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Giant Verruca Vulgaris of the Auricle Mimicking Verrucous Carcinoma: A Diagnostic and Surgical Case Study

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A B S T R A C T

Introduction: Verruca vulgaris, universally recognized as the common wart, represents a benign epithelial proliferation induced by the human papillomavirus (HPV). While these lesions are ubiquitous in the general population, typically manifesting as small hyperkeratotic papules on the acral regions, the occurrence of giant variants exceeding 2 centimeters in diameter on the auricle is an exceptionally rare clinical entity. This unusual presentation creates a significant diagnostic dilemma for the head and neck surgeon, as giant auricular warts frequently mimic the clinical presentation of malignant neoplasms, specifically verrucous carcinoma and squamous cell carcinoma. **Case presentation:** We document the case of a 36-year-old Indonesian male who presented with a massive, painless, exophytic mass on the posterior aspect of the right auricle. The lesion had persisted for ten years, exhibiting a slow, linear growth trajectory without spontaneous regression. Physical examination revealed a solitary, sessile, papillomatous lesion measuring 3.3 cm × 1.5 cm × 1.0 cm. Despite its alarming size, the mass remained mobile over the underlying perichondrium. Given the giant dimensions and the inability to rule out malignancy via visual inspection, the patient underwent total surgical excision with primary closure under general anesthesia. Histopathological analysis confirmed the diagnosis of verruca vulgaris, characterized by marked hyperkeratosis, papillomatosis, and pathognomonic koilocytosis, with an intact basement membrane ruling out invasive carcinoma. **Conclusion:** Giant verruca vulgaris of the auricle is a rare pathology that requires a rigorous diagnostic approach. We conclude that auricular verrucous lesions exceeding 2 centimeters should be clinically managed as tumors of uncertain potential. Surgical excision remains the diagnostic and therapeutic gold standard, offering immediate aesthetic restoration and the essential histopathological confirmation required to differentiate benign giant warts from the deceptive pushing border of verrucous carcinoma.

1. Introduction

The human papillomavirus constitutes a vast and sophisticated family of non-enveloped, double-stranded DNA viruses that exhibit a strict and specific tropism for the squamous epithelium of the human body. This viral family, comprising over 200 distinct genotypes, is responsible for a wide spectrum of mucocutaneous pathologies ranging from benign, self-limiting cutaneous warts to aggressive, invasive malignancies.[1] Among the myriad of clinical manifestations associated with this viral pathogen, verruca vulgaris, or the common wart, is undoubtedly the most prevalent phenotype encountered in clinical practice. These lesions typically affect the keratinized skin of the acral regions, most notably the hands and feet, where environmental exposure and frequent micro-trauma facilitate the entry of viral particles into the basal keratinocytes. Epidemiological data indicate a high prevalence of cutaneous warts in the general population, with estimates suggesting that up to one-third of primary school children may be affected at any given time. However, despite the ubiquity of this virus,

the manifestation of verruca vulgaris on the external ear, and specifically the auricle, is an epidemiological anomaly that is seldom documented in the medical literature.[2]

The auricle represents a unique anatomical and immunological microenvironment that differs significantly from the glabrous skin of the palms and soles. Anatomically, the auricular skin is characterized by its thinness and its tight adherence to the underlying perichondrium on the anterior surface, while the posterior surface possesses a slightly more generous layer of subcutaneous tissue.[3] This region lacks the thick, protective hyperkeratotic layer found on the hands and feet, which is the preferred habitat for HPV types 1 and 2. Furthermore, the auricle is generally shielded from the heavy viral inoculum load typically found on communal surfaces, such as swimming pool decks or gymnasium floors, which drive the transmission of plantar and palmar warts.[4] Consequently, the establishment of an HPV infection in this region implies a specific and unusual breach in local defense mechanisms. It suggests a mechanism of direct inoculation, potentially through autoinoculation from digital warts or transmission via contaminated fomites such as ear-cleaning tools or barber equipment, which can introduce the virus into the delicate auricular epidermis.[5]

In the vast majority of cases, cutaneous warts present as small, discrete, hyperkeratotic papules measuring between one and ten millimeters in diameter. These lesions are frequently self-limiting, as the host immune system eventually recognizes the viral antigens and mounts a cell-mediated immune response that clears the infection.[6] However, a distinct and biologically intriguing subset of lesions defies this natural history. When a verrucous lesion exceeds two centimeters in diameter or demonstrates aggressive, unchecked localized growth, it is clinically classified as a giant verruca vulgaris. This designation denotes a profound deviation from the standard viral life cycle and implies a state of localized immune evasion or immunological ignorance that allows for unrestricted viral replication over a prolonged period. The emergence of a giant wart in an immunocompetent individual raises significant questions regarding host-virus interactions and the potential for localized immune tolerance in specific dermatological sites.[7]

The clinical significance of a giant auricular wart extends far beyond its rarity or cosmetic impact; its primary importance lies in its ability to mimic malignant neoplasms.[8] The giant dimensions and chronic duration of these lesions overlap significantly with the clinical presentation of Verrucous Carcinoma, also known as Ackerman's tumor, and well-differentiated squamous cell carcinoma. Both of these malignant entities can present as slow-growing, exophytic, cauliflower-like masses that are indistinguishable from a giant wart upon visual inspection. This mimicry creates a dangerous diagnostic gray zone for the clinician. A misdiagnosis of a malignant lesion as a benign wart can lead to inadequate treatment, recurrence, and local invasion, while the misdiagnosis of a benign wart as a malignancy can result in unnecessarily radical surgery and disfigurement. Therefore, the distinction between these entities is not merely academic but is the cornerstone of effective patient management.[9,10]

The novelty of this study lies in the detailed documentation of a giant verruca vulgaris measuring 3.3 centimeters on the posterior auricle of an immunocompetent adult male, which persisted and expanded over a decade-long period of clinical neglect. This case serves as a rare and valuable observation of the natural history of untreated HPV infection in a non-genital site, demonstrating the capacity of benign lesions to achieve massive proportions through mechanisms of localized immune evasion. The primary aim of this study is to delineate the forensic, clinical, and histopathological features that allow for the accurate differentiation of giant benign warts from auricular malignancies. Furthermore, we aim to establish a clear surgical management algorithm emphasizing that for auricular lesions exceeding two centimeters, excisional biopsy is not merely a therapeutic option but a diagnostic mandate essential to rule out the silent progression of verrucous

carcinoma and ensure long-term patient safety.

