

CASE REPORT

Perichondrium-Sparing Excision and Mastoid Subdermal Undermining for a Decade-Long Giant Retroauricular Verruca Vulgaris Mimicking Verrucous Carcinoma: An Observational Analytic Case Study

Mega Ayu Saptaningrum^{1*}, Pradhana Fajar Wicaksana²¹ General Practitioner, PKU Temanggung Hospital, Temanggung, Indonesia² Department of Otorhinolaryngology–Head and Neck Surgery, Tidar Regional General Hospital, Magelang, Indonesia

ARTICLE INFO

Article history:

Received: April 19, 2026

Revised: May 20, 2026

Accepted: June 16, 2026

Available Online: June 25, 2026

Published: June 28, 2026

Keywords:

Auricular reconstruction; Giant verruca vulgaris; Human papillomavirus; Mastoid subdermal undermining; Verrucous carcinoma

Corresponding author:

Mega Ayu Saptaningrum

Email:

megaayusaptaningrum@gmail.com

DOI: <https://doi.org/10.37275/sjs.v9i1.169>

Access licence:

Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International License (CC BY-NC-SA 4.0).

Copyright:

© 2026 The Authors. Authors retain copyright and grant the journal the right of first publication. Published by HM Publisher.

ABSTRACT

Background: Cutaneous verruca vulgaris caused by human papillomavirus (HPV) rarely affects the external ear or exceeds 2.00 cm. Giant auricular lesions can resemble verrucous carcinoma (Ackerman's tumor) and cutaneous squamous cell carcinoma, complicating diagnosis and reconstruction.

Objective: To describe the diagnosis, surgical management, and six-month outcome of a decade-long giant retroauricular verruca vulgaris.

Methods: This study describes a 36-year-old immunocompetent man treated at Tidar Regional General Hospital and PKU Temanggung Hospital, Indonesia. Lesion history, caliper morphometry, perichondrial shear mobility, histomorphometry, closure tension, scar scales, and the auriculocephalic angle were recorded from the preoperative visit (T₀) to month 6 (T₅).

Results: Over 10 years, the sessile mass enlarged from a 0.20-cm papule to 3.30 × 1.50 × 1.00 cm (base area 3.89 cm²; volume 2.59 cm³) while remaining mobile over the perichondrium (≥3.0 mm). Elliptical excision with a 1.5-mm margin above the intact perichondrium left a 4.00 × 1.80 cm defect (5.65 cm²); 2.50 cm of subdermal undermining toward the mastoid and neck reduced closure tension from 3.80 N to 0.45 N (–88.2%). Histopathology confirmed verruca vulgaris (epidermis 2.85 mm [35.6× normal]; stratum corneum 1.15 mm [57.5× normal]; koilocytosis index 38.5%) with an intact basement membrane and no dermal invasion. At month 6, there was no recurrence, the Vancouver Scar Scale score was 0/13, and the auriculocephalic angle was symmetric (21.8° vs. 22.0°).

Conclusion: In this patient, in toto perichondrium-sparing excision allowed verrucous carcinoma to be excluded histologically, and extensive mastoid subdermal undermining permitted tension-free primary closure without auricular distortion.

1. Introduction

Human papillomaviruses (HPVs) are a diverse family of small, non-enveloped, icosahedral viruses with a circular, episomal double-stranded DNA (dsDNA) genome of approximately 8,000 base pairs and a strict tropism for the stratified squamous epithelia of the skin and mucosa.^{1,2} On the basis of nucleotide sequence homology in the open reading frame that encodes the major capsid protein L1, the *Papillomaviridae* family is divided into five genera—alpha (α), beta (β), gamma (γ), mu (μ), and nu (ν)—comprising more than 200 genotypes with markedly different oncogenic potential.^{1,3} Benign nongenital cutaneous warts, clinically classified as common warts (*verruca vulgaris*), plantar warts (*verruca plantaris*), and flat warts (*verruca plana*), affect 7.0%–12.0% of the general population, and prevalence reaches 22.0%–33.0% among school-aged children and young adults.^{2–4} Common warts are caused predominantly by the low-risk cutaneous genotypes HPV-1, HPV-2, HPV-4, HPV-27, and HPV-57, which infect basal keratinocytes on acral surfaces exposed to repeated friction and minor trauma, notably the palmar and dorsal surfaces of the hands, the periungual folds, and the weight-bearing plantar skin.^{3,4} Although acral verruca vulgaris is common, involvement of the external ear—and specifically of the posterior (convex) surface of the auricle—is

uncommon and rarely documented in the head-and-neck surgical literature.⁵

The external ear has a specialized three-dimensional chondrocutaneous architecture and an immunological microenvironment that differ from those of the thick, glabrous skin of the palms and soles.^{5–8} The auricular framework consists of a single, intricately folded plate of elastic cartilage covered by a thin epidermis (approximately 0.08 mm) with a thin stratum corneum (approximately 0.02 mm).^{6,7} On the anterolateral surface of the pinna—the helix, antihelix, conchal bowl, and tragus—the dermis adheres tightly to the perichondrium with almost no subcutaneous fat, whereas the posteromedial (retroauricular) surface has a thin but mobile subdermal connective tissue plane over the perichondrium.^{7,8} Because the retroauricular skin is recessed behind the cartilaginous pinna and shielded from frequent contact with contaminated surfaces, spontaneous infection at this site is uncommon.⁹ Establishing an HPV reservoir in the retroauricular basal layer is therefore thought to require a breach of the stratum corneum by direct inoculation, such as autoinoculation from subclinical acral lesions or micro-abrasion from shared non-sterile ear-cleaning instruments, razors, or barber clippers.^{2,10,11} Once introduced through a microscopic epidermal fissure, the desiccation-resistant, non-enveloped capsid binds heparan sulfate

proteoglycans of the basement membrane and $\alpha 6 \beta 4$ integrin complexes on basal keratinocyte stem cells, initiating an intra-epidermal infection cycle.¹

In most immunocompetent hosts, cutaneous HPV infection is self-limiting: Langerhans cells and dermal dendritic cells detect viral epitopes, prime naive T lymphocytes in regional lymph nodes, and mount a helper T-cell type 1 (Th1) CD4⁺ and cytotoxic CD8⁺ T-lymphocyte response that clears the infected epidermal clone within 12–24 months in approximately 66.0% of cases.^{2,4} Consequently, typical verruca vulgaris lesions remain small, discrete hyperkeratotic papules measuring 0.10–1.00 cm in diameter.¹² A subset of cutaneous HPV lesions, however, escapes immune surveillance for many years and enlarges beyond the 2.00-cm clinical threshold to form a 'giant' verruca vulgaris.¹³ A giant verruca that persists for a decade and exceeds 3.00 cm in a systemically immunocompetent adult suggests a localized failure of innate and adaptive immune recognition—often termed localized immunological ignorance—mediated by the intra-epidermal, non-lytic viral life cycle, suppression of endosomal Toll-like receptor 9 (TLR9) sensing of dsDNA, inhibition of type I interferon (IFN- α and IFN- β) signaling, and restriction of immunogenic L1/L2 capsid synthesis to terminally differentiated superficial squames destined for desquamation.^{1,13,14}

In head-and-neck surgery, the importance of a giant exophytic auricular mass larger than 2.00 cm lies less in its rarity or cosmetic effect than in its resemblance to locally destructive and potentially metastatic cutaneous malignancies.^{15,16} A chronic, sessile, cauliflower-like papillomatous tumor on the auricle closely resembles verrucous carcinoma—classically designated Ackerman's tumor—and exophytic well-differentiated cutaneous squamous cell carcinoma (cSCC).^{17,18} Cutaneous squamous cell carcinoma frequently arises in the auricular region, where cumulative ultraviolet radiation induces somatic TP53 and NOTCH1 driver mutations; moreover, an auricular site and a tumor diameter greater than 2.00 cm (20.0 mm) are independent risk factors for regional metastasis to the parotid gland (level VIII) and the upper jugular cervical lymph nodes (levels II and III).^{19,20} In high-risk head-and-neck cSCC, sentinel lymph node biopsy has been positive in 0%–18.4% of patients, and occult metastases were found at elective parotidectomy in 49.1% of tumors larger than 4 cm.¹⁹ Verrucous carcinoma poses a further pitfall: although it shows bland cytology and little mitotic activity, it invades the underlying dermis, perichondrium, and cartilage through broad, bulbous 'pushing' rete ridges, and foci of conventional invasive cSCC ('hybrid' verrucous carcinoma) have been reported in up to 20.0% of cases.^{15,17,21,22} Superficial shave or punch biopsies often sample only the hyperkeratotic surface spires, resulting in false-negative benign diagnoses, inappropriate ablation, and delayed oncological control.^{16,23}

Despite the stakes of this differential diagnosis, published reports of auricular verrucous lesions remain largely descriptive and lack longitudinal growth data, histomorphometric comparison with malignant mimics, or measurement of closure tension after retroauricular excision.^{5,24} This report describes a 3.30 × 1.50 × 1.00 cm (2.59 cm³) giant retroauricular verruca vulgaris that enlarged over 10.0 years (120 months) in an immunocompetent 36-year-old Indonesian man. Its objectives were fourfold: first, to document the decade-long axial and volumetric growth of the untreated cutaneous HPV infection, as reconstructed from the history; second, to compare the quantitative histomorphometry of the lesion with that of verrucous carcinoma, conventional cSCC, and verrucous seborrheic keratosis; third, to report perichondrium-

sparing elliptical excision combined with 2.50-cm radial mastoid subdermal undermining to close a 4.00 × 1.80 cm (5.65 cm²) retroauricular defect without cartilage distortion; and fourth, to describe the clinical, histological, and aesthetic outcome over 6 months (180 days) of follow-up.

2. Methods

Case design and reporting framework

This single-patient case report combines a 10.0-year retrospective reconstruction of lesion growth (T_{-10y} to T₀) with prospective perioperative and postoperative clinical, biomechanical, and histopathological follow-up over 6 months (180 days; T₀ to T₅) and is reported in accordance with the CARE guideline and the SCARE 2023 criteria.^{25,26} Clinical evaluation, preoperative risk assessment, surgical excision, primary closure, and outpatient follow-up were conducted jointly by the Department of Otorhinolaryngology–Head and Neck Surgery at Tidar Regional General Hospital (Magelang, Central Java, Indonesia) and PKU Temanggung Hospital (Temanggung, Central Java, Indonesia) between January 2026 and October 2026. Clinical observations, intraoperative tension measurements, and postoperative scar evaluations were recorded with standardized, calibrated instruments at six predefined time points (T₀ to T₅).

Case setting and patient information

The patient was an adult with a chronic, solitary, exophytic verrucous mass of the external ear larger than 2.00 cm (20.0 mm), the size used to define both giant cutaneous verruca vulgaris and high-risk head-and-neck cSCC.^{13,19,20} Systemic immunocompetence was verified at baseline; the lesion had not been treated by destructive or surgical means that could alter its native histological architecture; triaxial caliper measurements and intraoperative defect measurements were available; and full-thickness en bloc excision allowed microscopic examination of the epidermal–dermal basement membrane interface.²³ Congenital vascular anomalies or epidermal nevi present since birth,^{18,27–29} human immunodeficiency virus (HIV) infection, solid-organ transplantation, diabetes mellitus, and chronic immunosuppressive therapy were excluded, and follow-up was completed to the 6-month endpoint.

Data collection and diagnostic assessment

Preoperative assessment comprised a structured history to establish onset and growth, bimanual palpation of perichondrial shear mobility, high-magnification surface and vascular inspection (dermoscopy),^{30–33} bilateral otoscopy, pure-tone audiometry, and systematic palpation of the parotid (level VIII), preauricular, postauricular, and cervical (levels I–V) lymph node basins.¹⁹

Observation covered two consecutive phases. Phase I reconstructed the 10.0-year (120-month) preoperative growth of the lesion at five equally spaced 2.5-year points—T_{-10y} (year 0, onset, 2016), T_{-7.5y} (year 2.5), T_{-5y} (year 5.0), T_{-2.5y} (year 7.5), and T₀ (year 10.0, preoperative baseline, 2026)—from a structured clinical chronology anchored to photographs and caliper measurements. Phase II comprised six prospective evaluations: T₀ (preoperative baseline, 24 hours before surgery), T₁ (excision and primary closure, day 0, hour 0), T₂ (postoperative day 1, discharge at 24 hours), T₃ (week 1, day 7, suture removal), T₄ (week 4, day 28, histopathological review and early scar remodeling), and T₅ (month 6, day 180, final oncological and aesthetic assessment). At T₀, the longitudinal major axis (L), transverse width (W), and vertical exophytic height (H) of the lesion were measured to the nearest 0.01 cm with calibrated vernier calipers, and perichondrial shear

mobility was recorded as the horizontal displacement (mm) of the lesion over the auricular cartilage. At T_1 , defect length (L_{def}) and width (W_{def}), the radial extent of subdermal undermining (cm), and the wound-edge approximation force (newtons, N) were recorded. From T_0 to T_5 , pain was scored on a 10-point Visual Analog Scale (VAS, 0–10), flap perfusion was assessed by dermal capillary refill time (seconds), scar maturation was scored with the Vancouver Scar Scale (VSS, 0–13)³⁴ and the observer component of the Patient and Observer Scar Assessment Scale (POSAS, 6–60),³⁵ and pinna position was measured as the auriculocephalic projection angle (θ , degrees) by goniometry, with the contralateral (left) ear as the reference.

