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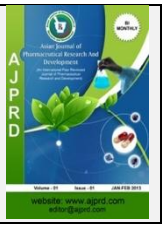

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Review Article

Review in Acne Vulgaris along with its treatment

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A B S T R A C T

Acne is a prevalent inflammatory skin condition that impacts the skin's sebaceous units. Both significant psychological effects and severe skin scarring may result from it for the patient.Acne is caused by four well-known pathological factors, which are the focus of acnetreatment. Several treatment options are covered in this review, including topical (retinoids and antibiotics) and systemic (retinoids, hormones, and antibiotics) treatments. Complementary and alternative medicine (CAM) was also suggested as a potential acne treatment since the general public has been expressing interest in safer and more natural treatment options. There is also mention of the application of physical therapies such cryoslush therapy, electrocauterization, comedone extraction, cryotherapy, intralesional corticosteroids, and optical treatments. Considerable study has been done on acne's underlying causes and available therapies.However, new therapeutic approaches are required since Propionibacterium acnes is becoming more resistant to the drugs that are already accessible. Researchers must also look at these therapy options more thoroughly because there is insufficient data to support the effectiveness of complementary and alternative medicine.

Keywords: adult acne, skin conditions, Propionibacterium acne, treatment, management

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INTRODUCTION

The skin makes up almost 15% of an adult's total body weight, making it the biggest organ in the body.The mucous membranes that line the body's surface form one continuous layer of skin. The skin and its derived structure make up the integumentary system. The epidermis, dermis, and subcutaneous tissue are the three layers that make up the skin. The outermost layer, the epidermis, is made up of a particular kind of cells called keratinocytes, which are responsible for producing the protective protein keratin, which is a long, thread-like strand.

Collagen, a fibrillar structural protein, is the main component of the dermis, the intermediate layer. The dermis is located on the panniculus, or subcutaneous tissue, which is made up of tiny fat cell lobes called lipocytes.[1]

Organization

Three layers comprise the skin.

The skin's outermost layer, the epidermis, influences skin tone and acts as a waterproof barrier.

Underneath the epidermis is the dermis, which is home to sweat glands, blood vessels, lymphatic vessels, hair follicles, and connective tissue. The hypodermis, or deeper subcutaneous tissue, is composed of connective tissue and fat.[2]

LAYERS EPIDERMIS-

Epidermis Layers

Stratum basale-The lowest layer is called stratum basale, or stratum germinativum..The cuboidal to columnar mitotically active stem cells that make up this layer are continuously generating keratinocytes. Melanocytes are also present in this stratum basale.The prickle cell layer, or stratum spinosum, is made up of eight to ten layers of irregular, polyhedral cells with cytoplasmic processes that jut outward and connect to other cells through desmosomes.

Stratum granulosum, which consists of three to five cell layers, has diamond-shaped cells that have lamellar and keratohyalin granules. Keratin precursors seen in keratohyalin granules eventually agglomerate, crosslink, and form bundles. The glycolipids found in the lamellar granules

are secreted onto the cell surface where they act as a glue to hold the cells together

Stratum corneum, consists of 20–30 layers of keratin and horny scales composed of dead keratinocytes, or anucleate squamous cells. The thickness of this layer fluctuates the most, notably in callused skin. The dead keratinocytes in this layer release defensins, which are a component of our first line of defense.

The epidermis' cells
Keratinocytes
Melanocytes
The Langerhans cells
Merkel's cellular.[4]

HYPODERMIS

Hypodermis The hypodermis is the subcutaneous layer located beneath the dermis and is primarily composed of fat. It serves as the skin's primary structural support while also insulating the body from cold and aiding in shock absorption. It is intricately linked with blood vessels and nerves.[33]

ACNE VULGARIS

Acne vulgaris, also referred to as acne, is a skin disorder that affects people and is characterised by scaly red skin (seborrhea), comedones, blackheads, papules, big papules (nodules), pimples, and scarring . Skin with dense sebaceous follicles, such as that on the face, chest, and back, is affected by acne.[5]There are visible lesions on the chest, upper back, neck, and face. There are various types of acne, including drug-induced excoriated acne, acne conglobata, acne fulminans, acne mechanica, and acne in newborns and infants.[6] Seborrhea (excess grease), non-inflammatory lesions (open and closed comedones), inflammatory lesions (papules and pustules), and varying degrees of scarring are among the clinical characteristics of acne.[7].:

PATHOGENESIS

A number of host variables, such as the activation of sebaceous glands by circulating androgens, dysbiosis of the pilosebaceous follicle microbiome, and cellular immunological responses, interact in the development of acne vulgaris[8].Sebum overproduction is the result of excessive androgen Hormones or a heightened sebaceous gland sensitivity to Normal levels of androgen hormones[9].Acne can occasionally indicate hyperandrogenism. When female teenagers or adults with acne come in, it's important to always inquire about hirsutism, irregular menstruation, and unexplained weight gain. Then, a polycystic ovary syndrome evaluation can be required[10].The recruited inflammatory cells subsequently release pro-inflammatory cytokines, like IL-1, IL-8, IL-12, and defensins, which cause inflammatory papules, pustules, and, in extreme cases, cysts and nodules.[11]

