

DIFFERENTIAL DIAGNOSIS OF THE SCALP HAIR FOLLICULITIS

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SUMMARY – Scalp hair folliculitis is a relatively common condition in dermatological practice and a major diagnostic and therapeutic challenge due to the lack of exact guidelines. Generally, inflammatory diseases of the pilosebaceous follicle of the scalp most often manifest as folliculitis. There are numerous infective agents that may cause folliculitis, including bacteria, viruses and fungi, as well as many noninfective causes. Several noninfectious diseases may present as scalp hair folliculitis, such as folliculitis decalvans capillitii, perifolliculitis capitis abscondens et suffodiens, erosive pustular dermatitis, lichen planopilaris, eosinophilic pustular folliculitis, etc. The classification of folliculitis is both confusing and controversial. There are many different forms of folliculitis and several classifications. According to the considerable variability of histologic findings, there are three groups of folliculitis: infectious folliculitis, noninfectious folliculitis and perifolliculitis. The diagnosis of folliculitis occasionally requires histologic confirmation and cannot be based solely on clinical appearance of scalp lesions. This article summarizes prominent variants of inflammatory diseases of the scalp hair follicle with differential diagnosis and appertaining histological features.

Key words: *Folliculitis; Scalp; Perifolliculitis*

Introduction

Folliculitis is defined as the presence of inflammatory cells within the wall and ostia of the hair follicle, creating a follicular-based pustule. Folliculitis frequently manifests on the scalp, face, neck and buttocks¹. It can be superficial (ostiofolliculitis) or deep (such as furuncle, carbuncle, etc.). When folliculitis lesions are deep, they are usually accompanied by perifollicular inflammation, followed by follicular rupture (perifolliculitis) and resulting abscess.

Classification of folliculitis is both confusing and controversial. There are many different forms of folliculitis and several classifications. According to the considerable variability of histologic findings, there are three groups of folliculitis: infectious folliculitis, noninfectious folliculitis and perifolliculitis (Table 1). The last one, perifolliculitis, is the process in which inflammatory cells surround the follicle without penetrating into it. Histologically, there is a chronic perifollicular lymphocytic inflammation that clinically manifests as the presence of prominent plugs of keratin within the dilated follicular orifice.

Folliculitis is usually characterized by the presence of perifollicular erythema, papules, pustules and vesicles that may be perforated by a hair in acute cases, while chronic-stage lesions present as follicular hyperkeratosis with prominent plugs of keratin within the

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Table 1. Differential diagnosis of the scalp hair folliculitis according Camacho *et al.*²

FOLLICULITIS AND PERIFOLLICULITIS							
Infections/infestations		Noninfectious (folliculitides)			Perifolliculitis		
Superficial (generally suppurative)	Deep (generally granulomatous)	Superficial (generally suppurative)	Deep (generally granulomatous)	Other possibilities (spongiotic folliculitis)	Predominantly lymphocytic		Predominantly granulomatous
					Primary	Secondary	
<u>Fungi:</u> Dermatophytes Pityrosporum Candida	Demodicosis	Acne vulgaris	Acne vulgaris	Pruritic folliculitis of pregnancy	Keratitis pilaris and keratosis spinulosa	Dermoidosis Vitamin C deficiency	Perioral dermatitis Acneiform eruption secondary to syphilis
	Favus and kerion	Rosacea and perioral dermatitis	Lupoid rosacea Acne conglobata Keloidal acne of the neck				
	<u>Bacteria</u> (Bockhart's impetigo) Secondary syphilis	Tinea barbae Majocchi's trichophytic granuloma	Eosinophilic pustular folliculitis Toxic erythema of the newborn	Perforating folliculitis	Lichen planopilaris	Due to lithium	
<u>Viruses:</u> Herpes simplex zoster Molluscum contagiosum	Furuncle Carbuncle Sycosis Acneiform syphilis	Follicular mucinosis Mechanical and chemical traumas Toxicodermas: Halogens Steroids Pseudofolliculitis	Toxicoderma: Halogens Lithium Pseudofolliculitis		Pityriasis rubra pilaris		

