Review Article

Malassezia Folliculitis: A Review Article

Anon Paichitrojjana MD[1]

[1] School of Anti-Aging and Regenerative Medicine, Mae Fah Luang University, Bangkok, Thailand

Malassezia folliculitis (MF) results from overgrowth of *Malassezia* yeasts, which are normal skin flora. This condition is caused by a loss of balance between *Malassezia* yeasts, microenvironment, and human immunity. MF presented as small, monomorphic, itchy papules, and pustules particularly on hair line, face, and upper trunk. Because the appearance and location of MF are similar to acne, this makes it difficult to distinguish between the two conditions. MF is an under-recognized disease that is often misdiagnosed as acne vulgaris, recalcitrant acne, neonatal cephalic pustulosis or neonatal acne, and steroid acne. In addition, MF can occur simultaneously with acne vulgaris. The definite diagnosis is based on clinical presentations, direct microscopy, histopathological examination, and good response to antifungal treatments. MF may persist for years without complete resolution with standard acne treatment. Dermatologists should be aware of this disease when encountering patients with acne problems to provide proper management.

Keywords: *Malassezia* folliculitis; Acne vulgaris; Recalcitrant acne; Neonatal acne; Steroid acne

Received 25 August 2021 | Revised 31 January 2022 | Accepted 31 January 2022

J Med Assoc Thai 2022;105(2):160-7

Website: http://www.jmatonline.com

Malassezia yeast is a normal flora of the human skin surface and only causes disease under specific conditions. It has been linked to skin diseases including seborrheic dermatitis, dandruff, *Malassezia* folliculitis (MF), and pityriasis versicolor[1,2]. MF, also known as *Pityrosporum* folliculitis (PF), typically manifests as a pruritic, follicular papulopustular eruption distributed on the upper trunk of young adults.

Malassezia

Malassezia is an anthropophilic fungus, classified as one of normal skin flora. This fungus can grow in a yeast phase as well as in a mycelial phase. In normal condition, *Malassezia* is usually found in the yeast form and live in the infundibular portion of the hair follicle. It survives on the lipid composition of sebum as nutrient for growth and proliferation. *Malassezia* belongs to Fungi kingdom, phylum Deuteromycota, class Blastomycetes, order Cryptococcales, and the family Cryptococcaceae. *Malassezia furfur* species was first described in 1889. Early taxonomic classifications of *Malassezia* yeasts are performed under limited conditions. They are distinguished only by microscopic findings from skin samples. Microbiological inoculation became possible after the lipophilic nature of this fungus was discovered in 1939 and was used as a tool to identify *Malassezia* species. After that, *M. furfur* (serotype A, B, C), *M. pachydermatis*, and *M. sympodialis* were identified. Guého et al isolated four new species, *M. globosa*, *M. obtusa*, *M. restricta*, and *M. slooffiae* based on morphology, ultrastructure, physiology, and molecular biology[3]. Since 2002, the use of molecular biology has made it possible to discover new species, such as, *M. dermatis*, *M. japonica*, *M. yamatoensis*, and *M. nana*[4-7]. The genus *Malassezia* currently consists of 22 species of *Malassezia*, which have been isolated from both human and animal skin, and new species continue to be discovered. Studies have investigated the distribution of *Malassezia* yeasts on normal human skin according to body region. Lee et al conducted the quantitative study on the distribution of *Malassezia* yeasts in a healthy Korean population and found that *M. restricta* was mostly found on the scalp and forehead, whereas *M. globosa* was the predominant species on the chest. Quantitative analysis also showed that the yeast count per unit area of skin was higher for the chest and scalp and lower for the upper arm and thigh. The *Malassezia* yeast count

Correspondence to:

Paichitrojjana A.

School of Anti-Aging and Regenerative Medicine, Mae Fah Luang University, 36/87-88 PS Tower 25Fl, Asoke Road, Sukhumvit 21, Klong Toey Nua, Wattana, Bangkok 10110, Thailand.

Phone: +66-81-9343050

Email: anonpaic@gmail.com

How to cite this article:

Paichitrojjana A. *Malassezia* Folliculitis: A Review Article. J Med Assoc Thai 2022;105:160-7.

