A Short Review on Acne Vulgaris and Its Treatment

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Abstract- Acne is a skin infection caused by changes in sebaceous glands, with the most common form being acne vulgaris. It is characterized by scaly red skin, blackheads and whiteheads, pinheads, large papules, pimples, and scarring. Acne is a pleomorphic disorder that can manifest at any time during life, but is most common between ages 12-24, affecting 85% of the population. It affects dense sebaceous follicles in areas like the face, chest, and back. Acne can be inflammatory or non-inflammatory and is mainly caused by an increase in androgens like testosterone during puberty. There are two types of lesions: non-inflammatory, open and closed comedones, as well as inflammatory papules, pustules, nodules, and cysts. Treatment for acne requires patience, proper care, and perseverance. It may take two or three months for treatments to work effectively, and it is important to allow ample time for each regimen or drug to work before proceeding with other treatments.

Keywords: Common Acne, Sebaceous Glands, Acne Vulgaris, Acne, Skin.

Introduction

Acne is an infection of the skin, caused by changes in the sebaceous glands. The most common form of acne is called acne vulgaris, which means "common acne". The redness comes from the inflammation of the skin in response to the infection. (1) Acne vulgaris or simply known as acne is a human skin disease characterized by skin with scaly red skin (seborrhea), blackheads and whiteheads (comedones), pinheads (papules), large papules (nodules), pimples and scarring (2)

The term acne is derived from Greek word acme which means prime of life. Although generally considered to be a benign, self-limiting condition, acne may cause severe psychological problems or disfiguring scars that can persist for a lifetime. It is a pleomorphic disorder and can manifest at any time during life but it most commonly presents between ages of 12-24, which estimates of 85% of population affected. (10)

Acne affects skin having dense sebaceous follicles in areas including face, chest and back (3). Acne may be of inflammatory or non-inflammatory forms (4). Due to changes in pilosebaceous unit lesions are caused by androgen stimulation. Acne occurs commonly during adolescence, affecting about 80–90% of teenagers in the Western world and lower rate are reported in rural societies (5-8). Acne is usually caused by increase in androgens level like testosterone mainly during puberty in both male and female (9).



Figure 1: Acne on the Back



Figure 2: Acne on the Chest



Figure 3: Acne on the Face.

Types of acne lesions -

It is differentiated by two types of lesions: non-inflammatory, open and closed comedones, as well as inflammatory papules, pustules, nodules, and cysts (figure 4). The comedones are of two types: a comedo that is closed is a whitehead, while another that is open is a blackhead type. (11)

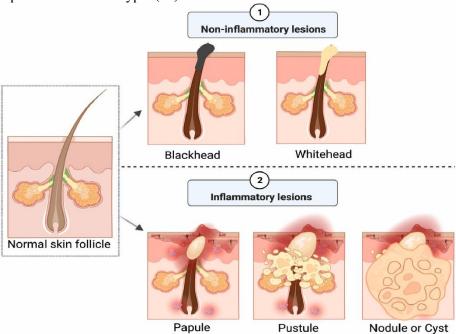


Figure 4: Schematic illustration of major distinguishing of the two types of lesions (non-inflammatory, inflammatory)

Blackheads: Blackheads are non-inflammatory acne lesions that develop on the skin due to excess oil and dead skin cells obstructing hair shafts. A blackhead is referred to as an open comedo because the skin surface remains exposed and has a dark look, such as black or brown. Blackheads are mild acne that usually appears on the face, arms, chest, neck, back and shoulders.



Figure 6: blackheads on face

Whiteheads: Whiteheads are small bumps and non-inflammatory acne lesion that develops on the skin when oil, bacteria and skin cells block the opening of hair follicle pores. Whiteheads are referred to as closed comedones since the bumps are closed and white. Whiteheads can develop anywhere on the body, but they are most frequent in the T-zone, which includes the nose, chin, and forehead.



