Obesity and the Skin

Abstract and Introduction

Abstract

Obesity is a serious global health problem, perhaps the biggest public health issue of our times. Excess body weight may be a factor in carcinogenesis in general, as well as contributing to the pathogenesis of metabolic, cardiovascular and musculoskeletal disorders. Obesity also has many cutaneous features, which form the basis for this review article. Many of these clinical entities are common to the majority of obese patients, e.g. striae distensae, plantar hyperkeratosis and an increased risk of skin infections. However, it may also be associated with poor wound healing, malignant melanoma and an increased risk of inflammatory dermatoses, such as psoriasis, as well as some rarer disorders. Therapeutic interventions for obesity, whether over-the-counter, prescription medicines or surgical interventions, are increasingly commonplace. All of these treatment modalities potentially have dermatological side-effects too.

Introduction

Obesity [a body mass index (BMI) > 30 kg m^{-2}] is a medical problem of increasing prevalence. It may present at any age from early childhood onwards, but it most commonly develops in mid-life.[1] In 1980, 8% of women and 6% of men in England were obese, but by 1998 this had increased to 21% of women and 17% of men.[1] This trend shows no signs of abating and it has a major impact on virtually all aspects of healthcare provision, including in dermatology.

As clinicians, we will see more patients with medical problems linked to obesity and this article covers some of the dermatological associations of excess fat deposition. One of the commonest consequences of obesity is insulin resistance and consequent type 2 diabetes mellitus[2] (). The presence of diabetes[2] will amplify the effects of obesity on other common skin problems, such as prolonged wound healing,[3] ulcers,[4] cutaneous infections,[3] psoriasis[5] and the effects of androgenization.[6] So diabetes and its associations form a secondary theme throughout this review.[2–6] The complex interactions between psoriasis, obesity and other cardiovascular risk factors, including diabetes mellitus in particular, are discussed in two separate sections.[5]

Table 1. Skin diseases associated with diabetes mellitus

Pebble fingers
Acanthosis nigricans
Infections
Diabetic rubeosis
Diabetic bullae
Oral leucoplakia
Neuropathic ulcers
Pigmentation in haemochromatosis
Vitiligo
Porphyria cutanea tarda
Scleroderma
Granuloma annulare
Necrobiosis lipoidica diabeticorum

Atherosclerosis, arterial ulcers and gangrene
Diabetic dermopathy (pigmented pretibial patches)
Genital pruritus
Acne keloidalis nuchae
Increased skin thickness and cheiroarthropathy
Dupuytren's contracture
Oral lichen planus
Acquired perforation dermatoses
Reactions to oral hypoglycaemics and insulin

Other conditions more specific to obesity, such as varicose eczema and lymphoedema, are covered in more general terms, as these topics are well reviewed elsewhere.[7] There may also be links between obesity and other inflammatory dermatoses such as atopic eczema,[8] and also with skin cancer.[9] Finally, rarer associations and the complications of the treatment of obesity are outlined in the final paragraphs. This review starts, however, with an outline of the physiology and pathophysiology of cutaneous adipose tissue.[6,10–22]

Physiological Function of Adipose Tissue

In adults, subcutaneous fat consists almost entirely of white adipose tissue (WAT; adipocytes), which provides insulation and acts as an energy source.[10–13] Where energy intake exceeds its expenditure, this excess energy is stored in adipocytes, leading to obesity.[10–13] WAT also produces many peptide hormones, cytokines and paracrine transmitters.[10–13] These include leptin, tumour necrosis factor (TNF)-α, transforming growth factor-β, interleukin (IL)-1 and IL-6, acylation stimulating protein, adiponectin, resistin, visfatin, plasminogen activator inhibitor, androgens and nonesterified fatty acids.[6,10–13] WAT also acts as a source of functional mast cell progenitors.[14] The hyperinsulinaemia associated with obesity[2] augments the production of androgens from WAT[6] and reduces circulating sex-hormone binding globulin (SHBG) levels, which further increases the supply of available free androgens.[6]

