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REVIEW ARTICLE

Pressure and Skin: A Review of Disease Entities Driven or Influenced by Mechanical Pressure

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Abstract

Skin perceives and reacts to external mechanical forces to create resistance against the external environment. Excessive or inappropriate stimuli of pressure may lead to cellular alterations of the skin and the development of both benign and malignant skin disorders. We conducted a comprehensive literature review to delve into the pressure-induced and aggravated skin disorders and their underlying pressure-related mechanisms. Dysregulated mechanical responses of the skin give rise to local inflammation, ischemia, necrosis, proliferation, hyperkeratosis, impaired regeneration, atrophy, or other injurious reactions, resulting in various disease entities. The use of personal devices, activities, occupations, weight bearing, and even unintentional object contact and postures are potential scenarios that account for the development of pressure-related skin disorders. The spectrum of these skin disorders may involve the epidermis (keratinocytes and melanocytes), hair follicles, eccrine glands, nail apparatuses, dermis (fibroblasts, mast cells, and vasculature), subcutis, and fascia. Clarifying the clinical context of each patient and recognizing how pressure at the cellular and tissue levels leads to skin lesions can enhance our comprehension of pressure-related skin disorders to attain better management.

Key Points

Mechanical pressure impacts skin integrity in conjunction with other physical forces that lead to inflammation, ischemia, necrosis, proliferation, hyperkeratosis, impaired regeneration, atrophy, or other injurious reactions, resulting in diseased skin at macroscopic, cellular, and tissue levels.

The use of personal devices, activities, occupations, weight bearing, and even unintentional object contact and postures are common causes of pressure-related skin disorders, and thus a thorough clinical history and a dermatological examination are crucial to reach an accurate diagnosis and to provide suitable management.

Alterations in skin cells such as fibroblasts, keratinocytes, mast cells, and melanocytes as well as in different levels of tissues can be elicited by pressure and result in various pathological changes based on different clinical contexts.

1 Introduction

Human skin perceives, withstands, and reacts to various mechanical factors, including friction, occlusion, and pressure. There are several reviews on the effects of friction [1, 2] and occlusion [3, 4] on the skin. However, aside from pressure ulcers (PUs), few papers provide an integrated narration of the influence of mechanical pressure on skin disorders. Normal force or its component creates pressure unevenly on different areas of the skin, particularly bony prominences, distal ends of limbs, and gravity-dependent body parts. Though the skin can resist and modulate most pressure and other physical forces, excessive or inappropriate mechanical stimuli may lead to the development of skin disorders [5]. This article aims to examine the effect of mechanical pressure on dermatological disorders, reviewing the clinical characteristics and mechanisms, diagnosis, and management.

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2 Methods

We performed a literature search in electronic databases (PubMed, Embase, Google Scholar) for relevant articles from inception to 31 May, 2023 utilizing the keywords: (pressure OR compression OR gravity OR weight-bearing OR mechanical load) AND (skin OR cutaneous OR dermatologic OR dermatosis OR dermatitis). Titles and abstracts were inspected to include articles concerning pressure-related skin disorders for review. Pressure-related skin disorders, as defined in this review, are primarily characterized by their significant mechanical etiology, and these disorders are either directly driven, induced, or caused by pressure or they may have pressure acting as a contributory co-factor where other mechanical forces such as friction are also involved. Articles regarding pressure as psychological stress were removed to avoid polysemy. Pressure referring to the potential effect of hydrostatic pressure was not considered, such as the diseases of aquagenic syringeal acrokeratoderma and hereditary papulotranslucent acrokeratoderma. Articles focusing on pressure or compression as therapeutics were also excluded. Only articles available in English were included.

3 Keratotic Diseases

The effect of pressure on cutaneous disorders can result in various pathological alterations such as inflammation, hyperkeratosis, ischemia, proliferation, and atrophy, either individually or in combination. These manifestations occur in diverse life situations, such as using a personal device, performing activities, and even maintaining a posture. We review and categorize these diseases based on their major pathological changes (Table 1, Fig. 1).

3.1 Clavus (Corn) and Callus (Callosity)

Clavi are well-circumscribed painful hyperkeratotic lesions that demonstrate a central thickening of the stratum corneum (Fig. 2a), while calluses are broader, more ill-defined lesions and are sometimes advantageous to populations such as athletes and musicians because of the alleviation of pain as a result of the thickened skin [6]. These common lesions are apt to emerge at body sites with repetitive exposure to mechanical pressure and friction, especially the feet. Increased plantar pressure may enhance the turnover rate of keratinocytes in localized skin, thereby leading to epidermal proliferation [7]. The mechanical load of weight bearing is more pronounced at the metatarsophalangeal joints, where calluses form and serve as a mechanical trigger to further increase plantar pressure. Management of the disorder relies on sharp debridement and protection, for which regular paring and covering with foam pads or silicone toe sleeves as cushioning can be effective [8]. Surgical intervention should only be considered for the purpose of correcting an anatomical misalignment to reduce mechanical pressure once conservative treatment fails. Nevertheless, metatarsal osteotomy is usually discouraged because of its unpredictable clinical outcome [8].

3.2 Palmoplantar Keratoderma

As sites frequently loaded with physical pressure, the palms and soles are inclined to abnormal keratinization to resist mechanical stress and protect underlying structures [9]. Palmoplantar keratoderma (PPK) represents an extensive category of hyperkeratotic disorders of acral regions that often associates with a myriad of extracutaneous manifestations, such as cardiomyopathy, deafness, and malignancies, in an acquired or hereditary manner [10, 11]. Mechanical pressure may play a crucial role in the development of hereditary PPKs apart from genetic factors [12]. Among different morphological patterns, focal [13], striate [14], and punctate [15] PPKs are particularly mentioned in relation to pressure.

To date, PPKs are incurable and mostly managed through symptomatic relief. Application of topical keratolytics, emollients, and corticosteroids along with wet dressings may combat hyperkeratosis and inflammation, while systemic retinoids, especially acitretin, provide effective symptomatic alleviation in non-epidermolytic PPKs [11, 16]. The intricate alterations in palmoplantar homeostasis caused by mechanical pressure make the PPK family a conundrum that warrants more investigations.

3.3 Senile Gluteal Dermatosis

Senile gluteal dermatosis demonstrates hyperkeratotic, lichenified, brownish plaques on the gluteal cleft and bilateral buttocks along with horizontal hyperkeratotic ridges, forming the characteristic “three corners of triangle” bordered by coccyx and ischial tuberosities [17, 18]. The dermatosis is more prevalent in lean elderly Asian men [18]. A biopsy of the lesion reveals orthokeratotic hyperkeratosis, epidermal psoriasiform hyperplasia, follicular plugging, and papillary dermal vascular dilatation with a perivascular lymphohistiocytic infiltrate [17–19]. Prolonged sitting is a major exacerbating factor of senile gluteal dermatosis, as sitting upright concentrates forces of pressure and chafing at the gluteal area medial to bony prominences, leading to local inflammation [18, 20].

