



Fungal Zoonotic Diseases in HIV Patients

Tafese Hailu*

Ethiopian Veterinary Drug and Feed Quality Testing Center, Finfine/Addis Ababa, Oromia, Ethiopia

Abstract

Zoonoses are infections transmitted either directly or indirectly from vertebrate animals to humans and less frequently vice versa. Humans suffer from numerous fungal zoonoses. The most common fungal zoonotic diseases are aspergillosis, cryptococcosis, dermatophytosis, Candidiasis and coccidioidomycosis. They are opportunistic pathogens that their occurrence is facilitated by an increase in the number of immune suppressed and compromised persons due to widespread viral epidemics and aging differences. It is difficult to diagnose these fungal zoonotic diseases because there are no common signs and symptoms of zoonotic disease. In this study the most important zoonotic fungal diseases in HIV patients are reviewed.

Keywords: Animal; Infections; Diseases; Pathogens; Zoonosis; Human

Introduction

Zoonoses are defined as those diseases and infections naturally transmitted between people and vertebrate animals [1]. They are caused by an array of bacteria, viruses, fungi, or parasites. Fungal zoonotic diseases are high in HIV patients due to the immunosuppressive effect of the HIV. The most predominant are *Aspergillosis*, *Cryptococcosis*, *Dermatophytosis*, *Sporotrichosis*, *Candidiasis* and *Coccidioidomycosis*. The worldwide increase in the incidence of these diseases is mainly due to human settlements in areas where animal population is high and increased ownership of domesticated animals [2].

More than 600 fungal species are reported to infect animal and humans [3] and are associated with a wide range of diseases from skin conditions [4] to meningitis [5]. The predominant pathogens which cause severe infection in human and animal are *Aspergillus* species, *Microsporium* species, *Trichophyton* species, *Cryptococcus* species, *Candida* species and *Coccidioides* species. They are opportunistic pathogens that can occupy and colonize diverse bio compartments in individuals who come in contact with in them both outdoor environments where fungi are ubiquitous and indoor environments where fungi are found on food, in the air and many surfaces [6,7]. The emergence of fungi as a prevalent class of human pathogen has been a relatively recent occurrence, mapping temporally to the past several decades and thought to be facilitated by an increase in the number of immune suppressed persons due to widespread viral epidemics [8].

It is difficult to diagnose these fungal zoonotic diseases and can be capable of confusing even for the most experienced physicians, especially if they occur in countries where the disease is not endemic because, there is no common signs and symptoms of zoonotic disease [9]. The main objective of this paper is to highlight fungal zoonotic diseases in HIV patients.

Literature Review

Aspergillosis

Etiology: Aspergillosis is a disease caused by *Aspergillus fumigates* and occasionally other species of the genus *Aspergillus*, such as *A. terreus* and *A. niger*. These fungi are common components of the soil microflora; they play an important role in the decomposition of organic matter (Tables 1-3) [10].

Epidemiology

Geographic distribution and occurrence: The fungus is ubiquitous and distributed worldwide. The diseases have no particular distribution [11].

Source of infection and mode of transmission: The reservoir is the soil. The infecting element is conidia (exospores) of the fungus, which are transmitted through air. An important source of infection is fodder and bedding contaminated by fungus conidia. Air borne conidia are found in incubators, hatcheries, incubation room and air ducts; this may be the source of infection for chickens or turkeys and humans [11].

Disease in animals: In cattle it causes 75% of mycotic abortions. The cotyledons swell and turn a brownish gray color. The fungus may invade the fetus, causing dermatitis and bronchopneumonia. Retention of placenta is common [12]. In dog, the disease is characterized by granulomas in several organs, particularly in the kidney, spleen and bones. Lumbar discospondylitis and focal osteomyelitis are also common [13]. The symptoms in bird include fever, loss of appetite, labored breathing, diarrhea and emaciation. Localized guttural pouch mycosis will be involved in horse. Fungal granuloma in paranasal sinuses or nasal passage is also common [14].

Disease in human: Humans acquire infection through inhalation of fungal conidia. In HIV infected patients' aspergillosis commonly presents as a respiratory illness that can be a necrotizing pneumonia or a tracheobronchitis [15]. Symptoms of pneumonia include fever, dyspnoea, cough, and chest pain. Extra pulmonary forms of aspergillosis include sinusitis, cutaneous disease, osteomyelitis, and brain abscess. In several series, the most common sites for aspergillosis include the lung, gastrointestinal tract, and central nervous system, followed by other sites, including the liver, kidneys, thyroid, spleen,

*Corresponding author: Tafese Hailu, Ethiopian Veterinary Drug and Feed Quality Testing Center, Finfine/Addis Ababa, Oromia, Ethiopia, Tel: +251912727053; E-mail: tafesehailu@vet@gmail.com

Received June 14, 2018; Accepted August 21, 2018; Published August 29, 2018

Citation: Hailu T (2018) Fungal Zoonotic Diseases in HIV Patients. J Microbiol Pathol 2: 111.

