

A Review on Aspergillosis in Poultry

Genene Girma¹, Mengestie Abebaw¹, Mebrie Zemene^{1*}, Yergashewa Mamuye¹ and Gashaw Getaneh²

¹Faculty of Veterinary Medicine, Department of Veterinary Pharmacy, University of Gondar, Gondar, Ethiopia

²Faculty of Veterinary Medicine, Department of Biomedical Science, University of Gondar, Gondar, Ethiopia

Abstract

Aspergillosis is a respiratory disease of chicken, turkey, humans and other mammals and less frequently ducks, pigeon, geese and other wild and domestic birds. It is caused by a fungal species under the genus *Aspergillus*. In chickens and turkey, the disease may be endemic on some farm. In wild birds, it appears too sporadic, frequently affecting only an individual bird. It is usually seen in birds of 7-40 days old. *Aspergillus* spp are ubiquitous and the disease is found where ever environmental condition is favorable for growth. *Aspergillus fumigatus* is frequently isolated in affected bird. Infection occurs by inhalation of spores. The disease has two forms, acute and chronic form. The acute form occurs when large amount of spores are ingested by the bird, where as, the chronic form affects birds under immune suppression. The clinical signs are non-specific and a variety of diagnostic procedures are required for the diagnosis of aspergillosis in poultry, of which culturing is very important. 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.

Keywords: Aspergillosis; *Aspergillus fumigatus*; Pevenetion

Introduction

Aspergillosis is non-contagious disease of avian. It is caused by a fungal species under the genus *Aspergillus*. The disease occurs under immune compromised situations of the host or when the bird is exposed to an overwhelming number of spores. Stress is the main predisposing factor for the development of the disease [1]. It is a common mismanagement problem in commercial and back yard poultry. The disease primarily affects lower respiratory system [2].

Aspergillus spp can penetrate egg shell and infect the embryo. The infected embryo may die or hatch with well developed lesion. If infected eggs are broken, large numbers of spores are released, which contaminate the hatchery equipment [3]. Aspergillosis can be acute or chronic form. Acute aspergillosis generally occurs in young birds and resulting in high morbidity and mortality. The chronic form is sporadic and it causes lesser mortality and generally affects older birds, especially a compromised immune system due to poor husbandry condition [2].

Poor sanitation in the house as well as food contamination promotes fungal growth. Poor ventilation in conjunction with these factor increase the possibility of invasion and infection of birds' respiratory system by air borne spore [2]. Mycotoxins are toxic secondary metabolites produced by organisms of the fungus. Aflatoxin is a type of mycotoxins produced by *Aspergillus flavus* and *Aspergillus parasiticus* and ochratoxin is produced by *Aspergillus ochraecus* that affect poultry when they consume contaminated food [4-6]. Therefore, the objectives of this paper are to highlight about poultry Aspergillosis (Table 1).

Etiology

Aspergillosis in poultry is caused by a fungal species under the genus *Aspergillus*. Organisms cultured from affected organ in decreasing frequency are: *Aspergillus fumigates*, *A. flavus*, *A. niger*, *A. glaucus* and *A. terreus*. From these spp, *A. fumigatus* is a common cause of the disease. These organisms are common soil saprophytes which grow on organic matter in warm (>25°C) and also humid environment, and damaged egg in hatchery [7,8].

Most of the species under the genus *Aspergillus* are classified as fungi imperfect (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 mycelia 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 [6].

The function of the conidia is dissemination of the fungus. *Aspergillus* is useable as a food in many different substances because of the number of enzyme that it can produce. The two main requirement of substance for fungal growth are organic matter and moisture, if both factors are present the fungus can grow in almost any substance. The shape can be globose, elliptical or oval, and the surface can be smooth or finely rough. The color of conidia determines the color of the conidial head which also determine the color of the colony [7].

Kingdom	Fungi
Division	Ascomycota
Class	Eurotiomycetes
Order	Eurotiales
Family	Trichocomaceae
Genus	<i>Aspergillus</i>

Source: [5]

Table 1: Taxonomy of *Aspergillus*.