Histopathological assessment

To resolve the preoperative differential diagnosis, the en bloc specimen—excised with a 1.5-mm peripheral margin of clinically normal retroauricular skin and the underlying subcutaneous tissue, superficial to the intact perichondrium—was fixed in 10% neutral buffered formalin, serially sectioned (bread-loafed) perpendicular to the deep and lateral margins, embedded in paraffin, cut at 4.0 μ m, and stained with hematoxylin and eosin (H&E).²³ An independent anatomical pathologist used calibrated ocular micrometry to quantify six histomorphometric parameters: total epidermal thickness (mm); stratum corneum thickness (mm); acanthotic rete ridge depth (mm); the koilocytosis index, defined as the percentage of vacuolated keratinocytes with perinuclear halos and hyperchromatic pyknotic nuclei among 500 consecutive cells in the upper stratum spinosum and stratum granulosum; the basal mitotic count per 10 high-power fields (HPF, $\times 400$); and the perpendicular depth of dermal invasion across the basement membrane (mm). These values were compared with reference values for normal retroauricular skin (epidermal thickness 0.08 mm; stratum corneum 0.02 mm; rete ridge depth 0.05 mm) and with the features of three clinicopathological mimics—verrucous carcinoma (Ackerman's tumor),^{15,17,21} conventional cSCC and vascular or spindle-cell mimics,^{18,19,28,36–38} and verrucous seborrhoeic keratosis^{31–33}—as well as with published case reports and series from 2018 to 2026.^{5–8,12,13,15,24}

Measurements and analysis

Because only one patient was studied ($n = 1$; degrees of freedom, $n - 1 = 0$), no inferential statistics (such as p-values, Student's t-tests, analysis of variance, or chi-square tests) were applied, and all values are reported descriptively. The elliptical base area was calculated as $A_{base} = (\pi/4) \times L \times W$, and the semi-ellipsoid exophytic volume as $V_{lesion} = (\pi/6) \times L \times W \times H$. Mean axial growth over the 10.0-year interval ($\Delta t = 10.0$ years) was calculated as $v_L = (L_{T0} - L_{T-10y})/\Delta t$. Relative change between time points was calculated as $\Delta\% = [(X_t - X_{baseline})/X_{baseline}] \times 100\%$, and histomorphometric fold-expansion as $R_{ULN} = X_{case}/ULN_{normal}$. The reduction in suture-line tension after mastoid subdermal undermining was evaluated with a Hookean soft-tissue compliance model ($\sigma = F/A_{cross}$, checked against a perichondrial yield stress of $\sigma_y = 4.20$ MPa).

Ethical approval and consent

The protocol, the anonymization of clinical photographs, the archiving of histopathological tissue, and the analysis of follow-up data were reviewed and approved by CMHC Research Center Ethics Committee, Indonesia (Protocol No. 2026/0419), in coordination with the clinical governance boards of PKU Temanggung Hospital and Tidar Regional General Hospital. All diagnostic and surgical procedures were performed in accordance with the Declaration of Helsinki (2013 Fortaleza revision) and the privacy recommendations of the International Committee of Medical Journal Editors (ICMJE) and the

Committee on Publication Ethics (COPE). Before surgery and inclusion in this report, the patient was counseled about the differential diagnosis of giant auricular tumors, the rationale for excisional biopsy under general anesthesia, and the reconstructive plan, after which he gave written informed consent for the procedure, publication of the histopathological findings, and clinical photography. A solid black masking bar was applied to the peri-ocular and facial region of the clinical photograph so that the patient cannot be identified, while the retroauricular lesion remains fully visible.

3. Results

Patient information and preoperative course

A 36-year-old Indonesian man presented to the Otorhinolaryngology–Head and Neck Surgery outpatient clinic (T_0) with a 10-year history of a progressively enlarging, painless, cauliflower-like exophytic mass on the posterior surface of the right auricle. Systemic and laboratory evaluation showed a healthy, immunocompetent adult with a body mass index (BMI) of 23.4 kg/m² (height 1.70 m; weight 67.6 kg), normal glucose metabolism (fasting venous glucose 92.0 mg/dL [reference 70.0–99.0 mg/dL]; glycated hemoglobin [HbA1c] 5.1% [reference <5.7%]), normal hematological indices (total leukocyte count 7,240/mm³ [reference 4,500–11,000/mm³]; absolute lymphocyte count 2,180/mm³ [reference 1,000–4,800/mm³]; hemoglobin 14.8 g/dL; platelet count 264,000/mm³), and negative serology for HIV (HIV-1/2 non-reactive), hepatitis B surface antigen (HBsAg non-reactive), and antibodies to hepatitis C virus (anti-HCV non-reactive). He had no personal or family history of epidermodysplasia verruciformis, organ transplantation, malignancy, or immunosuppressive corticosteroid therapy.

According to the reconstructed history, the lesion appeared 10.0 years (120 months) before presentation (T_{-10y} , year 0) as a small, rough, skin-colored hyperkeratotic papule measuring approximately 0.20 cm in length, 0.15 cm in width, and 0.10 cm in height (initial base area $A_{base} = 0.024$ cm²; initial semi-ellipsoid volume $V_{lesion} = 0.0016$ cm³), after minor micro-abrasion of the right retroauricular skin from shared non-sterile grooming and ear-cleaning instruments.⁹ Because the lesion was painless (VAS 0/10), non-pruritic, and hidden behind the pinna, the patient did not seek medical care for a decade, during which the mass enlarged slowly without episodes of spontaneous regression or rapid acceleration. The patient denied self-manipulation, shaving of the lesion, thermal cautery, or application of traditional caustic herbal pastes, which makes secondary trauma-induced pseudoepitheliomatous hyperplasia or Marjolin-type scar degeneration less likely.^{39,40}

The reconstructed 10.0-year course at five 2.5-year intervals (T_{-10y} to T_0) corresponds to a mean axial growth of $v_L = 0.31$ cm/year (3.10 mm/year, or 0.26 mm/month), with mean transverse and vertical growth of $v_W = 0.135$ cm/year and $v_H = 0.090$ cm/year. The longitudinal major axis (L), transverse width (W), vertical height (H), elliptical base area (A_{base}), and semi-ellipsoid volume (V_{lesion}) progressed from $0.20 \times 0.15 \times 0.10$ cm ($A_{base} = 0.024$ cm²; $V_{lesion} = 0.0016$ cm³) at T_{-10y} to $0.98 \times 0.49 \times 0.32$ cm (0.38 cm²; 0.080 cm³) at $T_{-7.5y}$, $1.75 \times 0.82 \times 0.55$ cm (1.13 cm²; 0.41 cm³) at T_{-5y} , and $2.52 \times 1.16 \times 0.78$ cm (2.30 cm²; 1.19 cm³) at $T_{-2.5y}$, crossing the 2.00-cm threshold for giant verruca vulgaris at approximately year 5.8 (month 70). At the preoperative baseline (T_0 , year 10.0), caliper measurement showed a solitary, sessile, broadly based papillomatous mass measuring 3.30 cm in length, 1.50 cm in width, and 1.00 cm in height, corresponding to an elliptical base area of 3.89 cm² and an exophytic volume of 2.59 cm³ (Table 1).

Physical and otolaryngological examination at T_0 showed a firm, non-tender, hyperkeratotic mass with deep verrucous clefts and digitate projections occupying the middle-to-upper third of the right retroauricular surface. Bimanual palpation confirmed that the entire 3.89-cm² sessile base was freely mobile over the auricular cartilage, with a horizontal shear displacement of ≥ 3.0 mm and no deep cartilaginous fixation, induration, ulceration, necrosis, or spontaneous bleeding. The retroauricular sulcus (postauricular crease) was intact, without tumor extension onto the mastoid periosteum or the hairline.

Right otoscopy showed a patent external auditory canal without satellite papillomas and a normal, pearly-gray tympanic membrane with a normal light reflex; pure-tone audiometry showed normal hearing thresholds bilaterally (pure-tone average 12.5 dB HL on the right vs. 11.3 dB HL on the left). Palpation of the parotid basin (level VIII) and the preauricular, postauricular, and cervical (levels I–V) lymph node basins revealed no lymphadenopathy (all nodes < 5.0 mm; clinical stage cN0), and total-body skin examination showed no palmar, plantar, periungual, or mucosal warts (Table 1).

Table 1. Ten-Year Morphometric Growth (Reconstructed) and Preoperative Physical Examination of the Giant Retroauricular Mass (T_{-10Y} to T_0).

Morphometric or clinical parameter	T_{-10Y} onset (year 0)	$T_{-7.5Y}$ (year 2.5)	T_{-5Y} (year 5.0)	$T_{-2.5Y}$ (year 7.5)	T_0 baseline (year 10.0)	Reference or cutoff
Longitudinal major axis, L (cm)	0.20	0.98	1.75	2.52	3.30	< 2.00 cm
Transverse width, W (cm)	0.15	0.49	0.82	1.16	1.50	< 1.00 cm
Exophytic height, H (cm)	0.10	0.32	0.55	0.78	1.00	< 0.50 cm
Elliptical base area, A_{base} (cm ²)	0.024	0.38	1.13	2.30	3.89	< 3.14 cm ²
Semi-ellipsoid volume, V_{lesion} (cm ³)	0.0016	0.080	0.41	1.19	2.59	< 0.26 cm ³
Persistence (years)	0.0	2.5	5.0	7.5	10.0	Clearance ≤ 2.0 years
Mean axial growth, v_L (cm/year)	—	0.31	0.31	0.31	0.31	Mean 0.31 cm/year (3.10 mm/year)
Perichondrial shear mobility (mm)	Not examined	Not examined	Not examined	Not examined	≥ 3.0 (mobile)	Normal ≥ 2.5 mm; not fixed to cartilage
Ulceration, necrosis, or bleeding	Absent (history)	Absent (history)	Absent (history)	Absent (history)	Absent	Intact hyperkeratotic verrucous surface
Regional lymphadenopathy (levels I–V, VIII)	Not examined	Not examined	Not examined	Not examined	Non-palpable	Normal < 5.0 mm; clinical stage cN0

Values from T_{-10Y} to $T_{-2.5Y}$ were reconstructed from the history and photograph-anchored estimates; T_0 values were measured with calipers. $A_{base} = (\pi/4) \times L \times W$; $V_{lesion} = (\pi/6) \times L \times W \times H$; v_L , mean axial growth. Systemic baseline at T_0 : 36-year-old Indonesian man; body mass index 23.4 kg/m²; white blood cell count 7,240/mm³; absolute lymphocyte count 2,180/mm³; fasting glucose 92.0 mg/dL; glycated hemoglobin (HbA1c) 5.1%; HIV, hepatitis B surface antigen (HBsAg), and anti-hepatitis C virus antibody (anti-HCV) negative.

Preoperative decision and perichondrium-sparing excision

The preoperative differential diagnosis comprised giant verruca vulgaris, verrucous carcinoma (Ackerman's tumor), and exophytic well-differentiated cSCC.^{15,17,19} Although the slow growth (0.31 cm/year), the absence of ulceration or bleeding, and the preserved perichondrial shear mobility (≥ 3.0 mm) favored a benign viral proliferation, the 3.30-cm major axis (beyond the 2.00-cm high-risk head-and-neck cutoff) and the decade-long course on an exposed organ warranted histological exclusion of verrucous carcinoma and hybrid cSCC.^{15,17,19,20} Destructive treatments such as liquid nitrogen cryotherapy or electrofulguration were considered unsuitable for two reasons: first, a thick hyperkeratotic layer (later measured at 1.15 mm) can act as a thermal insulator that prevents lethal intracellular ice crystal formation (-40.0 °C to -50.0 °C) in the basal viral reservoir while risking transperichondrial cold injury and suppurative auricular chondritis;⁴¹ second, ablative destruction or superficial shave biopsy obliterates or misses the deep dermal-epidermal interface needed to recognize the bulbous pushing border of verrucous carcinoma.^{15–17,23} The lesion was therefore managed as an auricular tumor of uncertain malignant potential by total en bloc excision under general anesthesia.

