomy + ND	Adjuvant chemoradiotherapy
Melanoma	Wide local excision + sentinel node biopsy	Immunotherapy / targeted therapy disease
Adenoid cystic Ca	Wide resection with perineural clearance	Adjuvant RT mandatory; long-tern late recurrence
Keratoacanthoma	Excision (may observe if classic appearance)	Histology confirmation essential; t uncertain

[DIAGRAM: Surgical Diagrams — Sleeve Resection, Lateral TBR, Radical TBR, En Bloc TBR, Parotidectomy & Fre Reconstruction]

Insert labeled diagram here

SECTION 11 | Complications

11.1 Complications of the Disease

Local / Regional

- Facial nerve palsy (LMN) — involvement of facial nerve in temporal bone; causes: facial asymmetry, lagophthalmos, Bell phenomenon.
- Temporal bone destruction — loss of hearing, labyrinthine damage ? vertigo, SNHL.
- TMJ involvement ? trismus, ankylosis.
- Parotid gland invasion ? parotid mass, parotidectomy required.

Intracranial

- Dural involvement ? CSF leak, meningitis.
- Epidural or brain abscess.
- Encephalitis — direct spread.
- Cranial nerve palsies (V, IX, X, XI, XII) — extensive skull base invasion.
- Lateral sinus (sigmoid sinus) thrombosis.

Systemic

- Distant metastases: lung (most common), liver, bone, brain.
- Especially frequent in adenoid cystic carcinoma (lung; decades later).

11.2 Complications of Surgery

- Facial nerve palsy — intentional (sacrifice) or inadvertent injury.
- CSF leak — dural entry during temporal bone resection.
- Wound infection and flap necrosis.
- Haematoma.
- Hearing loss (total) — cochlear sacrifice.
- Balance disturbance — labyrinthine removal.
- Internal carotid artery injury — catastrophic; preoperative carotid balloon occlusion testing recommended.

11.3 Complications of Radiotherapy

- Osteoradionecrosis of temporal bone — chronic non-healing bone necrosis.
- Radiation-induced otitis media / otitis externa.
- Sensorineural hearing loss (cochlear damage).
- Xerostomia (if parotid in field).
- Secondary malignancy (rare).

SECTION 12 | Prognosis

12.1 Prognostic Factors

Most Important Adverse Prognostic Factors

- Facial nerve palsy at presentation — T4 disease; worst prognostic sign.
- Positive surgical margins — strongly predicts recurrence.
- Lymph node metastasis — significantly worsens survival.
- Intracranial extension — very poor prognosis.

- Perineural invasion — predicts local recurrence and need for wider margins.
- Tumour grade — poorly differentiated tumours have worse outcomes.
- T-stage at presentation — T1/T2 have good cure rates; T4 has poor survival.

12.2 Survival by Stage — EAC Carcinoma

Stage (Pittsburgh)	Approximate 5-Year Survival	Notes
T1 (Stage I)	70–100%	Excellent outcome with complete e
T2 (Stage II)	50–70%	Good outcome with lateral tempor
T3 (Stage III)	30–50%	Radical surgery + RT required
T4 (Stage IV)	<20%	Facial palsy, intracranial spread; p

12.3 Prognosis by Tumour Type

- BCC — excellent; rarely fatal; <1% distant metastasis rate.
- SCC — stage-dependent; early disease curable; advanced T4 disease: <20% survival.
- Melanoma — Breslow thickness most important; thin (<0.8 mm) lesions: >95% survival; thick/nodal disease: <50%.
- Adenoid cystic carcinoma — prolonged course; late recurrences (even at 10–20 years); lung metastasis common.
- Merkel cell carcinoma — aggressive; 5-year survival ~40–60% overall; worse with nodal involvement.

12.4 Recurrence Patterns

- Local recurrence — most common mode of failure; especially at surgical margins.
- Regional recurrence — parotid, cervical nodes.
- Intracranial recurrence — very poor prognosis; usually unresectable.
- Distant recurrence — adenoid cystic carcinoma: lung (late); SCC: uncommon.
- Follow-up: 3-monthly for first 2 years, 6-monthly for years 3–5, annually thereafter.