*Corresponding author: Mebrie Zemene, Lecturer, Faculty of Veterinary Medicine, Department of Veterinary Pharmacy, University of Gondar, Gondar, Ethiopia, Tel: 251918518866; E-mail: zemenezebrie@gmail.com

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Epidemiology

Aspergillus spp are 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 damaged egg in hatcheries and poor ventilation system. The disease more frequently occurs in tropical countries like Ethiopia. Sever outbreak associated with hatchery contamination may result in up to 15% chicken mortality during the first two weeks and decrease growth rate on affected survivors [2].

Concerning the distribution of Aspergillosis among different seasons, [8] reported that the disease is prevailing throughout the year but in hot and humid weather condition, the percentage of positive isolates in both lung and environmental samples was higher in summer (50% and 41.2%) respectively, followed by winter season which was (29.2% and 29.4%) respectively as compared with other seasons of the year. These are because of the warm, humid environment and the disease in winter season is common when indoor gas levels tend to be highest.

Transmission and predisposing factors

Aspergillosis affects avian spp, other animals and humans. All birds are susceptible to aspergillosis. It is reported in domestic birds like poultry, duck, and quails as well as in wild bird [2]. Inhalation of conidia or spores from contaminated feed, fecal material, soil and contamination of egg in ovo, infect the developing embryo. Higher susceptibility of birds to aspergillosis may be attributed to anatomic and physiologic characteristics of the avian respiratory system. The small non-expanding lungs and nine air sacs constitute a primary nidus for infection because the air (or conidia) reaches the caudal air sacs before it pass through those part of the lungs in which the gas exchange takes place [9]. Higher body temperature also allows quick fungal growth. Other factors include chronic stress, unsanitary conditions, overcrowding, malnutrition, vitamin deficiencies especially vitamin A and overuse of certain medications (corticosteroids) as well as respiratory irritants (disinfectant fumes and aerosol sprays). Birds that are otherwise ill or are very young or old are also susceptible to aspergillosis [10]. The incidence of aspergillosis increased when chicken reared on saw dust than rice husk as litter and incidence was 67.74% and 32.26% respectively might be due to high moisture content of saw dust allowing more fungal growth than rice husk [11].

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. Tissue invasion creates an inflammatory condition and inflammatory response with hetrophils, lymphocytes, monocytes and some giant cells infiltrating the lesion and produce lesion [12]. Chronic disease especially in turkey often terminate in impendence of pulmonary blood flow caused by enlarging pulmonary granulomas and this cause right ventricular dilatation and ascitis. Another mortality cause for aspergillosis included exudates to become lodged in the trachea or syrinx producing acute respiratory embarrassment in chronically infected individuals [13].

Clinical sign

Aspergillosis may involve many body systems; fungal spores most commonly invade trachea, air sacs and lung. Signs depend on the number of spore that enter the body and organ system affected but can be generally reflected as disease of respiratory tract, occasionally the CNS [7]. There are three common presentation of the disease; diffuse lower respiratory tract diseases, syringeal graunloma and focal CNS graunuloma particularly in the brain [13].

Within the first 3-5 days newly hatched chicken infected in the hatchery show a very rapid and difficult breathing and start breathing with an open mouth (gaspers) due to the gradual obstruction of the air passage [14]. Survivor may become dull and stunted, show sleepiness, lack of appetite, emaciation, increased thirst, develop eye swelling or blindness and show torticollis (twisting of the neck to one side). Other birds may remain subclinically affected for some times but later slowly develop breathing problems. This is because increasing body weight put demands on the reduced functional capacity of the lung [15].

Infected poultry usually show mortality in two phases: Acute respiratory disease may cause 5-50% mortality in the first 1-3 weeks of age. Those which survive usually develop chronic disease with up to 5% mortality due to pulmonary insufficiency, acitis, blindness or neurological fungal metastasis. A common feature of dyspnea associated with aspergillosis is the lack of respiratory noise [15] (Figure 1).