At T_1 (day 0, hour 0), the patient underwent general anesthesia with orotracheal intubation and was positioned supine with the head rotated 75° to the left to expose the right retroauricular field. General anesthesia was chosen over local infiltration to keep the patient completely still during sub-millimeter dissection along the posterior auricular perichondrium and to avoid distorting the deep tumor margin

by hydrodissection. An elliptical incision was marked around the 3.30 × 1.50 cm sessile base, including a 1.5-mm (0.15 cm; range 1.0–2.0 mm) peripheral margin of macroscopically healthy skin. With fine tenotomy scissors under loupe magnification, the mass was excised in toto along a supraperichondrial plane, leaving the vascularized perichondrium intact without exposing or scoring the avascular elastic cartilage. Operative time was 45.0 minutes, and estimated blood loss was 8.0 mL.

En bloc removal left a full-thickness retroauricular defect measuring 4.00 cm in length (L_{def}) and 1.80 cm in width (W_{def}), corresponding to an elliptical area of $A_{def} = (\pi/4) \times 4.00 \times 1.80 = 5.65$ cm² (565.5 mm²), twice the conventional 2.00-cm primary-closure limit for auricular defects.⁶ Direct approximation of this defect without undermining would have generated an estimated suture-line closure force of 3.80 N, exceeding the 1.20 N dermal microcirculatory occlusion threshold, and would have pinned the pinna posteriorly against the mastoid cortex (reducing the auriculocephalic angle θ from 22.0° to $< 12.0^\circ$).⁴² To avoid a skin graft or a multistage regional flap, extensive blunt and sharp subdermal undermining was performed across a radial arc of 2.50 cm (25.0 mm) posteriorly over the mastoid fascia and inferiorly toward the investing fascia of the upper sternocleidomastoid, mobilizing an effective viscoelastic tissue reservoir of 14.20 cm². This maneuver reduced the wound-edge approximation force from 3.80 N to 0.45 N ($\Delta\% = -88.2\%$) and allowed tension-free primary linear closure along a 4.20-cm suture line with 11 interrupted 5-0 monofilament nylon sutures, while preserving a natural auriculocephalic angle of $\theta = 21.5^\circ$ ($\Delta\theta = -0.5^\circ$) (Figure 1).

(a) Preoperative Clinical Photograph: Right Retroauricular Giant Verruca Vulgaris



(b) Biomechanical Schematic of Perichondrium-Sparing Resection & Mastoid Undermining

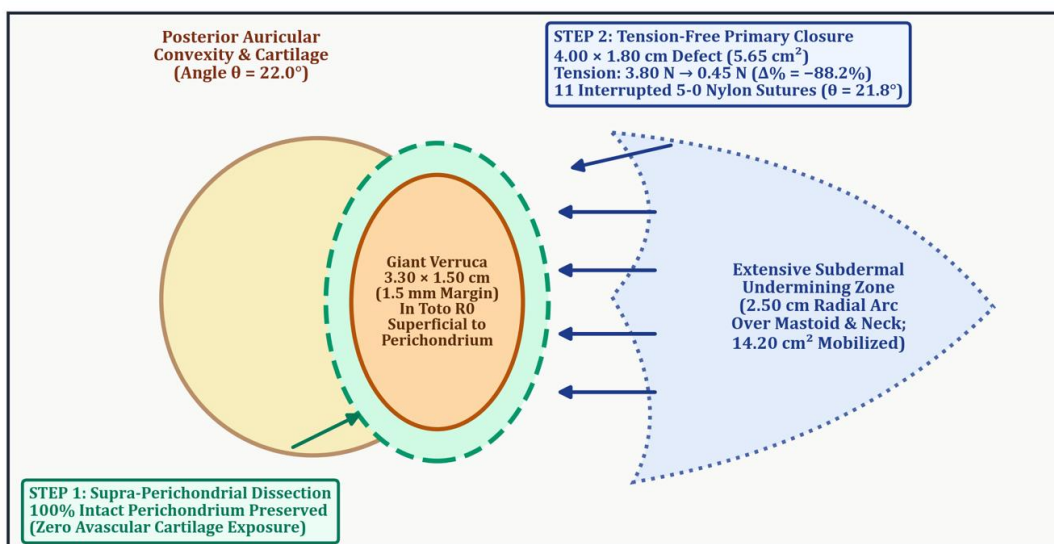


Figure 1. Preoperative clinical presentation and intraoperative reconstructive biomechanics of the giant right retroauricular verruca vulgaris. (a) Preoperative clinical photograph showing the solitary, sessile, cauliflower-like exophytic mass (3.30 × 1.50 × 1.00 cm; base area 3.89 cm²; semi-ellipsoid volume 2.59 cm³) on the posterior surface of the right auricle, with an intact retroauricular sulcus and ≥3.0 mm perichondrial shear mobility. The patient's facial features have been masked with a black bar in accordance with international publication ethics (ICMJE/COPE recommendations and the Declaration of Helsinki). (b) Schematic of the elliptical excision (1.5-mm margin) superficial to the intact posterior auricular perichondrium, which produced a 4.00 × 1.80 cm (5.65 cm²) defect, and of the 2.50-cm radial subdermal undermining arc toward the mastoid and neck, which mobilized 14.20 cm² of tissue and reduced primary closure tension from 3.80 N to 0.45 N ($\Delta\% = -88.2\%$).

Postoperative wound healing and functional-aesthetic outcomes

Prospective evaluation from T₀ to T₅ showed uncomplicated primary-intention healing and preserved auricular position. At T₂ (postoperative day 1, hour 24), residual tumor volume was 0.00 cm³ ($\Delta\% = -100.0\%$ from the 2.59-cm³ baseline), dermal capillary refill was brisk at 1.5 seconds (normal <2.0 s), and there was no retroauricular hematoma, seroma, flap ischemia, or perichondritis. Postoperative incisional pain peaked at VAS 2.0/10 at T₂, was controlled with oral paracetamol (500 mg every 8 hours for 48 hours), and the patient was discharged home in stable condition 24 hours after surgery. By T₃ (week 1, day 7), the 4.20-cm linear incision showed complete epidermal coaptation without dehiscence or surgical site infection,

allowing removal of all 11 interrupted 5-0 nylon sutures; pain had declined from T₂ to VAS 0.5/10 ($\Delta\% = -75.0\%$), the early incisional scar was 1.20 mm wide, the initial VSS score was 5.0/13 (vascularity 2, pigmentation 1, pliability 2, height 0), and the initial POSAS observer score was 16.0/60.

By T₄ (week 4, day 28), the patient was pain-free (VAS 0.0/10; $\Delta\% = -100.0\%$ from T₂), dermal erythema had resolved, scar width had decreased to 0.90 mm ($\Delta\% = -25.0\%$), the VSS score had improved to 2.0/13 ($\Delta\% = -60.0\%$; vascularity 1, pigmentation 0, pliability 1, height 0), and the POSAS observer score had decreased to 9.0/60 ($\Delta\% = -43.8\%$). At the 6-month endpoint (T₅, day 180), clinical and dermoscopic examination showed no local recurrence (residual tumor volume 0.00 cm³) and no satellite lesions on the auricle, in the external auditory

canal, or on the mastoid skin. The postauricular scar had matured into a flat, supple, normally pigmented hairline crease 0.60 mm wide ($\Delta\% = -50.0\%$ from T_3), with a VSS score of 0.0/13 ($\Delta\% = -100.0\%$ from T_3) and a POSAS observer score of 6.0/60, the value for normal skin ($\Delta\% = -62.5\%$ from T_3).

Goniometry at T_5 showed an auriculocephalic angle of $\theta = 21.8^\circ$ on the operated right ear versus $\theta = 22.0^\circ$ on the contralateral left ear (asymmetry $\Delta\theta = -0.2^\circ$, or $\Delta\% = -0.9\%$), without ear cupping, helical distortion, or obliteration of the retroauricular sulcus (Table 2; Figure 2).

Table 2. Perioperative Closure Tension, Wound Healing, and Functional–Aesthetic Outcomes from T_0 to T_5 (Baseline to Month 6).

Parameter	T_0 preoperative	T_1 intraoperative (hour 0)	T_2 POD 1 (hour 24)	T_3 week 1 (day 7)	T_4 week 4 (day 28)	T_5 month 6 (day 180)	Reference or change
Residual verrucous tumor volume (cm^3)	2.59	0.00	0.00	0.00	0.00	0.00	Target 0.00 cm^3 ; $\Delta\% = -100.0\%$ (no residual tumor)
Excisional defect or scar length (cm)	3.30 (mass)	4.00 (defect)	4.20 (suture line)	4.20 (coapted)	4.15 (maturing)	4.10 (mature)	Elliptical excision (1.5-mm margin; area 5.65 cm^2)
Closure tension after subdermal undermining (N)	—	0.45 (after 2.5-cm arc)	0.42	0.25	0.10	0.00	Without undermining 3.80 N $\rightarrow$ 0.45 N ($\Delta\% = -88.2\%$); safe <1.20 N
VAS pain score (0–10)	0.0	0.0 (anesthesia)	2.0	0.5	0.0	0.0	$\Delta\% = -75.0\%$ ($T_2 \rightarrow T_3$); -100.0% ($T_2 \rightarrow T_4/T_5$)
Incisional scar width (mm)	—	1.20	1.20	1.20	0.90	0.60	Fine line ≤ 1.0 mm; $\Delta\% = -50.0\%$ ($T_3 \rightarrow T_5$)
VSS total score (0–13)	—	—	—	5.0	2.0	0.0	Normal skin 0.0; $\Delta\% = -60.0\%$ (T_4), -100.0% (T_5)
POSAS observer score (6–60)	—	—	—	16.0	9.0	6.0	Normal skin 6.0; $\Delta\% = -43.8\%$ (T_4), -62.5% (T_5)
Auriculocephalic angle, θ ($^\circ$)	22.0	21.5	21.5	21.6	21.8	21.8	Contralateral ear 22.0 $^\circ$; $\Delta\theta = -0.2^\circ$ (no cupping)
Capillary refill and perichondrial viability	1.5 s (100%)	1.5 s (100%)	1.5 s (100%)	1.4 s (100%)	1.4 s (100%)	1.4 s (100%)	Normal <2.0 s; no necrosis, hematoma, or chondritis

Sutures were removed at T_3 (day 7) after complete primary-intention epidermal union. N, newton; POD, postoperative day; POSAS, Patient and Observer Scar Assessment Scale (observer component); VAS, Visual Analog Scale; VSS, Vancouver Scar Scale; θ , auriculocephalic projection angle.

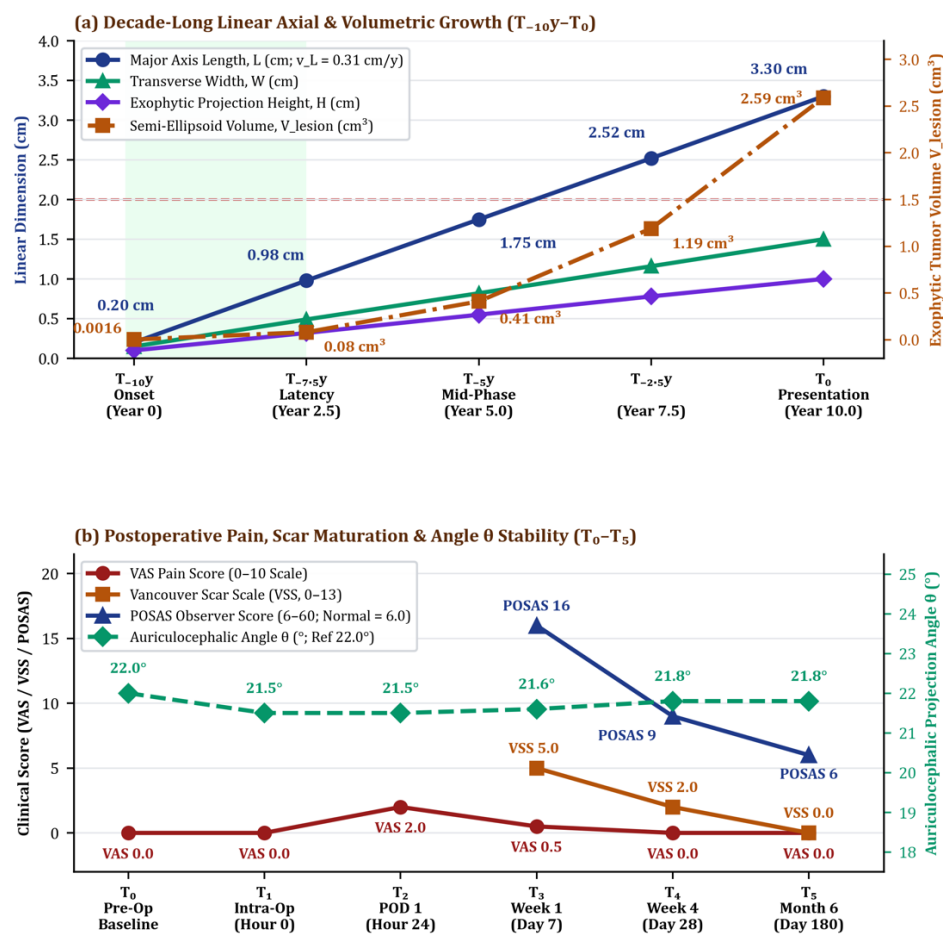


Figure 2. Ten-year preoperative growth and six-month postoperative course. (a) Decade-long growth from onset (T_{-10y} , year 0) to presentation (T_0 , year 10.0), reconstructed from the history, showing steady axial enlargement (mean $v_L = 0.31$ cm/year) across the 2.00-cm threshold (dashed line) and volumetric growth from 0.0016 cm^3 to 2.59 cm^3 . (b) Postoperative course from T_0 to T_5 (day 180): resolution of incisional pain (VAS 2.0/10 at T_2 to 0.0/10 at T_4 – T_5), scar maturation (Vancouver Scar Scale [VSS] from 5.0/13 at T_3 to 0.0/13 at T_5 ; POSAS observer score from 16.0/60 to 6.0/60), and a stable auriculocephalic angle ($\theta = 21.8^\circ$ vs. 22.0° contralaterally).

