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Review on Major Fungal Disease of Poultry

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Abstract: Fungal infections are common in all kinds of poultry birds but are less prevalent as compared to bacterial and viral infections. Fungal diseases cause significant economic losses to the poultry industry either due to their direct infectious nature or due to production of mycotoxin, the secondary fungal metabolites produced in grains or poultry feed. Several fungi have created widespread destruction in the poultry industry and some of them cause direct harm to human health due to their zoonotic implications. Most of the fungal diseases of poultry occur sporadically but sometimes they may occur in the form of an outbreak. They are responsible for high morbidity and mortality, especially in young birds. Mycotoxin are the leading cause of producing immune suppressions in birds, which make them prone to several bacterial and viral infections leading to huge economic losses to the poultry industry. The control of fungal disease in poultry is difficult due to lack of proper bio-security measures, intense farming and greater germ load in the farm premises. In comparison to bacterial and viral diseases, advances in diagnosis, treatment, prevention and control of fungal diseases in poultry have not taken much attention. It requires appropriate attention in terms of effective prevention and control of fungal disease.

Key words: Fungal Diseases · Poultry Industry · Birds · Fungus

INTRODUCTION

In recent years, a fungal infection have emerged as a world-wide health problem and has become an important cause of respiratory infection in poultry owing to extensive use of broad spectrum antibiotics, corticosteroids and immune-suppressive agents and increasing population of terminally ill and debilitated patients [1] which trigger an interest to examine the source and reservoir of such fungi. Among the infectious diseases fungal diseases have their own importance and seem to one of the great obstacle for the poultry farmers in the form of high morbidity, mortality and production losses [2].

Fungal infections are common in all kinds of poultry birds but are less prevalent as compared to bacterial and viral infections. Fungi are unicellular or multi cellular heterotrophic eukaryotes that derive nutrition by absorption; reproduce by asexual means, sexual means, or both; and possess cell walls [3].

Fungal diseases of poultry includes Aspergillosis, Candidiasis, Dactylariosis, Favus, Rhodotorulosis, Torulosis, Mucormycoses, Mycotoxicoses, Histoplasmosis and Cryptococcosis; Aspergillosis, Candidiasis and Mycotoxicoses are the most important and impact on poultry production. Fungi produce disease in two ways via producing pathogenic signs and lesions of disease by invading, harming and destroying body tissues of the host and by producing some toxins known as mycotoxin in food grains and feed during crop production, harvesting and storage steps, the intake, consumption and subsequent intoxication of which produce disease, immunosuppressive condition and lowers production potential [4].

Most of the fungal diseases of poultry occur sporadically but sometimes they may occur in the form of an outbreak [5]. Seasonal variation plays important role in the spread of fungal infections. Predominance of infection in closed housing during summer and the presence of fungi in the poultry litter material during autumn make the eradication difficult [6].

The fungal pathogen, mainly target the respiratory and nervous system of poultry and cause specific pathological changes in the host characterized by inflammation, lesions and sickness leading to death [7]. Propagation and dissemination occur as a result of their

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saprophyte lifestyle; infection is a dead end, with the exception of favus (Dermatophytosis), because mycoses are not contagious [8].

In native type of poultry husbandry, the control of fungal diseases in poultry is difficult due to lack of proper bio security measures, intense farming and greater germ load in the farm premises. Fungal infection requires appropriate attention in terms of timely diagnosis and effective treatment regimens to be followed. Advances in the treatment and control of bacterial and viral diseases of poultry have been outstanding in the recent years but the situation is not so good in case of fungal infections and thus is a matter of concern [4].

Major Fungal Diseases of Poultry in Ethiopia

Aspergillosis (Brooder Pneumonia): Aspergillosis has emerged as a significant poultry health concern for poultry producers and humans health officials as it causes economic losses in poultry industry. Aspergillosis is an infectious, non-contagious disease, which affects humans, mammals and mainly wild or domestic birds. It is a large spectrum of fungal diseases, which primarily affect the lungs and are caused by members of the genus Aspergillus; is ubiquitous molds in the environment and are especially common in the soil and decaying vegetation [9].

The disease mainly affects respiratory tract of birds and develops as broncho-pneumonia. It occurs in both acute and chronic forms in poultry. The clinical manifestation of acute Aspergillosis is usually observed in young birds, often with episodes of outbreaks in poultry with high morbidity and mortality, whereas chronic Aspergillosis is more frequently observed in adult birds [10, 11].

Outbreaks occur when the organism is present in sufficient quantities to establish disease or when the bird's resistance is impaired by factors such as environmental stress, immunosuppressant from concomitant diseases or inadequate nutrition [12].

Etiology: Aspergillosis is mostly caused by *A. fumigates* and other Aspergillus species or mixed infections can play a role in the disease. The reason why *A. fumigates* is the predominant species of airborne fungal infections might be that the spores are much smaller than the spores of other Aspergillus species. Chickens and poults may become infected during hatching as a result of inhaling large number of spores in heavily contaminated hatching machines or from contaminated litter, in older birds, infection is caused primarily by inhalation of spores laden dust from contaminated litter or feed or dust range area [13].

