

Primary External Auditory Canal Cholesteatoma: A Case Series of Diagnostic Delay and Stage-Directed Management

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Abstract

Background: Primary external auditory canal cholesteatoma (EACC) is uncommon and is regularly labelled as otitis externa or keratosis obturans, so diagnosis is often delayed.

Case Series. We describe a series of Five adults (three women; age 30–70 years) with primary EACC are described. Four had He stage II disease confined to the canal with an intact tympanic membrane (one bilateral). One had stage III disease with middle-ear soft tissue and scutum erosion. All five had attended several practitioners for presumed external otitis or wax. Two ears were treated by canalplasty and meatoplasty (one combined with modified radical mastoidectomy and type III tympanoplasty). Three stage II ears were cleared by scheduled microscopic debridement and topical ointment. At 3–6 months the operated stage II canal had healed and conservatively treated ears were dry, without clinical progression.

Conclusion. Persistent otalgia after treatment of otitis externa warrants microscopy after toilet and HRCT. Selected stage II pockets with an intact drum can be controlled by disciplined outpatient cleaning; canalplasty or mastoidectomy is required when follow-up is unreliable or disease has left the canal.

Keywords: Cholesteatoma; Ear Canal; Otologic Surgical Procedures.

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Introduction

Cholesteatoma is a locally destructive accumulation of keratinizing squamous epithelium. It is familiar in the middle ear and mastoid, but the same process may arise in the external auditory canal (EAC) [1–3]. Primary EACC is uncommon: older clinic series estimated roughly one to seven cases per 1,000 new ear consultations, whereas population rates are closer to 0.15–0.30 per 100,000 per year [2,3].

Secondary EACC follows surgery, trauma, or chronic canal inflammation [1,3,5]. Otalgia, otorrhoea, fullness, and conductive hearing loss are the usual complaints [3,4]. These symptoms are shared with otitis externa and with keratosis

obturans, so patients often receive repeated topical therapy or wax removal before bone erosion is recognized. Diagnosis requires demonstration of focal keratin over exposed or eroded canal bone, confirmed on high-resolution computed tomography (HRCT) of the temporal bone. Keratosis obturans produces a circumferential plug and canal widening without osteitis and is managed by debridement alone; EACC shows focal osteitis and may need canalplasty or mastoidectomy [5–7].

He's classification separates canal disease without bony change (stage I), canal-limited erosion (stage II), extension into the mastoid or tympanic cavity but still within the temporal bone (stage III,

substages A–C), and extra-temporal or complicated disease (stage IV), and links each stage to a treatment strategy [8]. We report five consecutive primary EACC patients to illustrate the diagnostic delay that remains typical of this condition and to show that management can be individualized by stage, tympanic-membrane status, and the reliability of follow-up.

Case Series: This retrospective consecutive series was compiled from the otology outpatient clinic of a tertiary teaching hospital. Primary EACC was defined as keratin debris plus focal bony canal erosion on otoendoscopy after toilet, corroborated by HRCT, in the absence of prior ear surgery or trauma. Disease was staged by He [8]. Outcome was a dry, easily toilettable canal without residual keratin at the last visit (3–6 months).

Case 1: A 48-year-old woman had six years of intermittent right otalgia and hearing loss. Early foul otorrhoea had settled, but discomfort persisted despite repeated treatment by general practitioners.

Otoendoscopy showed impacted keratin, exposed canal bone, and scanty foul mucopus; the tympanic membrane was intact. HRCT demonstrated floor erosion of the bony EAC.

The left ear showed the same keratin and floor erosion. Pure-tone audiometry (PTA) recorded mild bilateral conductive loss (He stage II both sides). Right canalplasty and meatoplasty were performed through a post-auricular incision with posterior meatotomy. The cavity was healed at three months. The left ear remains on scheduled cleaning.

Case 2: A 30-year-old woman reported two years of right otalgia with intermittent hearing loss and many visits for ear cleaning. Otoendoscopy showed keratin and posterior canal-wall erosion with an intact drum. HRCT confirmed isolated posterior-wall erosion. The left ear was normal. PTA showed mild right conductive loss (He stage II).

Canalplasty was offered; she chose conservative care. Fortnightly clinic debridement and topical neomycin-polymyxin ointment cleared the pocket by three months. Monthly surveillance cleaning continues.

Case 3: A 35-year-old man had three years of right otalgia and fullness treated as otitis externa. After toilet, keratin and posterior-wall erosion were visible; the drum was intact. HRCT showed isolated posterior-wall erosion. Left hearing was normal; the right ear had mild conductive loss (He stage II). He declined canalplasty. Microscopic cleaning every two weeks for two months, with ointment after each sitting, produced a dry, disease-free canal at three months.

