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DavidPolly T
Assistant Professor, Department of Dermatology, Venereology and Leprosy, Palakkad Institute of Medical Sciences, Opp Deer Park, Walayar, Palakkad, Kerala, India

Corresponding Author:
DavidPolly T
Assistant Professor, Department of Dermatology, Venereology and Leprosy, Palakkad Institute of Medical Sciences, Opp Deer Park, Walayar, Palakkad, Kerala, India

Skin and obesity: A Narrative review

DavidPolly T

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Abstract
Obesity is one of the most significant health challenges of our time, affecting every part of the body-including the skin. The skin problems associated with obesity can increase overall morbidity, which forms the basis of this review article. While many cutaneous manifestations are common among all obese individuals, obesity also serves as an important risk factor for inflammatory dermatoses such as psoriasis, atopic eczema, and certain rare disorders.

Keywords: Obesity, acanthosis nigricans, body mass index (BMI), abdominal striae

Introduction
Obesity is a medical condition characterized by an abnormal or excessive accumulation of adipose tissue, resulting in excess body weight [1, 2]. It develops from a chronic imbalance between energy expenditure and food intake. Key metabolic factors that predict weight gain include low energy expenditure during sedentary activities, a high respiratory quotient, and reduced levels of spontaneous physical activity[3].Although obesity is a significant health issue, it has received relatively little attention. However, it induces a range of changes affecting the skin and its appendages. These include alterations in skin barrier function, modifications in the activity of sweat and sebaceous glands (and sebum production), changes in collagen structure and function, as well as impacts on the lymphatic system, wound healing, microcirculation, macrocirculation, and subcutaneous fat distribution [3, 4].

Obesity affects every part of the body and skin is no exception
Some of the common cutaneous manifestation of obesity in Indian scenario are as follows

1. Acanthosis nigricans
2. Acrochordons
3. DPN (Dermatosis papulose nigra)
4. Abdominal striae
5. Keratosis pilaris
6. Chronic venous insufficiency
7. Gynaecomastia

Infectious
- Intertrigo
- Candida
- Dermatophytes
- Folliculitis
- Necrotizing cellulitis/fasciitis

Inflammatory
- Hidradenitis suppurativa
- Psoriasis

Physiological skin changes
Obesity is associated with numerous physiological skin changes. Loeffler, Aramaki, and Effendy[5] in their study found increased transepidermal water loss in obese patient because of altered epidermal barrier suggesting changes in skin barrier function. In obese condition

there can be increase in harmones like insulin, androgens, growth harmone and insulin like growth factors which can activate sebaceous gland and it can effect acne severity [6, 7]. Obese patients can have increased sweating because of larger subcutaneous fat which can further lead to increased frictional and moistural components [8]. Obesity can also damage the lymphatic vasculature which can result in secondary lymphedema [9]. Other physiological changes obesity can cause are microvascular dysfunction which can lead to microangiopathy and hypertension [10].

Acanthosis nigricans

Acanthosis nigricans is the most common cutaneous manifestation of obesity in both adults and children [1, 3] [Figure 1]. This hyperplastic skin lesion is closely associated with insulin resistance and hyperinsulinemia [11]. It appears as skin that is thickened, coarse, and darker than the surrounding areas. Common sites of involvement include the neck, axilla, groins, perineum, elbows, knees, knuckles, the inner surfaces of the thighs, and the abdominal skin folds [11,12,13].

Acanthosis nigricans is classified into four types (Curth *et al*.) [12, 14].

a) Malignant AN (type 1) - a cutaneous paraneoplastic syndrome associated with adenocarcinoma
b) True benign AN (type 2) - familial present at birth or beginning in childhood
c) Pseudo-AN (type 3) - associated with several syndromes in which obesity and endocrinopathies especially insulin resistant state coexist
d) Drug induced AN (type 4)

Hud *et al*. observed that acanthosis nigricans in obese patients is associated with elevated plasma insulin levels [15]. Similarly, Anuradha *et al*. found that acanthosis nigricans occurs more frequently in individuals with metabolic syndrome [13]. Furthermore, Varthakavi *et al*. demonstrated that patients with acanthosis nigricans exhibit a decreased glucose disposal rate, thereby establishing an association between acanthosis nigricans and insulin resistance [12].