Necropsy finding

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 <1 mm in diameter) white to yellowish granulomas up to the size of a pea. Roughly spherical granulomatous nodules (>2 cm) may also be observed in serosa and parenchyma of the other organs involved [16].

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 (Figures 2a and 2b). The plaques also found in the syrinx, air sacs,

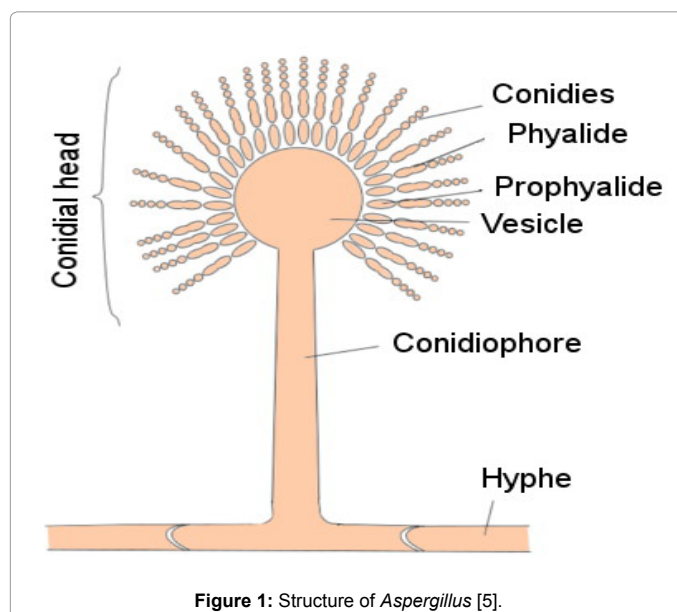


Figure 1: Structure of *Aspergillus* [5].

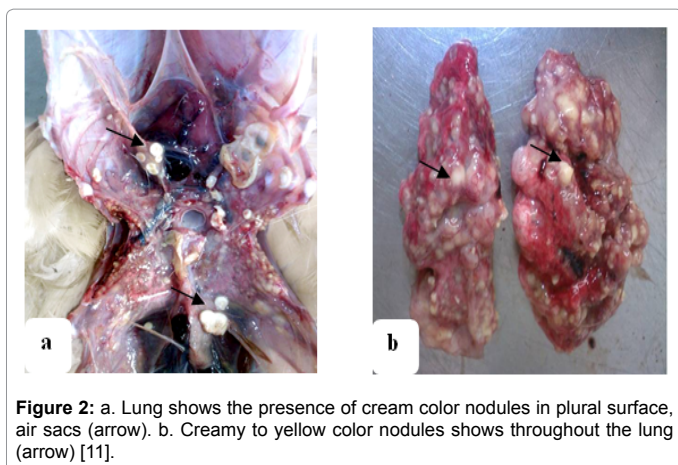


Figure 2: a. Lung shows the presence of cream color nodules in plural surface, air sacs (arrow). b. Creamy to yellow color nodules shows throughout the lung (arrow) [11].

liver and intestines. Lung parenchyma was consolidated and single or multiple necrotic areas are visible on cut surfaces of lungs [11].

Mycotic pododermatitis along with pulmonary aspergillosis was reported in turkeys. In footpads, keratinized epidermal disruption, encrustations and acute inflammation were noted [17]. Epidermal cysts associated with *A. fumigatus* have been described in the comb of a silky bantam chicken [18]. It leads to periorbital swelling, swollen and adhered eyelids with turbid discharge, cloudy cornea and cheesy yellow exudates within the conjunctival sac [19]. Osteo-arthritis and granulomatous osteoarthritis of the hip joints with necrosis of the femur head was observed in turkey [20].

Visceral organs were involved in aspergillosis with formation of nodular granulomatous lesions. Right ventricular dilatation or cor pulmonale due to pulmonary hypertension may occur with or without ascites in poultry [21]. Abscesses in the cerebellum and cerebrum were reported. It may occur with or without pulmonary and other lesions. In the cerebellum of broiler breeders and turkeys, circumscribed white to greyish areas were observed. Granuloma formation was also seen in the brain and lungs of layer chicken [22].

