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ACNE VULGARIS AND ITS TREATMENT A REVIEW

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ABSTRACT: Acne vulgaris is nothing but it is a skin condition that affects the sebaceous follicle. It begins to occur shortly just after the puberty and can last for up to 40-50 years. Up till now, the pathophysiology of acne has remained a mystery. Several anti-acne agents are currently available that affect one or more of these pathogenic factors and are effective against one or more acne lesion types. Acne treatment is difficult and necessitates an awareness of etiopathological variables. Treatment of acne vary according to the patients and the disease conditions they have. Treatment of acne in adult women specifically has its challenges due to the considerations of patient preferences, pregnancy, and lactation. Agents effective against inflammatory lesions include tretinoin, benzoyl peroxide, and topical and systemic antibiotics. Agents effective against nodules and cysts include oral antibiotics and isotretinoin. However, the successful utilization of the available agents and techniques is highly dependent on an accurate and thorough assessment of each patient's needs and concerns, followed by the implementation of an individualized treatment program that has been clearly communicated to the patient. Now a days Novel drug delivery system is used for the treatment of acne. This article examines the numerous elements that contribute to acne's occurrence, etiology, and treatment.

INTRODUCTION:

Acne is regarded as one of the most common skin disorders [1]. When young people experience significant disfigurement, it can lead to serious effects such as depression and suicide. Among skin illnesses, acne vulgaris is the second most common cause of suicide. When a person with acne is compared to a person who does not have acne, it is discovered that the former has a higher level of anxiety, more socio inhibition, and is more aggressive [2]. Acne is a skin ailment that arises when the sebaceous glands (SGs) reach a certain level of activity. Males and females are both affected by this condition; there is no gender preference, but males' symptoms are more severe [3].

EPIDEMIOLOGY:

Acne affects 9.4 percent of the population, according to statistics from 2010 [72]. It affects over 90% of persons during their adolescent years and occasionally into adulthood [5]. Moderate and severe instances affect about 20% of the population. Acne is uncommon in rural places, and it may not exist in non-westernized Paraguayans and Papua New Guineans [7]. Females have a higher prevalence of 9.8% compared to men' 9.0% [72]. Approximately 1% of males and 5% of females in over 40-year-old subjects experience issues [5]. It affects persons of all ethnic groups, and it is unclear whether race has an impact on disease rates [73,74]. Acne affects 40 to 50 million people in the United States, or around 16 percent of the population, and 3 to 5 million people in Australia, or about 23 percent of the population [75]. It is more severe in Caucasians than in people of African heritage in the United States [6].

Types of Acne:

Blackheads: Open bumps on the skin that fill with excess oil and dead skin. They look as if dirt has deposited in the bump, but the dark spots are actually caused by an irregular light reflection off the clogged follicle.

Whiteheads: Bumps that remain closed by oil and dead skin.

Papules: Small red or pink bumps that become inflamed.

Pustules: Pimples containing pus. They look like whiteheads surrounded by red rings. They can cause scarring if picked or scratched.

Fungal acne (pityrosporum folliculitis): This type occurs when an excess of yeast develops in the hair follicles. They can become itchy and inflamed.

Nodules: Solid pimples that are deep in your skin. They are large and painful.

Cysts: Pus-filled pimples. These can cause scars.