Elevated circulating insulin levels lead to a reduction in the number of functional insulin receptors [16, 17]. These receptors are crucial for regulating glucose uptake, cell growth, DNA synthesis, and the metabolism of proteins and fats through tyrosine kinase activity. Both keratinocytes and fibroblasts express insulin-like growth factor (IGF) receptors, which can bind insulin and promote cellular growth [12, 18]. A decrease in functional insulin receptors results in a relative increase in insulin binding to IGF receptors, which contributes to the development of acanthosis nigricans [16]. Histopathologically, acanthosis nigricans is characterized by papillomatosis and hyperkeratosis of the skin [19].

Acrochordon

Acrochordons, also known as skin tags or fibroepithelial stromal polyps, are common benign tumors observed in middle-aged men and women [20]. Clinically, these lesions appear as soft, pedunculated, brown papules most frequently found on the neck, axilla, and groin [3]. They can also occur at less common sites, including the eyelids, penis, vulva, cervix, and other extra-genital areas [20, 21].

Acrochordons are associated with several systemic conditions, such as obesity, diabetes, hyperlipidemia, colonic polyps (as seen in Gardner syndrome), insulin resistance, and acromegaly. Three types of acrochordons have been described:

1. **Small, furrowed papules:** Measuring approximately one to two millimeters in width and height, commonly located on the neck and axilla.
2. **Filiform lesions:** Single or multiple lesions about two millimeters in width and five millimeters in length, occurring on various parts of the body.
3. **Large, pedunculated tumors:** Also described as nevoid, bag-like soft fibromas, typically found on the lower part of the trunk [21].

Histopathologically, acrochordons consist of a fibrovascular core and fat cells, covered by either a normal epidermis or by an acanthotic, flattened, frond-like epithelium [20, 21]. Shah *et al*. demonstrated a significant association between acrochordons and diabetes mellitus, suggesting that these lesions may serve as a cutaneous marker of metabolic syndrome [22].

Keratosis pilaris

Keratosis pilaris typically presents as small, spiny, perifollicular papules, most commonly affecting the extremities [3]. This condition is associated with a high BMI, leg skin dryness, and atopic disorders [23]. In obese individuals, elevated insulin levels and insulin resistance are believed to play key roles in its pathogenesis [24].

Striae distensae

Striae are a cutaneous condition characterized by linear atrophic plaques arranged perpendicular to the lines of greatest tension, occurring in areas where stretching has caused dermal damage [3, 25] [Figure 2]. They typically begin as red to purple, raised, wavy lesions (striae rubra) that eventually fade into white, atrophic patches with a wrinkled appearance (striae alba) [8, 26]. These lesions are common in physiological conditions such as adolescent growth spurts and pregnancy, but they are also seen in pathological states like obesity, Cushing's syndrome, Marfan's syndrome, and with long-term systemic or topical corticosteroid use.[27] They most frequently appear on the breasts, arms, thighs, abdomen, lumbosacral region, buttocks, face, and flexural areas, particularly in cases induced by Cushing syndrome or steroid therapy [25, 26].

The exact pathogenesis of striae has yet to be fully elucidated. Proposed mechanisms include dermal scarring resulting from the rupture of collagen fibers [8]. Sheu *et al*. suggested that sequential changes involving elastolysis coupled with mast cell degeneration occur in the early stages of striae distensae [28]. Additionally, striae tend to develop in skin with a high proportion of rigid, cross-linked collagen [25]. Histopathologically, early striae demonstrate dermal edema and perivascular lymphocytic cuffing, while later stages are marked by epidermal atrophy and the loss of rete ridges [27, 29].

Skin Diseases exacerbated by Obesity

Skin diseases aggravated by obesity in adults are lymphoedema, skin infections, chronic venous insufficiency, plantar hyperkeratosis, cellulitis, hidradenitis suppurativa, psoriasis, insulin resistance syndrome and tophaceous gout [3]. Most commonly associated skin disease in children are intertrigo, psoriasis and atopic dermatitis [1].

Obesity and lymphedema

Lymphedema results from either congenital malformation or injury to the lymphatic system (or its vasculature), leading to progressive swelling that commonly affects the limbs-and sometimes the genital area as well [30]. The lower extremities are more frequently involved, potentially due to a greater accumulation of adipose tissue there compared to the upper extremities, along with the fact that the legs' dependent position impedes lymphatic flow against gravity [9].