Epidemiology: Aspergillus species are common soil saprophytes, worldwide and grow on organic matter in warm (>25°C) humid environments including damaged eggs in hatcheries, poor ventilation systems, poultry litter and feed; and the fungal spores are ubiquitous in nature. Certain infectious diseases may contribute to Aspergillosis, e.g. infectious bronchitis, chronic respiratory disease, Laryngotracheitis, Newcastle *disease* and Fowl pox. It has been speculated that extremely dry air and dust can cause the infection with Aspergillus because they dry out the respiratory mucosa and protective effect of mucus is absent [14].

Host: Aspergillosis affects all avian species, animals and humans. Newly hatched turkeys, chickens and ducks are highly susceptible to infection, but disease also occurs frequently in neonates of other avian species. High mortality rates are seen in chicks and poults that inhale large numbers of spores during hatching in contaminated incubators or when placed on mold-bearing litter. In older birds, infection is caused primarily by inhalation of spore-laden dust from contaminated litter or feed or dusty range areas. Morbidity can be underestimated in finishing flocks until slaughter inspection reveals pulmonary lesions [15].

Predisposing Factor: The disease occurs typically as a result of inhalation of the ubiquitously available spores. Multiple infections in a single facility imply common exposure rather than bird-to-bird spread. Infection can be overwhelming and acute, as when a bird is exposed to a point source of heavy spore contamination, or it can occur as a result of low-level ambient exposure coupled with compromised immune function in the host. Some factors that have been implicated as causal in the development of Aspergillosis include: recent capture, change of ownership, poor ventilation, neonatal and genetic condition and birds subjected to multiple corticosteroids, exposure to respiratory irritants and lead poisoning [16].

Transmission and Source of Infection: Aspergillosis is not a transmissible disease. Infection is acquired from environmental exposure; disturbances of soil or movement of hay, compost, or litter can produce aerosols that furnish occasion for respiratory exposure to conidia.

Fresh litter contaminated with *A. fumigates* can precipitate out breaks of Aspergillosis. Aspergillosis can also be acquired *inovo* [3].

Exposure is by inhalation of spores. These often originate from infected eggs that are opened during hatch mates. Infection within the hatchery may also occur from contaminated air ducts or other equipment. After infection in the hatchery, lateral transmission after placement is not usually a significant source of new infections. Aspergillosis can also produced by inhalation of spores from contaminated feed or poultry house litter. Fungal growth in wet litter produces large number of spores that become aerosolized as this litter is dried. In such instances, new cases may continue to appear for some time after placement [9].

Pathogenesis: Infection generally occurs when the bird inhales airborne spores. The air sacs are usually considered as the primary infection sites, since inhaled air reaches the posterior thoracic and abdominal air sacs prior to contacting epithelial surfaces in the lungs [17]. The organism may then penetrate respiratory tissues, reproducing by simple division of tubular hyphae to form mycelia. Tissue invasion incites an inflammatory response, with heterophils, lymphocytes, monocytes and some giant cells infiltrating the lesion. Severity of lesions depends on chronicity of infection, organs affected and the number of spore's inhaled [18].

When there are too many spores or the bird has an impaired immune response, the innate defense mechanisms do not succeed in eliminating infection at the site of the air capillaries. This may lead to the development of loosely attached plaques, which may or may not become overgrown by connective tissue of the host. These plaques or necrotic debris in the respiratory tract can obstruct air sacs. Hyphae containing fruiting bodies can fill the lumen and may penetrate the air sacs, causing serositis and superficial necrosis in the adjacent organs [17, 19].

Besides direct extension of the infection through the air sac wall, disseminated mycosis also occurs by haematogenous spread. Macrophages in the respiratory tract ingest spores and find their way through the interstitial into the blood and lymphatic stream and thus to other organs [20].

Clinical Sign and Lesion: Acute Aspergillosis may include a variety of nonspecific clinical signs: anorexia, lethargy, ruffled feathers, respiratory signs, polydipsia, polyuria, stunting, or sudden death. In chicks, contaminated during hatching, the disease, commonly known as brooder pneumonia, is highly fatal in the first ten days of life and results in a major respiratory distress. Respiratory signs include dyspnea, gasping, hyperpnoea with panting, nonproductive coughing, wheezing, cyanosis and sometimes nasal discharge [21].

In the chronic form, dyspnea, depression, dehydration and emaciation are described. Nervous system involvement causes ataxia, tremor, opisthotonos, lateral recumbence, torticollis, seizures, convulsions, lameness and hind limb paresis. Mortality in young birds averages 5–20%, but may be as high as 50% and less than 5% in adult [22].

Typically, lesions consist of white to yellowish granulomas ranging from miliary (<1□mm in diameter) to large roughly spherical granulomatous nodules (>2□cm) involving serosae and parenchyma of one or multiple organs. Single or multiple necrotic areas are visible on cut surfaces. The primary location of lesions is the air sacs and lungs although esophagus, proventriculus, gizzard, small intestine, liver, kidney, spleen, skin, trachea, peritoneum, brain, eye, muscle, or heart may be involved [19].