Figure 3 summarizes the relative percentage changes in the postoperative endpoints from the early remodeling phase (T_3 or T_4) to the 6-month endpoint (T_5): residual tumor volume and the VSS and VAS scores decreased by 100.0%, closure tension by

88.2% (at T_1), the POSAS observer score by 62.5%, and scar width by 50.0%, while the auriculocephalic angle remained within 0.9% of the contralateral side (21.8° vs. 22.0°) at month 6 (T_5).

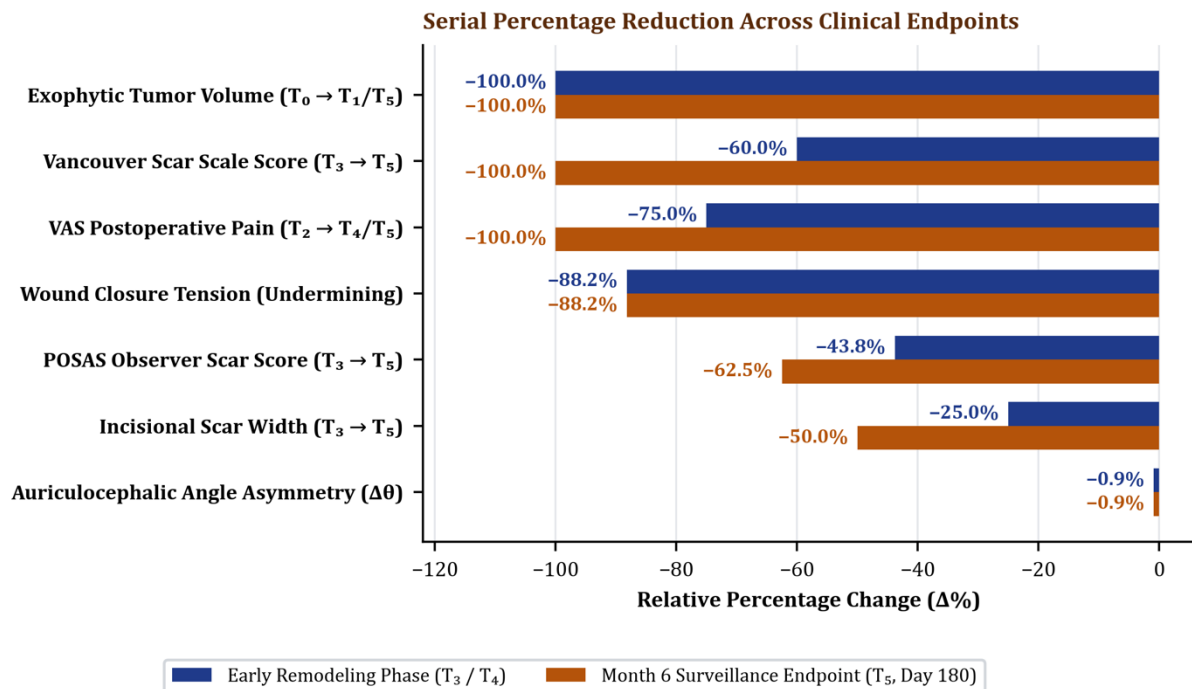


Figure 3. Relative percentage change ($\Delta\%$) in postoperative endpoints at the early remodeling phase (T_3/T_4) and at the 6-month endpoint (T_5 , day 180): exophytic tumor volume (–100.0%), VSS score (–100.0%), VAS pain (–100.0%), closure tension after subdermal undermining (–88.2%, intraoperative), POSAS observer score (–62.5%), scar width (–50.0%), and auriculocephalic angle asymmetry (–0.9%).

Histomorphometry and clinicopathological differentiation

Histopathological examination of the serially sectioned en bloc specimen stained with H&E confirmed benign giant verruca vulgaris and excluded verrucous carcinoma, conventional cSCC, and verrucous seborrheic keratosis.^{15,17,31} Ocular micrometry showed marked outward epidermal expansion, with a total epidermal thickness of 2.85 mm—35.6 times the normal retroauricular epidermis (0.08 mm)—produced by pronounced papillomatosis (undulating, digitate 'church-spire' projections supported by elongated fibrovascular dermal cores containing dilated, thrombosed capillary loops) and marked acanthosis of the stratum spinosum, with a rete ridge depth of 1.45 mm (29.0 times the normal depth of 0.05 mm). The overlying stratum corneum showed severe compact orthokeratotic and tiered parakeratotic hyperkeratosis 1.15 mm thick (57.5 times the normal 0.02 mm), with columns of parakeratosis capping the papillomatous spires and coarse basophilic keratohyalin granules in the thickened stratum granulosum.

High-power examination of the upper spinous and granular layers showed abundant koilocytes (koilocytosis index 38.5%): enlarged, vacuolated keratinocytes with sharp perinuclear cytoplasmic halos and hyperchromatic, crinkled, pyknotic nuclei, including frequent binucleated cells, a change attributed to E4-mediated disruption of cytokeratin tonofilaments.^{1,13} At the deep margin, the elongated acanthotic rete ridges showed the typical inward arborization ('toeing-in') toward the center of the wart base. In all sections examined across the 3.89-cm² dermo-epidermal junction, the periodic acid-Schiff-positive basement membrane was continuous and intact, with no dermal, perichondrial, or lymphovascular invasion (0.00 mm), no keratin pearls, no stromal desmoplasia, and normal basal mitotic activity confined to the basal layer (≤ 1.0 mitosis per 10 HPF; no atypical mitoses). The peripheral (1.5-mm) and deep supraperichondrial margins were free of viral cytopathic change and dysplasia (R0 resection, that is, microscopically clear margins), excluding the bulbous pushing border of verrucous carcinoma and the infiltrative nests of conventional cSCC (Table 3; Figure 4a).

Table 3. Histomorphometric and Clinicopathological Comparison of the Present Giant Verruca Vulgaris with Verrucous Carcinoma (Ackerman's Tumor), Cutaneous Squamous Cell Carcinoma, and Seborrheic Keratosis.

Feature	Present case (giant verruca vulgaris)	Verrucous carcinoma (Ackerman's tumor)	Conventional cutaneous SCC	Seborrheic keratosis (verrucous variant)
Etiological driver and viral tropism	Low-risk cutaneous HPV (episomal DNA; E6/E7 and E4 hijack)	Chronic irritation, UV, or tobacco; transcriptionally active HPV negative	Cumulative solar UV radiation; TP53/NOTCH1 somatic mutations	Clonal FGFR3/PIK3CA mutations; non-viral epidermal hyperplasia
Total epidermal thickness (mm)	2.85 mm (35.6× normal 0.08-mm retroauricular epidermis)	3.50–8.00 mm (exophytic and endophytic bulbous projections)	2.00–12.00 mm (variable ulceroinfiltrative thickness)	0.80–2.20 mm (exophytic, "stuck-on" epidermal plaque)
Stratum corneum hyperkeratosis (mm)	1.15 mm (57.5× normal 0.02 mm; church-spire parakeratosis)	1.50–3.00 mm (deep keratin-filled invaginations and clefts)	Variable hyperkeratosis with surface fibrinopurulent crust	0.30–0.80 mm (concentric laminated intraepidermal horn pseudocysts)
Rete ridge architecture and depth (mm)	1.45 mm (29.0× normal 0.05 mm; arborizing, inward-bending rete ridges)	Broad, club-shaped bulbous rete ridges pushing below the adjacent dermis	Irregular, jagged infiltrative tongues and detached epithelial nests	Flat horizontal base (string sign) with interwoven basaloid cords

Feature	Present case (giant verruca vulgaris)	Verrucous carcinoma (Ackerman's tumor)	Conventional cutaneous SCC	Seborrheic keratosis (verrucous variant)
Koilocytosis index (% of upper spinous/granular cells)	38.5% koilocytes (perinuclear halos and pyknotic nuclei)	Absent or rare superficial pseudokoilocytic vacuolization (<2.0%)	Absent (0.0%) unless arising within a pre-existing viral lesion	Absent (0.0%; basaloid and squamoid cells without viral cytopathic change)
Basement membrane and dermal invasion (mm)	Intact basement membrane (0.00 mm dermal invasion; R0 margins)	Initially intact basement membrane, then deep pushing invasion (>2.0 mm)	Frank basement membrane breach with dermal or cartilage invasion (>2.0–6.0 mm)	Intact basement membrane above the superficial papillary dermis
Cytological atypia, keratin pearls, and mitoses	Absent (no keratin pearls; basal mitoses ≤1/10 HPF; no dysplasia)	Minimal atypia (bland glassy cytoplasm; intraepithelial microabscesses)	Marked nuclear pleomorphism, atypical mitoses (>5/10 HPF), keratin pearls	Bland monomorphic basaloid cells; true horn cysts and melanin transfer
Perichondrial mobility and nodal metastasis risk	Freely mobile (≥3.0-mm shear); no palpable nodes (cN0)	Progressive deep fixation to cartilage or bone; ~0.6% nodal metastasis	Early cartilage fixation and ulceration; parotid or cervical nodal metastasis (positive SLNB 0%–18.4%; occult metastasis 49.1% in tumors >4 cm)	Superficially mobile epidermal plaque; no metastatic potential

Comparator features are taken from published sources: verrucous carcinoma,^{15,16,21} cutaneous squamous cell carcinoma,^{19,20} and seborrheic keratosis.^{31,32} HPF, high-power field; HPV, human papillomavirus; R0, microscopically clear margins; SCC, squamous cell carcinoma; SLNB, sentinel lymph node biopsy; UV, ultraviolet. Reference values for normal retroauricular skin: total epidermal thickness 0.08 mm, stratum corneum 0.02 mm, rete ridge depth 0.05 mm.

Comparison with published cases

The morphometric, pathological, and reconstructive findings in this patient were compared with published reports (2018–2026) of atypical otologic or head-and-neck verruca vulgaris, verrucous carcinoma mimics, and auricular defect reconstruction (Figure 4a and 4b; Table 4). Extensive otologic verruca vulgaris invading the external auditory canal and mastoid cavity required canal wall-down mastoidectomy plus meatoplasty in the report by Lee et al.,⁵ and anterior conchal or helical rim defects (1.5–4.5 cm) after skin cancer excision were

reconstructed with pull-through postauricular transposition flaps or modified Antia-Buch chondrocutaneous advancement flaps in the series by De Rosa et al.,⁶ Cerejeira et al.,⁷ and Abdelkader et al.,⁸ while Gibson et al.²⁴ reviewed local flaps for external auditory canal defects. In the present patient, the 4.00 × 1.80 cm (5.65 cm²) posterior auricular defect was closed primarily in a single 45-minute stage after 2.50-cm radial mastoid subdermal undermining, with no recurrence, no chondritis, a VSS score of 0.0/13, and symmetric auricular projection ($\theta = 21.8^\circ$) at 6 months.