Obesity and lymphedema have a bidirectional relationship. For example, research by Aschen *et al*. demonstrated that impaired lymphatic flow can induce inflammation and significantly upregulate genes involved in adipocyte differentiation-such as peroxisome proliferator-activated receptor gamma (PPAR-Ɣ) and CCAAT-enhancer-binding protein alpha (CEBP-α)-as well as elevate adipokine expression [9, 31].

Recent studies also suggest that dietary factors can influence lymphatic function. In one experiment by Kim *et al*. [32]., mice with a defect in the apolipoprotein E gene exhibited abnormally high circulating cholesterol levels and developed notable lymphatic abnormalities, including reduced interstitial fluid transport capacity, defective lymphatic valves, and impaired trafficking of immune cells through tissue.

If left unchecked, chronic lymphedema may progress to conditions such as elephantiasis nostra verrucosa and can eventually lead to serious complications like angiosarcomas [3].

Obesity and chronic venous insufficiency

Obesity is a significant risk factor for developing both venous thromboembolism and chronic venous insufficiency [3, 33]. An increased body weight can trigger alterations in the coagulation system-diminishing fibrinolytic activity while elevating plasma concentrations of clotting factors-which, in turn, may contribute to endothelial dysfunction and the formation of venous thromboses [33].

Central obesity often leads to increased intra-abdominal pressure, which opposes the venous return from the lower extremities. This pressure imbalance can result in the failure of venous valves and subsequent venous dilation, manifesting as varicosities [8]. The elevated hydrostatic pressure may also force components of the intravascular fluid to leak into the surrounding tissues. When red blood cells escape from the veins, their hemoglobin deposits within the dermis, thereby inciting an inflammatory response characterized by erythema and warmth. Clinically, this process is observed as pitting edema, brown macular hyperpigmentation, and scaling [8].

Stasis dermatitis typically arises from the irritation of superficial nerve fibers, a consequence of the increased local pressure and the accumulation of metabolic by-products that elevate tissue pH [34]. Over time, chronic venous insufficiency may further progress to complications such as lipodermatosclerosis and venous ulcerations [35]. These venous ulcers are most commonly found along the medial aspect of the lower extremity-between the mid-calf and the medial malleolus, following the course of the greater saphenous vein-with overweight individuals exhibiting a higher risk for ulceration compared to those with a normal body mass and similar levels of venous reflux [36, 37].

Effect of Obesity on the folliculosebaceous unit

Mirmirani *et al*. [38] a study on skin disorders associated with obesity hypothesised that early onset of obesity can be associated with alterations in folliculosebaceous unit which can manifest as hirsutism and alopecia.

Yang *et al*. [39] in his study found that higher BMI is significantly associated with greater severity of hair loss in men with male pattern and androgenetic alopecia especially in those with early onset androgenetic alopecia.

Obesity and skin infections

Infections are among the most common complications associated with obesity. Several risk factors contribute to this increased susceptibility, including impaired barrier function, a humid and macerated microenvironment, and limitations in mobility and hygiene practices [1]. Intertrigo is one of the most frequent manifestations; it appears as macerated, erythematous plaques in skin folds-such as the inframammary, genitocrural, axillary, and abdominal regions-resulting from increased friction and moisture [Figure 3]. These issues are further compounded by large skin folds that trap heat and sweat profusely, a consequence of the thick layers of subcutaneous fat. [1, 8]. Superinfection with yeast mainly Candida albicans is a common complication of intertrigo [1]. Other fungal infections associated with obesity are tinae paedis, pityriasis versicolor [Figure 4] and toe nail onychomycosis.

Bacterial Infections in Obesity

Bacterial infections in obese individuals can range from mild conditions such as folliculitis and furunculosis to more severe infections like erysipelas and necrotizing fasciitis.

Erysipelas

Erysipelas is an infectious disease affecting the dermis and subcutaneous tissue, most commonly caused by streptococcal bacteria. A study by Krasagakis suggests that obesity is an independent risk factor for local complications of erysipelas. Due to the potential severity of the disease and the risk of empirical treatment failure, obese patients require careful evaluation and monitoring [40].

