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Attic Reconstruction After Cholesteatoma Surgery: Bone Versus Cartilage

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Abstract: *Cholesteatoma is a destructive, keratinizing epithelial lesion frequently associated with chronic suppurative otitis media (CSOM), leading to bone erosion and complications such as ossicular damage and labyrinthine fistula. Surgical intervention remains the definitive treatment, with goals centered on complete disease eradication, prevention of recurrence, restoration of hearing, and creation of a dry, self-cleansing cavity. Canal wall up (CWU) and canal wall down (CWD) techniques each have advantages and limitations, often influenced by anatomical and disease-related factors. Reconstruction of the attic wall plays a vital role in surgical success, particularly in cases involving epitympanic cholesteatoma. Materials for attic reconstruction commonly include autologous bone and cartilage, each offering distinct benefits. Cortical bone provides mechanical strength and reliable osseointegration but may be limited by donor site morbidity and risk of graft resorption. In contrast, cartilage is flexible, durable, and metabolically stable, promoting healing and epithelialization, though it lacks osteogenic properties and may mask residual disease.*
Keywords: *Attic reconstruction, Cholesteatoma surgery, Cartilage graft, Bone graft, Middle ear repair*

1. INTRODUCTION

Chronic suppurative otitis media (CSOM) affects all demographics and can be safe or dangerous, with the latter commonly involving cholesteatoma and significant consequences (Bhutta et al., 2024). Due to its aggressive erosion of temporal bone structures, cholesteatoma is a destructive, keratin-filled cyst detected clinically and radiologically (Anne et al., 2024). The intact canal wall (ICW) and canal wall down (CWD) are surgical options. Canal wall down clears disease but sacrifices hearing and cavity maintenance (Bhat et al., 2021). Intact canal walls preserve hearing but increases recurrence. Canal wall reconstruction (CWR) mixes both methods (Swain et al., 2024).

When the outer attic walls (OAW) is lost, disease recurrence rate increase. Robinson found that scutumplasty during canal wall-up mastoidectomy reduced retraction rates from 15% to 5% (Robinson & Surgery, 1997). Scutumplasty grafts should be biocompatible, stable, accessible, and infection-resistant. Autologous bone, cartilage, titanium, ceramics, and hydroxyapatite are common. Cortical bone is strong and integrative, but harvesting it near diseased regions may increase infection and resorption risks (Vasile et al., 2024). The flexibility and durability of cartilage are preferable, but it lacks regeneration potential and may not be suited for revision cases owing to limited availability or fixation failure, which can cause recurrence (Su et al., 2024).

Anatomy of the Ear

The external, middle, and inner ear have different hearing and balancing functions. The pinna, external auditory canal, and tympanic membrane (TM) gather and transfer sound waves inward (Önerci et al., 2021). The oval window transmits vibrations from the middle ear's ossicular chain—malleus, incus, and stapes. In the petrous temporal bone, the cochlea and vestibular structures (semicircular canals, utricle, and saccule) are filled with endolymph for hearing and balance (Sasan et al., 2025).

Eustachian tube (ET) , Middle ear (ME) proper and mastoid air cells make up the middle ear cleft (Rashad et al., 2025). The 36-mm ET is two-thirds cartilaginous and one-third bony, allowing middle ear ventilation, drainage, and protection. Middle ear pathology revolves around ET dysfunction (Öztürk et al., 2025).

The six-walled middle ear houses the ossicles and neurovascular system. The middle ear is divided into several compartments by mucosal folds and ligaments. Disease extension can be limited for a considerable period of time to one or more compartments by these mucosal folds (Zaki et al., 2023). Epitympanum, mesotympanum and hypotympanum are the three main divisions of the ME. The

malleus head and incus body are in the epitympanum, which communicates with the mastoid antrum and is split by the cog (Soloperto et al., 2020). The TM-bordered mesotympanum is the thinnest, has ossicles , oval and round windows. The hypotympanum, a crescent-shaped area 1–6 mm next to the jugular bulb, shrinks when the bulb is elevated (Troyan et al., 2020).