There are treatments for acne that successfully inhibit the overproduction of sebum, the aberrant desquamation of sebaceous follicle epithelial cells, and the growth of P. Acnes.[12]

Within acne-infected hair follicles, at least four pathophysiologic events occur: androgen-mediated stimulation of sebaceous gland activity; aberrant keratinization resulting in follicular plugging, or the formation of comedones); Propionibacterium acnes proliferates within the follicle; and inflammation. Apart from these four fundamental methods, genetic factor also included [13].The initial morphological alteration associated with acne vulgaris is aberrant follicular epithelial differentiation in the pilosebaceous unit.8 Desquamated cornified cells in the follicle's upper canal exhibit abnormally high levels of adhesion; rather of going through the regular shedding and discharge process through the follicular orifice, these cells form a microscopic hyperkeratotic plug that is maintained in the follicular canal, known as the microcomedo. Although the exact mechanism underlying this process, sometimes called comedogenesis, is unknown, a linoleic acid follicular deficit may be involved.9. Comedones are non-inflammatory acne lesions that become clinically evident as a result of the progressive enlargement of microcomedones[14].

TYPES OF ACNE

Blackheads: are open pimples on the skin that accumulate dead skin cells and extra oil. The dark patches, which give the impression that debris has accumulated in the bump, are actually the result of uneven light reflection off the obstructed follicle.

Whiteheads: Pimples that are sealed shut by dead skin and oil.

Papules: Tiny, inflammatory red or pink pimples.

Pustules are pus-filled pimples. They resemble whiteheads with red circles around them. If they are scratched or plucked at, they may leave scars.

Pityrosporum folliculitis (fungal acne):often known as fungal acne, is a condition in which the hair follicles become overabundant with yeast. They may become irritated and scratchy.

Nodules: Firm zits located deep within the skin. They hurt and are big

Cysts: pimples packed with pus. These cause scars [15]

Occurs on the face, neck, chest, upper back, and upper arm regions of affected persons, where there are a lot of big, hormone-responsive sebaceous glands. From grade 1 to grade 4, acne manifests as a range of polymorphic lesions, the first of which are comedones, as follows:

Grade 1: Also referred to as "comedones," this group is divided into two categories: open and closed. When sebum clogs the pilosebaceous orifice, papules with a central, dilated follicular aperture filled with grey, brown, or black keratotic material are known as open comedones. Conversely, sebum and keratin obstruct the pilosebaceous orifice beneath the skin's surface to generate closed comedones.

Grade 2: Small papules with erythema are indicative of inflammatory lesions.

Grade3: pimples.

Grade 4: Known as nodulocystic acne, a large number of pustules combine to create nodules and cysts. [16]

TYPES OF SCARS

Table 1: Various types of scars and their characteristics [17].

Scars	Characteristics
Box car scars	Similar to chickenpox scars, angular scars can be deep or minor and appear on the cheeks.
Ice pick scars	Deep pits are most common sign of Ice pick scars
Rolling scars	Wave like appearancein the skin
Hypertrophic scars	Thickened or keloid scars
Pigmented scars	True scars, change in the skin's pigmentation as a result ofxVÀg nodular cystic acne , inflamed red mark

An altered wound healing response to cutaneous inflammation causes acne scars; in approximately 80% of atrophic scars, inflammatory cell infiltrates are the cause of the scars. Papules, pustules, and post-inflammatory lesions are the primary causes of almost all scars (99%).Variations in acne severity are partly explained by the variable activation of epidermal innate immunity by distinct P. Acnes phylotypes. On the other hand, fewer skin-homing CD4+ T-cells are present in early lesions in patients who are more likely to scar than in individuals who do not, a response that intensifies as lesions heal.[16]

ETIOLOGY

Diet: Acne and food are not well understood, yet a high-glycemic diet is linked to acne exacerbation. There is a direct correlation between rising acne prevalence and milk consumption. Studies revealed that eating chocolate and salt did not contribute to the development of acne. Chocolate has a high glycemic load due to its high sugar content. There's a chance that insulin metabolism and obesity are related to acne. [b]

Personal factors: Acne connected to certain personal characteristics, such as body mass index (BMI), alcohol consumption, and smoking status, to the appearance of acne. The majority of studies have found that the prevalence of acne is higher in overweight and obese people (defined as BMI ≥23 kg/m2 and ≥25 kg/m2, respectively) than in underweight people (BMI <18.5 kg/m2) or people with a normal weight (18.5 kg/m2≤BMI<23 kg/m2). The obtained OR of 2.36 (95% CI 1.97–2.83), which compares overweight/obese BMI to normal/underweight BMI, indicates that acne presentation is significantly influenced by BMI.[18].