DOI: 10.35755/jmedassocthai.2022.02.13268

on the scalp, forehead, and chest was the highest in the 11 to 30 years age group[8]. *Malassezia* yeast becomes pathogen in certain predisposing factors such as immunosuppression, hot and humid climates causing increased episodes of sweating, antibiotic treatment, and corticosteroid therapy[9-12]. *Malassezia* yeasts are the causative agents of pityriasis versicolor, seborrheic dermatitis, dandruff formation, and MF. Studies showed that the pathogenic *Malassezia* yeasts are the same species that was found on normal skin of the patients. *M. globosa* and *M. restricta* were the most frequently discovered species from the skin lesions of patients with seborrheic dermatitis[13]. *M. globosa* was found to be a major causative agent of pityriasis versicolor on the back[14]. *M. restricta* and *M. globosa* are the most common causative species for MF of the face and trunk, respectively[15].

MF

Skin flora or skin microbiota refers to the microorganisms that reside on human skin. Many of them are bacteria but are also fungi and ecto-parasite. Most are found in stratum corneum layer of epidermis and upper part of the hair follicles. A change in equilibrium has caused human skin flora to become pathogenic organism for skin diseases such as *Cutibacterium acnes* in acne vulgaris, *Corynebacterium* species in erythrasma, *Malassezia* species in pityriasis versicolor, MF, and others. MF results from overgrowth of *Malassezia* yeast in hair follicles, caused by changes in microenvironment and human immunity. MF is an acneiform eruption, described first by Weary et al in 1969[16]. This condition has clinical presentations similar to acne vulgaris. It is easy to miss and thus is likely underdiagnosed[17-20]. MF was originally perceived as folliculitis that caused by *P. ovale*, so it was named PF. Later, Potter confirmed the relationship between MF, *P. orbiculare*, and *P. ovale*, which is currently classified in the same species name *M. furfur*[21]. *M. furfur* is an oval, monopolar budding yeast. It is a polymorphic, lipophilic micro-organism with a thick, multilayered cell wall. This yeast is found in the stratum corneum and infundibular of hair follicles. *Malassezia* yeast can transform into a pathogenic organism under certain conditions, such as increased temperature, humidity, greasy skin, sweating, and immunosuppression[22]. In addition, one study found that this changes is associated with composition of fatty acids of the sebaceous gland due to an increase in androgen concentration[23].There are two possible mechanisms that may cause follicular inflammation by *Malassezia* yeast. The first mechanism is caused by lipase and phospholipase activity of *Malassezia* yeast that damage skin barrier function and cause inflammation by inducing irritation and non-immunogenic stimulation of the immune system[24,25]. This mechanism is supported by the presence of an increased number of NK1+ and CD16+ cells within biopsies from the lesional skin[25]. No differences were identified between the number of interleukin associated cells and the lesional or non-lesional skin. The second possible mechanism is caused by the ability of *Malassezia* yeast in vitro that can stimulate keratinocyte production of inflammatory cytokines via Toll-like receptor 2[24]. Among these inflammatory cytokines are interleukin (IL)-lα, IL-6, IL-8, IL-12, and tumor necrosis factor-α along with anti-inflammatory cytokines IL-4 and IL-10[26]. *Malassezia* yeast can also activate complement cascades by both the classical and alternative pathways[27]. Although there are many species that cause MF, all species have the same clinical presentation[28]. The most common species identified from lesional skin were *M. globosa*, *M. restricta*, and *M. sympodialis*[15,20]. These species were not only identified as most common on lesional skin, but also as non-lesional skin of the same patient as well as healthy controls[26]. This knowledge was confirmed by a study based on recombinant deoxyribonucleic acid (rDNA) analysis that identified the most common species from MF samples to be *M. globosa*, *M. sympodialis*, *M. restricta*, and *M. furfur* in order of most to least common[28]. It was also found that the same species were identified from both lesional and non-lesional samples of the same patient. From the results of all studies, it can be concluded that MF is caused by *Malassezia* species of the normal skin flora and is not caused by an exogenous species[15].

MF and neonatal cephalic pustulosis (neonatal acne)

Neonatal cephalic pustulosis is a variant of neonatal acne. Clinical symptom is the presence of pustular eruption on the face or scalp of newborn babies, often during the third week. The cause of neonatal cephalic pustulosis is strongly related to colonization of *Malassezia* yeasts, which can be detected through a microscope from pustules. It is typically a self-limited disease, but severe cases can be treated with topical ketoconazole[29]. This relationship of causative *Malassezia* yeast has been confirmed by many studies. Rapelanoro et al studied about papulopustular eruptions of the face in neonates. Direct examination of pustule smears