Copyright: © 2018 Hailu T. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Diseases	Pathogenic fungi	Common animal	Clinical outcome	Geographical location
Aspergillosis	<i>Aspergillus fumigates</i> , <i>Aspergillus niger</i>	Birds and domestic mammal	Pneumonia or thracheobronchitis. and Extra pulmonary illness	Worldwide
Cryptococcosis	<i>Cryptococcus neoformans</i> , <i>Cryptococcus gattii</i>	Birds, mammals, reptiles and amphibians	local or generalized infection of central nervous system	Worldwide with <i>C. gattii</i> restricted to tropical and sub-tropical region
Dermatophytosis	<i>Microsporumcanis</i> , <i>T. mentagrophytes</i> and <i>verrucosum</i> .	All domestic animal and wildlife	Peculiar ring lesion	America, Africa, sometimes Europe.
Sporotrichosis	<i>Sporothrix schenckii</i> <i>Sporothrix brasiliensis</i>	Cats, dogs, horse, cow, camel, goat, bird, pig, rat, and armadillo	Localized cutaneous, lymphocutaneous and disseminated infection	Worldwide with main endemicity in Japan, India, Mexico, Brazil, Uruguay and Peru
Candidiasis	<i>Candida albicans</i>	Chicken or pigeon coops, bird roosts and bat	-oral cavity Lesions (erosions, ulcers, and fissuring of the lips)-vulvovaginitis	Worldwide
Coccidioidomycosis	<i>Coccidioides immitis</i>	Dogs, llamas, cats, horses	Asymptomatic to severe and fatal infection	USA, Mexico, Central and South America

Table 1: Pathogenic fungi with the potential of transmission to human and its clinical outcome in different geographical locations.

Disease	Global estimate of annual occurrence	References
Pulmonary Aspergillosis	3,000,000	[3,5,74-82]
Invasive aspergillosis	250,000	
Cryptococcosis	223,000	
Dermatophytosis	1,000,000,000	
Sporotrichosis	740,000	
Invasive Candidiasis	700,000	
Coccidioidomycosis	25,000	

Table 2: Estimates of occurrence of fungal diseases per year for the last 20 years on globe.

Fungal infection	Severity	Disease stage (in HIV)	Route of transmission
Pulmonary aspergillosis	Chronic	Stage 4	Inhalation of conidia
Invasive Aspergillosis	Acute	Stage 3	
Cryptococcosis	Acute	Stage 4	Inhalation, sometimes through skin damage
Dermatophytosis	Acute	Stage 1	Direct or indirect contact with infected animals or material
Sporotrichosis	Chronic	Stage 3	Scratches or bites from animals and traumatic inoculation
Invasive candidiasis	Acute	Stage 3	
Coccidioidomycosis	Chronic	Stage 3	Skin damage and Inhalation of arthroconidia

Table 3: Severity of fungal zoonotic infection and its route of transmission in HIV patients [3].

and heart. Bronchoscope examination demonstrates that ulcerative or plaque like lesions adherent to tracheal wall.

Diagnosis: Diagnosis in both human and animal should be based on clinical sign and laboratory results. Definitive diagnosis of the diseases is based on demonstration of the organisms within lesion by culture. Ant-mortem demonstration of high concentration of antibodies to *Aspergillus* species provides tentative diagnosis. Both agar gel immune diffusion assays and ELISA are available for serological examination. These assays may not be useful in immunosuppressed patients [16].

Control and prevention: Due to the ubiquitous nature of the fungus, it is impossible to establish practical control measures. It is advisable to take special precautionary measures to avoid nosocomial outbreak. HIV patients should receive prophylactic treatment with amphotericin B. Moldy bedding or fodder should not be handled or given to domestic mammals and birds [17].

Treatment: Treatment of aspergillosis in HIV infected patient has not been systematically examined. Voriconazole is the recommended treatment for aspergillosis in infected patients [18]. Alternatively, lipid formulation amphotericin B can be used as the second line of agent; which include echinocandins (such as caspofungin, anidulafungin or micafungin) and posaconazole.

Cryptococcosis

Etiology: Cryptococcosis is the commonest systemic fungal infections caused by *Cryptococcus neoformans* and *Cryptococcus gattii* [19], which have a predilection site of pulmonary and central nervous system [20]. The major virulence factors associated with *Cryptococcus* are the ability to grow at 37°C [21], the production of the pigment melanin [22,23] and the formation of polysaccharide capsule [24].

Epidemiology

Geographic distribution: *Cryptococcus neoformans* is found worldwide, whereas *C. gattii* most often is found in Australia. Approximately 5% to 8% of HIV infected patients in developed countries were diagnosed with disseminated *Cryptococcosis* [25]. *C. gattii* was believed to be restricted to the tropics and subtropical regions [26].

Source of infection and mode of transmission: *C. neoformans* var. *Neoformans* is located most frequently in soil containing high amounts of bird excreta. People can stir up the dust and then breathe it in when they work, play, or walk in areas. Infection occurs *via* inhalation [27].

Disease in animal: This disease has been recognized in cattle, horse, sheep, goat, dog, and several species of wild animals. Various cases have been described in sheep and goat with localized pulmonary disease and mastitis. In goat the disease is characterized by accumulated fluid in

pleural and peritoneal cavities, atelectatic lungs, dark red plaque in trachea, and alopecic lesion on the head from which yellow exudates seeped [28].

The disseminated form of the disease is most commonly diagnosed in dog and cats. The primary diagnosis in cat is the disorder of central nervous system, with granulomas in the eye and nasal passage, meningoencephalitis and optical neuritis [29].

Disease in human: In HIV infected patients *Cryptococcosis* is commonly presents as a sub-acute meningitis or meningoencephalitis with fever, malaise, and headache [25]. Classic meningeal symptoms and signs such as neck stiffness and photophobia also occur. Some patients experience encephalopathic symptoms such as lethargy, altered mentation, personality change, and memory and visual loss that are usually a result of increased intra cranial pressure, though to result from CSF absorption or yeast infection of the brain [30]. Cryptococcal pneumonia may be either asymptomatic or symptomatic, with or without evidence of dissemination, and clinical manifestations include fever and cough that produces scant sputum, shortness of breath, and pleuritic pain. Physical examination may reveal localized nodular lesions, pleural effusions, tachypnea, and thoracic lymphadenopathy [31].

