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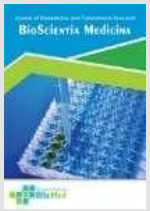

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Psoriasiform Digital Bowen's Disease: A Diagnostic Challenge and Short-Term Response to Liquid Nitrogen Cryotherapy

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A B S T R A C T

Background: Bowen's disease (BD), or squamous cell carcinoma in situ, classically presents as a slowly enlarging erythematous plaque on sun-exposed skin. However, digital Bowen's disease represents a distinct and rare clinical subset that frequently poses a significant diagnostic dilemma. Due to its unique anatomical location and morphological variability, digital BD often masquerades as benign inflammatory dermatoses, particularly psoriasis or chronic eczema, leading to dangerous therapeutic delays. **Case presentation:** We report the case of a 46-year-old male presenting with a solitary, rough, erythematous plaque on the dorsal aspect of the left index finger that had persisted for one year. The lesion was initially misdiagnosed and treated as an inflammatory condition without success. Detailed dermoscopic evaluation revealed a specific "psoriasiform" vascular pattern characterized by clustered glomerular vessels and surface scaling, raising suspicion for malignancy. Histopathological analysis confirmed the diagnosis of Bowen's disease, demonstrating full-thickness epidermal atypia with psoriasiform hyperplasia. Notably, the presence of histological koilocytic atypia suggested a potential synergistic etiology involving Human Papillomavirus (HPV) infection alongside chronic ultraviolet exposure. The patient was treated with a tissue-sparing protocol of liquid nitrogen cryotherapy to preserve digital function. **Conclusion:** Complete clinical resolution of the lesion was observed at the three-week follow-up interval, resulting in a hypopigmented macule with full preservation of joint mobility. This case highlights the critical necessity of distinguishing "psoriasiform" malignancies from true inflammatory diseases through the recognition of specific vascular arrangements in dermoscopy. Furthermore, it suggests that cryotherapy is a pragmatic, function-sparing alternative to surgical excision for digital malignancies, provided that rigorous long-term surveillance is maintained to monitor for recurrence.

1. Introduction

Bowen's disease (BD) represents the intraepidermal stage of squamous cell carcinoma (SCC), a malignant proliferation of atypical keratinocytes strictly confined to the epidermis without violation of the basement membrane zone.[1] Since its initial description by John T. Bowen in 1912, the condition has been recognized as a definitive precursor to invasive carcinoma, carrying a transformation risk estimated between 3% and 5% in immunocompetent individuals, extending significantly higher in immunocompromised populations. While the classic clinical phenotype involves a slowly enlarging, erythematous, scaly plaque on the head and neck of elderly individuals, the epidemiological landscape of the disease is evolving.[2]

Contemporary data indicate a shifting demographic toward younger patients and an increasing incidence of acral manifestations, particularly on the digits and periungual regions, which present unique diagnostic and therapeutic challenges.[3,4]

The pathogenesis of BD is multifactorial, involving a complex and often synergistic interplay between environmental carcinogens and host genetic susceptibility.[4] Chronic ultraviolet (UV) radiation remains the established primary driver for cutaneous BD, inducing specific signature mutations in the TP53 tumor suppressor gene. However, digital BD constitutes a unique pathophysiological entity. In this anatomical niche, high-risk human papillomavirus (HPV) strains—specifically the mucosal types 16 and 18, as well as the cutaneous types 34 and 48—are increasingly implicated as co-carcinogens. This viral association often dictates a specific histological architecture characterized by koilocytic atypia and psoriasiform hyperplasia, differing significantly from the atrophic variants often seen on the trunk.[5,6]

Diagnostic precision in digital BD is frequently compromised by its chameleon-like clinical behavior. The lesion's predilection for the fingers often leads clinicians to favor diagnoses of verruca vulgaris, chronic contact dermatitis, or localized psoriasis. This mimicry is not merely clinical but also extends to dermoscopy, as both psoriasis and BD can exhibit glomerular vessels.[7,8] Therefore, distinguishing these entities requires a rigorous synthesis of clinical history, specific dermoscopic vascular pattern analysis, and histopathological confirmation.

The novelty of this study rests upon the detailed clinicopathological analysis of a rare case of digital Bowen's disease in a 46-year-old male that perfectly mimicked psoriasis vulgaris, providing a comprehensive guide for clinicians to recognize this malignant mimic through the correlation of psoriasiform dermoscopic vascular patterns with psoriasiform histopathological architecture.[9,10] Furthermore, the aim of this study is to scientifically demonstrate the efficacy and functional advantages of liquid nitrogen cryotherapy as a first-line intervention for digital malignancies, utilizing a specific thermodynamic protocol designed to maximize tumor destruction while preserving the intricate mobility of the hand joints.

2. Case Presentation

The clinical investigation began with a comprehensive anamnesis of a 46-year-old male patient who presented to the Dermatology and Venereology outpatient clinic at Prof. Dr. I.G.N.G. Ngoerah General Hospital. The patient's primary concern was a persistent, evolving skin lesion located on the dorsal aspect of his left hand, specifically overlying the proximal interphalangeal region of the index finger. The lesion had been present for approximately 12 months, exhibiting a recalcitrant course that had defied previous therapeutic interventions. This prolonged duration of one year is clinically significant, as it immediately raises the index of suspicion for a neoplastic process or a chronic infectious etiology rather than an acute inflammatory dermatitis. The patient described the initial onset of the lesion as a small, asymptomatic, purple-bluish macule, a morphological description that initially suggested a vascular or traumatic etiology to previous clinicians. However, over the subsequent months, the lesion underwent a gradual centrifugal expansion, transforming from a flat macule into a roughened, indurated plaque. This morphological evolution was accompanied by the development of subjective symptomatology, including intermittent pruritus (itching) and localized nociception (pain), which are atypical for early-stage Bowen's disease but can occur when the lesion becomes hyperkeratotic or fissured, stimulating cutaneous nerve endings, as detailed in Figure 1.

