Regulatory Mechanisms in Biosystems

ISSN 2519-8521 (Print)
ISSN 2520-2588 (Online)
Regul. Mech. Biosyst.

Pulmonary aspergillosis in broiler chickens: A case description from a private poultry farm in Lviv Region, Ukraine

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Article info

Received 10.12.2025
Received in revised form 19.01.2026
Accepted 14.02.2026

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Shchebentovska, O., Blishch, H., & Kostynuk, A. (2026). Pulmonary aspergillosis in broiler chickens: A case description from a private poultry farm in Lviv Region, Ukraine. Regulatory Mechanisms in Biosystems, 17(1), e26024. doi:10.15421/0226024

Aspergillosis is one of the most common mycotic infections affecting birds, mammals, and humans. It is recorded worldwide and causes substantial losses to enterprises. The disease develops predominantly following the entry of *Aspergillus* spores into the body of animals and birds, and may run an acute or chronic course. Important factors in disease development include litter or grain contaminated with fungi, poor ventilation in livestock facilities, failure to comply with veterinary and sanitary rules when preparing premises for chick placement, and increased stocking density, which increases the likelihood of infection. The intensity of infection and clinical manifestations depend on the host's defensive capacity, pathogen virulence, and the number of spores entering the body. This article describes a case of pulmonary aspergillosis in 4-day-old Cobb-500 broiler chicks, in which increased mortality was observed over 10 days and exceeded 5%. The affected chicks were inactive, lay mostly with the neck extended forward, had labored breathing, and did not consume feed or water. During necropsy, solitary and multiple miliary greyish-yellow granulomatous nodules were detected, localized on the air sacs and within the pulmonary parenchyma. In most chicks, the air sacs were thickened and, in some instances, covered with fibrinous deposits with a grey-white and greenish-grey mold-like surface. Histological examination of the air sacs and lungs revealed massive accumulations of septate hyphae, arranged chaotically and invading the tissues. Septate hyphae with dichotomous branching had a clearly defined cell wall and, by morphological features, corresponded to fungi of the genus *Aspergillus*. Occasional conidiophores were also detected within the hyphal masses, indicating active sporulation of the agent. In the lungs of broiler chicks, multiple granulomatous lesions were identified; centrally they contained septate, dichotomously branching hyphae that invasively extended into the surrounding parenchyma and vessels. At the periphery of granulomas, heterophilic-macrophagic infiltration and formation of multinucleated giant cells were characteristically present. Grocott staining confirmed the presence of numerous hyphae typical of *Aspergillus* spp.

Keywords: broiler chicks; air sacs; lungs; fungi; hyphae; conidia; granulomatous nodules.

Introduction

In recent years a major problem in industrial poultry production in Ukraine has been an increase in fungal diseases, particularly aspergillosis. This is primarily associated with rearing large numbers of poultry in confined spaces, high temperature and humidity in poultry houses, inadequate ventilation, contamination of feed and litter, and the anatomical and physiological features of the avian respiratory system. High metabolic activity and low immune reactivity in broiler chicks also contribute to rapid infection. This is especially critical during the first two weeks of life. Mortality in acute aspergillosis is quite high and often reaches 5–10% as early as the first week, causing significant economic losses in poultry farming. In addition, aspergillosis should be considered an anthropozoonotic disease: it is transmitted to humans via contact with diseased birds, inhalation of spores from contaminated feed and litter, and consumption of insufficiently cooked meat from affected poultry (Calnek et al., 1997).

