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Dermatological manifestations of endocrine diseases

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Abstract: Almost all endocrine diseases can be accompanied by damage to the skin and its appendages (hair and nails). Naturally, this is not always the leading symptom and often has a non-specific character. In other words, manifestations can occur in other conditions, not necessarily in diseases of the endocrine organ. Therefore, we, endocrinologists, do not rely on skin manifestations when making a diagnosis, since they are an additional plus in favor of any pathological condition.
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Skin damage in endocrine diseases is a rather complex process. There are several mechanisms by which endocrine diseases lead to skin lesions, namely:

• Hormones interact with the receptor of the cell surface in order to regulate their function. Many types of cells located in the skin have hormonal receptors. A deficiency or excess of hormones can disrupt the metabolism of the skin. Hot and moist skin, for example, often indicates hyperthyroidism.

• An excess or deficiency of the hormone can act on the skin indirectly (and most often it happens that way), and not through specific receptors. An example of this is hyperglycemia (in diabetes, which causes an increase in the frequency and severity of skin infections). In some rare skin diseases, the appearance of which suggests the presence of endocrine diseases, the pathogenesis remains unclear. Examples of such diseases are diabetic lipoid necrobiosis, scleredema (in diabetes mellitus) and pretibial myxedema (in hyperthyroidism).

So, what are the skin manifestations of endocrine diseases that practitioners need to remember? Let's consider each nosology separately.

Addison's disease develops when more than 90% of the adrenal cortex tissue is destroyed. It can develop at any age, but more often from 30 to 50 years, has no sexual differences. The main skin manifestation is darkening of the skin. The mechanism of hyperpigmentation development is due to the increased production of ACTH, and with it MSG or melanocyte-stimulating fraction of ACTH. The insufficient level of ascorbic acid in the body in Addison's disease suggests a similarity in the pathogenesis of pigmentation disorders in this disease and C-vitamin deficiency. High copper content in the blood of Addison's disease patients increases the activity of tyrosinase and enhance the synthesis of melanin.

Clinical picture: Skin pigmentation can have a very different shade. Previously, this disease was called "bronze disease". The most common is golden-brown, earthy, lemon-yellow shade or the color of a sun tan. The development of pigmentation can begin with a short-term inflammation of the skin, after which a persistent hyperpigmented spot remains. Spots can appear on any part of the body, but more often open skin areas, places of constant pressure and friction (lower back, elbows, knees), large skin folds are pigmented. It is characterized by increased pigmentation of the folds of

the palms, soles, while the rest of the surface is not pigmented. There is an increase in pigmentation in the area of postoperative and post-traumatic scars, in physiologically hyperpigmented places (breast nipples, genitals), new nevi appear and there is an increase in pigmentation of old melanocytic nevi. A number of patients have dark hair**.**

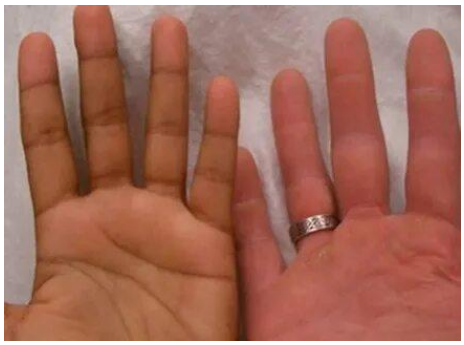

Fig.1.Hyperpigmentation

Pigmentation of the oral mucosa is an important sign. The pigment is deposited in the form of a dark strip along the edge of the gums at the roots of the teeth, on the mucous membrane of the cheeks against the molars "buccal pigmentation". However, the absence of mucosal lesions does not exclude the disease. The mucous membranes of the rectum and vagina are often pigmented, less often the conjunctiva of the eye. In 5-20% of cases, the opposite phenomenon occurs – loss of pigmentation and the appearance of areas with depigmented skin – vitiligo.

Diabetes mellitus

This is a group of endocrine diseases associated with impaired glucose uptake and developing due to absolute or relative (violation of interaction with target cells) insufficiency of the hormone insulin, resulting in hyperglycemia. There are types 1 and 2 diabetes mellitus, gestational diabetes and symptomatic diabetes. Doctors have described more than 30 types of dermatoses that can occur with diabetes. They can be divided into three groups: primary — caused by metabolic disorders (xanthomatosis, lipid necrobiosis, diabetic blisters and dermatopathies, etc.); secondary — when bacterial or fungal infection is attached; skin problems during drug treatment, i.e. allergic and adverse reactions.

