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Literature Review
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The Use of Dermoscopy to Distinguish Between Pityrosporum Folliculitis and Acne Vulgaris

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ABSTRACT

Background: Both *Pityrosporum folliculitis* (PF) and *Acne vulgaris* (AV) are chronic inflammations of pilosebaceous follicles but with different etiologies. Similar findings may lead to a misdiagnosis that worsens symptoms, especially if PF is treated with antibiotics meant for AV. A simple kalium hydroxide (KOH) examination helps to differentiate PF and AV, but it is not always readily available in a clinical setting**. Purpose**: To find an alternative, practical tool to confirm the diagnosis of PF and AV**. Review:** The dermoscopy is a handheld microscope equipped with 10x magnification and a light source for microscopic visualization of the subcutaneous structures. The monomorphic lesions in PF appear as hypopigmented, round lesions with coiled/looped hair in the center. Dermoscopy images of AV vary according to their polymorphic manifestations. A non-inflammatory AV shows a yellow-brown blockage in the center, while an inflammatory AV is depicted as round, whitish lesions with thin brownish borders and erythematous marginal lesions. Pustular AV appears as raised lesions with indistinct borders, white or yellowish in the middle, and surrounding reddish borders. **Conclusion:** Dermoscopy helps to see skin structures invisible to the naked eye and thus helps to diagnose PF and AV.

Keywords: Dermoscopy, Pityrosporum folliculitis, Acne vulgaris.

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BACKGROUND

Pityrosporum folliculitis (PF), also known as *Malassezia folliculitis*, is a chronic pruritic eruption in the pilosebaceous follicle due to *Malassezia sp.* infection.[1,2] *Acne vulgaris* (AV) is a chronic inflammation of the pilosebaceous follicle that is often associated with infection with *Cutibacterium acnes*.[3] Both infections may clinically appear as erythematous papules and pustules on the follicle, although AV can further develop into the formation of follicular plugs, and, hence, microcomedones.[4] Despite the clinical similarity, both require different treatments. The etiology of PF is fungal infection; thus, oral or topical antifungals may alleviate the symptoms.[1] On the other hand, AV, which pathogenesis involves the overgrowth of bacteria, requires the help of antibiotics. Misdiagnosing PF as AV and giving antibiotics as treatment may worsen the symptoms.[1,4] The antibiotic is hypothesized to disturb the normal flora of the skin and hence give room for the growth of *Malassezia sp.,* the pathogen of PF.[2,4]

A simple kalium hydroxide (KOH) examination helps to differentiate PF and AV. A microscope, a tool used to perform a KOH examination, is not always readily available in a clinical setting.[5,6] Therefore, an alternative, practical tool is needed to confirm the diagnosis and thus provide a better prognosis for the patient.Dermoscopy is a non-invasive diagnostic tool that helps clinicians observe skin lesions sub-macroscopically and show features that will not be accessible to the naked eye. The use of a dermoscopy, which initially focused more on skin pigment tumors, has also been used for other

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diseases based on the shape of the lesion, vascular vessels, scale pattern, color, follicular abnormalities, and others.[7] A retrospective report by Jakhar D. *et al.*[8] in 2018 showed the possibility of using dermoscopy as a tool to distinguish PF from AV. Therefore, we aim to further study the possibility of using dermoscopy as a diagnostic tool for differentiating PF and AV.

REVIEW

Pityrosporum folliculitis (PF) is a pruritic eruption of pilosebaceous follicles caused by the fungal infection of *Malassezia sp*., especially *Malassezia furfur*.[1,2,9] Malassezia triggers an inflammatory reaction with lymphocytes, histiocytes, and neutrophils, along with a rupture of the follicular epithelium.[6,10] The predisposing factors include occlusion of hair follicles and skin due to skin care products and cosmetics, warm and sweaty skin, sun exposure, Human Immunodeficiency Virus (HIV) infection, diabetes mellitus, and the use of corticosteroids or immunosuppressants.[2,6,10] The incidence increases by the year, affecting predominantly teenagers of young adult males.[2] Its similarity to other skin diseases, especially AV, hinders the diagnosis and treatment of PF, thus affecting the quality of life of PF patients.Malassezia is a commensal dimorphic lipophilic fungus found in 40-80% of the stratum corneum, especially in the infundibulum of the hair follicle. [2,10,11] In the condition of yeast, this fungus is commonly called Pityrosporum, and in the form of mycelia, it is called Malassezia. The yeast form of the Malassezia fungus is cited as the cause of the development of PF.[12] The focus of the link between Malassezia and folliculitis lies in follicular occlusion and the overgrowth of the associated fungi.[13] Malassezia can hydrolyze triglycerides from sebum into free fatty acids, thereby inviting inflammatory reactions.[10] However, another study suggests that the produced fatty acids are insufficient to cause inflammatory reactions.[13] The activity of Malassezia in the hair follicle gives rise to the itchy, monomorphic appearance of papular or pustular eruptions in PF. Pityrosporum folliculitis lesions can be found mainly on the upper trunk, then on the neck, back, and sides of the extensor arms.[12,14]