Diagnosis: Diagnosis is made based on the history of disease in area, lesion and clinical sign. Cryptococcal disease can be diagnosed through culture of blood or CSF on Sabouraud’s dextrose agar with incubation at 30°C to 37°C, CSF microscopy with India ink staining and Cryptococcal antigen detection. Three methods exist for Cryptococcal capsular antigen detection: Latex agglutination, enzyme immunoassays, and lateral flow assays [32].

Control and prevention: There are no specific measures for preventing the disease. However, it is important to control the underlying diseases. According to the World Health Organization (WHO), early antiretroviral therapy (ART) initiation is the most important and cost-effective preventive strategy to reduce the incidence and high mortality associated with Cryptococcal meningitis in HIV-infected adults, adolescents and children. Controlling the bird population and removal of their excrement followed by chemical decontamination might prevent some cases [33]. To date, there is no effective vaccine to prevent Cryptococcosis. In truth, the best way to prevent this disease is to not inhale the fungus. Therefore, the use of masks can be of help to prevent inhalation.

Treatment: The combination of amphotericin B plus Flucytosine for 4 to 6 weeks has been considered standard treatment for patients without HIV infection. However, concern about increased toxicity and decreased efficacy [34] has led to a reconsideration of this regimen in HIV-infected patients. Several studies have examined the use of oral fluconazole instead of amphotericin B for initial therapy of Cryptococcal meningitis in patients with HIV infection [35].

Dermatophytosis

Etiology: Over 90% of worldwide feline dermatophytosis cases are caused by *Microsporium canis* [36]. Others are caused by infection with *M. gypseum*, *Trichophyton mentagrophytes*, *T. quinckeanum*, *T. verrucosum* or other agents. With the exception of *M. gypseum*, all produce proteolytic and keratolytic enzymes that enable them to utilize keratin as the sole source of nutrition after colonization of the dead, keratinized portion of epidermal tissue (mostly Stratum corneum and hairs, sometimes nails) [37].

Epidemiology

Geographic distribution

Source of infection and mode of transmission: Ring worm occurs in all animal species in all countries but more commonly where animals are accommodated in dense groups; especially indoors. Cat is the most common host and reservoir of *M. canis*. Dermatophytes can be transmitted from animal to human or animal either directly through contact with asymptotically infected animals or skin lesions on infected animals, or indirect contact with contaminated bedding, harness, grooming kits and horse blankets, fungi in the air, in dust, or on surfaces of the room. Spore can exist on the skin without causing lesion, and up to 20% of normal animals in an infected group will act as carrier animal (Table 4) [38].

Disease in animal: The typical presentation of ringworm in animal is regular and circular alopecia about 3cm, with hair breakage, desquamation, sometimes an erythematous margin and central healing. The lesions are located on head, neck, tail and perineum but general distribution over the body will occur. Multiple lesions may coalesce in "map-like" appearance with soreness to touch [39].

Disease in human: Symptoms of *Dermatophytosis* in human are mild and self-limiting. Skin lesions show circular pattern of development, which is clear in the center forming a ring and categorized as Tinea Capitis (infection on scalp and hair), Tinea barbae (infection of beard) Tinea Corporis (infection on extremities, arm and hand) [40], Tinea Pedis (athlete’s foot) and Tinea Unguium (infection on nails) [41].

Diagnosis: Clinical diagnosis can be confirmed by Woods light test which is a black light (ultraviolet light) with magnifying lens. However, it is not very sensitive: only about 50% of *M. canis* strains fluoresce and other dermatophytes do not at all [37]. Microscopic examination is another simple and rapid method to detect dermatophytes on hairs or scales. The sample should be cleared with 10-20% KOH before examination to improve the visualization of fungal elements on the hair shafts [36].

Culture on Sabouraud dextrose agar is the gold standard for the detection of dermatophytes. This method is very sensitive and can determine the species. Samples (hairs, scales) should be collected from the margin of new lesions after gently swabbing with alcohol to reduce contamination. Polymerase chain reaction (PCR) has been proposed for the detection of *M. canis* sequences [42].

Control and prevention: Avoiding contact with animals that are obviously sick can prevent a certain percentage of human cases.

Organism	Geographic distribution
<i>E. floccosum</i>	Worldwide
<i>M. auduninii</i>	Europe, rare in N. America
<i>M. canis</i>	N. America, some of Europe
<i>M. ferrineum</i>	Africa, India, China, Japan
<i>M. gypseum</i>	Worldwide
<i>M. nanum</i>	Worldwide
<i>M. persicolor</i>	Worldwide
<i>T. equinum</i>	Worldwide
<i>T. mentagrophytes</i>	Worldwide
<i>T. soudanese</i>	Africa
<i>T. tonsurans</i>	Worldwide
<i>T. verucosum</i>	Worldwide

Table 4: Geographic distribution of dermatophytosis.

Remains of hair and scales should have to be burned and rooms, stables, and all utensils should be disinfected. Advice often given includes washing clothes in hot water with fungicidal soap after suspected exposure to ringworm, avoid walking barefoot; instead wear appropriate protective shoes in locker rooms and sandals at the beach [43], and avoid touching pets with bald spots as they are carriers of the fungus [44,45]. A vaccine consisting of killed *M. canis* components in adjuvant was licensed in the USA for feline use [46].

Treatment: Antifungal treatments include topical agents such as miconazole, terbinafine, clotrimazole, ketoconazole, or tolnaftate applied twice daily until symptoms resolve usually within one or two weeks. Topical treatments should then be continued for a further 7 days after resolution of visible symptoms to prevent recurrence [47].

In the USA, lime-sulphur solution is commonly used. In some countries, the fungistatic drug griseofulvin is still used. It is administered orally for at least 4-6 weeks at 25-50 mg/kg twice daily [48].