SECTION 13 | High-Yield Exam Pearls

? ANATOMY PEARLS

• Most common benign bony tumour of EAC: Exostosis (associated with cold water swimming)

• Osteoma: solitary, pedunculated, unilateral — near suture line

• Exostosis: multiple, sessile, bilateral — cold water swimming (surfer's ear)

• Fissures of Santorini: allow EAC tumour spread to PAROTID

• Foramen of Huschke: allows spread to TMJ and infratemporal fossa

• Preauricular lymph nodes: drain pinna (anterior) and EAC ? first echelon for pinna SCC

? CLASSIFICATION / DIAGNOSIS PEARLS

• Most common malignancy of EAC: SCC

• Most common malignancy of pinna: BCC

• Most common premalignant lesion of pinna: Actinic keratosis

• Most common benign lesion of EAC: Osteoma / Exostosis

• Most common benign glandular tumour of EAC: Ceruminous gland adenoma

• Keratoacanthoma vs SCC: KA self-involutes; SCC does not — biopsy essential

• Bowen disease = SCC in situ (full-thickness atypia; basement membrane intact)

• Persistent granulation tissue in EAC unresponsive to treatment = biopsy immediately

? HISTOPATHOLOGY PEARLS

• Keratin pearls = well-differentiated SCC

• Peripheral palisading + retraction from stroma = BCC

• Cribriform (Swiss-cheese) pattern + perineural invasion = Adenoid cystic carcinoma

• Perinuclear CK20 dot + synaptophysin + chromogranin = Merkel cell carcinoma

• Breslow thickness = most important prognostic factor in melanoma

• Pagetoid spread = melanoma in radial growth phase

• Verrucous carcinoma: pushing base (not infiltrating); minimal cytological atypia

• Deep biopsy essential for verrucous carcinoma — superficial biopsy misses diagnosis

? STAGING / SURGICAL PEARLS

• Pittsburgh staging used for EAC carcinoma

• Facial palsy = T4 disease = worst prognostic indicator in EAC carcinoma

• Lateral temporal bone resection: T1–T2 EAC tumours

• Radical temporal bone resection: T3 EAC tumours

• En bloc temporal bone resection: T4 (extensive skull base) — rare

• Mohs surgery: highest cure rate for BCC and SCC; best for peri-auricular lesions

• Adenoid cystic carcinoma: perineural invasion ? wide margins + mandatory adjuvant RT

• EAC carcinoma: wide excision alone insufficient — temporal bone resection needed

? CLINICAL / MCQ PEARLS

• Surfer's ear = exostosis = multiple bilateral broad-based bony EAC swellings

• Cauliflower ear = haematoma auris complication (perichondritis ? fibrosis)

• Preauricular sinus infection: organism = Staphylococcus aureus; treatment = excision

• Keloid after ear piercing: more common in dark-skinned individuals

• Keratosis obturans: widened canal + keratin plug + associated with sinusitis/bronchiectasis

• Gorlin-Goltz syndrome: multiple BCCs + odontogenic keratocysts + rib/skeletal anomalies

• Merkel cell carcinoma: MCPyV + immunosuppression; CK20 perinuclear dot pattern

• Bloody otorrhoea in adult = rule out malignancy first

• Adenoid cystic carcinoma: late lung metastasis even 10–20 years post-treatment

• COWS mnemonic: Cold ? Opposite; Warm ? Same (for caloric test nystagmus direction)

13.1 Important Viva Questions

1. **What is the most common malignancy of the EAC?**

- SCC is the most common malignancy of the EAC. BCC is the most common malignancy of the pinna skin.

2. **What are the pathways of spread of EAC carcinoma?**

- Local (bone erosion), anterior (Fissures of Santorini ? parotid; Foramen of Huschke ? TMJ/IFR), perineural, lymphatic (preauricular nodes), intracranial (tegmen ? MCA fossa).

3. **What is Pittsburgh staging? What is T4 disease?**

- Pittsburgh T4 = tumour eroding cochlea, petrous apex, medial tympanic wall, carotid canal, jugular foramen, dura, OR extensive soft tissue involvement (>0.5 cm) OR facial nerve involvement.

4. **What is the difference between osteoma and exostosis?**

- Osteoma: solitary, pedunculated, unilateral, near suture lines. Exostosis: multiple, bilateral, broad-based, cold water swimming association.

5. **What is Mohs surgery? When is it used?**

- Staged excision with intraoperative frozen section margin control. Best for BCC/SCC of pinna and EAC — ensures complete removal while preserving maximum normal tissue.

6. **What is the histopathological hallmark of adenoid cystic carcinoma?**

- Cribriform (Swiss-cheese) pattern with perineural invasion. Late distant (lung) metastases are characteristic.

7. **Why is facial nerve palsy a poor prognostic sign in EAC carcinoma?**

- Indicates T4 disease with temporal bone involvement up to geniculate ganglion region. Only ~20% 5-year survival. Requires radical resection.