Microscopic examination with a 10% KOH stain is sufficient to confirm the diagnosis of PF,[1,10] although Tu *et al.*[11] suggested the use of an additional gram stain to simultaneously diagnose the possibility of bacterial folliculitis or mixed infection. A biopsy is not necessary for diagnosis.[1,10] A biopsy of pityrosporum folliculitis shows dilated follicles with many round or oval yeast cells and inflammation of the hair follicles, or perifollicular, in the dermis.[1,11]. Pityrosporum folliculitis is treated with oral and/or topical antifungals. Oral antifungal in the form of ketoconazole 200 mg daily for 4 weeks, fluconazole 150 mg every week for 2-4 weeks, and itraconazole 200 mg daily for 2 weeks. Oral ketoconazole is still widely used in our clinical setting, despite the possible side effects on the liver and adrenal glands. Fluconazole may be a safer option with better disease resolution (100% vs. 91%). Alternative therapy with topical antifungals such as 2% ketoconazole shampoo, 2.5% selenium sulfate lotion, and 0.77% ciclopirox olamine cream shows promising results with 100% case resolution.[4] Administration of both oral and topical antifungals may provide better efficacy with fewer side effects.[12]. Pityrosporum folliculitis has a high rate of recurrence. Using 2.5% selenium sulfide lotion once a week, 2% ketoconazole cream once a week, or 2% ketoconazole shampoo 2-3 times a week can prevent recurrences of PF. Recurrence can also be prevented by consuming 400 mg of itraconazole or 200 mg of fluconazole once a month.[1]

Acne vulgaris is a self-limiting chronic inflammation of the pilosebaceous follicular unit.[3] The prevalence of acne peaks in mid-to-late adolescence, affecting more than 85% of adolescents, and then gradually decreases. However, acne can persist into the third decade or more, especially in women.[15,16] Acne is the most common complaint for patients visiting the cosmetic division of an outpatient skin clinic in Cipto Mangunkusumo General Hospital in Jakarta (45%)[3] and in dr. Soetomo General Academic Hospital, Surabaya (31.88%).[17] The average cases per year during the 2008-2010 period ranged from 2,204 in Cipto Mangunkusumo General Hospital to 1,149 in dr. Soetomo General Academic Hospital. The pathogenesis of AV involves the activity of *Cutibacterium acnes*, increasing sebum production, the proliferation of epidermal follicles, and inflammatory reactions. The complex pathogenesis of AV gives rise to its polymorphic manifestations. *Cutibacterium acnes*, formerly known as *Propionibacterium acnes*,[18] is a gram-positive, anaerobic bacteria that constitutes 90% of the commensal bacteria in the pilosebaceous unit. [19,20] The pathogenesis of acne has always been associated with the activity of these bacteria in the production of lipase, protease, hyaluronidation, and chemotaxis factors, which stimulate the body to form anti-Propionibacterium antibodies, causing an inflammatory chain. [19,21] Increased concentrations of *C. acnes* are often found in individuals with cases of AV.[18,19,21] Ahluwalia *et al.*[18] found that the concentration of *C. acnes* was higher on the forehead and nose compared to the cheeks and chin. Although *C. acnes* is common, it does not rule out the possibility that other species, such as Staphylococcus, Corynebacteria, other gram-negative bacteria, and Malassezia, may also play a role in the pathogenesis of acne.[18,20,21]. Sebum is produced by the sebaceous glands to lubricate the skin, distribute antioxidants, and express antimicrobial proteins.[22] However, excessive sebum production is associated with the pathogenesis of acne,[23] in which triglycerides from sebum are broken down into free fatty acids by *C. acnes*, helping

the metabolism of these bacteria.[19,20] *C. acnes* then produces diacylglycerol acyltransferase which in turn increases sebum production.[20] This series of activities causes an inflammatory process and plays a role in the formation of blackheads in the pathogenesis of acne.[19,20] Therefore, acne lesions are commonly found in areas rich in sebaceous glands, such as the face, back, chest, and shoulders.[19]

