Practice of Medicine

Acne Vulgaris And Homoeopathic Approach

ACNE VULGARIS AND HOMOEOPATHIC APPROACH
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Abstract: Acne vulgaris is a disease of the pilosebaceous unit resulting from the interplay of different factors: seborrhea, P. acnes colonization, hyperkeratinization of the follicular duct and release of inflammatory mediators. Increased sebum lipogenesis by sebaceous gland is considered, among all features, the major one involved in the pathophysiology of acne.This article provide information about epistaxis along with homoeopathy.
Keywords: Acne vulgaris, hyperkeratinization , homoeopathy
Introduction: Acne is a chronic disease of the pilosebaceous follicle that causes polymorph cutaneous lesions, among them comedones (as a primary lesion), papules, cysts, pustules, and abscesses, which after regression may leave scars. It was thought that the primary change in the sebaceous follicle is the alteration in the pattern of keratinization within the follicle. Initial alteration is in the infrainfundibular portion where there is hyperproliferation. The keratin is also qualitatively altered as it tends to become densely packed along with monofilaments and lipid droplets
Propionobacterium acnes colonises the follicular duct and proliferates, breaking down the sebum to triglycerides, irritants that contribute to the development of inflammation. When the follicular epithelium is invaded by lymphocytes it ruptures, releasing sebum, micro-organisms, and keratin into the dermis. Neutrophils, lymphocytes, and foreign body giant cells accumulate and produce the erythematous papules, pustules, and nodular swelling characteristic of inflammatory acne.
Causes of Acne –
• Excess oil production

• Bacteria
• Excess activity of a type of hormone (androgens)
• Excess oil production
• Certain medications
• Stress
• Family history

Classification of Acne :

• Whitehead
• Blackheads
• Papules
• Cysts
• Nodular
• Pustules

Pathogenesis :

Acne vulgaris is a skin disorder of the sebaceous follicles that commonly occurs in adolescence and in young adulthood. The major pathogenic factors involved are hyperkeratinization, obstruction of sebaceous follicles resulting from abnormal keratinization of the infundibular epithelium, stimulation of sebaceous gland secretion by androgens, and microbial colonization of pilosebaceous units by Propionibacterium acnes, which promotes perifollicular inflammation.

The clinical presentation of acne can range from a mild comedonal form to severe inflammatory cystic acne of the face, chest, and back. At the ultrastructural level, follicular keratinocytes in comedones can be seen to possess increased numbers of desmosomes and tonofilaments, which result in ductal hypercornification.

The increased activity of sebaceous glands elicited by androgen causes proliferation of P. acnes, an anaerobe present within the retained sebum in the pilosebaceous ducts. The organism possesses a ribosome-rich cytoplasm and a relatively thick cell wall, and produces several biologically active mediators that may contribute to inflammation, for instance, by promoting leukocyte migration and follicular rupture. In inflamed lesions, numerous neutrophils and macrophages infiltrate around hair follicles and sometimes phagocytose P. acnes. To examine the participation of neurogenic factors in the pathogenesis of acne, we quantitatively assessed the effects of neuropeptides on the morphology of sebaceous glands in vitro using electron microscopy. Substance P, which can be elicited by stress, promoted the development of cytoplasmic organelles in sebaceous cells, stimulated sebaceous germinative cells, and induced significant increases in the area of sebaceous glands. It also increased the size of individual sebaceous cells and the number of sebum vacuoles for each differentiated sebaceous cell, all of which suggests that substance P promotes both the proliferation and the differentiation of sebaceous glands. In this review, we introduce the general concept of pathogenic factors involved in acne, including typical electron microscopic findings and recent evidence of stress-induced exacerbation of acne from a neurological point of view. An improved understanding of the pathogenesis of acne should lead to a rational therapy to successfully treat this skin disease.

Clinical features :

• Blackheads (black spots the size of a pinhead).
• Whiteheads (white spots similar to blackheads).
• Pustules (small pus-filled lesions).
• Redness and inflammation around eruptions.
• If acne is severe, cysts (larger, firm swellings in the skin), and abscesses (swollen, inflamed, tender area of infection containing pus)