from papulopustular lesions of face, neck, and scalp showed *M. furfur* yeasts in eight of 13 cases. All cases responded well to 2% ketoconazole cream treatment within one week[30]. Niamba et al conducted a prospective case-control study about correlation between neonates with cephalic pustulosis and *M. sympodialis*. Cultures from swabs and smears of pustules were obtained from patients, and swabs from healthy site-matched skin were obtained from controls. They found that the prevalence of *Malassezia* yeasts increased with age, and the severity of the pustulosis was correlated with the isolation of *M. sympodialis*[31]. This was confirmed by a prospective study conducted by Bernier et al about skin colonization by *Malassezia* yeasts in neonates and relationship with neonatal cephalic pustulosis. This study found *Malassezia* colonization begins at birth and increases in the first weeks of life. A high prevalence of *M. sympodialis* in neonates was observed from birth and a correlation was found between *Malassezia* yeasts and neonatal cephalic pustulosis. *Malassezia* colonization was higher when pustulosis was more severe and *M. sympodialis* was found in pustules[32].

MF and steroid acne

Steroid acne refers to an acne-like skin eruption that occurs in patients with high levels of circulating corticosteroids. They may have Cushing disease or may be undergoing treatment with systemic steroid such as prednisolone, dexamethasone, or anabolic steroids. Steroid acne is most common in young adults who have been taking moderate or high dose of oral steroid for several weeks. Steroid acne is presented as uniform, monomorphic inflammatory papules, and pustules on the chest but may also develop on the face, neck, back, and upper arms. Steroid acne exhibited clinical symptoms similar to those seen in MF, but there are few reports about the incidence of *Malassezia* yeast and steroid acne. Yu et al studied about the incidence of *P. ovale* and the effect of antifungal drugs in steroid acne. The results of this study showed that more than 80% of patients with acneiform eruption receiving systemic steroid revealed significant numbers of *P. ovale* in the papulopustular lesions. Furthermore, it was also found that this group of patients responded well to antifungal treatment[18].

MF and acne vulgaris

MF is commonly misdiagnosed as acne due to clinical presentations and location of the skin lesions. Besides that, it can often be associated with acne vulgaris and may persist for years without complete resolution with conventional acne treatment[33]. Patients usually receive unnecessary and prolonged antibiotic treatment. A retrospective study review of 110 patients with MF found that more than 75% had acne vulgaris and had recently been treated with antibiotics. MF was more common after antibiotic use. It presents as monomorphic, pruritic papulopustular lesions along the hairline and on the upper part of the back, and it improved with topical or oral antifungal therapy[34]. Pürnak et al studied about the prevalence of MF among patients with acne vulgaris. MF was diagnosed based on clinical presentations and laboratory results. This study had shown that 25% of acne patients were also diagnosed with MF. Most of these patients had good results from antifungal treatment. The lesions decreased by more than 50% in 68.4% of the patients, which reduced the number of closed comedones and inflammatory papules. It was also found that the average number of spores in lesional samples was significantly decreased after treatment. They concluded that MF could present as acne vulgaris-like lesions, or the two diseases may coexist[35]. Ayers et al reported patients with combination of acne vulgaris and MF. These patients demonstrated follicular papulopustular lesions on the face, back, and chest. Symptoms often wax and wane depending on the patient's activities, time of the year, and current treatment regimens. This condition often got worse when treated with antibiotics and dramatically responded with antifungal therapy[10].

Clinical

MF is common in young adults[10]. The density of colonization of *Malassezia* yeasts is related to age and sebaceous gland activity[36]. It is also more common in men than women and in people living in hot, humid climates, which may be due to excessive sweating. Other predisposing factors include topical or oral antibiotic use, corticosteroid use, and other immunosuppressants[28,37]. MF presents as small, uniform, itchy papules, and pustules particularly on the upper chest and back. Other sites include the forehead, hair line, face, neck, and extensor aspect of upper arms (Figure 1-4). Durdu et al found 71.4% of patients with MF developed skin lesions in more than one area of the body. The most common location was the face (57.1%), followed by the back (53%), extensor surfaces of the arms (38.8%), chest (36.7%), and neck (18.3%). Most patients experience itching in the lesional area[38]. It also found that MF patients are more likely to experience seborrheic dermatitis and

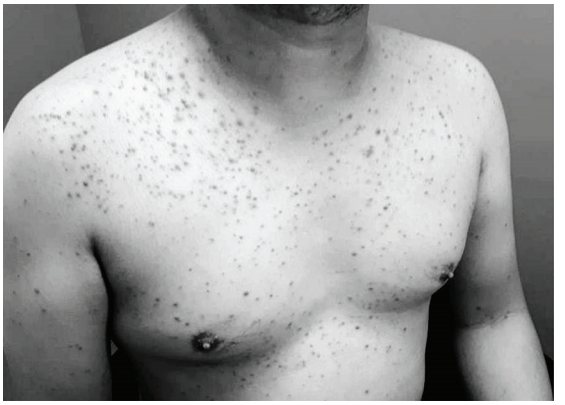

Figure 1. *Malassezia* folliculitis on upper chest.