Six ligaments within the tympanic cavity support the ossicles The anterior malleolar ligament originates from the neck of the malleus and extends to the anterior attic bony wall. The superior malleolar ligament extends between the malleus head and the tegmen tympani (Hassan et al., 2024). The lateral maleolar ligament is a thick fold starts from the middle portion of the neck of the malleus to the outer attic wall, it is considered to be strong to prevent progression of pars flaccida retraction pockets. The posterior malleal ligament extends from the neck of the malleus to the posterior tympanic spine. The superior incudal ligament passes from the superior surface of the body of incus to the tegmen, it separates the upper lateral attic from the upper medial attic.The posterior incudal ligament secures the short process in the incudal fossa then passes from the incudal fossa to the floor of the antrum (Sasan et al., 2025).

Prussak's Space is the small middle ear recess, bordered laterally by Shrapnell's membrane, superiorly by the scutum and lateral maleolar ligament, inferiorly by the short process of the malleus, and medially by the neck of the malleus. From the neck of the malleus, the anterior maleolar fold and the anterior ligament arise, demarcating Prussak's space anteriorly. Prussak's space is a site for primary acquired cholesteatoma formation (da Costa et al., 2023).

The middle ear communicates posteriorly with the mastoid antrum and its air cells through a large irregular opening, the aditus ad antrum.The antrum, which is pneumatized in 80% of adults and measures around 1×1×1 cm, is an air filled sinus within the petrous part of the temporal bone. It is marked by the MacEwen's triangle on the surface of the skull (Abdelkareem et al., 2025), it is well developed at birth, unlike the other air cells..Temporal bones may be classified by the degree of air cells development into pnuematized when air cells are well developed, diploic when bone marrow is present or sclerotic when the bone is dense with no air spaces beyond the antrum; 80 percent of the temporal bones are pnumatized. The mastoid air cells are classified according to their anatomical location into the following groups: zygomatic, angular, marginal, perisinus , peridural , periantral , retrofacial , perilayrinthine and tip cells (Koç et al. ,2004).

ET function, mastoid pressure buffering, and transmucosal gas exchange provide middle ear ventilation. Damage to this system causes negative pressure, effusion, and infection, affecting hearing. (Hamela et al., 2025).

Chronic Suppurative Otitis Media (CSOM)

Chronic suppurative otitis media (CSOM) is a middle ear mucoperiosteal lining disease that causes otorrhea through a TM perforation. It usually accompanies poorly resolved childhood acute otitis media (AOM). Due to their underdeveloped immune systems, children are more likely to develop the disorder (Hurst & Denne, 2020), which is linked to repeated upper respiratory tract infections, poor hygiene, and poverty. Pseudomonas aeruginosa and Staphylococcus aureus are most commonly isolated (Leach et al., 2021).

Two primary types of CSOM: tubotympanic (safe) and atticoantral (unsafe). A central pars tensa perforation causes mucopurulent discharge and conductive hearing loss in the safe kind. Tympanoplasty is the conventional treatment, however mastoidectomy may needed (Khairkar et al., 2023). The unsafe type of CSOM affects the pars flaccida or posterosuperior margin is associated with cholesteatoma and increases the risk of serious consequences (Tan et al., 2025).

Cholesteatoma, a non-neoplastic, keratinizing squamous epithelial lesion that may be congenital, primary acquired or secondary acquired, is aggressive and destructive (Casazza et al., 2021).

Congenital cholesteatoma, by definition, originates from embryonic areas of keratinizing epithelium within the middle ear cleft. Although congenital cholesteatoma generally are seen as pearl like masses behind an intact tympanic membrane, it can advance to perforate the TM and become chronically infected, taking the appearance of an acquired cholesteatoma (Potsic et al. ,2002).

Patients with acquired cholesteatoma usually present with a history of recurrent ear discharge and with

a reduced pneumatization of the mastoid would be seen on CT. According to the condition of the tympanic membrane, acquired cholesteatoma can be divided into primary and secondary. Primary acquired cholesteatomas develop behind an intact tympanic membrane through retraction of pars flaccida as a result of obstructive ET dysfunction. Prolonged exposure to negative middle ear pressure causes the TM to retract medially. A retraction pocket may eventually become trapped behind the TM, inducing inflammatory changes. This epithelium-lined pocket accumulates keratinized squamous debris, which forms a cholesteatoma (Lou, 2023).

