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Short Communication

Coccidioidomycosis: A Highly Infectious Airborne Mycosis of Public Health Concern

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An airborne disease is one, which is transmitted by the inhalation of infectious microbe through the respiratory tract from the environment. There are many airborne diseases caused by multiple etiological agents, such as virus (Influenza), bacterium (Tuberculosis), fungus (Histoplasmosis), actinomycetes (Nocardiosis), Chlamydia (Chlamydiosis), and Rickettsia (Coxiellosis) [1]. A large number of respiratory mycoses like adiaspiromycosis, aspergillois, blastomycosis, coccidioidomycosis, cryptococcosis, geotrichosis, histoplasmosis, paracoccioidomycosis, pneumocystosis, pseudoallescheriasis, rhodotorulosis, sporotrichosis, and zygomycosis are reported from many developing as well as developed countries of the world [2,3]. These pulmonary mycotic diseases pose a great challenge to medical and veterinary professionals. Among these, coccidioidomycosis (Desert fever, Desert rheumatism, Posada-Wernicke disease, San Joaquin fever, Valley fever) is an emerging and re-emerging life threatening fungal disease, particularly among the immunocompromised patients. Coccidioidomycosis is an important saprozoonotic mycotic disease, which presents a major public health problem in endemic regions of the world. The imported cases of disease are observed in persons who travelled to endemic zones.

Coccidioidomycosis is reported from Argentina, Bolivia, Brazil, Chile, Columbia, Ecuador, Peru, Uruguay, USA, and Venezuela, The disease is endemic in certain desert areas of USA, such as Arizona, California, New Mexico and Texas. It is estimated that approximately 150, 000 cases of coccidioidomycosis occur annually in USA [3]. In hyper endemic regions of the United States, about one-fifth of the persons become sick and therefore, seeks medical help [2]. There is a dramatic upsurge in the incidence of coccidioidomycosis in endemic regions. The last decade has shown the significant increase in the incidence of pulmonary coccidioidomycosis in the United States. It is a common cause of community acquired pneumonia in endemic areas. Pulmonary coccidioidomycosis is described in healthy Japanese individuals. Coccidioidomycosis is a saprozoonosis, which is encountered in immunocompetent as well as immunocompromised subjects.

An epidemic of coccidioidomycosis associated with climate change was described in Arizona, USA. An airborne dissemination of pathogen was responsible for an epidemic in San Joaquin Valley. The point outbreak of disease is reported in archeologists, military personnel, outdoor construction employees, and agricultural workers. The patients who are suffering with diabetes mellitus, AIDS/HIV, Hodgkin's disease, cancer or receiving long term corticosteroid therapy, or undergoing organ transplantation or having advanced pregnancy have more risk of getting disseminated coccidioidomycosis [2,3].

The first case of human coccidioidomycosis was described in a 36- year- old soldier by Alejandro Posadas in 1892 from Bunenos Aires, Argentina. Coccidioidomycosis is reported from USA, Argentina, and Mexico. The imported cases of coccidioidomycosis are also recorded from other countries. The disease is caused by *Coccidioides* immitis and *C. posadasii,* which are geophilic dimorphic fungi. The former species appears to be restricted to California, USA whereas the later one may occur outside California [3]. These fungi can be used as potential agents of bioterrorism. The etiologic agents of coccidioidomycosis occur in mold (mycelial) stage in the soil, and in yeast phase in host tissue. The mold form in the soil produces a large number of arthtrospores/ arthoconidia that act as source of infection to humans as well as animals. The natural habitat of *Coccidioides* immitis was not known until 1932 when Stewart and Meyer first recovered the fungus from the soil in the San Joaquin Valley of California, USA. A plethora of factors, such as sandy and alkaline soil, hot and dry season, an annual rainfall of 5 to 20 inches, an arid or semi-arid climate, and high concentration of calcium, magnesium, sodium chloride, and sulphate favor the growth of *Coccidioides* species in the soil [2].

The respiratory tract is recognized as the principal portal of entry of the fungi. The source of infection is exogenous. The arthroconidia, which are disseminated by air current in the environment, are inhaled by humans and animals during digging or playing at the sites where the fungi occur in the soil. Infection can also be contracted by direct inoculation of the fungus through the skin. Person to person transmission may occur very rarely when the patient is looked after by the nursing staff in the hospital [1]. Airborne arthroconidia may constitute a serious hazard to the laboratory worker who can acquire accidental infection. Pathologist who performs postmortem examination may also get infection through accidental inoculation of the fungus. Transmission of infection can also occur through fomites. The individual with pulmonary coccidioidomycosis develops the disseminated form involving meninges, visceral organs, bones, subcutaneuous tissues, and skin [2].