8. **What is the management of EAC carcinoma?**

- T1: Sleeve/lateral temporal bone resection. T2: Lateral TBR + parotidectomy. T3: Radical TBR + parotidectomy + ND. T4: En bloc resection + chemoradiotherapy. All advanced stages require adjuvant RT.

SECTION 14 | Important Histopathology Slides & Clinical Photographs

14.1 Histopathology Slides — Key Findings

Tumour	Histopathology Slide Finding	Examination Tip
SCC — Well-differentiated	Keratin pearls (concentric whorls of keratinized cells) + intercellular bridges + nuclear pleomorphism	MOST COMMON MCQ slide i
SCC — Perineural invasion	Tumour cells in and around nerve sheaths; nerve cross-sections surrounded by malignant cells	Important for adverse prognosis
BCC	Basaloid cell nests with peripheral palisading; peri-tumoral retraction artefact from stroma; mucinous stroma	Peripheral palisading = pathogn
Adenoid cystic carcinoma	Cribriform (Swiss-cheese) pattern: cylindrical mucoid spaces surrounded by tumour cells; also tubular/solid patterns	CRIBRIFORM = hallmark. Peri mandatory to mention
Melanoma	Malignant melanocytes at dermo-epidermal junction; pagetoid spread; melanin granules; mitoses; vertical growth phase	Breslow thickness = most impor measurement

Ceruminous gland carcinoma	Glandular/tubular structures with nuclear atypia; decapitation (apocrine) secretion; invasion into surrounding tissue	May resemble benign ceruminou small biopsy
Verrucous carcinoma	Exophytic, highly keratinized; broad pushing (non-infiltrating) base; minimal cytological atypia; no keratin pearls	Requires deep biopsy to demons base
Merkel cell carcinoma	Small round blue cells, scanty cytoplasm; paranuclear dot of CK20 on IHC; synaptophysin+; chromogranin+	CK20 perinuclear dot = pathogn
Bone invasion	Tumour cells permeating between bony trabeculae; reactive new bone; osteoclastic resorption at tumour-bone interface	Important to report in temporal l specimens

14.2 Clinical Photographs — Recognised Appearances

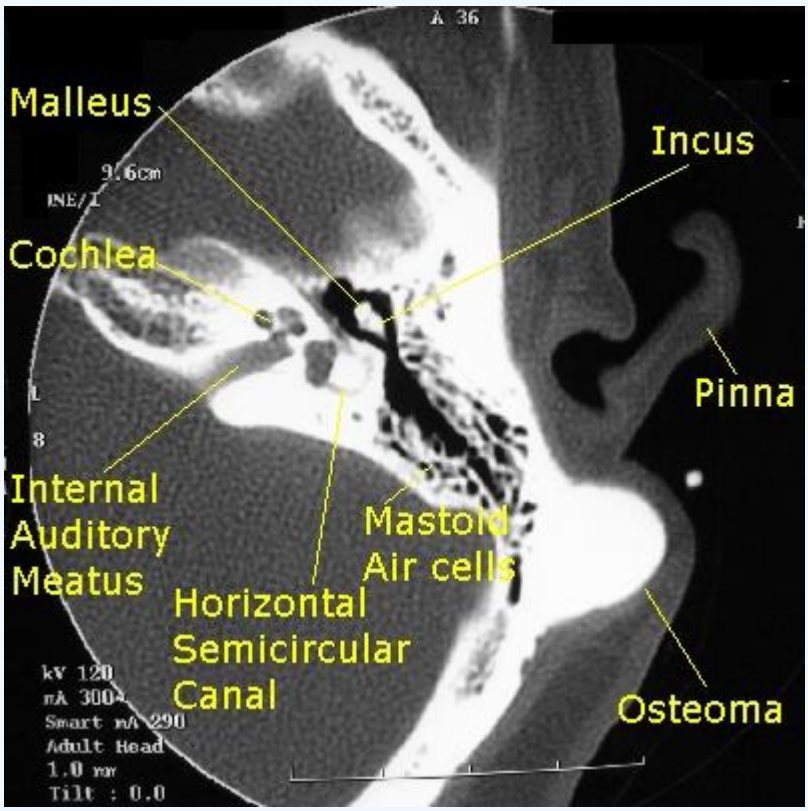
Lesion	Classic Clinical Appearance	Key Exam Point
SCC of pinna	Indurated ulcer with hard rolled edges; bleeding; helix most common site	Most common pinna malignancy EAC; most common overall = B
EAC carcinoma	Granulation tissue or polypoid mass filling EAC; bleeds on touch; associated otalgia	Biopsy any non-healing EAC m