Various *Aspergillus* species are ubiquitous, but they are most often detected where environmental conditions favor fungal growth. They are common soil saprophytes that grow well in warm (>25 °C) and humid environments. Growth is often found on damaged eggshells. Serious outbreaks associated with incubator contamination cause the death of more than 15% of chicks during the first two weeks of life and subsequently result in reduced weight gain (Girma et al., 2016). Aspergillosis, as a disease caused by *Aspergillus* spp., was first described in birds by Réaumur in 1749. The pathogenic *Aspergillus candidus* was identified by Rayer and Montagne in 1842 while describing characteristic lesions of the air sacs in the bullfinch (Samanta, 2015). The term "aspergillosis" was first introduced while describing a respiratory disease with pulmonary involvement in the great bustard (*Otis tarda*) at the Frankfurt Zoological Garden in 1863 by Fresenius, whereas the first description of human disease caused by *Aspergillus* spp. was registered by Sluiter in 1847 (Schmidt, 1998; Knoke et al., 2003; Beernaert et al., 2010; Sugui et al., 2014). The genus *Aspergillus* belongs to Ascomycetes, class Eurotiomycetes, and includes more than 250 species grouped into subgenera.

Aspergillus fumigatus is considered the primary respiratory pathogen in birds. Other species, such as *A. flavus*, *A. niger*, *A. nidulans*, and *A. terreus*, have also been isolated from air sacs and lungs in birds (sometimes during mixed infections), but much less frequently than *A. fumigatus* (Okoye et al., 1989; Throne Steinlage et al., 2003; Martin et al., 2007; Arné et al., 2011). Predisposing factors include inadequate ventilation in poultry houses, which increases the risk of aerogenous spread of fungal spores as well as active sporulation of *A. fumigatus* on poor-quality litter and contaminated feed. Acute aspergillosis is reported in young animals and poultry after inhalation of spores, leading to high morbidity and mortality. According to authors, the chronic form is registered less often and mainly affects adult birds (Corkish, 1982; Zafra et al., 2008).

Since the early 1800s, fungi of the genus *A. fumigatus* have been isolated from affected lungs of wild birds, particularly waterfowl, gulls, and corvids, especially after feeding moldy grain and grain waste (Adrian et al., 1978; Zinkl et al., 1997). Some authors described cases of aspergillosis caused by *A. fumigatus* in birds of prey, penguins, and parrots kept in captivity (Alvarez-Perez et al., 2010).

Disease caused by fungi of the genus *Aspergillus* spp. has been recorded in almost all domestic bird species: roosters and laying hens (Corkish, 1982; Throne Steinlage et al., 2003; Talbot et al., 2018), broilers (Akan et al., 2002; Martin et al., 2007), turkeys (Dyar et al., 1984; Kunkle & Rimler, 1996; Cortes et al., 2005; Olias et al., 2010), ducks and geese, ostriches (Khosravi et al., 2008), Japanese quail and pigeons (Singh et al., 1994). In spontaneous outbreaks, mortality ranged from 4.5% to 90%, while the age of birds varied from 3 days to 20 weeks (Arné et al., 2011). Economic losses for industrial poultry

farms are most significant in turkey production, especially when clinical signs appear at the end of the production cycle.

Aspergillus fungi are considered the most common contaminants of litter. Certain species can be identified microscopically based on morphological characteristics (Frisvad et al., 2009). The fungal mycelium consists of septate hyphae with dichotomous branching. The primary branch originating from the vegetative part of the thallus is termed the "foot cell". It branches into conidiophores bearing vesicles, the shape of which varies among species. Portions of the vesicle are covered with rows of phialides arranged in different ways. Asexual spores (conidia) arise from these phialides. The vesicle area covered by phialides, the number of phialide rows as well as their arrangement on the vesicle depend on the *Aspergillus* species. In affected tissues, only mycelium may be visible, whereas conidiophores are sometimes detected in body cavities (Gauthier et al., 2012; Samson et al., 2014). *Aspergillus* fungi are cultured on Sabouraud dextrose agar or Czapek agar (Guinea et al., 2005). *A. fumigatus* colonies appear as white mold that becomes grey-green after several days. *A. niger* colonies are initially white but later turn black. *A. terreus* colonies are also initially white but later become cinnamon-colored with a "sugary texture". *A. flavus* forms pronounced yellow-green colonies with phialides over the entire surface, whereas *A. nidulans* contains a brown pigment that helps it blend with the soil where it typically grows (Tell, 2005).