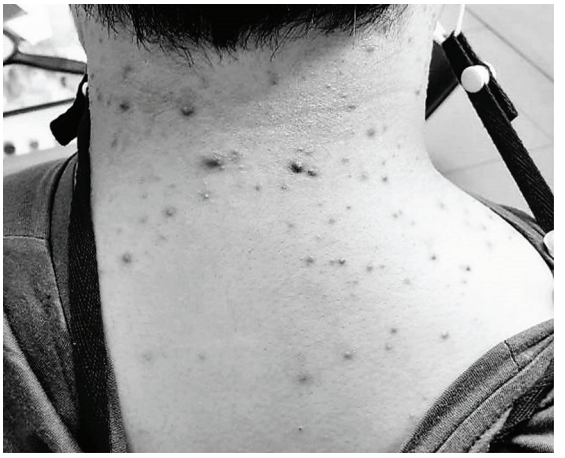

Figure 2. *Malassezia* folliculitis on neck and upper back.

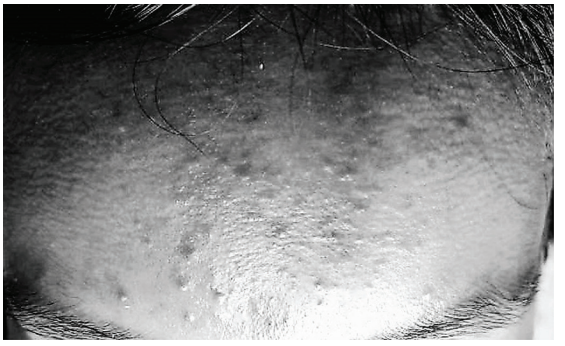

Figure 3. *Malassezia* folliculitis on forehead.

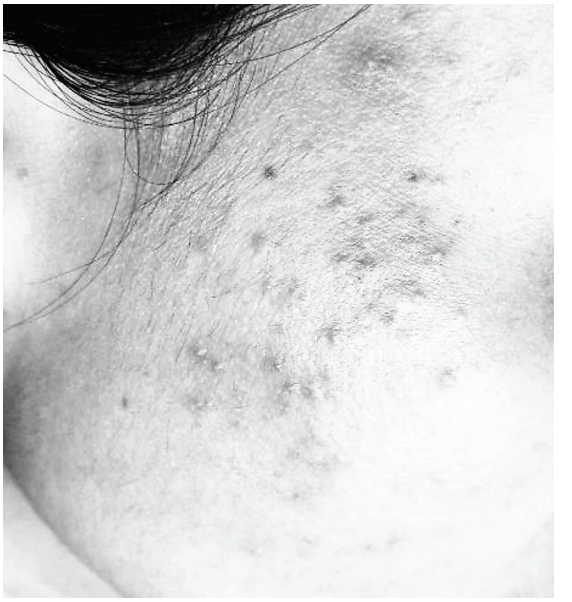

Figure 4. *Malassezia* folliculitis on right cheek.

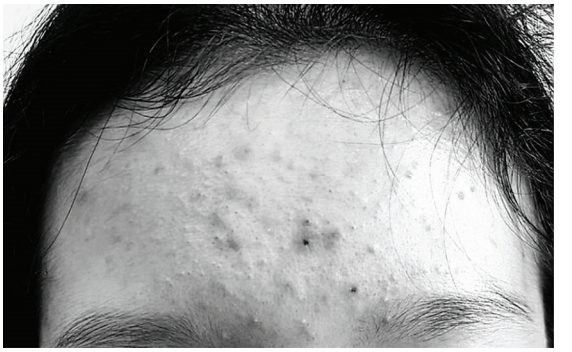

Figure 5. *Malassezia* folliculitis associated with acne vulgaris is characterized by inflammatory papules with multiple comedones of acne vulgaris and small, itchy, monomorphic papulopustular lesions of *Malassezia* folliculitis on forehead.

