ISSN: 2831-3887

Research Article

Journal of Veterinary Health Science

Animal Aspergillosis; Diagnostic Aspects and the One Health Perspectives

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Submitted: 2026, Feb 10; **Accepted:** 2026, Mar 16; **Published:** 2026, Mar 27

Citation: Denamo, A. N., Kalacho, N. D. (2026). Animal Aspergillosis; Diagnostic Aspects and the One Health Perspectives. *J Vet Heal Sci, 7*(1), 01-08.

List of Abbreviations

ABPA	- Allergic broncho-pulmonary aspergillosis
BAL	- Broncho alveolar lavage
BHI	- Brain heart infusion
BMA	- Bovine mycotic abortion
CAP	- Chronic pulmonary aspergillosis
CT	- Computerized tomography
DCA	- Disseminated canine aspergillosis
ELISA	- Enzyme linked immune sorbent assay
GP	- Guttural poach
IA	- Invasive aspergillosis
IPA	- Invasive pulmonary aspergillosis
ITS	-Internal transcribed spacer
MRI	- Magnetic resonance imaging
PCR	- Polymerase chain reaction
PDA	- Potato-dextrose agar
SDA	- Sabouroud –dextrose agar
TNF	- ά- Tumor necrosis factor –Alpha

Abstract

Aspergillosis is a respiratory disease of mammals, humans, chicken and turkey and less frequently ducks, pigeon, geese and other wild and domestic birds. It is caused by a fungal species under the genus aspergillus. The genus Aspergillus is composed of more than 300 species, a fraction of which are involved in animal or human infections mostly following environmental exposure. Aspergillus fumigatus is frequently isolated in affected animals and humans and may lead to a variety of allergic reactions and life-threatening systemic infections in Animals and humans. Aspergillus species are ubiquitous and the disease is found where ever environmental condition is favorable for growth. Various risk factors (i.e., immunosuppression, tuberculosis) have been recognized for human whereas for veterinary infections, unhygienic management, trauma, anatomical conformation of the skull or suspected immunological deficiencies have been suggested. Infection occurs by inhalation of spores. Diagnosis is based on the clinical manifestation, supported and confirmed by laboratory means, involving the classical approach of demonstrating the etiological agent in the clinical specimens and in culture. Non-cultural methods, such as antigen detection and/or molecular assays to detect fungal nucleic acids or protein profiles, are used as well. Treatment of aspergillosis is not effective and prevention is the best way of controlling the disease. Good managemental practices such as sanitation, avoiding wet litter or soil and

moldy or dusty feeds, providing adequate ventilation, and disinfecting feed and water lines should be implemented to prevent and control the disease. Therefore, this review provides an update on the evolving epidemiology and risk factors of aspergillosis in animals as well as its public health significance.

Keywords: Aspergillosis, Animal, Diagnosis, Human, Prevention, Zoonoses

1. Introduction

Zoonoses are infections that can be naturally transmitted between vertebrate animals and humans [1]. From global prospective, zoonotic infections have been recognized for many centuries, and account for the majority of emerging and reemerging infectious diseases, worldwide [2]. The increase in fungal diseases challenges healthcare providers globally, with more than a billion people still falling ill with fungal infections and over 1.6 million related deaths each year [3]. Aspergillosis is a fungal disease caused by Aspergillus, which can affect humans, dogs, cats, horses, marine mammals, wild and domestic birds and even invertebrates, such as bees and corals. Aspergillus species are widespread in the environment, growing on plants, decaying organic matter, and in soils, air/bio-aerosols, in/on animal systems and in freshwater and marine habitats [4,5].

The most commonly implicated pathogens in aspergillosis are representative of the following species complexes: *Aspergillus fumigatus, Aspergillus favus, Aspergillus terreus*, and *Aspergillus niger*. The most frequently reported agent accounting for 60 to 90% of all infections is *A. fumigatus*. Non-fumigatus species, on the other hand, are also now being identified in a wide range of cases led by *A. favus* [6]. Aspergillosis includes various diseases caused by Aspergillus species dependent upon the host immunological responses, ranging from non-invasive allergic illness to chronic and invasive lung infection [7].