The primary route of infection with *A. fumigatus* in poultry is considered to be aerogenous (Barton et al., 1992; Beernaert et al., 2010). *A. fumigatus* spores are small enough that they are not fully retained in the nasal cavity or trachea, thus some reach the air sacs and lungs. Typically, the air sacs become the primary sites of lesions because inspired air first enters the posterior thoracic and abdominal air sacs and only then proceeds to the lungs (Fedde, 1998; Nardoni et al., 2006).

In the pulmonary parenchyma, spores settle in the parabronchi, where they are ingested by (surface) phagocytic epithelial cells (Maina, 2002). Defensive mechanisms cannot fully localize infection within the air capillary region when large numbers of spores enter the body or if the immune system is weakened. This leads to the formation of large lesions resembling "cottony plaques", which may undergo fibrous organization (Oglesbee, 1997). Sporulation may sometimes occur in the lungs and air sacs (Nardoni et al., 2006; Cacciuttolo et al., 2009). Hyphae fill the lumen of alveoli and penetrate through the air sacs, inducing fibrinous inflammation (Tsai et al., 1992).

In addition to direct extension through the air sac wall, a hematogenous route may lead to disseminated mycosis. Macrophages in the respiratory tract ingest spores and then, via the interstitium, enter the blood and lymphatic circulation, disseminating infection to other organs (Richard & Thurston, 1983).

Therefore, understanding the clinical and morphological manifestations of aspergillosis and applying mycological and pathohistological diagnostics remain highly relevant, because *Aspergillus*-associated lesions are characterized by high mortality in broiler chicks and considerable variability in pathogenesis between species, while limited therapeutic efficacy necessitates deeper analysis of risk factors and improvement of preventive strategies on farms.

Materials and methods

During this study, ethical requirements for the use of animals in experimental research were fully observed (Strasbourg, 1986; Kyiv, 2002). An outbreak of aspergillosis in Cobb-500 broiler chicks was registered at a private poultry farm in Lviv Region. At 4 days of age, increased mortality was recorded, exceeding 5% by day 10. Clinically, affected chicks did not consume water or feed, remained largely recumbent with the neck outstretched, and exhibited labored respiration. For laboratory investigation, 50 chick carcasses were delivered and necropsied in the post-mortem examination room of the Department of Normal and Pathological Morphology and Forensic Veterinary Medicine of Stepan Gzhytskyi National University of Veterinary Medicine and Biotechnologies.

For pathohistological examination, fragments of air sacs and lungs were fixed in 10% aqueous formalin followed by washing in tap water. Tissue fragments were dehydrated through an ascending alcohol series starting from 700, increasing the concentration by 10% with an exposure of 12 hours at each step. They were then transferred to a 1:1 mixture of xylene and absolute ethanol for 1 hour, then to xylene I for 1 hour and xylene II for 1 hour, and then held in a 1:1 mixture of xylene and paraffin for 1 hour. In a thermostat, the tissues were infiltrated with paraffin in two changes at 56 °C, with an exposure time of 1 hour in each change. The material was embedded in blocks and cooled. From the obtained blocks, 7-µm histological sections were prepared on an MC-2 sliding microtome and, after deparaffinization, stained with Mayer's hematoxylin and eosin.