The natural infection is reported in many species of animals, such as alpaca, armadillo, baboon, badger, bear, cat, cattle, coyote, chinchilla, deer, dog, dolphin, ferret, gorilla, horse, kangaroo, rat, lion, llama, monkey, mouse, rhinoceros, pig, sheep, squirrel, tiger, and zebra [1,2]. It is now well established that the soil of desert regions serve as an ecological niche for *Coccidioides* species.

The disease is endemic in certain desert areas of Arizona, California, Texas, and New Mexico. It is estimated that approximately 150, 000 cases of coccidioidomycosis occur annually in USA [3]. In hyper endemic regions of the United States, about one-fifth of the persons become sick and therefore, seeks medical help [2]. There is a dramatic upsurge in the incidence of coccidioidomycosis in endemic regions. The last decade has shown the significant increase in the incidence of pulmonary coccidioidomycosis in the United States. It is a common cause of community acquired pneumonia in endemic areas. Pulmonary coccidioidomycosis is described in healthy Japanese individuals. Coccidioidomycosis is a saprozoonosis, which is encountered in immunocompetent as well as immunocompromised subjects.

In humans, clinical manifestations of coccidioidomycosis are varied, which include fever, cough, sputum, chest pain, dyspnoea, haemoptysis, brocopneumonia, pleurisy, pneumonia, empyema, pneumothorax, malaise, anorexia, headache, night sweats, myalgia, generalized pain, weakness, reduced body weight, fatigue, erythema nodosum, lymphadenopathy, lymphangitis, cellulitis, synovitis, osteomyelitis, cholecystitis, meningitis, and percarditis [1-3]. In addition, the infection of prostate, liver, and tongue is also reported. Primary pulmonary infections are inapparent or produce transient flu like illness. Lung cavities are detected in 5% of the patients. Disseminated form becomes fatal, if not timely treated. The skin lesions are observed in about 15-20% of the patients with disseminated disease. The severe involvement of the lungs and central nervous system may carry about 60% mortalities in USA.

There are no specific clinical signs, which can help to make a tentative diagnosis. Radiography computed tomography of chest can reveal lesions in the lungs. Diagnosis can be confirmed by demonstration of spherules in brochioalveolar lavage (BAL), sputum, pus, and exudates by potassium hydroxide (10% solution), calcoflour stain, and Papanicolaou stain. The biopsied and autopsied tissues are examined for the fungus by histopathology with Grocott-methenamine silver (GMS), periodic acid Schiff (PAS), and haemotoxylin-eosin techniques [1,2]. The examination of blood reveals peripheral eosinophilia. The isolation of the causative agent from biologic specimens should be attempted only in a well-equipped reference laboratory as the fungus is highly virulent and airborne. The detailed morphology of isolates recovered from clinical materials can be easily studied in PHOL (Pal, Hasegawa, Ono, Lee) stain [4] or Narayan stain (2004) [5]. Real- time PCR and nested PCR assay can give rapid diagnosis of disease.

Cutaneous hypersensitivity test with coccidioidin, which is analogous to tuberculin, is very useful in epidemiological investigation. The test becomes positive within 21 days after the onset of pulmonary symptoms. The coccidioidin (0.1ml) is injected intradermally and an induration of 5 mm or more is considered as positive [1]. Coccidioidomycosis should be differentiated from blastomycosis, histoplasmosis, paracoccidiodomycosis, and tuberculosis. It is advised that patient presented to the hospital with pneumonia following recent travel to endemic area should be meticulously investigated for coccidioidomycosis.

The most of the patients of coccidioidomycosis hardly require any therapy as they recover without chemotherapeutic agents. Patients, who are either immunocompromised or suffering with disseminated infection, should be treated with antifungal drugs. The duration of treatment is based on the severity of infection, and immune status of the patient. A number of antifungal drugs, such as liposomal amphotericin B, fluconazole, itraconazole, ketoconazole, posaconazole, and voriconazloe are used for the management of disease [1-3]. The combination therapy with caspofungin and fluconazole is suggested in patients who are unresponsive to amphotericin B or suffering with diffuse pulmonary coccodioidomycosis [3]. It is imperative to perform surgical debridement of subcutaneous abscesses. It is advised that drug susceptibility of mould form of *Coccidioides* should not be performed as the fungus is highly infectious.

Currently, no vaccine is available to immunize the high risk groups. Therefore, the prevention and control of disease depends on dust control methods (oiling airstrips, seeding lawns, planting vegetation, paving roads), increasing awareness among the physicians and travellers in endemic regions about the disease, use of face mask while working in laboratory with mold culture, prompt medical attention to skin injury, and avoidance of visit by pregnant women and immunocompromised patients to endemic areas. It is pertinent to mention that patient with primary pulmonary should be immediately treated with antifungal drugs to prevent the dissemination of infection to other organs [1,2].

There is a need to undertake further research work on the development of safe, potent, and low cost vaccine for the prevention of coccidioidomycosis.

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Conflict of Interest

None

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