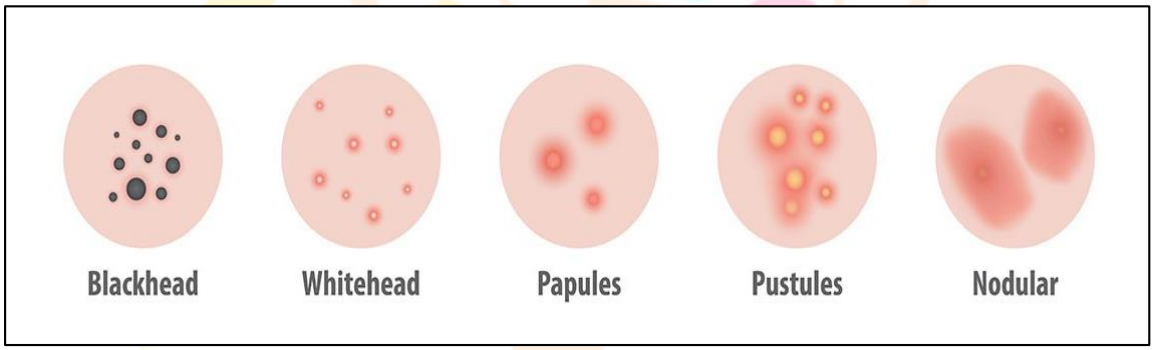

fig. 1: types of acne vulgaris

ETIOLOGY OF ACNE

SG produces more sebum, hypercornification of the sebaceous ducts, colonisation of Propionibacterium acnes in the pilosebaceous ducts, and inflammation are all factors that might lead to acne. Acne severity is linked to seborrhea, which is linked to follicular infundibulum. Hypercornification, hyperkeratinization, and hypodesquamation of keratinocytes of the infundibulum occur in milder acne, resulting in the formation of comedones. In severe acne, the infundibulum ruptures, releasing sebum into the dermis, triggering inflammatory reactions.

Environmental Factors

It includes various factors like High-humidity, Prolonged sweating, Increase in skin hydration, Exposure to dirt or vaporized cooking oil or certain chemicals like petroleum derivatives.

Drug Use

Drugs like Phenytoin, Isoniazid, Phenobarbital, Lithium, Ethionamide, Steroids, Azathioprine, Quinine and Rifampin causes acne [4].

Hormonal:

Acne can also be caused by menstrual cycles and puberty. Increased androgen levels drive follicular gland growth, as well as increased sebum production, throughout puberty [6, 8]. The effects of anabolic steroids are similar [9]. The androgens testosterone, dihydrotestosterone, dehydroepiandrosterone sulphate, and insulin-like growth factor 1 (IGF -I) are all connected to acne. Acne vulgaris is less common in later years, while rosacea, which has comparable symptoms in older age groups, is becoming more common.

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 [10].

Psychological:

Increased stress levels have been linked to increased acne severity in studies [11]. According to the National Institutes of Health (USA), stress can promote acne flare-ups [12]. A study of adolescents in Singapore discovered a link between stress levels and acne severity [13].

Infectious:

Propionibacterium acnes (P. acnes) is an anaerobic bacterium that is primarily responsible for acne. Since normal pores are solely colonised by Propionibacterium acnes, Staphylococcus aureus has been discovered to play a crucial function [14]. P. acnes clonal substrains have also been linked to both normal skin health and long-term acne problems. These strains have the ability to change, perpetuate, or adapt to the aberrant cycle of acne pore inflammation, oil production, and insufficient sloughing activity. One virulent strain of Propionibacterium acnes has been circulating throughout Europe for at least 87 years [15]. In vitro, antibiotic resistance to P. acnes has been steadily growing [16].

Diet:

Although a high-glycemic diet has been linked to acne worsening [17–19], the association between acne and food is yet unknown. There is a link between milk consumption and an increase in the prevalence of acne [18, 20, 21]. Consumption of chocolate and salt is not linked to the development of acne, according to studies [18]. Chocolate includes a lot of sugar, which can cause a high glycemic index. It's probable that acne has something to do with fat and insulin metabolism [22].

PATHOGENESIS:

Seborrhea causes an increase in androgen concentration due to genetic reasons as well as puberty, which leads to an increase in sebum production. The production of androgen in the body increases during puberty. Androgens are produced, as well as reuptake, in the sebocyte. Within the cytoplasm, these androgens form an androgen-receptor complex. These then reach the nucleus via nucleopore, altering the specific gene sequence and so affecting the reading rate, causing the sebocyte to produce more sebum. Sebum is created in this way and flows through the pilosebaceous ducts to the skin's surface. The pilosebaceous ducts carry the Produced sebum to the skin's surface. During the flow, this sebum delivers linoleic acid to the hair follicle's keratinocytes. As a result, there is a local linoleic acid shortage, which compromises the follicular barrier. This permits P. acnes' free fatty acid to enter the follicle through the activity of its enzyme lipase or other processes on triglycerides. The follicular wall can potentially be damaged as a result of oxygen stress or the production of free radicals by phagocytes in reaction to invading microbes. The free fatty acids that are consumed are highly chemotactic, resulting in the generation of cytokines such as IL-8 and IL-1, which cause inflammation and keratinocyte proliferation to increase. This causes hypercornification of the ducts and the production of thick horny lamellae. As a result, retention-proliferation hyperkeratosis develops. Retention-proliferation hyperkeratosis produces microcomedone, which grows and transforms into a comedone, which then develops into acne [23-53,54].