2. Case Presentation

A 36-year-old male patient of Indonesian descent presented to the Otorhinolaryngology outpatient clinic with a primary complaint of a large, disfiguring mass located on the posterior aspect of his right ear. The clinical history was particularly notable for the extreme chronicity of the lesion, which the patient reported had been present for approximately ten years. Forensic inquiry into the growth kinetics of the mass revealed a linear growth velocity; the patient described the lesion initiating as a minute, non-specific skin tag a decade prior, which then underwent a slow, inexorable expansion without periods of rapid acceleration or spontaneous regression. This history of slow, steady growth is clinically distinct from the rapid, exponential doubling often seen in aggressive high-grade malignancies, yet it mimics the deceptive pushing growth pattern of low-grade verrucous carcinoma.

When questioned regarding the significant delay in seeking medical attention for such a prominent lesion, the patient cited a complete lack of pain and a slow, gradual acceptance of the cosmetic deformity as part of his normal appearance. He denied any history of trauma to the ear prior to the onset of the lesion, and crucially, he reported no history of self-surgery, manipulation, or the application of traditional caustic remedies. This negative history is significant because chronic irritation and inflammation from attempted self-removal can sometimes induce malignant transformation in benign lesions or alter the histological architecture, complicating the diagnosis. The patient denied any associated otologic symptoms, such as otorrhea, hearing loss, tinnitus, or vertigo, suggesting the pathology was confined to the external ear. He was immunocompetent, with no history of HIV, organ transplantation, diabetes mellitus, or chronic use of immunosuppressive medications, detailed in Figure 1.

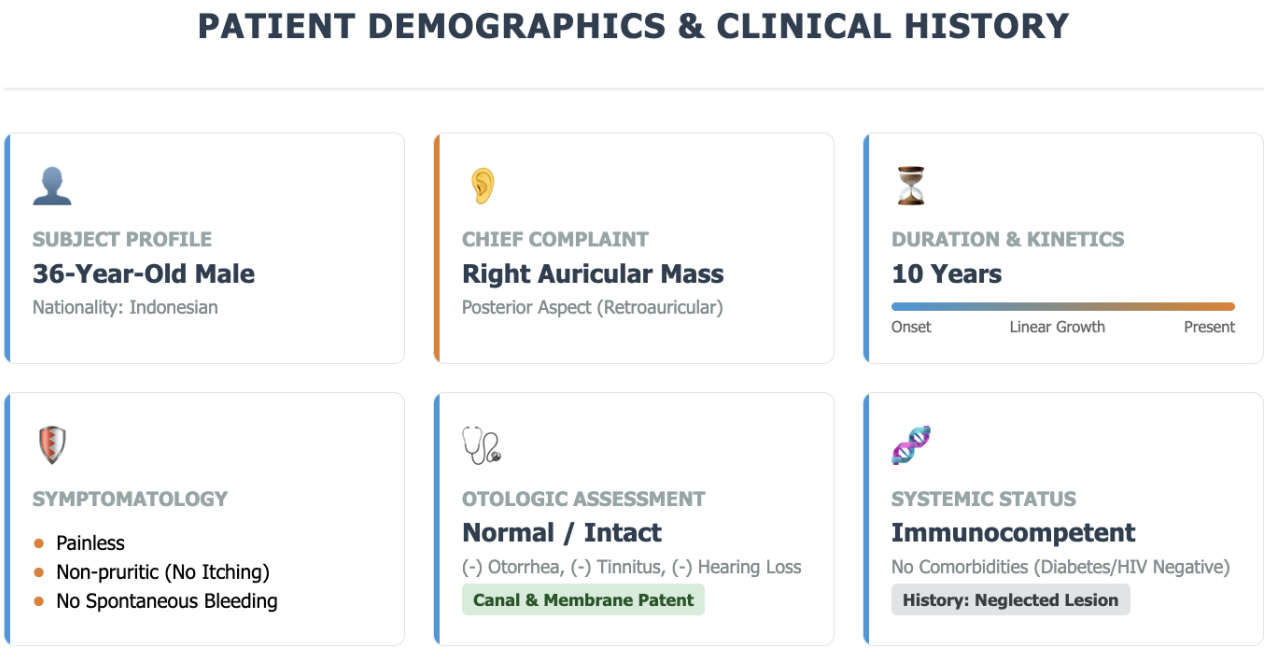

Figure 1. Schematic overview of the patient's demographics and clinical presentation. The diagram highlights the significant chronicity (10 years) of the lesion in an immunocompetent host, juxtaposed with the absence of alarming symptoms (pain, bleeding) or otologic involvement. This clinical profile—massive size with benign behavior—formed the basis for the preoperative diagnostic dilemma.

Upon physical examination, a striking and massive pathology was identified on the retroauricular surface of the right auricle. The lesion presented as a solitary, exophytic mass with a distinct papillomatous, cauliflower-like surface architecture that is characteristic of hyperkeratotic proliferation. The surface was rough and irregular, with deep clefts and fissures typical of verrucous growth. Morphological assessment revealed a sessile configuration, meaning the lesion possessed a broad base attached directly to the skin, rather than being pedunculated on a stalk. This finding is surgically significant as sessile lesions require a wider disruption of the cutaneous surface for complete removal compared to pedunculated lesions, which can often be simply snipped at the base.