Sporotrichosis

Etiology: Sporotrichosis is a chronic granulomatous disease caused by *Sporothrix schenckii* which is a dimorphic fungus [49]. It survives in the environment and becomes pathogenic in animals as a result of the dimorphic abilities of the organism; this dimorphism is the conversion from a yeast-like form at temperatures between 35 and 37°C to a mycelial phase (with branching, septate hyphae) at environmental or laboratory temperatures of 25 to 30°C [50].

Epidemiology

Geographic distribution: Sporotrichosis occurs in Africa, Australia, India, Japan and the Americas, but is rare in Europe. High occurrence has been observed in Latin America, especially in Brazil [51].

Source of infection and mode of transmission: This fungus is isolated from decaying vegetation such as thorns, straw, hay, wood, and infected animals. *Sporotrichosis* usually results from the traumatic inoculation of *Sporothrix* spp. in the dermis. Transmission from animal to man is by contact with lesion, contaminated soil, penetration of thorns or wood chips, scratching, direct contact with or biting from infected animals and inhalation [52].

Disease in animal: Lesion can be anywhere but usually located on distal extremities, head, or base of tail. In animals the disease is usually cutaneous or lymphocutaneous lesions generally develop in association with the lymphatic chain and are detected initially as nodules that are painful upon palpation; the nodules eventually ulcerate and suppurative lymphadenitis develops. Lesions may be with microabscesses at the border; the exudate from these lesions is usually thick and brownish red. Cutaneous lesions of *sporotrichosis* in the cat are most often observed on the legs, face, or nasal plenum [53].

Although the localized cutaneous form remains localized to the skin, there may be regional multicentric lesions. In cats, the lungs and liver are the primary sites for dissemination of *S. schenckii*; however, involvement of bones, eye, CNS, gastrointestinal tract, spleen, kidney, mammary gland, testis, and epididymis also occur [50].

Disease in human: Clinical presentations of *sporotrichosis* may vary according to the immunological status of the host, the load and depth of the inoculum, and the pathogenicity and thermal tolerance of the strain, among other factors [54].

According to the location of the lesions, *Sporotrichosis* can be classified into cutaneous, mucosal, and extracutaneous forms. The

most common clinical form is cutaneous form. Initially, a papule or pustule, followed by formation of a subcutaneous nodule is formed at the site of inoculation. The nodules may ulcerate and yellowish or gray pus appears. In mucosal form the lesions in the nasal mucosa often involve the septum, with drainage of bloody secretions and detachment of crusts. In the conjunctiva, the granulomatous lesion is accompanied by a serous-purulent presence or not of lid edema [55]. In the extracutaneous forms one or several joints and bones can be involved, as well as tenosynovitis or bursitis. Pulmonary sporotrichosis, a rare form of disease results from inhalation of the fungus. The most common symptoms are cough, expectoration, dyspnea, pleuritic pain, and hemoptysis [56].

Diagnosis: *Sporotrichosis* can be diagnosed through a correlation of clinical, epidemiological, and laboratory data. *Sporotrichosis* in cats is most often diagnosed via cytological evaluation of samples obtained from aspiration of abscesses or nodules, impression smears of ulcerated skin or exudate, smears of swab specimens, or skin scrapings. Smears are air-dried and stained with Wright's or a Romanowsky-type stain [57].

Results of fungal culture of specimens from lesions are needed for definitive diagnosis of *Sporotrichosis* in humans and animals. This fungus is cultured on Sabouraud Mycologic medium. When *Sporotrichosis* is suspected, but yeast like cells are not observed cytologically or cultured from specimens obtained from lesions, results of *Sporothrix* whole yeast agglutination or latex agglutination testing may be required for diagnosis [58].

Control and prevention: Veterinarians, veterinary technicians, and animal owners should always wear gloves when handling cats with ulcerative lesions or open draining tracts. Without doubt, owners should be advised of the zoonotic potential of cutaneous *Sporotrichosis*. After handling pets with *Sporotrichosis*, hands and arms should be washed with an antiseptic solution of known antifungal activity such as povidone iodine or chlorhexidine solutions [59].

Treatment: Treatment of *Sporotrichosis* depends on the severity and location of the disease. The treatment options for this condition are: Saturated potassium iodide solution, Itraconazole (Sporanox), fluconazole, Amphotericin B, Newer triazoles and Surgery in cases of bone infection and cavitary nodules in the lungs [15].

Candidiasis

Etiology: Candidiasis is the fungal zoonotic diseases caused by infection with species of the genus *Candida*, predominantly with *Candida albicans*. Other less frequent species are *candida tropicalis*, *candida krusei*, *candida parapsilosis*, *candida pseudotropicalis*, etc. In Sabouraud's medium it forms creamy white, convex colonies [60].

Epidemiology

Geographic distribution: Bovine abortions due to *Candida* spp., while sporadic, have a worldwide distribution with reports from England, Poland, Hungary, Germany, New Zealand, Israel, India, and the USA [61].

Source of infection and mode of transmission: *C. albicans* as a component of the normal flora in the digestive system of a high percentage of healthy individuals and animals. The infection can spread through contact with oral secretions, skin, and vagina of sick individuals or carriers [62].

Disease in animal: Infection may be localized or disseminated to

other part of body via hematogenous route, resulting in micro abscesses at multiple sites. Localized *Candidiasis* is usually characterized by ulcers in oral cavity, gastrointestinal tract, or genitourinary mucosa. Disseminated disease is typified by fever and erythematous skin lesion [14].

Aspergillus fumigatus accounts for approximately 75% of the cases of mycotic abortion in cattle, with the remaining 25% caused by species in the genus *Candida*. *Candida* spp. can on rare occasions, lead to mastitis. Avian *Candidiasis* is an infection of upper respiratory system and sometimes nervous system. However, the disease is generally asymptomatic and diagnosis occur post-mortem. The most frequent lesion is found in the crop and consists of plaques that resemble curdled milk that adhere to mucosa. It also causes thickening of the crop wall [10].