In contrast, brown fat is most prominent in the newborn and its role appears to be physiologically distinct from that of white adipocytes.[15] The role of brown fat in the older child and adult is a subject of much investigation and has been well reviewed recently.[15]

Cutaneous Pathophysiology in the Obese

Obesity alters the epidermal barrier of the skin in some way, so that obese individuals have increased transepidermal water loss and dry skin.[16] Erythema is more pronounced, compared with controls,[17] and there is reduced microvascular reactivity.[18] The obese have larger skin folds and will sweat more profusely when overheated than the nonobese.[7] Obesity inhibits lymphatic flow[7] and alters collagen formation.[19] Delayed-type hypersensitivity is increased in obesity and reduces with weight reduction,[20] which may relate to an alteration in the balance of adipocyte cytokine production.[12,21]

The shape of the foot changes with obesity.[22] For example, obese children have a lower footprint angle and obese individuals have a wider forefoot width and also higher plantar pressures during standing and walking.[22] These pressure effects may eventually lead to plantar hyperkeratosis, a cutaneous sign of severe obesity.[22]

Interestingly, no differences have been found between the activity of sebaceous, apocrine or eccrine glands in the obese compared with the nonobese,[7] despite the evidence of changes in endocrine

homeostasis with increasing weight gain (see also the section 'Adipocytokines, obesity and psoriasis').[2,5,6,10–13,23]

Obesity and Wound Healing

Obesity predisposes to poor wound healing.[24] Incisional hernias are one of the most common delayed complications.[25,26] Several clinical factors have been implicated, including an increased incidence of surgical wound infection, technical failure and haemorrhage.[25] Leptin, the adipocytokine, promotes wound healing as well as inhibiting feeding and other immunological roles.[13,27,28] Leptin resistance, associated with obesity, may contribute to the pathophysiology of impaired wound repair.[13,27,28]

Obesity and Psoriasis

The association between psoriasis and obesity is the subject of a recent evidence-based review.[5] A key question is whether obesity is causal[29–33] or a consequence of psoriasis.[30,34,35] It could be argued that a lack of physical activity, due either to the cosmetic impact of psoriasis or to the locomotor effect of psoriatic arthropathy, might predispose an individual to obesity.[35] Childhood-onset obesity may particularly predispose to both psoriasis[36] and psoriatic arthropathy,[37] suggesting a genetic link. The association between psoriasis and obesity forms part of a wider association of psoriasis with a range of cardiovascular risk factors, namely atherosclerosis, diabetes mellitus and hypertension.[38–53] The separate association of psoriasis with smoking may be an important confounding variable, particularly with regard to hypertension and atherosclerosis.[52,53]

There is increasing evidence that progressive weight loss can produce significant improvements in the severity of psoriasis.[54–58] Perhaps the most direct evidence that obesity may be causal in psoriasis is the fact that bariatric sugery can produce rapid remission from psoriasis.[54,57,58] A confounding variable is the fact that the quality of life of the average patient undergoing bariatric surgery is significantly improved,[59] which would reduce psychological stress and thus potentially the disease burden of psoriasis.[5]

Adipocytokines, Obesity and Psoriasis

Macrophages in adipose tissue produce TNF-α, as well as other cytokines involved in psoriasis, such as IL-1, IL-6, IL-17 and interferon-γ.[5,60] These adipocytokines, as well as leptin, are recruited and stimulated in obesity and may have an autocrine and paracrine effect on nearby skin.[5,29,60] Leptin, produced by adipocytes,[13] decreases T-cell autoregulation and is involved in inflammatory processes stimulating cytokine release,[28] as well as its more established role in appetite suppression and metabolic control.[27] Leptin levels have been shown to correlate with psoriasis severity.[61] Resistin is also produced in adipose tissue and leads to insulin resistance and upregulation of inflammatory processes including TNF-α secretion.[62,63] Resistin levels are increased in patients with psoriasis, correlating with obesity and increased severity of psoriasis.[62,63] Similarly, levels of adiponectin (an anti-inflammatory mediator produced by adipocytes that reduces oxidative stress) are lower in the obese psoriatic compared with the nonobese psoriatic and the inverse is true of interleukin.[13,64,65]

