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SKIN LESION AND DISEASES IN OBESITY - PART II: THE SKIN DISORDERS RELATED TO MECHANICAL INJURES, DERMATOLOGIC DISEASES AGGRAVATED BY OBESITY AND CONSEQUENCES OF BARIATRIC SURGERY.

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ABSTRACT

Obesity is a growing epidemic affecting millions worldwide. It has numerous effects on various systems of the body leading to physiological changes that may cause the development of diseases like high blood pressure, heart disease, high blood cholesterol and type 2 diabetes. The impact of obesity on the skin has received minimal attention. The purpose of this article is to highlight the association between obesity and dermatologic conditions. We presented skin diseases related to mechanical injures due to obesity (plantar keratosis, stretch marks, cellulitis, lymphatic edema, chronic venous insufficiency), dermatologic diseases aggravated by obesity (psoriasis, acne vulgaris, hydradenitis suppurativa) and consequences of bariatric surgery. Moreover many studies confirmed that obesity is a risk factor for developing autoimmune disease (rheumatoid arthritis, systemic lupus erythematosus, systemic sclerosis and Sjögren's syndrome). Visceral fatty tissue produces adipokines (leptin, adiponectin, resistin, visfatin), which are involved in the development of autoimmune disorders. Skin lesions and disorders associated with obesity are symptoms of an increased risk of atherosclerosis and coronary artery disease too. Awareness of these disorders may help to diagnose the subclinical phase, and thus early implement the appropriate management for patients. The treatment of obesity is a difficult task and requires the action of multidisciplinary teams of physicians, dieticians, physiotherapists and psychologists. Knowledge of skin disorders and dermatological diseases in obese people is necessary because of global epidemic of obesity.

Keywords:

I. Skin diseases related to mechanical injures

❖ Stretch marks

Stretch marks are perpendicular to the direction of the greatest stress tissues. Initially red, then purple and finally white. They are described in 40% of children with moderate to severe obesity, and their number increases with duration of obesity [Hsu, et al. 1996; Tobin, et al., 2013; Yosipovich, et al., 2007]. They are a result of skin tension - stretching and thinning of connective tissue, whereby there is a crack of collagen fibers. Histological examination reveals poorly organized collagen and elastin fibers from degradation and the formation of scar tissue [Cordeiro, et al., 2010; Yosipovich, et al., 2007]. Another reason is the influence of the relative hyperandrogenism in obese women. Cordeiro et al. showed an increased expression of estrogen receptors, androgen and glucocorticoid in these lesions [Cordeiro, et al., 2010]. In the urine of obese patients with stretch marks increased levels of adrenocorticosteroids were found compared to obese patients without stretch marks [Yosipovich, et al., 2007].

Stretch marks can be regarded as 'scars' '' resulting as a damage of the tissue in which new production of collagen is an answear for local tension. In the early stage of development we observe elastosis, degranulation of mast cells and macrophages with no skin appendages [Yosipovich, et al., 2007]. Interestingly, the test of papillary and reticular skin layer reveal a big weakness of collagen and elastic fibers with reduced tensile strength in obese patients with rapid weight loss as compared to patients whose weight was diminished gradually after bariatric surgery [Sami, et al, 2015] .

Currently available treatment options are unsatisfactory. Topical tretinoin, carboxytheraphy, mesotherapy, IPL, CO_2 laser can be use.

❖ Plantar keratosis

Plantar keratosis is a consequence of increased pressure caused by body weight. It can be regarded as a physiological response to mechanical injury, which promote the accelerated proliferation of skin cells and reduce the peeling, leads to hypertrophy of the stratum corneum, it can be often seen in metatarsophalangeal joints areas [Spink, et al., 2009]. Over time, excessive hyperkeratosis causes tissue damage and pain, possibly through the release of inflammatory mediators, or the pressure of the keratin masses on the nerves [Spink, et al., 2009]. This is the type of ortokeratosis called Haxthausens symptom. This abnormality is often associated with menopause, but in obese patient occur in both sexes [Scheinfeld, et al., 2007]. Patients need a weight reduction, protective inserts for shoes and keratolytic ointments.

Excessive plantar keratosis and the formation of calluses and corns lead to feet deformation in obesity. Obese children have a wider forefoot plantar pressure and higher

when standing and walking [Shipman, et al., 2011].

Moreover, dry skin, as a consequence of impaired lymphatic and venous circulation, is the next trigger factor for plantar keratosis.