Necrotizing Fasciitis

Necrotizing fasciitis is a life-threatening soft tissue infection characterized by widespread necrosis of the fascia and subcutaneous tissue [41]. Obesity is recognized as a significant risk factor for necrotizing fasciitis, with infections often occurring in the perineal and trunk regions [42]. Clinical features include extensive tissue destruction, systemic toxicity, and high mortality rates. Pathophysiological mechanisms involve thrombosis of blood vessels, bacterial spread along fascial planes, and limited infiltration of acute inflammatory cells [3].

Obesity and Psoriasis

Psoriasis is a common, debilitating chronic skin disorder that has recently been linked to increased adiposity and obesity [43] [Figure 5]. Adipose tissue secretes several inflammatory mediators—including adiponectin, leptin, plasminogen activator inhibitor-1 (PAI-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α)—which may contribute to its pathogenesis [44, 45].

The inverse variant of psoriasis, which is associated with obesity, can sometimes be indistinguishable from intertrigo

in overweight individuals [46]. Moreover, psoriatic patients over the age of 18 with a history of obesity are at a higher risk of developing psoriatic arthritis compared to those of normal weight [45, 47]. Psoriatic arthritis is characterized by involvement of the distal interphalangeal joints, asymmetric arthritis, dactylitis, enthesitis, spinal involvement, and a frequent association with HLA-B27 [48]. TNF-α plays a central role by enhancing the pro-inflammatory activity in the synovial tissue, thereby contributing to oxidative stress and the recruitment of leukocytes into atherosclerotic plaques [49]. It also increases serum levels of atherogenic lipoproteins and promotes atherosclerotic lesion development by upregulating adhesion molecules on endothelial cells, recruiting and activating inflammatory cells, and initiating an inflammatory cascade within the arterial wall [50, 51, 52]. Consequently, psoriatic arthritis is regarded as a multisystem disease that can involve the coronary arteries and heart [48].

A study conducted by Zhang *et al*. [53] in a Chinese Han population found that psoriatic patients have a higher prevalence of overweight and obesity compared with non-psoriatic individuals. Similarly, Vahid *et al*. [54] observed that the prevalence of obesity and severe obesity was higher among patients with psoriasis than in the control group. Diet also plays an important role; an Italian study reported that psoriatic patients consumed a higher proportion of high-fat and saturated foods compared to the normal group, suggesting that dietary habits may contribute to the increased prevalence of metabolic abnormalities in psoriasis [55].

Obesity further complicates the treatment of psoriasis. In a prospective study by Di Minno *et al*. [56]. involving 135 obese psoriatic arthritis patients and 135 normal-weight controls, the presence of abdominal obesity was associated with an increased risk of failing to achieve minimal disease activity (MDA) during treatment with TNF-α blockers. Obesity is linked to a decreased response to both systemic and biologic therapies. This effect appears to be related to pharmacokinetic factors, being more pronounced for drugs administered in fixed doses than for those whose doses are adjusted according to patient weight. For instance, biological drugs such as etanercept and adalimumab—which are typically given in fixed doses—may be less effective in obese patients [57, 58]. Weight reduction, on the other hand, can reduce drug toxicity and enhance both the effectiveness and tolerance of these therapies, particularly in the context of fixed-dose regimens [58, 59].

Obesity, Insulin resistance and comorbidities

Obesity induces insulin resistance and involves molecules that leads individual to inflammatory state and metabolic complications [60]. It is thought to be because of resistance to insulin action in peripheral tissues. An important consequence of insulin resistance is an increase in free fatty acids, which further exacerbates insulin resistance. Increased insulin resistance in hepatic, muscular, and adipose tissues is associated with overproduction of proinflammatory cytokines such as interleukin-6 and tumor necrosis factor, alongside a decrease in anti-inflammatory cytokines like adiponectin. These factors collectively contribute to a chronic inflammatory state [60].

Hyperinsulinemia can also alter endocrine pathways, particularly those involving insulin-like growth factor-1 and androgens. These hormonal changes lead to altered cellular proliferation and growth in various tissues, including the skin. Skin conditions exacerbated by insulin resistance include keratosis pilaris, hyperandrogenism, hirsutism, acne, acrochordons, acanthosis nigricans, and polycystic ovary syndrome [61].