Thus, there is some indirect evidence that the immunological and metabolic alterations associated with obesity may be linked with the pathophysiology of psoriasis.[5,13,27–29,60–65] This is supported by the series of clinical observations that obesity does not appear to impair the treatment of psoriasis, except when treatments with the newer biologic therapies (that block cytokines) are being used.[5]

Obesity, Diabetes Mellitus and the Skin

Obesity is associated with insulin resistance and type 2 diabetes mellitus.[2,66,67] Therefore, there is a secondary association of obesity with all the cutaneous associations of diabetes mellitus (see).[2,66,67] Specific dermatological associations with diabetes and obesity would include fibroepithelial polyps,

acanthosis nigricans and acne keloidalis nuchae,[7,22,68,69] although acanthosis nigricans can also be familial or associated with malignancies[70,71] and some consider fibroepithelial polyps to be associated with diabetes alone, in the absence of obesity.[72] On the other hand, fibroepithelial polyps express increased amounts of leptin and mast cells, which might favour a link with obesity, via an altered adipocytokine environment.[13,14,27,28,57]

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Obesity and Disorders Associated With Hyperandrogenism

Hyperinsulinaemia,[2,66,67] even in the absence of diabetes, increases the production of androgens and reduces circulating SHBG in the obese.[6] This may be a contributory factor in the association of the following disorders with increasing BMI: acne, hirsuitism, androgenetic alopecia (in both sexes) and polycystic ovarian syndrome.[6,22,23,73,74] It should be noted that the anatomy of the sebaceous glands is not altered by obesity.[7]

Obesity, Vascular Disorders and Ulceration

Excess body weight puts extra stresses on the vascular system.[75,76] There is an association with varicose veins, possibly in part due to valve failure due to the high intra-abdominal pressures.[75] Venous insufficiency then leads to varicose eczema and ulceration.[76] Obese persons may have a higher pain threshold than the nonobese.[77] This decreased pain sensitivity might partly be the reason for the development of distal venous and pressure ulcers in obese individuals.[78]

Occasionally, severe obesity is associated with the development of massive necrosis of both subcutaneous fat and the overlying skin.[79] This may lead to secondary infection and ulcer formation.[76,80] The lymphatics are a fragile network of vessels that cause huge morbidity if they fail, with gross oedema and increased risk of infection due to the collection of protein-rich fluid in the tissues.[80,81] Lymphoedema presents as a soft, pitting swelling most commonly over the shins. It is often associated with obesity,[7,22] particularly the massive localized form of lymphoedema.[82]

Cutaneous Infections in the Obese

Cutaneous infections, including methicillin-resistant *Staphylococcus aureus*, are more common in those with an elevated BMI.[83] This may relate to the physical effects of larger skin folds[7] but may equally relate to the immunological alterations seen in the skin in the obese.[5,29,59,62,63] Friction and increased moisture in the deep skin creases lead to maceration and inflammation, so that intertrigo is a common association with obesity.[73,84] Poor mobility and inability to maintain adequate levels of hygiene may exacerbate this problem. For example, urinary incontinence may be more common in obese than nonobese women, exacerbating any tendency to infection and ulceration in the groins and genital area.[73]

Cellulitis occurs particularly in legs with coexisting lymphoedema.[85] This cellulitis can be particularly difficult to clear, as it may require higher initial doses of antibiotics than used normally (partly because of the higher BMI) and may also need prophylactic antibiotics to prevent recurrence.[85] Recurrent soft tissue bacterial infections in the damaged lymphatics lead to a condition called elephantiasis nostras verrucosa, which is particularly common in the obese.[86]