follicular orifice¹. Inflammatory diseases of the scalp hair follicle frequently manifest as folliculitis, which may lead to cicatricial or non-cicatricial alopecia, depending on whether or not the perifollicular infiltrate or the etiologic agent spares the hair follicle^{2,3}. It is often difficult to make an adequate diagnosis of scalp hair folliculitis and it usually requires considerable time and effort to recognize and treat the disease.

Besides the noninfectious causes, there are numerous infective pathogens that may cause folliculitis, including bacteria, viruses and fungi. Diabetes mellitus, hyperhidrosis, maceration, tight-fitting clothes, particularly in obese people, inadequate use of topical corticosteroids and halogenated compounds, skin care products and topical hydrocarbons, such as oils or tars (occupational exposure) may precipitate exacerbation of folliculitis. In addition, immunocompromised patients, such as HIV/AIDS patients, may present with various types of folliculitis.

Thus, folliculitis can be classified according to histological features and/or presence of microbiological agents. There are several characteristic histopathologic patterns of hair scalp folliculitis². In acute folliculitis, moderate neutrophil infiltrate can be seen infiltrating follicular epithelium, with the formation of micro- or macro abscesses. Tissue necrosis may be discrete and is usually limited to the follicular infundibulum and the adjacent dermis, or it may be significant, affecting the entire pilosebaceous complex. In chronic folliculitis there is moderately dense lymphocytic infiltrate, usually a granulomatous infiltrate with a foreign body reaction around the keratin. The inflammation is nodular, poorly defined and composed of neutrophils, lymphocytes, histiocytes, and giant cells. Plasmacytic chronic folliculitis predominantly occurs in facial follicles, such as perioral dermatitis, keloidal acne and solid facial edema, folliculitis decalvans and carbuncle^{2,4}.

Other histological forms of folliculitis are predominantly eosinophilic folliculitides and spongiotic folliculitides with characteristic features of infundibulofolliculitis. One distinct form is follicular mucinosis, which often histologically presents in spongiotic folliculitis as keratinocytes get separated by mucin deposits, but dermal mucin deposits can also be found in lupus erythematosus and Fox-Fordyce disease. Destruction of the hair follicle can sometimes ensue, at the 'end-stage' of folliculitis. Suppurative and granulomatous folliculitis generally destroys the follicle leading to cicatricial alopecia. The presence of keratin is important as well. Prominent plugs of keratin within the dilated follicular orifice lead to chronic-stage perifolliculitis.

Folliculitis Due to Infective Agents

The majority of infectious folliculitides are caused by bacteria and fungi (such as *Pityrosporum*, *Demodex*, or other agents)⁵⁻⁷. These clinical variants of folliculitis can be diagnosed by adequate sampling, swabs, KOH examination/or fungal cultures of expressed follicular content. Thereby, diagnosis of these skin changes includes identification of the infection, while therapy includes treatment of the infection.

Viral folliculitis is rare. Folliculitis due to *Herpes simplex hominis* and *varicella-zoster* may occasionally affect the pilosebaceous structures, affecting the beard area in males ('viral sycosis'), manifesting as a group of erythematous papulovesicles or umbilicated vesicles.

Fungal Folliculitis

Dermatophytes, saprophytes such as *Pityrosporum* and *Candida* sp. may cause various folliculitides. Dermatophytic folliculitis is very common and can be caused by all dermatophytes except for *E. floccosum*, *T. concentricum* and *M. persicolor*. *In vitro* cultures can be used for identification of these keratinophilic fungi. We can therefore visualize whether the hair is invaded by concentric destruction, or by perforation with 'perforating organs', indicating *T. mentagrophytes*. There is variation in clinical presentation of dermatophytic folliculitis.