tinea versicolor[9], which is because both associated diseases result from the *Malassezia* yeasts. Studies have shown that there is a strong correlation between MF and acne vulgaris with various incidence[12,34,35]. Jacinto-Jamora et al studied about MF in Philippines and found that most patients that presented with papulopustular lesions had comedones on the face. MF coexisted with acne vulgaris in 56% of patients, and the addition of antimycotics to the acne regimen produced dramatic improvement of the lesions[12]. MF is often an underdiagnosed disease. It can be misdiagnosed as acne vulgaris and can often be associated with acne vulgaris (Figure 5). This condition requires a high index of clinical suspicion on initial presentation. It should be considered in patients with neonatal cephalic pustulosis or neonatal acne, young adult patients with acne on hairline, face, neck, or upper trunk, patients with recalcitrant acne, and patients with diabetes mellitus, AIDS, or a history of using antibiotic, corticosteroid and other

immunosuppressants, which may be responsible for the imbalance between *Malassezia* yeast and host immunity.

Diagnosis

Like other types of normal skin flora that can cause skin diseases, such as *Cutibacterium acnes* in acne vulgaris and *Malassezia* yeasts in pityriasis versicolor, the definite diagnosis is based on the clinical presentation, direct microscopy, histopathology, and response to antimicrobial treatment. MF usually presents as small, uniform, itchy papulopustular lesions on face, upper chest, and back. Wood lamp examination may demonstrate a yellow-green fluorescence on these skin lesions. This diagnostic tool was observed to be positive in 66.7% of MF patients, so it can be used in conjunction with physical examination[28]. Dermoscopic examination can be used to differentiate between MF and acne vulgaris. Dermoscopic features of MF have been described as follicular papule and pustules with surrounding erythema, dirty white perilesional scales, coiled hairs with perifollicular erythema and scaling, hypopigmentation of involved hair follicles, and perilesional brownish discoloration in resolving lesions[38]. Laboratory investigations by skin scraping with potassium hydroxide (KOH), tape stripping with Parker blue ink to confirm the presence of *Malassezia* yeasts is not recommended, as this can be misleading because *Malassezia* yeasts are presented as a normal part of skin flora in 75% to 98% of healthy individuals[10,20,28]. Usage of comedone extractor is recommended by Yu et al, rather than a simple skin scraping for KOH preparation. This will reveal levels of yeast within the hair follicle rather than the stratum corneum[18]. From the study conducted by Durdu et al, May-Grunwald-Giemsa smears showed higher positivity (100%) compared to KOH (81.6%) as confirmed by fungal culture[28]. Tu et al showed sensitivity and specificity of Gram staining in diagnosis of MF are 84.6% and 100%, respectively[39]. Diagnosis criteria about numbers of *Malassezia* yeasts from the skin sample are defined. Suzuki et al proposed diagnosis criteria for MF based on characteristic clinical features and direct microscopic findings of 10 or more yeast-like fungi per one hair follicle[40]. Pürnak et al suggested that MF will be diagnosed with more than six spores in one high power microscopic field (×400)[35]. At present, these diagnostic criteria are not conclusive. The author recommends using comedone extractor for collecting samples from papulopustular lesions and staining it with Gram stain to look for the presence of *Malassezia* yeasts. This method is simple, not expensive, and can rule out other causes of folliculitis such as, *C. acnes*, Gram-negative bacteria, Dermatophytes, and *Demodex* mites. Culture of *Malassezia* species typically requires special media, Dixon's or Leeming-Notman agar, and growth at 32°C to 35°C under aerobic conditions, so it is not routinely performed. Histopathological sections reveal dilated follicles plugged with keratinous material, and perifollicular inflammatory cells infiltration with neutrophils. When the follicle contains round yeast organisms without hyphae, it demonstrates positivity with periodic acid-Schiff stain[41]. An et al demonstrated that significant differences in histopathologic findings exist between MF and another acneiform eruption lesions relative to the presence of necrotic keratinocytes in the follicular wall[42].

Treatment

There are many treatments for MF, but it is important to address any predisposing factors of the patients at the beginning, because MF tends to recur. Educating patients about how to prevent this disease is as important as the treatments. Topical treatments such as selenium sulfide shampoo, 1.5% ciclopirox olamine, 2% ketoconazole shampoo, and topical ketoconazole have been reported to be effective in patients with MF. However, the most effective treatment is oral antifungal medication, because *Malassezia* yeast is located deep within the hair follicle. Topical treatment may not be sufficiently effective for the treatment. Otherwise, oral antifungal medications are also useful for their anti-inflammatory actions[43]. Nevertheless, topical antifungals are useful as adjunctive therapy as well as maintenance and prophylactic therapy. Investigators have studied the efficacy of itraconazole, as this antifungal is excreted in high concentrations in sebum. Itraconazole is a broad-spectrum triazole, which is highly lipophilic with good oral absorption and extensive tissue distribution[44]. Two weeks of 200 mg itraconazole daily resulted in complete recovery of 79.6% of patients with MF[28]. Antifungal drug sensitivities of *Malassezia* yeasts from MF were listed as itraconazole, which is better than ketoconazole, which is better than Amphotericin B, which is better than fluconazole. Itraconazole and ketoconazole have the best antifungal activity against *Malassezia* yeasts, but ketoconazole has a higher chance of causing hepatitis[45]. In practice, fluconazole is used more commonly due to its low side effect profile.