Clinical syndromes are dependent on the immunological status of the host; immunodeficient people are more likely to experience invasive syndromes. An infection can spread locally or to distant locations after injection or inhalation, depending on the immunological status of the patient [8]. Inhaling environmental conidia is a typical way for animals to become infected, and the respiratory system is the most prevalent anatomical site for the initial site of infection [9]. Aspergillus is a common filamentous fungus that mainly infects people with underlying respiratory diseases and immunocompromised hosts. Aspergillus species in the environment use dead material as a source of nutrients and reproduce asexually through conidia [10].

In animals, Aspergillosis is primarily a respiratory infection that may become generalized; however, tissue predilection is variable between species. Similar to infections in humans, animals exhibiting inability to produce a normal immune response are at higher risk of infection. Aspergillosis may also occur in healthy animals under environmental stress and other immune-compromising conditions [11].

Human aspergillosis is a multifaceted disease, including ear, sinus, eye, skin, lung, or disseminated infection [12]. The pulmonary tract is the major target system for *Aspergillus spp*. and pulmonary involvement is represented in several clinical entities: the allergic broncho-pulmonary aspergillosis (ABPA), aspergilloma, chronic pulmonary aspergillosis (CPA), and invasive pulmonary aspergillosis (IPA), leading possibly to disseminated invasive aspergillosis (IA). According to the Global Action Fund for Fungal Infections, it is estimated that the health of more than 15 million people is affected by aspergillosis, causing more than 1 million deaths per year. As fungal infections increase in clinical medicine, the number of cases of aspergillosis is likewise increasing through the past decades [13].

The diagnosis of aspergillosis with a combination of microbiological and histopathology findings, host factors, and clinical and radiological evidence is needed to obtain rapid and accurate diagnosis. Despite significant advances in aspergillosis diagnosis and treatment, severe fungal diseases continue to occur and are not easy to treat [14]. The isolation and identification of the fungus allows the determination of its susceptibility to antifungal drugs. Thus, antifungal susceptibility testing maybe considered as part of the diagnostic process, which is of relevance for management of the infection [15]. In closing, the management of aspergillosis should take into account the susceptibility of Aspergillus species to antifungals or antifungal combinations. Thus, the objectives of this review paper are to give an update on general animal aspergillosis its zoonotic implications and to indicate the diagnostic approaches, control and prevention in animals and humans.

2. General Overview of Aspergilosis

2.1. Taxonomic Classification and Structure of Aspergillosis

The genus aspergillus is classified in the family *aspergillaceae*, order Eurotiale [16]. Aspergillus is ascomycetes and is classified in the form subdivision Deuteromycotina, because most species do not have a sexual reproductive cycle. Most of the species under the genus Aspergillus are classified as fungi imperfecti (asexual reproduction) but the perfect state has been found in *Aspergillus nidulans* that can be reproduced by ascospores. When grow start hyphae, together forming mycelia. Vegetative mycelium consists of superficial hyphae and hyphae on the surface are aerial mycelia. This structure produce conidiophore, these are formed in foot cells and end in a vesicle, in this stricter grow one line of phyalides or one line of mutulae and over this stricter one line of phyalides that produce a chain of conidia or spores. Conidia are uni or multi nucleated but always single celled [17].

Kingdom	Fungi
Division	Ascomycota
Class	Eurotiomycetes
Order	Eurotiales
Family	Trichocomaceae
Genus	Aspergillus

Table 1: Taxonomy and classification of Aspergillus

2.2. Etiology

Aspergillosis cuased by a fungal species under the genus Aspergillus. The most common species of Aspergillus causing invasive disease include *A. fumigatus* (90% in some series), *Aspergillus flavus*, *Aspergillus niger*, *Aspergillus terreus*, and Aspergillus nidulans, and the most common allergens include *A. fumigatus* and *Aspergillus clavatusm* [18]. These organisms are common soil saprophytes which grow on organic matter in warm (>25°C) and also humid environment [19].