A thorough review of the patient's occupational and environmental history revealed critical risk factors. The patient worked as a merchant, a profession that, in his specific context, involved significant outdoor activity. Crucially, he utilized a motorcycle as his primary mode of daily transportation. This detail is of paramount epidemiological importance; motorcycle

riders often grip the handlebars with the dorsal aspects of their hands fully exposed to direct sunlight. The patient confirmed that his hands were subjected to intense, cumulative ultraviolet (UV) radiation for prolonged periods daily. Furthermore, he engaged in regular gardening activities, adding another layer of actinic exposure. In a revealing admission regarding his photoprotective behavior, he stated a complete absence of sunscreen use or physical barriers such as protective gloves. This chronic, cumulative UV exposure is the single most established environmental driver for cutaneous squamous cell carcinoma and its precursors, inducing direct DNA damage in keratinocytes, as detailed in Figure 1.

The patient's medical history was meticulously reviewed to identify potential predisposing factors. He denied any history of direct trauma to the site, arsenic exposure (often found in contaminated groundwater or traditional medicines), or prior radiation therapy. However, the family history provided a potential genetic clue; his biological mother had succumbed to breast cancer. While breast cancer and cutaneous squamous cell carcinoma are distinct entities, a family history of malignancy can sometimes indicate a broader, underlying germline susceptibility to neoplastic disease or defects in DNA repair mechanisms. The patient had no history of systemic autoimmune diseases, diabetes mellitus, or known immunosuppression, which are traditional risk factors for accelerated carcinogenesis. Prior to this consultation, the patient had been managed by a general practitioner with a diagnosis of localized inflammatory dermatitis. He was prescribed a course of topical corticosteroids, a standard intervention for inflammatory dermatoses. The complete lack of clinical response to this anti-inflammatory regimen was a critical diagnostic pivot point, strongly suggesting that the underlying pathology was not inflammatory but rather neoplastic. Notably, the patient had previously refused a suggestion for electrocautery, citing specific concerns regarding potential scarring and functional impairment of the finger joint, a valid concern that guided our subsequent choice of tissue-sparing cryotherapy, detailed in Figure 1.

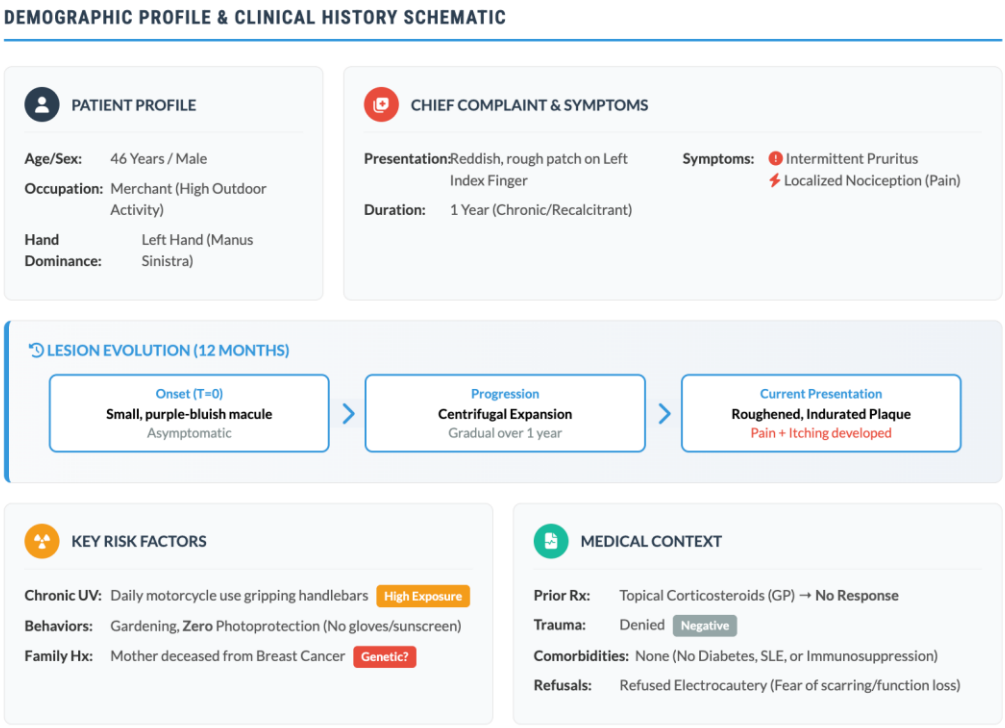

Figure 1. Demographic profile and clinical history.

Following the anamnesis, a systematic physical examination was conducted to characterize the lesion's morphology and assess the surrounding integument. The patient's general status was within normal limits, with stable vital signs, indicating a localized cutaneous process rather than a systemic syndrome. The dermatological assessment was focused on the acral region of the left hand. Inspection revealed a solitary, indurated plaque located on the dorsal aspect of the second digit (index finger), specifically overlying the proximal phalanx. The anatomical localization is clinically relevant; the dorsal hand is a "high-tension" area where the skin is thin and tightly adherent to the underlying extensor tendons, making surgical excision challenging due to the lack of tissue laxity for primary closure, detailed in Figure 2.

The lesion measured 1.2 cm x 0.8 cm in diameter, a size that falls within the manageable range for non-surgical interventions but is large enough to warrant concern for invasive progression. Morphologically, the plaque presented with an erythematous background interspersed with areas of hyperpigmentation, creating a variegated color palette that differed from the uniform "salmon-pink" often seen in psoriasis. The surface texture was notably rough and hyperkeratotic, covered by fine, adherent white scales. This squamous character is the clinical hallmark of abnormal keratinization, a feature shared by both psoriasis (parakeratosis due to accelerated turnover) and Bowen's disease (parakeratosis due to dysplastic maturation). The borders of the lesion provided a subtle but critical diagnostic clue. unlike the sharp, punched-out demarcation typically associated with psoriasis plaques, this lesion exhibited indistinct, irregular, and somewhat geographic borders. This irregularity is often a sign of the radial growth phase of an intraepidermal carcinoma, where the neoplastic clone expands asymmetrically into the surrounding epidermis, as detailed in Figure 2.

