**The tale of microabscesses: A review**

Suma Srinivasan¹, Amrita Samanta², B. Veerendra Kumar¹, M. G. Madhura¹, Sarita Yanduri¹

¹Department of Oral and Maxillofacial Pathology, D. A. Pandu Memorial RV Dental College and Hospital, Bengaluru, Karnataka, India; ²Department of Oral Pathology, NSVK Sri Venkateshwara Dental College and Hospital, Bengaluru, Karnataka, India

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**Introduction**

The immune system is a powerful, complex and diverse defensive mechanism of the body. Any antigenic invasion and infections are well-shielded by these immune cells in a variety of mechanisms. One such advanced procedure being the formation of microabscesses.

Microabscess refers to a small accumulation of polymorphs, lymphoid cells, or eosinophils in the epidermis and/or adnexal structures of the skin and epithelium of mucous membranes. Munro’s microabscess found in psoriasis vulgaris and psoriasiform oral lesions is an accumulation of neutrophils in stratum granulosum, stratum corneum, or stratum malpighi. Neutrophilic microabscess formation is also evident in various bacterial infections, Reiter’s disease, and fissure tongue. In mycosis fungoides, intraepidermal accumulation of monocytoïd neoplastic lymphoid cells is referred to as Darier Pautrier microabscess. Lymphocytic accumulation in the epidermis is found in allergic contact dermatitis, lichen simplex chronicus, and pityriasis rosea. Microabscess of eosinophils is seen in pemphigus vegetans and incontinentia pigmenti, while dermatitis herpetiformis shows admixture of neutrophilic and eosinophilic microabscesses. Thus, microabscesses are characteristic of certain pathologies and are helpful in their diagnosis. Furthermore, insight into formation of these microabscesses may have a tale to tell regarding the disease process. Thus, this article attempts to review microabscesses and their significance in oral diseases.

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**Etymology**

*Mikros* (Greek) meaning: Small; *Abscedere* (Latin), meaning: to go away.¹

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**Abstract**

Microabscess refers to a small accumulation of polymorphs, lymphoid cells, or eosinophils in the epidermis and/or adnexal structures of the skin and epithelium of mucous membranes. Munro’s microabscess found in psoriasis vulgaris and psoriasiform oral lesions is an accumulation of neutrophils in stratum granulosum, stratum corneum, or stratum malpighi. Neutrophilic microabscess formation is also evident in various bacterial infections, Reiter’s disease, and fissure tongue. In mycosis fungoides, intraepidermal accumulation of monocytoïd neoplastic lymphoid cells is referred to as Darier Pautrier microabscess. Lymphocytic accumulation in the epidermis is found in allergic contact dermatitis, lichen simplex chronicus, and pityriasis rosea. Microabscess of eosinophils is seen in pemphigus vegetans and incontinentia pigmenti, while dermatitis herpetiformis shows admixture of neutrophilic and eosinophilic microabscesses. Thus, microabscesses are characteristic of certain pathologies and are helpful in their diagnosis. Furthermore, insight into formation of these microabscesses may have a tale to tell regarding the disease process. Thus, this article attempts to review microabscesses and their significance in oral diseases.

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**Keywords:**

Epidermis, eosinophil, lymphocyte, microabscess, neutrophil

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**Correspondence:**

Amrita Samanta, Department of Oral Pathology, NSVK Sri Venkateshwara Dental College and Hospital, Bengaluru, Karnataka, India. E-mail: dramritasamanta@gmail.com

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**Microabscess**

A focal aggregate of PMNs is seen in skin disease, for example, mycosis fungoides: Pautrier’s microabscess, psoriasis: Munro’s microabscess, and bullous pemphigoid: papillary microabscess, or elsewhere, for example, in perivascular tissues of lung in Wegener’s granulomatosis.²

- An abscess visible only under a microscope.³
- A very small circumscribed collection of leukocytes in solid tissues.⁴

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**Classification**

I. According to predominant cell types:
   a) Lymphocytic
   b) Neutrophilic
   c) Eosinophilic
   d) Mixed

II. According to their attributed names:
   a) Munro’s Microabscesses
   b) Pautrier’s Microabscesses
   c) Papillary Microabscesses

III. According to Location in Skin:
   a) Epidermal Microabscesses:
      - Polymorphonuclear microabscesses (Munro’s type):
        Seen in psoriasis, Candida infection
• Lymphocytic Microabscesses: Pautrier microabscesses in mycosis fungoides
• Dermal Microabscesses:
  • Dermal microabscess of polymorphs is seen in dermatitis herpetiformis, some forms of vasculitis, and infections such as furuncle and cellulitis