Table 1 List of pressure-related skin disorders categorized by major pathological changes

Major pathological changes	Disease entities
Keratotic diseases	Clavus (corn) and callus (callosity)
	Palmoplantar keratoderma
	Senile gluteal dermatosis
	Traumatic anserine folliculosis
Ischemic diseases	Chondrodermatitis nodularis
	Chondrodermatitis nodularis helicis
	Chondrodermatitis nodularis nasi
	Coma blister
	Pressure ulcer
	Pressure alopecia
Proliferative diseases	Acanthoma fissuratum
	Atypical decubital fibroplasia (decubital ischemic fasciitis)
	Cutaneous metaplastic synovial cyst
	Acral melanoma
Atrophic diseases	Lipoatrophia semicircularis
	Anetoderma of prematurity
Immune-mediated diseases	Delayed-pressure urticaria and pressure-aggravated urticaria
Miscellaneous[a]	Ingrown nail (onychocryptosis)
	Sports-related skin disorders
	Skater's/athlete's nodule
	Skater's pad
	Skate/lace bite
	Biker's panniculitis
	Rower's rump/saddle sore of cyclist
	Perineal nodular induration of cyclist
	Swimmer's nose deformity
	Tennis/jogger's toe
	Golfer's nail
	Talon noir
	Musical instrument-related skin disorders
	Finger/lip callosity
	Fiddler's neck (submandibular)
	Nodular cervical induration of violinist
	Cheilitis
	Prosthesis-related skin disorders
	Blister
	Contact dermatitis
	Acroangiodermatitis
	Lichenification
	Ulceration
	Malignancy
	Marjolin ulcer
	Epidermal hyperplastic changes
	Hyperkeratotic papule
	Epidermoid cyst
	Mechanical acne

[a]Lesions listed in this category may result from various pathological changes, making an accurate classification of each lesion into the categories mentioned above trivial and challenging

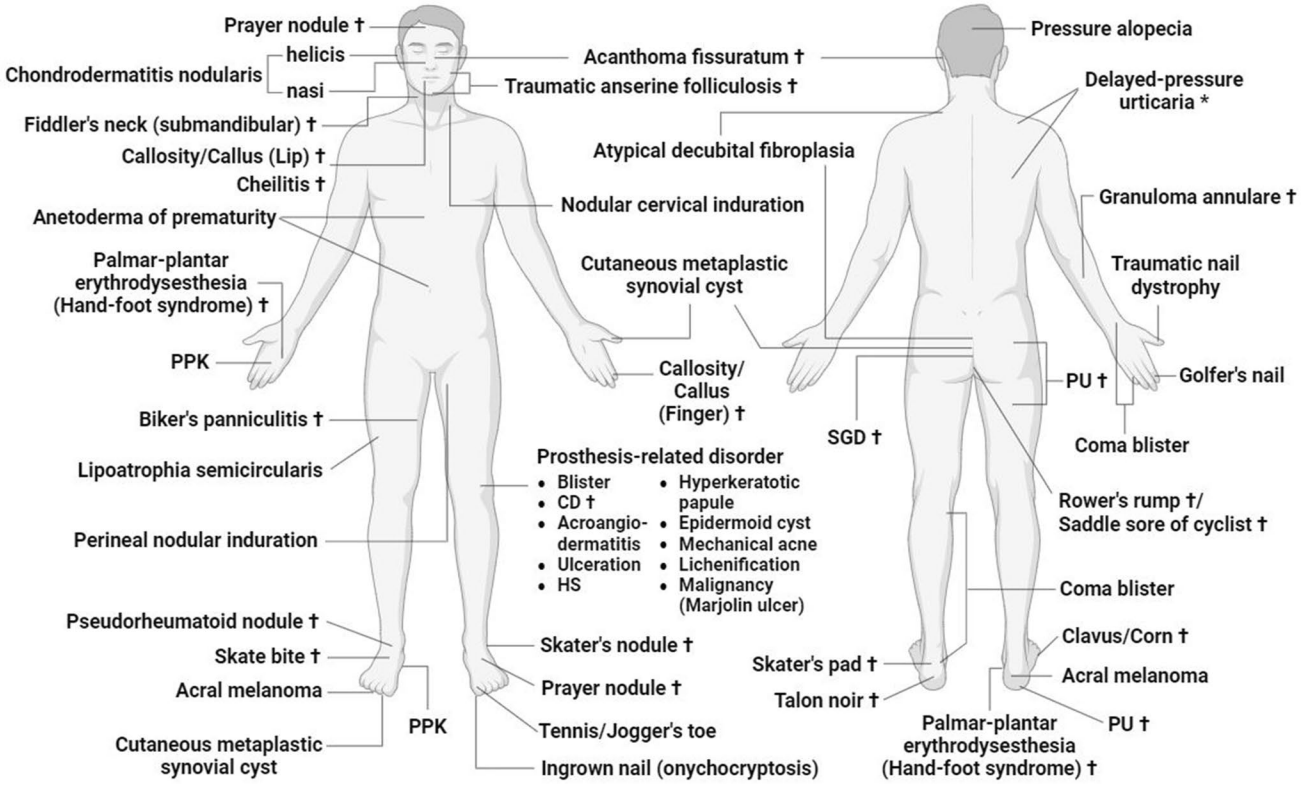

Fig. 1 Common representative sites of pressure-related skin disorders. Note that the cutaneous lesion sites depicted here represent typical locations for each disease entity. Manifestations can vary and are not exclusive to these sites, depending on the clinical scenario. Created with BioRender.com. *CD* contact dermatitis, *HS* hidradenitis suppurativa, *PPK* palmoplantar keratoderma, *PU* pressure ulcer, *SGD* senile gluteal dermatosis, * indicates a representative site of a lesion caused by the pressure from backpack shoulder straps, † indicates disease entities where pressure may act as a co-factor, either alongside other mechanical forces such as friction and shearing stress or as a less common etiological factor

Japanese and Korean researchers reported a relatively high prevalence of senile gluteal dermatosis as a result of their conventional flooring of tatami, a woven straw mat, and the Korean-style mattress, respectively, which is frequently used for sitting and lying [21, 22].

Therapeutically, aside from pressure offloading through a lifestyle modification, topical calcipotriol has been effective in reducing keratinocyte proliferation [18], whereas topical corticosteroids and keratolytics are of limited treatment value [20]. Topical and systemic retinoids might be beneficial once ulcerative or erosive lesions develop [22].

3.4 Traumatic Anserine Folliculosis

Traumatic anserine folliculosis is characterized by grouped follicular papules that give the skin an appearance reminiscent of goose skin (anserine) and a texture similar to sandpaper [23]. Pressure and friction are the most well-established etiological factors. This is evidenced by the development of lesions on the chin, mandibular area, and cheek, where pressure is typically applied when patients rest their chins and cheeks on their palms while studying or viewing smartphones and tablets [23, 24]. Histopathologically, hyperkeratosis, hypergranulosis, rudimentary follicles, and a dilated follicular opening with retention of keratotic materials are generally observed [23, 25]. The papules are often asymptomatic, and thus the clinical history of the patients' habitual chin-on-palm posture can be overlooked. This stresses the importance of a thorough history taking by clinicians, as the lesion distribution practically complies with the contact site of the palms, and these papules themselves might go unnoticed by the patients [26]. Once traumatic anserine folliculosis is diagnosed, removal of the source of pressure by postural and habitual modifications is crucial, while topical keratolytics, retinoids, and vitamin D analogs have been prescribed to accelerate lesion resolution [23, 25–27].