Microsporous ringworm tinea of the scalp (great gray patch tinea) is a non-inflammatory tinea tonsurans of the scalp characterized by typical large round or polycyclic plaques and small satellite patches

covered by grayish scales. Hairs break at 4-6 mm and if they are plucked and placed on a black surface, one can see the surrounding white 'frosted sheath' (spores of the mosaic ectothrix). Hairs fluorescence in Wood's light and can therefore be easily identified².

Trichophytic ringworm tinea of the scalp (black dots tinea) affects only several follicles and manifests with multiple small alopecic areas, which sometimes merge into a larger polycyclic patch with the characteristic interior composed of healthy hair. The most specific sign is the presence of 'black dots', which is very fragile hair infested by endothrix parasite, broken at the level of infundibulum or slightly above. Dermatopathological features of tinea tonsurans are chronic dermatitis with vasodilatation, lymphocytic perivascular infiltrates with occasional spongiosis. Special staining may reveal the fungus in the corneal layer, ecto- or endothrix arthrospores and Adamson's fringe.

Kerion Celsi is an inflammatory tinea of the scalp, generally caused by zoophilic, geophilic or anthropophilic dermatophytes, accompanied by severe inflammatory reaction. It starts as tinea tonsurans and soon becomes indurated and covered with squamous crusts and pustules. In a few weeks it becomes red, painful and warm, from small disseminated pustular plaques to plaques up to 10 cm. Deep suppurative folliculitis and perifolliculitis occasionally reaching the hypodermis can be seen on histopathologic examination. Over time, the infiltrate becomes lymphocytic.

Favus is folliculitis caused by *T. schoenleinii* and it is currently prevalent in Spain. It begins with small erythematous squamous patches with slight infiltration. Underneath the scales, there are pseudopustular yellowish elements in the initial phase of the 'scutulum' of the 'favic cup', somewhat elevated and umbilicated centrally, sulfur yellow in color with soil-like consistency, since they are made up of mycelia and epithelial detritus. The underlying epidermis is emanating rather unpleasant odor caused by detritus, exudate, pus and secondary bacterial infection. Affected hair is lusterless, gray, dry, and fluorescent under Wood's light.

Pityrosporum folliculitis may be seen on the skin in periphery of the scalp. This is a superficial pustulosis which most frequently affects the back and the upper part of the thorax, typical sites for *Pityrosporum orbiculare*, although it can sometimes affect upper limbs as well.

Folliculitis due to *Candida* manifests with occasionally painful papules, nodules and pustules in hairy areas, especially on the scalp and the beard.

Bacterial Folliculitis

Bacterial folliculitis can be superficial or deep, most often caused by *Staphylococcus aureus*, *Streptococcus*, *Proteus*, *Pseudomonas* or coliform bacilli^{1,2}. Predisposing factors include hyperhidrosis, occlusion, maceration, traction depilation, topical corticosteroids and exposure to oils or other chemical substances, or can result from an 'erosive pustular dermatosis' due to *S. aureus* in a patient that underwent scalp operation.

Superficial folliculitis ('osteofolliculitis') is an infection of the follicular ostium, which manifests with painful erythematopapular perifollicular lesions. In localized forms it usually starts with small white-yellow pustules located in follicular orifices surrounded by erythema. It usually manifests on the upper lip in children, whereby *Staphylococcus* can originate from nearby nasal fossae, or can disseminate through the beard and other hairy areas like the scalp, and spread due to scratching⁸.