**Cell type predominant microabscesses in diseases**

I. Eosinophilic microabscesses

a) Eosinophilic Pustular Folliculitis: Classical eosinophilic pustular folliculitis, or Ofuji’s disease (Ofuji et al., 1970) is a chronic and relapsing dermatosis that is predominantly reported in East Asian populations. The lesions are characterized by pruritic follicular papulopustules, with a predilection for seborrhoeic areas. Eosinophilic pustular folliculitis presents as annular clusters of sterile follicular papules and pustules predominantly on the face and trunk that heal with post-inflammatory hyperpigmentation but tend to recur periodically. There is male preponderance, with a male-female ratio of 5:1. The peak age of occurrence being the third and fourth decades of life. Histopathologic examination of skin in the early phase reveals spongiosis of the outer root sheath of the infundibulum associated with a mixed perifollicular inflammatory infiltrate comprising predominantly eosinophils plus lymphocytes and neutrophils. With chronicity, eosinophilic microabscesses are noted and may be associated with disruption of the pilosebaceous structures. The epidermis is typically uninvolved.

b) Kimura’s Disease: A chronic non-specific lymphadenitis with numerous eosinophilic infiltrate (Kimm and Szeto; 1937; Kimura et al., 1948). Commonly seen in third decade of life with male is to female ratio of 3.5:1. The disease occurs in endemic form among Chinese and Japanese and is sporadic in non-asians. Histopathologic examination of sections reveals preservation of follicular lymphoid architecture with the presence of follicular hyperplasia. Interfollicular areas showed massive infiltration of mature eosinophils with eosinophilic microabscesses. Lymph node and perinodal adipose tissue showed eosinophilic folliculolysis. Hyalinized vessels in paracortical region were present along with sclerosis.

II. Neutrophilic Microabscesses

A. Psoriasis vulgaris: Psoriasis is a chronic, scaling inflammatory skin disease that affects approximately 1.5–3% of the population. Although it is not contagious or life threatening, it can affect the patient’s quality of life, with negative psychosocial implications. The presence of a well-defined margin and a silvery white scale, over a glossy homogenous membrane, is clinically diagnostic of psoriasis. The successive removal of the psoriatic scales usually reveals an underlying smooth, glossy red membrane with multiple bleeding points where thin suprapapillary epithelium is torn off (Auspitz’s sign). Psoriasis is characterized by keratinocyte hyperproliferation with parakeratosis, increased angiogenesis and dermal and epidermal infiltration of inflammatory cells, including CD4+ and CD8+ T-cells, neutrophils, macrophages, and dendritic cells (Monteleone et al., 2011). Munro’s microabscesses are a characteristic hallmark of psoriasis pathology (Munro, 1898; Steffen, 2002). These sites of inflammation contain polymorphonuclear leukocytes and form specifically in the epidermal layer of the skin (Munro, 1898; Steffen, 2002). Pustular Psoriasis: Generalized pustular psoriasis (GPP) is an inflammatory skin disorder which occurs in patients with or without psoriasis vulgaris and is characterized by sudden fever and extensive erythemas with pustules and edema, sometimes with life-threatening circulatory and/or respiratory disturbances. Histopathologically, pustular psoriasis shows sponge like array of neutrophils in the upper half of epidermis (Spongiform pustule of Kogoj), collection of neutrophils beneath the cornified layer (subcorneal pustules), and presence of pockets of neutrophils within the cornified layer (intracornal pustules). However, they display less marked epidermal hyperplasia compared to plaque type psoriasis.

ii. Reiter’s Syndrome: Classically, Reiter’s syndrome is a triad of non-specific urethritis, conjunctivitis, and arthritis that follows bacterial dysentery or exposure to a sexually transmissible disease. A male with HLA-B27 has a 20% risk for Reiter’s disease after an episode of Shigella dysentery. The histology of the epithelium reveals parakeratosis, acanthosis, inflammatory infiltration with microabscess formation.

B. Pustular drug eruptions: Drug eruptions are among the most common diseases of the skin. Because of their frequency and the wide spectrum of clinical presentations, drug eruptions are biopsied often and are encountered by histopathologists. The spectrum of the histopathologic presentations of drug eruptions includes a normal basket-woven cornified layer, edema of the papillary dermis, lymphohistiocytic perivascular infiltrate, subcorneal, or intraepidermal microabscesses. Keratinocyte necrosis and vacuolar interface dermatitis with few apoptotic epidermal cells can also be observed.