TREATMENT OF ACNE:

Treatment of mild acne:

The major treatment method is to use a topical preparation, and the preparations that can be used to treat mild acne are shown in Fig. 2.

The above-mentioned treatments are the most popular among patients, but they require clear instructions on how to use them in order for patients to comply. Furthermore, they are long-acting helpful medications [55].

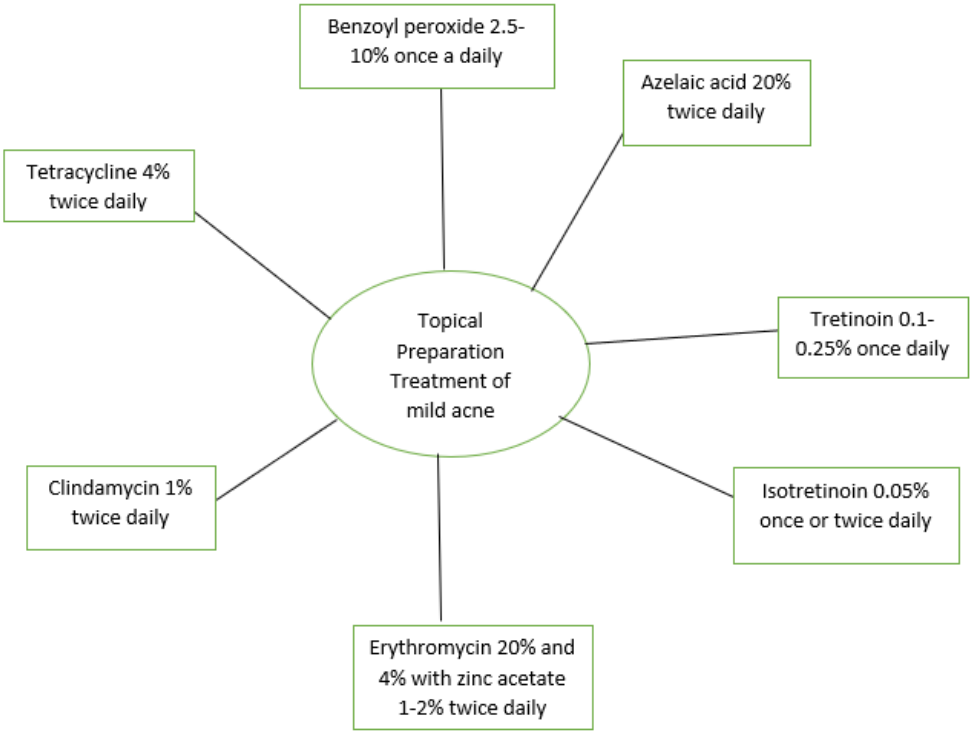

fig. 2: treatment of mild acne

Benzoyl Peroxide:

It's a potent oxidizer with antibacterial and keratolytic characteristics. Benzoyl peroxide does not cause any changes in the resistance pattern of aerobic bacteria to antibiotics, but when taken in conjunction with erythromycin topical formulation, it can prevent such resistance.

It comes in the form of lotions and creams with a concentration of 2.5-10% on the market. They are only to be used once a day. There isn't a dose-response profile that can indicate how efficacy increases with greater doses. The most common side effects of benzoyl peroxide include transitory skin irritation, allergic contact dermatitis, and clothing whitening. They can be used to treat moderate A. vulgaris for a long time or in combination with oral antibiotics [56].

Azelaic Acid:

It is keratolytic in nature and can induce local irritation and photosensitization, as well as a change in the composition of free fatty acids in skin surface lipids. It can also drastically lower follicular bacterial density. The duration of treatment is usually 6 months. It comes in a 20 percent concentration and can be taken twice a day [57].