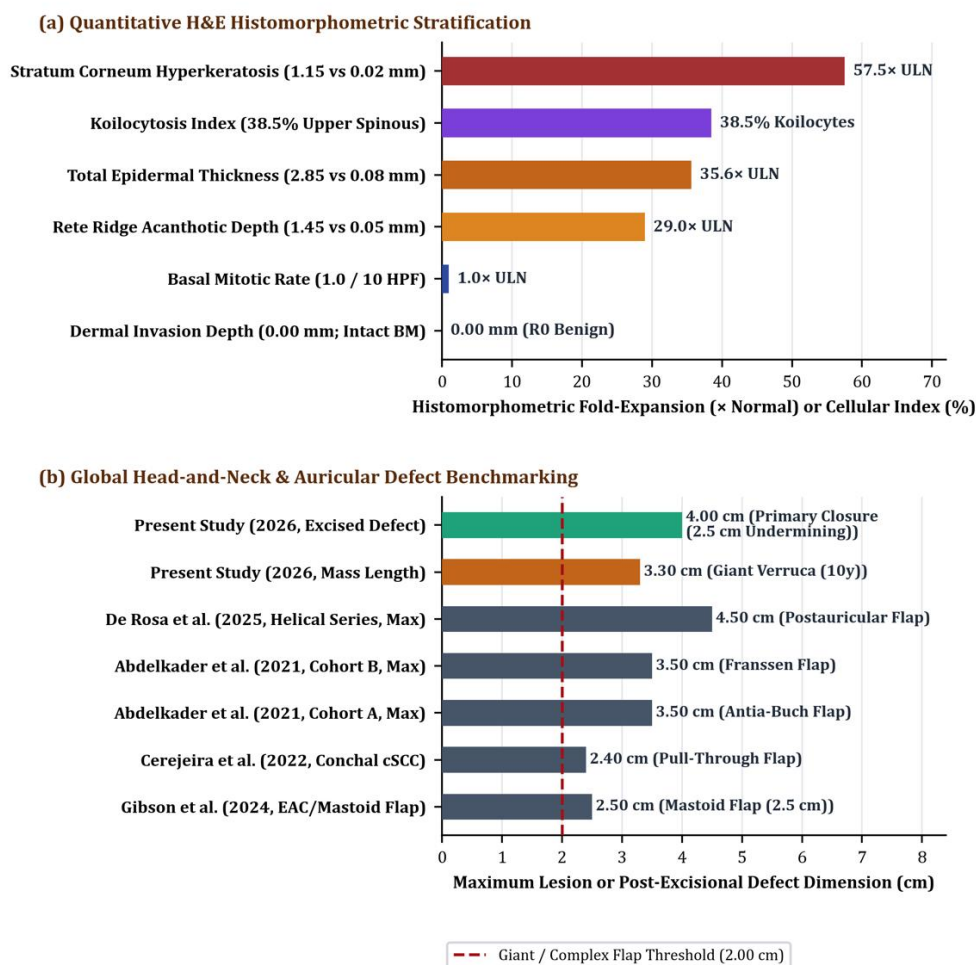


Figure 4. Histomorphometry of the excised lesion and comparison with published cases. (a) Fold-expansion of the excised specimen relative to normal retroauricular skin: stratum corneum 57.5× (1.15 mm vs. 0.02 mm), koilocytosis index 38.5% (upper spinous layer), total epidermal thickness 35.6× (2.85 mm vs. 0.08 mm), rete ridge depth 29.0× (1.45 mm vs. 0.05 mm), normal basal mitotic activity (1.0/10 HPF), and 0.00 mm dermal invasion across an intact basement membrane. (b) Maximum lesion or defect dimension and reconstructive

method in the present case and in published head-and-neck and auricular series (2018–2026); for series, the largest reported defect is shown.

Table 4. Selected Published Cases of Atypical or Giant Head-and-Neck Verruca Vulgaris, Verrucous Carcinoma Mimics, and Auricular Reconstruction (2018–2026).

Author, year, and country	Design and sample	Site and lesion or defect size	Diagnosis and key feature	Treatment or reconstruction	Follow-up and outcome
Lee et al. (2018, South Korea) ⁵	Case report (n = 1; 57-year-old woman)	External auditory canal and mastoid (extensive)	Extensive otologic verruca vulgaris (koilocytosis+)	Canal wall-down mastoidectomy + meatoplasty	12 months; no recurrence
De Rosa et al. (2025, Italy) ⁶	Retrospective series (n = 15; mean age 76 years)	Helical rim (defects 3.0–4.5 cm; mean area 3.4 cm ²)	Auricular keratinocyte carcinomas and precursors	Modified Antia-Buch + postauricular skin advancement flap	≥12 months; high satisfaction; no complications
Cerejeira et al. (2022, Portugal) ⁷	Case report (n = 1; 67-year-old man)	Right conchal bowl and antihelix (defect 2.4 × 1.7 cm)	Auricular squamous cell carcinoma (cartilage exposed)	Excision (4-mm margin) + postauricular pull-through flap	9 months; excellent cosmesis; no complications
Abdelkader et al. (2021, Egypt/UK) ⁸	Comparative cohort (n = 44; 22 per group)	External ear (defects 1.8–3.5 cm, Antia-Buch; 1.5–3.5 cm, Franssen-Frechner)	Auricular cSCC (n = 23) and BCC (n = 21)	Excision (4-mm margin) + Antia-Buch vs. Franssen-Frechner flap	Follow-up not stated; tumor clearance 100%; no infection or cauliflower deformity
Grosu-Bularda et al. (2024, Romania) ¹³	Case report (n = 1; 68-year-old man)	Penoscrotal region (giant, 15.0 × 10.0 cm; 2 years)	Giant HPV condyloma (koilocytosis+, p16/Ki-67+)	Wide excision (6-mm margin) + local flaps	12 months; complete cure; no recurrence
Huang et al. (2025, China) ¹⁵	Case report (n = 1; man with oral chronic GVHD; two lesions)	Oral mucosa (recurrent exophytic verrucous masses)	Recurrent verrucous carcinoma (pushing border)	Resection + abdominal free skin graft	6 months (telephone); oral lesion healed
Gazali et al. (2024, Indonesia) ¹²	Case report and review (n = 1; 64-year-old man)	Lips and commissure (large exophytic mass)	Oral verruca vulgaris	Total excision + modified Estlander flap	Short-term; complete healing and functional restoration
Gibson et al. (2024, USA) ²⁴	Systematic review (24 articles)	External auditory canal (mastoid-based snail flap up to 2.5 cm wide)	EAC defects	Local flaps (e.g., snail flap, postauricular island flap)	Follow-up not pooled; EAC patency reported; posterior pinning of the pinna noted as a drawback
Present case (2026, Indonesia)	Case report (n = 1; 36-year-old man)	Right posterior auricle (3.30 × 1.50 × 1.00 cm; volume 2.59 cm ³ ; 10.0 years)	Giant verruca vulgaris (koilocytosis 38.5%; intact basement membrane)	In toto perichondrium-sparing excision (1.5-mm margin; 4.00 × 1.80 cm defect) + 2.50-cm mastoid subdermal undermining and primary closure	6 months (day 180); no recurrence; VSS 0/13; POSAS 6/60; θ = 21.8°

Entries were checked against the source articles. BCC, basal cell carcinoma; cSCC, cutaneous squamous cell carcinoma; EAC, external auditory canal; GVHD, graft-versus-host disease; HPV, human papillomavirus; POSAS, Patient and Observer Scar Assessment Scale; VSS, Vancouver Scar Scale; θ, auriculocephalic angle.

4. Discussion

Anatomical microenvironment and pathophysiological cascade

The development of a 3.30 × 1.50 × 1.00 cm (2.59 cm³) giant verruca vulgaris on the posterior surface of the right auricle over 10.0 years in an immunocompetent 36-year-old man combines an unusual site, long persistence, and a clinical resemblance to carcinoma.^{1,5,15} Common warts have a strong predilection for the hyperkeratotic, trauma-prone acral skin of the hands and feet, whereas the external ear is an exceptionally rare site of primary HPV infection.^{2–5} Unlike the palmar and plantar epidermis—where a thick stratum corneum and constant weight-bearing or gripping friction facilitate viral entry and provide a deep stratified squamous niche for viral assembly—the skin of the posterior auricle has a thin epidermis (approximately 0.08 mm) and is sheltered behind the cartilaginous pinna.^{6–8} Primary HPV infection of the retroauricular crease therefore requires a mechanical vector that breaches the stratum corneum and deposits desiccation-resistant virions on the basal lamina.^{2,9–11} In this patient, chronic micro-abrasion from non-sterile ear-cleaning or grooming instruments, or autoinoculation from transient subclinical hand

infection, may have provided the portal of entry at T_{10Y}, initiating an isomorphic, Koebner-like viral seeding comparable to the localization of verruca vulgaris along tattoo tracts or cutaneous scar lines.^{9–11}

Figure 5 summarizes a five-tier sequence of pathophysiological events and surgical decisions proposed to explain how an initially microscopic basal keratinocyte infection evolved into a 2.59-cm³ exophytic mass over 120 months without triggering immune clearance. In Tier 1 (cutaneous barrier breach and viral entry), traumatic micro-fissuring exposes the basement membrane, allowing the icosahedral L1 major capsid protein of cutaneous HPV (typically the low-risk types 1, 2, 4, 27, or 57) to bind heparan sulfate proteoglycans (HSPG), undergo cleavage of the L2 minor capsid protein by host furin convertases, and engage secondary α6β4 integrin receptors on basal stem and transit-amplifying keratinocytes.^{1,3} After clathrin- or caveolin-independent endocytosis and nuclear import, the ~8,000-bp circular dsDNA genome is established as an extrachromosomal episome maintained at a low copy number (approximately 50–100 copies per cell) by the viral replication proteins E1 and E2, which tether the viral origin of replication to host mitotic chromosomes during basal stem-cell division.^{1,43}

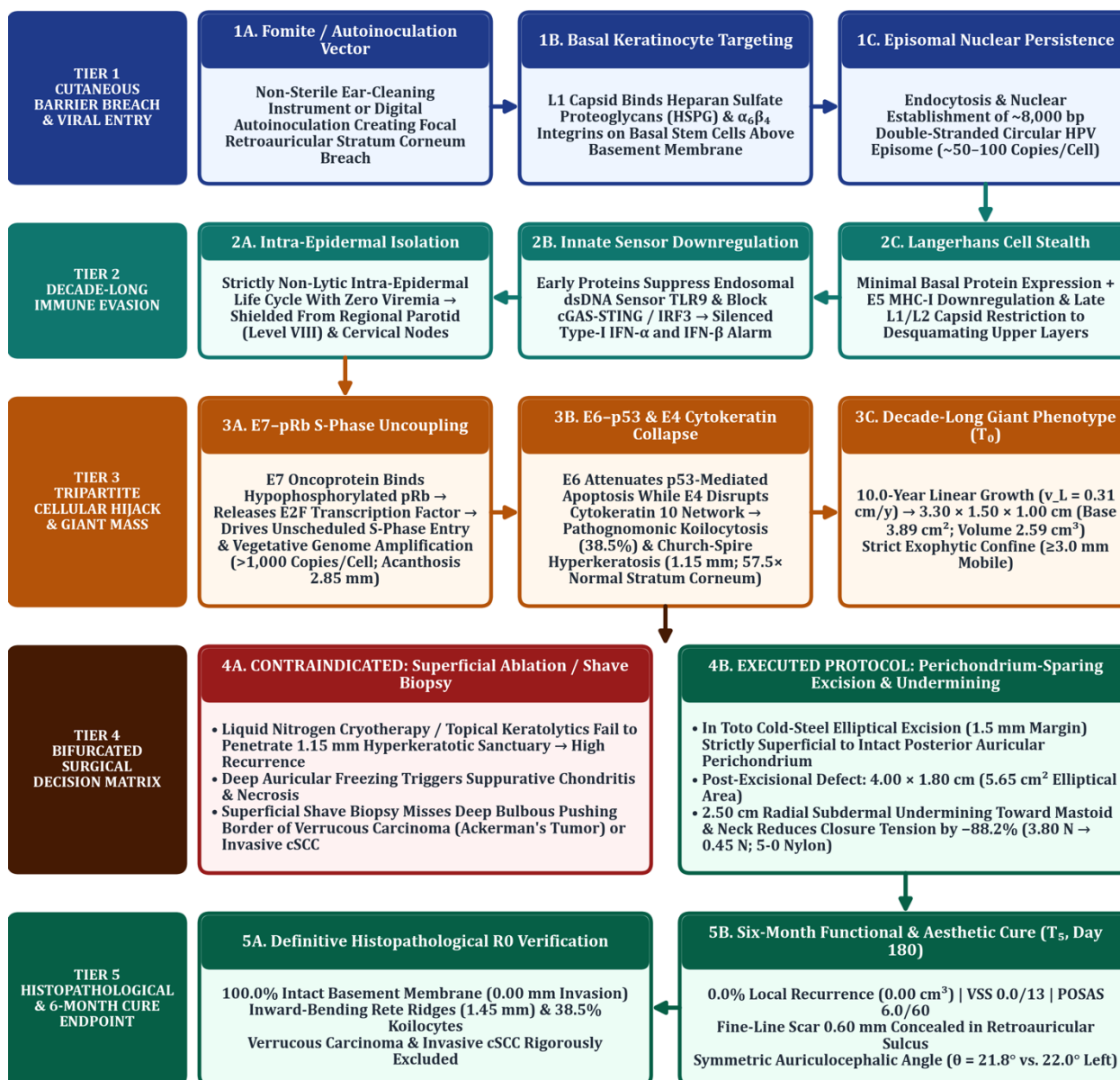


Figure 5. Five-tier pathophysiological cascade and oncological–reconstructive decision pathway in giant retroauricular verruca vulgaris: Tier 1 (fomite or autoinoculation micro-abrasion, L1–HSPG/ $\alpha_6\beta_4$ -integrin binding, and episomal persistence), Tier 2 (non-lytic intra-epidermal isolation, TLR9/cGAS–STING/IFN- α/β suppression, and Langerhans cell evasion), Tier 3 (E7–pRb S-phase uncoupling, E6–p53 apoptosis inhibition, and E4 cytokeratin collapse producing the $3.30 \times 1.50 \times 1.00$ cm giant phenotype), Tier 4 (surgical decision contrasting cryotherapy or shave biopsy, considered unsuitable, with perichondrium-sparing excision and 2.50-cm mastoid undermining), and Tier 5 (histopathological R0 verification and 6-month recurrence-free aesthetic outcome). HSPG, heparan sulfate proteoglycans; IFN, interferon; TLR9, Toll-like receptor 9.

