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# Introductory Chapter: Acne and Acneiform Dermatoses

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http://dx.doi.org/10.5772/66979

# 1. Introduction

#### 1.1. The definition of 'acne' and 'acneiform'

Acne vulgaris (AV) is a multifactorial disease of pilosebaceous unit and is a prototype of acneiform dermatoses. The term 'acneiform' refers to dermatoses which resemble acne vulgaris clinically but have different etiopathogenesis [1]. Acneiform dermatoses include a wide range of diseases which can occur in all age groups from infancy to elderly. They are so prevalent in all around the world that only AV rate has been reported to be 40–50 million/year in the USA and its prevalence has been estimated as 100% among adolescents in United Kingdom [2, 3]. Acneiform dermatoses have also important economic burden on governments due to high prevalence [2, 4].

# 2. Acne

AV lesions develop due to increased sebum production, follicular epidermal hyperproliferation, inflammation and *Propionibacterium acnes* (*P. acnes*) colonization [5]. Formation of microcomedones was originally thought to be the first step in AV pathogenesis. However, recent studies supported that inflammation has a role at all stages of acne lesion development, perhaps subclinically even before microcomedone formation. The immunochemical pathways underlying the initiation and extension of the inflammation in AV are not known completely but several inflammatory mediators including cytokines, defensins, peptidases, sebum lipids and neuropeptides may play role in complex pathogenesis of AV [6]. AV is clinically characterized by open and closed comedones, papules, pustules, nodules, cysts and scar formations [4, 7]. It primarily affects adolescents so it has negative psychosocial effects on this age group [8]. Also, there is a risk of postinflammatory hypo/hyperpigmentation and scar formation, so early treatment of AV is important. Topical (retinoids, benzoyl peroxide, antibiotics, salicylic acid, azelaic acid) and systemic (antibiotics, hormonal treatment, isotretinoin) agents and surgical procedures can be used in the treatment of AV [4, 7]. In pregnant women, topical benzoyl peroxide and antibiotics (erythromycin, clindamycin)



can be preferred. When necessary, systemic antibiotics such as penicillins, erythromycin and clindamycin can be added. Due to the risk of teratogenicity, retinoids should not be used in pregnant women [9].

Although acne is mostly seen in adolescence period, it may be seen in pediatric age group. Neonatal acne is a temporary and benign acne variant characterized by millimetric inflammatory papules on cheeks and across the nasal bridge which appears usually during the neonatal period [10]. Increased sebum secretion due to stimulation of neonatal sebaceous glands with maternal hormones, and *Malassezia furfur* and *Malassezia smpodialis* colonization have been implicated in the etiopathogenesis of this disease [7, 10, 11]. Lesions usually resolve by 3 months of age without any treatment [10]. Infantile acne usually begins between 3 and 6 months of age. It results from hormonal imbalance in this age period. Clinically, comedones, papules, pustules and nodules may be present similar to adolescent acne. Although it has a tendency to heal spontaneously within 1 or 2 years, it should be treated due to the risk of scar formation [7, 10].

Severity of AV may change from mild to severe. Moreover, in acne fulminans (acute febrile ulcerative acne), which is the most severe acne form, systemic symptoms such as fever, arthralgia, myalgia, fatigue, hepatosplenomegaly and osteolytic bone lesions may accompany to skin lesions that are characterized by suddenly appeared multiple, tender, inflammatory nodular, suppurative lesions and ulcers with hemorrhagic crusts on the back and chest. Increased sedimentation rate, proteinuria, leukocytosis and anemia may also be seen. Acne conglobata is another severe nodulocystic acne form; however, in contrast to acne fulminans, it is not accompanied by any systemic symptoms. It is characterized by comedones, papules, pustules, nodules, abscesses and scars on the chest, back, buttocks, face, shoulders and thighs. Solid facial edema (Morbihan disease) is an important complication of acne characterized by woody enduration and erythema on midface and cheeks [7, 10].