Topical Retinoid formulation:

They are beneficial in the treatment of both mild and moderate acne. It's best to use it once or twice a day. Tretinoin comes in the form of a cream or a lotion. As a gel formulation, isotretinoin is used. Comedolytic action is present in both formulations. Desquamation, occasional hyperpigmentation or hypopigmentation, erythema, and skin reactivity to sunlight are some of the side effects. It reveals that infants born to women who used topical retinoids during early pregnancy have malformations [58,59].

Topical Antibiotics:

Antibiotics are utilised in more severe cases because of their antibacterial and anti-inflammatory capabilities against P. acnes. They are becoming less effective as P. acnes resistance grows over the world [6, 60]. Topically applied or orally administered antibiotics such as clindamycin, erythromycin, and tetracyclines such as doxycycline, oxytetracycline, lymecycline, and minocycline are used to treat acne.

Treatment of Moderate Acne:

Oral Antibiotics:

The use of systemic antibiotics to treat the condition is still the most common therapeutic option, with tetracycline being the primary choice. Oral antibiotics should be taken half an hour before a meal to ensure adequate absorption, and patients should avoid taking iron supplements or drinking milk at the same time. The development of resistance to erythromycin by P. acnes and Staphylococcus epidermidis is a limiting issue for its use in treatment. Minocycline is the most commonly recommended systemic antibiotic for acne therapy. Clinically, there is a modest difference between minocycline and tetracycline because minocycline requires less dietary restriction. As an alternative, doxycycline or trimethoprim can be utilised. When high doses of minocycline are administered, adverse medication reactions linked with oral antibiotics include gastrointestinal distress, vaginal candidiasis, and hyperpigmentation [61].

Hormonal Treatment:

Acne is caused by the inflammation of the SGs. There is minimal evidence to suggest any hormonal disruptions in girls with acne, although 46 percent of women with acne between the ages of 18 and 32 have little rise in testosterone levels and also have inhibition of sex hormone binding globulin [62]. Cure can be achieved with antiandrogens such as cyproterone acetate 2 mg in combination with ethinyl estradiol 35 mg, which is as effective as oral tetracycline but takes 3-6 months to work [63]. Cyproterone acetate (50 or 100 mg) from days 5 to 15 of the menstrual cycle, along with 35 mg of ethinyl estradiol from days 5-26 of the menstrual cycle, has been shown to have greater positive benefits [64].

Combined contraceptive tablets containing norethisterone or levonorgestrel, for example, may worsen acne, but this is not true in other situations, such as those containing desogestrel or gestodene [65].