The dimensions of the mass were measured precisely using calipers, revealing a size of 3.3 cm in length, 1.5 cm in width, and 1.0 cm in height. These dimensions firmly categorized the lesion as a giant variant, exceeding the standard 2-centimeter threshold used in clinical dermatology. Assessing the mobility of the lesion was paramount to the pre-surgical staging and risk stratification. Palpation demonstrated that the mass was firm to the touch but non-tender. Most importantly, the mass was found to be mobile relative to the underlying auricular cartilage. This mobility indicated that the lesion was confined to the cutaneous and subcutaneous layers and had not invaded the perichondrium or the cartilage itself. Fixation to the underlying cartilage would have been an ominous sign suggestive of invasive squamous cell carcinoma. The retroauricular sulcus was intact, and there was no extension of the lesion onto the mastoid process or into the post-auricular hairline. Otoscopic examination of the right ear demonstrated a patent external auditory canal and a pristine, pearly-grey tympanic membrane, confirming that the middle and inner ear were unaffected. A comprehensive head and neck examination revealed no palpable pre-auricular, post-auricular, or cervical lymphadenopathy, ruling out regional metastasis at the clinical level, detailed in Figure 2.

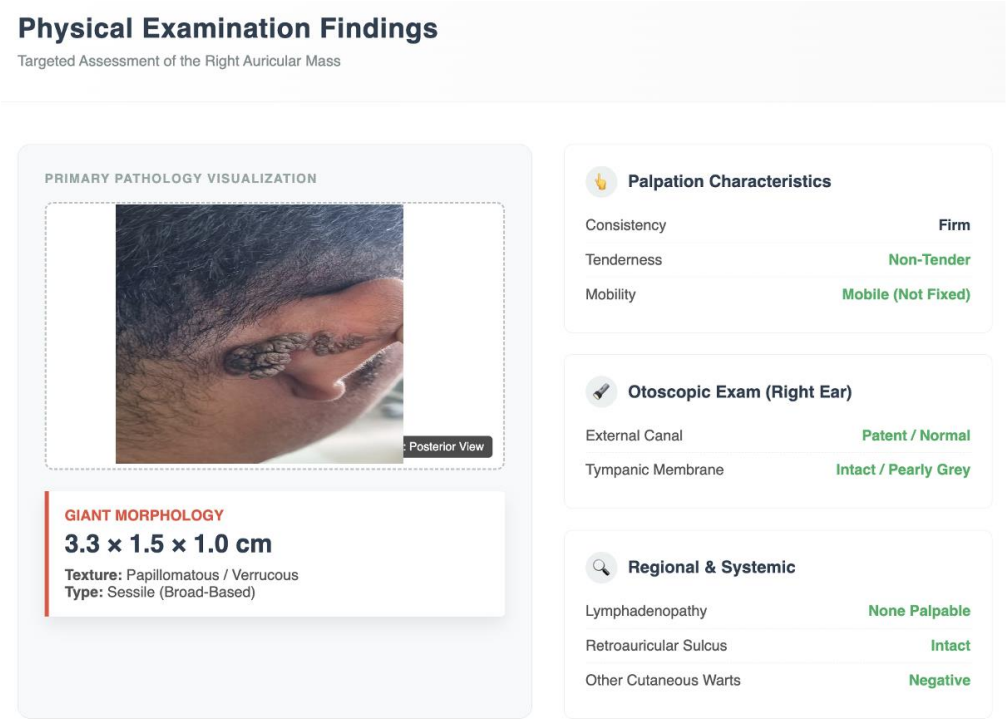

Figure 2. Comprehensive physical examination findings of the right auricle. The schematic (left) illustrates the sessile, verrucous mass located on the posterior aspect, measuring 3.3 cm in length. The clinical data (right) emphasizes critical negative findings: the preservation of mobility over the cartilage and the absence of regional lymphadenopathy, features which clinically supported a benign diagnosis despite the giant dimensions.

The preoperative assessment presented a complex diagnostic triad of possibilities: Giant verruca vulgaris, verrucous carcinoma, and squamous cell carcinoma. The clinical features supporting a benign diagnosis included the extremely slow growth over ten years, the absence of ulceration or necrosis, the lack of spontaneous bleeding, and the persistent mobility of the lesion over the underlying cartilage. However, the giant size exceeding 2 centimeters and the location on a sun-exposed organ in a patient in his 30s necessitated the rigorous exclusion of malignancy. Squamous cell carcinoma typically presents as an ulcerative, faster-growing lesion that may be fixed to underlying structures, but well-differentiated variants can be exophytic. Verrucous carcinoma presents the greatest diagnostic challenge as it is a low-grade malignancy that looks almost identical to a giant wart but possesses a locally destructive pushing border. Given these considerations, a working diagnosis of a benign auricular tumor, specifically giant verruca vulgaris, was established. However, the decision was made to clinically manage the lesion as a tumor of uncertain potential, mandating excisional biopsy rather than destructive ablation to ensure a definitive histological diagnosis, detailed in Figure 3.

Figure 3. Schematic of the preoperative differential diagnosis. The diagram contrasts the clinical features of the patient's lesion (Giant Verruca Vulgaris) against its three primary mimics. While the size and chronicity strongly raised suspicion for malignancy (Red), the specific absence of fixation and ulceration, combined with eventual histopathological confirmation, supported the benign diagnosis (Green).