Acne lesions sometimes may occur due to chronic friction, and this form is named as acne mechanica. It is characterized by comedones, which occur secondary to mechanic obstruction of pilosebaceous unit [7, 12]. Cosmetic acne is most commonly seen among young females and is associated with the application of cosmetics containing comedogenic substances. It is characterized by multiple comedones at the sites of cosmetic used [11, 12]. Excessive heat exposure may cause inflammatory nodulocystic acneiform lesions on body and arms and this entity is named as tropical acne [4, 7]. Acne aestivalis (Mallorca acne) is characterized by multiple papular lesions on cheeks, sides of the neck, chest, shoulders and upper arms that develop after sun exposure. UV radiation, especially UVA, is the responsible factor in the development of these lesions. Recurrences are common in successive years [1]. Radiation acne refers to eruption, consisting of multiple comedone-like papules on the sites that are previously exposed to ionized radiation for treatment. Acne *excoriee des jeunes filles* is an acne form that is mainly seen among young women and characterized by crusted, excoriated papules. It is generally accompanied by psychiatric disorders [4, 7].

Another form of acne, occupational acne, occurs as a result of occupational exposure to follicular occlusive compounds such as chlorinated hydrocarbons, coal tar derivatives. It is characterized by especially comedones on exposure sites, but inflammatory papules, pustules and cysts may be seen. Chloracne is the main example of occupational acne. It is characterized by multiple cysts, papules and nodules on malar, retroauricular, mandibular, aksillar regions and scrotum [7, 12].

With an increasing frequency in recent years, drug-induced acne characterized by a monomorphic eruption composed of inflammatory papule and pustules which occurs after usage of certain drugs. The distribution of lesions may be similar to AV; on the contrary, the onset is mostly acute, lesions are predominantly inflammatory and monomorphic and comedones are usually absent [1]. Involvement of unusual localizations for AV (e.g. distal extremities), occurrence at an unusual age for AV, the history of exposure to one of the known causative drugs and regression of lesions after the cessation the offending agent are other clues for diagnoses [1, 13]. Steroid acne is the best known example of drug-induced acne. It may occur on chest and back as a result of systemic steroid usage or on face due to inappropriate or prolonged usage of topical steroids [7]. Anabolic steroids, fenitoin, lithium, vitamins, cyclosporine, isoniazid, azathioprine, iodides, bromides and epidermal growth factor receptor inhibitors are the other major drugs which are responsible from acneiform eruptions [1, 13]. Lesions regress after the cessation of responsible agent [1].

Acne lesions are mostly seen in isolated manner however may develop as a component of some syndromes. SAPHO syndrome is characterized by any combination of synovitis, *a*cne, *p*ustulosis, *h*yperostosis and *o*steitis. Characteristic skin lesions in SAPHO syndrome include acne fulminans, hidradenitis suppurativa and palmoplantar pustulosis [7, 14]. PAPA syndrome is an autosomal dominantly inherited, autoinflammatory disorder characterized by *p*yogenic *a*rthritis, *p*yoderma gangrenosum and *a*cne, which is usually in severe nodulocystic type. Apert syndrome (acrocephalosyndactyly type I) is an autosomal dominant congenital disorder characterized by craniofacial deformities, dental abnormalities, syndactyly and hypertelorism. Early onset, widespread, severe, comedonal and inflammatory acne involving the face, chest, back and unusual sites like forearms is a common clinical feature of this syndrome [14]. AV is also seen more frequently in patients with *p*oly*c*ystic *o*vary *s*yndrome (PCOS) due to hormonal disturbances. HAIR-AN syndrome, a subset of PCOS, is characterized by *h*yperandrogenism, *a*cne, *i*nsulin resistance and *a*canthosis *n*igricans [7, 14].

# 3. Acneifom dermatoses

The characteristic papulopustular lesions of Behcet disease are indistinguishable from AV clinically, but arms and legs involved preferentially in contrast to AV [14]. Histologically vasculitis might be seen [15]. It was reported that these lesions are usually observed in combination with arthritis [14].