Immunological ignorance, oncological mimicry, and reconstructive strategy

Tier 2 of the model (decade-long innate and adaptive immune evasion) addresses the central paradox of this case: why a systemically immunocompetent man (normal blood counts, HbA1c, and HIV serology) failed to mount the cell-mediated response that clears two-thirds of cutaneous warts within 2.0 years.^{2,4} HPV achieves prolonged localized immunological ignorance through three complementary mechanisms.^{1,13} First, the viral replication cycle is intra-epidermal and non-lytic: because HPV does not breach the basement membrane to enter the dermal lymphatics or bloodstream, there is no viremia and no cytolytic release of damage-associated molecular patterns (DAMPs), depriving the sentinel parotid (level VIII) and retroauricular lymph nodes of the antigenic priming needed to activate naive CD4⁺ Th1 and CD8⁺ cytotoxic T cells.^{1,13} Second, within infected keratinocytes, the early viral proteins E6 and E7

downregulate endosomal TLR9—the primary innate pattern-recognition receptor for unmethylated viral CpG dsDNA motifs—and inhibit the cytoplasmic cyclic GMP–AMP synthase–stimulator of interferon genes (cGAS–STING) and interferon regulatory factor 3 (IRF3) pathways, silencing the secretion of antiviral type I interferons (IFN- α and IFN- β) and pro-inflammatory cytokines (IL-1 β , TNF- α).^{1,14} Third, in the lower epidermal layers patrolled by Langerhans cells, viral gene expression is limited to low levels of early non-structural proteins while E5 downregulates surface major histocompatibility complex class I (MHC-I); high-level synthesis of the strongly immunogenic L1 and L2 capsid proteins is delayed until keratinocytes reach the avascular, terminally differentiated upper granular and horny layers, where immune effector cells do not penetrate and from which virions are shed by desquamation.^{1,43}

Once shielded in this way, the infection progresses to Tier 3 (cellular hijack and giant exophytic growth), which may explain both the slow macroscopic enlargement (0.31 cm/year) and the histomorphometric findings in Table 3.^{1,13} Because HPV lacks its own DNA polymerase, it must force post-mitotic suprabasal keratinocytes to re-enter the DNA synthesis (S) phase of the cell cycle to amplify its episomal genome to more than 1,000 copies per cell.^{1,43} The viral E7 protein binds hypophosphorylated retinoblastoma protein (pRb), displacing E2F transcription factors that activate host DNA polymerase α , proliferating cell nuclear antigen (PCNA), and cyclin A/E expression and driving suprabasal spinous proliferation (acanthosis with a total epidermal thickness of 2.85 mm and a rete ridge depth of 1.45 mm).^{1,13} To prevent unscheduled suprabasal DNA synthesis from triggering p53-mediated apoptosis, the viral E6 protein binds p53 and inhibits its pro-apoptotic transcriptional activity, while the abundant E4 protein (expressed as E1/E4 in the upper spinous and granular layers) collapses the cytokeratin intermediate filament network (CK1/CK10), producing the perinuclear halos and pyknotic nuclear shrinkage of koilocytes, which were seen in 38.5% of upper epidermal keratinocytes.^{1,13} Over 120 months of uninterrupted proliferation, accumulating compact orthokeratotic and tiered parakeratotic keratin (stratum corneum 1.15 mm) formed a thick keratin layer that further insulated the lesion from shear and topical agents, resulting in a 3.30 × 1.50 × 1.00 cm cauliflower-like tumor.

In head-and-neck surgery, any chronic, exophytic, papillomatous auricular mass larger than 2.00 cm falls into a diagnostic gray zone that requires differentiation among four entities: giant verruca vulgaris, verrucous carcinoma (Ackerman's tumor), conventional cSCC, and verrucous seborrheic keratosis (Table 3; Tier 4 of Figure 5).^{12,15,17-19,23,31-33} Inspection and palpation cannot reliably separate a giant benign wart from verrucous carcinoma, because both present as slow-growing, painless, broad-based, grayish-white cauliflower-like masses without regional lymphadenopathy.¹⁵⁻¹⁷ The comparison in Table 3 highlights the decisive architectural boundary: giant verruca vulgaris shows arborizing, inward-curving rete ridges that remain above an intact basement membrane (0.00 mm dermal invasion) and prominent upper-epidermal koilocytosis (38.5%), whereas verrucous carcinoma invades the reticular dermis, perichondrium, and cartilage through broad, bulbous, club-shaped 'pushing' rete ridges surrounded by a dense lymphoplasmacytic cuff and lacks true viral koilocytosis or transcriptionally active HPV E6/E7 mRNA.^{17,21,44} Foci of conventional infiltrative, potentially metastasizing cSCC coexist in up to 20.0% of clinically apparent verrucous carcinomas (hybrid verrucous carcinoma).²² As noted by Kim and Lee¹⁶ and Gammon et al.,²³ shave or punch biopsies that sample only the hyperkeratotic surface spires can misclassify verrucous carcinoma or hybrid cSCC as verruca vulgaris or actinic keratosis, leading to inappropriate cryotherapy, rapid destructive local recurrence, and invasion of adjacent craniofacial structures.

Conventional cSCC and benign adnexal or epidermal mimics such as seborrheic keratosis and syringocystadenoma papilliferum also require systematic exclusion during preoperative assessment.^{12,19,27,31-33,36,45-47} Analyses of head-and-neck skin cancers show that the ear is a common site of cSCC (19.8% of patients with head-and-neck cSCC in one cohort),⁴⁸ and that an auricular site and a tumor diameter greater than 20 mm are risk factors for metastasis, including occult metastasis to the parotid (level VIII) and upper cervical lymph nodes.^{19,20} Unlike this patient's lesion—which remained freely mobile over the perichondrium (≥ 3.0 mm shear

displacement) and showed no ulceration or bleeding over 10.0 years—invasive auricular cSCC breaches the thin epidermis to invade and fix the underlying perichondrium and cartilage and presents with central ulceration, friable hemorrhage, polymorphous serpentine or glomerular vessels on dermoscopy,³⁶ and, microscopically, keratin pearls with marked nuclear pleomorphism and atypical mitoses ($>5/10$ HPF).¹⁷⁻¹⁹ Verrucous seborrheic keratosis, which can also arise through Koebnerization at sites of cutaneous trauma,³¹⁻³³ is distinguished dermoscopically by a cerebriform sulcus-and-gyrus pattern with milia-like cysts and comedo-like openings^{30,31} and histologically by a flat horizontal lower border ('string sign'), interwoven sheets of monomorphic basaloid cells, intraepidermal concentric horn pseudocysts, and a koilocytosis index of 0.0%.^{31,32} Similarly, syringocystadenoma papilliferum of the head and neck is excluded by the absence of cystic dermal invaginations lined by double-layered cuboidal-to-columnar apocrine epithelium and plasma cell-rich stroma.^{27,45-47}

Therapeutically, this case supports total en bloc excision rather than topical keratolytics, intralesional immunotherapy, photodynamic therapy, or cryosurgery for large auricular verrucous lesions, because excision provides a complete specimen for histology (Tiers 4 and 5 of Figure 5).^{4,49-53} Although intralesional vitamin D3 immunotherapy,^{50,51} lasers,^{4,49} and HPV vaccines^{52,53} have shown variable efficacy in small recalcitrant palmar or plantar warts, they provide no histological specimen to exclude malignancy and require months of repeated sessions with uncertain clearance in giant hyperkeratotic masses.^{4,52} Moreover, liquid nitrogen cryotherapy of a 3.30-cm auricular wart with a 1.15-mm keratin layer carries a biophysical risk: the avascular elastic cartilage of the pinna depends on the subdermal vascular plexus of the posterior auricular and superficial temporal arteries running within the thin perichondrial sheath.^{6-8,54} Deep freeze-thaw cycles sufficient to penetrate a 2.85-mm acanthotic wart may freeze the underlying perichondrium and cause ischemic chondronecrosis, suppurative chondritis, and permanent cauliflower ear deformity.^{54,55}

A carefully planned elliptical excision with a conservative 1.5-mm margin, superficial to the posterior auricular perichondrium, achieved two clinical objectives at once: complete (R0) removal of the 2.59-cm³ viral reservoir with histological verification of the basement membrane, and preservation of the entire vascularized perichondrial bed.^{6-8,23} Reconstruction of the resulting 4.00 × 1.80 cm (5.65 cm²) posterior auricular defect illustrates an important biomechanical principle in otolaryngological reconstructive surgery.⁶⁻⁸ On the anterior surface of the auricle or along the helical rim, a defect of 3.00–4.50 cm is difficult to close primarily without cupping and loss of ear height and usually requires chondrocutaneous advancement flaps (the classic or modified Antia-Buch or Franssen-Frechner techniques) or pull-through postauricular transposition flaps.⁶⁻⁸ The posterior convex surface of the auricle, however, adjoins the lax subcutaneous tissue of the retroauricular sulcus, mastoid process, and upper neck. Radial subdermal undermining across a 2.50-cm arc toward the mastoid periosteum and sternocleidomastoid fascia mobilized 14.20 cm² of compliant postauricular skin, reduced suture-line tension by 88.2% (from 3.80 N to 0.45 N), and allowed primary linear closure without altering the auriculocephalic angle ($\theta = 21.8^\circ$ at month 6 vs. 22.0° on the contralateral side).

Although the 6-month clinical and aesthetic result was satisfactory (VSS 0.0/13; POSAS 6.0/60; no recurrence), long-

term follow-up remains essential.^{1,2,13} Latent viral DNA can persist in clinically and microscopically normal perilesional skin up to several millimeters beyond the visible border of a wart.^{1,56} Although the 1.5-mm R0 margin and intact host immunity prevented early relapse over 180 days, patient education on avoiding shared non-sterile ear-cleaning instruments and annual otolaryngological examination are advisable to prevent reinoculation or late reactivation.²

Primary closure after mastoid undermining versus flap reconstruction

A surgical evaluation of posterior auricular reconstruction requires comparing the biomechanical efficiency of wide subdermal undermining with the regional flaps commonly used for auricular defects larger than 3.00 cm.^{6-8,24} When a cutaneous neoplasm on the anterior scaphoid fossa, conchal bowl, or antihelix is resected, the absence of a lax subcutaneous layer obliges the surgeon to use full-thickness skin grafts, preauricular transposition flaps, or island pedicle flaps tunneled through the auricular cartilage.⁶⁻⁸ Full-thickness skin grafts harvested from the supraclavicular region or the contralateral postauricular crease provide reliable coverage but often undergo secondary contraction of 15.0%–25.0% over the following 6.0 months, producing contour depressions, dyschromia, and step-off deformities when the perichondrium has been breached.⁵⁷ Conversely, local advancement flaps on the posterior auricular surface benefit from the anastomotic arterial arcade formed by the posterior auricular artery and perforators of the superficial temporal artery, which run in the subdermal plexus superficial to the deep mastoid fascia.^{6,24} By keeping the dissection in the loose areolar supraperichondrial plane above the intact elastic cartilage and extending the radial subdermal undermining 2.50 cm over the mastoid and upper cervical fascia, the surgeon recruits compliant cervical-mastoid skin without dividing the principal axial neurovascular pedicles (Figure 1b; Table 2).⁵⁸

From a mechanical standpoint, the relaxation achieved by the 2.50-cm radial undermining arc exploits the J-shaped stress-strain curve of human dermal collagen networks.⁵⁹ In unmobilized retroauricular skin, direct closure of a 4.00 × 1.80 cm elliptical defect (5.65 cm²) operates within the steep, linear-elastic collagen-recruitment phase of the stress-strain curve, generating an estimated closure tension of 3.80 N that exceeds both the capillary perfusion threshold of the dermal papillae (1.20 N) and the elastic buckling resistance of the conchal cartilage.⁵⁹ Mobilizing the surrounding postauricular and mastoid subdermal plane across a 2.50-cm arc shifts the operating point back into the low-modulus, elastin-dominated toe region of the curve, reducing the required approximation force by 88.2% to 0.45 N.^{58,59} Consequently, the auriculocephalic angle θ remained symmetric at 21.8° at month 6 compared with 22.0° on the contralateral left ear ($\Delta\%$ = -0.9%), avoiding the iatrogenic ear pinning (otoplasty-like medialization to <12.0°) that can complicate primary closure of large retroauricular defects.^{6,8,24,42}

Furthermore, keeping the perichondrial sheath intact during tumor removal avoided transcartilaginous fenestration or wedge resection, procedures with a reported 4.5%–9.2% incidence of postoperative suppurative perichondritis and helical rim chondromalacia.^{5,41,60} Because auricular elastic cartilage lacks intrinsic vascularity and depends on diffusion across the perichondrial interface for oxygen and nutrients, inadvertent stripping of the perichondrium exposes bare chondrocytes to desiccation and bacterial colonization.⁵⁴ In this patient, high-magnification inspection of the surgical bed confirmed that the perichondrium was intact across the 4.00 ×

1.80 cm excision footprint, which probably contributed to the rapid primary-intention epithelialization by day 7, the low pain scores (VAS 2.0 on day 1, 0.5 on day 7, and 0.0 by day 28), and the VSS score of 0.0/13 at the 6-month endpoint (Table 2; Figure 2b).