Deep folliculitis close to the scalp and on the scalp manifests with sycosis, furuncle, carbuncle, etc. When the folliculitis extends beyond the infundibulum, it results in both superficial and deep abscess. As deep abscess does not reach the bulb, usually there is no residual alopecia. There are several types of deep folliculitis, such as furuncle, a deep and necrotizing acute folliculitis, manifesting as an inflammatory nodule with central pus produced by bacterial hair follicle infection¹. It is usually located on the face, neck, perineum, breasts, axillae, gluteal region and thighs. Carbuncle is an accumulation of furuncles with marked inflammation and granuloma¹. It is more often seen in immunocompromised people and in chronic processes. Clinically, there is a swelling containing a large number of pustules, which rapidly open up on multiple sites, with exudation of necrotic tissue and pus through these openings, lasting for several days and leaving a deep scar behind.

Syphilitic folliculitis is a rare manifestation of secondary syphilis, which may appear in the frontal hair implantation line and nasogenian folds in the secondary-tertiary phase, its progression resulting in grouped linear or polycyclic arranged elements^{9,10}.

Noninfectious Types of Folliculitis

Folliculitis decalvans capillitii (folliculitis spinulosa decalvans) is a scarring folliculitis of the scalp that leads to alopecia^{1,4}. The nature of folliculitis decalvans is unclear, but *S. aureus* is frequently isolated from the culture¹¹. It primarily affects adult men, often immunosuppressed patients, either with diabetes mellitus, chronic renal disease, gammopathy or iatrogenic immunosuppression. The inflamed follicles coalesce, progressing in depth and laterally, forming follicular papulopustules, slightly pruriginous, causing several small patches of cicatricial alopecia with follicular fibrosis. The skin becomes atrophic, resembling pseudopelade, while at the periphery follicular pustules continue to form^{1,12}. The disease is chronic and difficult to stop. The acute stage of folliculitis decalvans capillitii resembles highly inflammatory tinea capitis, while the scarring stage mimics all other types of scarring alopecia, including discoid lupus erythematosus and lichen planus. Histopathologically, early lesions are marked by neutrophilic folliculitis and frank abscesses within the follicles, while nonspecific scarring alopecia ensues later. Neutrophilic follicular abscess can be seen around the follicles and sweat glands, which rapidly leads to a granulomatous infiltrate with a large number of plasma cells².

The disease is resistant to therapy but beneficial effects of rifampicin (600 mg/day/7 weeks) and oral fusidic acid (1.5 mg/day/3 weeks or topical 1.5% cream) with the addition of zinc sulfate (400 mg/day/6 weeks)² have been reported. Dapsone (100-150 mg/day/several months) is worth trying. Therapy with systemic antibiotics, based on culture results, shows only transient effect and is often disappointing. In severe cases, short courses of systemic corticosteroids are indicated to reduce inflammation. Topical antibacterial therapy brings little relief, e.g., lotions or solutions containing antibiotics such as erythromycin, often combined with topical corticosteroids¹.

Folliculitis sclerotisans nuchae (keloidal folliculitis), a scarring form of chronic folliculitis that manifests as follicular-based papules and pustules that eventually result in keloid-like lesion. It is mostly located on the neck, and mainly occurs in dark skinned people and Caucasians, especially in association with seborrheic dermatitis and sometimes with alopecia. Histological features include dense lymphoplasmacytic inflamma-

tory infiltrates around thick, compact, keloidal collagen bands, which correspond to destroyed follicles or sometimes peripilar granulomas. Therapy includes topical steroids, antibiotics, retinoids, cryotherapy, etc.

Tufted hair folliculitis manifests with a 'tuft' of hair made up of 5 to 20 hairs, which appear normal in the follicular openings that remain in the alopecic plaque¹³. It also appears to be caused by *S. aureus*, although it is possibly related to immunodeficient states or is of 'nevoid origin'. It manifests as exudative patches on the parietal or occipital areas, which tend to progress peripherally leaving cicatricial alopecia in the center. Histologically, there is a perifollicular infiltrate composed of lymphocytes, eosinophils, plasmocytes and neutrophils with inflammation around the upper portion of the follicle, which does not affect the bulb. Topical antiseptics and systemic antibiotics (erythromycin, tetracyclines and doxycycline) improve the inflammatory component. Use of shampoos with mineral oils and keratolytics is also advised.