Histopathology

The microscopical examination showed congestion of pulmonary and perialveolar blood vessel and perivascular edema (Figure 3). The normal architecture of the lung and air sacs were replaced by disseminated granulomatous foci. The center of the granulomatous foci contained caseous necrosis and necrotic cellular debris surrounded by rims of heterophils, lymphocytes, macrophages and multinucleated giant cells was seen (Figure 3). The nodules consisted of coagulative necrotic center (Figure 4). A few, more severe, densification and inflammatory lesions were focally present on the pleura and the underlying pulmonary lobules (Figure 4) [11].

Diagnosis

Signs of aspergillosis are non-specific and making diagnosis is difficult. Moreover, no single test provides certainty. Diagnosis usually rely upon an accumulation of evidence from history, clinical presentation, postmortem findings, hematology, biochemistry, serology, radiographic changes, endoscopy, and culture of the fungus. Cases of aspergillosis in birds are often diagnosed based on postmortem findings of white caseous nodules in the lungs or air sacs of affected birds since clinical diagnosis is difficult [23]. The history of the bird can reveal a stressful event and some under lining environmental factors and immune suppressive condition or treatment. It may also reveal chronic

debilitating, voice change or exercise intolerance [24]. Granulomatous nodules and /or cheesy plaques on the serosa and parenchyma of respiratory tracts as well as other organs are observed. But, definitive diagnosis is based on the isolation of *Aspergillus* species by culture or by the detection of the organism during histological examination [25]. Identification can also be made by preparing a wet smear. For this, a nodule can be dissected out and crushed on a slide beneath a cover slip in a drop of 20% potassium hydroxide and lactophenol cotton blue. The lactophenol cotton blue stains the fungal hyphae. Wet mounts can also be prepared from sputum or nasal swabs in either 10% KOH and Calcofluor or Parker ink and/or Gram stain [26].

The tissue samples (lungs, trachea, pharynx and thoracic air sacs as well as other organs) fixed in 10% neutral buffered formalin are processed and embedded in paraffin blocks and stain with haematoxylin and eosin (HE) method. *Aspergillus hyphae* stained poorly in H and E stained sections. Differential stains such as Periodic acid-Schiff (PAS), Bauer's and Gridley's stains differentiate and easily identify the hyphae and mycelia. Special stains for fungus Grocott's and Gomori Methanamine Silver stain should be employed to detect the presence of fungal hyphae. For proper identification of the species, the pathogenic organism must be isolated by culturing it on differential media. Small pieces of lesions aseptically removed are placed onto plates or slants containing malt agar, Sabouraud's glucose agar or antibiotics and incubated at 37°C for 24 hours. Species of *Aspergillus* can be identified by observing the characteristic conidial head and colony [16].

Differential Diagnosis

The differential diagnosis includes infectious bronchitis (dyspnea), newcastle disease (watery greenish diarrhea), infectious larngeo tracheatias (gaspings, cough and extension of the neck during inspiration), dactylaria infection (nervous sign) and nutritional encephalomalacia [27].

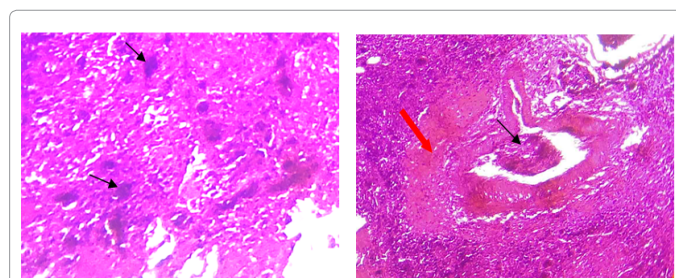


Figure 3: a. Showing congestion of pulmonary, perialveolar blood vessel and diffuse edema of pulmonary tissues (arrow) in Lung. b. Areas of caseous necrosis (black arrow) and cellular debris (red arrow) in lung [11].