Palpation of the lesion revealed a firm, indurated consistency, distinct from the palpable elevation but softer consistency of an inflammatory plaque. There was no tenderness on palpation, suggesting the absence of acute inflammation or perineural invasion. Furthermore, there was no evidence of ulceration or bleeding, which are typically ominous signs of progression to invasive squamous cell carcinoma. A thorough examination of the regional lymphatic basins, specifically the epitrochlear and axillary nodes, revealed no lymphadenopathy, clinically staging the lesion as N0 (no regional lymph node metastasis). The surrounding skin on the dorsal hand showed signs of chronic actinic damage, including solar lentigines and mild atrophy, consistent with the patient's history of prolonged motorcycle use without photoprotection. This field cancerization effect suggests that the surrounding keratinocytes may also harbor subclinical UV-induced mutations, reinforcing the need for a treatment modality that addresses margins effectively, as detailed in Figure 2.

To bridge the gap between clinical impression and definitive diagnosis, and to specifically address the psoriasiform mimicry, advanced diagnostic modalities were employed. Dermoscopy was utilized as a non-invasive bridge to histology, providing an *in vivo* visualization of the subsurface skin structures. The dermoscopic examination was performed using polarized light to eliminate surface reflection and visualize the vascular and pigmentary structures of the papillary dermis. The examination revealed a striking and highly specific vascular pattern dominated by glomerular vessels. These vessels appear as coiled, tortuous capillaries that resemble the renal glomerulus. While glomerular vessels are the classic dermoscopic hallmark of psoriasis, representing dilated capillaries within elongated dermal papillae, their arrangement in this lesion was distinct. In psoriasis, glomerular vessels are typically distributed in a uniform, symmetric, and homogeneous array, resembling a "pearl necklace" or a regimented field.

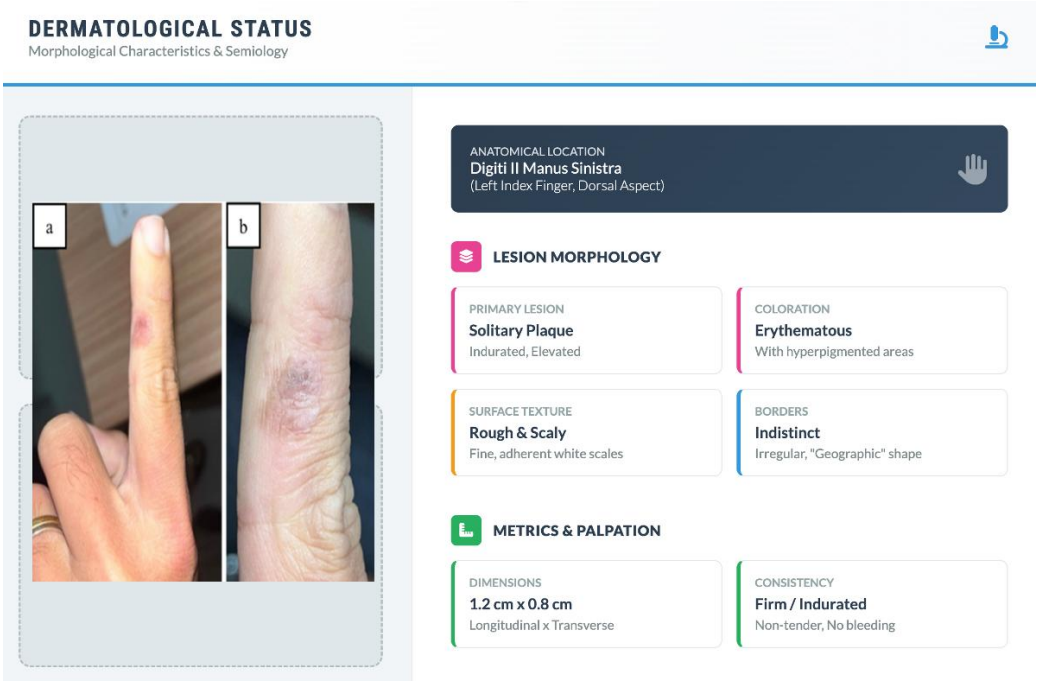

Figure 2. Dermatological status.

In contrast, the glomerular vessels observed in this patient were clustered, irregular, and distributed asymmetrically, reflecting the chaotic and unregulated neo-angiogenesis driven by the tumor's VEGF expression. Interspersed with these vascular structures were amorphous white structureless areas, representing significant hyperkeratosis and acanthosis. Importantly, there was a complete absence of a pigment network, globules, or streaks, findings that effectively ruled out a melanocytic origin (such as amelanotic melanoma) and focused the differential diagnosis on keratinocytic neoplasms, detailed in Figure 3.

Following the dermoscopic evaluation, a confirmatory 3-mm punch biopsy was performed to obtain a definitive histopathological diagnosis. The biopsy site was infiltrated with 2% lidocaine with epinephrine, and the tissue was processed for Hematoxylin and Eosin (H&E) staining. The histopathological analysis revealed a disordered epidermis characterized by marked acanthosis and elongation of the rete ridges, a pattern termed psoriasiform hyperplasia. This architectural feature explains the clinical mimicry of psoriasis. However, distinct from the reactive hyperplasia of psoriasis, the keratinocytes in this specimen exhibited full-thickness atypia, characterized by a loss of normal maturation polarity from the basal layer to the stratum corneum. Cytological examination revealed significant nuclear pleomorphism, hyperchromasia (darkly staining nuclei), and dyskeratosis (premature keratinization of single cells). These features are the hallmarks of malignancy. Crucially, the upper epidermis contained numerous cells with perinuclear halos and irregular, hyperchromatic nuclei, morphologically consistent with koilocytes. This finding is highly significant as it points towards a viral cytopathic effect, suggesting that Human Papillomavirus (HPV) infection may be a co-factor in the oncogenesis of this digital lesion. The basement membrane remained intact, confirming the in situ nature of the malignancy and ruling out invasion into the dermis. Special stains, including Periodic Acid-Schiff (PAS) and Ziehl-Neelsen, were negative, definitively excluding fungal (tinea) and mycobacterial infections as potential mimics, detailed in Figure 3.