2.3. Epidemiology

Aspergillus species are widespread in the environment and are commonly isolated from both the outdoor environment (i.e. soil, plant debris) and indoor environment, including hospitals [20]. Aspergillus spp.is ubiquitous and the disease has a worldwide distribution and may be found wherever environmental condition is favorable for fungal growth. These organisms are common soil saprophytes and grow on organic matter in warm (>25°C) humid environments including poor ventilation system [21]. Globally, the prevalence of aspergillosis is increasing as a result of developing advanced medical practices with a rise in the proportion of immunocompromised populations due to cancer treatment, organ transplantation, and prolonged immunosuppressive therapy. A ten-fold increase in the frequency of invasive aspergillosis (IA) was seen over the last two decades [3].

Aspergillus spores are frequently found in the air, water, soil, rotting vegetation, plant debris, manure, sawdust litter, bagasse litter, animal feed, and indoor air environments. According to, Aspergillosis is prevalent throughout the year, but in hot, humid weather, the percentage of positive isolates in lung and environmental samples was higher in the summer (50% and 41.2%, respectively), followed by the winter season (29.2% and 29.4%, respectively) when compared to other seasons of the year [19]. These are caused by the warm, humid climate and the fact that indoor gas levels are often at their peak during the winter.

Environmental factors play an important role in the development of the disease include the number of spores to which the bird exposed, poor sanitation in the house as well as food contaminated with faces promote for fungal growth. *Aspergillus fumigatus* can withstand and survive in a wide range of pH and temperature and its hydrophobic cell wall allows this species to be efficiently dispersed by even slight air currents. Similarly, a number of features allow this species to be the most predominant mould species causing infections in humans [22].

Animals who are immunocompromised such as patients undergoing hematopoietic stem cell transplantation, chemotherapy for leukaemia are at an increased risk for invasive aspergillosis infections. These animals may have neutropenia or corticoid-induced immunosuppression as a result of medical treatments. Neutropenia is often caused by extremely cytotoxic medications such as cyclophosphamide. Cyclophosphamide interferes with cellular replication including that of white blood cells such as neutrophils. A decreased neutrophil count inhibits the ability of the body to mount immune responses against pathogens. Although tumor necrosis factor alpha (TNF-α) a signaling molecule related to acute inflammation responses is produced, the abnormally low number of neutrophils present in neutropenic patients leads to a depressed inflammatory response [23].

2.4. Pathogenesis

Aspergillosis is caused by inhalation of overwhelming numbers of small, hydrophobic fungal spores (conidia) into the respiratory tract. After infective spores invade tracheal, nasal, bronchial and air sac epithelium, they penetrate the respiratory tissue and reproduce by single division of tubular hyphae to form mycelia and they initiate granulomas at this site. Then they are disseminated hematogeneously to the other tissue like brain, pericardium, bone marrow, kidney and other soft tissue [22]. Small conidia size allows penetration to the lower respiratory tract system and escaping clearance by mucociliary forces, presence of melanin in the cell wall enables withstanding reactive oxygen species and phagocytosis, and abundance of negatively charged sialic acid on the surface permits. *A. fumigatus* to effectively bind to the basal lamina proteins once inside the host lung. Although conidia can be easily cleared by counteracting host mechanisms in the lung [24].

Alveolar macrophages are the first line of defence against inhaled Aspergillus conidia. In the lungs, pathogen recognition receptors, such as Toll-like receptors, dectin-1 and mannose-binding lectin, identify specific fungal wall components and produce cytokines that stimulate neutrophil recruitment, the main defence mechanism against Aspergillus hyphae [25]. The primary location of lesions is the lungs and air sacs although other organs may be involved. Extensive involvement of the respiratory tract can occur before development of clinical signs. Lesions vary in size from pinhead or miller seed (milliary 2 cm) may also be observed in serosa and parenchyma of the other organs involved [26].