With type 2 diabetes, when there is insensitivity to insulin, Acanthosis nigricans can develop. These are areas of hyperpigmentation with simultaneous thickening of the skin and possible hyperkeratosis. Looks like dirty skin. There are many reasons for the development of insulin resistance. Cushing's syndrome, acromegaly and polycystic ovaries can also cause the development of Acanthosis nigricans. Familial obesity, certain malignant tumors, mainly adenocarcinomas of the gastrointestinal tract, can by themselves produce insulin-like growth factors and thus cause Acanthosis nigricans (Fig.2).

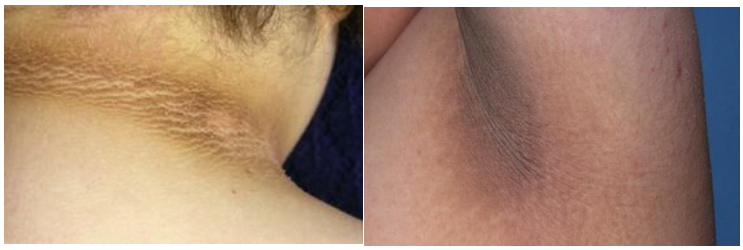

Fig.2.Acanthosis nigricans

n the treatment of diabetes with insulin injections, the following may occur: diabetic lipodystrophy (thinning of the fat layer at the injection sites), as well as the reverse condition, when fat "bumps" form in these places. To avoid this, you just need to regularly change the injection sites (Fig.3).

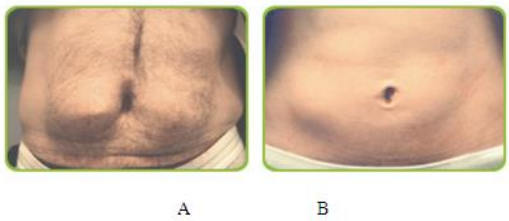

Fig. 3 Diabetic lipodystrophy

Lipoid necrobiosis is a disease that most often occurs in the tibia area on the shins, although it can develop in other places. The early manifestation is erythematous papules that have no special signs, which transform into annular lesions of yellowish-brown color with clearly visible dilated vessels and central epidermal atrophy (Fig.4). Lipid necrobiosis only in 0.3% of diabetics and develops in both insulin-dependent and insulin-resistant patients.

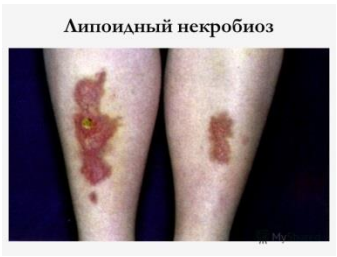

Fig. 4. Lipoid necrobiosis

Scleredema is characterized by pronounced induration and thickening of the skin of the neck, back, upper extremities. Histopathological examination reveals a thickening of the dermis with an underlying increase in the amount of a mucus-like substance. Scleredema is often associated with previously transmitted streptococcal infections, monoclonal gammopathy and diabetes. In adults with scleredema (adult scleredema), insulin-resistant diabetes is noted with a certain frequency, which causes a long-term chronic course of sclerodema, unlike post-streptococcal sclerodema, which in most cases resolves within 1-2 years.