To visualize the fungal structures that stain intensely black, the Grocott method was used. Paraffin sections were deparaffinized and brought to water, then treated with 5% chromic acid for 1 hour followed by rinsing in running water for 5 minutes and in three changes of distilled water. Silver impregnation was carried out in a mixture of 25 mL of the basic methenamine silver solution, 25 mL distilled water, and 1–2 mL of 5% sodium tetraborate (at 45–60 °C) under microscopic control until fungi began to stain dark brown. The sections were then rinsed in two changes of distilled water. Toning was performed in 0.1% gold chloride for 5 minutes. Residual silver was thoroughly removed with 2–3% sodium thiosulfate followed by rinsing in running water. Counterstaining was performed with 0.2% Light Green SF in 0.2% acetic acid. Sections were dehydrated, cleared, and mounted in synthetic balsam (Mulisch & Welsch, 2010). In this research, we used a combination of Grocott and hematoxylin and eosin. Such approach allowed detailed assessment of the cellular composition of granulomas and inflammatory infiltrates, whereas counterstaining with Light Green alone provides only background staining with limited cellular detail. Light microscopy and microphotography of the obtained histological specimens were performed using a Leica DM-2500 microscope and a Leica DFC 450C camera.

Results

Necropsy of broiler chicks revealed multiple small, round, whitish-grey and yellow granulomatous nodules on the membranes of the air sacs (Fig. 1a, 1b). In some broiler chicks, the air sacs were thickened, with multifocal fibrinous deposits (Fig. 1d). In others, large caseous-necrotic foci with a characteristic greenish-grey mold-like surface were detected in the area of the caudal air sacs (Fig. 1c). Such macroscopic changes are typical of colonization by fungi of the genus *Aspergillus*. The kidneys were hyperaemic, the liver was mildly enlarged, and small yellow granulomas were occasionally observed between the intestinal loops. The pericardium had a whitish fibrinous deposit. In the pulmonary parenchyma, moderate venous congestion and multifocal miliary greyish granulomas were noted. In several broiler chicks, a large caseous-granulomatous mass was present in the ventral part of the body cavity, replacing the normal air sac structures (Figs. 1d, 2a). Thus, the main lesions in broiler chicks were confined to the air sacs and lungs. Their size ranged from pea-sized nodules to diffuse lesions (Fig. 2a, 2b). In the lungs, multiple miliary, firm, yellow lesions were observed (Fig. 2a, 2b).

Histological examination of broiler chick air sacs revealed massive accumulations of septate fungal hyphae in the serosal and subzerosal layers, arranged chaotically (Fig. 3a). Around the hyphae, cellular infiltration was detected, represented by heterophils, macrophages, and lymphocytes (Fig. 3c). Septate, dichotomously branching hyphae with a clearly identified wall were morphologically consistent with fungi of the genus *Aspergillus* (Fig. 3d). The hyphae were surrounded by dense epithelioid and heterophilic infiltration. Marked edema was noted in the intercellular matrix (Fig. 3b). Hyphae invaded the surrounding tissues, causing focal tissue necrosis.

In Figure 4a and 4b, hyphae are clearly visible invading and disrupting the air sac wall; the wall itself is thickened due to massive lymphohistiocytic infiltration and edema. Multinucleated giant cells and macrophages are noted. Within the lumen and on the surface of the air sac wall, dense aggregates of septate fungal hyphae with dichotomous branching were present (Fig. 4c, 4d), along with occasional rounded, thick-walled structures consistent with conidiophores. Surrounding tissues are abundantly infiltrated with macrophages and lymphocytes.