According to, the Pulmonary lesions are characterized by multiple hard creams to yellow colored, circumscribe plaques a few mm to several cm in diameter seen throughout the lungs surface, inside the lungs, scattered in ventral surface of sternum and air passages on gross examination [27]. *A. fumigatus* can cause a wide range of infections in both immunocompromised and immunocompetent individuals including an estimated annual number of 16 million pulmonary-infections with fatal outcomes in many hundred thousand patients annually [28-30]. Tissue invasion creates an inflammatory condition and inflammatory response with hetrophils, lymphocytes, monocytes and some giant cells infiltrating the lesion and produce lesion [31].

2.5. Clinical Signs

Clinical signs are usually non-specific (lethargy, inappetance, and anorexia) or can be related to compromise of the respiratory system (rhinitis, change in vocalization, dyspnea). A rapidly invasive *Aspergillus* infection in the lungs often causes cough, fever, chest pain, and difficulty breathing. Poorly controlled aspergillosis can disseminate through the blood to cause widespread organ damage [32]. Symptoms include fever, chills, shock, delirium, seizures, and blood clots. Aspergillosis of the ear canal causes itching and occasionally pain. Fluid draining overnight from the ear may leave a stain on the pillow. Aspergillosis of the sinuses causes a feeling of congestion and sometimes pain or discharge [33].

In canines, clinical signs include sneezing, epistaxis, unilateral or bilateral mucopurulent discharge, ulceration of the nasal planum, and facial pain and muscle wasting with the more chronic cases. Pathology associated with this disease is concentrated in the region of the nasal cavity and paranasal sinuses. In some cases where the cribriform plate is invaded, central nervous system infections can occur. In certain canine species, disseminated aspergillosis has been reported and the kidney is one of the target organs [34]. Cows typically abort in their second or third trimester of pregnancy. Placentitis is the hallmark pathologic finding where the placenta has a leathery appearance that is almost pathognemonic; the intercodylendary tissue is thickened and the cotyledons are hypertrophied and have a rough and irregular surface [35]. In avian species, clinically, the first signs include breathing difficulties and wheezing due to the obstruction of airways by fungal granulomata. Subsequently general symptoms such as lethargy, inappetence, diarrhea, and feather ruffling may appear. In other cases, wasting may be the only symptom Pathological changes include granulomata in the lungs and fungal plaques in the air-sacs are [36].

3. Aspargilosis In Different Animal Species

3.1. Avian Aspergillosis

Both domestic and wild birds, as well as those kept in captivity, can contract avian aspergillosis. Although some species, like penguins, appear to be overrepresented, there is no concrete proof that they are more vulnerable [37]. According to, young birds appear to be more vulnerable to acute aspergillosis. Birds have rather high body temperatures, often exceeding 40°C, thus fungi that infect them must be able to flourish there [38]. The primary etiological agents implicated are Aspergillus spp. in general and A. fumigatus in particular [35]. Tests for individual birds can involve radiography, CT, or MRI scans [39] and endoscopy to identify occlusions in birds with straight tracheae (this method is not feasible if this organ is convoluted, as in certain crane species).

Additionally, endoscopy enables the etiological agent to be cultured and lesions to be sampled. Fumigaclavin, a fungal toxin, has been proposed as a sign of avian aspergillosis, but more research is needed to determine the circumstances under which it can be found [1]. Aberrant clinical pathological test results are mostly the function of the organ involved. Other methods include protein electrophoresis on cellulose acetate or agarose gel film. A decrease in albumin or serum proteins was found to be negative prognostic indicators in penguins [40]. Biopsies may reveal fungal hyphae whereas immunohistochemistry may indicate whether the infecting fungus belongs to the genus. assessed the specificity of these tests by using polyclonal anti-Aspergillus rabbit antibodies on 50 paraffin-embedded samples [41].

3.2. Canine Aspergillosis

Disseminated canine aspergillosis (DCA) affects primarily German shepherd breed dogs, with females being overrepresented [42]. It is caused most frequently by *Aspergillus terreus* or, more rarely by two other species belonging to the Terrei group, namely *A. carneus* and *A. alabamensis*. Other Aspergillus spp. that may cause disseminated canine mycoses include A. deflectus, *A. fumigatus*, *A. niger*, *A. flavus*, *A. flavipes, A. versicolor*, or unspecified Aspergillus spp. Clinical signs are non-specific and include lethargy, weight loss, central nervous system signs, and ataxia due to musculoskeletal lesions [43].

