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Editors' welcome

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In this edition, we put the spotlight on pythiosis, a difficult-to-treat infection caused by a water mold – the fungus-like *Pythium insidiosum*. The rare septate hyphae morphology from both lesions and culture of the organism can be easily misdiagnosed; Dr Ariya Chindamporn provides a detailed run-down on how best to identify it in the laboratory. Dr Methee Chayakulkeeree shares an interesting case of a teenager who was correctly diagnosed with pythiosis after certain risk factors were identified.

Also in this issue, the mystery of mycetoma in Asia is tackled: "Data on mycetoma in Asia are scarce," according to Dr Harsimran Kaur and Professor Arunaloke Chakrabarti. Dr Atul Patel gives a quick guide on echinocandins that will prove helpful to practicing clinicians.

We take this opportunity as well to introduce two new committee members of our growing team, Dr Lee Lee Low and Dr Fairuz Amran, both from Malaysia.

We are very pleased with the direction and accomplishements of the group, and are even happier that we are now able to share to a bigger audience, and hear from you instantly as well. We also share with you updates from our official website, AFWGonline.com, as well as our social media presence. We invite you all to visit the site and drop us a line on Facebook, Instagram or LinkedIn. See you online!

Laboratory Diagnosis of Pythiosis

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The rare septate hyphae morphology from both lesions and culture of *Pythium insidiosum* is so similar to those of the molds Mucorales and Entomophthorales that it is easy to misdiagnose. Early diagnosis of pythiosis is crucial: this infection has a high mortality rate, especially in the vascular type; and a corneal transplant or penetrating keratoplasty may be needed for the keratitis type. Several case reports indicate that early diagnosis leads to lower risk or greater survival for patients. This article details the proper steps to making an accurate laboratory diagnosis.

A. Isolation and identification

1. Sample collection: The sample should be collected aseptically and placed in a sterile container. To keep the sample moist, a small amount of normal saline or sterile water, not formalin, is suggested. If the specimens need to be kept longer, a few drops of antibiotics (chloramphenicol/penicillin), should be added to prevent bacterial overgrowth.

   a. Keratitis type: scraping or corneal biopsy
   b. Subcutaneous type: biopsy from the lesion (for bovine pythiosis, kunkers are the proper specimens for diagnosis)
   c. Vascular type: occlusion in the arterial aneurysm – not the muscle or tissue around the infected artery – because the organism grows inside the endothelium

2. Transport: Avoid transporting the specimens on ice because some isolates are sensitive to low temperatures.

3. Direct examination: KOH preparation with 10% KOH solution allows simple and rapid detection in scraping, tissue biopsy and occlusion samples. This simple test can show whether hyphae are present in the clinical specimens or not. The solution of 10% KOH with calcofluor is also recommended. A positive sample shows rare septate hyphae with several vesicles inside (see Figure). The diameter of the hyphae is about 2.6-6.4 µm, similar to that of *Aspergillus* but not Zygomycetes.

4. Culturing: The clinical specimens should be cut into very small pieces in sterile conditions before inoculation on Sabouraud dextrose agar or Sabouraud dextrose broth and incubated at 25-35°C. The growth of small hair-like projections of *P. insidiosum* around the inoculated sample is detected within 12-24 hours. When the culture is mature, it becomes a hyaline, submerged colony. Blood agar is another recommended medium for first isolation. *P. insidiosum* does not produce any spores on medium.

5. Identification: Two screening methods are induction of zoospores, the natural form of oomycetes, and biochemical assay. Both *P. insidiosum* and *Lagenidium* produce sterile, rare septate hyphae and hydrolyze maltose, but only *P. insidiosum* utilizes sucrose. A sucrose-positive result can also differentiate *Pythium* spp. from Entomophthoromycota and Mucoromycota. Presently, the definitive identification is via molecular approach using polymerase chain reaction (PCR), which is discussed further in 'Molecular diagnosis'.

B. Histopathology

Physicians often send to the laboratory formalin-fixed, paraffin-embedded (FFPE) samples. Short, longitudinal, rare septate hyphae and transverse hyphae with a diameter similar to that of the septate hyphae would be seen. Hematoxylin and eosin (H&E) and periodic acid-Schiff (PAS) stains show necrotic eosinophilic granulomas around the hyphae, similar to the tissue reaction of Entomophthoromycota infection. Gomori methenamine silver (GMS) is the preferred stain for *P. insidiosum*, with the sizes mentioned above.