The patient was scheduled for total surgical excision under general anesthesia. While local anesthesia is often sufficient for routine auricular lesions, the decision to utilize general anesthesia in this case was multifaceted. It was justified to ensure absolute patient immobility, which is crucial for precise dissection around the delicate auricular cartilage, and to manage potential patient anxiety regarding the resection of such a large mass near the face. The surgical approach involved a meticulously planned elliptical incision surrounding the broad base of the lesion. A narrow margin of approximately 1 to 2 millimeters of clinically healthy skin was included in the resection. This decision to use a narrow margin

rather than the wide margins typically reserved for malignancy was a calculated clinical risk based on the benign gross appearance and the need to preserve tissue for closure.

The mass was excised in toto, with the surgeon strictly maintaining the dissection plane superficial to the perichondrium. Great care was taken to preserve the perichondrium intact, as its removal would expose the avascular cartilage, increasing the risk of necrosis and infection. Following the removal of the mass, the resulting defect measured approximately 4 centimeters in length. Closing a defect of this magnitude on the ear without causing distortion or cupping presents a reconstructive challenge. To achieve primary closure, the surrounding post-auricular skin was undermined extensively towards the mastoid and the neck. This maneuver released the tension on the skin flaps, allowing the wound edges to be approximated and sutured without pinning the ear back against the head. The wound was closed using interrupted non-absorbable sutures to ensure secure healing, detailed in Figure 4.

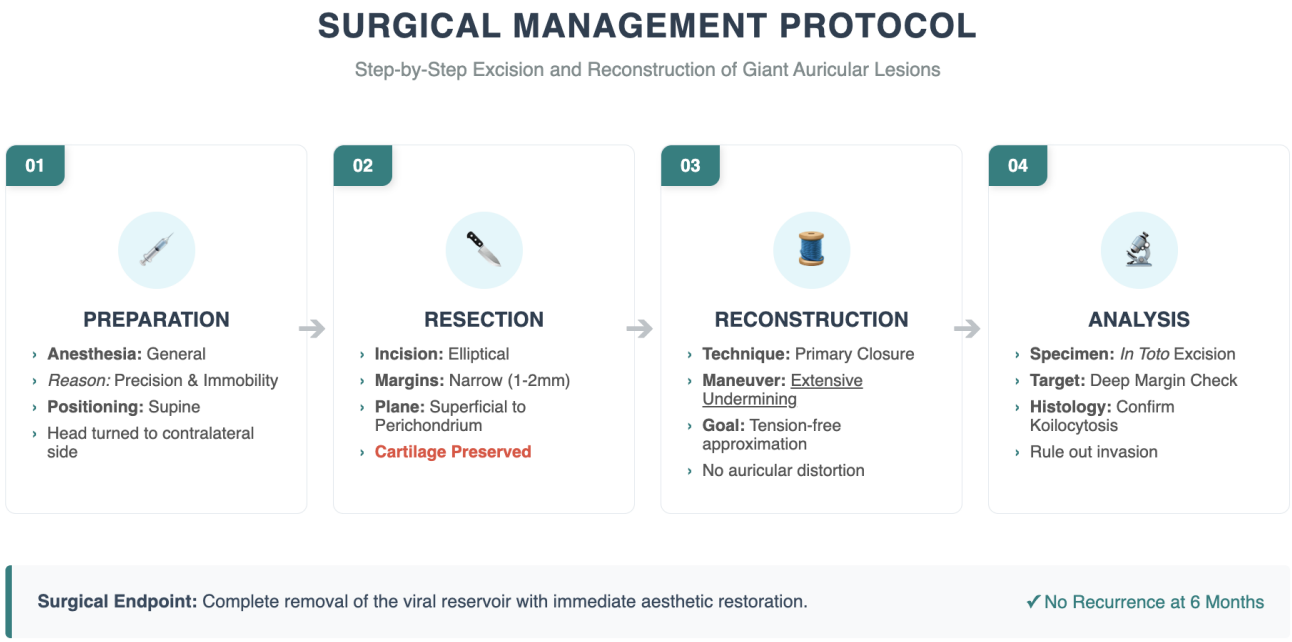

Figure 4. Schematic of the surgical protocol employed. The process emphasizes the preservation of the underlying perichondrium during resection (Step 02) and the critical role of tissue undermining during reconstruction (Step 03) to achieve primary closure of the 4cm defect without distorting the auricular cartilage.

The excised specimen was submitted for histopathological analysis using standard Hematoxylin and Eosin staining. Microscopic examination revealed the classic architectural features associated with a viral wart. The epidermis demonstrated marked hyperkeratosis, a thickening of the stratum corneum, along with acanthosis, a thickening of the spinous layer, and papillomatosis, characterized by undulating, finger-like projections of the epidermis. Crucially, the diagnostic hallmark of HPV infection, koilocytosis, was identified. Koilocytes are vacuolated keratinocytes with perinuclear halos and pyknotic nuclei, typically found in the upper layers of the epidermis. The presence of these cells provided definitive evidence of the viral etiology of the lesion. Furthermore, the basement membrane was found to be completely intact, with no evidence of keratin pearls, atypical mitoses, or invasive nests of

cells penetrating into the dermis. This absence of invasion effectively ruled out both squamous cell carcinoma and verrucous carcinoma, confirming the diagnosis of a benign giant verruca vulgaris, detailed in Figure 5.