There are reports of MF treatment with isotretinoin, but its efficacy is not conclusive. Friedman found that 20 weeks of therapy with low-dose isotretinoin has beneficial effect[46], while Goodfield and Saihan reported failure of isotretinoin therapy in MF patients[47]. Photodynamic therapy (PDT) is a new alternative therapeutic option for MF. Lee et al did a pilot study using topical PDT with methyl aminolevulinate cream as a photosensitizer treatment for MF. Out of the six patients included in this study, three presented with strong improvement, one with moderate improvement, one with mild improvement, and one with no improvement. This study also reported no recurrence after four months[48]. Recurrence of MF is common even after successful treatment. Therefore, analyzing and eliminating predisposing factors is recommended. Long-term prophylaxis with topical treatments should be considered in patients with a history of recurrences.

Conclusion

MF is typically presented as monomorphic, pruritic papules and pustules along the hairline, neck, and upper part of the trunk and responds well to treatment with topical or oral antifungal medications[40]. Definite diagnosis of MF is based primarily on clinical presentation, direct microscopy, histopathological examination, and rapid response with antifungal treatments[16,17]. Due to the clinical presentation and location of the lesions being similar to acne, MF is often misdiagnosed as acne. In addition, MF can also occur simultaneously with acne vulgaris, thus making an accurate diagnosis even more difficult[40]. The association with acne vulgaris may require combinations of both antifungal and acne medications[14,34]. Dermatologists should be aware of MF when encountering neonatal acne, young adults with acne on hairline, face, neck and upper trunk, recalcitrant acne, steroid acne, and immunocompromised patients to provide proper management for these patients.

What is already known about this topic?

Malassezia folliculitis results from overgrowth of *Malassezia* yeasts present in the normal skin flora. This condition is caused by a loss of balance between *Malassezia* yeasts, microenvironment, and human immunity. *Malassezia* yeast becomes pathogenic organism in certain predisposing factors such as immunosuppression, hot and humid climates causing increased episodes of sweating, antibiotic treatment, and corticosteroid therapy. MF typically presents as small, monomorphic, itchy papules, and pustules particularly on hair line, face, and upper trunk of young adults.

What does this study add?

This article reviews knowledge about MF, new data about *Malassezia* yeast, a variety of clinical manifestations, investigations, and diagnostic criteria for MF. MF is an under-recognized disease, often misdiagnosed as acne vulgaris, recalcitrant acne, neonatal cephalic pustulosis or neonatal acne, and steroid acne. This condition can occur simultaneously with acne, making it more difficult to get an accurate diagnosis. The definite diagnosis is based on clinical presentations, direct microscopy, histopathological examination, and good response to antifungal treatments. Dermatologists should be aware of this disease when encountering patients with acne problems to provide proper management for the patients.

Conflicts of interest

The author declares no conflicts of interest.

References

1. Saunte DML, Gaitanis G, Hay RJ. Malassezia-associated skin diseases, the use of diagnostics and treatment. Front Cell Infect Microbiol 2020;10:112.
2. Saxena R, Mittal P, Clavaud C, Dhakan DB, Hegde P, Veeranagaiah MM, et al. Comparison of healthy and dandruff scalp microbiome reveals the role of commensals in scalp health. Front Cell Infect Microbiol 2018;8:346.
3. Guého E, Midgley G, Guillot J. The genus Malassezia with description of four new species. Antonie Van Leeuwenhoek 1996;69:337-55.
4. Sugita T, Takashima M, Shinoda T, Suto H, Unno T, Tsuboi R, et al. New yeast species, Malassezia dermatis, isolated from patients with atopic dermatitis. J Clin Microbiol 2002;40:1363-7.
5. Sugita T, Takashima M, Kodama M, Tsuboi R, Nishikawa A. Description of a new yeast species, Malassezia japonica, and its detection in patients with atopic dermatitis and healthy subjects. J Clin Microbiol 2003;41:4695-9.
6. Sugita T, Tajima M, Takashima M, Amaya M, Saito M, Tsuboi R, et al. A new yeast, Malassezia yamatoensis, isolated from a patient with seborrheic dermatitis, and its distribution in patients and healthy subjects. Microbiol Immunol 2004;48:579-83.
7. Hirai A, Kano R, Makimura K, Duarte ER, Hamdan JS, Lachance MA, et al. Malassezia nana sp. nov., a novel lipid-dependent yeast species isolated from animals. Int J Syst Evol Microbiol 2004;54:623-7.
8. Lee YW, Yim SM, Lim SH, Choe YB, Ahn KJ.