Rosacea is a multifactorial disorder presented with acneiform lesions. It generally affects individuals with fair skin and older age compared to AV. There is a female predilection, but phymatous type occurs mostly in men. Vascular hyperreactivity and *P. acnes* and *Demodex folliculorum* colonization have been proposed in the etiopathogenesis. Food and drugs that can cause vasodilatation and thermal stimulation may precipitate rosacea development. It

is clinically characterized by erythema, papule, pustule and telangiectasias with subjective symptoms such as burning and stinging. Four main clinical subtypes have been described, namely erythematotelangiectatic, papulopustular, phymatous and ocular. In addition to them, there are other rosacea types such as, granulomatous rosacea, periorificial dermatitis and pyoderma faciale. Typically, it starts as a transient erythema attacks (flushing) on cheeks and nose. Erythema becomes permanent with time and telangiectasias occur. After that, millimetric papules, pustules, plaques and nodules with varying severities may occur. Granulomatous rosacea is a variant of rosacea characterized by red-brown papules which show granulomatous inflammation histopathologically. In phymatous type rosacea, follicular orifices become prominent, nodularities and irregularities occur on the thickened skin due to sebaceous hyperplasia. These phymatous changes most often develop on the nose (rhinophyma), but may also occur on chin (gnatophyma), ear (otophyma), forehead (metophyma) and eyelids (blepharophyma). Ocular symptoms such as burning, stinging, dryness, tearing, blepharitis and corneal damage may be present in the nearly half of the patients independently severity of cutaneous signs (Ocular rosacea) [12, 16–18].

Perioral and periorbital dermatitis is characterized by discrete, erythematous papules, plaques and pustules on erythematous base around mouth and eye, which characteristically spare the vermillion border. Although the relationship with rosacea is not clear, it has similar histopathological findings. Also, it responses to similar treatments. Pyoderma faciale is another entity that resembles rosacea histopathologically and responds similar treatment modalities. This disease is characterized by inflammatory papules, pustules and plaques on the midface. Systemic symptoms, such as fever, myalgia and laboratory findings (increased sedimentation rate and white blood cell count) may accompany with cutaneous symptoms in these patients. Inappropriate or long-term steroid usage may also cause rosacea formation or exacerbation (Steroid rosacea) [17, 18].

Usage of sun protection and avoidance from the factors that cause vasodilatation and irritation should be recommended to rosacea patients. Topical (metronidazole, sodium sulfacetamide, azelaic acid, tretioin) and systemic (antibiotics and retinoic acid) agents can be used in the treatment. Vascular lasers and intense pulsed light therapy can be used in the treatment of telangiectasias. Electrosurgery and carbon dioxide laser are the treatment options for the rhinophyma [19].

Demodicosis is a disease of the pilosebaceous unit associated with human *Demodex* mites (*D. folliculorum and Demodex brevis*). *Demodex* mites can be found in normal adult skin with a density of less than 5 mites per cm<sup>2</sup>. Demodicosis may occur if the density of these mite increases due to the defect in host immune mechanism, in association with systemic disease or immunosuppressive drug usage (topical corticosteroids, topical calcineurin inhibitors). There are different clinical manifestations of demodicosis that can mimic many other skin disorders, such as rosacea, perioral dermatitis and folliculitis. Spinulate demodicosis (pityriasis folliculorum) characterized by facial erythema with follicular plugs, scaling and sandpaper-like appearance. There is a pronounced inflammation in papulopustular/nodulocystic demodicosis (rosacea-like demodicosis), and it is most com-

monly affects perioral and periorbital areas. Ocular and auricular types of demodicosis were also described [12, 20].

#### 4. Conclusion

Acne and acneiform eruptions are frequent disorders in dermatology practice and the severity of diseases may change from self-limiting, benign condition to fulminant systemic disease. Etiopathogenesis of them are different. They may be seen as isolated or as a component of any syndrome. So, differential diagnoses of these diseases are important. Patient's age, occupation, cosmetic usage, drug history, concomitant diseases, menstrual cycle irregularities and morphology and localization of lesions are important clues for diagnoses. To make an accurate diagnoses, detailed history, physical examination and, if necessary, histopathological examinations are essential. In this book, acne vulgaris and acneiform eruptions will be discussed in detail.

