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Squamous Papilloma of the External Auditory Canal as a High-Fidelity Mimic of Malignant Otitis Externa: A Case Report

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A B S T R A C T

Malignant otitis externa (MOE) is a life-threatening osteomyelitis of the skull base, typically affecting geriatric patients with diabetes mellitus. Its initial presentation of otalgia, otorrhea, and an external auditory canal (EAC) mass demands an immediate, high index of suspicion. Squamous papilloma, a benign human papillomavirus (HPV)-related neoplasm, is exceedingly rare in the EAC. This report details a case where this rare benign entity presented as a high-fidelity clinical and laboratory mimic of MOE. We present the case of a 71-year-old female with poorly controlled Type 2 Diabetes Mellitus who presented with a three-week history of severe, refractory otalgia. A systematic diagnostic evaluation was performed, including clinical examination, full audiological assessment, serum inflammatory markers, and high-resolution computed tomography (HRCT) of the temporal bones. The patient's presentation was a textbook surrogate for MOE, including severe otalgia, purulent otorrhea, a friable EAC mass, and markedly elevated erythrocyte sedimentation rate (ESR) (78 mm/hr) and C-Reactive Protein (CRP) (45.2 mg/L). However, HRCT demonstrated an occluding soft-tissue mass without the hallmark finding of temporal bone erosion. The patient underwent transcanal excisional biopsy. Histopathological (H&E) analysis provided the definitive diagnosis of a benign squamous papilloma, with pathognomonic koilocytosis consistent with HPV infection. The patient's severe symptoms resolved completely upon excision. In conclusion, this case highlights a critical diagnostic pitfall. A secondarily infected EAC squamous papilloma can create a clinical and laboratory picture indistinguishable from early-stage MOE. The absence of bony erosion on HRCT is the single most critical finding to pivot the diagnosis away from invasive osteomyelitis. This report underscores the mandatory role of a systematic diagnostic pathway combining imaging and histopathology to prevent misdiagnosis and avoid unnecessary, prolonged, and toxic systemic antimicrobial therapy.

1. Introduction

The presentation of severe, unrelenting otalgia in a geriatric or immunocompromised patient, particularly one with diabetes mellitus, constitutes an otolaryngological emergency.[1] This clinical scenario immediately activates a high-stakes diagnostic algorithm focused on a single, paramount imperative: the exclusion of malignant otitis externa (MOE), also known as necrotizing otitis externa. This condition, first described by Toulmouche in 1838 and later characterized by Chandler in 1968, is not a simple external otitis but a relentless, invasive, and potentially fatal osteomyelitis of the temporal bone and adjacent skull base.[2] The mortality rate, once

approaching 50%, remains significant (10-20%) even with modern antimicrobial and surgical therapies, underscoring the necessity for prompt and accurate diagnosis.[3]

MOE is a classic example of a "susceptible host" disease, with over 90% of adult cases occurring in patients with diabetes mellitus.[4] The pathophysiology is a "perfect storm," a confluence of host vulnerability and microbial virulence. The diabetic patient provides a fertile ground for invasive infection via a triad of factors. Hyperglycemia directly impairs the innate immune response. It reduces neutrophil chemotaxis, phagocytosis, and intracellular killing capabilities. This blunted frontline defense allows a superficial infection to become invasive. Chronic diabetes induces progressive microvascular disease, characterized by endothelial dysfunction and thickening of capillary basement membranes. This compromises blood supply to the tissues of the EAC, creating a relatively ischemic and hypoxic environment that is both vulnerable to infection and difficult for systemic antibiotics to penetrate. Some evidence suggests that the cerumen in diabetic patients is more alkaline (higher pH) than in non-diabetic individuals, a local microenvironment that further favors the proliferation of the primary pathogen. Pseudomonas aeruginosa, a Gram-negative aerobic bacillus, is the causative agent in over 95% of MOE cases. This organism is not merely an opportunistic colonizer; it possesses a formidable arsenal of virulence factors. These include exotoxin A (which inhibits host protein synthesis), proteases, and elastases (which facilitate tissue degradation), and a profound ability to form biofilms. This biofilm protects the bacteria from host defenses and antibiotics, contributing to the refractory nature of the disease.[5]