C. Serology

Immunodiffusion test to detect antibodies against *P. insidiosum* is specific but has low sensitivity. Several other tests focusing on antibodies have been developed, such as enzyme-linked immunosorbent assay (ELISA), immunochromatography, hemagglutination and Western blot assays. Detection of antibodies is an important test for vascular pythiosis to aid management and to determine whether the patient was infected with the fungus or not. Immunofluorescence or immunoperoxidase staining are also used.

D. Molecular diagnosis

Molecular techniques have been used for the identification of *P. insidiosum* from cultures, clinical specimens and FFPE samples. The target site in the internal transcribed spacer (ITS) in the ribosomal RNA region of *P. insidiosum* has been used for amplification by PCR. In addition, specific primers in the cytochrome oxidase gene (*COX2*) have been reported to identify the species. To differentiate *P. insidiosum* and subcutaneous zygomycosis and entomophthoromycosis, thermophilic helicase-dependent DNA amplification (tHDA) and restriction fragment length polymorphism (RFLP) in the *COX2* region could be used.

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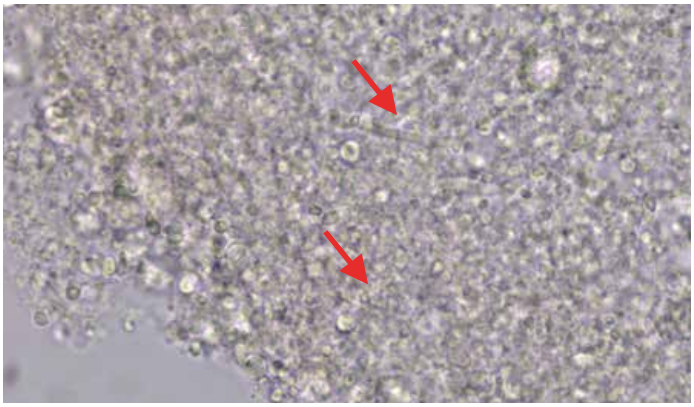
Figure. KOH preparation from infected artery showing rare septate hyphae



Human Pythiosis

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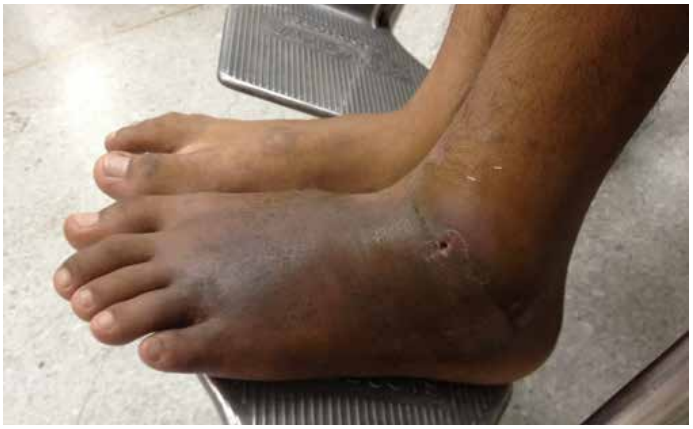
Case details

A 17-year-old man was punctured by a wooden stick that was soaked in floodwater. He sought medical advice and received antibiotics to prevent wound infection. One month later, his left foot became swollen and the puncture site developed into a chronic ulcer (Figure 1). He went to another hospital where he was diagnosed with chronic osteomyelitis of the left foot. Antibiotics were given, but his symptoms did not improve. He underwent magnetic resonance imaging followed by tissue biopsy of his left foot. Histopathologic findings revealed fungal granuloma.

His medical history revealed that the patient was diagnosed with beta thalassemia major at 3 years old and received blood transfusion regularly. Because of his underlying disease, human pythiosis was suspected and his serum was tested for antibodies against *Pythium insidiosum*. The test turned out positive.