Diagnosis: Ante-mortem Aspergillosis diagnosis can be difficult, principally in chronic cases. The clinical signs are nonspecific and fungus mycelia are generally intimately associated with the tissues, making them rarely visible in exudates or body fluids. Moreover, no single test provides certainty. Diagnosis usually relies up on an accumulation of evidence from the history, clinical presentation, hematology and biochemistry, serology, radiographic changes, endoscopy and culture of the fungus [20].

History: A careful clinical history can reveal the presence of poor sanitation in the environment; an immune suppressing factor, chronic weakness history, weight loss, change in voice or exercise intolerance, also species susceptibility to this disease must be considered. Aspergillosis must be suspected in cases of weak animals that do not response to or get worst with antibiotic treatment [23].

Isolation and Cultivation: Aspergillus grows rapidly on blood and sabouraud dextrose agar at room temperature. Colonies are white at first but later turn green to dark green, flat and velvety [24].

Direct Examination: Small pieces of tissues or deep scrapings are examined in 10% NaOH. Short pieces of thick, septet hophead are characteristic. The typical conidial heads are seen only in the lungs and air sacs, where there is access to oxygen [24].

Serology: Based on growth phase or hyphae-specific antigen of *Aspergillus* species [25].

Differential Diagnosis: Aspergillosis should be differentiated from Infectious bronchitis, Infectious Laryngotracheitis, Dactylariosis and Nutritional encephalomalacia [26].

Treatment: There is no effective treatment for Aspergillosis, because the drugs used do not reach the fungus that is walled off by the bird's inflammatory response and therefore isolated of the blood son in the tissues is extensive and when only systemic drugs are used. The best treatments result if the granulomatous lesions are debrided and a topic treatment, in conjunction with a systemic therapy is given. Options for the treatment of Aspergillosis are limited, the drugs used include: itraconazole, flucytosine, fluconazolionazole and amphotericin B [27].

Prevention and Control: *Aspergillus genus* is an opportunistic pathogen; therefore every attempt should be made to reduce predisposing immunosuppressive factors such as stress and malnutrition, good management of the birds is essential; to avoid inhalation of a large number of spores, birds should be housed in a well-ventilated area, with bedding changes daily. When treating other illnesses, the benefits of long term or repeated antibiotic usage, or the use of immunosuppressive doses of corticosteroids, must be weighed against the possibility of opportunistic deep mycotic infections [28].

Spontaneous recovery from pulmonary Aspergillosis can occur if re-exposure to the mold is prevented. Strict adherence to sanitation procedures in the hatchery minimizes early outbreaks. Grossly contaminated or cracked eggs should not be set for incubation, because they enable bacterial and fungal growth and may explode and disseminate spores throughout the hatching machine. Contaminated hatchers should be thoroughly cleaned and fumigated with formaldehyde. Avoiding moldy litter or ranges serves to prevent outbreaks in older birds. Cleaned pens should be sprayed or fumigated with enilconazole following label directions and all equipment should be cleaned and disinfected [29].

Candidiasis: It is also called moniliasis, thrush or sour crop**.** Candidiasis is an occasional opportunistic fungal disease of importance in poultry. It has also been reported to be a disease or an intestinal infection in numerous species of wild birds that are being raised in captivity. Candidiasis is the most common opportunistic endogenous mycosis; in that perturbance of the micro flora or other debilitation of the host, rather than exposure to an external source and it is initiator of pathologic infection. Candida affects digestive tract and cause infection of the mouth, esophagus (Food pipe), or crop. Off these, the more common is infection of the crop and is called 'Crop mycosis' [30].

Etiology: Candidiasis is a fungal disease caused by yeasts of the genus Candida having nearly 200 species. Among them, six are most frequently isolated [31].

Epidemiology: Candida species occur worldwide on plant materials and as commensalisms in the digestive and urogenital tracts of animals and humans. *Candida albicans* isolated from environmental source less frequent than other Candida species suggested adaptation towards a parasitic rather than saprophytic existence. *Candida albicans* is also normal in habitats of upper respiratory mucosa and primarily associated with infection of skin and mucus membrane; however, they can invade every organ in the body [32]. *Candida albicans* is part of the normal flora of endogenous microbial flora and infection is believed to endogenous in origin [33].

Host: Susceptible hosts include domestic poultry, water fowls and wild birds. Infections are more common in birds under 3 weeks of age. This suggests acquired or age resistance. Mortality directly due to Candidiasis is low to nil and most of the symptoms are due to other concurrent diseases or reduced feed intake [34].

Predisposing Factor: One of the most predisposing factors is prolonged antibiotic administration which suppresses normal bacterial flora and competition for nutrients, thus allowing Candida to proliferate. Other risk factors include highly contaminated drinkers or feeders, eating litters, concurrent immunosuppressant, environmental stress or nutritional disease [15].