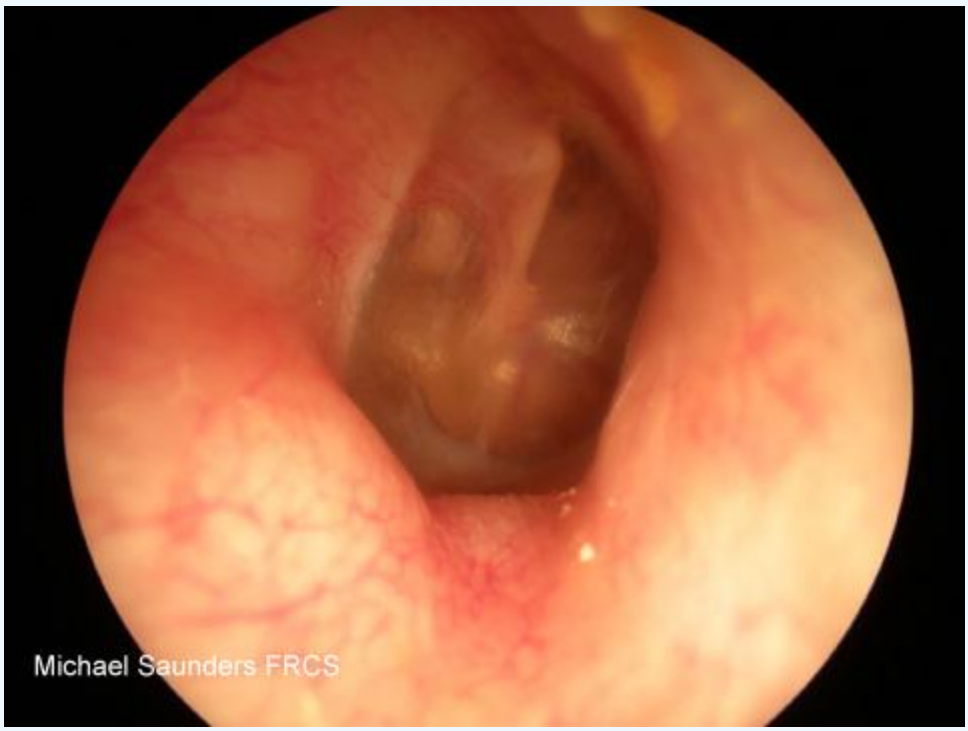
BCC of ear	Pearly translucent papule with central ulcer (rodent ulcer); telangiectasia; rolled edges	Most common malignancy of pi
Melanoma of pinna	Asymmetric pigmented lesion; irregular border; variegated color; >6 mm	ABCDE rule: Asymmetry, Bord Diameter, Evolution
Osteoma	Firm, smooth, sessile or pedunculated hard swelling at bony EAC — near TM; unilateral	Distinguish from exostosis (bila swimming history)
Exostosis	Multiple hard nodular protrusions in deep bony EAC; bilateral; narrows canal	Cold water swimmers; bilateral; symptomatic
Ear keloid	Firm, rubbery, shiny, overgrown scar tissue at lobule; post-piercing; extends beyond wound boundary	Keloid vs hypertrophic scar: kel beyond wound
Keratosis obturans	Widened bony EAC filled with hard keratin plug; extremely painful; bilateral	Associated with sinusitis + bron differs from EAC cholesteatoma aggressive bone erosion)

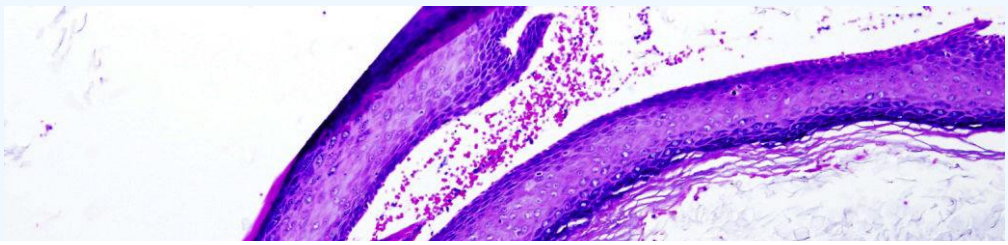
[DIAGRAM: Histopathology Slides: SCC Keratin Pearls | BCC Peripheral Palisading | Adenoid Cystic Cribriform Pa Merkel Cell CK20 Dot]

Insert labeled diagram here

- Osteoma EAC
- Exostosis EAC
- Keratosis Obturans







End of Chapter — Tumours of the External Ear

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