The infection typically begins as a seemingly banal otitis externa but rapidly progresses. It breaches the epithelial barrier at the osteocartilaginous junction (the Fissures of Santorini), invading the underlying soft tissue, cartilage, and bone.[6] From here, it spreads relentlessly along the skull base, causing an obliterative endarteritis that furthers tissue necrosis. Its progression leads to the classic, ominous sequence of cranial neuropathies, beginning with the facial nerve (CN VII) at the stylomastoid foramen, followed by the jugular foramen (CN IX, X, XI), and potentially the hypoglossal nerve (CN XII). The clinical diagnosis is based on a constellation of findings. Levenson's criteria remain highly relevant, requiring otalgia, otorrhea, granulation tissue in the EAC, microabscesses on biopsy, and a failure to respond to topical treatment. Cohen and Friedman (1987) proposed criteria including diabetes/immunocompromise, otalgia, otorrhea, edema, granulation tissue, and, critically, a positive bone scan or CT scan demonstrating osteomyelitis. The presence of severe, nocturnal otalgia disproportionate to physical findings, coupled with an elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), is highly suggestive. However, the diagnosis is not always straightforward. In the early stages, before extensive bony erosion or cranial neuropathies develop, the clinical picture can be ambiguous. The "granulation tissue" at the EAC floor, while classic, can be mimicked by other inflammatory or neoplastic processes. This creates a significant diagnostic pitfall.[7]

Neoplasms of the external auditory canal are, in stark contrast to otitis externa, exceptionally rare. They account for less than 0.2% of all head and neck tumors. The differential diagnosis is broad and includes both malignant and benign entities. Squamous cell carcinoma (SCCa) is the most common malignancy, representing 80% of EAC cancers. It is an invasive, destructive lesion with a poor prognosis. Other malignancies include basal cell carcinoma, adenoid cystic carcinoma, and ceruminous adenocarcinoma.[8] A key diagnostic challenge is that an invasive, ulcerated SCCa can present with otalgia, otorrhea, and a "friable" mass, also mimicking MOE. Benign tumors are even rarer. They include osteomas, exostoses, ceruminous adenomas, and pleomorphic

adenomas. These are typically slow-growing, non-tender masses.

Within the category of benign neoplasms, the squamous papilloma is an exceptional finding in the EAC. Squamous papillomas are benign epithelial proliferations etiologically linked to the human papillomavirus (HPV), a small, non-enveloped, double-stranded DNA virus.[9] This entity is common in the oral cavity, larynx (as Recurrent Respiratory Papillomatosis or RRP), and anogenital region (as condylomata acuminata). The vast majority of benign papillomatous lesions are caused by "low-risk" HPV subtypes, primarily 6 and 11. The virus infects the basal keratinocytes of the epithelium, typically through micro-abrasions. The viral life cycle is tied to epithelial differentiation. The expression of the low-risk viral oncoproteins E6 and E7 dysregulates the cell cycle by binding to and promoting the degradation of tumor suppressor proteins p53 and pRb, respectively. However, the binding affinity of low-risk E6/E7 is much weaker than that of their "high-risk" (such as HPV-16, -18) counterparts. This leads to a controlled, benign proliferation and the formation of the characteristic exophytic, "wart-like" lesion, rather than the genomic instability and malignant transformation seen in high-risk HPV infections. The presence of a squamous papilloma in the EAC is a true medical curiosity, with only a small number of single-case reports and small case series in the global literature. The presumed mechanism of transmission is autoinoculation from a site of active infection (such as the hand) via a contaminated object or digit, introduced into the EAC, often during attempts at pruritus relief.[10]