C. Geographic tongue: It is also known as lingua geographica refers to psoriasiform mucositis of dorsum of the tongue. Clinically, they characteristically show irregular, depapillated, and red patches surrounded by serpiginous white boundaries. Histologic features exhibit irregular thickening of the epithelium with the presence of keratin (at the white serpiginous lines), acanthosis, and neutrophilic infiltration that produce Munro’s microabscesses in the superficial and spinous layers. Thin, elongated rete pegs drop down into the connective tissue.
D. Pustular secondary syphilis: Pustular secondary syphilis is a rare form of secondary syphilis, accounting for about 2% of all secondary syphilis. It has been thought to occur commonly in debilitated patients. Lesions of pustular syphilis consist of crops of pustules which may be follicular, perifollicular, or associated with other forms of secondary syphilis such as framboesiform, papulosquamous, and ulcerated. Histopathology of the biopsy specimen reveals psoriasiform hyperplasia with spongiosis. The presence of papillary dermal edema and basilar vacuolar alterations is observed. There are intraepidermal neutrophilic microabscesses. Scattered necrotic keratinocytes are also seen.

E. Impetigo: Impetigo is a highly contagious superficial bacterial skin infection seen in children. It occurs annually in around 2.8% of children aged up to 4 years old and 1.6% of those aged 5–15 years (National Institute for Health and Care Excellence, 2013). The third most common skin disease seen in children after eczema and viral warts (Sladden and Johnston, 2004), it is usually transmitted by direct contact. Bullous impetigo starts with smaller vesicles, which become flaccid blisters, measuring up to 2 cm in diameter, initially with clear content that later becomes purulent. Non-bullous impetigo presents as vesicles or pustules commonly present around the mouth and nose. The lesions rapidly burst and develop into gold-crusted plaques, typically 2 cm in diameter, which have been said to resemble “glued-on cornflakes”. On histopathology, the vesicopustule arise in the upper layers of epidermis with involvement of the granular layer. There is subcorneal collection of neutrophils forming microabscesses, along with few acantholytic cells which are infrequently observed in the floor of vesicopustule. Gram-positive cocci are usually demonstrated extracellularly or within the neutrophils.

F. Subcorneal Pustular Dermatosis: Subcorneal pustular dermatosis (SPD) of Sneddon and Wilkinson is a rare disorder presenting with recurrent sterile vesiculopustules affecting predominantly the intertriginous and flexural regions of the body, and rarely the extremities, palms, and soles. The exact cause of SPD is not yet proven. There is growing evidence that a network of cytokines (TNF-α, IL-18), synthesized by monocytes and keratinocytes might activate neutrophils and induce their migration, thus promoting SPD. Histopathology shows a subcorneal accumulation of neutrophils, with few spongiform pustules and acantholytic cells. There is a superficial split between the stratum corneum and the layers of epidermis beneath. Few eosinophils may be seen with the neutrophils in the blister.

G. Halogenodermas: Prolonged intake of halogens (bromide, iodides, and fluorides) leads to formation of vegetating, papillomatous plaques on the skin. Histopathologically, there is epidermal proliferation, acanthosis of follicular epithelium, and intraepidermal microabscess formation composed of predominantly neutrophils and eosinophils along with desquamated keratinocytes.

H. Keratoacanthoma: Squamoproliferative tumor of low grade malignancy originating in the pilosebaceous glands. Common among the elderly, fair-skinned males during the 6th to 7th decades of life. Keratoacanthomas occur mostly in sun-exposed skin, predominantly in the face and arms. The lesion has a rapid growth phase of 2–6 weeks followed by period of latency of another 2–6 weeks after which there is a phase of involution of 2–6 weeks which leaves a slightly depressed scar. On histopathologic examination, there is the presence of a crater filled with eosinophilic keratin plug in the center of the lesion. Epithelial “lip” or “marginal buttress” extends over the keratin-filled crater. Acantholytic epithelium and highly keratinized keratinocytes with eosinophilic, glassy cytoplasm is frequently observed. The stroma reveals inflammatory cell infiltrate predominantly composed of neutrophils, which extends into epithelial nests and form microabscesses. Perineural invasion and atypical mitosis are infrequent findings.