Disease in human: In HIV-infected patients, *Candidiasis* is virtually always mucocutaneous, involving the oropharynx, the esophagus, and the vagina. The most frequent form of the mucosal infection presents clinically as a mycotic stomatitis (thrush) characterized by lightly adhering white plaques on the tongue and other parts of the mouth that can leave a bloody surface when removed [63].

Infection of vulva or vagina causes severe itching, burning, soreness, and whitish or whitish gray cheese like discharge, often with curd-like appearance. Infection of male genital (Balanitis candidiasis) include red skin around the head of penis, swelling, irritation, and soreness of penis, thick lumpy discharge under the skin, unpleasant odour, difficulty retracting the penis (phimosis) and pain when passing urine or during sex [61]. Perianal infection can cause pruritis with erythematous, papular or ulcerative lesions. Esophageal *Candidiasis* can cause dysphagia or odynophagia (painful swelling) [64].

Diagnosis: The diagnosis of almost any form of *Candida* disease requires an integration of clinical, epidemiological, and laboratory findings. For identification by light microscope a scraping or swab of affected part is placed on a microscopic slide and 10% of KOH is added to specimen to enhance visualization. The most widely used serological test is immunodiffusion. Definitive diagnosis is made by culturing on Sabouraud's medium at 37°C [65].

Control and prevention: The most important measure for preventing the spread of pathogens is effective hand washing, Cleaning, disinfection and sterilization (Miller, 2010). Neonatal *Candidiasis* can be prevented by treating the mother's vaginal *candidiasis* with nystatin. To prevent epidemics in nurseries, patients with oral *candidiasis* should be isolated and strict hygiene measures should have to be established [66].

Treatment: The latest recommendations include the echinocandins caspofungin micafungin, and anidulafungin, along with voriconazole and posaconazole, as well as lipid formulations of amphotericin B in various situations. Fluconazole is still considered a first-line agent in non-neutropenic patients with *candidemia* or suspected invasive *candidiasis* [67].

Coccidioidomycosis

Etiology: *Coccidioidomycosis* is caused by soil dwelling fungus that consists of two species; *Coccidioides immitis* and *Coccidioides posadasii*. The fungus occurs in one phase in natural environment (soil), and parasitic phase in the mammalian host [68].

Epidemiology

Geographic distribution: *Coccidioidomycosis* is currently increasing in the United States due to significant growth in population and

tourism in endemic area. The disease is enzootic in the south-western United States and up to 20% of cattle finished in the area may harbor the fungus [69].

Source of infection and mode of transmission: *C. immitis* is a soil saprophyte in arid and semiarid regions. The infection is transmitted to man and animals through inhalation of wind-borne arthrospores of the fungus; it occurs more frequently after dust storms. The infection can be contracted in laboratory by inhaling the spores from fungus cultures [70].

Disease in animal: In horse findings include weight loss up to severe emaciation, a fluctuating temperature, persistent cough, and superficial abscesses, often recurring and most commonly in pectoral area, increased lung sounds, wheezing, and dullness are audible over the ventral chest. Thickening of placenta, plaque like lesion on the umbilical cord and nodules in the lung of fetus were recorded in the case of abortion. The lesion produced in cattle, sheep, and pigs is disseminated mycosis with granulomas in the lung, liver, and kidneys [14].

Disease in human: Common syndromes of *Coccidioidomycosis* in HIV infected patients are focal pneumonia, diffuse pneumonia, cutaneous disease, meningitis, liver, and lymph node involvement. Focal pneumonia is the most common and difficult to distinguish from bacterial pneumonia. Patients present symptoms such as cough, fever and pleuritic chest Pain. Diffuse pulmonary disease present with fever and dyspnoea [71].

Diagnosis: Diagnosis is usually based on the history of animal case from an endemic area, clinical finding and histopathology. Diagnosis is confirmed by culture of the fungus on appropriate media. Coccidioidal Immunoglobulin M (IgM) immunoglobulin G (IgG) serology performed by ELISA, immuno-diffusion is useful in diagnosis. It has been shown to detect antigen in urine and serum samples from HIV infected patients with active *coccidioidomycosis* [72].

Control and prevention: Care should be taken in handling, changing, and discarding dressings, casts, and similar materials in which contamination could occur. Measures to control dust are recommended in areas with endemic infection, including construction sites and other activities that cause excessive soil disturbance. Immunosuppressed people residing in or traveling to areas with endemic infection should be counseled to avoid exposure to activities that may aerosolize spores in contaminated soil.

Treatment: Initial therapy with a triazole antifungal is appropriate. Fluconazole or itraconazole of 400mg daily is effective. For the patients who fail to respond to fluconazole or itraconazole the newer triazoles (posaconazole and voriconazole) may be useful. Amphotericin B is the preferred therapy for diffuse pulmonary involvements [68,73].

Conclusion and Recommendations

Fungal zoonoses are the most important public health problem on the world. The increased incidence of these infections and the diversity of fungi causing them throughout world have paralleled the emergence of HIV infection that lowered the immunity of the host. Contemporarily, those diseases are becoming widespread due to human settlements along with animal population, increased ownership of domesticated animals, mutation rates of virus (HIV), poor understanding of the epidemiology and public health importance of the diseases. The diagnosis of these diseases is difficult resulting in challenge in the prevention and control strategies.

Based on the above conclusions, the following recommendations are forwarded:

1. Improved diagnostic methods have to be developed for an early diagnosis of opportunistic mycosis for control and prevention of the disease and save more lives.
2. The community has to be aware the public health importance of fungal zoonosis.
3. The coordination between medical, veterinary and public health department has to be encouraged.
4. Immuno-deficient individuals (HIV patients) have to be counseled to avoid exposure to infected animal and take prophylactic antibiotics.