This report addresses the intersection of these disparate pathologies: the rare benign neoplasm (EAC squamous papilloma) presenting in the ideal host (geriatric, uncontrolled diabetic) for the life-threatening infection (MOE). A review of the literature reveals that while the existence of EAC papilloma is documented, no report has detailed a case that presented as a high-fidelity clinical and laboratory mimic of MOE, complete with markedly elevated inflammatory markers. This creates a dangerous diagnostic dilemma: one condition requires 6-12 weeks of IV antipseudomonal antibiotics, while the other is cured by simple excision. A misdiagnosis carries severe consequences—either a fatal, untreated infection or months of unnecessary, toxic, and costly therapy. The aim of this report is, therefore, fourfold: (1) to present a unique case of a benign eac squamous papilloma that was clinically and biochemically indistinguishable from early-stage malignant otitis externa; (2) to detail the systematic, stepwise diagnostic pathway that successfully differentiated these two entities; (3) to review and contrast the distinct pathophysiologies of moe and hpv-driven papillomatosis; and (4) to propose a clear, evidence-based diagnostic algorithm for clinicians faced with this "dangerous mimicry."

2. Case Presentation

This case report has been prepared in accordance with the CARE (CAse REport) guidelines. Written informed consent was obtained from the patient for the publication of this case report, including all de-identified clinical details and accompanying medical images. A 71-year-old Balinese female presented to our Otorhinolaryngology-Head and Neck Surgery outpatient clinic with a chief complaint of severe, progressively worsening left-sided otalgia for three weeks. The patient described her experience as "frightening." The left-sided ear pain was not a minor ache but a "debilitating, throbbing, 10/10 pain" that was refractory to over-the-counter analgesics and frequently awakened her from sleep. She reported significant anxiety related to the severity of the pain and the purulent discharge, fearing she had a "terrible infection or cancer." She expressed frustration that her "frequent ear cleaning" with cotton swabs and a metal hairpin, which she did to relieve a persistent itch, had preceded the onset of the severe pain. Post-

surgery, she described the resolution of pain as "immediate and total," and expressed profound relief that her condition was benign and curable. Her symptoms began approximately one month prior with intermittent pruritus in the left ear, which she admittedly managed by "cleaning" the canal with cotton swabs and, on occasion, a metal hairpin. Three weeks prior to presentation, this pruritus was replaced by a dull, aching pain that rapidly intensified over two weeks into a severe, constant, throbbing otalgia. One week prior to admission, she noted the onset of a purulent, non-foul-smelling, whitish-yellow otorrhea. This was accompanied by a subjective decrease in hearing and a persistent sensation of aural fullness on the left side. She denied tinnitus, vertigo, dizziness, or any facial weakness, asymmetry, or dysphagia. Her past medical history was significant for a 15-year history of Type 2 Diabetes Mellitus, for which she was prescribed metformin and glibenclamide. She reported poor compliance and admitted to not regularly monitoring her blood glucose. She also had a history of hypertension, adequately controlled with amlodipine.

On physical examination, vital signs were stable. The patient was in mild distress from pain. Otoscopic examination of the right ear was unremarkable. Examination of the left ear, after gentle suctioning of purulent debris, revealed a polypoid, friable, pale-white papillary mass that appeared to arise from the inferior wall of the cartilaginous EAC. The mass was exquisitely tender to palpation with a sterile cotton wisp and occluded approximately 70-80% of the canal lumen. The tympanic membrane was obscured by the mass. There was no periauricular erythema or edema, and no tenderness over the mastoid tip. Critically, the facial nerve (CN VII) was symmetric at rest and with full voluntary movement (House-Brackmann Grade I). Tuning fork tests (512 Hz) were consistent with a left-sided conductive hearing loss (Rinne negative on the left, Weber lateralized to the left).

Given the high-risk host and clinical findings, the patient was admitted for an urgent workup to rule out MOE. Laboratory and audiological results are summarized in Table 1. The most salient findings were the significantly elevated inflammatory markers: an ESR of 78 mm/hr (Ref: <20 mm/hr) and a CRP of 45.2 mg/L (Ref: <5 mg/L). Her metabolic panel confirmed poorly controlled diabetes (HbA1c 7.4%). Pure Tone Audiometry (PTA) demonstrated a moderate sensorineural hearing loss (SNHL) on the right, consistent with presbycusis. The left ear demonstrated a severe mixed hearing loss (MHL), with a significant average air-bone gap (ABG) of 38.75 dB. Tympanometry of the left ear showed a Type B (flat) tympanogram, consistent with canal occlusion. The initial working diagnosis was malignant otitis externa sinistra. The patient was started on empirical intravenous Ceftriaxone, otic Ofloxacin drops, and analgesics, and an urgent consultation was made with internal medicine for glycemic control.