Source and Route of Transmission: Most infections have an endogenous source; infection can be spread through contact, with oral secretions, skin and dropping of sick birds and carriers. An exogenous infection probably occurred due to indirect contact between affected and carrier birds. Candidiasis also transmitted through contaminated water recirculation systems, hence *Candida albicans can* survive chlorination, ultra violate light, filtration and turbidity [35].

Pathogenesis: Candida is acquired by ingestion and probably becomes parts of the resident flora of the mouth, esophagus and crop; under the predisposing conditions it proliferates on the surface and hyphae or pseudo hyphae invades superficial epithelial layers. This invasion stimulates hyperplasia and pseudo membrane or diphtheritic membrane formation [15].

Clinical Signs and Lesions: Signs are variable and non-specific may be associated with the primary or predisposing conditions than with Candidiasis itself. Mortality directly caused by Candidiasis is low to non-existence. Affected chicks show unsatisfactory growth, stunted appearance, listlessness and roughness of feathers. When Candidiasis occurs as a secondary infection, the signs of the predisposing disease predominates the clinical signs. Young birds are more susceptible than older birds to mycosis of the digestive tract. At necropsy, no significant gross lesions were recorded in most visceral organs, except the liver. The liver showed mottling. Thickening of the crop mucosa were noted. However, on opening the oral cavity, gray to well-like lesions at the opening of the pharynx and larynx were seen and this yellowish white pseudo membrane on the mucosa continued to the proximal esophagus [5].

Diagnosis

Direct Microscopic Examination: Direct examination can be performed using various chemicals based on the type of the sample such as lesions in crops, esophagus or the mucus membrane in lacto phenol-cotton blue [36] tissue section stained by PAS (Periodic acid-Schiff) or GMS (Gomorimethylamine silver) [37] fixed smears stained with Wright's and Gimsa [38]. Under microscope *Candida albicans* thick walled resting cells called chlamydospores or chlamydoconidia, ovoid or round, budding yeast cells, hyphae or pseudo hyphae are observed [36, 38].

Culture Examination: The *Candida albicans* culture is carried out aerobically at 37°C2 to 5 days, on sabouraud dextrose agar, with or without cycloheximide [31]. On blood and corn meal agar *C. albicans* grow at 24-48 Hrs [36].

Serological Diagnosis: The most widely used test for systemic Candidiasis is immune diffusion for detection of antigen, which is more specific and sensitive [36].

Treatment: In normal situations, Candida is not harmful because the bodies of poultry are able to keep it under control, mainly by immune cells and probiotic bacteria. However, some factor directly affect the normal balance of intestinal environment, can kill the friendly bacteria and stimulate the overgrowth of pathogenic microorganisms. Therefore, the best treatment is to reduce and control Candida levels. Candidiasis is treated by copper sulphate in drinking water and nystatin in feed or water can be given [38].

Prevention and Control: Prevention and control involves maintaining good sanitation and elimination of factors that predispose poultry to infection. Affected birds should be segregated for protection against cannibalism. Appearance of the disease in very young chicks suggests that the surface of the egg is a source of infection. Such a possibility could be removed by dipping eggs in an iodine preparation prior to incubation [32].

Mycotoxicoses: Mycotoxicoses are diseases of animals caused by ingesting mycotoxin; is diverse group of toxic secondary metabolites produced by certain molds when they grow on agricultural products. They do not belong to a single class of chemical compounds and they differ in their toxicological effects. Avian Mycotoxicoses refers to all the diseases caused by the effect of mycotoxin in birds. It is a great constraint in poultry industry, because the disease is characterized by immune suppression, hepatotoxicity and nephrotoxicity, loss of egg production, mutagenicity and teratogenicity. Mycotoxin are anti-nutritive factor present in feed ingredients and in concentrated feed, they are a group of secondary fungal metabolites of low molecular weight, diverse and ambiguous in nature, which are specifically implicated in causing toxic effects [39].

Mycotoxin are biologically active, toxic metabolites produced by toxigenic fungi mainly belonging to aspergillums', fusarium and penicillium species, which

invade crops in the field and may grow on foods during storage under favorable conditions of temperature and humidity. Mycotoxin may have additive or even synergistic effects with other mycotoxin, infectious agents and nutritional deficiencies. Many are chemically stable and maintain toxicity over time [40].

There are numerous mycotoxins in the food chain that cause unwanted biological effects inside human and animal organisms upon ingestion. High levels of mycotoxin in food and feed results in the appearances of acute mycotoxin and high mortality rates. Lower levels cause the occurrence of chronic mycotoxin with or without manifested clinical symptoms, but followed by a considerable decrease in production performance, immune suppression effects and the occurrence of residues in poultry meat and eggs. Toxicity of mycotoxin primarily depends on the species of mycotoxin, quantity and duration of ingestion, type, sex and age of the bird, general health and immune status, as well as environmental factors and nutritive status. Since fungi frequently produce more than one mycotoxin, the animal simultaneously takes in more mycotoxin through ingestion [41].

Mycotoxicoses in poultry include Aflatoxicosis, Ochratoxicosis and Trichothecene are the most commonly seen mycotoxin in commercial poultry; out of this Aflatoxicosis is the most challenging problem in poultry industry [9].