Quantitative investigation on the distribution of Malassezia species on healthy human skin in Korea. Mycoses 2006;49:405-10.
9. Bäck O, Faergemann J, Hörnqvist R. Pityrosporum folliculitis: a common disease of the young and middle-aged. J Am Acad Dermatol 1985;12:56-61.
10. Ayers K, Sweeney SM, Wiss K. Pityrosporum folliculitis: diagnosis and management in 6 female adolescents with acne vulgaris. Arch Pediatr Adolesc Med 2005;159:64-7.
11. Bulmer GS, Pu XM, Yi LX. Malassezia folliculitis in China. Mycopathologia 2008;165:411-2.
12. Jacinto-Jamora S, Tamesis J, Katigbak ML. Pityrosporum folliculitis in the Philippines: diagnosis, prevalence, and management. J Am Acad Dermatol 1991;24:693-6.
13. Zhang H, Ran Y, Xie Z, Zhang R. Identification of Malassezia species in patients with seborrheic dermatitis in China. Mycopathologia 2013;175:83-9.
14. Cam VT, Van TN, Hau KT, Huu DL, Minh PPT, Huu SN, et al. Distribution of Malassezia species from scales of patient with pityriasis versicolor by culture in Vietnam. Open Access Maced J Med Sci 2019;7:184-6.
15. Akaza N, Akamatsu H, Sasaki Y, Kishi M, Mizutani H, Sano A, et al. Malassezia folliculitis is caused by cutaneous resident Malassezia species. Med Mycol 2009;47:618-24.
16. Weary PE, Russell CM, Butler HK, Hsu YT. Acneform eruption resulting from antibiotic administration. Arch Dermatol 1969;100:179-83.
17. Gaitanis G, Velegraki A, Mayser P, Bassukas ID. Skin diseases associated with Malassezia yeasts: facts and controversies. Clin Dermatol 2013;31:455-63.
18. Yu HJ, Lee SK, Son SJ, Kim YS, Yang HY, Kim JH. Steroid acne vs. Pityrosporum folliculitis: the incidence of Pityrosporum ovale and the effect of antifungal drugs in steroid acne. Int J Dermatol 1998;37:772-7.
19. Abdel-Razek M, Fadaly G, Abdel-Raheim M, al-Morsy F. Pityrosporum (Malassezia) folliculitis in Saudi Arabia--diagnosis and therapeutic trials. Clin Exp Dermatol 1995;20:406-9.
20. Crespo Erchiga V, Delgado Florencio V. Malassezia species in skin diseases. Curr Opin Infect Dis 2002;15:133-42.
21. Potter BS, Burgoon CF Jr, Johnson WC. Pityrosporum folliculitis. Report of seven cases and review of the Pityrosporum organism relative to cutaneous disease. Arch Dermatol 1973;107:388-91.
22. Roberts SO. Pityriasis versicolor: a clinical and mycological investigation. Br J Dermatol 1969;81:315-26.
23. Mokronosova MA, Arzumanian VG, Gervazieva VB. Yeast-like fungi Malassezia (Pityrosporum): clinical and immunological aspects of study. Vestn Ross Akad Med Nauk 1998:47-50.
24. Baroni A, Orlando M, Donnarumma G, Farro P, Iovene MR, Tufano MA, et al. Toll-like receptor 2 (TLR2) mediates intracellular signalling in human keratinocytes in response to Malassezia furfur. Arch Dermatol Res 2006;297:280-8.
25. Faergemann J, Bergbrant IM, Dohsé M, Scott A, Westgate G. Seborrhoeic dermatitis and Pityrosporum (Malassezia) folliculitis: characterization of inflammatory cells and mediators in the skin by immunohistochemistry. Br J Dermatol 2001;144:549-56.
26. Gaitanis G, Magiatis P, Hantschke M, Bassukas ID, Velegraki A. The Malassezia genus in skin and systemic diseases. Clin Microbiol Rev 2012;25:106-41.
27. Ljubojević S, Skerlev M, Lipozencić J, Basta-Juzbasić A. The role of Malassezia furfur in dermatology. Clin Dermatol 2002;20:179-82.
28. Durdu M, Güran M, Ilkit M. Epidemiological characteristics of Malassezia folliculitis and use of the May-Grünwald-Giemsa stain to diagnose the infection. Diagn Microbiol Infect Dis 2013;76:450-7.
29. Zuniga R, Nguyen T. Skin conditions: common skin rashes in infants. FP Essent 2013;407:31-41.
30. Rapelanoro R, Mortureux P, Couprie B, Maleville J, Taïeb A. Neonatal Malassezia furfur pustulosis. Arch Dermatol 1996;132:190-3.
31. Niamba P, Weill FX, Sarlangue J, Labrèze C, Couprie B, Taïeh A. Is common neonatal cephalic pustulosis (neonatal acne) triggered by Malassezia sympodialis? Arch Dermatol 1998;134:995-8.
32. Bernier V, Weill FX, Hirigoyen V, Elleau C, Feyler A, Labrèze C, et al. Skin colonization by Malassezia species in neonates: a prospective study and relationship with neonatal cephalic pustulosis. Arch Dermatol 2002;138:215-8.
33. Rubenstein RM, Malerich SA. Malassezia (pityrosporum) folliculitis. J Clin Aesthet Dermatol 2014;7:37-41.
34. Prindaville B, Belazarian L, Levin NA, Wiss K. Pityrosporum folliculitis: A retrospective review of 110 cases. J Am Acad Dermatol 2018;78:511-4.
35. Pürnak S, Durdu M, Tekindal MA, Güleç AT, Seçkin D. The prevalence of Malassezia folliculitis in patients with papulopustular/comedonal acne, and their response to antifungal treatment. Skinmed 2018;16:99-104.
36. Marcon MJ, Powell DA. Human infections due to Malassezia spp. Clin Microbiol Rev 1992;5:101-19.
37. Alves EV, Martins JE, Ribeiro EB, Sotto MN. Pityrosporum folliculitis: renal transplantation case report. J Dermatol 2000;27:49-51.
38. Jakhar D, Kaur I, Chaudhary R. Dermoscopy of Pityrosporum folliculitis. J Am Acad Dermatol 2019;80:e43-4.
39. Tu WT, Chin SY, Chou CL, Hsu CY, Chen YT, Liu D, et al. Utility of Gram staining for diagnosis of Malassezia folliculitis. J Dermatol 2018;45:228-31.
40. Suzuki C, Hase M, Shimoyama H, Sei Y. Treatment outcomes for Malassezia folliculitis in the dermatology