An urgent high-resolution computed tomography (HRCT) scan of the temporal bones (axial and coronal planes, 1mm cuts) was performed to assess for the hallmark of MOE: bony erosion. The scan demonstrated a well-defined, non-enhancing soft tissue mass confined to the cartilaginous portion of the left EAC, measuring approximately 0.8 x 0.6 cm. There was associated fluid and debris filling the EAC medial to the mass. Crucially, there was no evidence of bony erosion. The bony walls of the EAC, the tympanic plate, and the temporal bone were smooth and intact. There was no enhancement or inflammatory stranding in the periauricular soft tissues. The middle ear cavity, ossicular chain, and mastoid air cells were clear and well-aerated. This critical negative finding—the absence of osteomyelitis—made the diagnosis of established MOE highly unlikely, despite the compelling clinical and laboratory picture. The diagnostic focus immediately pivoted toward a neoplastic process.

Table 1. Summary of initial laboratory and audiological findings.

Clinical data for the 71-year-old female patient upon admission, detailing inflammatory, hematological, metabolic, and audiological results.

CATEGORY	TEST	RESULT	REFERENCE RANGE	INTERPRETATION
Inflammatory	Erythrocyte Sedimentation Rate (ESR)	**78 mm/hr**	< 20 mm/hr	**Significantly elevated**
	C-Reactive Protein (CRP)	**45.2 mg/L**	< 5.0 mg/L	**Significantly elevated**
Hematology	White Blood Cell (WBC) Count	6.95 × 10^3/uL	4.0 - 11.0 x 10^3/uL	**Normal**
	Hemoglobin	**10.96 g/dL**	12.0 - 15.5 g/dL	**Mild normocytic anemia**
	Platelet Count	209 × 10^3/uL	150 - 450 × 10^3/uL	**Normal**
Metabolic	Random Blood Glucose	**185 mg/dL**	70 - 140 mg/dL	**Elevated**
	Hemoglobin A1c (HbA1c)	**7.4%**	< 5.7%	**Poorly controlled diabetes**
	Creatinine	**1.52 mg/dL**	0.6 - 1.2 mg/dL	**CKD Stage III**
Audiology (Left)	Pure Tone Audiometry (PTA)	Severe Mixed Hearing Loss	Normal	**Significant ABG (38.75 dB)**
	Tympanometry	Type B (Flat)	Type A (Peaked)	**Canal occlusion / fluid**

The patient was taken to the operating theater for examination under anesthesia and transcanal excisional biopsy of the left EAC mass. Under the operating microscope, the mass was confirmed to be a pedunculated, papillary lesion arising from the inferior wall of the cartilaginous EAC. It was meticulously excised *in toto* from its base. The excised mass consisted of multiple fragments of pink-tan, rubbery tissue. After removal, the underlying canal skin was intact. The tympanic membrane was fully visualized and found to be normal. Microscopic examination on H&E staining revealed tissue sections lined by a markedly hyperkeratotic, acanthotic, and papillomatous stratified squamous epithelium. The proliferation was exophytic, forming branching, finger-like projections with central fibrovascular cores. Throughout the upper layers of the epithelium (stratum spinosum and granulosum), there were numerous koilocytes. These pathognomonic cells displayed enlarged, hyperchromatic, and "raisinoid" nuclei, surrounded by a clear perinuclear halo. There was no evidence of nuclear atypia, loss of polarity, or invasion of the basement membrane.

Final pathological diagnosis was squamous papilloma, external auditory canal, consistent with HPV infection. The patient's severe, refractory otalgia resolved completely within 24 hours of surgery. The empirical IV antibiotics were discontinued upon receiving the definitive pathological diagnosis. She was discharged on postoperative day 2. At her one-month follow-up visit, the patient was asymptomatic and "felt wonderful." Her patient-reported anxiety was resolved. Otoscopy of the left ear showed a fully healed EAC with a patent canal and a normal, intact tympanic membrane.