Aflatoxicosis: Avian Aflatoxicosis is a disease of poultry caused by ingestion or inhalation of aflatoxins. It was the death of about 100,000 turkey poults in the United Kingdom in 1960 following the ingestion of poultry feed containing Brazilian groundnut cake which led to the discovery of a group of compounds now called the aflatoxins. Among mycotoxin, aflatoxins induce greater damages. It is a common condition after rainy season. Aflatoxins are toxic and carcinogenic metabolites of *Aflatoxins flavus, Aflatoxins parasiticus* and others. Aflatoxins are the most prevalent and economically significant mycotoxin to be consumed by poultry [42].

Etiology: Aflatoxins (AF) are widely distributed toxins produced by Aspergillus. Over 180 species of Aspergillus, only a few are Aflatoxigenic. After the discovery of AF in the 1960s, *A. flavus* and *A. parasiticus* of the section *Flavi* were the only known AF producers producing the B and B/G types of AF, respectively [43, 44].

Factor Enhancing Aflatoxigenic Fungi Occurrence and Prevalence in Poultry Feed Ingredients: There are several factors enhancing the prevalence of aflatoxigenic fungi and aflatoxins production. The factors include the following: aflatoxigenic fungi, trace elements and moisture, size and integrity of the seed, hot and humid conditions, Soil type and agricultural practices also affect AF (*Aflatoxigenic fungi)* contamination of feed ingredient [45].

AF has been found as contaminants in animal feed ingredient worldwide. Aflatoxins contamination is thus more likely in grains grown or handled in the tropics or subtropics. Individual fungus usually produces more than one toxin. It is uncommon to find a single mycotoxin occurring under field conditions; usually they occur in combination of two or more [30].

The occurrence in developing countries is more because there is no strict food and feed quality control programmes to reduce the burden of AF. Also their environmental condition presented as hot and humid climate makes most developing country vulnerable to AF in poultry feed. Naturally occurring aflatoxins contain aflatoxins B1, B2, G1 and G2.Designations B and G are given after their blue (B) or green (G) color reaction to fluorescent light. Off all, aflatoxins B1 are usually found in the highest concentration and are also the most toxic. It damages mainly liver [15].

Host affected: Young poultry are most sensitive to aflatoxins than adults. Ducks are being 10 times more sensitive than chickens and turkeys intermediate between the two [30].

Pathogenesis: The toxic effect of aflatoxins manifests itself on the level of interaction with genetic material. The aflatoxins molecule penetrates the cell and the nucleus, subsequently placing itself between the base pairs of DNA. The inserted aflatoxins molecules decelerate to a great extent in the process of DNA information transfer. Mistakes in DNA transcription are very frequent, which result in the inhibition of protein synthesis, i.e. “wrong” proteins are synthesized. Aflatoxicosis cause several effects in poultry. This include: reduced feed intake and weight gain, reduced feed efficiency, reduced immunity, increases mortality, results in liver damage ;such as fatty liver, cause hemorrhaging of the kidney and intestine and cause carcinogenesis and teratogenesis [46].

Clinical Sign and Lesion of Aflatoxicosis: Poultry is considerably resistant to aflatoxins, due to which the acute intoxication is relatively rare. Chronic intoxication

with aflatoxins demands ingestion of aflatoxins for several weeks (One week minimum). The clinical picture is the consequence of the mechanism of aflatoxins effects in the organisms of poultry. Aflatoxicosis does not usually induce mortality directly, although high level (> 10ppm) may be lethal. Lesions will depend on the age of the host and the dose of toxin and include enlarged livers which become friable and yellow with increasing dose, kidney and spleen enlargement and diminution of the bursa of fabricius, thymus and tests. Petechial hemorrhages or bruises after trauma are increased due to decreased clotting factor synthesis and increased capillary fragility [47].

Diagnosis of Mycotoxicoses in Poultry: These diseases may not be pathognomonic and sometimes subclinical and difficult to diagnose. Diagnosis is made through observing the appropriate field signs, finding gross as well as microscopic tissue lesions and detecting the suspected toxin in grains, forages, or the ingesta of affected animals. However, the tests required to detect these toxins are complex and few diagnostic laboratories offer tests for multiple Mycotoxicoses in developing countries. The samples of choice include both refrigerated and frozen carcasses for necropsy examination and are representative sample of the suspected contaminated grain source. Because the toxin is produced under cold conditions, the grain sample should be frozen rather than refrigerated for shipment to the diagnostic laboratory [48].

These samples can be rapidly screened for Mycotoxicoses with commercially available solid or liquid phase competitive enzyme linked immunosorbent assay (ELISA) tests. Positive tests should be confirmed by more rigorous analytical chemical methods including thin layer chromatography, high performance liquid chromatography gas chromatography, mass spectrometry, or monoclonal antibody technology [9].

Treatment of Mycotoxicoses: The toxic feed should be removed and replaced with unadulterated feed. Concurrent disease should be treated to alleviate disease interactions and substandard management practices must be corrected. Nonspecific toxicological therapies using activated charcoal (Digestive tract adsorption) in the feed have a sparing effect; but are not practical for larger production units [47].