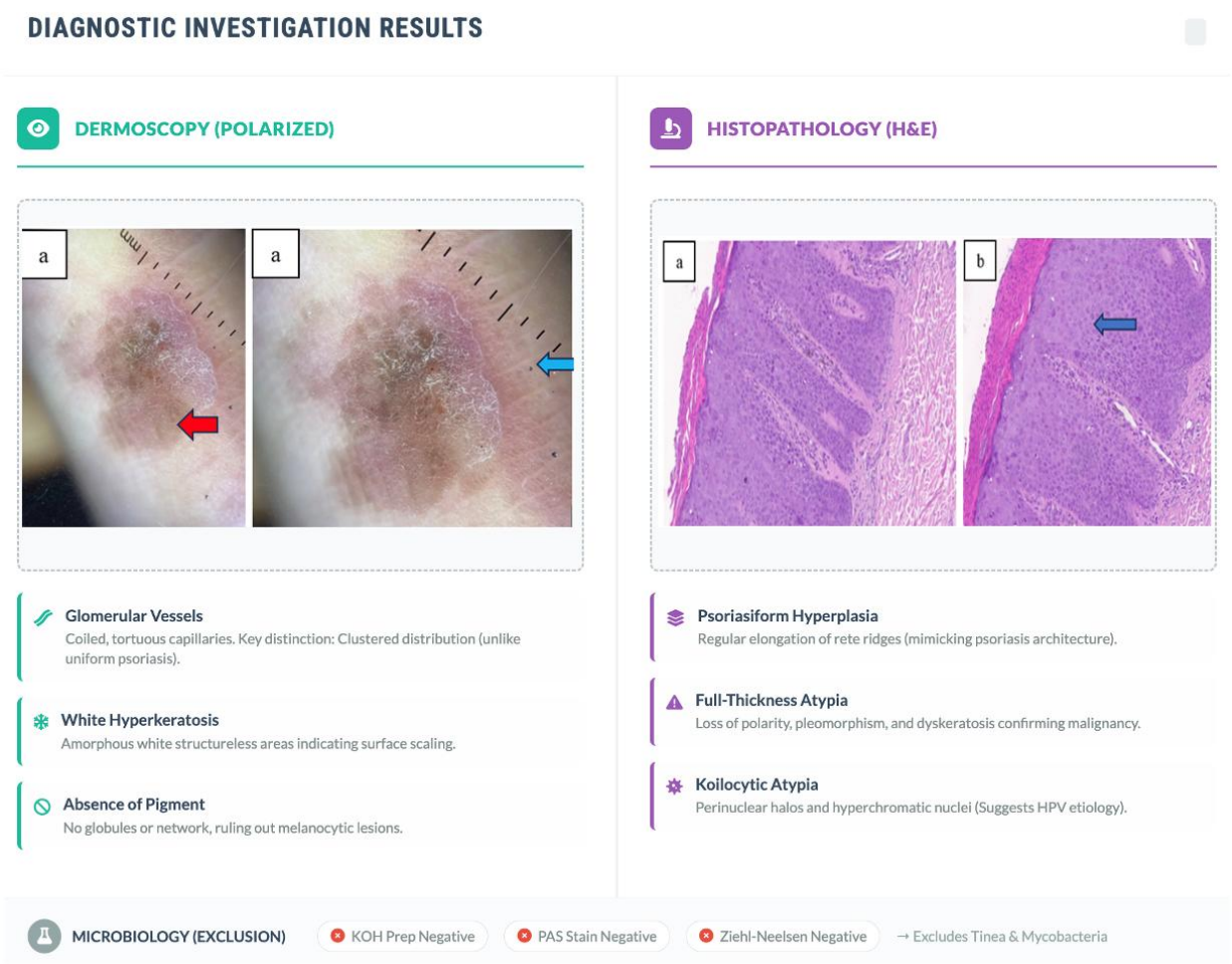

Figure 3. Diagnostic investigation results.

The therapeutic decision-making process was guided by the dual goals of ensuring oncological clearance and preserving the functional integrity of the hand. The location of the lesion over the proximal phalanx presented a specific surgical challenge; the skin in this area is relatively tight, and a standard elliptical excision with appropriate margins would likely result in significant tension, potentially requiring a skin graft or flap reconstruction. Such surgical interventions carry the risk of postoperative scarring, contracture, and limited joint mobility. Therefore, a tissue-sparing approach using cryotherapy was selected as the optimal modality. Cryotherapy utilizes the principles of cryobiology to induce targeted tissue necrosis through controlled freezing. The procedure utilized liquid nitrogen, which has a boiling point of -196°C, delivered via a handheld cryogun using the open spray technique. This method allows for precise control over the freeze field. To ensure an adequate depth of necrosis sufficient to treat the full thickness of the thickened, acanthotic epidermis, the nozzle was held approximately 1.5 cm from the skin surface. A crucial component of the protocol was the establishment of a safety margin; a 3 mm rim of clinically normal skin was included in the freeze field. This margin is essential to address subclinical tumor extension—microscopic foci of neoplastic cells that extend beyond the visible borders of the plaque. To protect the surrounding healthy tissue from collateral thermal damage, a layer of Vaseline album and gauze was applied to the periphery. The lesion was subjected to a single freeze-

thaw cycle with a freeze time of 20 seconds. This duration was carefully selected to be aggressive enough to destroy the neoplastic keratinocytes, which have a lethal temperature threshold of approximately -50°C, while minimizing the risk of damage to the underlying extensor tendon and digital nerves. The formation of a solid white ice ball and immediate blanching of the lesion confirmed that the target temperature had been reached. Following the freeze, the lesion was allowed to thaw passively in ambient air. The slow thaw phase is thermodynamically critical, as it promotes the phenomenon of recrystallization, where ice crystals fuse to form larger, more destructive crystals that mechanically disrupt cell membranes, enhancing the cytodestructive effect, detailed in Figure 4.