A repeat audiological assessment was performed one month post-surgery. The postoperative PTA showed a complete closure of the air-bone gap. The conductive component of her hearing loss was entirely resolved, and her hearing threshold improved by an

average of 22.5 dB. Her final diagnosis was bilateral, symmetric, mild-to-moderate sensorineural hearing loss, consistent with presbycusis, which had been masked on the left by the obstructive papilloma.

3. Discussion

This case presents a "perfect storm" of diagnostic confusion: a rare, benign neoplasm (EAC squamous papilloma) presenting in the archetypal host (geriatric, uncontrolled diabetic) with the classic clinical and laboratory signs (severe otalgia, otorrhea, EAC mass, high ESR/CRP) of a life-threatening infection (MOE). This high-fidelity mimicry forms the core of this report's educational value. The discussion will systematically deconstruct this mimicry, analyze the diagnostic pathway, and place this unique presentation within the context of the existing literature.[11]

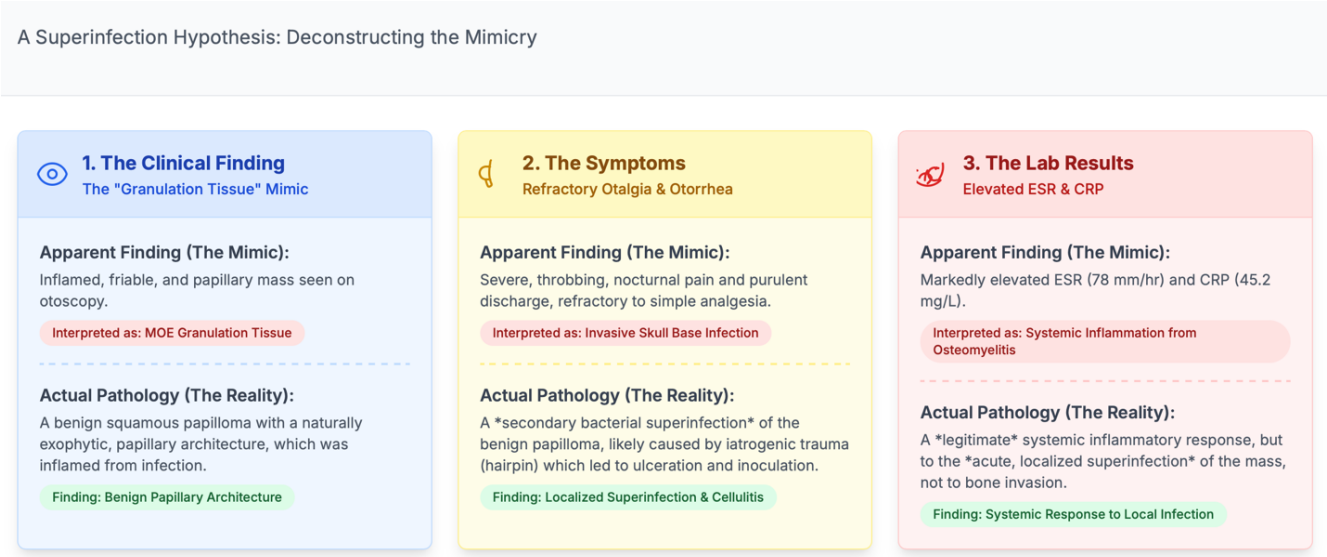

Figure 1. A superinfection hypothesis illustration.

The central thesis of this case—that a benign, non-invasive neoplasm produced the complete clinical, symptomatic, and laboratory signature of a life-threatening, invasive osteomyelitis—presents a profound diagnostic paradox. This situation, a "high-fidelity diagnostic mimicry," is far more than a simple medical curiosity. It represents a critical intersection of immunology, microbiology, cognitive science, and diagnostic technology. The consequences of misinterpreting this mimicry are severe, branching into two polar-opposite outcomes: the potential fatality of an untreated malignant otitis externa (MOE) or the significant morbidity, cost, and toxicity of a prolonged, unnecessary antibiotic regimen for a condition curable by simple excision.[12] A detailed, academic deconstruction of this paradox requires a systematic, multi-domain analysis. We must unpack how this mimicry was constructed, why it was so effective, and which specific diagnostic tools ultimately held the power to resolve it.