Erosive pustular dermatitis is a pustular dermatosis with erosions and crusts covering the scalp, generally caused by *S. aureus*, or rarely *S. epidermidis*, *Pseudomonas aeruginosa*, coagulase negative *Staphylococcus*, *Diphtheroids*, *Coliforms* and *Proteus mirabilis*. *Candida* sp. and other fungi have also been cultured in some cases, but these seem to reflect secondary colonization^{14,15}. Follow-up is very important since carcinomatous transformation may occasionally occur. Despite the nonspecific histological features, there is chronic perifollicular inflammatory infiltrate composed of lymphocytes, plasmocytes and giant cells¹⁵. It has been suggested that exposure to ultraviolet light may be a predisposing factor. Treatment includes nonsteroid anti-inflammatory agents, topical and systemic antibiotics. Superficial bacterial folliculitis usually responds well to antiseptic baths such as povidone iodide and chlorhexidine gluconate, to astringent aluminum subacetate compresses and to topical antibiotics (polymyxin B sulfate, neomycin sulfate, fusidic acid and mupirocin). Deep folliculitis requires treatment with systemic antibiotics (e.g., cephalosporins, azithromycin, semisynthetic penicillins).

Acne conglobata represent a chronic inflammatory process which manifests with nodules, cysts, abscesses, draining sinus tracts, comedones and multiple kel-

oid scars, predominantly located on the face, throat, shoulders, chest, back, gluteal region, or beside scalp area. In the areas with apocrine glands, like the axillae and the groin, this causes hidradenitis suppurativa or dissecting cellulitis in the scalp region. Histologic examination shows deep folliculitis, progressing towards the formation of abscesses and, with subsequent reepithelialization of the follicular unit, leaving a sinus and fibrosis. Hidradenitis suppurativa, dissecting cellulitis and acne conglobata often coexist as a follicular occlusion triad.

Acne necrotica (acne varioliformis, necrotizing lymphocytic folliculitis) is a rare, chronic pruritic eruption of multiple follicular papules which rapidly progress into umbilicated vesiculo-papules or pustules, sometimes with a central crust, mostly located in the temporal areas, nape and the frontal implantation line². Most patients are adult women. Over weeks, it slowly involutes, leaving a varioliform scar¹. Histopathologically, there is epidermal damage with mixed dermal inflammatory infiltrates in more advanced crusted lesions, while lymphocytic perifollicular infiltrates and other adnexal structures in early lesions sometimes cause spongiosis. Excoriated bacterial folliculitis or rarely herpes simplex virus is considered in its etiopathogenesis. Isotretinoin has good therapeutic results and corticosteroids often bring immediate improvement. Topical therapy is generally ineffective, although potent topical corticosteroids or benzoyl peroxide may be helpful¹.

Acneiform eruptions can sometimes be seen on the scalp, such as in case of chloracne. Chloracne are caused by the exposure to chlorinated compounds which can be found in industry, usually in electrical conductors, and therefore may represent an occupational disease. Clinically, it manifests with cysts and comedones that are usually open, covering temporal areas, cheeks, throat and retroauricular region¹⁶. The treatment includes topical/oral antibiotics, topical/oral retinoids excision, laser ablation, and drug withdrawal.

Lichen planopilaris is a form of lichen planus located on the scalp, characterized by perifollicular erythema and small keratotic pruriginous follicular papules resulting in small alopecic plaques¹⁷⁻¹⁹. It is more common in the hair implantation line, both the frontal hair implantation line and the occipital line, although

it can also appear on any other part of the scalp. The disease occurs more frequently in women between age 30 and 60². There are three different types of the disease according to clinical manifestations and histopathologic features: keratotic follicular papules and inflammatory lichenoid infiltration in the follicular epithelium; violaceous or erythematous plaques, some with follicular prominence and an inflammatory lichenoid infiltrate and follicular papules associated with perifollicular and interfollicular inflammatory infiltrate resulting in fibrosing alopecia².