Folliculitis, furunculosis and paronychia are problems encountered by many and increased incidence of *Candida albicans* is also associated with obesity.[22] Erythrasma (an infection caused by *Corynebacterium minutissimum*) occurs in the obese and superficial fungal infections including onychomycoses are also a problem, particularly if patients also have diabetes.[87] Less common infections include necrotizing fasciitis and gas gangrene.[7,22,88,89]

Obesity and Atopic Eczema

Obesity has been shown to alter the inflammatory state of an individual, leading to a low-level chronic inflammatory response and IL-6 secretion.[90] There is an association between obesity, asthma and atopic eczema.[8,91,92] Mast cells can be synthesized from WAT,[14] increased leptin levels correlate with allergen sensitization[93,94] and obesity correlates with skin infections.[7,83] All these observations could perhaps provide part of the pathogenic link between increased BMI and atopic eczema.

Obesity and Skin Cancer

Obesity is associated with a number of noncutaneous cancers.[95,96] One Canadian study has found an association with nonmelanoma skin cancers (NMSCs),[97] whereas another Australian study found no association with basal cell carcinoma.[98] Various epidemiological studies have found a link between excess adiposity and malignant melanoma (MM).[9,99–103] However, others have found no significant association with MM.[104] It may be that obesity is a more relevant risk factor for skin cancer in areas of lower ultraviolet radiation (UVR) exposure,[97] but this needs further evaluation.

There is some clinical genetic evidence of a link between obesity and phototype I skin with red hair, which are features of the rare syndrome associated with a homozygous mutation in the proopiomelanocortin (*POMC*) gene.[105] Linkage between *POMC* and other traits suggests that it forms part of a common genetic obesity predisposition.[106,107] Also, experimental evidence shows that POMC neurones in the brain are the target of circulating leptin (thus inhibiting feeding), suggesting that the two are linked crucially in body weight homeostasis.[27] Heterozygous mutations in *POMC* are characterized by obesity in the absence of red hair.[108] Similarly, homozygous and heterozygous mutations in *MC1R* (the gene for the cutaneous

receptor for the *POMC* gene products) are commonly associated with phototype I skin, red hair and an increased risk of both NMSC and MM.[105] *MC1R* heterozygous mutations in the absence of red hair are even commoner than in those expressing a red hair phenotype and they still give an increased risk of both NMSC and MM over *MC1R* wild-type genotypes.[105] What is not known is whether *POMC* heterozygosity (associated with obesity without red hair) is associated with increased skin cancer risk. A recent genetic epidemiology study identified a *POMC* single nucleotide polymorphism in caucasians which was not found to correlate with an increased risk of either NMSC or MM,[109] but this may not be a relevant mutation with respect to alteration of the function of the POMC protein with regard to skin cancer risk.

Other evidence from animal experiments suggests that alteration in the leptin pathway, leading to obesity, correlates with an aberrant cytokine response to UVR.[110] It is quite possible that an alteration in the expression of adipocytokines associated with obesity, compounded by the effects of UVR, could contribute to cutaneous carcinogenesis.[110] This model might fit more with the observation linking obesity with cancer in general.

Obesity and Other Common Skin Conditions

Obesity may result in a range of respiratory complications,[111] which may necessitate a tracheostomy being performed. Cutaneous complications of this procedure include erosions, ulceration and infection.[112]

Other conditions that may be associated with or aggravated by obesity include hidradenitis suppurativa, psoriasis, keratosis pilaris, striae, seborrhoeic dermatitis, scleredema, livedo reticularis, cutis verticis gyrata, lichen myxoedematosus, lipodermatosclerosis, lichen sclerosus and pilonidal sinus.[13,22,73,113–118] However, other factors may be relevant. For example, *Malassezia furfur* infection is necessary for seborrhoeic dermatitis to occur.[119] Finger pebbles, a papular thickening over the fingers, occurs in both diabetes and obesity.[120]

Obesity and Rarer Skin Conditions

Obesity is linked with a number of rarer conditions. Keratosis follicularis squamosa is a condition consisting of scaly patches, symmetrically scattered over the trunk, buttocks and thighs.[121] Histopathological examination shows a dilated hair follicle with a follicular plug, surrounded by lamellar orthohyperkeratosis.[121] There may be an association with obesity.[121]