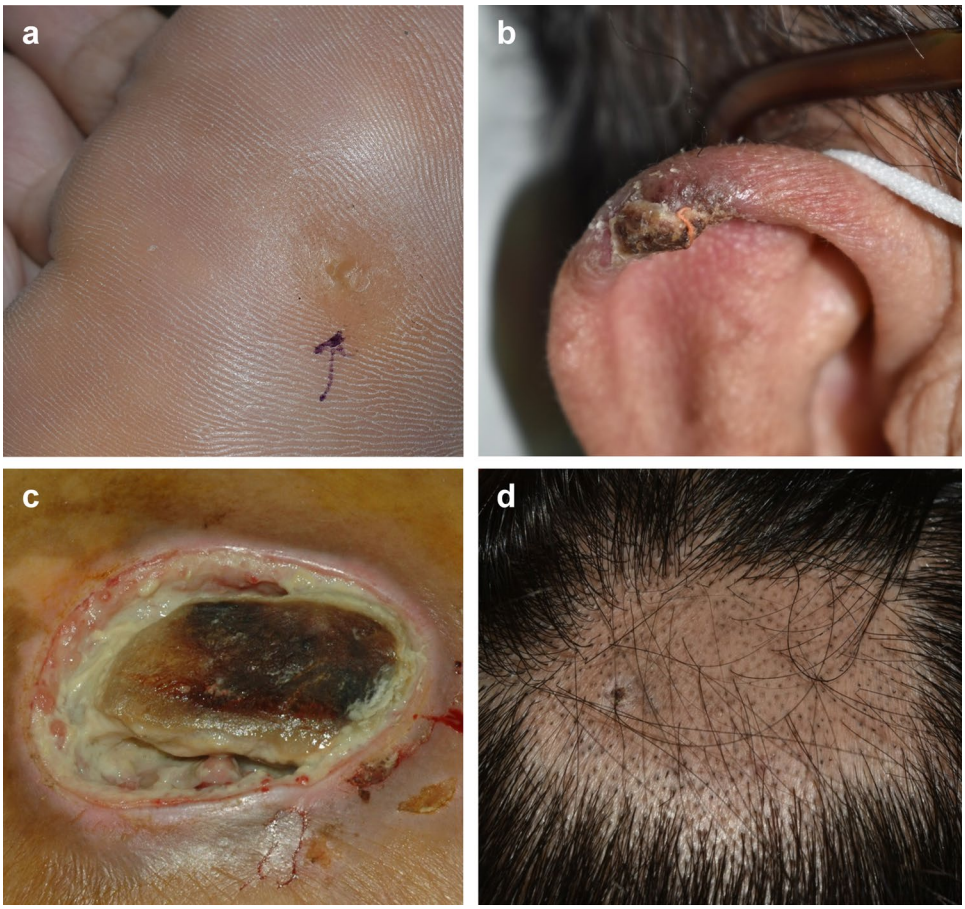

Fig. 2 Clinical images of pressure-related skin disorders: **a** clavus on the sole (arrow), **b** chondrodermatitis nodularis helicis, **c** pressure ulcer at the sacral area, and **d** pressure alopecia

4 Ischemic Diseases

4.1 Chondrodermatitis Nodularis

Chondrodermatitis nodularis is characterized by benign inflammation of auricular cartilages. Patients often present with a painful, ulcerated, flesh-colored, or erythematous nodule on the helix (chondrodermatitis nodularis helicis), or sometimes antihelix, of the ear (Fig. 2b). Although the condition is more prevalent in male individuals, there has been a notable increase in the number of elderly female individuals with this condition [28]. Chronic exposure to mechanical pressure followed by ischemia of the perichondrium is regarded as the culprit [29, 30]. The long distance from the helix to the blood supply origin of pinna worsens the situation. Lesions are attributable to pressure induced by the uses of cellphone [31], headphones [32], earbuds [33], hearing aids, and a tight-fitting helmet and cap [34, 35]. Sleeping in a lateral position habitually also serves as a risk factor [30]. A rare variant on the nasal dorsum was coined chondrodermatitis nodularis nasi, which has been reported to be associated with the oxygen devices in patients with obstructive sleep apnea [36, 37] as well as post-Mohs surgery [38].

Therapeutically, surgical interventions of excision, curettage, or the punch-and-graft technique are the most direct approaches with satisfying results in the recurrence rate and postoperative appearance [29, 39]. Meanwhile, relieving pressure by using specially designed cushions and avoiding the culprit sources are of utmost importance for patients regardless of undergoing surgery or not [29, 40–42]. Photodynamic therapy and topical nitroglycerin have also shown promising outcomes [43–46]. Intralesional hyaluronic acid may be efficacious for instant pain relief and the healing of ulcerations [47]. A systematic review suggested a treatment algorithm that prioritized surgery, followed by nitroglycerin and/or pressure-relieving measures [48].

4.2 Coma Blister

Coma blisters are tense bullae or vesicles with erythematous-to-violaceous plaques developed under various unconscious scenarios. A drug overdose involving barbiturates, benzodiazepines, opioids, ethanol, and psychotropics is the most well- known event [49, 50]. Local pressure originated from an unchanged posture during a drug-induced coma and the ensuing tissue hypoxia and ischemia contribute to the

degeneration of the epidermis and eccrine sweat ducts and glands, thereby inciting blister formation in pressure-prone areas including the forearms, fingers, malleoli, heels, buttocks, and medial knees [51, 52]. Common histopathological findings include intraepidermal or subepidermal bullae, as well as the pathognomonic occurrence of eccrine sweat gland necrosis [52]. Aside from pressure damage, metabolic acidosis, hypoxemia, hypokalemia, and hypothermia after a drug overdose also collaboratively predispose patients to pressure-related dermatosis [49, 53]. In many instances, these self-limited blisters form within 24–72 hours after a loss of consciousness and spontaneously subside within a month [51, 52]. Preventive measures should be taken for infectious complications in high-risk patients. If rhabdomyolysis co-occurs and leads to compartment syndrome, an emergent fasciotomy following debridement should be performed [51].

4.3 Pressure Ulcer

Pressure ulcers, also known as pressure injuries, are among the most common pressure-related medical conditions, particularly affecting populations with impaired mobility and sensation, such as patients with a spinal cord injury and those who are bedridden or wheelchair bound [54, 55]. Prolonged positional maintenance in recumbent, prone, or sitting positions predisposes individuals to PUs at certain sites. These sites often include, but are not limited to, the sacrum, ischium, greater trochanter, and calcaneus—often representing bony prominences of the body (Fig. 2c) [56]. With the emergence of COVID-19, hospital-acquired PUs have garnered increased attention from physicians and nurses, especially for patients with prolonged hospital stays or those who underwent prone positioning [57]. Medical devices such as nasal cannulas, endotracheal tubes, surgical drains, and compression wraps and garments are also potential sources of mechanical pressure that should raise concerns in not only adult but also pediatric patients [58, 59]. The deformation of epidermal, dermal, and subcutaneous tissues between internal stiff structures and external surfaces, influenced by both extrinsic (e.g., mechanical pressure, friction, skin moisture) and intrinsic (e.g., nutrition, vascular comorbidities) factors, underlies the formation of PUs [60, 61]. Various pathophysiologic processes often involve local tissue ischemia and edema, enhanced capillary permeability, cellular aging, and modified skin microclimate [56]. Alterations in skin temperature and humidity may reduce the damage threshold for the skin and thus serve as an indirect determinant of risk for PU development [62]. The effect of ischemia becomes pronounced when the external pressure applied surpasses both the arterial capillary filling pressure and venous capillary outflow pressure [54, 63]. This leads to subsequent tissue hypoxia, necrosis, damage, and prolonged inflammation, which can be further worsened by reactive oxygen species generated through a reperfusion injury [54, 56]. The greatest pressure often occurs at the interface of muscle and bone, followed by the subcutaneous tissue and skin, resulting in progressively decreasing hypoxic and mechanical stress across these layers [54]. Likewise, re-epithelialization of the superficial tissue often comes before the healing of the deeper counterpart [64].