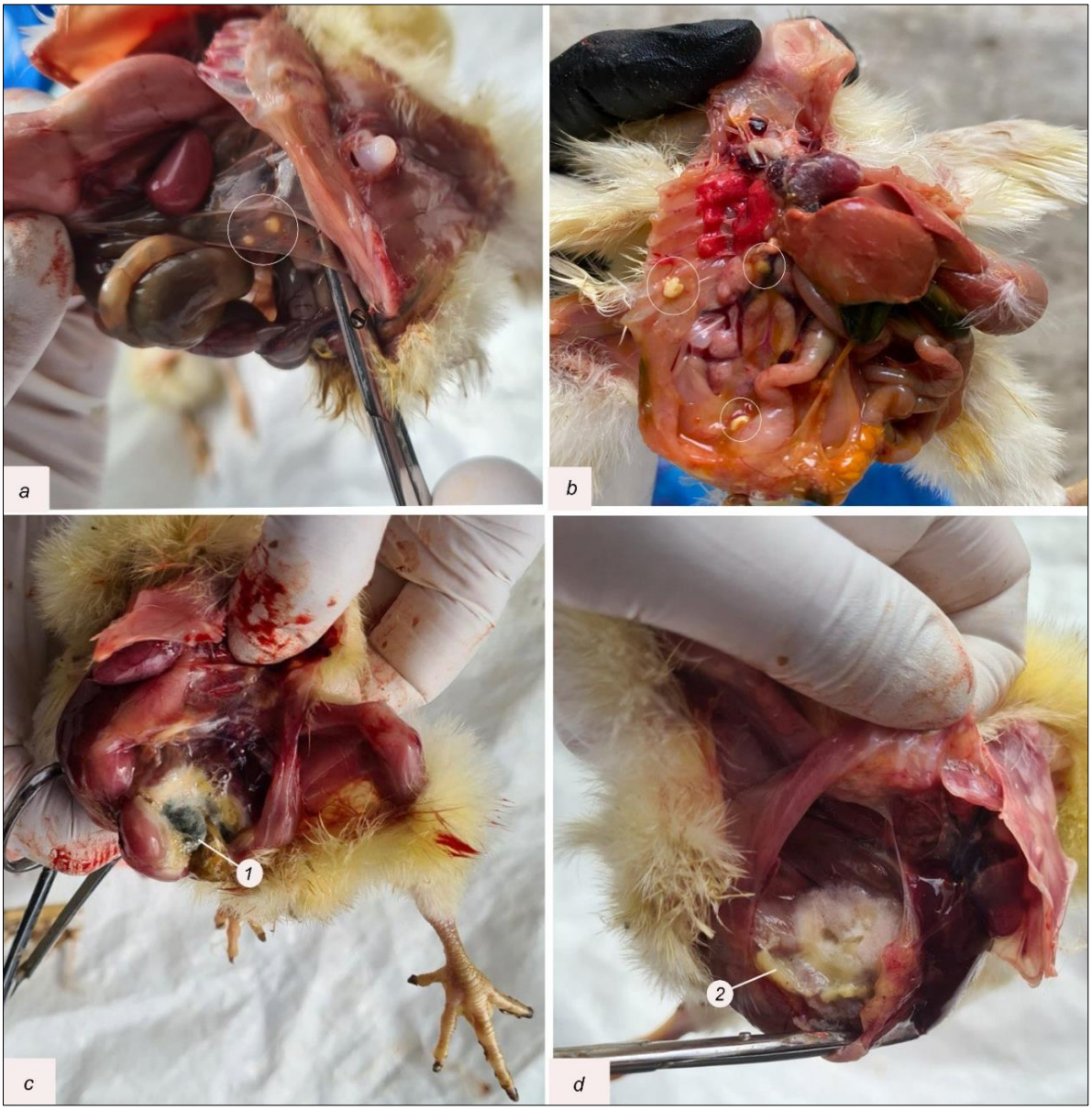

Fig. 1. Pathoanatomical changes in broiler chick aspergillosis: *a* – greyish-yellow granulomas on the air sacs (circled); *b* – marked peritonitis and airsacculitis with granulomas on the air sacs (circled); *c* – caseous-necrotic lesion of the caudal air sacs with characteristic greenish fungal colonies (*1*); *d* – massive caseous deposits and thickening of air sac walls (*2*)