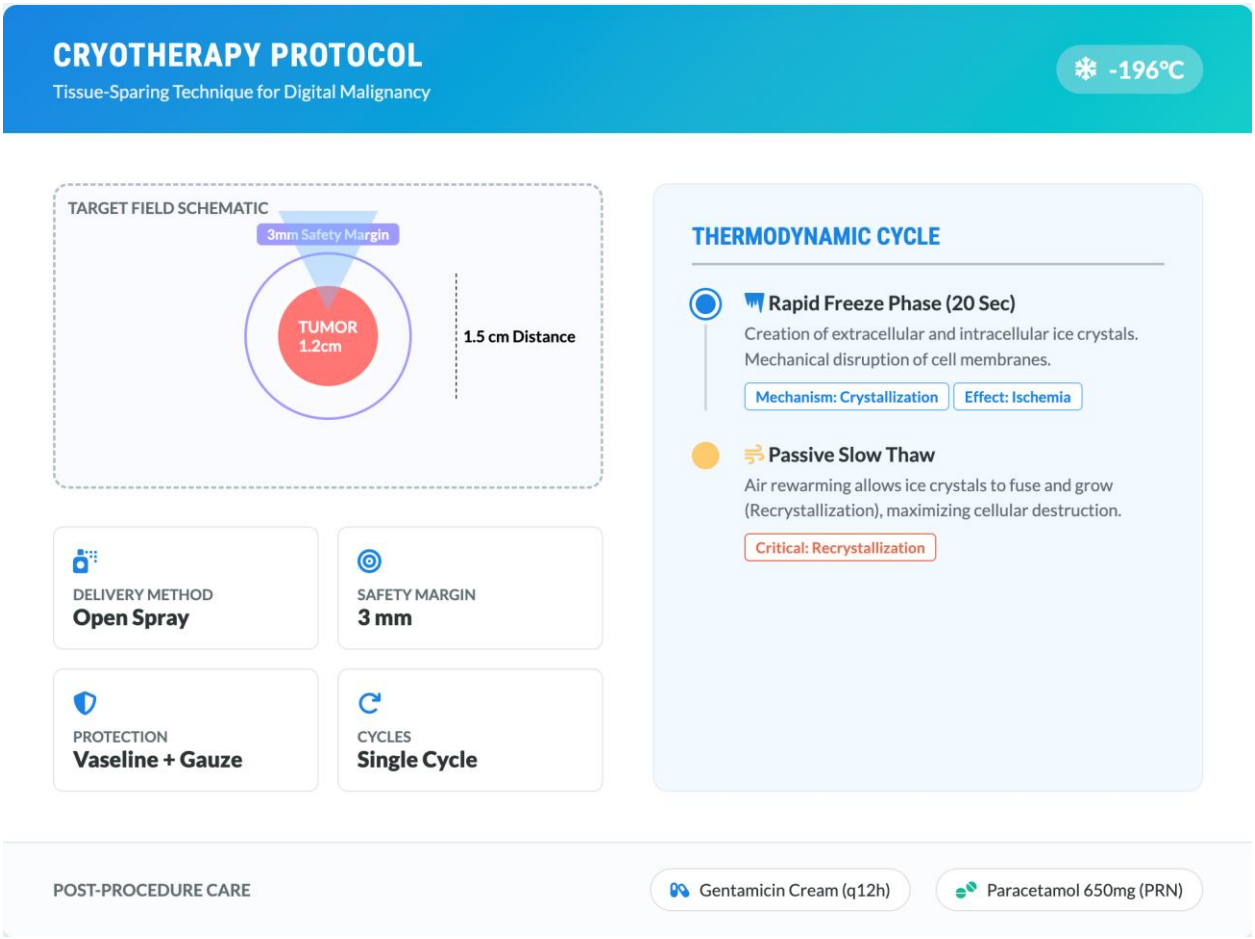

Figure 4. Cryotherapy protocol.

The patient was scheduled for a follow-up evaluation on Day 22 post-procedure to assess the therapeutic response and wound healing trajectory. The patient provided a detailed account of the post-operative course. He reported that within 24 hours of the procedure, the treated area developed a tense blister, a hallmark of second-degree frostbite, indicating successful cleavage at the dermo-epidermal junction. This blister subsequently ruptured and formed a necrotic crust (eschar) over the following week. By the time of the follow-up visit, the crust had spontaneously sloughed off, revealing the underlying re-epithelialized tissue. Clinical examination at this 3-week interval revealed complete clinical resolution of

the neoplastic plaque. The area where the rough, indurated lesion had once existed was now replaced by a solitary hypopigmented macule measuring 1.2 x 1 cm. The hypopigmentation is a known and expected sequela of cryotherapy, resulting from the high sensitivity of melanocytes to sub-zero temperatures, which are destroyed at temperatures warmer than those required to kill keratinocytes. A small central erosion covered by a thin, brownish-black crust was present, indicating the final stages of re-epithelialization. There was no clinical evidence of residual induration, erythema, or tumor activity, suggesting a complete clinical response. Importantly, the patient reported the complete resolution of the subjective symptoms of itching and pain that had plagued him for a year. Assessment of the digital function revealed a "Quo ad functionam bonam" outcome; the patient retained a full range of motion in the index finger, with no evidence of scar contracture or stiffness. This functional preservation validated the choice of cryotherapy over surgical excision. The patient was counseled on the importance of strict sun protection to prevent recurrence and the need for long-term surveillance given the potential for field cancerization in the surrounding photodamaged skin, detailed in Figure 5.