Weight reduction and increased physical activity are key strategies to improve insulin resistance and prevent the progression from impaired glucose tolerance to type 2 diabetes [60]. Additionally, pharmacologic treatments such as thiazolidinediones and metformin, which enhance insulin action, can also be effective [62].

Plantar Hyperkeratosis

Plantar hyperkeratosis is a condition resulting from physical pressure mechanisms and is closely linked to both the duration and severity of obesity.[63] In obese individuals, changes in foot structure—such as arch loss—lead to increased plantar pressure during walking and standing, along with a widened forefoot.[64] Weight reduction is the primary recommended treatment, as it helps reduce excessive pressure on the foot and minimize associated complications [8].

Hidradenitis suppurativa

Hidradenits suppurativa also known as acne inversa is a chronic recurrent inflammatory disorder of hair follicles in apocrine gland bearing sites resulting in abscesses and potentially fistula formation [65]. It is associated with smoking, obesity and inflammatory bowel disease.[66] Obesity is not always associated with hidradenitis suppurativa but it exacerbates the condition by shearing forces and androgen effects [67]. Androgen effect can result in coarsening of the hair shaft and subsequent follicular plugging [65].

Obesity and atopic dermatitis

Atopic dermatitis is a chronic, recurrent inflammatory skin condition influenced by genetic predisposition, immune dysregulation, and various environmental factors [68]. A strong correlation has been observed between obesity, atopic dermatitis, and bronchial asthma, particularly in obese children under five years old with persistent symptoms [69]. Obesity disrupts the epidermal barrier, leading to increased transepidermal water loss and dry skin. Adipocytes release adipokines such as leptin and adiponectin. Leptin, in particular, enhances T-cell survival and stimulates the production of pro-inflammatory cytokines—including tumor necrosis factor-α, interferon-gamma, and interleukins 6, 12, and 2—thereby significantly altering immune function [69]. Consequently, obese patients with atopic dermatitis tend to experience more frequent flare-ups of their condition, often requiring more intensive treatment [70].

Obesity and acne vulgaris

Obesity is frequently accompanied by peripheral hyperandrogenism, which can lead to increased sebum production and the development of severe acne [71]. In females, androgen-dependent acne results from elevated serum androgen levels, enhanced cutaneous utilization of androgens, or both. The pilosebaceous unit's responsiveness to androgens is a key mechanism in acne pathogenesis since sebum production is stimulated by androgens and inhibited by estrogens [72].

A study by Abulnaja examining hormonal and lipid profiles in obese adolescent females with acne vulgaris found that those with acne exhibited higher levels of interleukin-1β compared to their obese peers without acne [73]. IL-1β production, a primary function of sebaceous glands, reflects increased activity of sebocytes—the cells responsible for sebum secretion. Because sebum serves as an important nutrient source, its overproduction can promote the development of acne lesions [74]. Moreover, the secretion of pro-inflammatory lipids, cytokines, periglandular peptides, and neuropeptides by the sebaceous glands further contributes to the pathophysiology of acne [75].

Obesity and hyperandrogenism

Hyperandrogenism in obesity is primarily caused by alterations in the hypothalamo-pituitary-ovarian axis [76]. In obese individuals, increased adipose tissue and hyperinsulinemia lead to enhanced production of endogenous androgens [3]. Hyperinsulinemia, a consequence of peripheral insulin resistance, can result in a hyperglycemic state. Elevated insulin levels reduce the concentration of sex hormone-binding globulin (SHBG), which in turn increases free testosterone levels [77]. Moreover, insulin directly stimulates theca cells to produce androgens [78].

This combination of hyperandrogenism and hyperinsulinemia contributes to an atherogenic lipid profile. Both total cholesterol and triglyceride levels are elevated, while altered cholesterol metabolism due to increased lipase activity results in decreased levels of high-density lipoprotein cholesterol (HDL-C) [79].

Obesity and cancer

Overfeeding stimulates metabolic pathways in active cells that lead to increased cytokine production, which in turn recruits immune cells into the extracellular environment, resulting in systemic inflammation [80, 81]. This inflammatory response plays a critical role in the development of obesity-related diseases, as obesity is known to alter leukocyte counts and cell-mediated immune responses [80].