Control and Prevention of Mycotoxicoses: Mycotoxin is stable toxic compounds; once they have been produced, it is difficult to destroy them (Even with high temperatures). Several methods of controlling mycotoxin in feed and grain have been introduced such as irradiation, ammoniating, ozone degradation and fermentation. These methods are either expensive, can reduce nutrient quality, or can produce hazardous compounds. The most common and safest method used today is including a mycotoxin binder in the feed to absorb the toxins, resulting in the mycotoxin passing harmlessly through the animal. When untreated mycotoxin contaminated feeds are fed to birds, it may reduce growth and alter the immune system. No matter how strong and how solid the nutrition and health program, if mycotoxin are not under control, producers can experience reduced profitability [49].

Prevention of mycotoxin formation is essential since there are few ways to completely overcome problems once mycotoxin is present. Ammoniating of grains can destroy some mycotoxin, but there is no practical method to detoxify affected feed. Some additives are beneficial in reducing mold growth and therefore mycotoxin formation. Ammonia, propionic acid, sorbic acid and microbial or enzymatic feed additives are shown to be at least partially effective at inhibiting mold growth. Feed bunks should be cleaned regularly. Care should be taken to ensure that high moisture grains are stored at proper moisture contents and in a well maintained structure. Grains or other dry feed should be stored at a low moisture content (Less than14%) below which molds do not readily grow and then protected to remain dry. Aeration of grain is important to reduce moisture migration and to keep the feedstuffs in a good condition [50].

Obviously moldy feed should be avoided, if possible. If unacceptably high levels of mycotoxin occur, dilution or removal of the contaminated feed is preferable; however, it is often impossible to completely replace some feeds in the ration [51].

Public Health and Economic Importance of Fungal Diseases of Poultry: Most fungi are saprophytic and not pathogenic to plants, animals and humans. However, a relative few fungal species are phytopathogenic, cause disease in man and produce toxins that affect plants, animals and humans. Among such fungi are members of the Histoplasma, Cryptococcus and fusarium as well as other genera (e.g., Alternaria, Mucor) comprising the emerging pathogen group in humans. These fungi present a common threat to both agricultural production and the health of healthy and immune compromised individuals [52].

Few fungi can cause huge economic losses to agriculture, loss of food for consumption and serious, often fatal diseases in humans and animals. The Food and

Agriculture Organization (FAO) estimated that about 25% of human foods and animal feeds are contaminated with mycotoxin and strong efforts have been made to decontaminate them by the use of physical and chemical adsorbents but the success made so far is limited [53, 54].

Mycotoxins are currently considered as a serious threat to the poultry farming in terms of diseases leading to synergistic interactions with other infectious agents. The most obvious effect of mycotoxin on poultry is mortality which is produced when high levels of aflatoxins are present in feeds [55].

The most economically significant effect of Aflatoxicosis on growing birds is decreased growth and poor feed conversion (>1ppm). Consumption of even low levels of mycotoxin can lead to decreased feed consumption, poor growth rate and increased susceptibility to disease challenge. Higher levels lead to mortality. More frequent are sub-acute and often sub-clinical alterations of organ function that impair weight gain and reduce productivity. In layers, aflatoxins cause drop in egg production and poor hatchability [9].

CONCLUTION

Generally, fungal or mycotic infections can cause huge economic losses to poultry industry; loss of food for consumption and serious, often fatal diseases in humans and animals. Apart from the fungal infections, mycotoxin are also of major concern as they are the leading cause of immune suppression in birds, lowering their resistance level to various viral and bacterial diseases and increased mortality. Thus, a holistic approach is required to combat the adverse effects of mycotoxin and alleviate their adverse effects on economic returns from the poultry production.

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

- Timely adoption of good management practices, strict bio security, effective disease diagnosis and suitable preventive measure should be implied in order to prevent exposure of birds to fungal infection.
- Environmental modification should be made to decrease exposure to air born spores or decrease ingestion of spores.
- Appropriate control strategies should be designed for the control of mycotic disease.
- Soiled egg, died birds and moldy litter should be properly disposed.