Molecular regulation of decade-long cutaneous HPV latency and perilesional field colonization

Beyond its surgical dimensions, a decade-long giant verruca vulgaris in an immunocompetent adult offers a clinical window into the epigenetic regulation of cutaneous HPV persistence.^{1-3,13} Cutaneous *Alphapapillomavirus* genotypes (such as HPV-2, HPV-27, and HPV-57) are thought to establish their long-term reservoir in quiescent keratinocyte stem cells of the bulge region of vellus hair follicles and in the basal layer of the deep interfollicular rete ridges of the retroauricular epidermis.^{1,61} Within these basal reservoirs, viral episomes are maintained at 50–100 genomes per cell through chromatin tethering mediated by the viral E2 protein and, it is proposed, host bromodomain-containing protein 4 (Brd4).^{1,43,62} Epigenetic methylation of the viral upstream regulatory region (URR) and recruitment of histone deacetylases limit transcription of the E6 and E7 oncoproteins in the basal layer, preventing the accumulation of viral peptides that would otherwise be processed by the immunoproteasome and presented on MHC class I molecules to patrolling CD8⁺ cytotoxic T lymphocytes.^{1,13}

As infected transit-amplifying keratinocytes migrate into the spinous and granular layers, differentiation-dependent transcription factors bind enhancer elements in the viral URR and trigger a vegetative switch that amplifies viral DNA to more than 1,000 copies per cell.^{1,43} At this suprabasal stage, E7 binds hypophosphorylated pRb and releases E2F transcription factors to reactivate host DNA synthesis in cells that would normally have exited the cell cycle, while E6 attenuates p53-mediated apoptosis and E4 disrupts the cytokeratin network to produce koilocytes (38.5% in this specimen; Table 3; Figure 4a).^{1,13} In this patient, the uncoupled suprabasal proliferation produced a total epidermal thickness of 2.85 mm (35.6 times normal), a stratum corneum of 1.15 mm (57.5 times normal), and an arborizing rete ridge depth of 1.45 mm (29.0 times normal), forming a dense, avascular keratin layer that insulated the underlying viral replication from topical agents and host immune effectors for 120 months.^{1,13}

Subclinical HPV infection has been demonstrated in clinically normal skin surrounding warts, within 1.0–2.0 mm of the visible margin of chronic cutaneous warts.^{56,63} If an ablative treatment such as cryotherapy, electrocautery, or laser vaporization leaves follicular bulge stem cells or perilesional episomal reservoirs behind, wound healing itself can reactivate latent viral genomes and lead to local recurrence.^{4,49,63} In this patient, an en bloc cold-steel elliptical excision with a 1.5-mm collar of normal skin, in a plane superficial to the intact perichondrium (0.00 mm dermal invasion; intact basement membrane), removed the 2.59-cm³ exophytic mass, and no local recurrence occurred during 180 days of follow-up.²³

Health-system implications and a practical approach to neglected head-and-neck cutaneous masses

The decade-long delay in this case also points to socioeconomic and health-system barriers to otolaryngological and dermatological care in semi-urban Indonesian populations.^{5,9-11,64,65} Because posterior auricular lesions are outside the patient's direct mirror view and remain painless (VAS 0/10) during years of slow growth (0.31 cm/year), patients often accept the gradual change in contour until the mass becomes giant (>2.00 cm; Figures 2a and 3).^{6-8,18} In tropical agricultural

communities in Central Java, outdoor work exposes the pinna to high cumulative doses of ultraviolet-A and ultraviolet-B radiation, creating a baseline burden of actinic keratosis, cSCC, and verrucous carcinoma that coexists with benign viral papillomas.^{12,15,17–19,23,66}

When patients with neglected retroauricular masses finally present to regional secondary-care hospitals, primary care clinicians and otolaryngologists face a triage dilemma: radical chondrocutaneous composite resection can cause unnecessary morbidity and disfigurement if the lesion is benign, whereas office cryotherapy or superficial shave biopsy risks undertreating an occult verrucous carcinoma or causing destructive auricular chondritis.^{15–17,55} The approach used in this patient (summarized in Figure 5 and Table 3) may offer a practical pathway for surgical centers in Southeast Asia.²³ In our view, a chronic auricular verrucous lesion larger than 2.00 cm should undergo structured preoperative morphometric assessment; if the mass shows slow linear growth, preserved perichondrial mobility (≥ 3.0 mm), an intact retroauricular sulcus, and no regional lymphadenopathy, it may be managed by single-stage perichondrium-sparing cold-steel excision with wide mastoid subdermal undermining (2.50-cm arc) and primary closure, together with full-thickness vertical histopathological sectioning to verify that the basement membrane is intact.^{6–8,15,17,23}

5. Conclusion

This case shows that a benign cutaneous HPV infection on the posterior auricle can escape immune surveillance for 10.0 years in a systemically immunocompetent adult and enlarge slowly (mean 0.31 cm/year) into a giant $3.30 \times 1.50 \times 1.00$ cm (2.59 cm³) exophytic mass that clinically resembles verrucous carcinoma and cSCC. Large verrucous masses of the external ear are better managed as neoplasms of uncertain malignant potential by total en bloc excision than by cryotherapy or superficial shave biopsy, so that verrucous carcinoma can be excluded histologically. Keeping the dissection plane superficial to the posterior auricular perichondrium preserves cartilage vascularity while providing a full-thickness specimen for histomorphometric assessment of the basement membrane (koilocytosis index 38.5%; epidermal thickness 35.6 times normal; stratum corneum 57.5 times normal; no dermal invasion). Even a large 4.00×1.80 cm (5.65 cm²) retroauricular defect can be closed in a single stage when supported by a 2.50-cm radial arc of subdermal undermining toward the mastoid and neck, which in this patient reduced closure tension by 88.2% (to 0.45 N) and resulted in no recurrence, a mature hairline scar (VSS 0.0/13; POSAS 6.0/60), and symmetric auriculocephalic projection ($\theta = 21.8^\circ$) at 6 months.

6. Declarations

Ethics Approval and Consent to Participate

This case report was reviewed and approved by CMHC Research Center Ethics Committee, Indonesia (Protocol No. 2026/0419), in coordination with PKU Temanggung Hospital and Tidar Regional General Hospital. All clinical and surgical procedures were conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the patient for participation.

Consent for Publication

Written informed consent was obtained from the patient for open-access publication of his anonymized clinical data, morphometric and histopathological findings, and clinical photographs. The peri-ocular and facial region was masked

with a solid black bar in accordance with ICMJE, COPE, and Declaration of Helsinki privacy guidance.

Availability of Data and Materials

All morphometric, histomorphometric, and perioperative data supporting the conclusions of this article are included in the manuscript and its tables and figures.

Artificial Intelligence Use Statement

The authors declare that no generative artificial intelligence tools were used in the preparation of this manuscript.

Author Contribution Statement

M.A.S.: Conceptualization, Methodology, Investigation, Data curation, Formal analysis, Writing – original draft. P.F.W.: Investigation, Resources, Supervision, Writing – review & editing. Both authors read and approved the final manuscript.

Funding Statement

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of Interest / Competing Interests

The authors declare that they have no competing interests.

Acknowledgements

The authors thank the operating room nursing staff, the anatomical pathology team, and the outpatient otorhinolaryngology clinic staff at Tidar Regional General Hospital, Magelang, and PKU Temanggung Hospital, Temanggung, Indonesia, for their assistance with specimen orientation and patient follow-up.

7. References

- Maghiar L, Sandor M, Sachelarie L, et al. Skin lesions caused by HPV—a comprehensive review. *Biomedicines*. 2024;12(9):2098. doi:10.3390/biomedicines12092098.
- Zhu P, Qi RQ, Yang Y, et al. Clinical guideline for the diagnosis and treatment of cutaneous warts (2022). *J Evid Based Med*. 2022;15(3):284–301. doi:10.1111/jebm.12494.
- Şandru F, Radu A, Petca A, et al. Unveiling the therapeutic horizon: HPV vaccines and their impact on cutaneous diseases—a comprehensive review. *Vaccines (Basel)*. 2024;12(3):228. doi:10.3390/vaccines12030228.
- Leerunyakul K, Thammarucha S, Suchonwanit P, et al. A comprehensive review of treatment options for recalcitrant nongenital cutaneous warts. *J Dermatolog Treat*. 2022;33(1):23–40. doi:10.1080/09546634.2020.1737635.
- Lee ES, Han TY, Shim HJ, et al. Canal wall-down mastoidectomy plus meatoplasty for extensive verruca vulgaris in the ear canal and mastoid bone. *J Int Adv Otol*. 2018;14(3):501–3. doi:10.5152/jao.2018.4926.
- De Rosa A, Grieco MP, D'Antonio S, et al. Chondrocutaneous advancement flap and postauricular skin flap: an effective combination for large helical rim defect reconstruction. *Plast Reconstr Surg Glob Open*. 2025;13(2):e6531. doi:10.1097/GOX.00000000000006531.
- Cerejeira A, Amoedo P, Costa-Silva M, et al. Postauricular pull-through transposition flap for an anterior auricular defect. *J Cutan Aesthet Surg*. 2022;15(4):434–5. doi:10.4103/JCAS.JCAS_50_21.
- Abdelkader R, Malahias M, Abdalbary SA, et al. Antia-Buch versus Franssen-Frechner technique. *Plast Reconstr Surg Glob Open*. 2021;9(3):e3498. doi:10.1097/GOX.00000000000003498.
- Tampa M, Mitran MI, Mitran CI, et al. Viral infections confined to tattoos: a narrative review. *Medicina (Kaunas)*. 2022;58(3):342. doi:10.3390/medicina58030342.
- Zhang X, Lei L, Jiang L, et al. Characteristics and pathogenesis of Koebner phenomenon. *Exp Dermatol*. 2023;32(4):310–23. doi:10.1111/exd.14709.