Computed tomography (CT) angiography showed occlusion at the left distal third of the anterior tibial artery and distal end of the posterior tibial artery (Figure 2). He was then treated with *Pythium* immunotherapy and itraconazole in combination with terbinafine, and his left leg was amputated.

Figure 1. Chronic ulcer on the left foot



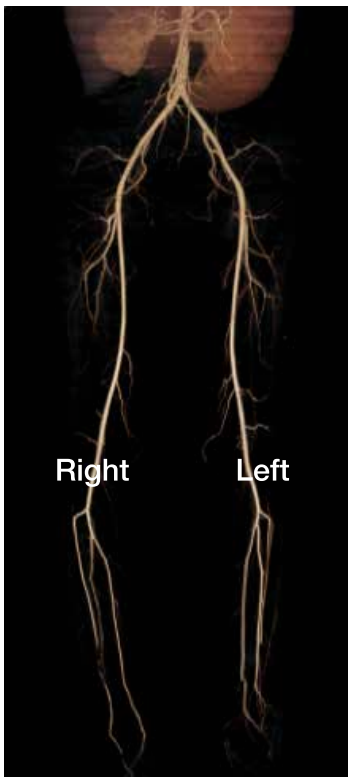
Clinical presentation of pythiosis

Pythiosis is a difficult-to-treat infection in animals and humans caused by a water mold – a fungus-like organism named *P. insidiosum*. The clinical presentation of human pythiosis can be classified into three forms: systemic or vascular; cutaneous; and ocular.[1] Patients at risk for acquiring *Pythium* infection include those with underlying thalassemia or hemoglobinopathy, although ocular pythiosis usually occurs in apparently immunocompetent hosts. The most common form of human pythiosis is vascular pythiosis, which is the most lethal form. Vascular pythiosis causes medium to large arterial occlusions, usually in the lower extremities, and can extend to involve the aortic bifurcation. Cutaneous pythiosis presents with a painful, subcutaneous granulomatous, infiltrative lump or ulcer on the arms or legs. It may present as acute necrotizing cellulitis. Ocular pythiosis normally presents as corneal ulcer or keratitis. The overall mortality of human pythiosis is approximately 40%.[2] For the patients who survive, however, most lose their limbs or eyes.

Diagnosis and treatment

Diagnosis of human pythiosis may be done by using an immunoassay to detect anti-*Pythium* antibodies.[3] Histopathology, polymerase chain reaction (PCR)[4] and culture can be performed on surgical tissue for definitive diagnosis. Therapy for human pythiosis involves surgery, medical treatment and immunotherapy. However, as medical treatment may not always be completely effective, radical surgery is the main therapeutic intervention: limb amputation for patients with vascular pythiosis; and eye enucleation for those with the ocular form of the disease. Approximately 90% of patients with vascular pythiosis require limb amputation for lifesaving treatment.[2] Similarly, 80% of those with the ocular form undergo eye enucleation for radical treatment. For medical treatment, saturated solution of potassium iodide (SSKI) or amphotericin B plus flucytosine may be used in localized cutaneous pythiosis, but these are not effective in systemic or vascular pythiosis. Although their clinical efficacy is still inconsistent, the combination of itraconazole and terbinafine has shown synergy in vitro and is currently recommended for medical treatment of systemic pythiosis.[5] Finally, immunotherapy with *P. insidiosum* antigenic extract has been reported to have some efficacy for treatment of systemic forms.[6] Further studies of pythiosis treatment are warranted to seek for more effective treatment modalities for improved outcomes of patients with *P. insidiosum* infection.

Figure 2.
CT angiography showing occlusion at the left distal third of anterior tibial artery and distal end of posterior tibial artery



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Mycetoma in Asia: Still Veiled in Mystery