Figure 7: whiteheads on face

Papules: Inflammation is the response of healthy skin tissue to bacteria, excess oil production, and excess androgen activity, and its symptoms include swelling, heat, redness, and pain. These inflamed lesions are known as papules and are considered an intermediary step between non-inflammatory and inflammatory lesions. Papules show on the skin as a little pink lump typically less than 5 mm in diameter and not pus-filled.



Figure 8: papules on face

Pustules: Pustules are small bumps and an inflammatory lesion that occurs on the skin by clogging the pores with excess oil and dead skin cells. Pustules are inflammatory lesions that contain fluid or pus in their centre. Often, they manifest as white pimples surrounded by red, irritated skin. Pustules can form on any part of the body, although they are most prevalent on the shoulders, chest, back, face, neck, underarms, pubic region, and hairline.



Figure 9: Pustules on face

Nodules: Acne nodules are a severe form of inflammatory acne that develops when the pores become clogged by bacteria, excess oil and dead skin cells. This type of combination usually causes whitehead or blackhead comedones, but if the infection penetrates underneath the surface of the skin and affects the pores as well as the surrounding area to become red and swollen and appear as a small bump. Acne nodules are not treatable with over-the-counter medications alone and might remain for weeks or months. Nodular acne is similar to papule acne, but its diameter is bigger than 5–10 mm, and it often develops on the face's jawline or chin.



Figure 10: Nodules on face

Cysts: Cystic acne is a severe kind of inflammatory acne that appears beneath the skin due to blocked pores caused by the accumulation of bacteria, dry skin cells, and oil People with the oily skin of all ages are most affected. Cyst typically appears as large white/red painful lesions filled with pus, sometimes leading to scars. Cystic acne can appear anywhere on the body, although it most frequently affects the face, arms, shoulders, back, chest, and neck. Most people with cystic acne experience both inflammatory and non-inflammatory acne symptoms. **(12)**



Figure 11: Cysts on face

Pathophysiology -

The pathophysiology of acne vulgaris is complex, with both internal and external triggers (see *How acne develops*). However, the underlying cause is increased sebum production and abnormal desquamation of epithelial cells. (13) One of the initial events in the evolution of acne lesions is the development of the microcomedo, or blockage of the follicular canal. Increased cohesiveness of corneocytes and hyperkeratosis of the follicular lining cause keratin and sebum to accumulate in the follicle. This creates a plug (comedo) above the sebaceous gland duct. As these cells continue to pack into the follicle, the comedo expands behind a small follicular opening to the skin. This results in distension of the follicle and formation of a closed comedone (firm, elevated, white or yellow papule). If the pore begins to dilate at the surface of the skin due to this retention keratosis, an open comedone results (blackhead). (13) The closed comedone is the precursor to the inflammatory lesions associated with acne. As the enlarging comedone causes increased force within the follicle, eventual rupture of the comedo wall results in extrusion of keratin and sebum as well as subsequent inflammation of the skin. (14)

Propionibacterium acnes (*P. acnes*) is the predominant bacteria associated with acne. It is considered part of the normal skin flora and is an inhabitant of the pilosebaceous follicle. However, the role of *P. acnes* in acne vulgaris is significant since the bacteria greatly contributes to the inflammation and irritation associated with acne. (13-14)

Hormones play a central role in the stimulation of sebaceous glands and development of acne. Sebaceous gland size and metabolic rate are directly stimulated by dihydrotestosterone, a derivative of testosterone (an androgenic/sex hormone). (13) Interestingly, an increase in estrogen will decrease sebum secretion. (15)

Development of acne often heralds the onset of puberty and increased sex hormone production. The severity of the acne generally correlates with the level of sex hormones being secreted (which tends to peak in the mid-teenage years). Females also tend to experience a flare in acne about a week before menstruation. (16)