Fig. 1. Patient 51 years old, male, BMI 30.4 - hyperkeratosis feet.

❖ Cellulite

Cellulite is a physiological disorder closely related to sex, which is one of the main aesthetic problems among women. It occurs in over 85% of women over the age of 20 [Kruglikov, 2012]. This is a non-inflammatory lipodystrophy of subcutaneous connective tissue with the result disturbances of venous and lymphatic, vasomotor and the sympathetic nervous system which leads to stagnation of water and swelling of the matrix causing fibrosclerosis. It lead to irregular skin surfaces of affected areas. Predisposing risk factors are obesity, sedentary lifestyle, high-fat diets, the effect of estrogen, genetic predisposition [Casa Almeida, et al., 2013; Emanuele, 2011; Scheinfeld, et al., 2007]. Recent studies showed that hypertrophy of adipose tissue, accompanied by a reduction of proteoglycans and overproduction of hyaluronic acid with low molecular weight, can cause various types of fibrosis, resulting in non-uniform tension in the subcutaneous space [Kruglikov, 2012].

One of the most important factors intensifying cellulite is obesity, but the disorder also involves women with normal BMI. Despite the co-existence, we should clearly distinguished cellulite from obesity. Obesity is a simple accumulation of fat in the available space. Whereas cellulite is formed as a result of changes in the type of lipodystrophy in the subcutaneous tissue [Goldman, et al., 2008]. In people with normal BMI large adipocytes are located in the femoral, gluteal and abdominal regions. Adipocytes increase gradually with increasing BMI and the difference between regional stored fat is reduced. It has been observed that some women with a normal BMI < 25 have adipocytes of a size comparable with the average size of adipocytes in women with BMI > 30. Additionally, there is a pronounced hyperplasia in the gluteofemoral region of women with an increased BMI. Although the biggest adipocytes are located in the gluteofemoral region and their number increases with BMI, hypertrophy can generally appear in people with different BMIs and even in slim subjects [Kruglikov, 2012]. Therefore, the development of cellulite, beyond simple obesity, must be triggered by other factors. The increase in the volume of adipocytes leads to the increase of the tension within the fat lobules. This tension affects the skin surface and cause recess, which resembles the effect of the mattress and enhances microcirculation disturbances [Goldman, et al., 2008].

The role of metabolic subcutaneous fatty tissue in areas affected by cellulite is also interesting [Emanuele, 2011; Rodriguez, et al., 2007]. While the thickness of the abdominal region is positively correlated with the risk factors for cardiovascular diseases (a visceral adipose tissue), it is located around the buttocks and thighs having a cardioprotective effect. This may be due to the ability of the capture and storage of fatty acids and lipids from circulation, so that they aren't deposited in the liver, muscle and pancreas [Emanuele, 2011; Rodriguez, et al., 2007]. This in turn has a positive effect on glucose homeostasis and insulin sensitivity, lowering oxidative stress and lipotoxicity (protection against damage to the vascular endothelium) [Emanuele, 2011].

❖ Lymphoedema

Lymphoedema is usually localized on the legs, but can also develop on the abdomen and scrotum [Kohli, et al., 2013]. 85% of patients with this disorder have BMI > 25 [Tobin, et al., 2013]. It develops as a result of impaired lymphatic drainage, leading to lymphatic stasis. Lymphatic vessels are not able to pump the lymph: there is a discrepancy between the speed of microvascular filtration (in the capillaries and veins) and lymphatic drainage [Ai-Naimi and Cox, 2009; Mortimer and Levick, 2004]. This causes the penetration of the protein from the lymph vessels to the surrounding tissues. They cause fibrosis processes of soft tissue, and reduce oxygenation which promotes the bacterial infections - the development of inflammation of the skin and subcutaneous tissue. The increased concentration of the protein in the tissues due to more oncotic pressure draws more fluid into the tissue, which increases further fibrosis. It may lead to the development of elephantiasis and verrucosis [Tobin, et al., 2013; Yosipovich, et al., 2007]. Treatment of these disorders should include a reduction of body weight and pressotherapy (elastic stockings or pneumatic compression devices). Relations between panniculitis (cellulitis) and each episode inflammation causes further damage the lymphatic system like a vicious circle, leading to the accumulation of secondary lymphedema and thus an increased risk of cellulitis [Ai-Naimi and Cox, 2009; Mortimer and Levick, 2004; Manolopoulos, et al., 2010].