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REFERENCES

1. Biswas, D., S. Agarwal, G. Sindhwani and J. Rawat, 2010. Fungal colonization in patients with chronic respiratory diseases from Himalayan region of India: Annals of Clinical Microbiology and Antimicrobials, 9(28): 1-7.
2. Sajid, M.A., I.A. Khan and U. Rauf, 2006. Aspergillus fumigates in commercial poultry flocks. Veterinary research institutes, Ghazi road Lahore Cant, Pakistan.
3. Saif, Y.M., A.M. Faddy, J.R. Glisson, L.R. Mcdougald, L.K. Nolan and D.E. Swayne, 2008. Disease of Poultry: Fungal infections. 12th ed. Blackwell Publishing, pp: 989-1008.
4. Dhama, K., S. Chakraborty, Mahima, M.Y. Wani and A.K. Verma, 2013. Novel and emerging therapies safeguarding health of humans and their companion animals: A review. Pak. J. Biol. Sci., 16: 101-111.
5. Mandal, A.B., A.S. Yadav, T.S. Johri and N.N. Pathak, 2004. Nutrition and disease management of poultry. Army printing press 33, Nehru Road, SadarCantt, pp: 137-143 and 307-311.
6. Solima, C., A.K.O. Alstrup and O.R. Therkildsen, 2012. A review of the factor causing paralysis in wild birds; implications for the paralytic syndrome observed in the Baltic Sea. Sci. total environment, 416: 32-39.
7. Shivachandra, S.B., R.L. Sah, S.D. Singh, J.M. Kataria and K. Manimaran, 2004. Comparative pathological changes in aflatoxins feed broilers infected with hydro pericardium syndrome. Indian J. Anim. Sci., 74: 600-604.
8. Saif, Y.M., 2003. Poultry disease: Fungal infections. Wiley-Blackwell, USA. ISBN-13:9780813804231. pp: 883-904.
9. Jordan, F., M. Pattison, D. Alexander and T. Frager, 2002. Poultry disease: Fungal disease. 5thed.

10. Charlton, B.R., R.P. Chin and H.J .Barnes, 2008. Diseases of Poultry: Fungal infections. In: Saif, Y.M., Fadly, A.M., Glisson, J.R., Mcdougald, L.R., Nolan, L.K. and Swayne, D.E. eds. Blackwell Publishing. Ames, Iowa, pp: 989-1001.
11. Tell, L.A., 2005. Aspergillosis in mammals and birds: Impact on veterinary medicine. Med. Mycol., 43: 71-73.
12. Ranck, J.R. and A.D. Miles, 2001. Aspergillosis: American association of avian pathologists. Available at: http:// www.Aap.Info/ Assets/Slides/Aspergillosis.
13. Joomla, M. and M. Simon, 2005. A hand book of poultry disease. 2nd ed. Available at: http//.www.Vet.Com.Viewed On: April 26, 2011.
14. Kristiansen, H.H. and C.M. Athes, 2000. Ammonia and poultry welfare: A review. World's Poultry Science Journal, 56: 235-245.
15. Jordan, F., M. Pattison, P.F. Mcmullin, J.M. Bradbury and D.J. Alexander, 2009. Poultry Disease: Fungal disease, 6th ed. pp: 428-441.
16. Samour, J., 2000. Avian medicine.Fahd Bin Sultan Falcon center, Riyadh kingdom of Saudi Arabia, pp: 275-290.
17. Nardoni, S., R. Ceccherelli, G. Rossi and F. Mancianti, 2006. Aspergillosis in larusCachinnansmicaellis: Survey of eight cases. Mycopathologia, 161: 317-321.
18. Perez, J. and L. Carrasco, 2000. Diagnostic histopathology of mycosis in veterinary pathology.
19. Cacciuttolo, E., G. Rossi, S. Nardoni, R. Legrottaglie and P. Mani, 2009. Anatomopathological aspects of avian aspergillosis. Veterinary Research Communications, 33: 521-527.
20. Dahlhausen, B., R. Abbott and P. Vanoverloop, 2004. Rapid detection of pathogenic Aspergillus species in avian samples by real-time PCR assay: A preliminary report. In: E. Bergman ed. Proceedings of the 25th annual conference and expo. Of the association of avian veterinarians. pp: 37. New Orleans, LA. USA.
21. Singh, S., M.K. Borah and D.K. Sharma, 2009. Aspergillosis in turkey poults.Indian Journal of Veterinary Pathology, 33(2): 220-221.
22. Butcher, G.B., J.P. Jacob and F.B. Mather, 2015. Common poultry disease. Gainesville, pp: 611.
23. Redig, R.D. and J. Ackermann, 2000. Raptors.In: Tully, T.N., Lawton, M.P., Dorrestein, G.M. editors. Avian medicine. Oxford: Butterworth Heinemann. Rev. IberoamMicol., 17: 18-22.
24. Carter, G.R., 1991. Essential of veterinary bacteriology and mycology.4th ed. London, pp: 266-268.
25. Quinn, P.J. and Markey, 2003. Concise review of veterinary microbiology.USA: Blackwell Publishing, pp: 72-86.
26. Kearns, R.D. and B. Loudis, 2003. Recent advances in avian infectious disease. Available at: http:www.vet.com.Viewed on: April, 26, 2011.
27. Abrams, G.A., J. Paul-Murphy, J.C. Ramer and C.J. Murphy, 2001. Aspergillusblepharitis and dermatitis in a peregrine falcon-gyrfalcon hybrid (Flacon peregrines x Falcon rusticolus). Journal of Avian Medicine and Surgery, 15: 114-120.
28. Oglesbee, B.L., 1997. Avian medicine and surgery: mycotic diseases. Philadelphia, PA: W.B. Saunders Company, pp: 323-361.
29. Amsalu, L., 2011. Review on Aspergillosis on poultry. A paper presented for the course seminar on livestock development (VCME-577), University of Gondar, Faculty of veterinary medicine, Gondar, Ethiopia.
30. Vegad, J.L., 2008. Poultry Disease: Guide for Farmers and Poultry Professionals. Fungal diseases, 2nd revised and enlarged ed, pp: 179-196.
31. Quinn, P.J., B.K. Markey, E.S. Fitzpatrick, S. Fanning and P.J. Hartigan, 2011. Veterinary microbiology and microbial disease. 2nd ed. Wiley: Blackwell Science, pp: 413-483.
32. Songer, J.G. and K.W. Post, 2005. Veterinary microbiology: Bacterial and fungal agents of animal disease. St. Louis: Elsevier, pp: 384-393.
33. Forbes, B.A., D.F. Sahm and A.S. Weissfield, 2002. Bailey and Scott's diagnostic microbiology.11th ed. St. Louis: Mosby, pp: 780.
34. Tiwari, R., M.Y. Wani and K. Dhama, 2011. Candidiasis in poultry: An overview. Poults technol., 6: 110-111.
35. Hungerford, L.L., C.L. Campbell and A.R. Smith, 1999. Veterinary mycology laboratory manual. Ames: Wesley, pp: 39-47.
36. Ach, P.N. and B. Szyfres, 2003. Zoonosis and communicable disease to man and animals. 3rd ed. Washington DC: Pan American Organization, pp: 315-330.
37. Quinn, P.J., B.K. Markey, M.E. Carter, W.J. Donnelly and Leonord, 2002. Veterinary microbiology and microbial disease.Blackwell Science, pp: 232-238.
38. Hirsh, D.C., N.J. MacLean and R.L. Walker, 2004. Veterinary microbiology. 2nded. USA: Blackwell, pp: 265-272.