For some Aspergillus spp., especially *A. terreus*, the presence of accessory conidia (aleurioconidia) in tissue and/or culture is an important indication as to the identity of the infection's etiology [44]. In addition, a commercial kit (Platelia Aspergillus, BioRad, United States), aimed at the detection of Aspergillus galactomannan in serum or bronchoalveolar lavage, by an immunoenzymatic sandwich microplate assay has been assessed in dogs [45].

3.3. Guttural Pouch Mycosis

The guttural pouch (GP) is a diverticulum of the Eustachian tubes in one toed ungulates (Peryssodactila), hyraxes, some bats, and the American forest mouse. Among these, horses are the only domestic animals that have this organ. The GPs are in contact with some major arteries and nerves which may be affected when the walls of the GP are eroded by the fungi [46]. This may result in potentially fatal epistaxis (while the animal is at rest) and/or paralysis of various cranial nerves leading, among others, to dysphagia [47]. Microorganisms, including fungi, have been found in healthy GPs and thus predisposing factors are necessary to allow the evolution of an infection. Although several hypotheses were suggested as to the nature of such factors, they thus far have remained unidentified. Fungi involved are primarily A. fumigatus and A. (Emericella) nidulans but other Aspergillus species have been reported [47].

3.4. Mycotic Abortion

Bovine mycotic abortions (BMAs) occur mostly in the third trimester of pregnancy and are mostly sporadic although they are an important cause of mycotic abortions in some countries. Symptoms of BMA include modifications to the placenta that becomes thick and "leathery." Cotyledons are thickened and may have necrotic centers. This may also occur in abortions caused by Brucella spp. and, considering the significant zoonotic potential of this microorganism; appropriate precautions should be taken until the abortion's etiology is ascertained. In addition, raised, hyperkeratotic plaques may be present on the fetus' skin although the frequency of this symptom is uncertain [48].

The pathogenesis of mycotic abortions in ruminants has significant implication for the interpretation of the diagnostic results. The contact between the fetal and the maternal part of the ruminant placenta is special mainly in two characteristics: (a) it is not contiguous but occurs in contact organs called placentomes, composed of the fetal part the cotyledon and the maternal part the caruncle and (b) there is no direct contact between the fetal and the maternal blood vessels [49].

4. Public Health Significance of Aspergillosis

Aspergillosis is considered a zoonotic disease as it can be transmitted from animals to humans and humans to animals. It also affects the animals particularly birds, but it is more common in domesticated animals like dogs and horses. In humans, it possesses high risk for development in those individuals having weak immune systems such as those undergoing chemotherapy, transplant recipients or those infected with HIV/AIDS. Workers in the industries like agriculture, construction, healthcare, food processing have chances of Aspergillus spores inhalation while working with contaminated material such as hay, grain or compost or may be while cleaning or maintaining contaminated environments [50]. The most clinically relevant Aspergillus species is *A. fumigatus*, followed by *A. flavus, A. terreus* and *A. niger*.

Noninvasive infections in immunocompetent patients (e.g. with cystic fibrosis or post-tuberculosis) are allergic sinusitis or allergic bronchopulmonary aspergillosis (ABPA), fungal balls in the sinus or lung, chronic pulmonary aspergillosis, otitis externa or onychomycosis. The zoonotic fungi can be directly transmitted from animals to humans. However, the endemic infections with indirect transmission from the environment and the zoophilic fungal pathogens with near-direct trans mission. Aspergillosis is transmitted to man by handling infected birds and animals, inhalation of spores from infected feed and litter, poor sanitation and poor hygienic condition and by eating under cooked contaminated animal product [51]. Most mycotoxins produced by aspergillus are not broken down by cooking temperature and there is no safe way to salvage grain or food that has been molded. It is transmitted via inhalation or ingestion [21].