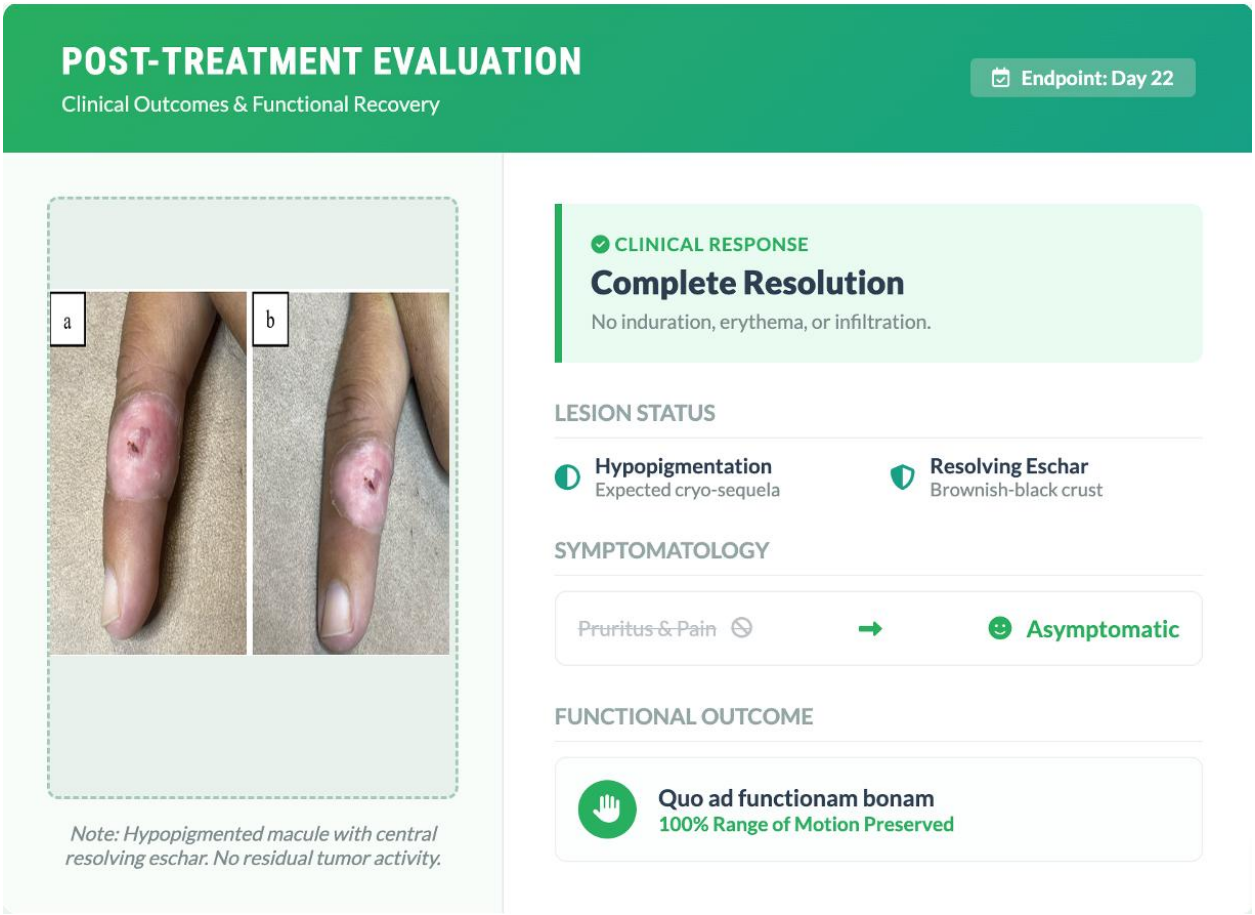

Figure 5. Post-treatment evaluation.

3. Discussion

The diagnosis and management of digital Bowen's disease (BD) represents a complex intersection of oncology, dermatology, and functional anatomy. This case report, detailing a psoriasiform presentation of digital BD in a relatively young male, serves as a

pivotal platform to discuss the intricate pathophysiological mechanisms, diagnostic nuances, and therapeutic principles governing this condition.[11] The most educationally significant aspect of this case is the perfect mimicry between the malignancy and psoriasis vulgaris. This psoriasiform presentation is not merely a visual coincidence but reflects shared architectural features in the skin that are driven by fundamentally different molecular engines.[12] The term "psoriasiform" in dermatopathology refers specifically to a pattern of epidermal reaction characterized by regular elongation of the rete ridges, often accompanied by acanthosis and parakeratosis. In benign inflammatory psoriasis, this hyperplasia is driven by a cytokine storm, predominantly involving the IL-23/Th17 axis. Pro-inflammatory cytokines such as Interleukin-17A (IL-17A), Interleukin-22 (IL-22), and tumor necrosis factor-alpha (TNF-α) stimulate the basal keratinocytes to proliferate rapidly, reducing the cell cycle from the normal 311 hours to approximately 36 hours. This accelerated turnover prevents proper maturation, leading to the retention of nuclei in the stratum corneum, manifested clinically as silvery scales and histologically as parakeratosis.[13]

In the context of digital Bowen's disease, however, this same architectural distortion is driven not by inflammation but by neoplastic autonomy. The elongation of the rete ridges seen in our patient's biopsy—termed psoriasiform hyperplasia—is a result of the clonal expansion of dysplastic keratinocytes.[14] Unlike the reactive hyperplasia of psoriasis, where the cellular architecture is maintained despite the speed of division, the neoplastic hyperplasia of Bowen's disease is characterized by full-thickness atypia.[15] This means that the normal polarity and maturation sequence of the epidermis are lost; atypical cells with pleomorphic, hyperchromatic nuclei are found in all layers, from the basal zone to the granular layer. This distinction is critical because it explains the clinical "roughness" and scaling reported by the patient. The dysplastic cells fail to produce a functional stratum corneum, resulting in a disordered, parakeratotic barrier that mimics the scale of psoriasis but lacks the silvery, micaceous quality typical of the inflammatory disease. Furthermore, the clinical finding of indistinct, geographic borders in our case serves as a subtle but vital differentiator. Psoriasis, being a regulated inflammatory process, typically creates sharply demarcated plaques where the transition from lesional to normal skin is abrupt. In contrast, the radial growth phase of an intraepidermal carcinoma is irregular; the neoplastic clone expands asymmetrically, creating borders that fade indistinctly into the surrounding tissue or exhibit jagged, irregular contours.[16]

Dermoscopy serves as a powerful, non-invasive tool to differentiate these entities, provided the clinician understands the underlying physics of light interaction and tumor angiogenesis. The hallmark dermoscopic feature observed in this case was the glomerular vessel.[17] Physiologically, these structures represent dilated, tortuous capillary loops located within the dermal papillae. In psoriasis, the elongation of the rete ridges is regular and rhythmic, leading to a uniform elongation of the intervening dermal papillae. Consequently, the dilated capillaries within these papillae are arranged in a symmetric, homogeneous, and evenly spaced distribution, often described as a pearl necklace or a regimented field of dots and coils. This uniformity reflects the orderly nature of the inflammatory signal.