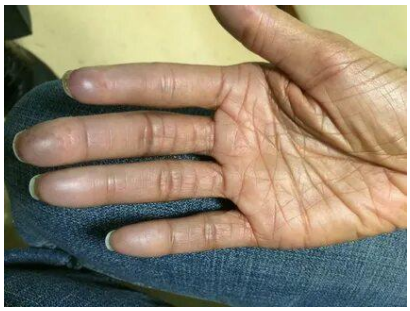

Fig. 5. Sclerodema

Bacterial skin infections in diabetic patients appear much more often and are more severe. Diabetic foot ulcers occupy a leading place among them. Numbness of the feet associated with diabetic neuropathy prevents the recognition of damage, and hyperglycemia, which disrupts the function of leukocytes, contributes to the development of bacterial infection. Staphylococcal folliculitis or skin abscesses in such patients are described in detail and treated well with antibiotics and surgical drainage of abscesses. Patients with diabetes may develop an external necrotizing ear infection caused by *Pseudomonas aeruginosa*. *Candidiasis*, usually caused by *Candida albicans*. Candidiasis of the skin and mucous membranes is characterized by red plaques with white exudate adhering to them and pustules located nearby. Candidiasis vulvovaginitis is extremely common. *Perianal dermatitis* in both men and women can be caused by *Candida*. Other forms of candidiasis of the skin and mucosa include thrush (infections of the oral mucosa), congestion (angular cheilitis), intertrigo (infection of skin folds), chronic interdigital erosive candidiasis (interdigital erosion), paranychia (infection of soft tissues around the nail plate) and onychomycosis (nail infection). The mechanism of development, apparently, involves an increased glucose content, which serves as a

substrate for the growth of *Candida*. Patients with recurrent candidiasis should be examined for the presence of diabetes. Sometimes, with diabetic ketoacidosis, the patient may develop mucoromycosis as a complication. Mucoromycosis is a severe progressive infection of soft tissues caused by the saprophyte fungi *Mucor, Phizopus and Absidia spp.* The infection is difficult to treat with systemic antifungal agents and often leads to death.

Hypothyroidism is a condition caused by a prolonged, persistent lack of thyroid hormones. The extreme degree of clinical symptoms of hypothyroidism in adults is myxedema, in children — cretinism. With hypothyroidism, there is a decrease in skin trophism, as well as a slowdown in collagen synthesis. As a result, the skin is dry, flaky, pale, with reduced turgor, as well as edematous due to metabolic disorders, xanthus may appear due to lipid metabolism disorders (Fig.6). Due to poor microcirculation, the skin is cold to the touch. With severe hypothyroidism, the skin becomes yellowish and thickened due to puffiness.

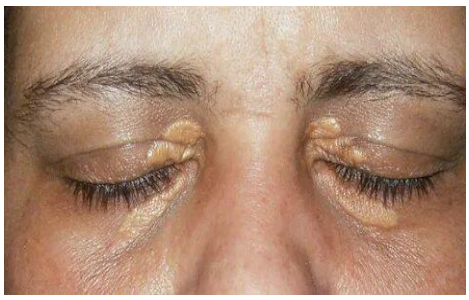

Fig. 6 Xanthoma of the eyelid

Due to the slowing down of tissue metabolism, not only the skin suffers, but also the appendages. Nails become brittle, layered with an uneven surface. The hair is dull and brittle, their natural renewal and growth stops, there is excessive loss. The yellow color of the skin is caused by an excessive content of carotene in the blood serum, which is deposited in the corneal layer. This excess of carotene is caused by a violation of the production of vitamin A from carotene in the liver. Thickening of the skin is caused by an increase in the amount of mucopolysaccharides and mucin in the skin (myxedema).

Thyrotoxicosis.

In a state of thyrotoxicosis, metabolism is accelerated, which means that the trophic of tissues is increased. This has a good effect on the quality of the skin. Due to increased blood circulation, the skin is hot, smooth, there is excessive sweating, and there is often an unhealthy blush on the cheeks. The quality of the hair is good, it grows well, is moisturized and has a shiny, smooth surface. With hyperthyroidism, pretibial myxedema may occur. This is swelling and infiltration of the skin of the feet and ankle joint. Rarely, pathological processes rise above the calf muscles. This condition often accompanies diffuse toxic goiter and is poorly treatable (Fig.7).

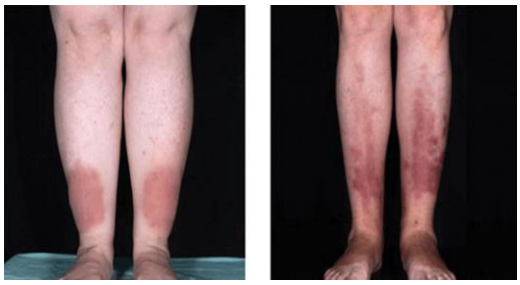

Fig. 7 Pretibial myxedema

Pretibial myxedema is specific to Graves' disease, but is observed only in 3-5% of patients with this disease. Pretibial myxedema is often observed in Graves' ophthalmopathy, leading to exophthalmos and acropathy (pin-shaped nails). Treatment of hyperthyroidism has no effect on the pretibial myxedema.