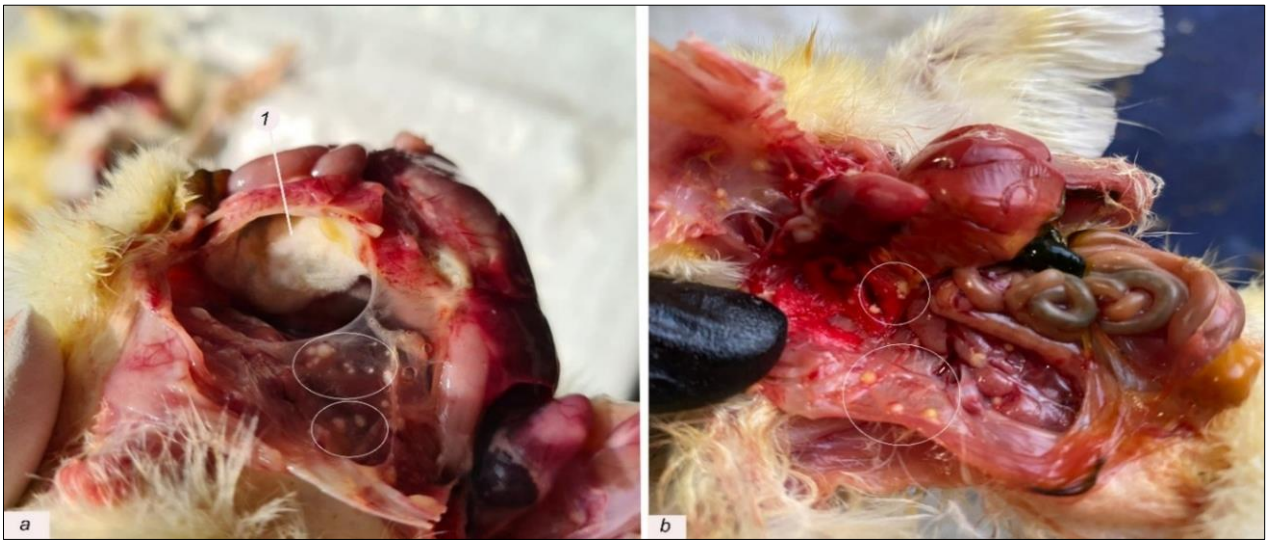

Fig. 2. Pathoanatomical changes in broiler chick aspergillosis: *a* – diffuse miliary lesions of the abdominal air sacs (circled), massive caseous deposits and thickening of the walls of the thoracic air sacs (*1*); *b* – multiple yellow granulomas in the pulmonary parenchyma and air sacs (circled)