department of a university hospital in Japan. Med Mycol J 2016;57:E63-6.
41. Marques SA, Silva SB, Camargo RM, Stolf HO, Marques ME. Exuberant clinical presentation of probable Malassezia folliculitis in a young nonimmunosuppressed patient. An Bras Dermatol 2012;87:459-62.
42. An MK, Hong EH, Cho EB, Park EJ, Kim KH, Kim KJ. Clinicopathological differentiation between Pityrosporum folliculitis and acneiform eruption. J Dermatol 2019;46:978-84.
43. Gupta AK, Batra R, Bluhm R, Boekhout T, Dawson TL Jr. Skin diseases associated with Malassezia species. J Am Acad Dermatol 2004;51:785-98.
44. Parsad D, Saini R, Negi KS. Short-term treatment of Pityrosporum folliculitis: a double blind placebo-controlled study. J Eur Acad Dermatol Venereol 1998;11:188-90.
45. Wang K, Cheng L, Li W, Jiang H, Zhang X, Liu S, et al. Susceptibilities of Malassezia strains from pityriasis versicolor, Malassezia folliculitis and seborrheic dermatitis to antifungal drugs. Heliyon 2020;6:e04203.
46. Friedman SJ. Pityrosporum folliculitis: treatment with isotretinoin. J Am Acad Dermatol 1987;16:632-3.
47. Goodfield MJ, Saihan EM. Failure of isotretinoin therapy in Pityrosporum folliculitis. J Am Acad Dermatol 1988;18:143-4.
48. Lee JW, Lee HI, Kim MN, Kim BJ, Chun YJ, Kim D. Topical photodynamic therapy with methyl aminolevulinate may be an alternative therapeutic option for the recalcitrant Malassezia folliculitis. Int J Dermatol 2011;50:488-90.