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Cystic Achne Vulgaris: A Review Study

Keerthika .S[1], Dr. Brundha[2]

[1]IIBDS, Saveetha dental college, 162, P.H. Road, Chennai, Tamilnadu 6000077, India
[2]Department of general pathology, Saveetha dental college, 162, P.H. Road, Chennai, Tamilnadu 6000077, India

Abstract: ***Aim:*** ***To review etiopathogenesis and treatment options available with us in present scenario.*** ***Background:*** ***Acne vulgaris is one of the commonest skin disorders, which dermatologists have to treat, mainly affect adolescents though it may present at any age. Acne is chronic inflammatory disease of pilosebaceous units. Clinically it can present as seborrhea, comedones, erythematous papules, pustules and nodules.In recent years, due to better understanding of etiopathogenesis of acne, new therapeutic modalities are designed.*** ***Reason:*** ***The purpose of this article is to determine possible factors of cystic acne vulgarisms.***

Keywords: acne, skin, disorders

1. Introduction

The term acne is derived from Greek word "acme" which means "prime of life"[1].Acne vulgaris (AV) is a chronic inflammatory disease of the pilosebaceous unit. Acne begins to develop at the time of adrenarche when the adrenal gland starts to produce large quantities of dehydroepiandrosterone sulfate, a precursor for testosterone. Acne begins to develop at the time of adrenarche when the adrenal gland starts to produce large quantities of dehydroepiandrosterone sulfate, a precursor for testosterone[2]. Acne can also affect neonate, infants and children. Acne neonatorum occurs in up to 20% of newborns. Moreover acne are mostly pleopmorphic, manifesting with a variety of lesions consisting of comedones, papules, pustules, nodules, and pitted and hypertrophic scars[3].Acne is characterised by areas of black heads, white heads, pimples, greasy skin and possibly scarring. Though acne is not life threatening condition it causes detrimental effects such as development of anxiety, reduced self esteem, depression or thoughts of suicide. This disease occurs both in males and females but the course is longer in males. It usually involves face but may also affect back and chest of the individual.[4]Acne vulgaris, the most common skin disorder, affects virtually all individuals at least once. Incidence peaks in 18-year-olds, but substantial numbers of 20- to 40-year-olds also develop the disease. The effects of acne should not be underestimated. It can persist for years; produce disfigurement and permanent scarring; and have significant psychosocial consequences, including diminished self-esteem, embarrassment, social withdrawal, depression, and unemployment.The extent and severity of these effects underline the importance of providing adequate therapy, which produces satisfactory results in most cases[5].

Etiology of Acne Vulgaris:

Acne vulgaris is multifactorial in origin involving both endogenous and exogenous factors. There are four primary pathogenic factors, which interact in a complex manner to produce acne lesions.

- Increased production of sebum by the sebaceous gland.
- Changes in keratinization process.
- Follicular colonization caused by propionibacterium spp.
- Release of inflammatory mediators

Others contributing factors include hormonal influences from estrogen and androgens, such as DHEAS (dehydro-epiandrosterone sulfate), which is responsible for increased sebum production in prepubescent children, leading to acne [6].

Seborrhea (increase in sebum secretion) and sebaceous gland hypertrophy and hyperplasia are the hallmarks of acne. The changes in the sebum production may cause irritation of the duct epithelium. squalene and wax esters are found in high levels in the sebum acne patients. Reduced levels of linoleic acid in sebum can lead to acne vulgaris by promoting the accumulation of cornified cells[7]. Ductal hypercornification is seen histologically as microcomedones, which are initial Lesions of acne. The stimulus to the hyperkeratosis of duct epithelium may be androgens, irritating effect of sebaceous lipids as they pass through the duct. The acceleration in the rate of sebum secretion and also its composition may irritate the infundibular keratinocytes, leading to release of inflammatory substance like IL-1α, this in turn causes reduction of sebaceous linoleic acid and 5α-reductase enzyme levels leading to induction of follicular hyperkeratosis. Comedogenesis occurs when abnormally desquamating corneocytes accumulate within the sebaceous follicle and form a keratinous plug [8]. It blocks the follicular ostium at the skin surface and becomes visible as closed comedone (white head). Follicular ostium dilation of follicular osmium causes open comedone filled with debris.

It was found that propionibacterium species (P. acne, P. granulosum) etc. are a major factor in the pathogenesis of acne. P. acne is a common skin resident and one of the major components among the microbial flora of the pilosebaceous follicle. These resident bacteria produce more lipases which are responsible for hydrolysis of triglycerides to free fatty acids and also follicular hyperkeratosis and even rupture of follicle.