References

1. World Health Organization, WHO (2005) The Control of Neglected Zoonotic Diseases. Report of a Joint WHO/DFID-AHP Meeting with the participation of FAO and OIE, Geneva.
2. Weiss LM (2008) Zoonotic parasitic diseases: emerging issues and problems. *Int J Parasitol* 38: 1209-1210.
3. Brown GD, Denning DW, Levitz SM (2012) Tackling Human Fungal Infections. *Sci* 336: 647-647.
4. Saunders CW, Scheynius A, Heitman J (2012) Malassezia Fungi are Specialized to live on skin and associated with dandruff, eczema and other skin diseases. *Clin Infect Dis* 24: 1-234.
5. Woerden HCV, Gregory C, Brown R, Marchesi JR, Hoogendoorn B (2013) Differences in fungi present in induced sputum samples from asthma patients and non-atopic controls: a community based case control study. *BMC Infect Dis* 13: 1471-2334.
6. Amend AS, Seifert KA, Samson R, Bruns TD (2010) Indoor fungal composition is geographically patterned and more diverse in temperate zones than in the tropics. *Proc Natl Acad Science* 107: 13748-13753.
7. Shelton BG, Kirkland KH, Flanders WD, Morris GK (2002) Profiles of airborne fungi buildings and outdoor environments in the United States. *Appl Environ Microbiol* 68: 1743-1753.
8. IOM (2011) Fungal Diseases: An Emerging Threat to Human, Animal and Plant Health. In: *Medicine*. 10th edition. The National Academies Press, Washington DC.
9. Cascio A, Bosilkoviski M, Rodriguez MAJ, Pappas G (2011) The socio ecology of zoonotic infections. *Clin Microbiol Infect* 17: 336-342.
10. Pedro N, Ach BS (2003) Zoonoses and communicable disease common to man and animals (3rd edn). ASM Press, Washington DC.
11. Chute HL, Richards JL (1991) Fungal infections, p: 234. In: Calnek, Barnes BWHJ, Beard CW, Reid WM, Yoder HW (eds.) *Disease of poultry* (9th edn). Ames: Iowa State University press.
12. Schmitt JA (1981) Mycotoxic disease. In: Ristic MI (ed.) *Diseases of cattle in the tropics*, (2nd edn), pp: 121-124. The Hague: Martinus Nijhoff, Dutch.
13. Day MJ, Penhale WJ, Eger CE (1986) Disseminated aspergillosis in dogs. *Aust Vet J* 63: 55-59.
14. Richard W, Nelson (2005) Systemic Mycosis. In: Ethinger JS, Feldman EC (eds.) *Textbook of Veterinary Internal Medicine*. Saunders Elsevier, (3rd edn), Philadelphia, PA, USA, pp: 667-690.
15. Lortholary O, Denning DW, Dupont B (1999) Endemic mycosis treatment update. *J Antimicrob Chemother* 43: 321-331.
16. Depaw B, Walsh TJ, Donney JP (2008) Revised definitions of invasive fungal disease from European organization for research and treatment of cancer/ invasive fungal infections cooperative groups and national institute of Allergy and infectious Disease Mycosis study Group (EORTC (MSG) consensus Group). *Clin Infect Dis* 48: 1813-1821.
17. Iwen PC, Reed JO, Armitage JO, Bierman PJ, Kessinger A, et. al (1993) Nosocomial invasive aspergillosis in lymphoma patients treated with bone marrow or peripheral stem cell transplants. *Infect cont Hosp Epidemiol* 14: 131-139.
18. Walsh TJ, Anaissie EJ, Denning DW (2008) Treatment of Aspergillosis: Clinical practice guidelines of infectious disease society of America. *Clin Infect Dis* 46: 327-360.
19. Gupta G, Fries BC (2010) Variability of phenotypic traits in *Cryptococcus* varieties and species and the resulting implications for pathogenesis. *Future Microbiol* 5: 775-787.
20. Junaid SA, Olabode AO, Udeani TKC, Aikoye S (2008) Prevalence of Pulmonary Cryptococcosis in HIV/AIDS Patients. *Afr J Infect Dis* 2: 74-79.
21. Nichols CB, Perfect ZH, Alspaugh JAA (2007) Ras1-Cdc24 signal transduction pathway mediates thermo-tolerance in the fungal pathogen *Cryptococcus neoformans*. *Mol Microbiol* 63: 1000-1118.
22. Jiang N, Xiao D, Zhang D, Sun N, Yan B, et al. (2009) Negative roles of a novel nitrogen metabolite repression-related gene, TAR1, in lactase production and nitrate utilization by the basidiomycete *Cryptococcus neoformans*. *Appl Environ Microbiol* 75: 6777-6782.
23. Panepinto J, Komperda K, Frases S (2009) Sec 6-dependent sorting of fungal extracellular exosomes and lactase of *Cryptococcus neoformans*. *Mol Microbiol* 71: 1165-1176.
24. Zaragoza O, Rodrigues ML, Jesus DM, Frases S, Dadachova E, et al. (2009) The capsule of the fungal pathogen *Cryptococcus neoformans*. *Adv Appl Microbiol* 68: 133-216.
25. Aberg JW (2002) Cryptococcosis. In: Dolin RMH, Saag MS (eds.) *AIDS Therapy*, pp: 498-510. Churchill Livingstone, New York, USA.
26. Ellis DH, Pfeiffer TJ (1990) Ecology life cycle and infectious propagule of *Cryptococcus neoformans*. *Lancet* 336: 923-925.
27. Jean SS, Fang CT, Shau WY (2002) Cryptococcaemia: Clinical features and prognostic factors. *Clin Dis Infect* 95: 511-518.
28. Chapman HM, Robinson WF, Bolton JR, Robertson JP (1990) *Cryptococcus neoformans* infection in goats. *Aust Vet J* 67: 263-265.
29. Malik R, Wigney DI, Muir DB (1992) Cryptococcosis in cats: Clinical and mycological assessment of 29 cases and evaluation of treatment using orally administered fluconazole. *J Med vet Mycol* 30: 133-144.
30. Mitchell TG, Perfect JR (1995) Cryptococcosis in the Era of AIDS-100 Years after the Discovery of *Cryptococcus neoformans*. *Clin Microbiol Rev* 8: 515-548.
31. Onen CL (2009) Clinical Diagnosis of AIDS and HIV-Related Diseases. *Afr* 2002: 297-321.
32. Powderly WG, Cloud GA, Mukes DW, Saag, MS (1994) Measurement of Cryptococcal antigen in serum and CSF Value in the management of AIDS-associated Cryptococcal meningitis. *Clin infect Dis* 18: 789-792.
33. World Health Organization, WHO (2011). Rapid advice: Diagnosis, Prevention and Management of Cryptococcal disease in HIV infected adults, adolescents, and children. Geneva.
34. Chuck SL, Sande MA (1989) Infections with *Cryptococcus neoformans* in the acquired immunodeficiency syndrome. *Engl J Med* 321: 793-799.
35. Larsen RA, Leal MAE, Chan LS (1990) Fluconazole compared with amphotericin B plus flucytosine for cryptococcal meningitis in AIDS: A randomized trial. *Ann Intern Med* 113:183-187.
36. DeBoer DJ, Moriello KA (2006) Cutaneous fungal infections. In: Greene CE (ed.) *Infectious diseases of the Dog and Cat*, St Louis, Missouri. Elsevier Saunders (4th edn), pp: 555-569.
37. Sparkes AH, Werrett G, Stokes CR, Gruffyd JTJ (1994b) Micro sporum canis in apparent carried by cats and the viability of arthrospores. *J Small Anim Pract* 35: 397-401.
38. Radostitis OM, Gay CC, Hinchliff KW, Constable PD (2007) *Veterinary Medicine: A text book of the disease of cattle, sheep, goats, pigs and horses* (10th edn), Saunders, Edinburg, London.
39. Chermette R, Ferreira L, Guillot J (2008) Dermatophytoses in animals. *Mycopathologia studies. Vet Dermatol* 15: 99-107.
40. Silva HM, Wetzman I, Rosenthal SA (1981) Cutaneous mycosis (Dermatomycosis). In: Balows A, Hausler WJ (eds.) *Diagnostic procedure for Bacterial, Mycotic and Parasitic Infections* (6th edn), American Public Health Association. Washington DC, p: 212.

