Short Communication
Volume 7:2, 2022

Journal of Molecular Histology and Medical Physiology

ISSN: 2684-494X

Open Access

Acne Histological and Immunocytochemical Study

Janaki Madhuri*

Department of Dermatology, General Infirmary, Leeds, UK

Introduction

Acne vulgaris is one of the most common inflammatory dermatoses seen in dermatology and can affect people of any gender or ethnicity. Many inflammatory acne patients have significant scarring, which is disfiguring and difficult to treat. A cell-mediated immune response is thought to be involved in the pathogenesis of acne, though the extent of this response varies between patients [1].

Acne vulgaris is a common pilosebaceous follicle cutaneous disorder that affects over 45 million people in the United States alone. Acne pathogenesis is multifactorial, with abnormal hyperkeratinization, increased sebum production, hormones, cutaneous microbes, and immunological mechanisms all playing a role. Many of the immunological processes that contribute to the formation of acne lesions occur on the skin itself. Skin is an important component of the innate immune system because it provides physical barriers as well as rapid cellular responses by keratinocytes, Langerhans cells, and other infiltrating inflammatory cells [2].

Description

A microcomedone is the first subclinical acne wound or scar distinguished by follicular epithelial hyperproliferation. On the periphery of these scars inflammatory cells have been discovered. The purpose of this study was to determine whether inflammatory events occur before or after hyperproliferative changes. Immunohistochemical techniques were used to look for cellular, vascular, and proliferative markers in biopsies of clinically normal follicles from uninvolved skin and early inflamed acne lesions. Control follicles were provided by non-acne subjects. Uninvolved skin follicles lacked microcomedonal characteristics [3].

Acne is a common cause of post-inflammatory hyperpigmentation (PIH), especially in patients of colour, and PIH is frequently more distressing to patients than the acne itself. Topical retinoids are approved for the treatment of acne as well as pigmentation disorders such as melasma or mottled hyperpigmentation caused by photo damage; they have also been shown to reduce hyperpigmentation in SOC patients. As a result, unless contraindicated, treatment with topical retinoids should begin as soon as possible. Irritation may be reduced by using novel formulations or applying commonly recommended moisturisers. Retinoids can be combined with other topical agents and procedures, such as superficial chemical peels, to help improve hyperpigmentation. Primary acne lesions will likely improve weeks before PIH resolves, so assisting patients in managing their expectations may reduce frustration. More education for clinicians and researchers about the presentation and management of dermatologic conditions in SOC patients is also recommended [4,5].

Conclusion

Cellular inflammatory events now appear to play a critical role in all stages of acne lesion development, from preclinical initiation to clinical presentation of active lesions and resolution. Acne has shifted from being primarily a hyperproliferative disorder of the sebaceous follicle to an inflammatory skin disorder. The sequence of events leading to lesion formation has become clearer, but the triggers for initiation remain unknown. Before triggers for initiation can be defined, non-invasive techniques for detecting preclinical "acne-prone" follicles must be developed. Finally, the differences in the inflammatory profiles of inflamed lesions from scarring acne patients versus other non-scarring acne patients support the notion that acne is a disorder encompassing a variety of pathologies.

Conflict of Interest

None.

References

1. Tuchayi, Sara Moradi, Evgenia Makrantonaki, Ruta Ganceviciene and Clio Dessinioti, et al. "Acne vulgaris." *Nat Rev Dis Primers* 1 (2015): 1-20.
2. Saurat, Jean-Hilaire. "Strategic targets in acne: the comedone switch in question." *Dermatol* 231 (2015): 105-111.
3. Cunliffe, William J., Holland D.B and Jeremy A. "Comedone formation: Etiology, clinical presentation, and treatment." *Clin Dermatol* 22 (2004): 367-374.
4. Do, Thy Thy, Semyon Zarkhin, Jeffrey S. Orringer and Shari Nemeth, et al. "Computer-assisted alignment and tracking of acne lesions indicate that most inflammatory lesions arise from comedones and *de novo*." *J Am Acad Dermatol* 58 (2008): 603-608.
5. Norris, J.F.B and Cunliffe W.J. "A histological and immunocytochemical study of early acne lesions." *Br J Dermatol* 118 (1988): 651-659.

How to cite this article: Madhuri, Janaki. "Acne Histological and Immunocytochemical Study." J Mol Hist Med Phys 7 (2022): 31.

***Address for Correspondence:** Janaki Madhuri, Department of Dermatology, General Infirmary, Leeds, UK, E-mail: madhuri_j@gmail.com*

Received: 03 March, 2022; Manuscript No: jmhmp-22-68051; **Editor assigned:** 05 March, 2022, PreQC No: P-68051; **Reviewed:** 17 March, 2022, QC No: Q-68051; **Revised:** 22 March, 2022, Manuscript No: R-68051; **Published:** 29 March, 2022, DOI: 10.37421/2684-494X.2022.7.31