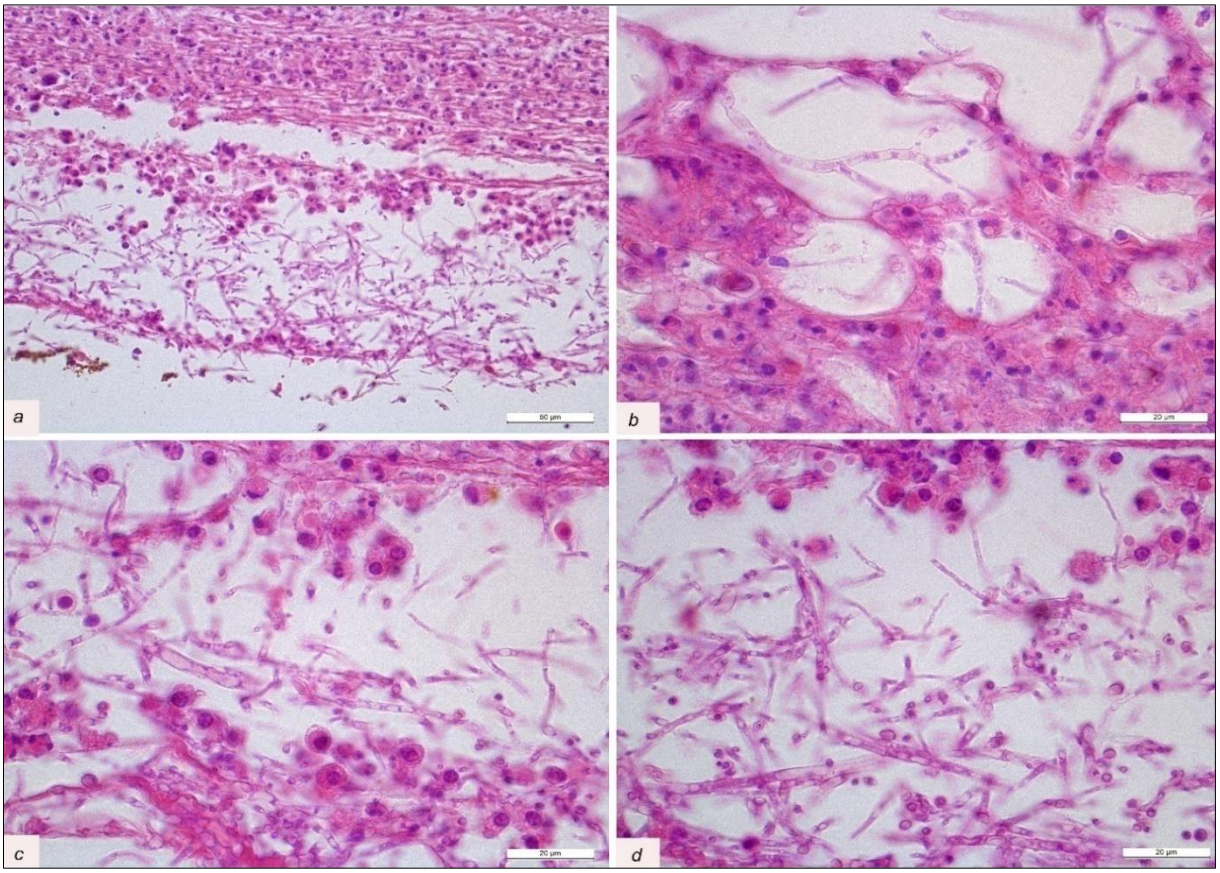

Fig. 3. Pathohistological changes in the air sacs of broiler chicks with aspergillosis:
a – extensive accumulations of fungal hyphae in the serosal and subserosal layers, surrounded by heterophilic cellular elements;
b – branched septate hyphae of *Aspergillus* spp.; *c*, *d* – branched mycelial masses; hematoxylin and eosin