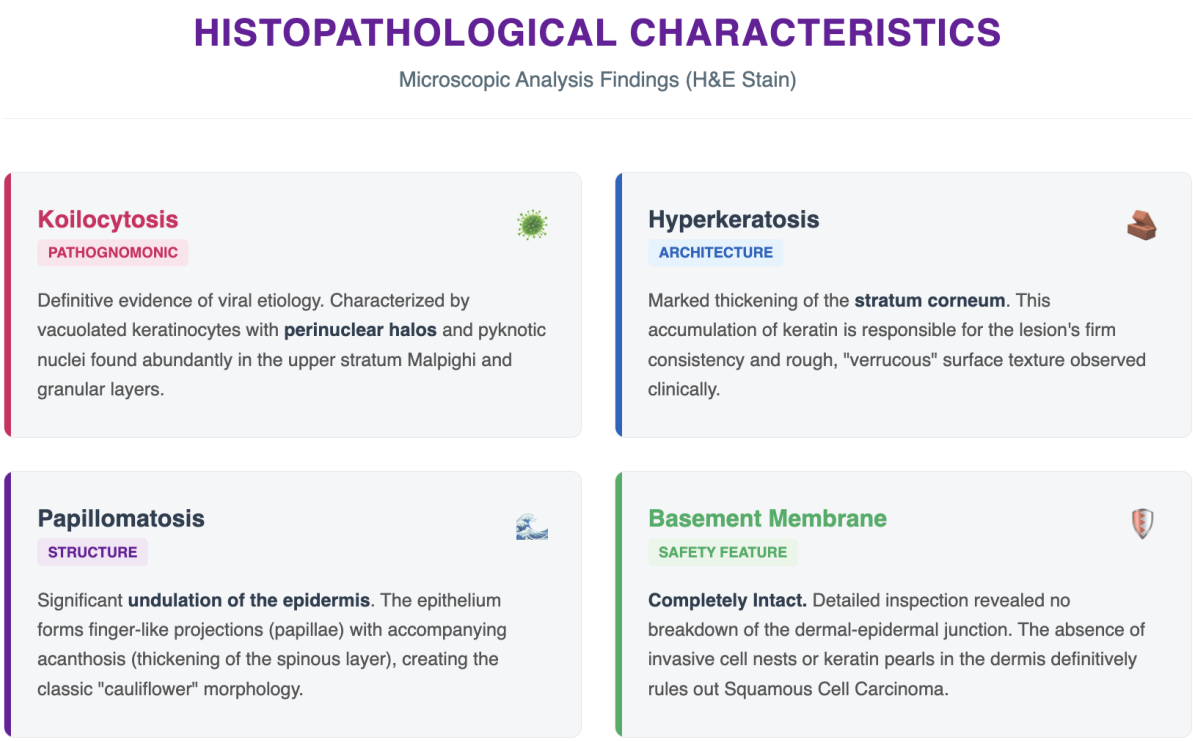

Figure 5. Summary of histopathological findings confirming the diagnosis of Verruca Vulgaris. The analysis highlights the presence of **Koilocytosis** as the pathognomonic sign of HPV infection. Crucially, the integrity of the **Basement Membrane** distinguishes this giant benign lesion from its malignant mimics (Verrucous Carcinoma and SCC), validating the safety of the surgical excision.

The patient's postoperative recovery was smooth and uncomplicated. The surgical wound healed by primary intention without any signs of dehiscence, hematoma, or infection. Sutures were removed one week after the surgery. At the six-month follow-up appointment, the patient showed no clinical evidence of recurrence. The cosmetic outcome was judged to be excellent, with the scar well-hidden in the post-auricular crease and no distortion of the auricular contour or position, detailed in Figure 6.

3. Discussion

The genesis of verruca vulgaris on the auricle represents a significant deviation from the standard epidemiological patterns of human papillomavirus infection. To fully understand the rarity of this case, one must first appreciate the unique anatomical and immunological microenvironment of the external ear. The auricular skin is biologically distinct; it is exceptionally thin, possesses a scant subcutaneous layer, and on the anterior surface, is tightly adherent to the underlying perichondrium.[11] This structural configuration creates a hostile environment for the establishment of viral reservoirs compared to the thick, hyperkeratotic skin of the palms and soles, which provides ample depth for the viral life cycle. Furthermore, the auricle is generally shielded from the heavy viral inoculum load typically found on

communal surfaces that drive the transmission of plantar and palmar warts. In this specific case, the mechanism of infection is hypothesized to be autoinoculation or fomite transmission. The human papillomavirus is a resilient, non-enveloped virus capable of surviving desiccation on inanimate objects for prolonged periods.[12] The use of shared or non-sterile ear-cleaning tools serves as a potent vector, creating the necessary micro-abrasions in the delicate auricular epidermis to allow viral particles to bypass the stratum corneum and access the basal keratinocytes.