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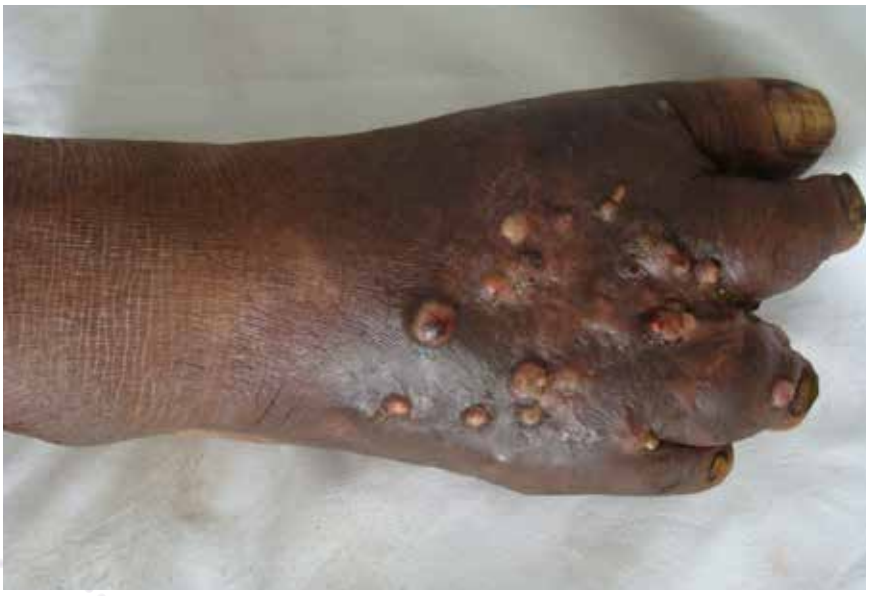
Epidemiology

Mycetoma is a chronic, granulomatous, subcutaneous disease caused by bacteria (actinomycetoma) or fungi (eumycetoma). The disease progresses over months to years from a localized, painless swelling to plaques, nodules and multiple discharging sinuses extruding grains (Figure), subsequently spreading to underlying muscle, tendons and bone, causing permanent deformities. Because of its typical tropical and subtropical distribution extending between 15° south and 30° north latitude (in a zone called the 'mycetoma belt'), and its neglected status, the World Health Organization (WHO) declared mycetoma as a neglected tropical disease.[1-3] However, its true distribution extends beyond this belt.

A rough estimate of mycetoma epidemiology by van de Sande indicated the most cases in Mexico, Sudan, Senegal and India, with few reports from Uganda, Romania, Nigeria, Bulgaria and Thailand.[3] The frequency varies with different geographic locations.[3-15] Actinomycetomas are predominant in dry areas, especially in North Africa, Central and South America and a few Asian countries, while eumycetoma prevails in tropical and subtropical regions of Asia (Mid-East, India) and Africa (Sub-Saharan Africa), where rainfall is abundant.[16] This is in contrast to the distribution in India, where eumycetoma is common in the dry western regions of Rajasthan, while eastern Rajasthan and southern India report a high rate of actinomycetomas despite sufficient rainfall in these regions.[3,17] Although the meta-analysis by van de Sande gave an overview of epidemiology, the true magnitude of the disease is still ambiguous, as majority of the cases from the endemic regions are not reported in literature.

Figure. Mycetoma of the foot with multiple discharging sinuses
Photo courtesy of Professor BM Hemashettar, India



Regional data

The data on mycetoma in Asia are scarce and mostly based on case reports from single centers. The heaviest burden of the disease is limited to Southeast Asia, the Middle East and, less commonly, in the Far East.[2] It was in Madurai (formerly Madura), India, where Gill, Colebrook and Godfrey first described this disease as 'Madura foot', which was later renamed 'mycetoma' by Carter.[2] Current available data show pockets of distribution of mycetoma in Rajasthan, Tamil Nadu and West Bengal provinces in India, with few scattered case reports from Punjab, Madhya Pradesh and Andhra Pradesh.[3] Overall, eumycetoma is more common in Rajasthan (62.5%), while actinomycetoma is more prevalent (54.3%-83.3%) in the rest of the country.[3] Causative agents also vary within India. *Madurella mycetomatis* is the most common agent, followed by *M. grisea* and *Aspergillus nidulans* in North West India, while species causing eumycetoma in South India include *M. mycetomatis*, *Neoscytalidium dimidiatum* and *A. flavus*.[17] The red grain mycetoma, *Actinomadura pelletieri*, is rare in India.[17]