Fig. 2. Patient 55 years old, male, BMI 43.4, t.2 diabetes, hypertension - chronic pyoderma verrucosa, which is the result of chronic venous insufficiency, post-thrombotic syndrome and secondary bacterial superinfection. Onychogryphosis and fungal infection of nails - a consequence of circulatory disorders.

Fig. 3. Patient 65 years old, female, BMI 48.09, hypertension, osteoarthritis, umbilical hernia - stasis lymphedema, secondary lipodermatosclerosis and onychomycosis (Trichophyton rubrum).

❖ Venous insufficiency and leg ulcers

Increased visceral fat mass interferes a venous outflow from the lower extremities, causing a widening of the veins and dysfunction of their valves. This leads to fluid penetration and red blood cells from the vessels into the subcutaneous tissue, and is a characteristic image of stasis edema ("stasis dermatitis") and the hyperpigmentation of skin. Hemoglobin derived from the red blood cells can cause an inflammatory reaction, and iron from heme induces characteristic colour [Tobin, et al., 2013;

Yosipovich, et al., 2007]. Increased blood pressure in the venous system causes damage to valves and reflux, resulting in the formation of varicose veins and extension. Developing symptoms of chronic venous insufficiency can lead to leg ulcers. The risk of ulcers is correlated with the degree of stasis edema [Tobin, et al., 2013; Danielsson, et al., 2002]. The study Iannuzzi et al., reveled that obesity is associated with the occurrence of varicose veins, independently of sex steroids in postmenopausal women [Iannuzzi, et al., 2002]. There are associations between BMI and the varicose veins, regarding age, the concentration of estradiol, testosterone and SHBG. This study showed that obesity interferes normal blood flow between the superficial and deep venous system of the lower limbs due to increased amounts of fat and fibrous tissue surrounding the vein, leading to a standstill [Iannuzzi, et al., 2002]. The consequence of these disturbations are symptoms of heaviness, reduction of local blood flow, lymph tissue oxygenation and reduced wound healing.

In addition, developing lipodermatosclerosis and venous ulcers further intensifies the symptoms of chronic venous insufficiency. 70% of ulcers develop the saphenous vein. There is a feeling of itching, pain and burning. Ulcer healing is impaired in obese. It has been shown that the skin obese mice had reduced mechanical strength due to the absence of collagen [Yosipovich, et al., 2007].

II. Dermatologic diseases aggravated by obesity

❖ Acne vulgaris and acne inversa (hidradenitis suppurativa)

Acne vulgaris is more severe in obese people. It is associated with hyperandrogenism, increased secretion of insulin, growth hormone and IGF-1 [Yosipovich, et al., 2007]. In PCOS, with acne and obesity, using antidiabetic medication - pioglitazone, reduces the symptoms of acne [Romualdi, et al., 2003]. In hair follicles, because of the hyperkeratosis, a lymphocytic-histiocytic inflammation involving pro-inflammatory cytokines (IL-1 β, IL-10, IL-12, IL-23 and TNF-α) and hyperactivity mTORC1 signaling complex is observed. Activated mTORC1 increases the secretion of androgens and contribute to the proliferation of the sebaceous glands [Lim and Oon, 2016; Melnik and Zouboulis, 2013].

Saudi study evaluating women with active acne between the ages of 13 and 42 years of age showed a positive correlation between the severity of acne lesions with BMI and elevated blood levels of testosterone, prolactin and DHEAS [Alan and Cenesizoglu, 2014]. In contrast, Del Prete et al. showed that males in an Italian population with severe course of acne had a high BMI and insulin resistance [Del Prete et al., 2012]. Exacerbation of acne lesions occur with increasing body weight and consumption of milk [Melnik, et al., 2013].

Hyperinsulinemia occurs in obesity, and increased production of IGF-1 by the keratinocytes receptors leads to follicular epidermal hyperproliferation with subsequent plugging of the pilosebaceous unit, contributing to the formation of comedones, lipogenesis in the sebaceous glands and androgen synthesis [Seleit, et al. 2014]. In addition, insulin stimulates the synthesis of androgens, increasing the production of sebum, exacerbating acne [Napolitano, et al., 2015]. Furthermore, IGF-1 stimulates the activity of 5-α-reductase, leading to increased proliferation of sebaceous glands, sebum production and lipogenesis, influencing acne development [Kumari and Thappa, 2013; Napolitano, et al., 2015]. The study Seleit et al. found a strong positive relationship between IGF-1, BMI and severity of acne [Seleit, et al. 2014].