Secondary acquired cholesteatomas grow in the middle ear mostly through marginal perforation of the tympanic membrane. Squamous epithelium can migrate through the TM defect into the middle ear space, with the accumulation of squamous debris resulting in development of the cholesteatoma (Dolhi & Weimer, 2022). Secondary acquired cholesteatoma can be iatrogenic (after surgery such tympanoplasty), residual (due to incomplete removal), recurrent (new development post-treatment), or retention cholesteatoma (trapped keratin in insufficiently exteriorized cavity) (Dolhi & Weimer, 2022). There are several theories of the pathogenesis of acquired middle ear cholesteatoma such as invagination theory, basal cell hyperplasia theory, epithelial invasion theory and squamous metaplasia theory. Chronic negative pressure produces TM retraction and pocket formation, according to the invagination theory Basal cell hyperplasia, epithelial migration through TM perforations, and chronic inflammation-induced middle ear lining squamous metaplasia are other potential processes (Godlewska et al., 2020).

Complications of cholesteatoma

Cholesteatoma is a non-neoplastic lesion of the temporal bone that can gradually expand and cause complications by bone erosion of the nearby structures. It is capable of causing significant ossicular erosion and may extend into the labyrinth, with labyrinthine fistula reported as a frequent complication. There are many theories to explain the destructive pattern of cholesteatoma such as pressure theory, chemical activity theory, bacterial infection, inflammatory mediators theory, enzymatic activity theory, proliferation markers theory, apoptosis and apoptotic activity theory (Hura et al., 2025). Histologically, the lesion consists of a hyperplastic squamous epithelial matrix, an inflammatory perimatrix, and keratinous debris. resorption occurs through chronic pressure effects, acidic breakdown products from keratin, and inflammatory cytokines stimulated by bacterial biofilms (Krahl et al., 2022).

Several molecular mediators contribute to this destructive process. Receptor activator of nuclear factor kappa ligand (RANKL) is a key driver of osteoclast differentiation, and elevated RANKL/ osteoprotegrin (OPG) ratios are strongly associated with more extensive bone erosion. Nitric oxide (NO) also enhances osteoclastic activity, while pro-inflammatory cytokines such as tumor necrosis factor alpha (TNF-α) and interleukin 1(IL-1), along with matrix metalloproteinases MMP-2 and MMP-9, are linked to aggressive disease behavior and tissue degradation (Huang et al., 2023). Collagenases and hexosaminidase (HEX) enzymes further degrade extracellular matrix components, and monoclonal antibody (MIB1) expression has been identified as a marker of aggressiveness in pediatric cholesteatoma (Xing et al., 2025).

Epithelial hyperproliferation is further promoted by elevated prostaglandins, particularly PGE2 and thromboxane E2. Resistance to apoptosis, through upregulation of anti-apoptotic proteins despite stable p53 expression, allows for the persistence and expansion of cholesteatoma tissue. Final common pathways of bone erosion involve osteoclast activation, mediated by M-CSF, IL-1, IL-6, TNF-α, and arachidonic acid derivatives (Xu et al., 2023). The immunological environment is also critical, with elevated levels of mast cells, T lymphocytes (CD3, CD6), histiocytes (CD68), and toll-like receptors (TLR2, TLR3, TLR4), further underscoring the role of immune-mediated mechanisms in cholesteatoma-induced bone resorption (Hada et al., 2025).

Radiologic Evaluation of Cholesteatoma

High-Resolution Computed Tomography (HRCT) is preferred for suspected cholesteatoma because to its high sensitivity and negative predictive value, especially when the middle ear and mastoid are clean. Ossicular erosion, scutum blunting, bony remodeling, labyrinthine fistula, and non-dependent soft tissue density are key findings. The diseased ear is compared to the contralateral side to prevent confusing typical anatomical variations for pathology. Middle ear aeration, mastoid pneumatization, and important bone elements such the lateral semicircular canal, tegmen tympani, and facial canal are

assessed (Anne et al., 2024). CT is less specific when soft tissue emerges without erosion, making cholesteatoma difficult to distinguish from effusion or granulation tissue, however air-fluid levels and an expanded antrum may indicate infection (Ferriastuti et al., 2022). CT specificity increases to 80% when associated with clinical findings. In revision patients with illness affecting the tegmen or petrous apex, HRCT is essential for surgical planning despite its tissue characterization limits. Routine HRCT before mastoid surgery is advised. (Saha et al., 2025).