Studies from Iran and Thailand have shown predominance of actinomycetoma (84.5% and 64.7%, respectively), while studies from Yemen demonstrate high prevalence of eumycetoma (71%).[15,18-21] The most common agents of actinomycetoma in Iran are *Actinomadura madurae* (23.5%), *Nocardia asteroides* (20.6%) and *Nocardia caviae* (13.2%); *Pseudoallescheria boydii* (10.3%) is the common cause of eumycetoma.[18,19,22] The prevalence of mycetoma in China is quite low, with around 19 cases reported between 1960 and 2010 (10 eumycetoma; 9 actinomycetoma). The etiologic agents reported from the Chinese population include *Nocardia brasiliensis*, *Nocardia asteroides*, *Nocardia otitidiscaviarum*, *Actinomadura madurae*, *Acremonium falciforme*, *Scopulariopsis maduromycosis*, *Pseudallescheria boydii*, *Madurella mycetomatis*, *Madurella pseudomycetomatis*, *Trichophyton verrucosum* and *Aspergillus* spp.[23] Sporadic cases of mycetoma are reported from Singapore (*Monosporium apiospermum*), Malaysia (*Phialophora jeanselmei*, *Madurella mycetomi*, *Streptomyces somaliensis*), Philippines (*Madurella grisea*), Indonesia (*Madurella tropicana*), Laos (*Actinomadura madurae*), Cambodia (*Pyrenochaeta romeroi*, *Madura mycetes*), Thailand (*Nocardia asteroids*, *N. caviae*, *N. brasiliensis*, *N. rosatii*, *Madurella mycetomii*, *Pseudallescheria boydii*, *Exophiala jeanselmei*, *Actinomadura madurae*, *Cladosporium carrionii*), and Vietnam (*Nocardia otitidiscaviarum*).[24]

Presentation and diagnosis

The disease generally affects young men from rural areas working barefoot outdoors (for activities such as farming, for instance). However, in Thailand, there is an equal prevalence of mycetoma in men and women.[3] The disease usually occurs by inoculation of the etiologic agent at the body site, most commonly foot (80%), followed by leg, trunk (less common in Asia) and arm.[3]

An early diagnosis of the disease is necessary to prevent disfigurement, and identification of the etiologic agent is required for guiding management. The diagnosis is quite challenging in developing countries, where there is a lack of facilities, forcing the clinicians to assess the disease clinically and manage the patient without accurate identification of the pathogen or determining the extent of disease by imaging. Ultrasound and fine needle aspiration are the minimum requirements to accurately diagnose the disease. Other imaging modalities include X-ray, computed tomography (CT) and magnetic resonance imaging (MRI), which are lacking in peripheral and remote areas of developing nations. Culture methods, the gold standard for identification, have limitations of long turnaround time, difficulty isolating the true causative agent from contaminating bacteria and saprophytic fungi, and the need for experienced personnel. Matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF MS) is a rapid technique of identification but is available only in a few reference centers. Other modalities like histology, cytology, skin test and serology lack specificity for identification. Molecular methods may improve the diagnostic capabilities, but are too expensive to be available at all centers of developing countries.

Treatment and prognosis

The treatment is chosen only after distinguishing if the disease is an actinomycetoma or a eumycetoma. Surgical debridement and medical management by antibiotics or antifungal agents are the cornerstones of mycetoma treatment.[2] However, the rate of recurrence is quite high, probably due to poor compliance or poor response to the drugs. The prognosis of actinomycetoma is better than eumycetoma.

Conclusion

There are many gaps in knowledge regarding the epidemiology and management of mycetoma. The exact magnitude of disease burden in Asia is still a mystery that needs to be solved to fill in these gaps. Clearly, awareness must be raised among health professionals for early diagnosis and treatment of mycetoma.

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Echinocandins: A Quick Guide for Practicing Clinicians

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Echinocandins are some of the most well tolerated and safest antifungal drugs. All three echinocandins are structurally similar with limited oral bioavailability. They irreversibly inhibit β-1,3-D-glucan synthase, the enzyme complex that forms glucan polymers in the fungal cell wall. Glucan polymers are responsible for providing rigidity to the cell wall, so disrupting β-1,3-D glucan synthesis leads to reduced cell wall integrity, cell rupture and cell death.[1,2] Echinocandins have fungicidal activity against *Candida* species and are fungistatic for *Aspergillus* species. These drugs cause damage to the hyphal tips and branch points of growing *Aspergillus* cells and decrease invasion potential.[1,2]

Spectrum of activity and pharmacology[2-4]

- Activity against all *Candida* species (with higher minimum inhibitory concentrations [MICs] for *C. parapsilosis* and *C. guilliermondii*) and *Aspergillus* species
- No activity against *Cryptococcus*, *Histoplasma*, *Fusarium*, *Scedosporium* and *Mucorales* except *Rhizopus oryzae*

A comparative pharmacology of all three echinocandins is described in the Table.