Obesity appears to be a risk factor for the development of acne inversa. The visceral fat tissue produce the proinflammatory cytokines, including IL-6 and TNF-α, which are involved in the pathogenesis of acne. In particular, the high glycemic index and high protein milk diet increases insulin and IGF-1 level. FoxO1 and mTORC1 are involved in stimulating the production of androgens, keratinocyte proliferation and sebaceous glands [Lim and Oon, 2016; Melnik and Zouboulis, 2013]. This explains the impact of obesity and insulin resistance at the worsening of acne inversa. Among patients with this disease 26% of men and 33% of women were obese [Slade, et al., 2003; Tobin, et al., 2013]. In other studies, this percentage was as high as 75% [Tobin, et al., 2013]. Obesity increases the occlusion, maceration and friction by overlapping folds of skin [Tobin, et al., 2013; Yosipovich, et al., 2007]. Miller et al. performed a cross-sectional study on a Danish patients and compared with controls using Bioelectrical Impedance Analysis to assess body composition. They revealed that hidradenitis suppurativa is associated with a high fat percentage, high visceral fat, and low muscle percentage adding to the morbidity of the disease. The higher predicted estimate of basal metabolic rate may reflect a dysfunctional metabolism contributing to the high-fat-body composition [Miller, et al., 2016]. In turn, weight loss can alleviate the disease [Slade, et al., 2003]. Another factor may be relative hyperandrogenism.

Fig. 4. Patient 52 years old, male, hydradenitis suppurativa for 17 years, BMI 37, diabetes t.2, high blood pressure, hypertrigliceridemia: the picture above - comedolike follicular occlusion, chronic relapsing inflammation, mucopurulent discharge, and progressive scarring on the right axillary region; picture below - symptoms of hydradenitis suppurativa accompanied by acrochordons on the left axillary region.

❖ Psoriasis

Numerous epidemiological studies confirm the connection between obesity and psoriasis. Obesity is an independent factor in the development and worsening of psoriasis [Owczarczyk-Saczonek and Nowicki, 2013]. Overweight (BMI 26-29) slightly increases a risk of developing the disease, and obesity (BMI> 29) even more than 2-times [Naldi, et al., 2015]. Meta-analysis of 16 observational studies revealed that the odds ratio for the coexistence of obesity (BMI ≥ 30) among patients with psoriasis was 1,66 compared to healthy subjects: a mild form 1,46, and a severe 2,23. This means that obesity

increases the risk the disease by more than 50% [Armstrong, et al., 2012]. In addition, weight reduction improves psoriasis [Gottlieb, et al., 2008]. Moreover, Jin et al., in Chinese population study found that the coexistence of obesity and the presence of HLA-Cw6 increases the risk of developing psoriasis by 35-times compared to slim individuals without this gene [Jin, et al., 2008]. Similar data apply to children and adolescents: the coexistence of psoriasis with obesity is much more frequent than in adults and is associated with many complications in the future, such as sleep apnea, insulin resistance and an increase in cardiovascular mortality in adulthood [Mercy and Paller, 2013]. It also increases the risk of developing psoriatic arthritis [Soltani-Arabshahi, 2010].

Macrophages of visceral fatty tissue are the source of pro-inflammatory cytokines responsible for the development of chronic inflammation in adipose tissue, such as, TNF-α, IL-1, IL-6, IL-17 [Carcarossa, et al., 2014]. They are also very important cytokines not only involved in the development of psoriatic lesions, but also influence of systemic inflammation. The consequence is the insulin resistance because of TNF-α, resulting in: inhibition of insulin receptor and glucose transport protein (GLUT-4), as well as lack of insulin secretion in pancreas and adiponectin production from adipocytes, responsible for insulin sensitivity [Olszanecka, et al., 2005]. Moreover, insulin resistance affects endothelial cell dysfunction, leads to arteriosclerosis and finally, myocardial infarction or stroke. This explains the concept of "psoriatic march" and the observations of the frequent coexistence of psoriasis with obesity [Boehncke, et al., 2011].

Many studies confirm that adipokines produced in visceral fatty tissue affect the severity of psoriasis [Owczarczyk-Saczonek and Nowicki, 2013]. Observations show a relationship between increased levels of some proinflammatory adipokines such as chemerin, visfatin or resistin, with severe course of disease and lowering their concentration along with the treatment [Ismail, et al., 2012; Gerkowicz, et al., 2012; Takahashi, et al., 2013]. The concentration of anti-inflammatory adipokines (adiponectin, omentin and waspin) are reduced in the serum of patients with severe disease compared with patients with mild forms of psoriasis [Ismail, et al., 2012; Gerkowicz, et al., 2012; Takahashi, et al., 2013].

