CASE REPORT

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Cutaneous and subcutaneous abdominal necrosis in a morbidly obese patient – a case report

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Abstract: Obesity leads to many health problems; it is a well-known risk factor for type 2 diabetes, hyperlipidaemia, arterial hypertension, cardiovascular complications as well as skin infections. A 57-year-old woman was addmited to the Department of Internal Medicine because of type 2 diabetes mellitus, morbid obesity, and deep cutaneous and subcutaneous abdominal necrosis. The patient underwent local necrectomy - the removal of necrotic deep cutaneous and subcutaneous tissue. Despite intensive pharmacoteraphy, the patient died suddenly on the eighth day after hospitalization.

Key words: obesity, type 2 diabetes, skin necrosis

INTRODUCTION

Obesity is ultimately the result of an imbalance between energy intake and energy expenditure, but this simplistic equation belies the fact that obesity is also a multifactorial disease with complex causes. Until recently, obesity was considered to be the direct result of a sedentary lifestyle plus chronic ingestion of excess calories. Twin studies demonstrate substantial genetic influences on body mass index (BMI), with little influence from the childhood environment. As much as 40-70% of obesity may be explained by genetic influences. Genetic determinants of some types of obesity have now been established and five genes affecting the control of appetite have been identified. Mutations of each gene result in obesity.

Morbidly obese patients are those individuals who weigh $\geqslant$ 45 kg over ideal body weight. This approximates a body mass index $\geqslant$ 40 kg/m^2 [1]. It is widely recognized that obesity increases the risk of coronary heart disease, hypertension, hyperlipidemia, osteoarthrosis, and diabetes. Obesity is also known to be directly related to increased risk of sleep apnea, breast endometrial, colon cancer, gallbladder disease, musculoskeletal disorders, severe pancreatitis, diverticulitis, infertility, and skin infections.

In the presented case, morbid obesity together with type 2 diabetes led to deep cutaneous and subcutaneous abdominal necrosis.

CASE REPORT

A 57-year-old female patient presented with morbid obesity, general weakness, deep cutaneous and subcutaneous abdominal necrosis, and hyperglycaemia (437 mg/dl). She had a one-month history of skin lesions. The patient had not been treated for type 2 diabetes mellitus. For a year, the patient had irregularly been taking trandolapril and diltiazem due to arterial hypertension. She also had been treated for superficial and deep vein thrombosis of the lower limbs for 3 months prior to admission.

The patient reported that her quality of life was adversely affected by her extreme obesity. She avoided contacts with people, which led to social conflicts and isolation. Her physical activity was decreased to a minimum. For the last 2 weeks prior to admission the patient was bedridden. Her bad mental state and lack of family support were the reasons for her gluttony and health-related neglect, which led to increased functional disorders, anxiety, avoidance behaviour, and eventually resulted in depressive-somatic manifestations. Her everyday diet was rich in animal fats (mainly pork and beef meat fried with animal fat as well as bacon or lard) and carbohydrates (potatoes). Her diet was also excessive in sodium chloride (spices rich in salt) and low in vegetables (except for potatoes) and fruits. Unvaried meals and lack of physical activity very quickly led to enormous increase in body weight, with a weight gain of about 20 kg during the 6 months preceding hospitalization.

Current status. On admission, her body weight was 270 kg, height 175 cm, and body mass index (BMI) 88 kg/m^2. Arterial pressure was 140/100 mmHg. During hospitalization, the patient was febrile – up to 38.6 °C. Physical examination revealed the presence of extensive (17×7 cm) cutaneous and subcutaneous abdominal necrosis surrounded by an area of skin inflammation (Figures 1, 2).

No symptoms of peritoneal irritation were observed. Heart rate was regular and accelerated to 100-110/min. Diminished vesicular sound was heard over the lung fields. The trophic lesions following superficial and deep vein thrombosis were detected on the skin of the shin, with deep and exudating ulceration, 20×10 cm in size.

