

DEFINITION

1. **Acne vulgaris** is a chronic disorder of the pilosebaceous follicles of the skin. The lesions of acne consist of comedones, erythematous papules and pustules, nodules and cysts, and scarring.
2. The basic lesion of acne vulgaris is the **comedo**. This is produced by hyperkeratosis of the lining of the pilosebaceous follicles with retention of keratin and sebum. A closed comedo is known as a "whitehead" and an open comedo is called a "blackhead", the black colour being due to melanin. The comedo is a non-inflamed lesion and it usually regresses spontaneously with little or no scarring.
3. However the lesions may become inflamed and these inflamed lesions may heal leaving residual scarring. It is thought that the plugging produced by the comedo dilates the mouth of the follicle, forming papules by inflammation around the comedones. It is not uncommon to see comedones, pustules, cystic swellings and scars, often keloidal, in the same person.

CLINICAL MANIFESTATIONS

4. The lesions of acne occur predominantly on the face and to a lesser extent on the back and the chest.
5. The condition usually begins at puberty, when slight degrees of acne are common and are regarded as being physiological. Both sexes are affected with equal frequency. The condition diminishes in severity from the age of 20 years and tends to disappear around the age of 25, although occasionally it can persist throughout life. Its course is variable and recurrent exacerbations are common. In younger patients it more commonly affects the face, while in the middle-aged the back is the principal site.
6. Certain diseases which occlude the follicular pores, for example hidradenitis suppurativa and necrotising cellulitis of the scalp, may be associated with acne.
7. Severe acne variants are comparatively rare but they are clinically important. Four main variants are recognised:
8. **Pyoderma faciale** is seen usually in an older woman with existing acne who is subjected to stress, when a localised but explosive pattern of the disease appears.
9. **Acne conglobata** is a severe form of the disease which affects the face, back and limbs. Cystic and pustular lesions occur and scarring may be marked. It occurs mainly in men.
10. **Acne fulminans** is an immunologically induced severe systemic variant of acne conglobata. The clinical features are those of acne conglobata plus the classic delayed hypersensitivity systemic reaction with splenomegaly, arthropathy and rashes.

11. **Gram-negative folliculitis** is associated with the long-term antibiotic treatment of acne. Acne which was well controlled suddenly appears to "escape" from control. This condition takes the form of a sudden eruption of small follicular pustules.

AETIOLOGY

12. Four main factors cause or influence the development of acne vulgaris:
 - 12.1. increased sebum production
 - 12.2. an abnormality of the microbial flora
 - 12.3. cornification of the pilosebaceous duct
 - 12.4. inflammation.
13. These in turn are influenced by genetic and external factors.
14. The predominant importance of genetic factors in determining susceptibility to acne is suggested by racial studies and confirmed by the very high degree of concordance in identical twins. In the USA the disease is commonest in white Americans and less common in Negroes and Japanese.
15. Acne patients as a group have an enhanced rate of sebum secretion compared with controls. Sebaceous activity is dependent on male sex hormones of gonadal or adrenal origin. In the blood the sex hormones are protein bound. Increased levels of free sex hormone may result from their increased production or from decreased protein binding. This latter may arise from quantitative or qualitative changes in protein.
16. In males the levels of plasma testosterone are normal. In females the evidence is conflicting. It is generally agreed that the androgenic hormonal balance is disturbed in 50-75% of females suffering from acne. There is however no correlation between hirsutism or menstrual irregularity and the severity of the acne. These conflicting observations lead to the conclusion that male hormone balance is not the critical factor in producing clinical acne. Acne may also be associated with Cushing's syndrome, polycystic ovary syndrome and the adrenogenital syndrome.
17. The actual pathological mechanisms of the condition and of its characteristic lesions are unknown. Abnormality of fatty acid metabolism and immune factors play a part in follicular obstruction. This follicular obstruction leads to inflammatory changes, the keratin and sebum having a foreign-body effect. Most acne lesions are sterile but sometimes there is secondary infection, particularly with staphylococci.
18. The factors causing dermal inflammation in acne are unknown. It does not result directly from bacterial activity but may be secondary to chemical mediators produced in the follicles by bacteria.
19. While there is evidence to show that gross weight loss leads to decreased sebum secretion and subsequent reduction in acne, there is no evidence that dietary choice has any role in the cause or course of acne.

20. Sweating makes acne worse. This is particularly seen in catering workers and where people from temperate regions encounter hot humid climates. Troops in World War 2 were at risk when posted to the Far East. In the Vietnam War the condition was relatively rare as non-prone individuals were specifically selected to serve.
21. Patients often report that sunshine improves acne. However there is no proof that it does and there is some contradictory evidence. Ultraviolet radiation as used in PUVA therapy may actually induce acne lesions.
22. Physical or emotional stress does not cause acne. However the condition may itself cause depression and anxiety. There is evidence that patients with established acne may undergo exacerbations due to emotional stress.
23. Drugs may induce or aggravate the condition. The fact that the drugs do not produce such a reaction in all patients exposed indicates that endogenous factors are important. Acne may be exacerbated by corticosteroids, certain types of contraceptive pill, androgens and anabolic steroids, phenytoin, isoniazid, iodides and bromides.
24. A variety of chemicals may induce acne by external contact and these chemicals may also worsen pre-existing acne vulgaris. The majority of them are industrial hazards, the most important of which are DDT, insoluble cutting oils, crude petroleum and heavy coal-tar distillates such as tar and creosote. A characteristic syndrome called chloracne may follow exposure to certain toxic chlorinated hydrocarbons. Detergent acne occurs in those who wash obsessively in the vain hope of eliminating their existing acne.
25. Certain cosmetics may be comedogenic and they may produce perioral dermatitis. Similarly, pomades extending on to non-hair bearing skin may produce comedones.
26. Acne may follow repetitive mechanical skin trauma. This seems to occur in people who are genetically predisposed. The acne lesions are localised to the site of friction. An example is "fiddlers' neck", and it may be associated with bra straps, headbands and turtle or polo neck sweaters.

CONCLUSION

27. **Acne vulgaris** is a condition of the skin, the characteristic lesion of which is the comedo. It is primarily caused by genetic factors although the external factors listed above may precipitate the condition or exacerbate already existing lesions.

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