Studies in experimental animals have demonstrated that obese adipose tissue is infiltrated with numerous CD8+ cells, promoting macrophage accumulation while reducing the numbers of CD4+ and regulatory T-cells [82]. Similarly, research involving obese individuals has reported a decline in various T-cell subsets and their functions, alongside enhanced production of tumor necrosis factor-α (TNFα) [83]. Proinflammatory cytokines such as interleukin-6 (IL-6) and TNFα, produced by these infiltrating macrophages, may contribute to obesity-related pathologies, including cancer [84].

In addition, obesity significantly influences the extracellular matrix (ECM), altering its components and structure [85, 86]. Abnormal ECM remodeling can affect immune cell recruitment and activation, thereby actively contributing to inflammation [86]. Beyond metabolic complications, this tissue remodeling creates an environment that supports tumorigenesis [87].

Obesity and Pharmacotherapy

Weight loss drugs generally fall into two categories: those that suppress appetite and those that hinder nutrient absorption. Appetite suppressants include sympathomimetics, which work by stimulating the release of dopamine and norepinephrine [88].

Other agents approved for long-term obesity treatment include orlistat (a gastrointestinal lipase inhibitor), sibutramine (a centrally acting monoamine reuptake inhibitor), and rimonabant (an endocannabinoid receptor antagonist) [89].

In dermatology, medications with weight-adjusted dosing include oral isotretinoin and griseofulvin [3]. Obese patients treated with oral isotretinoin are more prone to elevated serum triglyceride levels, and systemic methotrexate therapy is associated with a higher risk of hepatotoxicity in this population [3]. Medications known to promote weight gain include oral corticosteroids, oral antihistamines, oral contraceptives, and certain antidepressants such as amitriptyline, mirtazapine, and paroxetine [90-93].

Obesity in the elderly

Obesity among older adults is increasing, and a significant condition associated with it is sarcopenia—the progressive loss of muscle mass coupled with an increase in fat that accompanies aging [88].

Sarcopenic Obesity

Sarcopenic obesity refers to the age-related decline in skeletal muscle mass combined with an increase and redistribution of body fat [94]. This change in body composition may result from factors such as illness or inactivity, leading to muscle loss while body fat is relatively preserved [95]. It is also linked to hormonal changes, including reduced production of growth hormone and testosterone, as well as decreased responsiveness to thyroid hormones and leptin [96]. As people age, decreased physical activity contributes further to muscle loss. The reduction in insulin-responsive tissue can result in insulin resistance, which promotes metabolic syndrome and additional fat accumulation. Moreover, increased adiposity leads to higher production of adipokines—such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6)—which further exacerbate insulin resistance [88].

Other studies related to skin and obesity

Several studies have demonstrated a strong association between obesity and a variety of cutaneous manifestations. For example, Divyashree *et al*. reported that conditions such as skin tags, acanthosis nigricans, plantar hyperkeratosis, fungal infections, intertrigo, striae, bacterial infections, and lymphedema are common in obese individuals, with fungal infections and intertrigo being particularly prevalent compared to bacterial infections [3]. Similarly, Nawaf Al-mutairi found that dermatoses including plantar hyperkeratosis, acanthosis nigricans, skin tags, striae, cutis distensae, and intertrigo are frequently observed among obese adult patients [2].

A systematic review and meta-analysis by April Zhang *et al*. highlighted an increased prevalence and severity of atopic dermatitis in overweight and obese individuals, noting that patients with eczema are more likely to be overweight or obese than the general population [68]. In a related study, Uzma Ahsan, through a case-control design, demonstrated that acanthosis nigricans, acrochordons, and striae have a statistically significant association with obesity ($P< 0.05$), whereas acne, plantar keratoderma, and lymphedema did not show such correlation ($P>0.05$) [97]. Furthermore, J.C. Boza *et al*. found that striae, plantar hyperkeratosis, acrochordons,

intertrigo, pseudoacanthosis nigricans, keratosis pilaris, lymphedema, and bacterial infections were significantly related to obesity in a comparative study [98].