Fig. 5. Patient 80 years old, female, BMI 47, t.2 diabetes, high blood pressure: the picture above - the symptoms of hirsutism; picture below - plaque psoriasis (PASI 11.6) accompanied by hirsutism and stretch marks on the abdomen.

Fig. 6. Patient 54 years old, female, BMI 36, severe plaque psoriasis for 40 years (PASI 12.4) - chronic venous insufficiency, cellulite and plantar keratosis.

III. Autoimmune diseases and obesity

Many studies confirmed that obesity is a risk factor for developing autoimmune disease. Italian studies indicated that the incidence of obesity was 12,4% in patients with early rheumatoid arthritis, 13,5% in seronegative spondylitis, 10% in systemic lupus erythematosus (SLE), 10,4% in systemic sclerosis and 11,3% in Sjögren's syndrome [Gremese, et al., 2014]. In turn, American studies showed that the prevalence of obesity in women with systemic lupus erythematosus ranges from 28 to 50%, but no correlation with disease activity was found [Katz, et al., 2012; Versini, et al., 2014]. However, other studies reportered a relationship between BMI in patients with SLE, lupus nephritis and hypertension [Chaiamnuay, et al., 2007; Nikpour, et al., 2009; Versini, et al., 2014]. Patient with SLE have a higher risk of cardiovascular disease and the Framingham score compared to the general population and SLE patients with a normal weight [Rizk, et al., 2012]. However, the frequency of obesity systemic scleroderma is from 9 to 18%, less than in the normal population [Gremese, et al., 2014].

Visceral fatty tissue produces adipokines (leptin, adiponectin, resistin, visfatin), which are involved in the development of autoimmune disorders. The connections between adipokines in rheumatoid arthritis, SLE, inflammatory bowel disease, multiple sclerosis, type 1 diabetes, psoriasis and psoriatic arthritis, and autoimmune thyroiditis has been proven [Versini, et al., 2014]. After low-calory diet in hypoleptynemia and in leptin deficient mice on decrease number of Th17 and an increase Treg is observed [Versini, et al., 2014]. Furthermore, obesity worsens the course of these conditions and impairs the response to therapy.

Adiponectin, which level is reduced in obesity, has an anti-inflammatory effects in rheumatic diseases [Gremese, et al., 2014]. Leptin is considered as a pro-inflammatory cytokine, which stimulates production of other proinflammatory cytokines from macrophages and enhances Th1 cytokine, and therefore can polarize Th1/Th2 balance towards Th1 phenotype [Gremese, et al., 2014]. In addition, leptin may control the modulation of immune tolerance by Treg [Procaccini, et al., 2011].

Another factor leading to the development of autoimmune disorders in obese is deficiency of vitamin D3, probably as a result of disorders of the intestinal flora, due to diet. It affects the imbalance between Th17 and Treg cells, resulting in autoimmune responses [Versini and Aljadeff, 2014; Versini, et al., 2014]. Furthermore, in obese fatty tissue indicates an increased production of IL-6 secreted by adipocytes and macrophages of visceral fat, which also affect the differentiation of naive T cells to Th17 [Wiener, et al., 2009]. Therefore, obese women have higher concentrations of cytokine Th17-dependent (IL-17, IL-23), but without an increase in the concentration of Th1 cytokines (IL-12, IFN-γ) [Sumarac-Dumanovic, et al., 2009].

Another factor responsible for the development of autoimmune diseases is a protein produced by the macrophages, which supports the survival of macrophages to various stimuli inducing apoptosis - macrophage apoptosis inhibitors (AIM macrophages). Its concentration rises in the blood of obese people and is responsible for the induction of lipolysis. By binding to natural

autoreactive antibodies IgM, they create immune complexes, which are presented to dendritic and B cells, leading to the production of IgG antibodies and stimulate the autoimmune processes [Arai, et al., 2014; Versini and Aljadeff, 2014].

Finally, an interesting concept is the influence of a high-salt diet and high-fat on intestinal microbiota, which leads to dysbiosis, causing an imbalance between Treg and Th17 cells [Brown-Coffee, Versini, et al., 2014]. In addition, it contributes to the prevalence of vitamin D deficiency among obese patients [Versini, et al., 2014].