Magnetic Resonance Imaging (MRI) complements HRCT, distinguishing cholesteatoma from granulation, fibrosis, or cholesterol granulomas, and assessing intracranial or labyrinthine extension, as shown in (Table.1). Cholesteatomas have hypointense T1 imaging, no gadolinium enhancement, and intermediate to high T2 signal (Nasef et al., 2024). Although artifacts might cause false positives, diffusion-weighted imaging (DWI) is very specific for cholesteatoma, which shows hyperintensity due to limited diffusion. Inflammatory tissues may increase with contrast, however cholesterol granulomas are hyperintense on T1 and T2. MRI is especially beneficial to detects residual or recurrent illness post-operatively, when CT generally fails. MRI using DWI sequences increases diagnostic precision and guides surgical decisions in difficult or recurring situations, but is not usually used preoperatively (Mansour et al., 2025).

Table (1): Differential diagnosis and imaging of cholesteatoma with MRI

	T1-weighted MRI	**T1-weighted MRI with gadolinium**	**T2-weighted MRI**	**Diffusion-weighted MRI**
Cholesteatoma	Hypo-intensity	Hypo-intensity Peripheral rim (matrix)	Hyper-intensity	Hyperintensity
Cholesterol granuloma	Hyper-intensity	Hyper-intensity	Hyper-intensity	Low intermediate
Inflammation/scar tissue	Hypo-intensity	Hyper-intensity	Hyper-intensity	No signal

Surgical Goals and Approaches

Cholesteatoma surgery aims to eliminate the disease, prevent recurrence, preserve function, create a dry, self-cleaning ear, and restore hearing through sound conduction mechanisms as possible (Tan et al., 2025). The major surgical methods are CWU and CWD. Canal wall up improves function and cosmetics but has greater residual and recurring disease rates, requiring second-look treatments. Canal wall down exposes and removes disease in hidden recesses more thoroughly, although it may cause cavity troubles and hearing loss (Hu et al., 2020).

Choice of Technique

Disease extention, location of the lesion, hearing status, mastoid pneumatization, and patient variables determine CWU or CWD. In situations with sclerotic mastoids, low dura, or complex anatomy, CWD is recommended over CWU (Swain et al., 2024). A hybrid method that temporarily removes and reconstructs the canal wall provides disease clearance and cavity repair.

Canal Wall Reconstruction (CWR)

It combines CWU and CWD advantages for enhanced hearing, disease access, and reduced morbidity in a single-stage operation (Askar et al., 2021). Atticotomy with cartilage or bone lateral attic wall repair improves function, notably in cholesteatoma. Reconstructing the posterior canal wall after CWD reduces recurrence and persistent otorrhea (Hasaballah et al., 2021). Autologous cartilage, bone pate, cortical bone, titanium mesh, hydroxyapatite, and ionomer cement are common used materials. Materials used should be durable, stable, accessible, and non-resorptive. Cost-effective and biocompatible cartilage and bone are recommended. Shaped autografts may slow surgery but lower extrusion risk, especially in chronic or revision patients (Alam & Chandra, 2022).

Attic Wall Reconstruction (Bone and cartilage grafting)

Autograft prosthesis like ossicles (incus, malleus) and cortical bone, have the advantages of a very low

extrusion rate, no risk of transmitting disease and biocompatibility. Their displacement, complete absorption, small remnant size and possibility of harboring microscopic disease have been blamed as potential disadvantages of their use (Georgeanu et al., 2023). The ability to initiate new bone growth is one of the greatest advantages of autologous bone graft over other substances. Well-fixed cortical bone grafts demonstrate a reliable bone graft survival and excellent osseointegration especially if combined with bone dust to fill the gaps between the bone graft and the surrounding bone. Many surgeons criticize using cortical bone in CWR due to the relative long surgery duration, possibility of bone resorption or infection (Albina, 2022). Cartilage with perichondrium is a good material for grafting in patients with poor Eustachian tube function, bilateral ear disease, smokers, anterior perforation, wet ears and also in revision cases. Reconstruction of attic can be carried out by a piece of tragal or conchal cartilage. The size and shape of the piece are decided according to the area of defect created by drilling or disease (Lucidi et al., 2022). The greatest advantage of the cartilage technique is its technical simplicity and easy harvesting, heal quickly and promote complete epithelialization of the mastoid cavity lining, leading to a dry, self-cleaning ear than bone graft. It lacks osteogenic potential, may not be available in revision operations, and may cause instability or recurrence. Its stiffness may reduce high-frequency sound transmission and hide remaining illness on imaging (Komori et al., 2021).

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