In sharp contrast, the vascular architecture in Bowen's disease is dictated by tumor-induced neo-angiogenesis. Malignant keratinocytes secrete high levels of vascular endothelial growth factor (VEGF) and other angiogenic factors to sustain their metabolic demands. However, unlike physiologic angiogenesis, tumor angiogenesis is chaotic and unregulated.[18] The resulting vessels are tortuous, leaky, and varied in caliber. Therefore, while glomerular vessels are present in Bowen's disease due to the acanthotic epidermis, their arrangement is clustered, irregular, and distinctively asymmetric. Our patient’s dermoscopic examination revealed precisely this pattern: glomerular vessels that were recognizable as coiled capillaries but lacked the orderly regiments

seen in psoriasis. They were clustered in peripheral zones and interspersed with amorphous white structureless areas. These white areas correspond to the hyperkeratotic scale that scatters light, preventing visualization of the underlying dermis. The combination of clustered glomerular vessels and white structureless areas in a solitary lesion has a specificity for Bowen's disease approaching 90%, far superior to clinical inspection alone. The absence of pigment networks or globules was equally important, as it effectively ruled out melanocytic mimics such as amelanotic melanoma, which would display milky-red areas or polymorphous vessels.[18]

Understanding the etiology of digital Bowen's disease requires navigating the complex intersection of environmental carcinogenesis and viral oncogenesis. We propose a "Two-Hit" pathogenic model for this case, positing a synergistic interaction between chronic ultraviolet (UV) radiation and high-risk human papillomavirus (HPV) infection. The patient's occupational history as a motorcycle rider is of paramount importance. The dorsal hands are among the most sun-exposed sites on the human body, receiving maximal incidence of UV radiation during gripping maneuvers. UV-B radiation (280–320 nm) acts as a potent mutagen by directly absorbing into the DNA of keratinocytes. This energy absorption causes the formation of covalent bonds between adjacent pyrimidine bases, resulting in cyclobutane pyrimidine dimers (CPDs) and 6-4 photoproducts. In a healthy cell, these DNA lesions are detected by the nucleotide excision repair (NER) pathway, and the cell cycle is arrested by the p53 protein to allow for repair. If the damage is irreparable, p53 initiates apoptosis. However, chronic, high-intensity UV exposure can overwhelm these repair mechanisms. Furthermore, UV radiation has a specific affinity for mutating the TP53 gene itself. The classic UV signature mutation involves a C→T transition at dipyrimidine sites (CC→TT). When the TP53 gene is mutated, the Guardian of the Genome is inactivated. The cell loses its G1/S checkpoint control, allowing keratinocytes with damaged DNA to enter the synthesis phase and replicate. This leads to the clonal expansion of mutated cells, forming the microscopic basis of actinic keratosis and Bowen's disease.

The histopathological finding of koilocytic atypia in our patient's specimen provides strong evidence for the Second Hit. Koilocytes—characterized by a large, acentric, hyperchromatic nucleus surrounded by a clear perinuclear halo—are the pathognomonic footprint of productive HPV infection. In the context of digital and periungual Bowen's disease, high-risk mucosal HPV types (such as HPV-16 and HPV-18) and cutaneous types (such as HPV-34 and HPV-48) are increasingly implicated. The oncogenic mechanism of high-risk HPV involves two key viral oncoproteins: E6 and E7. The E6 oncoprotein targets the p53 tumor suppressor protein. It binds to p53 and recruits the E6-associated protein (E6AP), a cellular ubiquitin ligase. This complex ubiquitinates p53, marking it for rapid degradation by the proteasome. In a cell already subjected to UV damage, the degradation of any remaining functional p53 by E6 represents a catastrophic failure of genomic surveillance. Simultaneously, the E7 oncoprotein targets the Retinoblastoma protein (pRb). In a resting cell, pRb binds to the E2F transcription factor, preventing it from activating the genes necessary for DNA synthesis. E7 binds to pRb with high affinity, disrupting the pRb-E2F complex. This releases E2F, which then translocates to the nucleus and drives the cell into the S-phase of the cell cycle, forcing replication even in the presence of DNA damage. This viral drive is often responsible for the specific psoriasiform architecture seen in this case. The virus forces the host keratinocytes to proliferate to increase the production of viral particles and facilitate shedding from the skin surface. This synergy between UV-induced DNA damage and HPV-mediated cell cycle deregulation creates a fertile environment for malignant transformation, explaining the occurrence of this lesion in a relatively young patient.[19]

While psoriasis was the primary differential diagnosis given the clinical presentation, rigorous medical practice demands the exclusion of other great

mimickers in the acral region. Amelanotic Melanoma: This is the most dangerous potential mimic. Acral amelanotic melanoma can present as a non-pigmented, erythematous nodule or plaque. It is often misdiagnosed as a pyogenic granuloma, wart, or eczema. Dermoscopy is the critical discriminator; melanoma typically displays milky-red areas, polymorphous vessels (combinations of dots, lines, and loops), and subtle remnants of pigmentation at the periphery.[20] The absence of these features and the specific finding of glomerular vessels in our case made melanoma unlikely, but histopathology remains the gold standard for exclusion. Verruca Vulgaris (Common Wart): Warts are ubiquitous on the fingers and share the histological feature of koilocytosis with HPV-associated Bowen's disease. However, clinically, warts typically present with a verrucous, vegetating surface rather than a scaly plaque. Dermoscopically, warts exhibit frog-spawn or papillomatous projections with central thrombosed capillaries (red or black dots), distinct from the coiled glomerular vessels of Bowen's disease. Chronic Eczema (Lichen Simplex Chronicus): Chronic rubbing or scratching can lead to lichenification, presenting as a thickened, scaly plaque. However, eczema typically presents with dotted vessels on dermoscopy and intense pruritus. While our patient reported itching, the unilateral, solitary nature of the lesion and the lack of response to steroids argued strongly against a primary eczematous process.