The current National Pressure Ulcer Advisory Panel staging system divides PUs into six categories. Four of these stages are based on the extent of skin loss, while the others include unstageable injuries obscured by slough or eschar and deep tissue injuries [65]. Proper decision making in PU management relies on prudent staging and a clinical evaluation. Principally, meticulous local wound care is sufficient for managing PUs of stages 1 and 2, while surgical intervention may be required in treating stages 3 and 4 PUs [61]. Pressure redistribution through frequent repositioning is crucial in reducing the burden of PUs regardless of their severity, while pressure offloading is essential to address the primary etiology of ulcer formation [66]. Establishing support surfaces with a constant low pressure and alternating pressure mattresses helps reduce the incidence and prevalence of PUs in debilitated patients [67]. Victims of stage 3 and 4 PUs may benefit from negative pressure wound therapy, as it provides better wound perfusion and growth of granulation tissue that improves the preoperative condition [66, 68]. Additionally, if surgery is indicated, the removal of bony prominences underlying the PU lesion can be considered to relieve pressure points [66].

4.4 Pressure Alopecia

Pressure alopecia (PA) is a rare postoperative complication occurring within 1 month after surgery [69]. During intraoperative anesthesia or an intensive care unit stay, a protracted period of immobilization in a recumbent position applies pressure to the patient's head, causing temporary occlusion of the blood vessels of the scalp, thereby inducing ischemia and hypoxia of the pressured site and resulting in vasculitis and cessation of hair follicle activity [70, 71]. Flesh-colored hairless patches emerge particularly on the vertex and occiput (Fig. 2d), and these lesions are often preceded by scalp tenderness, purpura, swelling, crusting, and ulceration [69, 70, 72]. Pressure alopecia has been reported in a myriad of operations, with cases of gynecologic surgery being first reported and those of cardiac surgery being most pronounced [70, 73]. A longer operation time implicates more accumulative pressure on the scalp and irreversibility of the impaired hair follicle activity that might lead to permanent PA [71]. Additionally, patients experiencing trauma [74], prone positioning because of COVID-19 [72], wearing orthodontic headgear

[75], and even watching television in a habitual lying position [76] have been reported to develop PA. This should prompt us to not think of postoperative alopecia as an equivalent to PA but a subcategory instead.

The novel concept of a hair shaft as a functional niche for hair follicle stem cells (HFSCs) in terms of mechanosensitization may provide additional insights into PA development [77, 78]. Terminal hair bulbs, which are located deeper than those of the vellus hair, are more susceptible to pressure-related injury and hypoxia, resulting in hair shaft loss [79]. Because the reduced number or diameter of hair shafts may decrease the size and functionality of the stem cell niche [77], victims of PA may consequently possess a defective HFSC niche. The shrinkage of the physical niche is followed by mechanical compression of HFSCs and alteration of the mechanical microenvironment, mediated by the PIEZO1-calcium-tumor necrosis factor-α (TNF-α) axis [77, 78]. Through continuous pressure, the intracellular calcium flux triggered by PIEZO1 contributes to long-term HFSC apoptosis and results in hair loss. This concept may suggest that we re-evaluate PA from a viewpoint beyond local ischemia and hypoxia.

Common trichoscopic findings of PA are dystrophic and fragmented hairs with haphazardly distributed yellow and black dots [74, 80]. Nevertheless, some vellus and thin hairs might remain visible, which has been explained by the lower metabolic rate of smaller follicles, along with the conical distribution of applied pressure on the curvilinear scalp that subjects deeper follicles to higher pressure, making the smaller and more superficial follicles less susceptible to hypoxia and ischemia [79]. In addition, circle hairs may prognosticate reversible PA [74], while comedone-like black dots unique to PA may differentiate it from alopecia areata [81].

Prevention of postoperative PA can be attained via frequent head repositioning, soft cushion usage, and a scalp massage during surgery to avoid continuous pressure, especially in patients in the Trendelenburg position or experiencing perioperative hypotension and hypoxemia [70, 71, 82]. A memory foam headrest and an alternating inflatable head pad are also helpful [83, 84].

5 Proliferative Diseases

5.1 Acanthoma fissuratum

Acanthoma fissuratum is a rare condition resulting from repetitive mechanical irritation, usually pressure and friction, at the contact site of the frame or nose pads of spectacles in glasses wearers. Initially described on ears as "granuloma fissuratum" [85], it is something of a misnomer as no granulomatous reactions were noted in this disorder.

The lesion commonly presents as a unilateral flesh-colored nodule, papule, or plaque with a central cleavage, resembling a coffee bean, with hyperkeratosis and acanthosis being major histopathological findings [86]. Retroauricular, superior auricular sulci, and the nasal bridge near the inner canthus prevail among possible sites of the lesion, while lesions at hip joints [87], genital organs [88], and the external auditory canal [89] have also been reported. Improper size and weight of glasses, comorbid cutaneous diseases, and craniofacial anatomical anomalies may pose a risk [86]. Spontaneous resolution of the lesion usually follows removal of the pressure origin, such as switching to a more fitted pair of glasses. Surgery and intralesional corticosteroids are also therapeutic options to treat persistent cases [86, 90].

5.2 Atypical Decubital Fibroplasia (Decubital Ischemic Fasciitis)

In 1992, Montgomery and colleagues reported 28 physically debilitated patients presenting with painless, ill-defined, focally myxoid masses on their shoulders, posterior chest walls, sacra, arms, thighs, and buttocks [91]. These unique lesions with a degenerative and regenerative pattern differed from PUs and were termed atypical decubital fibroplasia, as enlarged degenerated atypical fibroblasts with an abundant cytoplasm and hyperchromatic smudged nuclei were noted universally on a histopathological examination [91]. These fibroblasts are often admixed with zones of fibrinoid necrosis along with reactive fibrosis and focal myxoid changes surrounded by reactive neovascularization [92].

Atypical decubital fibroplasia has been recognized as a pseudosarcomatous fibroblastic proliferation mostly discovered on bony prominences or pressure points (e.g., sacrococcygeal, shoulder regions) of immobilized elderly patients [91–93]. Also known as decubital ischemic fasciitis, prolonged pressure-induced ischemia is regarded as the culprit of dermal and subcutaneous reactive changes that leads to benign atypical fibroblastic proliferation [91, 93], which occurs in perineal nodular induration as well. Border irregularity, fascial continuity, and pressure point location on a computed tomography image are representative radiographic features of the lesion [94]. It may recrudesce when pressure from a bedridden or wheelchair-bound status cannot be lessened or avoided [95], whereas surgical excision with debridement may mitigate the mechanical stress effectively and prevent recurrence [96].

5.3 Cutaneous Metaplastic Synovial Cyst

Repetitive mechanical trauma may result in the disruption of soft tissue, leading to the formation of a synovium-like membrane that surrounds the tender subcutaneous nodule known as a cutaneous metaplastic synovial cyst (CMSC) (Fig. 3a) [97]. Body parts with pressure from weight bearing, repeated manipulation, or previous local trauma, such as the first metatarsal head area [98], thumb [99], and buttock [100], respectively, are prone to chronic inflammation that may play a role in the pathogenesis of CMSC. Additionally, the fragility of the skin in patients with Ehlers–Danlos syndrome augments the effect of pressure on synovial metaplasia that makes them a specific population susceptible to CMSC [101]. Wound healing after skin microtrauma in these individuals is especially anomalous, resulting in synovial cell-like differentiation from mesenchymal stem cells [102]. Immunoreactivity against vimentin of the lining cells and the inward villous projections of the cystic wall corroborate the comparability between CMSCs and normal synovial cysts, which calls for a distinction of the two entities based on the presence or absence of antecedent local cutaneous trauma [99, 101]. Surgery is effective for CMSCs with minimal recurrence after cyst removal [103].