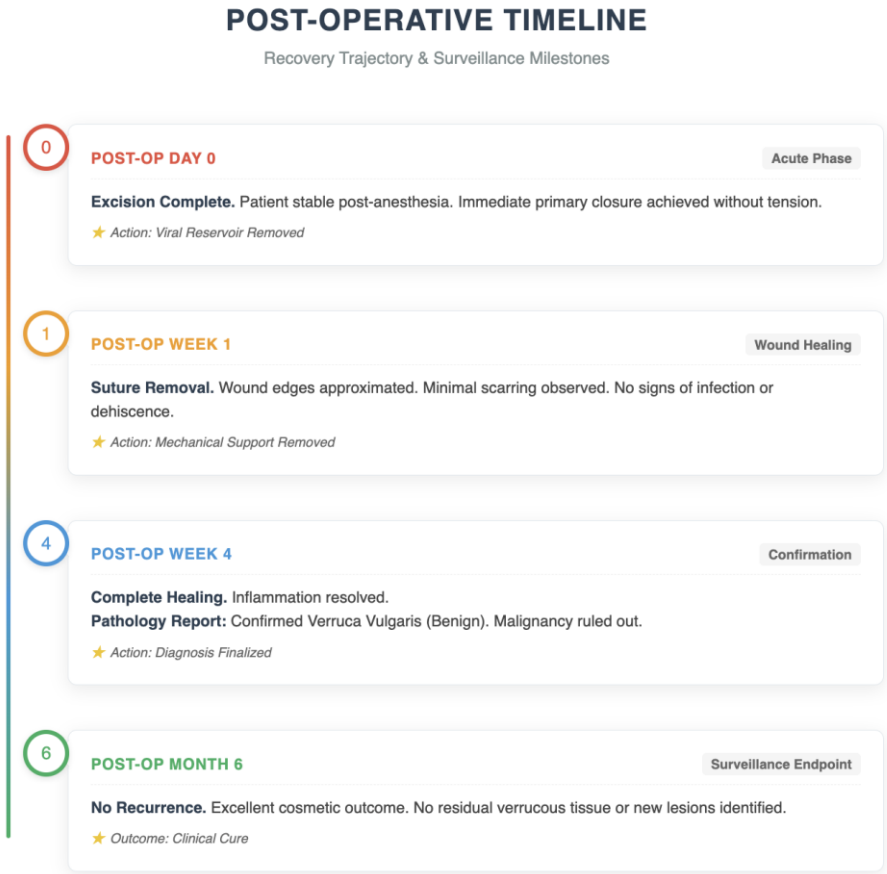

Figure 6. Longitudinal timeline of the post-operative course. The recovery was divided into four critical phases: Acute (Day 0), Wound Maturation (Week 1), Diagnostic Confirmation (Week 4), and Long-term Surveillance (Month 6). The absence of recurrence at the 6-month interval validates the efficacy of the surgical excision.

Once the virus successfully breaches this barrier and enters the basal stem cells, it initiates a complex hijack of the cellular machinery, establishing a latent infection that serves as the seed for the decade-long growth observed in this patient. The following narrative provides a detailed, scientific explication of the pathophysiological framework illustrated in Figure 7. This schematic diagram synthesizes the complex interaction between the human papillomavirus (HPV) and the host immune system over a ten-year timeline, delineating the specific biological mechanisms that permitted the development of a giant (3.3 cm) lesion in an immunocompetent host. The progression is categorized into four distinct evolutionary phases: Viral entry, immune evasion, cellular hijack, and the resultant giant phenotype. The pathogenesis of the giant auricular verruca begins with Phase I, titled viral entry, which represents the initial breach of the host's

anatomical defenses. The auricle, unlike the acral surfaces of the hands and feet, is not a primary reservoir for HPV. The skin of the posterior auricle is characterized by a thin epidermis and a paucity of the thick hyperkeratotic layers that typically harbor viral particles in plantar warts.[13] Therefore, the establishment of infection in this region necessitates a specific vector and a breach in the epithelial barrier. As depicted in the first quadrant of the figure, the virus exploits micro-abrasions within the stratum corneum to gain access to the basal keratinocytes. In the context of the presented case, the mechanism of inoculation is hypothesized to be either autoinoculation (transfer of viral particles from a subclinical lesion elsewhere on the body via digital manipulation) or fomite transmission (introduction of the virus via contaminated objects such as ear-cleaning tools or barber equipment).[14] HPV is a non-enveloped, double-stranded DNA virus with high environmental stability, capable of surviving desiccation on inanimate surfaces for prolonged periods.[15] Once the virus encounters a disruption in the epithelial integrity, it targets the basal stem cells or transit-amplifying cells located at the dermal-epidermal junction. Interaction with heparin sulfate proteoglycans and cell-surface receptors, such as alpha-6 integrins, facilitates viral internalization. This initial infection event is often clinically silent; the viral genome enters the nucleus and establishes itself as an episome (extrachromosomal DNA), marking the beginning of a latent infection that sets the stage for the subsequent decade of unchecked growth. Phase II, labeled immune evasion, constitutes the most scientifically critical component of the figure, explaining the chronological anomaly of a ten-year persistence in a healthy individual. Typically, cutaneous HPV infections are self-limiting, with the host's adaptive immune system—specifically CD4+ T-helper cells and CD8+ cytotoxic T-lymphocytes—recognizing and clearing the infection within 12 to 24 months. The progression of this specific lesion to a giant state over a decade implies a profound failure of local immune surveillance, a phenomenon often described as immunological ignorance. The figure highlights three primary mechanisms employed by the virus to maintain this stealth profile: 1. Absence of Viremia: Unlike many other viral pathogens, HPV does not have a blood-borne phase. The infection is strictly intra-epithelial, confined to the keratinocytes above the basement membrane. This physical isolation prevents the exposure of viral antigens to the systemic immune system and lymph nodes, significantly delaying the priming of an adaptive immune response. 2. Downregulation of Innate Sensors: The schematic emphasizes the downregulation of Toll-Like Receptors (TLRs), specifically TLR9, which acts as a sentinel for double-stranded DNA. The viral oncoproteins E6 and E7 actively interfere with the interferon signaling pathways (IFN-alpha and IFN-beta), effectively blinding the innate immune system to the presence of foreign viral DNA within the cell. 3. Low Protein Expression: In the basal layers where the virus resides, viral protein expression is kept to an absolute minimum. By limiting the production of capsid proteins (L1 and L2) to the most superficial, terminally differentiated layers of the skin (which are destined to desquamate), the virus avoids presenting antigenic targets to Langerhans cells in the lower epidermis. This phase represents a biological standoff where the virus replicates slowly and silently, avoiding the inflammatory triggers that would otherwise alert the host's defenses. It is this stealth capacity that allowed the lesion to persist and expand linearly from 2014 to 2024 without triggering the spontaneous regression typically seen in common warts. As the infection becomes established, the process moves to Phase III, cellular hijack, where the virus begins to actively reprogram the host cell's machinery to favor proliferation over differentiation. This phase is responsible for the histological architecture observed in the biopsy—specifically acanthosis (thickening of the spinous layer) and papillomatosis. The figure identifies the viral early proteins, E6 and E7, as the drivers of this cellular reprogramming. Even in low-risk HPV subtypes associated with benign warts (such as types 1, 2, or 57), E6 and E7 manipulate cell cycle