Fig. 7. Possible pathophysiological mechanisms between obesity and the stimulation of autoimmune disorders.

IV. Skin cancer and obesity

The relationship between obesity and increased risk of melanoma remain unexplored, although the population epidemiological studies provide reliable evidence on the relationship [de Giorgi, et al., 2012]. The mechanism is probably multifactorial. Brandon et al. found that obesity in mice results in a sharp increase in melanoma by supporting angiogenesis [Brandon, et al., 2009]. Hyperinsulinemia and IGF-1, as a consequence of insulin resistance in obesity, promote tumor growth, inhibiting apoptosis and stimulating cell proliferation. Secondly, leptin, produced in visceral fat, stimulates cell proliferation and angiogenesis too [de Giorgi, et al., 2012; Renehan, et al., 2008]. Homozygous mutation of proopiomelanocortin (POMC) concomitant with the red color of hair and phototype I skin is also a genetic predisposition to obesity. In addition, patients with 1,56-time higher serum leptin concentration have higher risk of melanoma [Millington, 2013]. Recent studies Skowron et al., showed that it is correlated with BMI and greater thickness infiltration of primary melanoma in Breslow scale [Skowron, et al., 2015].

Interestingly there is a negative association between obesity and cancers of the skin. In the population of American Caucasian the study found 32% lower risk of developing squamous cell carcinoma (SCC) and 19% basal carcinoma (BCC) in obese women with (BMI > 30). Probably it is a consequence of decreased physical activity, less exposure to ultraviolet rays, which it is a strong factor in the development of cancers [Olsen, et al., 2006; Pothiawala, et al., 2012].

V. Influence of the bariatric surgery on the skin

Bariatric surgery is a radical method of treatment of morbid obesity in patients not responding to conventional therapy. Numerous observations confirm an improvement in certain skin diseases related to obesity [Wollina, et al., 2015]. There is a reduction of inflammatory lesions in 15% of acne inversa patients and even 35% remission [Kromann, et al., 2014; Wollina, et al., 2015]. Similarly in psoriasis, meta-analysis of the effect of bariatric surgery in obese patients showed improvement of skin lesions in post-operative patients. Probably reducing systemic inflammation associated with obesity, has a crucial importance. After bariatric surgery there is a lower level of TNF-α in adipose tissue [Hossler, et al., 2013].

However, after treatment, especially in women, diffuse alopecia is observed as a result of deficiency of vitamins and microelements (zinc, iron, copper, selenium) [Rojas, et al., 2011; Wollina, et al., 2015]. This problem usually begins in the third month after surgery, and affects 19 to 60% of patients [da Silva, et al., 2014; Rojas, et al., 2011].

High level of proinflammatory leptin and reduction anti-inflammatory adiponectin is characteristic in obesity, and after bariatric surgery concentrations of these hormones act paradoxically. These variations, which can affect the function of the immune system are not fully understood. Perhaps the sudden reduction in body weight can become a factor provoking the development of autoimmunity. There are reports of the development of systemic autoimmune diseases after bariatric surgery: in 2 women - SLE (associated with antiphospholipid syndrome) and 2 men - rheumatoid arthritis [Cañas, et al., 2016].

Patients after bariatric surgery have a problem with the removal of excess of the skin. Studies have shown that the rapid reduction in weight loss is associated with damage to certain components of the extracellular matrix of the skin, especially elastin and collagen fibers, which leads to higher rate of complications and worse aesthetic results. In contrast, there is an increase of type III collagen expression which slows the correct healing process [Manzoni and Weber, 2015].

The rules of skin care in obese patients

- Proper personal hygiene (regular baths, low pH cleansers, hypoallergic emollients - moisturization of the skin);
- Prevention of friction (breathable clothing made of natural materials);
- Control perspiration and moisture (antiperspirants, talc);
- Self-control skin:
- Protection from the sun.

Conclusions

Skin lesions and disorders associated with obesity as acrochordons, acanthosis nigricans are symptoms of an increased risk of atherosclerosis and coronary artery disease. Their occurrence is associated not only with obesity, but insulin resistance, atherogenic lipidemia, or metabolic syndrome. Awareness of these disorders may help to diagnose the subclinical phase, and thus early implement the appropriate management for patients [Dwivedi and Jhamb, 2010].

The treatment of obesity is a difficult task and requires the action of multidisciplinary teams of physicians, dieticians, physiotherapists and psychologists. Knowledge of skin disorders and dermatological diseases in obese people is necessary because of global epidemy of obesity.

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