Granular parakeratosis is a recently recognized disorder of keratinization that is confined to intertriginous body sites, which may explain the connection with obesity.[122] The histological features are distinctive: a parakeratotic stratum corneum, with the presence of keratohyaline granules.[122] These granules are normally confined to the stratum granulosum.[122] The underlying pathophysiological defect is thought to be a failure in the normal degradation of profilaggrin and it is responsive to topical corticosteroids.[122]

Pretibial mucin deposition on the shins, in association with autoimmune thyroid disease, is known as pretibial myxoedema.[123] A similar condition (both clinically and histologically), in the absence of thyroid disease, is described in two case series of morbidly obese patients.[124,125] These papers proposed a new entity, chronic obesity lymphoedematous mucinosis, as a subtype of pretibial myxoedema.[124,125]

Polymorphic eruption of pregnancy is a self-limiting pruritic urticarial disorder of pregnancy, which starts in the last trimester or immediately postpartum.[126] Risk factors include primiparous women with excessive maternal weight gain, as well as multiple pregnancies.[126]

Dercum's disease (adiposis dolorosa) is a rare disease characterized by multiple, painful, subcutaneous lipomata that usually occur in obese, postmenopausal women.[127] It may have a familial basis.[128]

Keratoderma climacterum usually occurs in postmenopausal women, with no personal or family history of skin disorders.[128] There is a strong association with obesity.[129] The hyperkeratosis develops first at plantar pressure points and then the hyperkeratosis extends along the sole of the foot, with erythema and fissuring, making walking painful.[129] Acitretin orally can reduce the symptoms.[129]

Cutaneous Complications of Treatments for Obesity

Treatments for obesity can have cutaneous complications too. Some commercial products aimed at reducing the appearance of cellulite use heat, and erythema ab igne is a well-known complication of heat application.[130] Also, herbal remedies for obesity are not without risk.[131]

Established pharmacological therapies for obesity have also demonstrated cutaneous side-effects. For example, dexfenfluramine (now discontinued because of associated valvular heart disease and pulmonary hypertension) has been linked with urticarial vasculitis[132] and orlistat therapy with the development of a lichenoid drug eruption.[133]

Jejunoileal bypass surgery is no longer performed as regularly for weight loss, as there are newer, less invasive techniques (e.g. gastric banding) and also because a considerable proportion of patients experiences side-effects such as polyarthritis, tenosynovitis, myalgia, fever, anaemia and renal failure. This condition, known as bowel-associated dermatosis and arthritis syndrome,[134] often responds to antibiotic therapy.[135–138] Cutaneous features are also found with postjejunoileal bypass surgery, including a pustular erythematous macular eruption with a neutrophilic infiltrate similar to Sweet syndrome,[135–137] a nonpruritic papular eruption with IgG and C3 deposition,[135] and erythema nodosum.[135–138]

Conclusions

As the incidence of obesity continues to increase, all areas of medicine will encounter more patients with obesity-related complications. The dermatologist will be faced with trying to treat leg ulcers and infections due to lymphoedema, diabetes and venous disease. Intertrigo and fungal infections may present more frequently to clinics and cases of psoriasis may increase both in frequency and in difficulty in treating. Dermatological opinion may be sought in trying to reduce the appearance of striae or cellulite for cosmetic purposes. As far as the rarer dermatological conditions go, MM may become more frequent, but the dermatologist will also be expected to identify the rarer diseases associated with obesity. The public health message of weight loss may also become a more important part of a dermatologist's role than at present.

Sidebar 1

What's Already Known About This Topic?

- Obesity is associated with a number of skin conditions, including psoriasis, cutaneous infections, vascular disorders and possibly skin cancers.

Sidebar 2

What Does This Study Add?

- The review provides a structure for classifying the cutaneous problems associated with obesity.
- Psoriasis and other common skin problems are discussed in the context of obesity.

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