Table. Pharmacology of echinocandins[4]

	Caspofungin	Micafungin	Anidulafungin
Absorption/PK	Not orally absorbed/linear PK		
Distribution	Extensive into the tissues, liver, lungs, kidney and heart; minimal CNS penetration		
Metabolism	Hepatic Spontaneous degradation, hydrolysis and N-acetylation		Chemical degradation; not metabolized by the liver
Urine concentrations	Limited urinary excretion Not dialyzable		
CSF penetration	Penetrate the brain but not into the CSF		
Half-life	9-23 hours	11-21 hours	26.5 hours
Dose	70 mg IV on day 1, 50 mg	100 mg IV	200 mg IV on day 1, 100 mg
Dose adjustment Liver disease CYP inducers	70 mg day 1 then 35 mg daily 70 mg daily	None	None
CYP3A4 inhibition	No	Yes, weak	No
Drug interactions	Rifampin, efavirenz, nevirapine, tacrolimus, cyclosporine, phenytoin, dexamethasone, carbamazepine	Sirolimus, nifedipine	Some with cyclosporine

CNS, central nervous system; CSF, cerebrospinal fluid; PK, pharmacokinetics

Adverse drug reactions

Adverse drug reactions are uncommon with echinocandin use.[2] They include phlebitis, gastrointestinal side effects, hypokalemia and abnormal liver function tests. Caspofungin tends to have a higher frequency of liver-related laboratory abnormalities and a higher frequency of infusion-related pain and phlebitis. Histaminic reactions (rash, pruritus, flushing, hypotension, bronchospasm and angioedema) due to histamine release are reported with rapid infusion of anidulafungin, but these effects are transient and are easily managed by slowing the infusion rate with supportive care. Myocyte injury related to mitochondrial damage has been reported.

Echinocandin use

Echinocandins are used for the prevention, empiric treatment and treatment of *Candida* infections.

Candida infections: All major guidelines recommend echinocandins as the preferred agent for treatment of candidemia/invasive candidiasis.

Aspergillus infections: In patients who are intolerant or refractory to voriconazole treatment.

Who should not receive echinocandins?

Because of less-than-optimal pharmacokinetics in the central nervous system (CNS)/eyes/urine, echinocandins are not recommended for targeted therapy of fungal infections involving the CNS, eyes or the lower urinary tract

Advantages

- No breakthrough ophthalmic infections on echinocandin therapy for candidemia
- No negative outcomes in *C. parapsilosis* bloodstream infections treated with echinocandins (central venous catheter management is important)
- Echinocandins and liposomal amphotericin B have good activity in *Candida* biofilms[5]

Which echinocandins should I select in my practice?

All three are equal in efficacy so it would depend on host & cost

In HIV patients on efavirenz-based antiretroviral therapy; patients with tuberculosis (TB) taking rifampin-based anti-TB therapy; transplant recipients on triple immunosuppression; and patients with advanced liver disease – avoid caspofungin mainly because of drug-drug interactions and because it requires dosage adjustment (ie, when therapeutic drug monitoring is not done for echinocandins and drug levels are uncertain)

Summary

- Echinocandins are an important class of antifungal agent with cidal activity against *Candida* species
- Safe and drug of choice for candidemia
- Has fewest drug-drug interactions
- Active in biofilms but not in CNS/eye infections

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AFWG Updates

Introducing our new committee members

We warmly welcome two new faces to AFWG: Dr Fairuz Amran and Dr Lee Lee Low, both from Malaysia, and both valuable additions to our team. We asked them about themselves and their thoughts on medical mycology.