5.4 Acral Melanoma

Given its palmoplantar distribution, acral melanoma (AM) correlates more with mechanical stress than with skin phototype or sun exposure (Fig. 3b) [104]. Gravity and weight bearing can modify the microenvironment alongside melanocytes and melanoma cells. The protein of YAP and transcriptional coactivator with PDZ-binding motif act as transcriptional co-factors to not only facilitate tumorigenesis and cancer progression, but also to sense the extent of the extracellular matrix (ECM) stiffness and forces including mechanical compression and interstitial pressure [105, 106]. Weight-bearing subzones of the sole are inclined to AM development, particularly the heel and border of the foot, whereas the non-weight-bearing area of the arch is not as prevalent, with the exception of obese patients, who constitute nearly half of the arch-prone tumor victims [107]. Differences in patient demographics, tumor thickness, prognosis, and survival between lesions of weight-bearing and non-weight-bearing regions have been reported but without statistical significance [107–111]. On the contrary, acral melanocytic nevi (AMN) defy the pressure-prone distribution and are often located at non-weight-bearing sites [108, 112, 113]. Such a discrepancy might be the effect of chronic plantar pressure that modulates melanocyte migration and the associated subcellular damage, resulting in the inflammation-mediated carcinogenesis of AM [105, 114]. The spreading pattern along the long axis of AM that follows the skin creases potentially indicates migration and the pressure-dependent nature [109, 114].

While the distribution of AM and AMN may not be identical, the clinical resemblance of early AM to AMN suggests the need for further identification of distinguishing features.

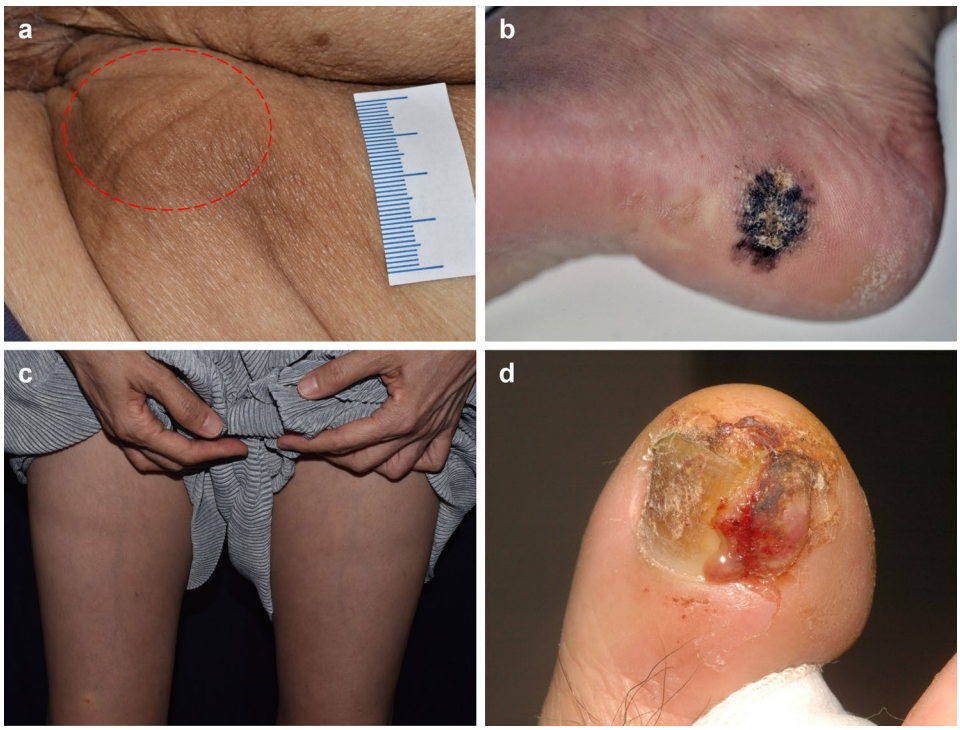

Fig. 3 Clinical images of pressure-related skin disorders: **a** cutaneous metaplastic synovial cyst (dotted circle), **b** acral melanoma, **c** lipoatrophia semicircularis, and **d** ingrown nail of the first toe

Dermoscopically, a parallel ridge pattern is indicative of AM, while fibrillar, parallel furrow, and lattice-like patterns are more common in AMN, with the fibrillar pattern showing a relatively stronger association with pressure by its lateral border distribution [104, 115, 116]. A thorough personal and occupational history of exposure to mechanical stress is also key to an early diagnosis of AM, as patients who are likely to experience chronic pressure such as farmers and those wearing tight shoes are susceptible to the malignancy [110].

Treatment of AM is centered on wide local excision, while slow Mohs micrographic surgery may be an alternative [117, 118]. Although adjuvant medical therapy demonstrates a poor response and provides uncertain benefits, anti-programmed cell death protein 1 immune checkpoint inhibitors and targeted therapy are still prospective options in treating primary and metastatic AM [117, 119]. Additionally, an excessive body mass index has been related to an increased risk of cutaneous melanoma [120], albeit the actual effect of body weight gain on pressure-related pathophysiology of AM requires further elucidation. Bariatric surgery-induced weight loss has proven advantageous to melanoma prevention [121], but likewise, whether pressure offloading in weight reduction makes a major contribution warrants clarification.

6 Atrophic Diseases

6.1 Lipoatrophia Semicircularis

Lipoatrophia semicircularis (LS), an unusual type of lipodystrophy presenting with band-like semicircular indentations of atrophic subcutaneous tissue, has mostly been detected on the anterolateral thighs of young to middle-aged women (Fig. 3c) [122, 123]. In particular, those who wear tight pants or work sedentarily in the office might be more at risk [124, 125]. Constant local pressure and the consequential repeated microtrauma with compromised microcirculation in the skin of thighs derived from such clothing, contact to the sharp edges of office desks, or inappropriate sitting postures are proposed to be etiological factors of the lesion [122, 123, 126–128]. Electromagnetic and electrostatic fields may also elicit perturbations to a cutaneous microenvironment that contribute to LS [124, 126, 127].

Histopathologically, some studies have assumed a chronological order of a LS pattern change, from normal in the early stage to the distinct inflammatory and involutional types 2 months beyond the disease onset [123, 129]. Examination often reveals a partial or complete loss of adipose tissue and diminutive fat lobules [123]. These findings suggest that LS may result from cellular atrophy and apoptosis induced by microtrauma. The lesion often regresses over time, from weeks to years variably, spontaneously, or after relief from mechanical pressure. Typical lesions (a bilateral symmetric horizontal band with length at least three times the width and limited to anterolateral thighs) have a more favorable clinical outcome compared with atypical cases in a recent follow-up study [124]. Amelioration of a work environment through redesigning an ergonomic profile and adjusting the electro-environment for office staff may be beneficial in preventing LS exacerbation and development [124, 125, 127].