General management

👇 Always face twice be gentle with your skin.
👇 Wash the face twice in a day and especially after sweating. Because sweat can make

acne worse.
👇 Keep your hands off your face.
👇 Let your skin heals naturally, if you pick or squeeze your acne, it increase the risk of getting acne scars.
👇 Rinse with luke warm.
👇 Avoid sunburn and suntan.
👇 Vitamin supplements: vit A and B6 are essential for skin.
👇 Exercise is also part of a natural acne healing process.
👇 Drink lots of water to clean your body off toxins and keep your skin hydrated form
Homoeopathic management:
1. Antim Crudum- Eczema with gastric derangements. Pimples, vesicles and pustules. Sensitive to cold bathing. Thick, hard, honey color scabes. Urticaria, measles like eruptions. Itching when warm in bed. Dry skin. Warts. Dry gangrene. Scaly, pustular eruption with burning and itching, worse at night.
2. Arnica Montana- Black and blue. Itching, burning, eruptions of small pimples. Crops of small boils. Ecchymosis. Bed sore. Acne indurate, characterized by symmetry in distribution.
3. Arsenicum Album- Dry, scaly, itching. Marked exfoliation of skin in large scales, leaving a raw exuding surface beneath. Itchthyosis. Enlarged scrofulous glands. Venereal bubo. Debilitating night sweats. Eczema of the beard. Watery, oozing, itching; worse, washing. Emaciation. Psoriasis. Acne hard, shotty, indurated base with pustules at apex.
4. Belladonna- Dry and hot. Swollen, sensitive; burns scarlet, smooth eruption like scarlatina, suddenly spreading. Erythema, pustules on face. Glands swollen, tender, red. Boils. Acne rosacea. Suppurative wounds alternate redness and paleness of the skin. Induration after inflammation.
5. Berberis aquafolium- pimply, dry, rough, scaly. Eruptions on scalp extending to face and neck. Acne. Dry eczema. Pruritus. .
6. Hepar Sulph- Abscesses; papules prone to suppurative and extend. Acne in youth. Suppurate with Prickly pain. Easily bleed. Unhealthy skin; every little injury suppurates. Chapped skin with deep crack on hand and feet. Wants to be wrapped up warmly. Putrid ulcers, surrounded by little pimples.
7. Kali bromatum-Acne of face, pustules, itching, worse on chest, shoulder and face. Anaesthesia of skin . psoriasis.
8. Kali arsenicosum –intolerable itching , worse undressing, dry ,scaly,wilted. Acne , pustules, worse during menses .
9. Ledum pal acne on forehead, sticking pain therein itching worse scratching and warmth of bed.
10. Lycopodium- Ulcerates. Abscesses beneath skin. Worse warm application. Hives, worse, warmth. Violent itching fissured eruptions. Acne, bleeds easily, thick and indurated. Brown
spots, freckles worse on left side of face and nose.
11. Natrum Muraticum- Greasy, oily esp. on hairy parts. Dry eruptions, esp. on margins of hairy scales and bends of joints. Fever blisters. Urticaria itch and burn. Itching after exertion
12. Nitric Acid- Warts, large jagged, bleed on washing. Ulcers bleed easily, sensitive, splinter like pain. Black pores on face, papules worse on forehead.
13. Sulphur- Dry, scaly, unhealthy; every little injury suppurates. Freckles. Itching, burning; worse scratching and washing. Pimply eruption, pustules, rhagades, hang nails. Excoriation eps. In folds. Skin affections after local medication. Pruritus esp. from warmth, in evening, often recurs in spring time, in damp weather.
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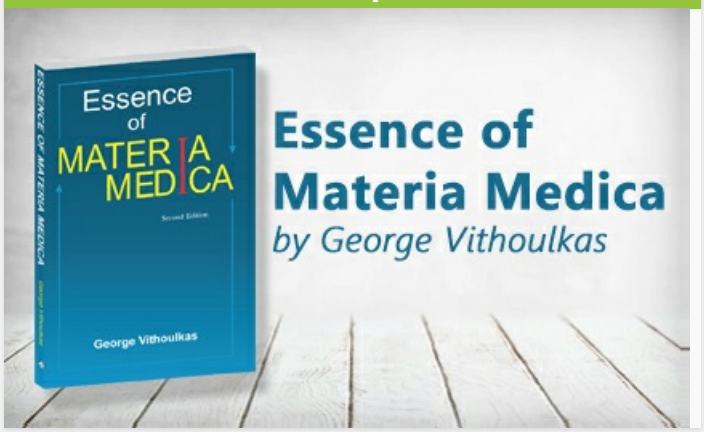

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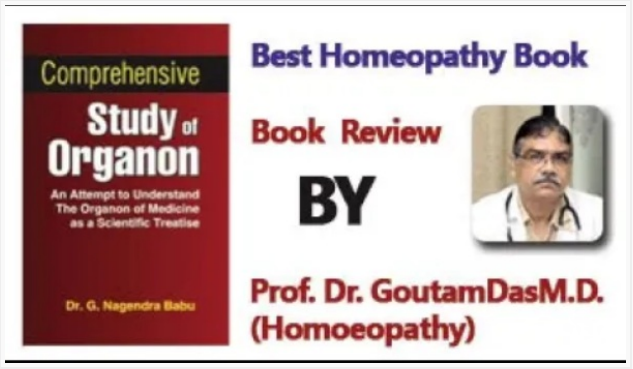

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