regulators. E7 binds to the Retinoblastoma protein (pRb), releasing the transcription factor E2F and pushing the keratinocyte into the S-phase of the cell cycle. Simultaneously, E6 interferes with p53 to prevent apoptosis, ensuring the cell survives despite this unscheduled replication. This uncoupling of differentiation and proliferation results in the massive expansion of the epidermal layers. The figure also notes the development of koilocytosis, the pathognomonic sign of HPV infection. Koilocytes are functionally compromised keratinocytes in the upper epidermis that exhibit perinuclear vacuolization due to the disruption of the cytokeratin network by the E4 viral protein.[16] This phase illustrates the transition from a latent infection to a productive, proliferative lesion, where the machinery of the host skin is completely co-opted to serve as a factory for viral assembly. The final quadrant, Phase IV, depicts the giant phenotype, which is the cumulative macroscopic result of the preceding three phases. After ten years of evading immunity and hijacking cellular proliferation, the lesion reaches the clinically giant dimension of 3.3 cm × 1.5 cm × 1.0 cm. The figure characterizes this phenotype as exophytic and verrucous. The term giant here is not merely a descriptor of size but a marker of biological potential. The sheer volume of the tumor burden (hyperkeratotic tissue) creates a physical sanctuary for the virus. The thick stratum corneum protects the active viral layers from environmental insults and topical treatments. Despite the alarming size, the figure crucially notes the benign status of the lesion. Unlike carcinomas, which invade the dermis, the giant verruca expands outward (exophytic growth) rather than downward (invasive growth). The basement membrane remains intact, acting as the definitive boundary between this massive viral factory and the systemic circulation. In synthesis, Figure 7 illustrates a linear biological narrative: a traumatic entry event (Phase I) is followed by a prolonged period of undetected latency and immune suppression (Phase II), allowing viral oncoproteins to drive abnormal cellular proliferation (Phase III), which ultimately manifests as a giant, disfiguring, yet benign tumor (Phase IV). This schematic provides the theoretical underpinning for the clinical case, explaining why a simple wart, usually a transient nuisance of childhood, evolved into a surgical challenge requiring extensive excision and reconstruction in an adult male. It underscores the critical concept that giant morphology is a function of time multiplied by immune evasion, rather than intrinsic malignancy.[17]

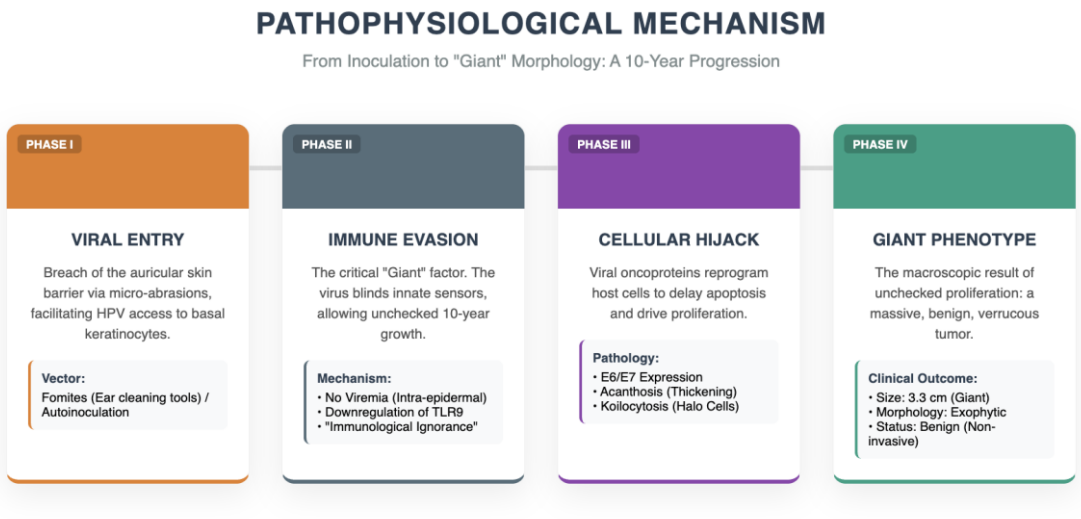

Figure 7. Schematic of the main pathophysiological concept. The progression begins with **autoinoculation** (Phase I) into the basal layer. The crucial factor permitting "Giant" growth is **Immune Evasion** (Phase II), where the virus downregulates Toll-Like Receptors (TLRs) to avoid detection for a decade. This allows the viral machinery (Phase III) to drive continuous **Acanthosis**, resulting in the massive 3.3 cm clinical lesion (Phase IV) without malignant invasion.

The most scientifically compelling aspect of this case is not merely the presence of the wart, but its persistence and giant growth over a ten-year period in an immunocompetent host.[18] Under normal physiological conditions, the host immune system is highly efficient at recognizing and clearing cutaneous HPV infections, typically resolving them within two years via a robust cell-mediated response driven by CD4+ Th1 cells and cytotoxic CD8+ T-cells. The fact that this lesion grew linearly for a decade implies a sophisticated state of localized immune evasion, or immunological ignorance. HPV has evolved multiple mechanisms to fly under the radar of the host's surveillance systems. Primary among these is the virus's ability to remain strictly intra-epidermal, thereby avoiding viremia and the subsequent exposure to blood-borne immune sentinels. Furthermore, the viral oncoproteins E6 and E7 actively downregulate the expression of Toll-Like Receptors, specifically TLR9, which are the innate immune sensors responsible for detecting foreign viral DNA. By blinding these sensors, the virus effectively prevents the activation of the interferon pathway, the body's primary antiviral alarm system. In the basal layer of the epithelium, the virus replicates its genome as episomes but maintains an extremely low protein expression profile, rendering the infected cells effectively invisible to immune patrols. In our patient, these mechanisms likely created a localized zone of immune tolerance at the posterior auricle, allowing the lesion to expand unchecked until it reached giant proportions.