6.2 Anetoderma of Prematurity

As a rare form of secondary anetoderma, anetoderma of prematurity exemplifies typical pressure-induced skin damage in neonates. The atrophic patches resulting from local hypoxia and inflammation through the compression of electrocardiographic electrodes or monitoring leads are exceptionally detected in neonatal intensive care unit patients [130]. Preceding cutaneous changes such as hyperpigmentation and telangiectasia were also reported at the sites of the medical compression devices [131]. Some investigators believed that growth retardation reflected by a lower birth weight is related to the decreased thickness of the neonatal epidermis, thereby predisposing these patients to anetoderma of prematurity [132]. Alterations in elastin production and matrix metalloproteinase activation may also be determinants of lesion development [133]. A history of extreme prematurity and neonatal intensive care unit hospitalization is especially valuable to early diagnose anetoderma of prematurity and prevent scarring and disfigurement of the pressured region through postural adjustments [134].

7 Immune-Mediated Diseases

7.1 Pressure-Induced/Aggravated Urticaria

Delayed-pressure urticaria (DPU) is a typical representative regarding the relationship between mechanical pressure and urticaria. Unlike other subtypes of physical urticaria, individuals with DPU often present with agonizing burning and pain, along with systemic manifestations such as flu-like symptoms and arthralgia, which can be attributed to the release of proinflammatory substances (e.g., TNF-α, interleukin-1, histamine, platelet factor 4) [135] by mast cells. The development of DPU involves tryptase-positive, chymase-positive mast cells, mechanosensitive neuropeptides, and proinflammatory cytokine release [136], following a non-immunologic mechanism [137]. Pressure applied by objects such as the shoulder straps of a backpack and a tight garment contributes to wheals or angioedema-like

swellings that often emerge 4–6 hours after pressure [138]. Uncommonly, vesiculobullous lesions may overlie the erythema in the bullous variant [139]. A large amount of dermal leukocyte infiltrate is seen because of a persistent pressure stimulus that contrasts DPU with other chronic inducible urticaria [137]. A pressure challenge test for skin provocation by using a dermographometer or weighted rods to observe the appearance of a delayed wheal serves as a sensitive diagnostic method [135, 138, 140–142]. While antihistamines often have limited efficacy in treating DPU, their status as first-line therapy remains owing to the absence of high-quality evidence for alternative treatments aside from pressure avoidance. Adding montelukast or theophylline to antihistamines may provide benefit, while sulfones, omalizumab, and anti-TNF-α therapies are potentially conducive to the treatment of refractory cases [143–145].

Furthermore, pressure also triggers or exacerbates lesions in patients with underlying urticarial diseases. Symptomatic dermographism, a subtype of chronic inducible urticaria, can be elicited through light stroking pressure [146, 147]. While DPU often co-occurs with chronic spontaneous urticaria [137, 147], physical stimuli in pressure-prone regions can in turn exacerbate chronic spontaneous urticaria lesions [138, 148]. Thus, it is sometimes hard to differentiate between lesions of DPU and comorbid chronic spontaneous urticaria [138]. The emergence of sock-line linear bands was also reported in infants with symptomatic dermographism, implying the role of upregulated mast cell activity triggered by pressure from the elastic sock top [149].

8 Miscellaneous

The development of certain disorders, such as an ingrown nail, results from a combination of mechanical factors and several pathological alterations. There are also pressure-related disease entities associated with specific activities or device equipment, such as playing sports, musical instrument performance, and wearing a prosthesis. These disease categories consist of a myriad of lesions, which are often collectively discussed in the literature. These cutaneous lesions may manifest diversely and result from various pathological changes, making an accurate classification of each lesion into the categories mentioned above trivial and challenging. In this section, we explore the impact of pressure on the development of these skin disorders.

8.1 Ingrown Nail (Onychocryptosis)

Ingrown nails commonly affect children and young adults that lead to pain, impaired ambulation, and heightened infection risk (Fig. 3d). During weight bearing, the lateral edges of the toenails can exert excessive pressure on the skin adjacent to the nail plate. This is particularly likely when the toenails are improperly trimmed or when one is wearing ill-fitting footwear, which can lead to skin penetration, subsequent inflammation, and pressure necrosis [150]. The internal compression resulting from a mechanical imbalance between the distal phalanges of the first and second toes as well as the external pressure to the lateral nail fold of the first toe have been identified as significant mechanical factors in the development of an ingrown nail [151, 152]. Thus, some researchers have studied the role of foot mechanics, considering a pathological hallux interphalangeal angle as a potential risk predictor [151]. Despite this, surgical correction of the anatomy should not be prioritized if no abnormalities exist, as a definitive link between forefoot alignment and an ingrown nail has yet to be established [152]. Conservative treatments such as gutter splint, cotton, and dental floss provide a physical separation between nail edges and lateral nail folds, as well as pain relief [153]. In contrast, surgeries that aim at nail plate narrowing or debulking excessive periungual tissues may result in fewer recurrences, such as chemical matricectomy and the Vandenbos procedure, respectively [154]. Additionally, the utilization of a toenail paronychium flap has been proposed [154]. This technique aims to redistribute periungual tissues and create shallower nail folds, which could potentially lessen the effect of pressure on the nail apparatus more persistently. Nevertheless, recurrences may occur with inadequate removal of the nail matrix during procedures, and with the regrowth of keratinized tissue, manifesting as either a nail or a spicule [150].

8.2 Sports-Related Skin Disorders

Athletes may be exposed to repetitive pressure from personal gear, motions, or surroundings that cause mechanical dermatoses [126]. For instance, swimmer's nose deformity is a common pressure-related complication resulting from tight goggles worn by swimmers that anatomically and mechanistically resembles acanthoma fissuratum [155]. In figure skaters, an indurated, inflammatory, and erythematous nodule near lateral malleoli (skater's/athlete's nodule, or prayer nodule, a histological equivalent on the forehead or foot of a religious worshipper [156]) or hyperkeratotic epidermal thickenings over the Achilles tendon (skater's pad) resulting from a combination of chronic pressure, irritation, and friction may be sought on their feet [157]. Tendinopathies of ankle extensors (skate/lace bite) also develop and discomfort skaters with pain on dorsiflexion when they wear skates with rigid tongues [157, 158].

Sports performed with a sitting posture may induce lesions such as biker's panniculitis on the inner thighs [126], rower's rump, and saddle sores of cyclists on the buttock [159] that result from pressure and friction applied by the seat. Perineal nodular induration is an unusual mesenchymal

pseudotumor mimicking an "accessory testicle" that occurs mostly in avid male cyclists, while female cases and participants of other saddle sports such as equestrians have also been reported [160, 161]. The oft tissue of the perineum posterior to the scrotum or adjacent to the labia majora is involved and withstands repetitive mechanical compression and vibration against the ischial tuberosities during sports, giving rise to the benign proliferation of fibroblastic components [162]. The mechanism of PNI corresponds to that of atypical decubital fibroplasia, and these two entities are also histopathologically similar [162, 163]. Unloading the perineum by transferring pressure to ischial tuberosities through modifying the saddle into a flatter harder seat may bring symptomatic relief [160], while surgical excision with a flap reconstruction can be an effective solution to treating an enlarging PNI [164]. Ultrasonography can provide real-time differential diagnoses of these disorders, especially those with a soft-tissue origin, such as identifying biker's panniculitis via visualizing thick hypoechogenic septations within the hyperechoic subcutaneous fatty lobules [126], and demonstrating cases of PNI through detecting a hypoechoic nodule with heterogeneous echogenicities and a void of Doppler signals [160].