The first layer of deception was visual. The otoscopic finding of an inflamed, friable, and papillary mass in the external auditory canal (EAC) of a diabetic patient with severe otalgia is, in the mind of any trained otolaryngologist, pathognomonic for malignant otitis externa.[13] This finding is colloquially and universally described as "granulation tissue." To understand the mimicry, one must first define what

this "granulation tissue" truly represents. In the context of MOE, it is not healthy, healing tissue. It is a pathological product of invasive, necrotizing infection. It is the visible manifestation of *Pseudomonas aeruginosa*-driven osteomyelitis of the temporal bone. The bacteria, thriving in the microangiopathic and immunocompromised tissue of the diabetic EAC, invade the underlying bone, causing necrosis. The body's failed healing response mounts a profound inflammatory reaction, characterized by neo-angiogenesis (the formation of new, fragile blood vessels) and a dense infiltrate of inflammatory cells. This creates a "tissue" that is intensely hyperemic, friable, and bleeds on contact. This is the "granulation tissue" of MOE.

Now, consider the mimic: the squamous papilloma. Structurally, this lesion is defined by its papillary, or finger-like, architecture. It is an exophytic, benign proliferation of squamous epithelium growing outwards from a central fibrovascular core.[14] In its normal, quiescent state, it appears as a pale, "warty," and non-tender growth. It is typically asymptomatic or, at most, causes a mild, non-painful conductive hearing loss or pruritus. The key to the mimicry, therefore, is the superinfection. The patient's own admission of iatrogenic trauma with a hairpin provides the inciting event. This non-sterile object breached the epithelial integrity of the papilloma, ulcerating its surface and directly inoculating it with bacteria. In this moist, occluded, and debris-filled environment, a fulminant secondary bacterial superinfection occurred. This acute, localized infection transformed the papilloma. The underlying papillary architecture, now acutely inflamed, edematous, and hyperemic from the infection, became visually indistinguishable from the neo-angiogenesis and inflammation of true MOE granulation tissue. Both are friable, both are red, both are "polypoid," and both are found at the classic location. The clinician, "primed" by the patient's diabetic status, sees this visual data and, understandably, makes the inductive leap to MOE.

The second, and perhaps more compelling, layer of mimicry was symptomatic. The cardinal symptom of MOE is severe, refractory, nocturnal otalgia. This pain is not the pain of a simple swimmer's ear; it is a deep, boring, relentless ache that signifies inflammation of the periosteum and bone itself.[15] A benign, non-infected papilloma is, by contrast, painless. The central paradox—the severe pain—is resolved by the same "superinfection hypothesis." The patient was not experiencing the pain of osteomyelitis. She was experiencing the pain of an acute, localized cellulitis and abscess of the papilloma itself. The bacterial infection, introduced by the hairpin, was not limited to the surface. It invaded the fibrovascular cores of the papilloma, creating a contained, pressurized, and intensely inflamed soft-tissue infection. This type of localized, purulent infection in a confined space—analogous to a dental abscess or a digital felon—is known to cause exquisite, throbbing pain due to the rapid increase in interstitial pressure and the release of inflammatory mediators (prostaglandins, bradykinin). The purulent otorrhea was simply the exudate from this active, localized bacterial battle. The patient's pain was real, severe, and refractory to simple analgesia. The "superinfection hypothesis" provides a complete, plausible, and sufficient pathological mechanism for the patient's entire symptomatic presentation without invoking bone invasion. The symptoms were identical to MOE, but the source was entirely different: a superficial soft-tissue infection, not a deep-seated osteomyelitis.