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# References

- [1] Plewig G, Jansen T. Acneiform dermatoses. Dermatology. 1998;196:102–7.
- [2] Bhate K, Williams HC. Epidemiology of acne vulgaris. Br J Dermatol. 2013;168:474–85. DOI: 10.1111/bjd.12149
- [3] Gollnick HP, Zouboulis CC. Not all acne is acne vulgaris. Dtsch Arztebl Int. 2014;**111**:301– 12. DOI: 10.3238/arztebl.2014.0301
- [4] Zaenglein AL, Thiboutot DM. Acne vulgaris. In: Bolognia JL, Jorizzo JL, Rapini RP, editors. Dermatology. 2nd ed. Spain: Mosby; 2008. p. 495–508.
- [5] Suh DH, Kwon HH. What's new in the physiopathology of acne? Br J Dermatol. 2015;**172**:13–9. DOI: 10.1111/bjd.13634
- [6] Tanghetti EA. The role of inflammation in the pathology of acne. J Clin Aesthet Dermatol. 2013;6:27–35.

- [7] Zaenglein AL, Graber EM, Thiboutot DM, Strauss JS. Acne vulgaris and acneiform eruptions. In: Wolff K, Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ, editors. Fitzpatrick's Dermatology in General Medicine. 7th ed. New York: McGraw-Hill; 2008. p. 690–702.
- [8] Revol O, Milliez N, Gerard D. Psychological impact of acne on 21st-century adolescents: decoding for better care. Br J Dermatol. 2015;**172**:52–8. DOI: 10.1111/bjd.13749.
- [9] Kartal Durmazlar SP, Eskioglu F. Cosmetic procedures in pregnancy: Review. Turkiye Klinikleri J Med Sci. 2008;28:942–6.
- [10] Mengesha YM, Bennett ML. Pustular skin disorders: diagnosis and treatment. Am J Clin Dermatol. 2002;3:389–400.
- [11] White GM. Recent findings in the epidemiologic evidence, classification, and subtypes of acne vulgaris. J Am Acad Dermatol. 1998;**39**:34–7.
- [12] Dessinioti C, Antoniou C, Katsambas A. Acneiform eruptions. Clin Dermatol. 2014;32:24– 34. DOI: 10.1016/j.clindermatol.2013.05.023.
- [13] Du-Thanh A, Kluger N, Bensalleh H, Guillot B. Drug-induced acneiform eruption. Am J Clin Dermatol. 2011;12:233–45. DOI: 10.2165/11588900-0000000-00000.
- [14] Lolis MS, Bowe WP, Shalita AR. Acne and systemic disease. Med Clin North Am. 2009;93:1161–81. DOI: 10.1016/j.mcna.2009.08.008.
- [15] Kartal Durmazlar SP. Venous Thrombosis in Behcet's Disease, Venous Thrombosis -Principles and Practice, Dr. Ertugrul Okuyan (Ed.), InTech, DOI: 10.5772/28082. Available from: http://www.intechopen.com/books/venous-thrombosis-principles-and-practice/ venous-thrombosis-in-behcet-s-disease.
- [16] Two AM, Wu W, Gallo RL, Hata TR. Rosacea: part I. Introduction, categorization, histology, pathogenesis, and risk factors. J Am Acad Dermatol. 2015;72:749–58. DOI: 10.1016/j. jaad.2014.08.028.
- [17] Pelle MT. Rosacea. In: Wolff K, Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ, editors. Fitzpatrick's Dermatology in General Medicine. 7th ed. New York: McGraw-Hill; 2008. p. 703–8.
- [18] Webster GF. Rosacea and related disorders. In: Bolognia JL, Jorizzo JL, Rapini RP, editors, Dermatology, Elsevier. 2nd ed. Spain: Mosby; 2008. p. 509–16.
- [19] Two AM, Wu W, Gallo RL, Hata TR. Rosacea: part II. Topical and systemic therapies in the treatment of rosacea. J Am Acad Dermatol. 2015;72:761–70. DOI: 10.1016/j. jaad.2014.08.027.
- [20] Chen W, Plewig G. Human demodicosis: revisit and a proposed classification. Br J Dermatol. 2014;170:1219–25. DOI: 10.1111/bjd.12850.