Along with excessive sebum production, there is stimulation of keratinocytes in the follicles with an unknown cause. Hyperproliferation of epidermal follicles causes blockage at the opening of the follicles, causing the accumulation of keratin, sebum, and bacteria in the follicles, thus forming microcomedones. Clinically, microcomedones appear as blackish papules when exposed to air or whitish papules when buried in the skin, measuring ≤1 mm.[19]. The microcomedones continue to enlarge in the follicle, causing the follicular wall to break. The rupture of the follicular wall causes the contents of the microcomedo, which are keratin, sebum, and bacteria, to enter the dermis and cause an inflammatory process. The shape of the lesion varies accordingly, from reddish papules to pustules, nodules, and cysts. The clinical findings alone are sufficient to support the diagnosis of AV.[19] The management of AV is related to the pathophysiology of acne, which is to treat the keratinization problem of follicles, reduce sebaceous gland activity, reduce the population of *C. acnes* bacteria, and treat inflammatory problems. Topical ingredients that can be applied to acne include sulfur, salicylic acid, azaleic acid, benzoyl peroxide, topical antibiotics, and retinoids. Systemically orally, antibiotics and antimicrobial agents, oral contraceptives, corticosteroids, gonadotropin-releasing hormone agonists, antiandrogens, and isotretinoin can be given. Other options include phototherapy and laser use, intralesional corticosteroid injections, acne surgery, and diet.[19] A dermoscopy, or dermatoscopy, is a handheld microscope equipped with 10x magnification and a light source for microscopic visualization of the subcutaneous structures. Dermoscopy helps to better visualize the distribution of skin pigment, layer surface skin keratin, vascular patterns, lesion margins, and ulceration. This makes dermoscopy useful not only for pigmented lesions but also for various skin conditions, including inflammatory diseases, infectious diseases, skin diseases, and skin reactions to treatment.[24]

Most of the light is reflected by the stratum corneum to be received by the retina. Light from the stratum corneum floods the retina, preventing the observer from visualizing the deeper layers of the skin. A clinical examiner without the aid of a dermoscopy can only see the outer surface of the skin. With a dermoscopy, the observer can see not only the structures in the outer layer of the skin but also the deeper structures, depending on the polarization of the dermoscopy. A non-polarized dermoscopy (NPD) showed a more translucent stratum corneum, to reveal deeper skin structures. Non-polarized dermoscopy requires direct skin contact. Orthokeratosis and cysts resembling milia are better seen in an NPD. A polarized dermoscopy (PD) uses 2 polarizing tools to capture backlight in the deeper layers of the skin. The mechanism enables PD to see even deep layers of the skin, such as abnormalities in blood vessels. Unlike NPD, PD requires no fluid immersion or direct contact, so it does not have a blanching effect on the blood vessels. Polarized dermoscopy is better at providing images of blood vessels, vascular blush, and areas that are whitish and shiny, such as scars or crystalline.[7]

Color is a very important clinical marker. The skin lesions' color depends on the chromophore's location and density. Melanin is the main chromophore in the skin. When melanin is located more superficially in the stratum corneum or slightly below it, it will appear black in the dermoscopy. If the location gets deeper, until it touches the dermis epidermal junction, it will appear light brown or dark brown, depending on the density of melanin. If it is deeper until it touches the dermis, the melanin will appear bluish or gray. Other chromophores are hemoglobin, which gives the skin its red color, and collagen fibers in the dermis, which gives it its white color.[7] Dermoscopy helps to visualize skin lesions. A few structures that can be observed through a dermoscopy are pigment networks, blotches, dots, globulus, streaks, hypopigmented areas, crystals, regression structures, blue-white cells, vascular structures, milia-like cysts, crypts, pseudo follicular openings, fissures and ridges, fingerprint-like structures, moth-eaten borders, leaf-like area, spoke-wheel-like structures, ovoid nests, lacuna, and others.[7]

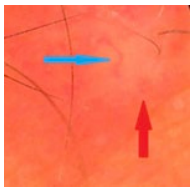
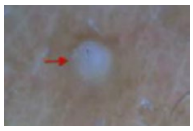
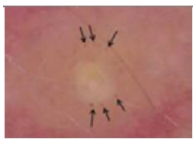
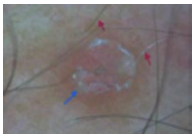
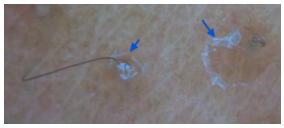
DISCUSSION