This leads to the most deceptive finding of all: the laboratory data. The markedly elevated erythrocyte sedimentation rate (ESR) at 78 mm/hr and C-reactive protein (CRP) at 45.2 mg/L are highly compelling. In established protocols for MOE, these acute-phase reactants are fundamental. They are used not only to support the initial diagnosis but, more critically, to monitor the response to therapy. A persistently elevated ESR/CRP in a patient on antipseudomonal

antibiotics is a red flag for treatment failure, occult abscess, or inadequate duration of therapy. This case is a masterclass in the correct interpretation of these tests. ESR and CRP are non-specific markers of systemic inflammation. They do not and cannot identify the source or nature of that inflammation. They are signaling molecules produced by the liver in response to circulating cytokines (like IL-6 and TNF-alpha). In the case of MOE, these cytokines are released from the deep-seated, chronic bone infection. In this case, the same cytokines were released in response to the acute, fulminant, localized bacterial superinfection of the papilloma. The patient's body was, in fact, mounting a legitimate and appropriate systemic inflammatory response. The error is not in the test; the error is in the interpretation. The clinician, anchored by the clinical picture, interprets "High ESR/CRP" as "proof of osteomyelitis." The correct, more precise interpretation is "High ESR/CRP confirms the presence of a significant systemic inflammatory response." This case powerfully demonstrates that a severe, localized soft-tissue infection can produce an inflammatory signature identical to that of invasive bone disease.[16]

If the clinical exam, patient-reported symptoms, and laboratory data all conspire to create a false-positive picture, the diagnostic pathway must rely on a modality that is objective, anatomical, and specific. This case highlights the indispensable, pivotal role of high-resolution computed tomography (HRCT) of the temporal bone. MOE is, by definition, an osteomyelitis. The sine qua non of the disease, its very hallmark, is the erosion and demineralization of bone. HRCT, with its sub-millimeter slices and high spatial resolution, is exceptionally sensitive (86-100%) for detecting the subtle, early erosion of the cortical bone of the EAC, particularly the tympanic plate. It can also identify inflammatory stranding in the adjacent periauricular fat pads, which is another key sign of invasive spread.[17]

In this patient, the HRCT was the single most important diagnostic branch point. It was the "arbiter of truth." The findings were unambiguous: a well-defined soft-tissue mass without any evidence of bony erosion, periosteal reaction, or inflammatory stranding in the skull base. This "hard negative" finding was a powerful negative predictor for established MOE. This finding creates a critical, logic-driven diagnostic junction. While one could argue that extremely early MOE might precede radiologic findings, the patient's three-week history of severe, refractory pain makes this highly unlikely. The intensity and duration of the symptoms were profoundly incongruent with the normal, intact bone. This single test effectively "ruled out" the working diagnosis of MOE. It forced the clinical team to pivot the entire differential diagnosis.[18] The diagnostic tree collapsed from "Invasive Infection" (MOE) to "EAC Neoplasm." At this point, the primary differential shifted to Squamous Cell Carcinoma (which *can* cause bony erosion) versus a benign process. This pivot was the single action that prevented the patient from undergoing a 6- to 12-week course of potentially nephrotoxic (in a patient with known CKD) and ototoxic antipseudomonal antibiotics.

This case is also a profound lesson in the human element of medicine: cognitive bias. The patient's demographic and history—"71-year-old female with uncontrolled diabetes"—is such a powerful, classic "red flag" for MOE that it can induce diagnostic anchoring. Diagnostic anchoring is a well-documented cognitive bias where a clinician latches onto the first, most prominent, or most high-stakes piece of information and uses it as an "anchor" to interpret all subsequent data. In this case, the anchor was "This patient profile = MOE." This report demonstrates the critical importance of respecting contradictory data. The "normal HRCT" was the one piece of data that did not fit the anchored diagnosis. A biased clinician might dismiss it ("The CT is wrong," or "It's just too early to see"). A systematic, algorithm-driven clinician respects

it as the "diagnostic pivot." This case serves as a powerful testament to the value of systematic diagnostic pathways (as outlined in Table 2 of the manuscript) to overcome the inherent and unavoidable cognitive biases of clinical practice.

With the diagnosis pivoted to "neoplasm," only histopathology could provide the definitive answer. The excisional biopsy and H&E analysis were the gold standard. The finding of a classic papillary architecture combined with the pathognomonic koilocyte was the final, conclusive piece of evidence.[19] The koilocyte—with its perinuclear halo and "raisinoid" nucleus—is the visible cytopathic effect of HPV replication, confirming the lesion as a benign squamous papilloma. The entire, terrifying clinical picture was, definitively, a masquerade.