A microhemorrhage of the nail bed vasculature induced by repetitive contact and pressure is a causative factor for tennis toe, jogger's toe, and golfer's nail [157, 159, 165–167]. Acceleration-deceleration movements when sprinting with ill-fitting shoes and a tight grip of golf clubs are why these athletes bear local pressure on their extremities. Notably, as these lesions usually present with melanonychia, clinicians should be aware of the differential diagnosis of subungual melanoma [159, 165, 167]. Talon noir is a similar hemorrhagic entity that often occurs in young athletes who experience a combination of pressure and shearing stress at the calcaneal fat pad during sports [159]. Participants of football, tennis, gymnastics, rock climbing [168, 169], baseball [170], skating, and ice hockey [157] have been reported to be affected. Intracorneal bleeding of posterolateral heel skin underlies these hyperpigmented asymptomatic macules [157]. Talon noir can be confirmed via a scratch test, in which scalpels are used to scrape away a partial thickness of the affected skin without bleeding [157, 168]. No intervention is needed, for the lesion often subsides spontaneously within several weeks [157, 170].

8.3 Musical Instrument-Related Skin Disorders

Industrious musicians are prone to a variety of skin diseases owing to the frequent use of musical instruments. Callosities, contact dermatitis, fiddler's neck, and onychodystrophy are some common disorders, especially in string and plucking instrument players [171, 172]. Pressing the strings repeatedly can form callosities on the fretting hands of guitarists, while holding the violin up to the neck for long practice hours can develop the fiddler's neck. While friction and shearing stress play important roles, long-term local pressure is also thought to be one of the contributing factors to fiddler's neck in the submandibular region, which often presents as hyperpigmented, lichenified, and erythematous plaques [173, 174]. Submandibular nodular indurations of a benign fibroblastic proliferation that are pathologically homologous to PNI of cyclists can also be seen after the exposure to repeated mechanical trauma caused by the string instrument [175]. For fiddler's neck, avoidance of inappropriate instrument handling and the application of a chin rest can lessen physical pressure and thus deal with the disease [173]. Surgery is often of limited effect and therefore not recommended [173]. For brass and woodwind instrumentalists, lip callosities and cheilitis are also adverse conditions ascribable to mechanical forces of local pressure, shearing stress, and occlusion during long-time practice [172].

8.4 Prosthesis-Related Skin Disorders

Limb prostheses are essential to amputees for functional and aesthetic purposes. While these devices offer close contact to the skin of a stump, which is poorly adapted to mechanical forces, dermatological disorders may ensue. According to a questionnaire-based study, skin problems, including those caused by mechanical pressure, were found to be sources of distress for one third of amputees [176]. Pressure on the prosthesis-stump interface results in a multitude of localized cutaneous alterations, especially on lower limb prostheses in that the extremities bear weight [177]. Maintaining sufficient pressure on the stump is important for securing it within the prosthesis, but this may cause pain, edema, soft-tissue necrosis, and local ischemia of the skin. Blisters, lichenification, acroangiodermatitis, contact dermatitis, epidermal hyperplastic changes (e.g., hyperkeratotic papules, epidermoid cysts, mechanical acne), and hidradenitis suppurativa-associated follicular diseases are all possible cutaneous manifestations of the interface elicited in part by mechanical factors [177–179].

Among various conditions, epidermal hyperplasia, contact dermatitis, and ulceration are thought to be specifically associated with mechanical forces including pressure [179]. Venolymphatic stasis followed by stump edema can arise from pressure, leading to verrucous hyperplasia, while pressure, prosthesis suitability, and local sensitization of the stump determine the development of hyperkeratotic papules [178]. Pressure and the humidity of the stump also synergistically heighten the risk of both allergic and irritant contact dermatitis [178]. Immunocompromised cutaneous district explains how skin damage, through the pathogeneses of chronic lymphatic stasis and neural injury, can predispose certain areas of the skin to dysregulated immune reactions

and antigen clearance, resulting in chronic inflammation, hyperplasia, fibrosis, and changes in vascular hemodynamics, which in turn exacerbate lymphatic stasis [180]. Because the stump skin is a unique trauma-induced area acting as an immunocompromised cutaneous district that bears significant mechanical forces including pressure and friction within the humid environment of the socket, its immunologic integrity can be especially fragile [181]. An amputation stump suffers from both the hindrance of normal trafficking of immune cells due to lymphatic stasis and the generation of immune-related peptides owing to damaged sensory nerves [182]. These factors are associated with the inclination to tissue breakdown, a vulnerability that is exacerbated if the patient has a past medical history of conditions such as diabetes mellitus, vascular diseases, or cancer [181]. In addition, chronic inflammatory and edematous changes are inevitable pathological processes provided pressure is constantly exerted on the stump. Not only does inflammation predispose the stump to ulceration or further infection, but it may result in a malignant transformation (Marjolin ulcer) [178]. The concept of stump skin as an immunocompromised cutaneous district is thus crucial for understanding its exceptional vulnerability, as the skin of these residual limb ends undergoes irreversible dysregulated immune responses, compromised circulation, and subsequent pathological alterations [183]. Therefore, stump skin is prone to various cutaneous disorders of mechanical pathomechanisms.

Certain designs of prosthetic sockets provide manual adjustments to tightness to best fit the stump, albeit some unadjusted areas may still receive disproportionate pressure. For instance, the full-contact type socket contains the stump either directly or with cushioning that lessens the average pressure per unit area. Nevertheless, these pressure-relieving advantages are gained at the cost of increased friction and poorer heat dissipation [177].

9 Discussion

The ubiquity of mechanical pressure in our daily life makes it an unavoidable insult and might imperil skin health to some extent. The effect of pressure on skin diseases is best exemplified in the use of personal devices, habitual or sociocultural behaviors, leisure activities, occupations, weight bearing, and even unintentional or unaware contact with objects and postures. In the future, new etiologies of pressure-related skin disorders may be added to the repertoire, such as nail dystrophies resulting from phubbing [184], gamer's nodules in avid game players simulating athlete's nodules [185], and personal protective equipment-induced dermatoses in the COVID-19 epoch [5, 186].

The mechanical properties of individual skin cells and their collective responses to external forces account for the development of cutaneous pathologies [187]. Although discussion on the detailed mechanotransduction pathways is beyond the scope of this review, understanding the potential mechanisms involved in mechanical pressure from a cellular perspective may provide us with insights into disease management (Table 2). Mechanosensitivity of cellular receptors and sensory nerves facilitates cutaneous cell–cell and cell–ECM interactions, whereby extrinsic forces including pressure can disrupt skin integrity and elicit local pathological alterations [114, 198]. Fibroblasts and keratinocytes, which play a major role in skin cell mechanotransduction, may be more implicated in the vast majority of pressure-related skin diseases, while mast cells and melanocytes also exhibit pathological alterations elicited by pressure through molecular and cellular interplay, inducing the development of urticaria and melanoma, respectively.