41. Del PA, Pereiro MM, Gimeno C, Cuetara MS, Rubio R, et al. (1992) Widespread dermatophytosis due to *Microsporum* (*Trichophyton*) *gallinae* in a patient with AIDS: A case report from Spain. *Clin Exp Dermatol* 17: 449-453.
42. Nardoni S, Franceschi A, Mancianti F (2007) Identification of *Microsporum canis* from dermatophytic pseudomycetoma in paraffin-embedded veterinary specimens using a common PCR protocol. *Mycoses* 50: 215-217.
43. Klemm L (2008) Keeping footloose on trips. *The Herald News*.
44. Hammer KA, Carson CF, Riley TV (2003) Antifungal activity of the components of *Melaleuca alternifolia* (tea tree) oil. *Journ of Appl Microbiol* 95: 853-860.
45. Fontenelle RO, Morais SM, Brito EH (2007) Chemical composition, toxicological aspects and antifungal activity of essential oil from *Lippiasidoides* Cham. *J Antimicrob Chemother* 59: 934-940.
46. DeBoer DJ, Moriello KA, Blum JL, Volk LM, Bredahl LK (2002) Safety and immunologic effects after inoculation of inactivated and combined live-inactivated dermatophytosis vaccines in cats. *Am J Vet Res* 63: 1532-1537.
47. Kyle AA, Dahl MV (2004) Topical therapy for fungal infections. *Am J Clin Dermatol* 5: 443-451.
48. Foil C (2005) Ringworm update. In: Plumb D (ed.) *Plumb's Veterinary Drug Handbook*, Blackwell Publishing, Iowa. Proceedings: Western Veter Conference (7th edn) 1-544.
49. Larone DH (1995) Thermally dimorphic fungi and medically important fungi. A guide to identification. 3rd edn. Washington DC.
50. Quinn PJ, Carter ME, Markey BK (1994) The dimorphic fungi. In: *Clinical veterinary microbiology*, (3rd edn), Microorganism by Year Book Europe, London, pp: 402-408.
51. Barros MB, Paes RA, Schubach AO (2011) *Sporothrix schenckii* and Sporotrichosis. *Clinical Microbiol Reviews*, Washington DC 24: 633-654.
52. Leme LRP, Schubach TMP, Santos IB, Figueiredo FB, Pereira SA, et al. (2007) Mycological evaluation of broncho alveolar lavage in cats with respiratory signs from Rio de Janeiro, Brazil. *Mycoses*, Berlin. *Clin Infect Dis* 50: 210-214.
53. Werner AH (1993) Feline sporotrichosis. *Compend Contin Educ Pract Vet* 15: 1189-1197.
54. Arrillaga MI (2009) Different virulence levels of the species of *Sporothrix* in a murine model. *Clin Microbiol Infect* 15: 651-655.
55. Schubach A, Lima MB, Schubach TM, Francesconi VAC (2005) Primary conjunctival sporotrichosis: two cases from a zoonotic epidemic in Rio de Janeiro, Brazil. *Cornea* 24: 491-493.
56. Kauffman CA, Bustamante B, Chapman SW, Pappas PG (2007) Clinical practice guidelines for the management of sporotrichosis: update by the Infectious Diseases Society of America. *Clin Infect Dis* 45: 1255-1265.
57. Clinkenbeard KD (1991) Diagnostic cytology: Sporotrichosis. *Compend Contin Educ Pract Vet* 13: 207-211.
58. Casserone S, Conti DIA, Zanetta E, Pereira MEP (1983) Serologia De la esporotricosis cutánea. *Sabouraudia* 21: 317-321.
59. Rutala WA (1995) Antisepsis, disinfection, and sterilization in hospitals and related institutions. In: Murray PR, Baron EJ, Pfaller MA (eds.) *Manual of Clinical Microbiology* (6th edn), American Society for Microbiology, Washington DC, pp: 227-245.
60. Pappas PG, Kauffman CA, Andes D, Benjamin DK Jr, Calandra TF, et al. (2009). Clinical practice guidelines for the management of candidiasis: 2009 update by the Infectious Diseases Society of America. *Clin Infect Dis* 48: 503-535.
61. David LM, Walzman RM, Rajamanoharan S (1997) Genitals colonization and infection with *Candida* in heterosexual and homosexual males. *Genitourin Med* 73: 394-396.