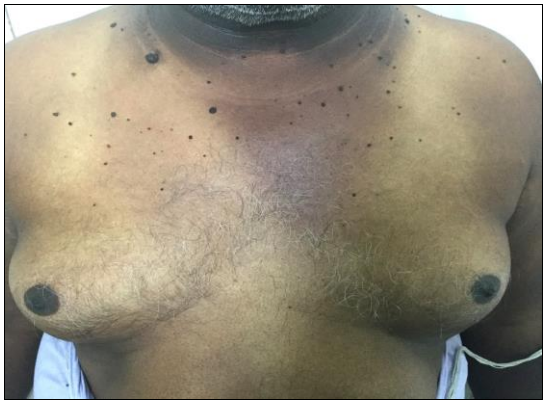

Fig 1: Acanthosis nigricans of neck with Achrocordons and Gynocomastia in a grade 3 obese patient.

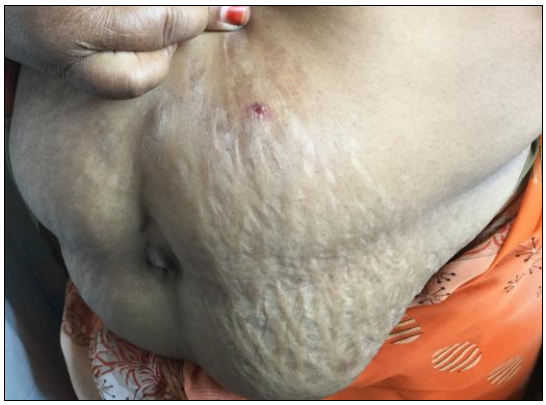

Fig 2: Abdominal striae

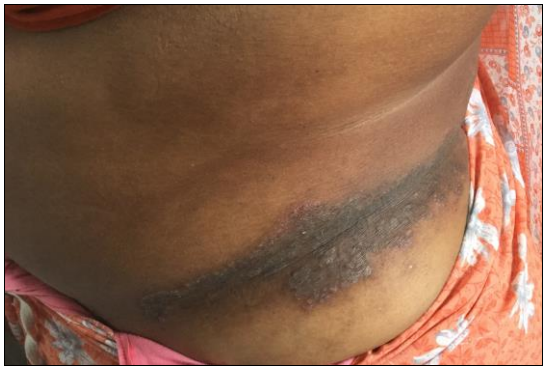

Fig 3: Tinea corporis in the abdominal folds due to increased friction and moisture.

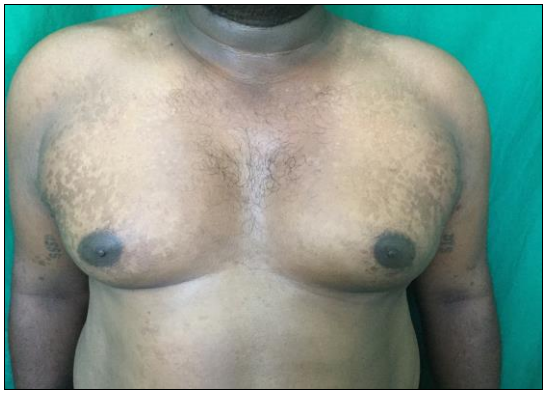

Fig 4: Chromic pityriasis versicolor on the anterior chest.

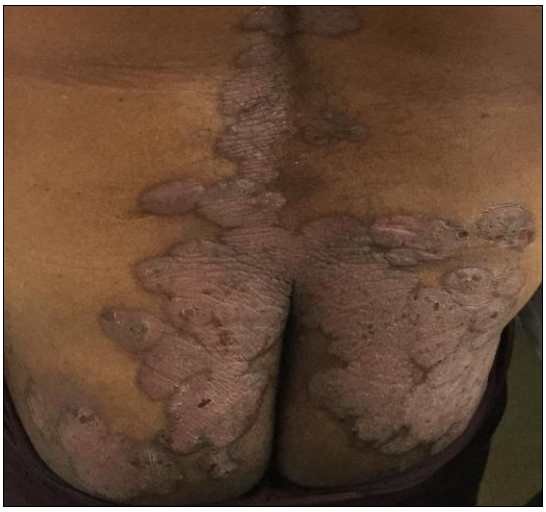

Fig 5: Psoriasis vulgaris over the lower back extending to gluteal region.

Conclusion

Obesity is a big growing problem in India. When studies are compared with western population, excess fat in Indian population generate high level of inflammation and metabolic disturbance at low BMI thresholds. In such situation the cutaneous problem associated with obesity can add to the burden of the condition. Obesity requires a multidisciplinary approach, recognition and control of dermatological complication of obesity helps in reducing the morbidity of the disease.

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