The definition of a giant verruca vulgaris is not merely a descriptive term regarding size; it is a clinical classification with profound prognostic implications. In this study, we define a giant wart as any verrucous lesion exceeding 2 centimeters in diameter.[19] This threshold is critical because it marks the inflection point where the clinical probability of malignancy begins to rise significantly. The gray zone between a giant benign wart and a verrucous carcinoma is navigated by the complex interaction between the viral genome and environmental co-carcinogens. The ear is a site of high cumulative ultraviolet radiation exposure. In the context of high-risk HPV subtypes, the E6 protein targets and degrades the tumor suppressor protein p53, leading to genomic instability. While verruca vulgaris is typically associated with low-risk HPV types such as 1, 2, or 57, the E6 proteins of these subtypes still bind to p53, albeit with lower affinity. Over a prolonged period of ten years, the combination of chronic viral replication, inflammation, and UV-induced somatic mutations can theoretically drive a benign lesion toward malignant transformation. This theoretical risk of oncogenesis is the primary justification for the aggressive surgical approach employed in this case, rather than a wait and see strategy.

The central challenge in managing giant auricular masses lies in the rigorous differentiation between three clinically similar but biologically distinct entities: Giant verruca vulgaris, verrucous carcinoma, and squamous cell carcinoma. This differentiation cannot be reliably achieved through visual inspection alone, as all three can present as exophytic, cauliflower-like masses. Giant Verruca Vulgaris, as confirmed in this case, is characterized histologically by koilocytosis, hyperkeratosis, and inward-bending rete ridges that remain contained above the basement membrane. Clinically, the lesion retains mobility over the underlying structures, indicating a lack of deep invasion. verrucous carcinoma, often referred to as Ackerman's tumor, represents the most dangerous mimic in this differential diagnosis. It is a low-grade, slow-growing malignancy that bears a striking resemblance to a giant wart. However, its histological signature is distinct and subtle; it lacks the severe cellular atypia seen in standard carcinoma and instead features a pushing border—broad, bulbous rete ridges that penetrate deep into the dermis and underlying tissues. This pushing border is the critical diagnostic feature that is frequently missed by superficial shave biopsies, which only capture the surface keratin. A misdiagnosis of verrucous carcinoma as a benign wart leads to inadequate treatment and high recurrence rates. Squamous cell

carcinoma represents the aggressive end of the spectrum, characterized by invasive islands of keratinocytes, the formation of keratin pearls, and high nuclear atypia. Clinically, these lesions are often fixed to the cartilage and may exhibit ulceration and bleeding, features that were notably absent in our patient.

The management of giant auricular lesions requires a delicate balance between oncological safety and cosmetic preservation. While cryotherapy and topical agents are the standard of care for small common warts, they are contraindicated for giant lesions for two reasons. First, the physics of thermal transfer limits the efficacy of liquid nitrogen in penetrating a thick, hyperkeratotic mass, often leaving the viral base intact. Second, aggressive freezing on the ear carries a high risk of chondritis, which can lead to cartilage necrosis and permanent deformity. Therefore, surgical excision is the gold standard. In this case, the decision to perform a total excision with a narrow margin was validated by the benign pathology. However, the reconstruction of the resulting defect presented a technical challenge. A 4-centimeter defect on the anterior surface of the ear would typically require a skin graft or a complex flap. However, the posterior surface of the auricle possesses a reservoir of loose, redundant skin. By utilizing a technique of extensive undermining—separating the skin from the underlying subcutaneous tissue towards the mastoid and the neck—we were able to mobilize sufficient tissue to advance the skin flaps and achieve primary closure. This approach effectively closed the giant defect without pinning the ear back or altering the auriculo-cephalic angle. This demonstrates that even massive defects on the posterior auricle can be managed with simple closure techniques if the surgeon possesses a thorough understanding of tissue biomechanics and undermining planes, avoiding the need for more complex and morbid procedures like the Postauricular Helix-based Adipodermal-pedicle Turnover flap.[20]

Although the patient demonstrated no evidence of recurrence at the six-month follow-up, the biology of HPV dictates a cautious long-term prognosis. HPV DNA is known to persist in clinically normal-appearing perilesional skin, a phenomenon known as latency. This viral reservoir can serve as a source for recurrence even after complete excision of the visible lesion. Consequently, while the patient is currently considered cured, the potential for reactivation remains, particularly if the local immune surveillance is compromised. Continued surveillance is therefore essential, not only to detect recurrence of the wart but to monitor for any delayed signs of malignant transformation that may have been missed in the initial assessment.

4. Conclusion

This case report documents the rare and instructive occurrence of a giant verruca vulgaris on the posterior auricle of a 36-year-old male, highlighting the diagnostic challenges posed by lesions that defy standard clinical dimensions. We conclude that size acts as an independent risk factor in the evaluation of auricular masses; any verrucous lesion exceeding 2 centimeters should be clinically classified as a tumor of uncertain potential. In such scenarios, excisional biopsy supersedes ablative therapies as the treatment of choice. This surgical approach provides the dual benefit of immediate aesthetic restoration and, most importantly, the provision of a complete, intact tissue specimen that allows the pathologist to evaluate the deep basement membrane—the only definitive method to rule out the deceptive pushing border of verrucous carcinoma. We further conclude that despite the daunting size of such lesions, primary surgical closure is often achievable through meticulous undermining, offering excellent cosmetic outcomes without the need for complex flaps. Clinicians are encouraged to maintain a high index of suspicion for malignancy in all chronic auricular masses and to adopt an aggressive diagnostic stance to prevent the misdiagnosis of treatable neoplasms.

5. References

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