Both PF and AV cause follicular obstruction and swelling in the infundibulum, giving rise to relatively similar clinical manifestations of papules or pustules in the follicles with signs of inflammation. A study done by Xu and Li[20] links the role of *Malassezia sp.* in the pathogenesis of acne. *Malassezia sp.* has lipase activity that is 100x higher than that of *C. acnes*, allowing Malassezia to hydrolyze the triglycerides in sebum and help the formation of free fatty acids, which is the precursor to the pathogenesis of acne.[20] Not only may the infection of *Malassezia sp.* in PF hypothetically precede AV formation, but these two conditions can also be found together.[8] The close relationship between the two diseases does not mean that they require the same therapy. Thus, finding a simple and fast diagnostic tool that is capable of differentiating PF and AV is very important in making the diagnosis. Before performing a dermoscopy examination, doctor or other medical personnel needs to establish a clinical diagnosis first. The main difference between PF and acne is mainly in the

lesions' variety. While PF is dominated by the findings of papules or pustules in hair follicles with signs of inflammation, AV tends to have polymorphic clinical manifestations ranging from elevated with a white base (papules), elevation with yellowish white in the middle (pustules), or brownish yellow lesions (blackheads).

Lesions in PF appear as monomorphous perifollicular papules or pustules. The dermoscopic image of PF as seen in Table 1 showed folliculocentric lesions with erythema base (100%). Studies suggest the findings of dotted/linear/tortuous vessels (88.9%), dirty white scaling (77.8%), hypopigmentation of proximal hair shaft (64.4%), coiled/looped hair (57.8%), and broken hairs (13.3%).[26] The hair follicles appear hypopigmented, possibly due to a fungal invasion of the hair. The lesions that heal leave a brownish appearance.[8,25]

Table 1. Dermoscopy images of pityrosporum folliculitis[8,25,26].

No	Dermoscopy Images	Descriptions
1.		Folliculocentric papule as indicated with round, white center, with perilesional erythema
2.		Folliculocentric pustule as indicated by yellowish white lesion, with perilesional erythema
3.		Peripheral, regularly distributed dotted vessels
4.		Dirty white scale
6.		Coiled/looped hairs with perifollicular erythema and scaling

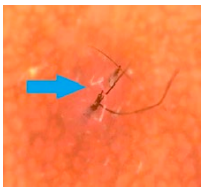
7.		Broken hair

Table 2. Dermoscopy images of acne vulgaris.[8,27,28]

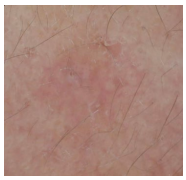
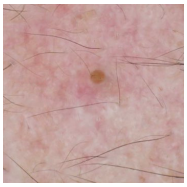
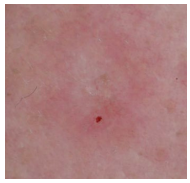
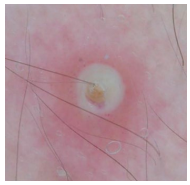
No	Dermoscopy Images	Descriptions
1.		Closed comedo
2.		Open comedo
3.		Papule
4.		Pustule

Table 3. Differences in the findings of the dermoscopy on pityrosporum folliculitis and acne vulgaris. [8,27,28]

	Pityrosporum folliculitis	Acne Vulgaris
Location of lesions	More often on the upper body, neck, back, and extensor sides of the hands	More often on the face, back, chest,and shoulders
Variation of lesions	Monomorphic	Polymorphic
On the dermoscope:		
Papules	+ (round, white center)	+ (round, white center)
Pustule	+ (yellowish white)	+ (yellowish white)
Hair follicles	+ (coiled hair / loop, hypopigmented follicle)	-
Inflammation	+	+
Scales	+	-
Blackheads	-	+ (Brownish yellow)

Acne can be both non-inflammatory and inflammatory. Dermoscopy images of AV, as shown in Table 2, may show a yellow-brown blockage in the center with rare signs of inflammation in non-inflammatory AV lesions, such as open and closed comedones.[8,27,28] The dermoscopy of inflammatory lesions in AV is depicted as round, whitish lesions with thin brownish borders and erythematous marginal lesions, which can show blood vessels and inflammation with a yellowish base.[8,27] Pustular AV appears as raised lesions with indistinct borders, white or yellowish in the middle, and surrounding reddish borders.[8,27,28] The difference between both PF and AF is further summarized in Table 3.

The main modalities for diagnosing PF and AV, including KOH and culture, are considered impractical and often not available in many clinical settings. A simple, readily available diagnostic tool may help establish the diagnosis of PF or AV. This gives room for a dermoscopy that is considered non-invasive and practical to be an alternative investigation in diagnosing PF, especially to differentiate it from AV.

This paper demonstrates that PF can be distinguished from acne on a dermoscopy. The monomorphic lesion of PF shows a damaged (coiled/looped) hair follicle with papules or pustules and signs of surrounding inflammation. Polymorphic lesions on acne have a variety of features, ranging from elevations with a white base (papules), elevations with yellowish white in the middle (pustules), or brownish-yellow lesions (blackheads). Therefore, dermoscopy may be considered a modality in locations where neither KOH nor culture is possible.

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