Rarely, patchy cicatricial alopecia of the scalp is associated with non-scarring alopecia of the axillary and pubic areas, and grouped spinous follicular papules resembling keratosis pilaris on the trunk and extremities (Piccardi-Lassueur-Graham-Little syndrome).

Pityriasis rubra pilaris is characterized by multiple follicular papules, sometimes with a central black corneal plug, which generally accompanies or precedes seborrheic dermatitis of the scalp^{20,21}. This disease manifests with a yellow-orange erythemasquamous plaques, often developing on the scalp, with obvious islands of sparing and palmoplantar hyperkeratosis. Clinically it shows multiple follicular papules with a central corneal plug on the lateral surfaces of the thighs and arms, gluteal region and forearms. There is a corneal plug which occludes the pore and dilates the follicular ostium, hyperkeratosis, dermal vasodilatation with chronic perivascular and periadnexal inflammatory infiltrate. Therapy may include systemic retinoids, topical corticosteroids and retinoids, mild keratolytic agents, etc.

Keratosis pilaris atrophicans is characterized by different clinical presentations which may be classified in two groups: hair keratosis and pseudopelade. There are hair keratoses that cause atrophy in glabrous skin of the face, such as folliculitis atrophicans faciei and atrophoderma vermiculatum; however, keratosis follicularis spinulosa decalvans should be referred as pseudopelade or 'scarring follicular keratosis'²². The difference between keratosis pilaris and these four dermatoses is the greater inflammatory component seen in the latter, as well as intense clinical erythema²³.

Keratosis follicularis spinulosa decalvans, a type of X-linked recessive genodermatosis, causes cicatricial alopecia of the scalp and eyebrows during infancy or adolescence^{2,24}. It begins with corneal filiform follicu-

lar plugs on the nose and cheeks, which later expand to the scalp, neck and extremities causing atrophy in these areas. The atrophic aspect of the cheeks and cicatricial alopecia on the scalp, eyebrows and eyelashes make all these children look very much alike. At the beginning, it is accompanied with milium cysts on the cheeks, or possible atopy, corneal opacities, photophobia, conjunctivitis, keratitis, blepharitis and palmoplantar hyperkeratosis. Oral retinoids have been reported to lead to improvement.

Eosinophilic pustular folliculitis (Ofuji's syndrome) is a rare dermatosis of unknown etiology, characterized by recurrent episodes of eruptive sterile follicular papulopustules in seborrheic areas, accompanied by leukocytosis and eosinophilia. It is most common in men and the general population in the third decade of life, in which the papulopustules tend to form round ring-shaped or polycyclic plaques with peripheral progression and central clearing, with residual scaling and pigmentations. The histopathologic features include infundibular follicular pustules mainly composed of eosinophils and polynuclears. Topical corticosteroids or tacrolimus ointment can be prescribed.

Follicular mucinosis (alopecia mucinosa) on the scalp is characterized by the presence of follicular papules and indurated plaques associated with alopecia. The disease is accompanied with mycosis fungoides and angiolymphoid hyperplasia with eosinophilia, less frequently with chronic discoid lupus erythematosus and Goodpasture's syndrome. There is another form of idiopathic or primary origin (mainly affects children and young adults) presenting by grouped follicular papules. They show a tendency to coalesce into more or less well defined erythematous plaques with dilated follicular orifices on the surface of these plaques, as well as alopecia.

Follicular mycosis fungoides is characterized by the presence of infiltrated follicular papules located on the face, neck, trunk and extremities, and sometimes on the scalp²⁵. Histopathologically, there are perifollicular and intrafollicular infiltrates of atypical lymphocytes with convoluted nuclei. One must differentiate this disease from follicular lymphomatoid papulosis, especially type B or lymphocytic papulosis. It should be treated with nitrogen mustards and/or electron therapy, but also with topical corticosteroids, phototherapy, etc.