Three disease states are observed in man and these are: first; Infection that can arise from weakening effect of aspergillosis, for example colonization of the lung cavities due to tuberculosis, neoplasm or new growth in the lung or kidney almost in organ system in human body may be involved. Onychomycosis, sinusitis, cerebral aspergillosis, pulmonary aspergillosis, cutaneous aspergillosis, hepatic aspergillosis as well as disseminated aspergillosis may develop. Nosocomial occurrence of aspergillosis due to catheters and other devices is also likely to occur in hospital environment and is a major risk for the development of Aspergillosis particularly neuropenic patients. Second; Allergic reaction to *aspergillus* spp, for example allergic broncho-pulmonary aspergillosis (ABPA). Third; toxic reactions occur as result of toxin produced by Aspergillus spp. Aflatoxins which are carcinogenic, induce hepatocellular carcinoma or liver cancer [52].

5. Diagnostic Approaches of Aspergilosis

An accurate diagnosis of animal fungal infections in general and aspergillosis in particular is of significant importance to determine the prognosis, remedial steps to be taken, and the choice of therapy, especially considering the differences in susceptibility of the various species of the fungus [36]. Precise species identification is crucial because of variations in the antifungal susceptibility profiles and of differences in the clinical presentations produced by the different specie [3]. The primary identification of the fungus may be based on morphology (this may necessitate the use of specific media such as Czapek-Dox agar). Due to differences in the prognosis and susceptibility of morphologically undistinguishable fungi, the exact identification of the etiology is of importance.

It may require the sequencing of the Internal Transcribed Spacer gene and often additional genes such as those of tubulin and/or calmodulin [53]. Serological methods were tested in animals in a limited number of cases and do not allow obtaining unequivocal conclusions as to the mycoses' nature [43]. On microscopy, Aspergillus species are reliably demonstrated by silver stains such as Gridley stain or Gomorimethenamine-silver. These give the fungal walls a gray-black colour. The hyphae of Aspergillus species range in diameter from 2.5 to 4.5 µm. They have septate hyphae, but these are not always apparent, and in such cases they may be mistaken for Zygomycota. Aspergillus hyphae tend to have dichotomous branching that is progressive and primarily at acute angles of around 45° [54].

Current laboratory diagnosis of aspergillosis employs also non-cultural methods, such as immunological assays, including detection of anti-Aspergillus antibodies and the Aspergillus antigen-galactomannan (the Platelia test). Additional non-cultural tests include detection of Aspergillus nucleic acids in patients' blood or other clinical samples, by using PCR technology.

Histopathology and the use of fungal-specific stains play an important role in the diagnosis of infections by Aspergillus species. The identification of fungal elements in histological sections and /or fresh clinical specimens in bronchoalveolar lavages (BALs) and biopsies (e.g., lung) results in the proof of an infection [55]. Microscopy does not support to definitively distinguish Aspergillus from other filamentous fungi, but the micromorphology may provide information on the fungal class. Aspergillus usually shows

typically dichotomous and septate hyphae, whereas Mucorales present with pauci-septate and 90◦C angle branching hyphae. The distinction between septate (e.g., Aspergillus) and non-septate hyphae (e.g., Mucorales) is important as it may affect the choice of antifungal treatment [56].

Culture from a clinical sample is the gold standard for diagnosis in general and has the advantage of yielding the specific etiological agent and allows antifungal susceptibility testing. However, a significant limitation is the fact that culturing may take several days to achieve a positive result. Deep site samples should be cultured on Sabouraud-dextrose-agar (SDA), brain-heart-infusion-agar (BHI), or potato-dextrose-agar (PDA) at 30◦C and 37◦C for 72 h. Supplementation of gentamicin plus chloramphenicol is recommended for non-sterile specimens such as sputum. Quantitative cultures may not be discriminative for infection or colonization due to Aspergillus species. Antigen and PCR tests are add on assays to culture and microscopy, sensitivity is dependent on the type of disease, the usage of antifungal drugs and the timing of testing relative to the disease process. The commercially available sandwich ELISA (Platelia Aspergillus, BioRad) has been validated for serum and BALs with a limit of detection of ~1 ng/ml [57].