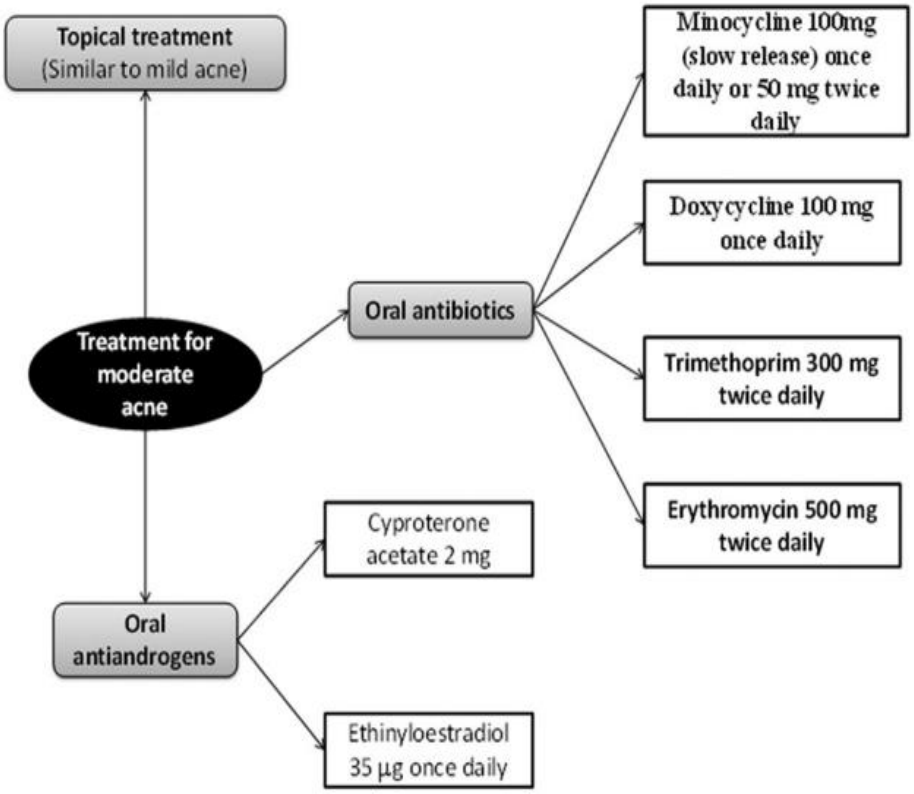

fig. 3: treatment of moderate acne

Treatment of severe acne:

Isotretinoin:

Isotretinoin reduces SG activity, which may lead to a reduction in sebum production, resulting in a significant drop in the population of P. acnes. The amount of retinol in the body increases cutaneous region and could indicate a metabolic conflict with vitamin A produced by the body [66-68]. The majority of patients require treatment for at least four months, although 15% of cases require even more time [69]. In total, 40% of patients are cured and do not require further therapy, while the remaining 21% require only topical treatment. Relapse occurred in 39 percent of those on the left within three years, and in 39 percent of those on the right after three years. Oral antibiotics are required in 16 percent of cases, while isotretinoin is required in 23 percent of cases [70]. Despite its high price, isotretinoin is a relatively inexpensive way to treat moderate to severe acne when compared to alternative antibiotics [71].

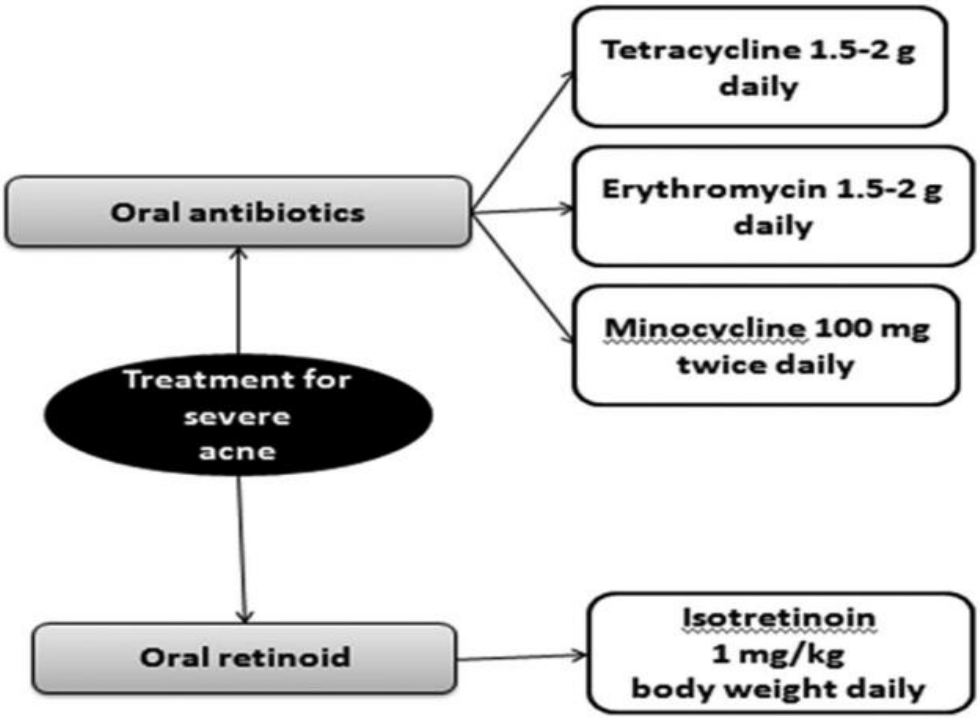

fig.4: treatment of severe acne

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