62. Vazquez JA, Snchez V, Dmuchowski C (1993) Nosocomial acquisition of *Candida albicans*. An epidemiologic study. *J Infect Dis*, pp: 168-201.
63. Edwards JE (1990) *Candida* species. In: Mandell GL, Dougllass JR, Bennett JA (eds.) *Principles and practice of infectious diseases* (3rd edn), New York. Churchill Livingstone.
64. Yamada T, Alpers DH (2009) *Textbook of gastroenterology*. Blackwell pub, Chichester, West Sussex, (5th edn).
65. Chakravarthi SK, Nagaraja HS (2010) A comprehensive review of the occurrence and management of systemic candidiasis as an opportunistic infection. *Microbiol Journ* 1: 1-5.
66. Ajelo L, Kaplan W (1980) In: *CRC Handbook series in zoonosis: Systemic mycosis*. Stonner HW, Kaplan M (eds.) CRC Press, Boca Raton, Florida.
67. Pappas PG, Rex JH, Sobel JD (2004) Guidelines for treatment of candidiasis. *Clin Infect Dis* 38: 161-189.
68. Galgiani JN, Ampel NM, Blair JE (2005) *Coccidioidomycosis*. *Clin Infect Dis* 41: 1217-1223.
69. Barker BM, Jewell KA, Kroken S, Orbach M (2007) The population biology of coccidioides: Epidemiologic implications for disease outbreaks. *Ann NY Acad Sci* 111: 147-149.
70. Ampel NM, Wieden MA, Galgiani JN (1989) *Coccidioidomycosis: Clinical update*. *Rev Infect Dis* 11: 897-911.
71. Kim MM, Blair JE, Carey EJ, Wu Q, Smilack JD (2009) *Coccidioidal pneumonia phoenix Arizona, USA*. *Clin infect Dis* 15: 397-401.
72. Durkin M, Estok L, Hospental D (2009) Detection of coccidioides antigenemia following dissociation of immune complex. *Clin Vaccine Immunol* 16: 1453-1456.
73. Stevens DA, Rendon A, Gaona FV (2007) Posaconazole therapy for chronic refractory coccidioidomycosis. *Clin Infect Dis* 132: 952-958.
74. Frasés S, Ferrer C, Sánchez M, Colom VM (2009) Molecular epidemiology of isolates of the *Cryptococcus neoformans* species complex from Spain. *Rev Iberoam Micol* 26: 112-117.
75. Guy JP, Raza S, Bondi E, Rosen Y, Kim DS, et al. (2012) *Cryptococcus pneumoniae* presenting in an immunocompetent host with pulmonary asbestosis: A case report. *J Med Case Rep* 6: 170-170.
76. Lupo P, Chang YC, Kelsall BL (2002) The presence of capsule in *Cryptococcus neoformans* the gene influences expression profile in dendritic cells during interaction with the fungus. *Infect Immun* 76: 1581-1590.
77. Miller CH, Palenik CJ (2014) *Infection Control and Management of Hazardous Materials for the Dental Team 5: Infection Control and Management of Hazardous Materials for the Dental Team*. Elsevier Health Sciences.
78. Rajasingham R, Rachel MS, Benjamin JP, Joseph NJ, Nelesh PG, et al. (2017) Global Burden of Disease of HIV-Associated Cryptococcal Meningitis: An Updated Analysis. *Lancet Infect Dis* 17: 873-881.
79. Saugier VP, Devergie A, Sulahian A (1993) Epidemiology and diagnosis of invasive pulmonary aspergillosis in bone marrow transplant patients: results of a 5-year retrospective study. *Bone Marr Transpl* 12: 121-124.
80. Tintelnot K, Lemmer K, Losert H, Schar G, Polak A (2004) Follow-up of epidemiological data of cryptococcosis in Austria, Germany and Switzerland with special focus on the characterization of clinical isolates 47: 455-464.
81. Wu G, Vilchez RA, Eidelman B, Fung J, Kormos R, et al. (2002) Cryptococcal meningitis: an analysis among 5,521 consecutive organ transplant recipients. *Transpl Infect Dis* 4: 183-188.
82. Fadok VA (1980) Dermatologic manifestations of the subcutaneous deep mycoses. *Compend Contin Educ Pract Vet* 2: 506-514.