The decision to utilize cryotherapy over surgical excision was driven by the anatomical constraints of the digit and the desire to preserve function. Understanding the biophysics of cryosurgery is essential for optimizing oncological outcomes. Liquid nitrogen, with a boiling point of -196°C, destroys tumor cells through distinct, sequential thermodynamic phases. The immediate effect of applying liquid nitrogen is rapid cooling. As the tissue temperature drops below 0°C, ice crystals begin to form in the extracellular space. This crystallization process removes pure water from the extracellular fluid, leaving behind a hyperosmotic solute. This osmotic gradient draws water out of the intracellular compartment, causing cell shrinkage and membrane collapse. As the cooling continues and the temperature drops below -15°C to -20°C, intracellular ice crystals begin to form. These crystals act as microscopic shards, mechanically piercing the cell membrane, mitochondrial membranes, and the nuclear envelope. This irreversible structural damage is the primary mechanism of cell death in the rapid freeze phase.[17,18]

The second mechanism of destruction targets the tumor's microvasculature. Freezing damages the endothelial lining of the capillaries and venules supplying the tumor. This endothelial injury exposes the subendothelial collagen, triggering platelet aggregation and the formation of microthrombi. The resulting stasis of blood flow leads to severe ischemic necrosis. This mechanism is particularly effective for Bowen's disease, as the metabolically active neoplastic cells are highly dependent on this neo-angiogenic vascular bed. The destruction of the vasculature ensures that even cells that might survive the initial thermal shock are subsequently starved of oxygen and nutrients, leading to delayed necrosis in the days following the procedure. The thaw phase is arguably the most critical and often overlooked component of the cryosurgical cycle. In our protocol, we utilized a passive, slow thaw. Physics dictates that during a slow thaw, small, unstable ice crystals fuse together to form larger, thermodynamically stable crystals—a process known as recrystallization. These growing crystals exert significant mechanical stress on the cell cytoskeleton and membranes, disrupting cellular integrity. A rapid thaw (e.g., applying heat) would minimize this effect; therefore, allowing the tissue to return to ambient temperature without assistance maximizes the cytodestructive efficacy. The lethal temperature threshold for keratinocytes is generally considered to be between -20°C and -50°C. By utilizing an open spray technique with a sufficient freeze time (20 seconds), we ensured that the ice ball extended laterally and deeply enough to encompass the entire neoplastic clone, including the follicular extensions

which can serve as a reservoir for recurrence.

The management of digital Bowen's disease requires a careful balance between cure rates and morbidity. Why was cryotherapy the superior choice in this specific case? Versus Surgical Excision: The dorsal skin of the finger is thin, with very little subcutaneous fat, and is tightly tethered to the underlying extensor tendon apparatus. A standard elliptical excision with the recommended 4-5 mm oncological margins would create a significant defect that could not be closed primarily (side-to-side) without excessive tension. Tension on a digital wound can lead to dehiscence, necrosis, or restriction of joint flexion. Therefore, excision would likely require a full-thickness skin graft or a local flap, both of which introduce secondary donor sites, increase the risk of infection, and require prolonged immobilization. Cryotherapy avoids these complications entirely, allowing for healing by secondary intention which, on the concave surfaces of the hand, often yields excellent cosmetic and functional results. Versus Topical 5-Fluorouracil (5-FU): Topical chemotherapy with 5-FU is an effective treatment for Bowen's disease. However, the protocol typically requires twice-daily application for 4 to 6 weeks. The mechanism of action involves interfering with DNA synthesis, which induces a vigorous inflammatory response characterized by erythema, erosion, crusting, and significant pain. On a functional part of the body like the index finger, this prolonged period of inflammation can limit daily activities and grip function. Furthermore, compliance is a major issue with topical regimens; patients often discontinue therapy prematurely due to the local skin reaction. Cryotherapy offers the advantage of a one-and-done intervention (or a limited number of sessions), eliminating the variable of patient compliance. Versus Photodynamic Therapy (PDT): PDT is a highly effective option with excellent cosmetic outcomes. However, it is significantly more expensive and time-consuming than cryotherapy. Furthermore, PDT in acral regions is notoriously painful due to the high density of nerve endings in the fingers. The intense burning sensation during illumination often requires nerve blocks for pain management. In resource-limited settings or for patients with a low pain threshold, cryotherapy remains a more pragmatic and accessible option.

While the short-term outcome in this case was excellent, scientific rigour demands the acknowledgment of limitations. The diagnosis of viral etiology relies on histomorphological evidence (koilocytes) rather than molecular confirmation. Definitive proof of HPV involvement would require polymerase chain reaction (PCR) or p16 immunohistochemistry, which were not available. Additionally, while clinical resolution was achieved at 22 days, this does not equate to a guaranteed long-term cure. Recurrence of Bowen's disease, particularly in acral sites where the neoplasm can extend down the hair follicle epithelium (the follicular reservoir), is a well-documented phenomenon. Recurrence rates for cryotherapy can range from 5% to 10% within 5 years. Therefore, the success reported here is provisional. The patient has been entered into a strict surveillance protocol, with scheduled dermoscopic monitoring every 6 months to detect any signs of repigmentation, scaling, or vascular atypia that would signal a recurrence.[19,20]

4. Conclusion

Digital Bowen’s disease serves as a master of disguise, capable of deceiving even experienced clinicians by mimicking benign inflammatory conditions such as psoriasis. This case reinforces the critical lesson that any persistent, solitary, psoriasiform plaque on the digits warrants a high index of suspicion and a low threshold for biopsy. This study validates the utility of dermoscopy in identifying the subtle chaotic arrangement of glomerular vessels that hints at malignancy. While the Two-Hit hypothesis of UV and HPV synergy offers a compelling biological explanation for the psoriasiform histology, clinicians must remain aware of the need for thorough histopathological evaluation. Finally, we conclude that liquid nitrogen cryotherapy represents a highly effective, function-sparing therapeutic modality for

digital BD. It offers the distinct advantage of preserving joint mobility in a functionally critical area. However, this clinical success must be tempered with caution; the preservation of tissue comes with the responsibility of rigorous, long-term surveillance to guard against the risk of recurrence inherent in non-surgical management.

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