I. Granuloma inguinale: Granuloma inguinale is an indolent, progressive, ulcerative, and granulomatous disease of low infectivity caused by Klebsiella granulomatis, previously known as Calymmatobacterium granulomatis (Donovania granulomatosi). It was first described by McLeod in 1882 in Madras. It occurs widely throughout the tropical and subtropical areas of the world and commonly affects the dermis and subcutaneous tissues of the genital, perineal, and perianal regions. It begins with a nodule or papule at the site of bacterial inoculation, which bursts, leading to the formation of an ulcer that grows slowly, bleeds easily and is painless. Histopathologic picture shows extensive ulceration with acanthosis and rete ridge elongation at the margins with massive cellular infiltrate formed predominantly by plasma cells, a paucity of lymphocytes, diffuse sprinkling of polymorphonuclear leukocytes with focal collections, pronounced epithelial proliferation at the margins, and the presence of large, mononuclear cells in the infiltrate which were considered to be pathognomonic. Intracellular and extracellular Donovan bodies of varying morphology and vascular proliferation and dilatation are prominent in the lower dermis. Neutrophilic microabscesses in the epidermis and localized collections of neutrophils in the upper dermis are evident.

J. Fungal Infections:

i. Candidiasis: Oropharyngeal candidiasis (OPC, thrush) is an opportunistic mucosal infection caused by the commensal fungus Candida albicans. C. albicans causes disease when exposure is combined with host susceptibility through immunodeficiency or a breach in normal barriers. Infections caused by Candida are most frequently superficial, occurring on moist mucosal surfaces in individuals
suffering with a mild debilitation. In severely immunocompromised patients, infections can be systemic and are significant because of their associated high mortality.[26] Histopathologically, there is the presence of one or several aggregates or hyphae within the dermis commonly at areas of vascular damage.[19] Hyperparakeratosis with elongated rete ridges is evident. There is chronic inflammatory infiltrate predominantly composed of neutrophils that form microabscesses often identified in parakeratin layer and superficial spinous layer.[14,26]

Neutrophilic microabscesses and small aggregates infiltrate around the hair follicles in the dermis. Of acute or chronic lympho-plasmacytic inflammatory epithelium is devoid of the fungi. There is the presence of fungal hyphae in hair follicles; however, the dermal Hoeppli phenomenon. The histopathological examination shows moderately acanthotic epidermis, with pseudoepitheliomatous hyperplasia. Discrete lymphoplasmacytic infiltrate, with rare polymorphonuclear cells and neutrophil microabscesses, with eosinophilic and amorphous granules, having fine basophilic stippling in its interior, in hematoxylin-eosin staining and Grocott method, associated to surrounding radiated intense eosinophilic substance, denominated as Splendore-Hoeppli phenomenon.[14,26,27]

iii. Dermatophytosis: Dermatophyte fungi of the genera Trichophyton, Epidermophyton, and Microsporum infect human skin, hair, and nails. These types of infections, termed “dermatophytoses” are widespread and increasing in the prevalence on a global scale. Histopathologic examination reveals the presence of fungal hyphae in hair follicles; however, the dermal epithelium is devoid of the fungi. There is the presence of acute or chronic lympho-plasmacytic inflammatory infiltrate around the hair follicles in the dermis. Neutrophilic microabscesses and small aggregates of foreign body giant cells are encountered in well-established lesions.[14]

III. Lymphocytic Microabscesses:

a) Allergic Contact Dermatitis: Contact dermatitis is a common inflammatory skin condition characterized by erythematous and pruritic skin lesions after contact with a foreign substance. The condition can be categorized as irritant or allergic. Allergic contact dermatitis is a delayed hypersensitivity reaction in which a foreign substance comes into contact with the skin; skin changes occur with re-exposure.[26]

b) Lichen simplex Chronicus: Lichen simplex chronicus (LSC) of the anogenital region, is a benign, but extremely uncomfortable disease characterized by skin thickening, hyperpigmentation and increased skin markings resulting from repetitive rubbing or scratching or picking of the skin. It is frequently a manifestation of an itch-scratch cycle.[29]

c) Pityriasis Rosea: Pityriasis rosea (PR) is a benign rash first described by Gilbert in 1860; the name means “fine pink scale.” It is a common skin disorder observed in otherwise healthy people, most frequently children and young adults. It is characterized by a herald patch and the later appearance of lesions arrayed along Langer’s lines (cleavage lines).[12] The smaller secondary lesions of pityriasis rosea follow Langer’s lines. When the lesions occur on the back, they align in atypical “Christmas tree” or “fir tree” pattern. On histopathologic examination, there is hyperkeratosis, focal parakeratosis, irregular acanthosis, exocytosis, spongiosis, extravasated erythrocytes, and perivascular mononuclear cell infiltration in dermis with formation of Munro’s microabscess and spongiform pustule.[12,14,30]

Conclusion

Microabscesses are thus the body’s defense mechanism to cordon off the pathogens. This review involves an attempt to classify the microabscesses and provides an overview of various diseases where microabscess formation is pathognomic and thereby helps in their easy diagnosis. Further studies are required to give a broader insight into the disease specific microabscesses.

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