Dr Fairuz Amran

Although Dr Amran has been working as a pathologist and clinical microbiologist since 1997, she is still very excited about her field. "We are seeing great discoveries in terms of epidemiology, diagnostics and management, especially of invasive mycoses," she shares. She is currently the head of the Bacteriology Unit of the Infectious Disease Research Centre of the Institute for Medical Research in Kuala Lumpur, Malaysia. She also serves as a consultant clinical microbiologist for the Malaysian Ministry of Health.

Dr Amran's interests are in general bacteriology and mycobacteriology, leptospirosis and mycology diagnostics. She has been heavily involved in research, has published several times in medical journals and presented multiple times at scientific meetings. She hopes that more of her peers would join her field. "Medical mycology is a niche area that only a few of my colleagues are interested in pursuing as a subspecialty. There are still a lot of issues in fungal infections in my country that need to be addressed, and we have yet to form a National Fungal Working Group," she explains. "We should have a platform to collaborate and discuss the various issues and challenges in our daily work."

Dr Amran has been working on improving their laboratory's diagnostic capabilities. "To me, the greatest challenge is making a diagnosis of invasive mycoses in a timely manner. The availability of adjunct diagnostic methods has improved diagnoses of invasive mycoses significantly over the past few years. Rapid, accurate diagnosis leads to early targeted therapy and better clinical outcome." When asked about what she would advise potential mycologists, she replied, "The most important thing is to be prepared to work hard as there is still a lot to discover and a lot of improvements to be made."

Dr Lee Lee Low

Dr Low has been an infectious disease physician since 2011 at the Hospital Sultanah Bahiyah (HSB) in Kedah, Malaysia. She shares an important turning point in her career, "I started my career as an infectious disease physician in a remote state in northern Malaysia. However, my fellowship training with Professor Debbie Marriott (Senior Specialist of the Department of Clinical Microbiology and Infectious Diseases at St Vincent's Hospital, Sydney, Australia) opened my eyes to clinical mycology. I realized there is much more to be learned in this field."

She considers laboratory diagnostics and therapeutic drug monitoring some of the biggest challenges that clinicians in Asia face in the management of fungal infections. "In terms of laboratory diagnostics and therapeutic drug monitoring, mycology is far less established than the field of bacteriology. This poses a challenge in managing difficult-to-treat fungal infections."

Dr Low serves as head of the Infection Control Unit at HSB, and is also a committee member of the antibiotic stewardship program at the same institution. In addition, she has significant research experience, being co-investigator of several major studies. However, her greatest satisfaction is still rooted in her clinical practice: "It is most satisfying for me to see patients turning the corner and showing improvement," she shares.

AFWGonline

The Asia Fungal Working Group is online!

www.AFWGonline.com

A comprehensive medical mycology resource for both researchers and healthcare professionals in Asia

November last year was an exciting time for us, as we launched our much-awaited official website. To provide our vast and varied AFWG network of professionals with updated and interesting resources, we developed AFWGonline.com to be a comprehensive medical mycology portal for both researchers and healthcare professionals in Asia.

The AFWGonline website features: original reviews, guidelines and case discussions from the AFWG Board and renowned mycology experts; downloadable resource materials and publications; details on AFWG-sponsored training courses; free online education modules and video lectures from past events; plus various other useful information for your practice.

We have recently uploaded slides and presentations from the highly successful 5th Medical Mycology Training Network (MMTN) Conference held in Bangkok, Thailand, in 2016. By simply visiting the website, mycology professionals can easily access these resources and download for their personal use or for educational events. To date, there are also three online CME modules that users can take simply by registering on the site. In the latest module, Professor Deborah Marriott of St Vincent's Hospital in Australia talks about optimizing dosing in invasive fungal infection management, and focuses on echinocandin pharmacokinetics, pharmacodynamics and therapeutic drug monitoring.

Social media presence

In our efforts to grow and interact more with the medical mycology community, we have established an online presence through social media. More and more professionals are joining our network and giving us lively feedback. We encourage all our readers to follow us on Facebook (Asia Fungal Working Group – AFWG), Instagram (@AFWGonline) and LinkedIn (AFWGonline), join the conversations, and share these with colleagues and peers.

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Visit us at AFWGonline.com and sign up for updates