# Microabscess reconnoiter

Mala Kamboj, Vanshika Makkar, Pooja Sharma, Rahul Kumar Raman

Department of Oral Pathology, Post Graduate Institute of Dental Sciences, Pt B D Sharma University of Health Sciences, Rohtak, Haryana, India

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## INTRODUCTION

Microabscess is derived from "*Mikro*" as Greek word meaning small and "*abscedere*" as Latin origin which means to go away, a very small abscess.[1] Dorland dictionary explained microabscess as a very small, localized collection of pus.[2] The American Heritage Medical Dictionary described it as a very small circumscribed collection of white blood cells in a solid tissue.[3] Inflammation is the primary response of host to varied biological irritants including microbes or traumatic injuries. The accumulation of neutrophils at the site of injury is the first line cellular response.[4] At few instances, it can lead to the formation of purulent exudation and these focal areas of suppuration are called microabscesses. It is characterized by an area comprehended of bacteria, immune cells chiefly neutrophils, fluids and dead cells.[5] In pyogenic infection, when immune reaction is inadequate to abolish the infection, this can lead to the development of microabscess.

## MECHANISM OF MICROABSCESS FORMATION AND REVERSION

The procedure of microabscess formation commences when immune system confronts the bacteria as an infectious agent[4,5] [Figure 1]. Interleukin-6 (IL-6) signaling is a crucial pathway which moderates neutrophils to the site of inflammation. IL-17A expression in the skin results in upregulated granulopoiesis and migration of IL-6 expressing neutrophils into the skin which exacerbates neutrophil microabscess development in psoriasiform lesions.[6] Microabscesses are found in many different diseases and at various locations. Out of them, well-known are liver microabscess, neutrophilic abscess in myocarditis, epidermal microabscess and tumor-infiltrating microabscess in pancreatic neoplasia. Microabscess can be evident in epithelium or connective tissue.[4,5]

## MICROABSCESS IN ORAL LESIONS

Various oral mucosal lesions are subjected to numerous bacterial and fungal pathogens. At times, these infections can lead to superficial microabscess formation [Figures 2 and 3]. Assorted oral lesions such as chronic osteomyelitis, plasma cell gingivitis, candidiasis, keratoacanthoma, pyogenic granuloma and measles can have such presentations.[7]

Munro's microabscess evinces infiltrating leukocytes within the horny layer of the epidermis. They are evident in psoriasiform lesions, such as in psoriasis and geographic tongue. In pustular psoriasis, multiple neutrophils pervade the overlying suprabasal layer and epidermal cells are destroyed to form spongiosis known as Kogoj's spongiform pustule. Reflectance confocal microscopy, a noninvasive technique reveals this microabscess as highly refractile and twinkling particles as compared to the surrounding keratinizing background.[8] Papillary microabscesses are characterized by the presence of acantholytic keratinocytes with neutrophils as seen in pemphigus vulgaris and dermatitis herpetiformis.[4]

Intraepithelial microabscess also cognates with verrucous carcinoma. One study on 426 cases of verrucous carcinoma showed 34% of cases with intraepithelial microabscess

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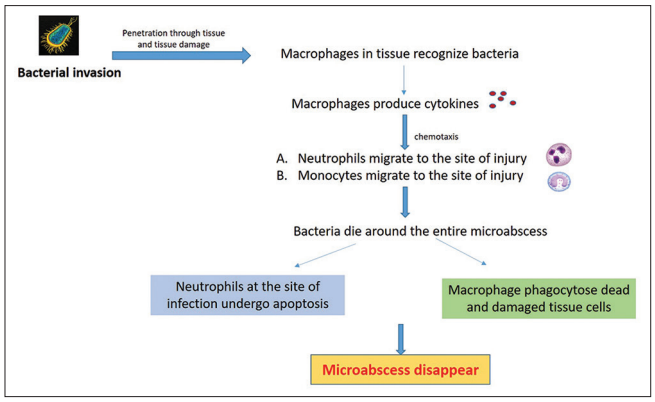