39. Muthomi, J.W., J.K. Ndung'u, J.K. Gathumbi, E.W. Mutitu and J.M. Wagacha, 2008. The occurrence of fusarium species and mycotoxin in Kenyan wheat. Crop protection, 27: 1215- 1219.
40. Shamsudeen, P., H.P. Shrivastava, S. Ram and D. Chandra, 2013. Effect of chelated and inorganic trace minerals on aflatoxins synthesis in maize. Journal of Poultry Science and Technology, 1(1): 13-16.
41. Binder, E.M., 2007. Managing the risk of Mycotoxin in modern feed production. Animal Science and Technology, 133: 149-166.
42. Jewers, K., 1987. Problems in relation to sampling of consignments for mycotoxin determination and interpretation of results. Proc. 2nd International Conference on mycotoxin, 28 September - 2nd October, Bangkok: Thailand, FAO: Rome.
43. Peterson, S.W., Y. Ito, B.W. Horn and T. Goto, 2001. Aspergillusbombycis, a new aflatoxigenic species and genetic variation in its sibling species, A. Nomiusmycological, 93: 689-703.
44. Eto, Y., S.W. Peterson, D.T. Wicklow and T. Goto, 2001. Aspergilluspseudotamarii, new aflatoxins producing species in Aspergillus section flair. Mycological Research, 105: 233-239.
45. Makun, H.A., M.F. Dutton, P.B. Njobeh, T.A. Gbodi and G.H. Ogbadu, 2012. Aflatoxins contamination in foods and feeds. A special focus on Africa. In: tech. avian mycotoxicoses in Developing Countries, pp: 111.
46. Njobeh, P.B., M.F. Dutton, A.T. Aberg and P. Haggblom, 2012. Estimation of multimycotoxin contamination in South African compound feeds. Toxins, 4: 83.
47. Kahn, C.M., 2005. The marks veterinary manual.9th ed. Washington DC.
48. Adeniran, L.A., O.P. Ajagbonna, N.A. Sani and H.O. Olabode, 2013. Avian mycotoxicoses in developing Countries. Available at: http://dx.doi.org/10.5772/56050.
49. Huwig, A., S. Freimund, O. Kappeli and H. Dultler, 2001. Mycotoxindetoxication of animal feed by different absorbents. Toxicol. Let., 122: 691-699.
50. Patil, R.D., R. Sharma and R.K. Asrani, 2014. Mycotoxicoses and its control in poultry: A review. Pradesh Agricultural University, Palampur, 176: 062.
51. Galvano, F., A. Piva, A. Ritieni and G. Galvano, 2001. Dietary strategies to counteract the effects of mycotoxin: a review's. Food Prot., 64: 120-131.
52. Lucca, D.A., 2007. Harmful fungi in both agriculture and medicine: National center for biotechnology information. Southern research center, USDA, ARS, New Orleans LA., 70124, USA.
53. Shetty, P.H., B. Hald and L. Jespersen, 2006. Surface bindings of aflatoxins B1 by saccharomyces cervisiae strains with potential decontaminating abilities in indigenous fermented foods.Int. J. Food Microbial, l113(1): 41-46.
54. Yiannikouris, A. and J.P. Jouany, 2002. Mycotoxins in feeds and their fate in animals: A review. Anim. Res., 51: 81-99.
55. Jand, S.K., P. Kaur and N.S. Sharma, 2005. Mycoses and Mycotoxicoses in poultry. Indian Journal of Animal Science, 75: 465-475.