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ROSACEA AND ITS ASPECTS

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Abstract: Rosacea (syn.: rosacea, acne rosacea, acne rosacea, gutta rosacea, cuperose, teleangiectasiasis faciei) is one of the most common dermatoses, the incidence of which ranges from 0.5 to 10% and occupies 7% of the local diseases. The exact etiology of rosacea is still unknown, but, of course, the disease is multifactorial, that is, the hereditary predisposition underlying it is realized under the influence of many external factors. The article deals with the issues of epidemiology, pathogenesis of rosacea. The features of classification, clinical course, diagnosis are outlined. A detailed description of the occurring clinical manifestations, supplemented by illustrations, is given.

Keywords: Rosacea, erythema, telangiectasias, Demodex mites, rhinophyma, rosacea-like diseases.

INTRODUCTION

The clinical and histological characteristics of rosacea remain poorly understood, despite the fact that rosacea is a relatively common dermatosis. There is no laboratory test that can easily confirm the diagnosis of rosacea. Additionally, the idea that certain conditions are “related” to rosacea is based on clinical similarities rather than scientific evidence.

MATERIALS AND METHODS

The term “rosacea” covers a set of symptoms and signs, which include persistent facial erythema, telangiectasia, inflammatory nodules and pustules, frequent flushing, non-pitting swelling on the face, inflammation changes in the eyes of various types, phymatous changes, mainly the nose, as well as ears, forehead, chin, eyelids. Some authors distinguish between rosacea fulminans, characterized by the rapid appearance of nodules and pustules superimposed on facial erythema, sometimes with fever, and rosacea conglobata, when inflammatory cysts are observed on the face with subsequent scarring. However, the attribution of these symptom complexes to rosacea is controversial, and many authors consider them to be more related to ordinary acne. Persistent red-brown nodules on the face with a characteristic granulomatous histology without caseous necrosis are called granulomatous rosacea, while frequent flushing and easily irritated skin faces are classified as “prerosacea”. The inclusion of the above-mentioned heterogeneous spectrum of clinical manifestations in the characteristics of rosacea makes it difficult to understand the pathogenesis of this disease [4].

RESULTS AND DISCUSSION

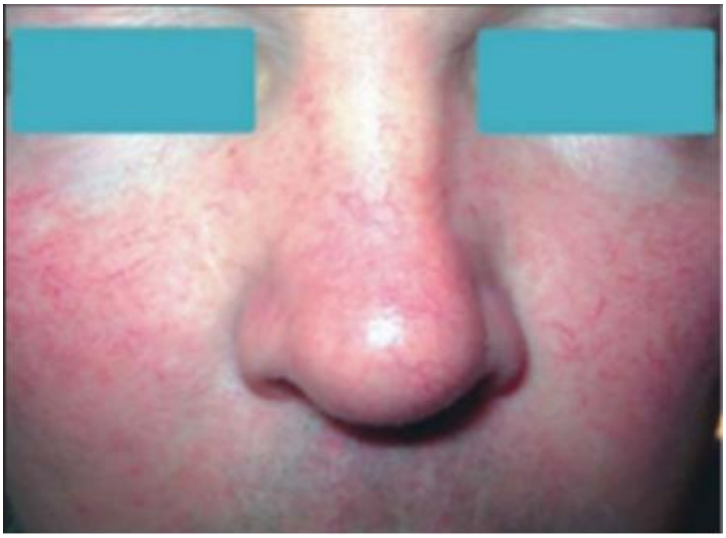
Table 1. Pathogenetic hypotheses of rosacea

Theory	Explanation

Abnormalities of the innate immune response	• The innate immune response protects against microbial infection without requiring specific recognition of the pathogenic irritant • Activation of innate immunity leads to the secretion of cytokines and antimicrobial molecules, for example cathelicidin peptide • In rosacea, there is increased regulation of cathelicidin and serine protease processing, which indicates dysfunction of the innate immune system
Ultraviolet radiation (UVR)	• It has been established that the impact of UVR: – stimulates angiogenesis – increases the production of chemically active oxygen particles, which leads to increased regulation of matrix metalloproteinases • Matrix metalloproteinases damage blood vessels and the main intercellular substance of the dermis
Vascular changes	• With rosacea, there is an increase in blood flow in the affected skin • Patients with rosacea blush more easily in response to heat compared to control groups • There is increased expression of VEGF* and markers of the endothelium of lymphatic vessels, which indicates stimulation of endothelial cells of blood and lymphatic vessels
Epidermal barrier dysfunction	• Increased transepidermal water loss • Reduced skin irritability threshold • Observed in both erythematous-telangiectatic and papulo-pustular forms of rosacea
Neurogenic inflammation	• Sensory nerves secrete neurotransmitters at sites of inflammation, leading to vasodilation, extravasation of plasma proteins and recruitment of inflammatory cells • The exact mechanisms are not fully understood
Microbes	• Demodex mites (folliculorum and brevis), which are commensals of normal skin, are found in large numbers in patients with rosacea • Demodex infestation is associated with a significant perifollicular infiltrate, predominantly with CD4+ T-helper cells • Presumably staphylococcal microflora plays a role in the pathogenesis of rosacea

The exact pathogenesis of rosacea is unknown. It will probably be established over time that the disease known today as rosacea includes several similar, possibly related, but different clinical conditions, each with an independent predominant pathogenetic mechanism. Some factors that

are significant for rosacea are listed in Table 1. Most often, rosacea is observed in people of the Caucasian race and in people with light skin that is sensitive to sunlight, i.e. with phototypes I and II. There may be a genetic predisposition to this disease, since 10-20% of patients report a family history of rosacea. Epidemiological studies indicate that erythematotelangiectatic rosacea may be associated with exposure to ultraviolet radiation and photodamage. It has been established that exposure to ultraviolet B radiation stimulates angiogenesis and increases the secretion of VEGF by keratinocytes.



Pic.-1 Erythematotelangiectatic rosacea

Patients with rosacea often report symptoms of burning, burning and dryness on the face. Studies have confirmed a reduced threshold of skin irritability in patients with rosacea. This may occur due to dysfunction of the epidermal barrier, which increases transepidermal water loss in patients with rosacea. In addition, it is assumed that damage or pathology of the stratum corneum leads to the penetration of sensory stimuli, which causes a burning sensation.

CONCLUSION

The conclusion is disappointing: there is no effective treatment for this pathology, although it has been reported that long-term courses of low-dose isotretinoin help some patients and that antihistamines for systemic use have a similar effect. Jansen and Plewig [5] proposed a treatment regimen including isotretinoin at a dose of 0.1-0.2 mg/kg per day for 2-4 months, which can be combined with ketotifen at a dose of 1-2 mg per day and an antihistamine means.

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