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PATHOGENESIS OF ACNE

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SUMMARY

Understanding the pathogenic process which leads to the development of acne lesions enables the clinician to design a logical therapeutic program directed at these pathogenic factors.

To date four principal pathogenic mechanisms have been defined in acne-these are:

1.- Sebum

2.- Propionibacterium acnes (P. acnes)

3.- Altered follicular keratinization

4.- Inflammation

Sebum production is androgen dependent and usually increases at or about puberty. This increase in sebum results in a relative deficiency of linoleic acid within the follicle and may precipitate the early changes seen in follicular keratinization. Sebum also provides an excellent culture medium for P. acnes since this organism uses the triglycerides of sebum as a source of sugar. P. acnes produces a variety of extracellular factors which are important in the subsequent development of acne lesions. P. acnes lipase hydrolyzes sebum triglycerides to free fatty acids. Free fatty acids are comedogenic and irritants. In addition, chemotactic factors released by P. acnes, attract neutrophils to the follicle resulting in the early inflammatory lesion of acne.

Subsequent breakdown of the follicle wall results in the classical inflammatory lesions of acne.

The final common pathway in acne lesion development is the formation of the microcomedo. Selected lipids of sebum and free fatty acids promote increased all turnover in the follicle wall and altered follicular keratinization. As a result the cells from the follicle are shed more rapidly and are more cohesive, producing a retention hyperkeratosis called the microcomedo. The microcomedo can then proceed to either mature closed and open comedones or, inflammatory lesions.

Finally, as a result of the above processes, changes may occur in the patient's immune system which may contribute to the inflamation process. As P. acnes enters the bloodstream antibodies to P. acnes are increased, but do not appear to be protective. As the severity of acne progresses there is diminished delayed type sensitivity, altered T-cell and phagocyte function and abnormal migration of neutrophils. The latter may be the result rather than the cause of chronic inflammation.

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