Itsenko-Cushing syndrome is caused by excessive synthesis of glucocorticoid hormones of the

adrenal glands, among which cortisol is considered the most recognizable. Excess cortisol affects the synthesis of collagen, suppressing it, as a result, the skin becomes thinner, loses elasticity and the ability to stretch. This is how stretch marks appear – vertical, wide (up to 5 cm) striae of bright burgundy color.

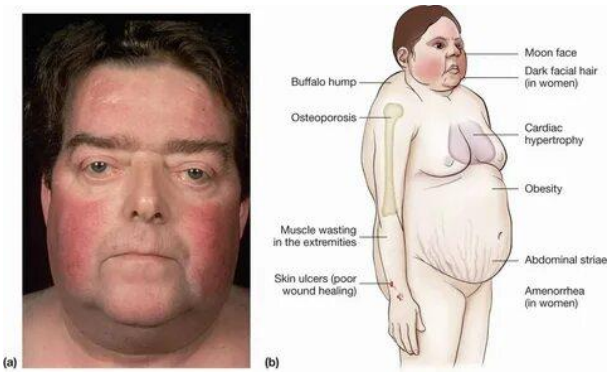

Fig. 8. Itsenko–Cushing's disease

In addition to the listed changes in this disease, there may be increased hair loss and acne. The skin is usually thin and atrophic. Wound healing is slow. The above-mentioned skin changes also occur with prolonged local treatment with strong steroid drugs. Also characterized by a wide (moon-shaped) face, increased deposition of subcutaneous fat on the back of the neck and upper back (climacteric hump) and on the trunk. The stripes on the skin and telangiectasia may turn pale during treatment, but they do not disappear.

The effect of sex hormones on hair and sebaceous glands. Androgens during maturation stimulate the activity of the sebaceous glands and the formation of acne. An excess of androgens in women leads to acne, hirsutism (increased facial hair) and baldness (male or female type). Concomitant symptoms include hyperpigmentation of the skin of the genitals and periarticular circles, as well as clitoromegaly.

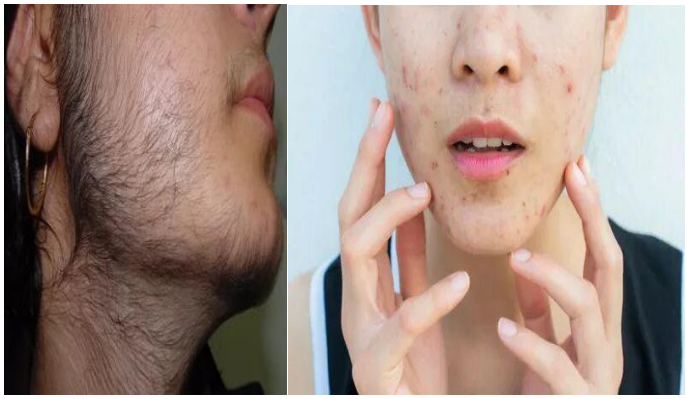

Fig.9 Hirsutism and acne

Possible causes of hirsutism and acne in women include adrenal and ovarian tumors, prolactin-producing pituitary tumors, polycystic ovarian diseases and adrenal enzyme deficiencies. Familial acne and hirsutism may be due to the hypersensitivity of the target organ to androgens circulating in the blood in normal amounts. Most cases of acne and hirsutism in women are familial benign in nature. Screening tests for tumors include testing for serum prolactin, dehydroepiandrosterone sulfate (an androgen secreted by the adrenal glands) and total testosterone.

Acromegaly

This is a disease that results from an excess of growth hormone in the body produced by the pituitary gland. Pronounced thickening of the bones leads to coarsening of facial features and an increase in the hands. Skin manifestations are the result of the production of a growth factor similar to insulin, and therefore partially coincide with skin changes in insulin resistance. The skin is hypertrophied and thickened, sometimes *Acanthosis nigricans* is observed. These changes can be

especially pronounced on the scalp as curling grooves (Cutis vertices gyrata). Thus, skin changes in endocrine diseases are one of the important criteria that are used in the diagnosis of diseases and, accordingly, require timely treatment**.**

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