Case 4: A 50-year-old woman presented with four years of bilateral hearing loss and earache. Both canals were widened and held keratin and pus. After toilet, grade II pars tensa retraction was seen on both sides. PTA showed mild mixed loss bilaterally. HRCT demonstrated bilateral posterior canal-wall erosion, right middle-ear soft tissue, and scutum erosion; the ossicles, facial canal, and inner ear appeared intact (He stage III). Right modified radical mastoidectomy with type III tympanoplasty and meatoplasty was performed. Postoperative audiometry and cavity status were not available in the source record at the time of writing.

Case 5: A 70-year-old man had about one year of left otalgia and discharge after several outside consultations. The tympanic membrane was intact; suctioning exposed posteroinferior canal-wall erosion. HRCT confirmed posterior-wall and floor defects without mastoid extension. PTA showed a 40 dB left conductive loss (He stage II). Canalplasty was deferred because of age-related comorbidity. Regular microscopic cleaning over six months produced a dry ear without clinical progression.

Table 1: Clinical profile, He stage, treatment, and short-term outcome

Case	Age/sex	Side	Duration	Clinical findings	HRCT	He stage	Treatment / outcome
1	48 F	Bilateral	6 y	Keratin, exposed bone; TM intact; mild CHL both ears	Floor erosion both EACs	II / II	R canalplasty + meatoplasty, healed 3 mo; L toilet
2	30 F	Right	2 y	Keratin + posterior wall erosion; TM intact; mild R CHL	Posterior wall erosion; TM intact	II	Declined surgery; fortnightly toilet — clear at 3 mo
3	35 M	Right	3 y	Treated as OE; keratin + posterior erosion; TM intact	Posterior wall erosion; TM intact	II	2-weekly microscopic toilet × 2 mo — dry at 3 mo
4	50 F	Bilateral	4 y	Widened canals, keratin, pus; grade II retraction; mixed HL	Bilateral posterior erosion; R ME soft tissue + scutum	III	R MRM + type III tympanoplasty + meatoplasty
5	70 M	Left	~1 y	Posteroinferior erosion after suction; TM intact; 40 dB CHL	Posterior wall and floor defect; no mastoid extension	II	Conservative (comorbidity) — dry, no progression at 6 mo

CHL conductive hearing loss; **EAC** external auditory canal; **HL** hearing loss; **ME** middle ear; **MRM** modified radical mastoidectomy; **OE** otitis externa; **TM** tympanic membrane.

Fig. 1 Otoendoscopic view and HRCT of a representative stage II canal (floor or posterior-wall erosion, intact tympanic membrane)—insert labelled images from cases 1 and 2. Original draft call-outs both read “Figure 2” and must be renumbered when files are uploaded (TIFF, ≥ 300 dpi).



Figure 1: otoendoscopic image showing inferior and posterior canal wall erosion

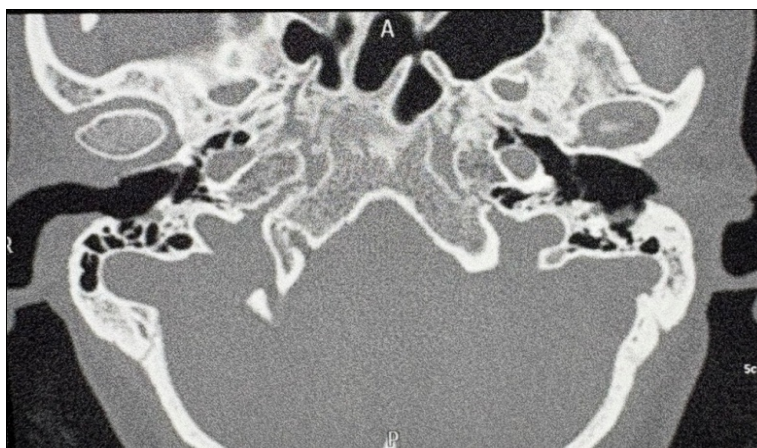


Figure 2: HRCT Temporal bone suggesting inferior wall erosion



Figure 3: HRCT Temporal bone coronal section showing cholesteatoma related inferior EAC wall erosion