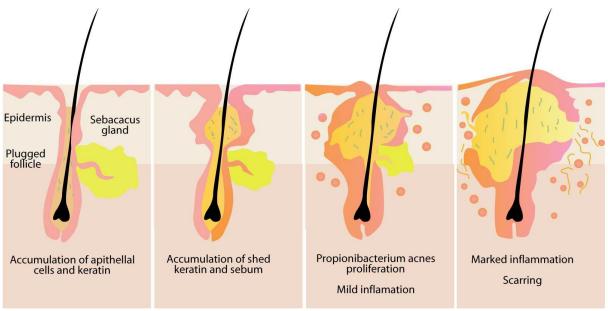


Figure 12: Acne Pathophysiology

At times, acne can be a sign of hyperandrogenism. Female teenagers and adult women presenting with acne should always be asked about irregular menses, hirsutism, or unexplained weight gain. Evaluation for polycystic ovary syndrome may then be necessary. Females with acne resistant to conventional treatment or sudden onset of severe acne may also warrant an endocrine evaluation. (16)

Other external factors that cause occlusion of the hair follicle can also trigger acne outbreaks. For instance, the use of comedogenic products such as cosmetics and greasy hair products can induce comedones and inflammatory lesions. Furthermore, occlusive garments such as collars, sports bras, hats, helmets, and chin straps can greatly exacerbate acne due to mechanical irritation and occlusion of the follicles. (16-17) Overzealous washing practices, especially with the use of exfoliants, also cause mechanical irritation that could exacerbate acne. (16)

There is a unique form of acne-like eruptions that are associated with certain medications. These eruptions are generally uniformly inflammatory papules, may be found in an atypical distribution, can present at an unusual age of onset, and are often resistant to conventional acne therapy. Examples of these medications include corticosteroids, androgens, anabolic steroids, neuropsychotherapeutic drugs (tricyclic antidepressants, selective serotonin reuptake inhibitors, lithium, antiepileptics), immunomodulators, and chemotherapeutic agents. (18)

Treatment -

Treating acne requires great patience and proper care and perseverance. Any of the treatments mentioned below may take two or three months to start working if no side effects such as excessive dryness or allergy is found, it is important to allow ample time to each regimen or drug to work well before stopping on it and proceed with other methods by the skin specialist (19)

Table 1. Different treatment options for acne (20)

Treatment Methods	Example
Topical	Retinoids: adapalene, isotretinoin, motretinide, retinoyl-β-glucuronide, tazarotene, tretinoin Antibiotics: clindamycin, erythromycin Diverse: azelaic acid, benzoyl peroxide, chemical peels, corticosteroids, dapsone, hydrogen peroxide, niacinamide, salicylic acid, sodium sulfacetamide, sulfur, triclosan
Systemic	Retinoids: isotretinoin Antibiotics: azithromycin, clindamycin, cotrimoxazole, doxycycline, erythromycin, levofloxacin, lymecycline, minocycline, roxithromycin Hormonal: contraceptives

	Diverse: clofazimine, corticosteroids, ibuprofen, zinc sulfate
Complementary and	Achillea millefolium, amaranth, antimicrobial
Alternative Medicines	peptides, arnica, asparagus, basil
(CAM)	oil, bay, benzoin, birch, bittersweet nightshade,
	black cumin, black walnut,
	borage, Brewer's yeast, burdock root, calendula, celandine, chamomile, chaste
	tree, Commiphora mukul, copaiba oil, coriander, cucumber, duckweed,
	Du Zhong extract, English walnut, Eucalyptus
	dives, fresh lemon, garlic,
	geranium, grapefruit seeds, green tea, jojoba oil,
	juniper twig, labrador tea,
	lemon grass, lemon, minerals, neem, oak bark,
	onion, orange peel, orange,
	Oregon grape root, patchouli, pea, petitgrain,
	pine, pomegranate rind extract,
	poplar, probiotics, pumpkin, resveratrol, rose myrtle, rhubarb, Rosa damascena,
	rosemary, rue, safflower oil, sandalwood, seaweed, soapwort,
	Sophora flavescens, specific antibodies, stinging
	nettle, sunflower oil,
	Taraxacum officinale, taurine bromamine, tea
	tree oil, thyme, turmeric, vinegar,
	vitex, witch hazel, Withania somnifera and yerba
	mate extract
Physical Treatment	Comedone extraction, cryoslush therapy,
	cryotherapy, electrocauterization,
	intralesional corticosteroids and optical
	treatments