Inflammatory process is a key component of acne.This is largely responsible for its morbidity and sequlae. Perifollicular T- cells are involved in genetically initiating the immunological events predisposed individuals, comedogenesis through release of IL-1 [9]. In addition, ductal corneocytes also produce IL-2, IL-8 and TNF- α..

Androgens increase sebum secretion and also cause sebaceous gland hyperplasia. Estrogen, suppresses sebaceous gland activity. There is a indirect relationship between acne severity and serum sex hormone binding globulin (SHBG) levels. Peripheral hyper-androgenism in many cases may balances with severity of acne in women, and can guide appropriate hormone therapy in most of the population. A hot and humid climate aggravates acne due to increased sweating leading to ductal hydration. Emotional stress also plays a significant role in the aggravation of the pre-existing acne. External application of oils, pomades, and other comedogenic chemicals eruptions Causes acneiform.A high glycemic diet induces hyper-insulinemia which results in androgen synthesis, predisposing to poly cystic ovarian disease (PCOD). Diet induced hyper-insulinemia also increases level of IGF-1 (Insulin Like growth factor) and reduce IGF binding proteins. The increased free IGF-1 level results in unregulated growth of follicular epithelium, increased sebum production and synthesis of androgens from gonads[11].

Dispelling Misconceptions in Acne:

- Acne is not a disease of poor hygiene. acne has no relation with cleanliness. The black tip of a comedo is oxidised sebum, not dirt, and it cannot be removed by scrubbing. Vigorous washing may actually make things worse[12].
- Also, diet has never been shown to have much effect on acne. patients need to understand that topical treatments prevents only new lesions, not shrinking ones that have already formed. Thus, the treatment should be applied faithfully to all skin that may be affected, not just to visible lesions[13].

Presentations of acne vulgaris:

- Acne usually presents with a greasy skin with a mixture of comedones, papules and pustules. It begins just after puberty and continue for a variable number of years, usually stopping in late teens or early 20s but uncommonly continuing well into adulthood.
- The face is affected in 99% of cases but also occurs in back and chest to a lesser extent.
- Nodulocystic acne: severe acne with cysts. Cysts can be painful. They may occur in isolation or be widespread over the face, neck, scalp, back, chest and shoulders.
- The severity of the condition varies enormously between individuals. But the degree of distress is sometimes disproportionate[14.]

Differential Diagnosis:

Although most acne diagnoses present no difficulties. However certain conditions may be confusing. These includes rosacea, ulery- thema ophryogenes, gram-negative folliculitis, steatocystoma multiplex, steroid acne, drug erup- tions, perioral dermatitis, iododermas, verruca vul- garis, verruca plana, bromodermas, syringomas, sarcoidosis, trichoepitheliomas, follicular muci- nosis, angiofibromas, infectious folliculitis, and keratosis pilaris[15].

Management of cystic acne vulgaris:

1)General Measures
2)Specific Measure

General measures:

- Elimination of stress by reassurance.
- Providing counselling to patient regarding nature of illness, treatment modalities and its outcome.
- Avoid scratching of lesions.
- Assessment of the endocrinal status and premenstrual flares.
- Avoidance of acnegenic drugs, oils, pomades and heavy cosmetics.
- Avoidance of high glycemic diet.
- Washing of face with soap and water periodically.

Specific measures:

- Decreasing secretion of sebaceous gland
- Modification of ductal hypercornification
- Decreasing P. acne population and associated flora.

Topical therapy:

Topical retinoids:

Retinoid preparations available are

- Tretinoin: 0.025%, 0.05%, 0.1% gel /cream.
- Isotretinoin: 0.05% gel.
- Adaplene: 0.03%, 0.1% gel.
- Tazarotene: 0.1%and 0.05% gel.
- Topical retinoids decreases the number and formation of precursor lesions; reduce mature comedones and inflammatory lesions. The main disadvantage of these these agents is primary irritant dermatitis which can present as erythema, scaling, and burning sensation and can vary depending on the skin type, sensitivity and formulation16.
- Benzoyl Peroxide: It is an efficient topical retinoids and an essential component in gel, cream or lotion in a strength varying from 2.5 to 10%. It is a broad-spectrum antimicrobial agent effective with oxidizing activity. It has anti- inflammatory, keratolytic and comedolytic activities. It is indicated in mild to moderate acne.
- Topical antibiotics such as picolinic acid, salicylic acid, tea tree oil can also be used for management of cystic acne vulagaris.

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