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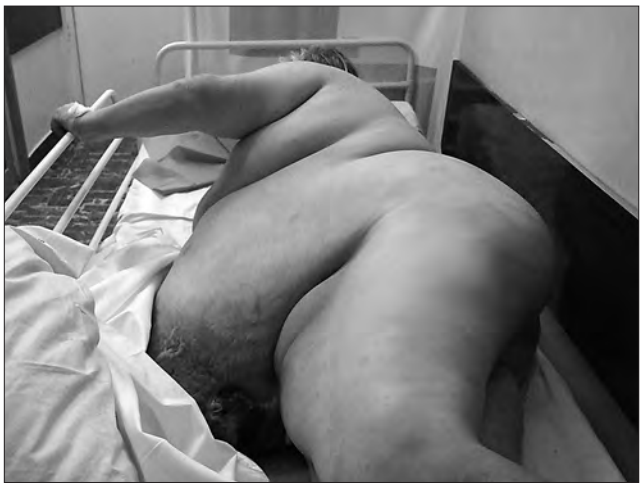
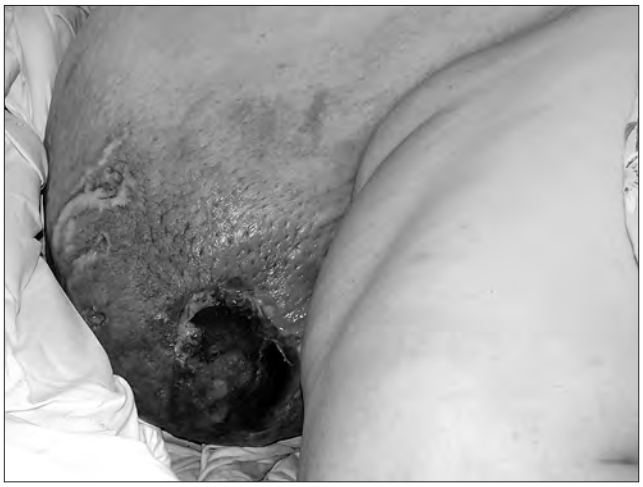


Figure 1 A 57-year-old woman with morbid obesity and deep cutaneous and subcutaneous abdominal necrosis.



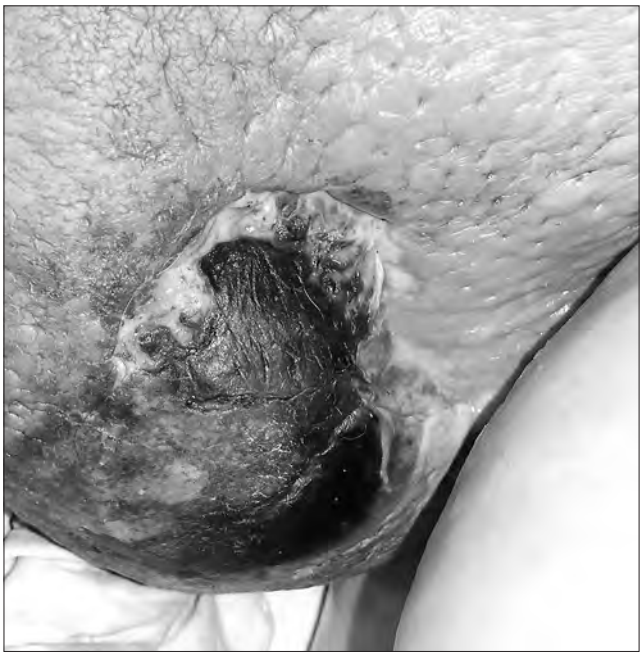


Figure 2 Cutaneous and subcutaneous abdominal necrosis surrounded by the area of skin inflammation.

Diagnostic procedures. There was no possibility to obtain good quality abdominal ultrasonography images because of the extreme obesity and thick skin folds. Chest X–ray was normal. Laboratory tests revealed normocytic anaemia with haemoglobin concentration of 8.7 g%, red blood count – 3.28 M/uL, thrombocytosis – 647 K/uL, leucocytosis – 12 K/uL and neutrophilia – 84.8%. Inflammatory markers were significantly increased: C-reactive protein (CRP) – 269 mg/l (normal 0-10), fibrinogen – 622 mg/dl (normal 200-400). The parameters of renal (creatinine, urea, electrolytes) and hepatic (ASPAT, ALAT, total protein, bilirubin, GGTP) function were normal. Serum glucose levels were high – up to 437 mg%, whereas the glycated haemoglobin level was 13.1% (reference value 4.5-6.0), which indicated poor glycaemia control during the last 3 months before admission. Repeated analyses of arterial blood gases did not demonstrate features of metabolic acidosis. Urine test disclosed urinary infection with leucocyturia and bacteriuria. The TSH level was normal – 0.5 uU/ml. The coagulation parameters were within the reference values. Due to persistent fever, the blood was sampled and the wound biopsied. After 6 days, the result of blood culture was negative. The wound culture demonstrated Escherichia coli, Streptococcus agalactiae, Candida albicans. The level of procalcytonine was normal - 0.47 ng/ml. The skin surrounding the necrotic lesions was biopsied for histopathological examinations. The biopsy specimen examined was covered with the epithelium; beneath the epithelium and in the dermis, lymphohistocytic inflammatory infiltrations were found, located primarily around small vessels.

Treatment. The patient was repeatedly consulted by surgeons due to cutaneous and subcutaneous abdominal necrosis, and surgical resection was recommended. The general condition of the patient, morbid obesity, lack of an operating table suitable for her weight of 270 kg, ruled out the possibility of administering general anaesthesia. She underwent local necrectomy and the removal of necrotic lesions resulted in a deep site in the cutaneous and subcutaneous tissue. The vascular surgeon diagnosed a post-thrombotic syndrome of the lower limbs. The patient was treated using low molecular weight heparin, ramipril (1 × 10 mg orally), amlodipine (2 × 10 mg orally), metronidazole (2 × 500 mg intravenous), amikacin (2 × 500 mg intravenous), amoxicillin with clavulanic acid (3 × 1.2 g intravenous), physiological saline infusions, short-acting insulin – 3 times a day (3 × 16 units), long-acting insulin – once a day (10 units), metformin (3 × 500 mg) and furosemide (1 × 1 ampule) intravenous.