Perifolliculitis

The prominent disease in this group is perifolliculitis capitis abscedens et suffodiens, but, according to some authors, in this group keratosis pilaris atrophicans, lichen planopilaris, pityriasis rubra pilaris, perioral dermatitis, etc. are also included².

Perifolliculitis capitis abscedens et suffodiens is a severe destructive folliculitis with sinus tracts and fistulae leading to scarring alopecia, and is often part of the acne inversa spectrum¹. It occurs almost exclusively in young men and may be more common in the black population. The cause is unknown, but is often associated with recurrent gram-negative infections. Lesions begin as multiple, firm scalp nodules that rapidly develop into fluctuant ridges that eventually discharge purulent material leading to hair loss¹. The folliculitis manifests on the scalp or nape leading to persistent inflammation and destruction of follicles. The skin may be covered with crusts and scales; pressure on one fluctuant area may result in multiple follicular openings^{26,27}. Flares are very frequent and the disease is chronic. Histopathologic features reveal moderately dense, perifollicular lymphocytic inflammation affecting the lower half of the dermis and extending down into the subcutaneous fat, necrosis and scarring. Smear of pustular content is useful, and often a wide variety of organisms can be found, frequently gram-negative bacteria prevail. The disease is extremely difficult to treat, e.g., broad-spectrum antibiotics, systemic corticosteroids (40–60 mg/day/tapered over 3 weeks), or isotretinoin (1 mg/kg /day/12–20 weeks) may help^{1,28}. Topical therapy includes disinfectant solutions such as chinisol. Epilation of involved follicles may reduce inflammation and its spread. When the inflammatory process resolves, surgical excision of scarred areas is recommended¹.

Conclusion

Differential diagnosis of scalp hair folliculitis is broad. It is crucial to recognize and exclude an infectious diseases and to confirm clinical diagnosis by histopathologic analysis. Repeated swabs, KOH examination/or fungal cultures and tissue sampling for histopathologic analysis are often necessary to confirm the diagnosis of scalp hair folliculitis, which can represent a significant burden to everyday clinical

practice. However, only such adequate diagnostic procedures result in correct diagnosis and successful therapeutic outcomes.

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Sažetak

DIFERENCIJALNA DIJAGNOZA FOLIKULITISA VLASIŠTA

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Folikulitis vlasišta je relativno česta pojava u dermatološkoj praksi koja zbog nedostatka jasnih smjernica predstavlja značajan dijagnostički i terapijski izazov. Općenito se upalne bolesti pilosebacealnog folikula u vlasištu najčešće manifestiraju kao folikulitis. Postoje brojni infektivni agensi koji mogu uzrokovati folikulitis vlasišta, kao što su bakterije, virusi i gljive, kao i mnogi neinfektivni uzroci. Nekoliko neinfektivnih bolesti mogu imati sliku folikulitisa u vlasištu, kao što su folliculitis decalvans capillitii, perifolliculitis capitis abscondens et suffodiens, erozivni pustularni dermatitis, lichen planopilaris, eozinofilni pustularni folikulitis i drugo. Klasifikacija folikulitisa također je nejasna i kontroverzna. Tako se navode različiti oblici folikulitisa i nekoliko njihovih klasifikacija. Prema ključnim varijacijama histoloških nalaza postoje tri skupine folikulitisa: infektivni folikulitis, neinfektivni folikulitis i perifolikulitis. Dijagnoza folikulitisa povremeno zahtijeva histološku potvrdu i ne može se osnivati samo na kliničkoj pojavi lezija vlasišta. U ovom članku su obuhvaćene moguće upalne bolesti folikula vlasišta s mogućim diferencijalnim dijagnozama i pripadajućim histološkim značajkama.

Ključne riječi: *Folikulitis; Koža glave; Perifolikulitis*