Hormones. Acne may result from an increase in androgens, or male sex hormones. During puberty, these naturally rise in both boys and girls, causing the sebaceous glands to grow and produce more sebum. Acne can also result from pregnancy-related hormonal changes.[19]

Genetics:Acne susceptibility genetics is polygenic, meaning the disease does not follow a traditional Mendelian inheritance pattern. Polymorphisms in Tumor necrosis factor-alpha, Interleukin-1 alpha, and CYP1A1 are among the candidates for genes linked to acne .[17]

Stress: Stress before exams can exacerbate acne, according to a research by Chin et al. Stress, however, has no influence on excessive sebum production. About 74% of 178 acne cases in a survey by Smith et al. indicated that stress was a contributing cause to their acne. It's a never-ending cycle where stress leads to acne, which in turn creates acne.[21].

TREATMENT:

Oral isotretinoin.

Accutane®, a naturally occurring metabolite of Vitamin A, is prescribed to treat severe acne. Isotretinoin reduces the size of the sebaceous gland, suppresses sebum production, and restores follicular epithelial desquamation. Isotretinoin has been shown in multiple studies to effectively treat severe acne. Treatment dosages varied from 0.1 to 0.5 mg/kg/day. The 1.0mg/kg/day dose resulted in an 89% reduction in total lesions.[9].Isotretinoin can cause dose-dependent side effects such as xerosis, cheilitis, acne flare-ups, dry eyes, headaches, and increased lipid and hepatic enzyme levels.[23]

Topical retinoids:

They are a variety of vitamin A compounds that influence gene expression. Topical retinoids approved by the US FDA for acne vulgaris treatment, including as adapalene, tretinoin, and tazarotene, regulate keratinocyte proliferation and differentiation, preventing comedone formation and providing anti-inflammatory benefits. Topical retinoids are the most effective treatment and maintenance for acne, reducing comedonal and inflammatory lesions.They also prevent and minimise the appearance of atrophic scars and dyspigmentation.[24]While topical retinoids may cause photosensitivity, using sunscreen on a daily basis can lessen the risk of sunburn. Adapalene 0.1% gel is accessible over-the-counter, but other topical retinoids require a prescription.[25]

Azelaic acid.

Azelaic acid is a naturally occurring saturated C9-dicarboxylic acid that has been demonstrated to be beneficial in the treatment of comedonal acne, inflammatory acne, and hyperpigmented skin problems. It is an acne treatment that

works by blocking the thioredoxin reductase enzyme in Propionibacterium acnes, which inhibits bacterial DNA production in the cytoplasm. Azelaic acid (20% cream or 15% gel) is indicated as the first line of treatment in monotherapy for both non-inflammatory and inflammatory acne, used twice daily.Azelaic acid 15% foam is effective and safe for treating face acne vulgaris.[26]

Salicylic Acid

Salicylic acid possesses bactericidal and keratolytic effects, which help reduce acne. Salicylic acid opens skin pores and sheds epithelial cells, but can induce hyperpigmentation in darker skin types.[10]

Benzoyl Peroxide

Benzoyl peroxide (BP) is commonly Prescribed topical medication for AvReduces P. Acnes colonisation and inflammation in acne lesions. It possesses keratolytic and sebostatic actions without the risk of developing drug-resistant germs. Benzoyl peroxide, a bactericidal agent with a stable formulation, effectively treats comedonal acne. The concentrations range from 2.5% to 10%. The Food and Drug Administration (FDA) has classified BP as Category C pregnancy risk.[7].

Topical antibiotics

Topical antibiotics (usually clindamycin or erythromycin) are useful for mild pustular and inflammatory acne. Antibiotics have anti-inflammatory qualities in addition to being antibacterial. They are often used in conjunction with retinoids (ZIANA) or benzoyl peroxide (Acanya and BenzaClin was). Clinical investigations have shown that topical clindamycin can be equally effective as oral antibiotics when used twice-daily.7,8 Clindamycin gel can be drying, and no visible residue on dry[16].

Acne fulminans.

Acne fulminans is an uncommon type of severe cystic acne. The condition is characterised by the abrupt emergence of large, tender inflammatory nodules and plaques on the trunk (Fig. 8). Systemic symptoms such as fever, arthralgias, anorexia, leukocytosis, and localised lytic bone lesions are common, although the face is rarely affected.[5]

Pyoderma faciale

(also known as rosacea fulminans) appears suddenly on the midface of young women. It may be similar to acne fulminans. The eruption comprises of erythematous plaques and pustules over the chin, cheeks, and forehead. Papules and nodules can form and become confluent[17]

Acne tropicalis

Acne tropicalis is an unusually severe acne eruption that occurs in older patients who may have had Acne vulgaris at an earlier age. It occurs in the tropics during seasons when the weather is hot and humid. This form of acne typically occurs on the back And buttocks, and has been especiallyCommon in soldiers stationed in the tropics who Carry backpacks.[18]

Acne conglobata

It is a rare but severe kind of nodulocystic acne in young men. It typically manifests as sensitive, disfiguring comedones, cysts, inflammatory nodules, and deep burrowing abscesseson the face, shoulders, back, chest, upper arms, buttocks, and thighs. Systemic signs are typically absent.

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