The patient died suddenly in her sleep on the eighth day after hospitalization. Her family did not give consent for a post-mortem examination. The most likely cause of sudden death was pulmonary embolism.

DISCUSSION

Obesity is now a growing worldwide problem because of its socio-economic and medical implications. According to different health surveys, the prevalence of obesity varies from 7% in France, and even up to 30% in the USA and Brazil [2]. Obesity leads to many health problems [3].

In the presented case, morbid obesity together with type 2 diabetes led gradually to deep cutaneous and subcutaneous abdominal necrosis. Obesity is very often connected with

different dermatoses, mainly because of its negative impact on the skin physiology. It alters skin barrier function leading to excessive transepidermal water loss and erythema. Obese patients more often have dry skin and impaired skin barrier repair. The association between increased sebaceous and sweat gland activity, leading to excessive sweating and sebum production, is also known. Moreover, the skin surface pH was found to be higher in the skin folds of obese women compared with slim ones, which resulted in a significantly higher rate of cutaneous infections [4]. Some studies have shown that even up to 50% of obese patients suffer from various skin infections [5]. Other studies have demonstrated that obesity may also be connected with inappropriate collagen structure and function, which leads to impaired wound healing [6]. The chronic inflammatory state of the skin in obese patients results also from poor lymphatic return, which is associated with lymph accumulation and lymphatic dilation. Lymphedema manifests as initially soft pitting oedema in the legs. With time, the accumulation of fluid and reduced tissue oxygenation facilitate the development of bacterial infections. Long-lasting lymphedema leads to elephantiasis nostras verrucosa, defined by hyperkeratosis and pappilomatosis of the epidermis of the affected area [7]. According to some authors chronic lymphedema can even result in angiosarcoma – a malignant vascular tumour [8].

Some authors suggest that obesity leads to significant changes in cutaneous microcirculation, as well as macrocirculation, contributing to microangiopathy and hypertension [9]. Obesity also influences the composition of subcutaneous fat. In normal people it is made up mainly of white adipose tissue, which plays an important role in metabolism of lipids and glucose and endocrine functions. Adipocytes secrete many substances, such as: proinflammatory cytokines (TNF α, IL-6), C-reactive protein (CRP), enzymes (lipoprotein lipase, angiotensinogen, dehydrogenase), plasminogen activator inhibitor-1 (PAI-1), steroid hormones and peptid hormones specific for adipose tissue: leptin, adiponectin, and recently discovered resistin and visfatin. These adipocytokines may be responsible for chronic subclinical inflammation, endothelial dysfunction, insulin resistance, and finally, the development of atherosclerosis [10].

Obesity is connected with a variety of dermatoses. Skin disorders can be connected with insulin resistance (e.x. acanthosis nigricans, acrochordons, keratosis pilaris), mechanical disorders (including plantar keratosis, striae, cellulite, lymphedema, chronic venous insufficiency), infectious factors (including intertrigo, candidiosis, folliculitis, dermatophytes), or inflammatory diseases (e.x. psoriasis, hidradenitis suppurativa).

Candidiasis [11], candida folliculitis [12], intertrigo, furunculosis, erythrasma [13], tinea cruris, and folliculitis are among the most common dermatoses in obese people. These infections occur most commonly in skin folds. In a French study, the percentage of obese patients affected by skin fold mycosis reached approximately 23%, the majority of whom were women [14]. Vascular disease, diabetes mellitus, and obesity are the three most prevalent predisposing factors to fungal disease of the foot and fungal nail disease [15].

In our patient, pathological obesity resulted in the development of intertrigo involving the body folds, which was strictly connected with predisposing factors present in skin folds, such as sweating, maceration, moisture, warmth and occlusion. In most patients it can be also exacerbated by colonization with bacteria and dermatophytes. Such a prolonged inflammatory state may result in deep ulcerations which are difficult to treat. In our patient, bacteria such as Escherichia coli and Streptococcus agalactiae, as well as Candida albicans, were found among the skin folds. Moreover, she suffered from leg ulcers, which are common in obese people. Such leg ulcers are usually a consequence of venous insufficiency due to mechanical obstruction, valvular incompetence and diabetes. A few years ago, our patient suffered from deep venous thrombosis, which resulted in the development of post-thrombotic syndrome. The combination of venous insufficiency and diabetes caused ulcerations localized in the lower parts of her legs. Inappropriate treatment of venous insufficiency, diabetes, and leg ulcers led to deep ulcerations resistant to long-lasting therapy.

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