Conclusion:

Acne is a skin ailment caused by changes in the sebaceous glands, and the most common type is acne vulgaris. It affects thick sebaceous follicles on the face, chest, and back. Acne can be inflammatory or non-inflammatory, and it is typically caused by an increase in androgens such as testosterone during puberty. Lesions are classified into two types: non-inflammatory comedones, open and closed, and inflammatory papules, pustules, nodules, and cysts. Acne treatment needs patience, careful care, and effort. Treatments may take two or three months to act successfully, so give each regimen or drug enough time to work before moving on to the next.

REFERENCES:

- 1. Chambers HF, Deleo FR (2009) waves of resistance: staphylococcus aureus in the antibiotic era. Rev Microbial, 7 (9): 629-641
- 2. Thappa D, Adityan B, Kumari R. Scoring Systems in Acne Vulgaris. Indian J Dermatol Ve 2009; 75(3): 323–6p.
- 3. Benner N; Sammons D. Overview of the Treatment of Acne Vulgaris, Osteopath Family Physic 2013; 5(5): 185–90p.
- 4. Harper JC. Acne Vulgaris, eMedicine, 2009.
- 5. Taylor M, Gonzalez M, Porter R. Pathways to Inflammation: Acne Pathophysiology, Eur J Dermatol 2011; 21(3): 323–33p.
- 6. Dawson AL, Dellavalle RP. Acne Vulgaris, BMJ 2013; 346: f2634p.
- 7. Berlin DJ, Goldberg AL. Acne and Rosacea Epidemiology, Diagnosis and Treatment, London: Manson Pub, 2012, 8p.

- 8. Spencer EH, Ferdowsian HR, Barnard ND. Diet and Acne: A Review of the Evidence, Int J Dermatol 2009; 48(4): 339–47p.
- 9. James WD. Acne, New Engl J Med 2005; 352(14): 1463–72p
- 10. Chaudhary S (2010) anti acne activity of some Indian Antiacne herbal drugs, international journal of pharma professional" s research, 1 (1):78-80
- 11. Ramli R, Malik AS, Hani AF, Jamil A. Acne analysis, grading and computational assessment methods: an overview. Skin research and technology. 2012 Feb;18(1):1-4.
- 12. Vasam M, Korutla S, Bohara RA. Acne vulgaris: A review of the pathophysiology, treatment, and recent nanotechnology based advances. Biochemistry and Biophysics Reports. 2023 Dec 1;36: 101578.
- 13. 6. Habif TP. *Clinical Dermatology: A Colour Guide to Diagnosis and Therapy*. 4th ed. Philadelphia: Mosby; 2004:162–194.
- 14. 7. Bolognia JL, Jorizzo JL, Rapini RP. *Dermatology*. 2nd ed. Spain: Mosby; 2008:496–508.
- 15. Zouboulis CC. Acne and sebaceous gland function. Clin Dermatol. 2004;22(5):360–366.
- 16. James WD, Berger T, Elston D. *Andrews' Diseases of the Skin: Clinical Dermatology*. 10th ed. Philadelphia, PA: Saunders; 2006:231–239.
- 17. Ramanathan S, Herbert AA. Management of acne vulgaris. J Pediatr Health Care. 2011;25(5):332–337.
- 18. Du-Thanh A, Kluger N, Bensalleh H, Guillot B. Drug-induced acneiform eruption. *Am J Clin Dermatol*. 2011;12(4):233–245.
- 19. Hamilton FL., Car J., Lyons C., Car M., Layton A., Majeed A, Laser and other light therapies for the treatment of acne vulgaris: systematic review, Br J Dermatol, 2009; 160:6: 1273-1285.
- 20. Fox L, Csongradi C, Aucamp M, Du Plessis J, Gerber M. Treatment modalities for acne. Molecules. 2016 Aug 13;21(8):1063.