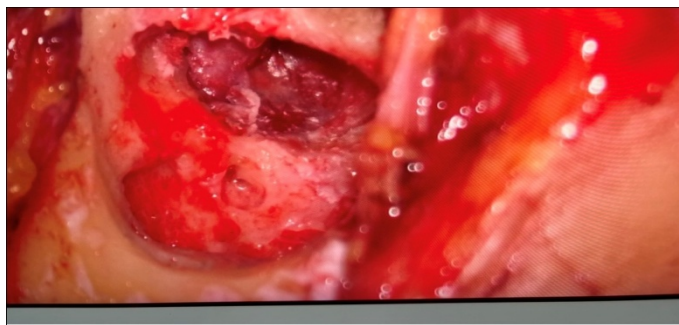


Figure 4: Intraoperative image showing extent of cavity

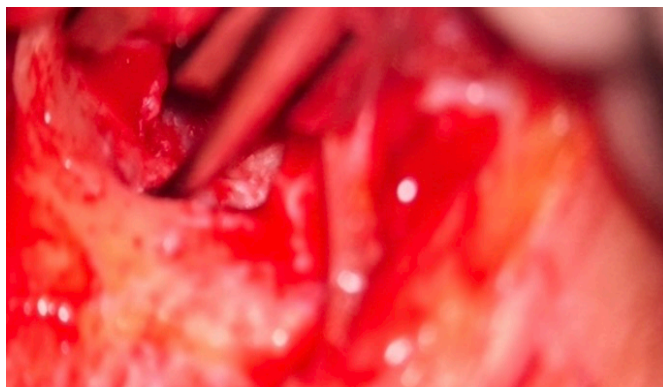


Figure 5: Intraoperative image showing posterior wall erosion in case of EAC cholesteatoma

Discussion

Primary EACC is attributed to localized osteitis after minor canal-skin injury, or to failed lateral epithelial migration with drier, adherent wax that traps keratin [3,5]. None of our patients had a surgical or traumatic antecedent.

Case 4 also showed pars tensa retraction and middle-ear soft tissue and may represent concurrent attic disease rather than purely canal-origin cholesteatoma; both pathways are treated by complete matrix removal and a safe cavity.

The educational point of this series is diagnostic delay. All five patients had multiple consultations for “otitis externa” or wax before erosion was recognized.

Keratosis obturans remains the main structural mimic: Naiberg et al. described circumferential distension and a tight plug without osteitis, in contrast to the focal erosion of EACC [6]. Maharjan and Rayamajhi warned that misclassification postpones surgery in aggressive canal disease [7]. Persistent otalgia after appropriate topical therapy should prompt microscopy after careful toilet and, if bone is exposed, HRCT. Treatment follows extent and the reliability of surveillance. The aim is a self-cleaning or easily toilettable canal so that keratin does not re-accumulate.

He stage II lesions with an intact drum can be offered either canalplasty or a documented debridement schedule; cases 2, 3, and 5 show that

the conservative limb can clear a shallow pocket when the patient attends.

It is not a substitute for canalplasty if the defect is deep, cleaning is painful or incomplete, or follow-up cannot be guaranteed. Recurrence after transcanal removal alone has been reported in stage II disease [8]. Stage III disease, as in case 4, requires mastoidectomy with reconstruction as indicated.

Facial palsy, labyrinthine fistula, mastoid abscess, and ossicular erosion are uncommon in isolated EACC but are the reason early recognition matters [9,10]. None occurred here, consistent with the predominance of stage II disease.

Limitations are those of a five-patient retrospective series: follow-up of only 3–6 months. Conservative “success” should be read as short-term control. Prospective registration of consecutive EACC with predefined imaging, histology, staging, and 12-month endoscopic review would test the algorithm suggested here.

Conclusion

Primary EACC is rare but regularly mistaken for otitis externa or keratosis obturans. Otoendoscopy after meticulous toilet plus HRCT establishes the diagnosis and assigns a He stage.

In selected stage II ears with an intact tympanic membrane, scheduled microscopic debridement can control disease; canalplasty, and for stage III disease mastoidectomy with tympanoplasty, remain

definitive when erosion is deeper or surveillance is impractical.

The teaching pearl is simple: bone exposed in the canal is not “just otitis externa”.

Key Messages

- Persistent otalgia or otorrhoea after treatment of otitis externa should trigger microscopy after toilet, not another course of drops alone.
- Focal bony erosion on otoendoscopy and HRCT distinguishes EACC from keratosis obturans and maps the walls involved.
- He staging directs care: scheduled debridement may suffice for shallow stage II pockets with an intact drum and reliable follow-up; canalplasty or mastoidectomy is required for deeper or extra-canal disease.
- Age and comorbidity justify a conservative first path only when the tympanic membrane is intact and the patient will attend for cleaning.

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