**Figure 1:** Schematic representation of mechanism of microabscess formation and its reversion

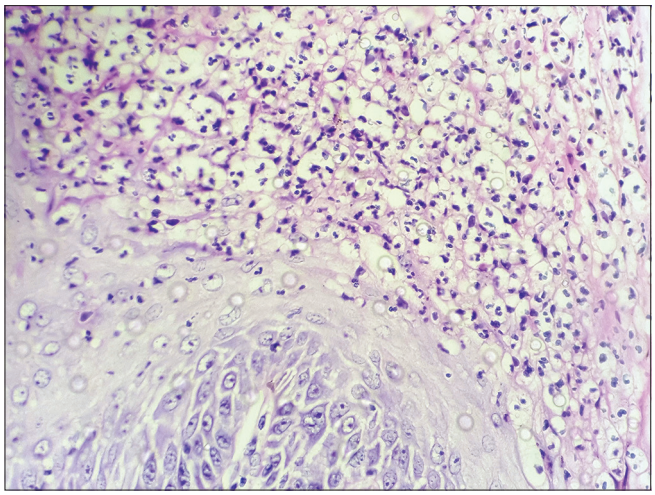

**Figure 2:** Accumulation of inflammatory cells chiefly neutrophils in superficial layers of epithelium (H and E, ×20)

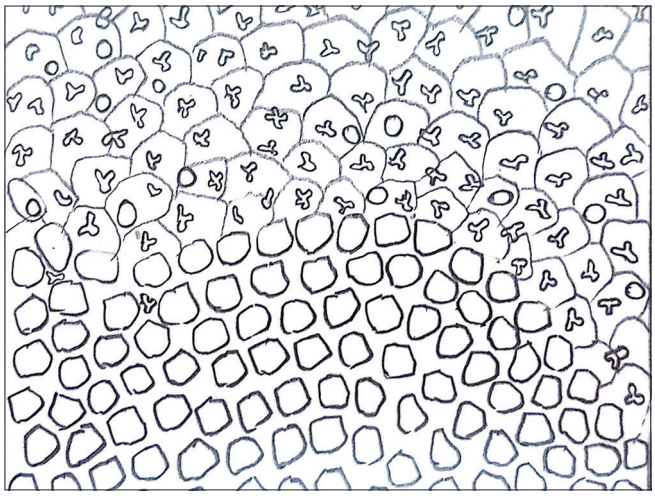

**Figure 3:** Hand-drawn illustration of superficial microabscess, representing purulent exudate chiefly neutrophils and few lymphocytes

formation.[9] Pautrier's microabscess is seen in the collection of malignant lymphocytes in epidermis as solid intra epidermal nodules as seen in lymphomas and mycosis fungoides. CD4+ lymphocytes lead to the formation of Pautrier's microabscess. Pseudopautrier's abscess is also seen in mycosis fungoides. Immunohistochemical markers CD1a, CD3, CD4, CD68 and high molecular cytokeratin helps in differentiating Pautrier's from pseudopautrier's abscess.

The rare variant of pemphigus with deposition of both IgG and IgA autoantibodies against keratinocyte cell surface has also been reported. Some of them were regarded as IgG/IgA pemphigus. Clinical features were atypical in most of these cases. Neutrophilic infiltration into the epidermis was shown in all of the cases.[10] Eosinophilic microabscess is seen at the luminal edge of epithelium as seen in eosinophilic esophagitis. Anti-IL-6 treatment reduces IL-17A-induced neutrophil microabscess formation. If the formation of fibrous tissue is evident around the microabscess, it is considered that microabscess could have lasted for a longer duration. To conclude microabscess can be considered as a natural strategy of the host to fight against infection.

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## Conflicts of interest

There are no conflicts of interest.

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