In the epidermis, both a prolonged duration and an increased magnitude of pressure can accelerate and induce an earlier increase in transepidermal water loss of the stratum corneum [199]. In turn, a dehydrated stratum corneum may be more vulnerable to the mechanical injury from pressure [200]. Moreover, mechanical pressure promotes entry into the mitotic cycle for epidermal cells; simultaneously, it can cause a temporary cell-cycle arrest at the G1/S transition as well as the upregulation of the proto-oncogene c-fos, which collectively manifest as tissue proliferation [201]. The release of proinflammatory cytokines and chemokines, including interleukin-1, interleukin-8, and TNF-α, is also detected upon sustained mechanical loading of the epidermis, preceding significant damage to the epidermal tissue [202]. In the deeper layers of the skin, mechanical pressure may upregulate matrix metalloproteinases and downregulate the transforming growth factor β1 receptor and SMAD family members 2 and 3, contributing to both impaired production and accelerated degradation of the dermal ECM [203, 204]. Tissue viability may be compromised by pressure loading, either directly through cellular deformation or indirectly through distortion of the vascular and lymphatic circulation from the supplying origin [205]. Seddone and colleagues observed that the rapid response of compression-induced hyperemia in cutaneous blood vessels was akin to that seen in skeletal muscles, indicating the vascular mechano-reactivity of cutaneous circulation [206]. While the functional implications of this are currently unclear, it might shed light on the underlying mechanisms of certain dermatological disorders, such as ischemia-reperfusion injury. Furthermore, cases where lipomatous masses were associated with chronic mechanical pressure demonstrated increased subcutaneous adipose tissue thickness at the lesion site, suggesting that inflammation-induced adipocytic proliferation may be elicited by pressure and underlie the pathomechanism of these tumors [207].

Table 2 Potential processes involved in the cellular mechanisms of mechanical pressure effects on skin in relevant dermatological disorders

Targeted cell	Potential processes elicited by pressure	References	Examples of relevant disease entities
Fibroblast	Myofibroblast differentiation Premature cellular senescence Compromised tensional homeostasis and tissue turnover	[188–191]	Atypical decubital fibroplasia, perineal nodular induration, nodular cervical induration, pressure ulcer
Keratinocyte	Dysregulated cellular differentiation, proliferation, regeneration, homeostasis, and epidermal integrity Stress keratin response, oxidative signaling, and inflammation (palmoplantar)	[192–195]	Acanthoma fissuratum, clavus, callosity, palmoplantar keratoderma, senile gluteal dermatosis
Mast cell	Mediation of Th2 immune response and activation of sensory neurons Upregulated mast cell activity and degranulation	[136, 149]	Delayed-pressure urticaria, pressure-aggravated chronic urticaria
Melanocyte	Dysregulated transcellular crosstalk between melanocytes, keratinocytes, and other cells Modulation of melanoblast differentiation Subcellular structural instability and damage Tumorigenesis and tumor aggression influenced by ECM stiffness	[105, 106, 196, 197]	Acral melanoma

ECM extracellular matrix, *Th2* T-helper 2

However, the complex interplay among cells, the nervous system, and the ECM as well as different layers of skin tissue complicates the attribution of individual pressure-related skin disorders to a single, discrete cellular, or tissue-level mechanism or pathological change [198]. A combination of alterations in various cellular structures and functions might more aptly explain the majority of these conditions.

Moreover, there are certain disease entities where mechanical pressure may instead serve as a co-factor; that is, pressure may be a sufficient but not a necessary condition in these instances. Granuloma annulare (GA) is a granulomatous inflammatory reaction that usually manifests as localized or generalized disc-shaped erythematous papules and plaques on the dorsal aspects of distal extremities [208]. Although the etiology of GA often remains undefined and has not been frequently correlated to physical factors, mechanical pressure may be an uncommon predisposing contributor. A case of GA emerging on the contact site of the pleated ridge defect of the elbow orthosis had been reported [209]. In this case, pressure provoked local inflammation and irritation, leading to the development of GA without the influence of other systemic factors. A study found that, for a majority of pediatric patients who had either just started wearing shoes or had recently changed their pair, the dorsum of the foot and ankle were the preferred sites for the pseudorheumatoid nodule, a subcutaneous variant of GA [210]. Repetitive traumatic forces, such as pressure applied by the footwear, are contributing factors to lesion development in not only children but also adult patients [210, 211]. T-cell-mediated delayed hypersensitivity and a subsequent panniculitis-type inflammation are involved in the pathological alterations following the mechanical trigger [212]. Another example is palmar-plantar erythrodysesthesia, also known as hand-foot syndrome. Aside from the palmoplantar regions, areas of the body susceptible to pressure and microtrauma, such as the intertriginous and sacral regions, can potentially develop palmar-plantar erythrodysesthesia [213, 214]. It has been hypothesized that pressure and friction may damage microcapillaries, resulting in drug extravasation (specifically liposomal doxorubicin) and a subsequent local inflammatory reaction, which potentially contribute to the pathogenesis of palmar-plantar erythrodysesthesia [213–215]. In addition, there is a case series suggesting a link between the development of lipomas and chronic pressure, though lipomas are not frequently associated with pressure in common medical discourse [207]. Pressure thus may act not only as a culprit but also as an accomplice in the evolution of various cutaneous lesions.

Notably, some disease entities manifest from similar or even identical pressure-related pathophysiology but are present at different body sites. For example, PNI appears at the perineum of cyclists, nodular cervical induration presents at the submandibular area of violinists, and atypical decubital fibroplasia can be seen in the sacrococcygeal and shoulder regions. All these conditions stem from the pathological proliferation of fibroblasts in response to different pressure-prone scenarios. The functional and immunologic heterogeneity of fibroblasts across different body areas may influence skin autoimmunity, which could underlie the site-specific development of these related lesions [216]. Further, the anatomical specificity of regional fibroblasts may be associated with a distinct metabolism and a varied interaction with epithelial components that influence skin fibrosis and other pathological processes [217], while this

regional heterogeneity also potentially affects epidermal differentiation of skin [218]. These may explain the similarities and differences in the lesion distribution of some pressure-related skin disorders. In contrast, at the same anatomical site, structures from different layers can be affected by pressure, leading to distinct cutaneous lesions. For example, at the dorsal ankles, a skate bite affects the tendon, while a pseudorheumatoid nodule impacts the subcutis.

Additionally, aging may contribute to the pathogenesis of some pressure-related skin disorders, such as chondrodermatitis nodularis, senile gluteal dermatosis, and atypical decubital fibroplasia. The structural fragility of senescent skin characterized by thinning of all skin layers and a defective ECM due to decreased transforming growth factor-β expression makes mechanical pressure particularly injurious to elderly individuals [204]. Similarly, neonates have under-developed skin and may be more vulnerable to external pressure. The age-related difference in fibroblastic functionality may determine the dermal ECM constitution and the proinflammatory cytokine level, which impacts skin homeostasis and regeneration, thereby predisposing these individuals to mechanical skin damage [218].

Nevertheless, the impact of pressure is often inseparable from lateral compression, friction, and occlusion, as they frequently parallel one another [2]. Meticulous history taking and dermatological examination remain crucial to unraveling the role of pressure in skin disorders.

10 Conclusions

The skin perceives and reacts to various external stimuli, including mechanical pressure, but the effect of pressure on the skin might go unperceived by physicians. The prolongation or inappropriateness of pressure bearing leads to dysregulated responses of cutaneous structures, thereby resulting in local inflammation, ischemia, necrosis, proliferation, hyperkeratosis, impaired regeneration, atrophy, or other injurious reactions that alter the skin microenvironment. The spectrum of pressure-related skin disorders may involve the epidermis (keratinocytes and melanocytes), hair follicles, eccrine glands, nail apparatuses, dermis (fibroblasts, mast cells, and vasculature), subcutis, and fascia. The use of personal devices, activities, occupations, weight bearing, and even unintentional object contact and postures can all be contributory scenarios of these disorders. Clarifying the clinical context of each patient and recognizing how pressure at the cellular and tissue levels leads to skin lesions can enhance our comprehension of pressure-related skin disorders to attain better management of this condition.

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