11. Karampinis E, Georgopoulou KE, Goudouras G, et al. Laser-induced Koebner-related skin reactions: a clinical overview. *Medicina (Kaunas)*. 2024;60(7):1177. doi:10.3390/medicina60071177.
12. Gazali M, Ruslin M, Stevanie C, et al. Short-term follow-up of surgical management verruca vulgaris with modified Estlander flap: a case report and recent literature review. *J Clin Exp Dent*. 2024;16(8):e1033-e1039. doi:10.4317/jced.61814.
13. Grosu-Bularda A, Hariga CS, Dumitru CS, et al. Clinicopathological findings and comprehensive review of Buschke-Lowenstein tumors based on a case study. *J Pers Med*. 2024;14(8):887. doi:10.3390/jpm14080887.
14. Lo Cigno I, Calati F, Albertini S, et al. Subversion of host innate immunity by human papillomavirus oncoproteins. *Pathogens*. 2020;9(4):292. doi:10.3390/pathogens9040292.
15. Huang J, Jiang Q, Liu J, et al. Two oral verrucous carcinomas in a patient with oral graft vs. host disease-recurrent verrucous carcinoma: a case report. *BMC Oral Health*. 2025;25(1):968. doi:10.1186/s12903-025-06333-3.
16. Kim DG, Lee KA. Verrucous carcinoma of the lower lip: a rare case mimicking benign lesion. *J Clin Med*. 2025;14(24):8763. doi:10.3390/jcm14248763.
17. Dey M, Grover K, Arora S, et al. Histopathological variants of oral squamous cell carcinoma operated at a cancer institute in North India. *Indian J Surg Oncol*. 2024;15(4):909-17. doi:10.1007/s13193-024-02011-y.
18. Tao J, Sun B, Chang SJ, et al. Unanticipated squamous cell carcinoma in a giant verrucous venous malformation. *Acta Derm Venereol*. 2025;105:adv44208. doi:10.2340/actadv.v105.44208.
19. Gontarz M, Urbańska M, Bargiel J, et al. Surgical management of head and neck squamous cell carcinoma metastatic to the parotid gland. *Front Oncol*. 2025;15:1579680. doi:10.3389/fonc.2025.1579680.
20. Thompson AK, Kelley BF, Prokop LJ, et al. Risk factors for cutaneous squamous cell carcinoma recurrence, metastasis, and disease-specific death: a systematic review and meta-analysis. *JAMA Dermatol*. 2016;152(4):419-28. doi:10.1001/jamadermatol.2015.4994.
21. Kristofelc N, Zidar N, Strojani P. Oral verrucous carcinoma: a diagnostic and therapeutic challenge. *Radiol Oncol*. 2023;57(1):1-11. doi:10.2478/raon-2023-0015.
22. Lee HM, Jang EJ, Na YC. Diagnosis and treatment of verrucous carcinoma of the lip: a case report. *Arch Craniofac Surg*. 2025;26(3):124-8. doi:10.7181/acfs.2024.0058.
23. Gammon VK, Simmons N, Yusuf Y, et al. Verruca actinic keratosis vs. verruca vulgaris - importance of communication, adequate specimen excision and tissue orientation. *SKIN J Cutan Med*. 2025;9(4):2559-62. doi:10.25251/ef03js48.
24. Gibson AC, Speed O, Boyette JR, et al. A systematic review of local flaps utilized for external auditory canal defects. *Craniofacial Trauma Reconstr*. 2024;17(4):NP345-57. doi:10.1177/19433875241262619.
25. Riley DS, Barber MS, Kienle GS, et al. CARE guidelines for case reports: explanation and elaboration document. *J Clin Epidemiol*. 2017;89:218-35. doi:10.1016/j.jclinepi.2017.04.026.
26. Sohrabi C, Mathew G, Maria N, et al. The SCARE 2023 guideline: updating consensus Surgical Case Report (SCARE) guidelines. *Int J Surg*. 2023;109(5):1136-40. doi:10.1097/js9.0000000000000373.
27. Jiang J, Chen Y, He Q, et al. Syringocystadenoma papilliferum and basal cell carcinoma arising in nevus sebaceous. *Clin Cosmet Investig Dermatol*. 2022;15:2021-6. doi:10.2147/CCID.S378746.
28. Schmidt BAR, El Zein S, Cuoto J, et al. Verrucous venous malformation-subcutaneous variant. *Am J Dermatopathol*. 2021;43(12):e181-e184. doi:10.1097/DAD.0000000000001963.
29. Azad A, Schweighart Gate E, Rinaldi G, et al. 30 years' experience in the use of cutaneous lasers for the treatment of verrucous venous malformations in children: a retrospective cohort study at Great Ormond Street Hospital for children. *Lasers Med Sci*. 2025;40(1):60. doi:10.1007/s10103-025-04326-w.
30. Sonthalia S, Agrawal M, Bhatia J, et al. Entodermoscopy update: a contemporary review on dermoscopy of cutaneous infections and infestations. *Indian Dermatol Online J*. 2021;12(2):220-36. doi:10.4103/idoj.IDOJ_559_20.
31. Marquez Maldonado CE, Achell Nava L, Arellano Vivero DM, et al. Vulgar verruca or seborrheic keratosis: a clinical challenge in tattooed skin. *Cureus*. 2025;17(12):e99397. doi:10.7759/cureus.99397.
32. Gorai S, Ahmad S, Raza SS, et al. Update of pathophysiology and treatment options of seborrheic keratosis. *Dermatol Ther*. 2022;35(12):e15934. doi:10.1111/dth.15934.
33. Yuan HJ, Feng Y, Sun ZZ, et al. A case of seborrheic keratosis lesions localized to eyebrow tattoos. *J Dtsch Dermatol Ges*. 2024;22(1):106-7. doi:10.1111/ddg.15264.
34. Sullivan T, Smith J, Kermod J, et al. Rating the burn scar. *J Burn Care Rehabil*. 1990;11(3):256-60. doi:10.1097/00004630-199005000-00014.
35. Draaijers LJ, Tempelman FRH, Botman YAM, et al. The Patient and Observer Scar Assessment Scale: a reliable and feasible tool for scar evaluation. *Plast Reconstr Surg*. 2004;113(7):1960-5. doi:10.1097/01.prs.0000122207.28773.56.
36. Mazur E, Kwiatkowska D, Trakatelli M, et al. Dermoscopy of subungual squamous cell carcinoma: a systematic review. *Cancers (Basel)*. 2026;18(3):446. doi:10.3390/cancers18030446.
37. Lee Y, Lee D, Yeo H, et al. Extraordinarily aggressive cutaneous sarcomatoid squamous cell carcinoma of the face: a case report. *Arch Craniofac Surg*. 2022;23(2):77-82. doi:10.7181/acfs.2022.00059.
38. Russo I, Marino D, Cozzolino C, et al. Kaposi's sarcoma: evaluation of clinical features, treatment outcomes, and prognosis in a single-center retrospective case series. *Cancers (Basel)*. 2024;16(4):691. doi:10.3390/cancers16040691.
39. El-Khoury J, Kibbi AG, Abbas O. Mucocutaneous pseudoepitheliomatous hyperplasia: a review. *Am J Dermatopathol*. 2012;34(2):165-75. doi:10.1097/dad.0b013e31821816ab.
40. Cheng KY, Yu J, Liu EW, et al. Prognostic factors of Marjolin ulcers: a meta-analysis and systematic review assisted by machine learning techniques. *Plast Reconstr Surg*. 2023;151(4):875-84. doi:10.1097/prs.00000000000010012.
41. Mokbel K, Kodresko A, Ghazal H, et al. Cryogenic media in biomedical applications: current advances, challenges, and future perspectives. *In Vivo*. 2024;38(1):1-39. doi:10.21873/in vivo.13407.
42. D'Arpa S, Cordova A, Moschella F. Further application of the bilobed flap: the split bilobed flap for reconstruction of composite posterior auricular and mastoid defects. *J Plast Reconstr Aesthet Surg*. 2006;59(12):1330-5. doi:10.1016/j.bjps.2006.06.022.
43. Moody C. Mechanisms by which HPV induces a replication competent environment in differentiating keratinocytes. *Viruses*. 2017;9(9):261. doi:10.3390/v9090261.
44. Patel KR, Chernock RD, Zhang TR, et al. Verrucous carcinomas of the head and neck, including those with associated squamous cell carcinoma, lack transcriptionally active high-risk human papillomavirus. *Hum Pathol*. 2013;44(11):2385-92. doi:10.1016/j.humpath.2013.07.011.
45. Jiang J, Liao Q, Yang J, et al. Syringocystadenoma papilliferum: a case series and literature review. *Clin Cosmet Investig Dermatol*. 2026;19:1-7. doi:10.2147/CCID.S595115.
46. Hazarika N, Taneja G, Dhanta A, et al. Dermoscopic clues to syringocystadenoma papilliferum. *J Cutan Aesthet Surg*. 2022;15(2):199-201. doi:10.4103/JCAS.JCAS_103_21.
47. Vigil SR, Yi AN, Elwood H, et al. Pediatric syringocystadenoma papilliferum arising de novo on the leg: characteristics observed with reflectance confocal microscopy. *Pediatr Dermatol*. 2025;42(4):882-4. doi:10.1111/pde.15869.
48. Leus AJG, Haisma MS, Terra JB, et al. Age-related differences in tumour characteristics and prognostic factors for disease progression in cutaneous squamous cell carcinoma of the head and neck. *Acta Derm Venereol*. 2022;102:adv00652. doi:10.2340/actadv.v101.347.
49. Jirys B, Khamaysi K, Avitan-Hersh E, et al. The role of Erbium-YAG in treating male genital warts. *J Clin Med*. 2025;14(5):1575. doi:10.3390/jcm14051575.

50. Al-Sabak H, Al-Hattab M, Al-Rammahi M, et al. The efficacy of intralesional vitamin D3 injection in the treatment of cutaneous warts: a clinical therapeutic trial study. *Skin Res Technol*. 2023;29(8):e13442. doi:10.1111/srt.13442.
51. Latif I, Sultan J, Aslam A, et al. Role of intralesional vitamin D3 in the treatment of cutaneous warts. *J Cutan Aesthet Surg*. 2021;14(4):404-8. doi:10.4103/JCAS.JCAS_170_20.
52. Kost Y, Deutsch A, Zhu TH, et al. Vaccination against human papillomavirus is not associated with resolution of verruca vulgaris in immunocompetent 9- to 21-year olds. *J Am Acad Dermatol*. 2022;87(1):250-2. doi:10.1016/j.jaad.2021.08.016.
53. Lee KH, Jeong JH, Park CJ, et al. Resolution of extensive plantar verruca vulgaris but not facial verruca plana following nonavalent human papillomavirus vaccine: a case report and literature review. *J Dermatol*. 2024;51(8):e279-e281. doi:10.1111/1346-8138.17190.
54. Long B, Mason J, Bridwell RE, et al. Managing auricular hematoma: an emergency medicine narrative review. *J Emerg Med*. 2025;69:62-75. doi:10.1016/j.jemermed.2024.08.021.
55. Burge SM, Shepherd JP, Dawber RPR. Effect of freezing the helix and the rim or edge of the human and pig ear. *J Dermatol Surg Oncol*. 1984;10(10):816-9. doi:10.1111/j.1524-4725.1984.tb01186.x.
56. Ferenczy A, Mitao M, Nagai N, et al. Latent papillomavirus and recurring genital warts. *N Engl J Med*. 1985;313(13):784-8. doi:10.1056/NEJM198509263131304.
57. Stekelenburg CM, Simons JM, Tuinebreijer WE, et al. Analyzing contraction of full thickness skin grafts in time: choosing the donor site does matter. *Burns*. 2016;42(7):1471-6. doi:10.1016/j.burns.2016.02.001.
58. Krishnan NM, Brown BJ, Davison SP, et al. Reducing wound tension with undermining or imbrication—do they work? *Plast Reconstr Surg Glob Open*. 2016;4(7):e799. doi:10.1097/gox.0000000000000799.
59. Zhou M, González PJ, Van Haasterecht L, et al. Uniaxial mechanical stretch properties correlated with three-dimensional microstructure of human dermal skin. *Biomech Model Mechanobiol*. 2024;23(3):911-25. doi:10.1007/s10237-023-01813-3.
60. Pokryvkova B, Smahelova J, Dalewska N, et al. ARG1 mRNA level is a promising prognostic marker in head and neck squamous cell carcinomas. *Diagnostics (Basel)*. 2021;11(4):628. doi:10.3390/diagnostics11040628.
61. Nunes EM, Talpe-Nunes V, Sichero L. Epidemiology and biology of cutaneous human papillomavirus. *Clinics (Sao Paulo)*. 2018;73:e489s. doi:10.6061/clinics/2018/e489s.
62. Della Fera AN, Warburton A, Coursey TL, et al. Persistent human papillomavirus infection. *Viruses*. 2021;13(2):321. doi:10.3390/v13020321.
63. Liang S, Miao J, Zheng J, et al. Clinical efficacy of 5-aminolevulinic acid photodynamic therapy combined with interferon spray in treating common and plantar warts. *Photodermatol Photoimmunol Photomed*. 2026;42(6):e70111. doi:10.1111/phpp.70111.
64. Ciccarese G, Herzum A, Serviddio G, et al. Efficacy of human papillomavirus vaccines for recalcitrant anogenital and oral warts. *J Clin Med*. 2023;12(23):7317. doi:10.3390/jcm12237317.
65. Smahelova J, Pokryvkova B, Stovickova E, et al. Aspartate- β -hydroxylase and hypoxia marker expression in head and neck carcinomas: implications for HPV-associated tumors. *Infect Agent Cancer*. 2024;19(1):26. doi:10.1186/s13027-024-00588-1.
66. Okuyama K, Tsuchiya M, Debnath KC, et al. Desmoplastic reaction in the microenvironment of head and neck and other solid tumors: the therapeutic barrier. *Ther Adv Med Oncol*. 2025;17:17588359251317144. doi:10.1177/17588359251317144.