Finally, this diagnosis has critical implications for prognosis and surveillance. The treatment—simple excision—is curative for the lesion. But the etiology—a chronic HPV 6/11 infection—carries a theoretical risk of recurrence. This is a well-established phenomenon in other anatomical sites, such as the larynx, where recurrent respiratory papillomatosis (RRP) can be a lifelong burden. While EAC recurrence has not been documented, the biological possibility mandates a prudent methodology of long-term, regular otoscopic surveillance. The patient is not just "cured"; they are in remission from a viral process, and follow-up is essential. In summary, this case deconstructs a complex diagnostic illusion, revealing how a secondary infection of a benign mass can perfectly mirror a life-threatening invasive process. It champions the primacy of objective, anatomical imaging (HRCT) over compelling but non-specific clinical and laboratory data, and serves as a powerful reminder of the cognitive biases that can lead clinicians astray. Based on this case, we propose a systematic diagnostic algorithm for clinicians faced with a high-risk (such as diabetic) patient with severe otalgia and an external auditory canal mass. This algorithm emphasizes the pivotal role of HRCT (Table 2).[20]

Table 2. Diagnostic algorithm for suspected MOE.

STEP	ACTION	FINDINGS & INTERPRETATION	NEXT STEP / MANAGEMENT DECISION
Step 1	**Initial Assessment & Suspicion** • Clinical Exam (Otoscopy) • Patient Demographics (Diabetes) • Key Symptoms (Severe Otalgia)	**High Index of Suspicion for MOE.**	• Admit patient • Establish IV access • Start Empiric IV Antipseudomonal Abx • Initiate Glycemic control
Step 2	**Laboratory Workup** • CBC, CMP, HbA1c • **ESR & CRP**	**If ESR/CRP Elevated:** Confirms significant inflammation. *Increases* suspicion for MOE but *does not confirm* it.	**URGENT Step 3**
Step 3	**The Diagnostic Pivot** • **URGENT High-Resolution CT (HRCT) Temporal Bone**	**PATHWAY A: Bony Erosion PRESENT** (EAC, tympanic plate, skull base)	**Diagnosis: MOE Confirmed.** • Continue IV Abx (6-12 wks) • Biopsy mass to rule out concurrent SCCa
		PATHWAY B: Bony Erosion ABSENT (Soft tissue mass *without* bone involvement)	**Diagnosis: MOE Highly Unlikely.** Pivots differential to Neoplasm (Benign/Malignant) or other.
Step 4	**Definitive Diagnosis (Pathway B)** • Surgical Biopsy • (Transcanal Excisional Biopsy of Mass)	Send for Histopathology. *This is now a diagnostic, not just therapeutic, step.*	**Step 5**
Step 5	**Pathological Confirmation** • H&E Analysis	**Findings: Papillomatosis, Koilocytosis (Pathognomonic for HPV)**	**Final Diagnosis: Squamous Papilloma.** • **ACTION: DISCONTINUE IV ANTIBIOTICS.** • Counsel patient. • Plan long-term surveillance.
		Findings: Invasive Carcinoma	**Final Diagnosis: SCCa.** • Oncology workup (PET, MRI) • Plan for surgery/radiation

4. Conclusion

Squamous papilloma of the external auditory canal is an extremely rare benign tumor. This case demonstrates that when secondarily infected and inflamed, this lesion can be a "dangerous mimic," presenting in a high-risk diabetic patient with the complete clinical and laboratory profile of life-threatening malignant otitis externa. The high index of suspicion for MOE in this patient population is appropriate and mandatory. However, this case powerfully underscores that clinical and laboratory findings alone are insufficient for a definitive diagnosis. The absence of bony erosion on HRCT is a critical negative predictor that must pivot the diagnostic pathway. Definitive diagnosis relies on histopathology. This systematic approach, integrating clinical suspicion with objective radiological and pathological data, is essential to differentiate these disparate entities, ensuring correct patient management and preventing months of unnecessary, toxic, and costly antimicrobial therapy.

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