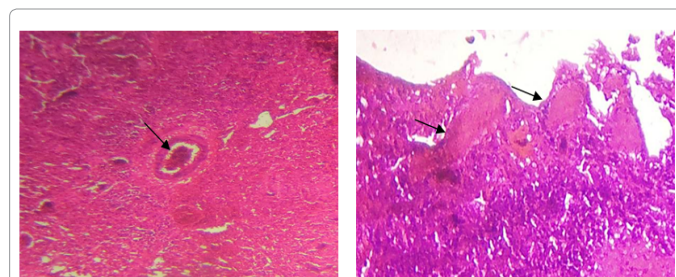


Figure 4: Aspergillosis showing granuloma formation with caseated center (arrow) in lung. b. Diffuse densification of the pleural parenchyma by congestion and an inflammatory cellular infiltration (arrows) in lung [11].

Treatment

Treatment for Aspergillosis is not effective because the drug used does not reach the fungus that is walled off by the bird's inflammatory response and therefore, isolated from the blood stream. This disease has a poor prognosis when the infection in the tissue is extensive and only systemic drugs are used. The best treatment results if the granulomatous lesion is dried and topical treatment in conjunction with systematic therapy is given. Treatment of aspergillosis involves the use of one or more systemic antifungal agent. Drugs which are commonly used include itraconazole, ketoconazole, clotrimazole, miconazole, fluconazole and Amphotericin B. From these drugs, itraconazole is a choice of treatment of the disease [11].

Prevention and control

Aspergillosis has no effective treatment and prevention by vaccination is not commercially practicable. Therefore, control depends on reducing exposure to the fungus and associated risk factors. *Aspergillus fumigatus* in young chicken has been somewhat controlled by hatchery sanitation. Moldy litter or feed should be avoided to prevent outbreak of aspergillosis. It is advisable to treat poultry house and litter with antifungal compounds [28]. Any moldy feed should be removed, bulk feed container should be cleaned, old litter should be removed from house and replaced with new. Hatching equipment and air ducts should be cleaned, disinfected and well monitored. Contaminated hatchery should be fumigated with formaldehyde or thiabendazole 120-360 g/m³ [13].

Public Health Significance of Aspergillosis

Aspergillosis has a zoonotic importance and recent outbreak of aspergillosis in chicken have left the chicken consuming population shocked and frightened. 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 poultry [29]. 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 [2].

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 neutropenic patients. Second; Allergic reaction to *Aspergillus* spp, for example allergic bronchopulmonary aspergillosis (ABPA). Third; toxic reactions occur as result of toxin produced by *Aspergillus* spp. Aflatoxins which are carcinogenic, induce hepatocellular carcinoma or liver cancer [10].

Aspergillosis establishes itself in patients debilitating by chronic disease such as diabetes, cancer, tuberculosis and disease of immune system, as well as in person treated with antibiotics, anti metabolites, and corticosteroids for prolonged period [30]. Persons occupationally exposed for long periods to materials contaminated by fungus spores (grain, hay, cotton, wool and other) run a greater risk. Normal children who inhale large number of conidia may develop fever and dyspnea. Allergic bronchopulmonary aspergillosis (ABPA) occurs in patients

with pre-existing asthma who present eosinophilia and intermittent bronchial obstruction [31-34].

Conclusion and Recommendations

Aspergillosis is a disease of respiratory system of chicken, humans, mammals and wild birds and caused by genus *Aspergillus*, which is distributed worldwide and it is soil saprophyte. It is a common mismanagement problem and causes a high mortality in chicken. 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 feces promote for fungal growth. Poor ventilation in conjunction with other factor increases the possibility of occurrence of the infection. There is no effective treatment for the disease.

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

- Cleaning and disinfection of feed and water utensils
- Poultry house must be well ventilated.
- Avoid overcrowding in poultry house.
- Avoid moldy or dusty feed.
- Proper Sanitation of hatching equipment.
- Treat poultry house and disinfect the litter with antifungal compound.
- Cull infected birds to prevent further contamination.

Use mold inhibitor in the feed for suspected outbreak.

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