Post mortem diagnosis is based on the presence of hyphae in the organ lesions, their isolation, and identification. It should be stressed that other molds cannot be differentiated from Aspergillus spp. at the histopathological examination unless immunohistochemistry is applied [58]. Moreover, to accurately identify the mold species involved, such as those belonging to the section Terrei, sequencing the ITS gene alone may be insufficient and additional genes such as that encoding for tubulin should be sequenced [59].

6. Treatment, Control and Prevention of Aspergilosis

6.1. Treatment

Aspergillosis has no effective treatment and prevention by vaccination is not commercially practicable but, Therapy of pulmonary aspergillosis is based on the use of voriconazole as a drug of choice. Posaconazole, isavuconazole, liposomal amphotericin B, or amphotericin are used as well. For allergic forms of aspergillosis such as ABPA or allergic Aspergillus sinusitis, the recommended treatment is itraconazole. Corticosteroids may also be helpful. The current medical treatments for aspergillosis include voriconazole and liposomal amphotericin B in combination with surgical debridement [60]. For the less aggressive allergic broncho-6+pulmonary aspergillosis, findings suggest the use of oral steroids for a prolonged period of time, preferably for 6–9 months in allergic aspergillosis of the lungs.

Itraconazole is given with the steroids, as it is considered to have a "steroid-sparing" effect, causing the steroids to be more effective, allowing a lower dose. Other drugs used, such as amphotericin B, caspofungin (in combination therapy only), flucytosine (in combination therapy only), or itraconazole, are used to treat this fungal infection. However, a growing proportion of infections are resistant to the triazoles. *A. fumigatus*, the most commonly infecting species, is intrinsically resistant to fluconazole [61]. Although antifungals have been proven to be effective in terms of ability to reduce the fungal burden, their clinical effectiveness is regrettably affected by the emergence of fungal resistance, resulting in a serious public health crisis worldwide [3].

6.2. Control and Prevention

Control and prevention depends on reducing exposure to the fungus and associated risk factors. *Aspergillus fumigatus* in young animals has been somewhat controlled by sanitation. Moldy housing or feed should be avoided to prevent outbreak of aspergillosis. It is advisable to treat housing and environment with antifungal compounds. Any moldy feed should be removed, bulk feed container should be cleaned; equipment should be cleaned, disinfected and well monitored in poultry old litter should be removed from house and replaced with new [62]. Improved diagnostic methods and economically sustainable therapeutic options could significantly improve the prognosis of animal aspergillosis. In addition, results of imaging techniques have also been found to be often unreliable and should be further investigated. A special case of future research should try to define the immunologic basis of canine disseminated aspergillosis. This would permit the exclusion of dogs carrying the faulty gene to be excluded from the pedigree lists (as has been done for hip dysplasia in GS dogs) and reduce the number of cases [36].

7. Conclusion and Recommendations

Aspergillosis is a spectrum of infections caused by fungi from the Aspergillus genus. The species most involved include *A. fumigatus*, *A. terreus*, *A. flavus*, and *A. niger*. The saprophytic fungus *Aspergillus fumigatus* is responsible for opportunistic infections affecting birds and mammals, including humans. The importance of fungal infections in both human and animals has increased over the last decades. Aspergillosis is a disease of respiratory system of chicken, humans, mammals and wild birds. Various risk factors (i.e., immunosuppression, tuberculosis) have been recognized for human whereas for veterinary infections, unhygienic management, trauma and suspected immunological deficiencies have been suggested. Clinical syndromes depend on the host's immune status, with invasive syndromes predominantly affecting immunodeficient individuals. Based on the above conclusion the following recommendation forwarded.

- Taking maximum attention in all aspect of Aspergillosis disease transmission to reach for effective treatment.
- Improve the unhygienic management and identify risk factors properly to overcome fungal infection control and prevention.
- Further advancements in this field of research as well as a growing spectrum of diseases for which it should be continuous research conducted for the applicable both preventatively and therapeutically.

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