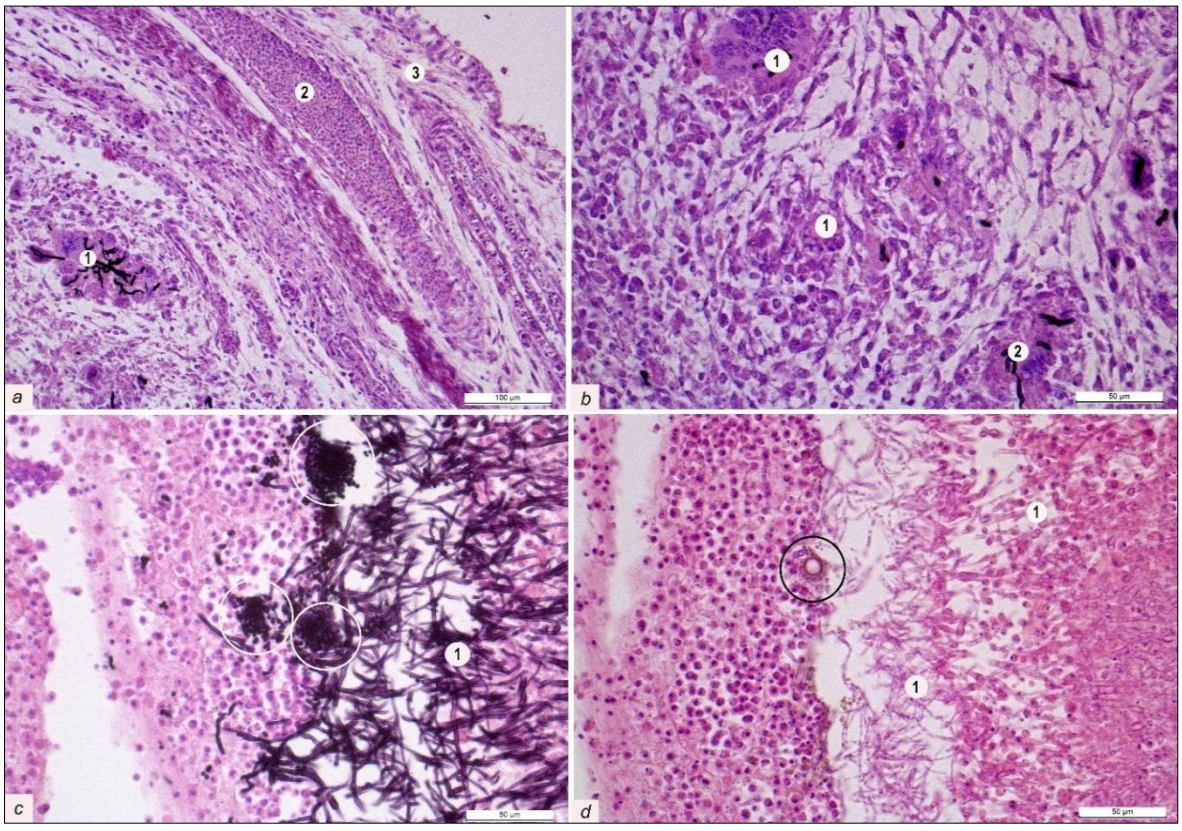

Fig. 4. Pathohistological changes in the air sacs of broiler chicks with aspergillosis:
a – invasion of septate hyphae into the air sac wall (*1*) with pronounced lymphohistiocytic infiltration (*2*) and edema (*3*);
b – giant epithelioid cells (*1*) with occasional hyphae (*2*); *c* – massive mycelial aggregates with conidiophores (circled), hyphae (*1*);
d – accumulations of septate hyphae (*1*) with a conidiophore (circled); hematoxylin and eosin + Grocott

Numerous granulomatous lesions were observed in the pulmonary parenchyma; their centers contained septate, dichotomously branching hyphae extending radially into the surrounding parenchyma (Fig. 5a). At the periphery of granulomatous nodules, pronounced cellular infiltration by macrophages, heterophils, and occasional multinucleated foreign-body-type giant cells was present (Fig. 5a, 5b). The morphological architecture of the pulmonary parenchyma in broiler chicks affected by *Aspergillus* is markedly altered; alveolar spaces are destroyed or filled with cellular elements. At the periphery of granulomas, alongside branched hyphal masses, occasional fragments of fungal elements are detected – they invasively damage vascular walls and enter the bloodstream (Fig. 5b). Diffuse heterophilic-macrophagic infiltration and moderate interstitial edema are also detected around the affected areas.

In histological sections stained using the Grocott method, numerous fungal structures are clearly demonstrated; they stain intensely black as a result of the interaction of methenamine silver with polysaccharide components of the fungal cell wall. Dense aggregates of septate *Aspergillus* spp. hyphae are observed within the lumen of the air sac and in adjacent tissues (Fig. 6a); the hyphae have thin, sharply demarcated walls and typical dichotomous branching. At the periphery, numerous small fragments and freely lying hyphae are visible, indicating active spread of infection. The air sac tissues and pulmonary parenchyma are stained greenish, allowing clear verification of fungal elements. In the pulmonary parenchyma, a granulomatous lesion is present with a central accumulation of invasive hyphae (Fig. 6b) extending radially and penetrating the interalveolar septa. In addition, hyphae are observed in perivascular and peribronchial regions, confirming an invasive growth pattern. The hyphae contrast well against the green background of the stroma, enabling visual assessment of the extent of tissue involvement.

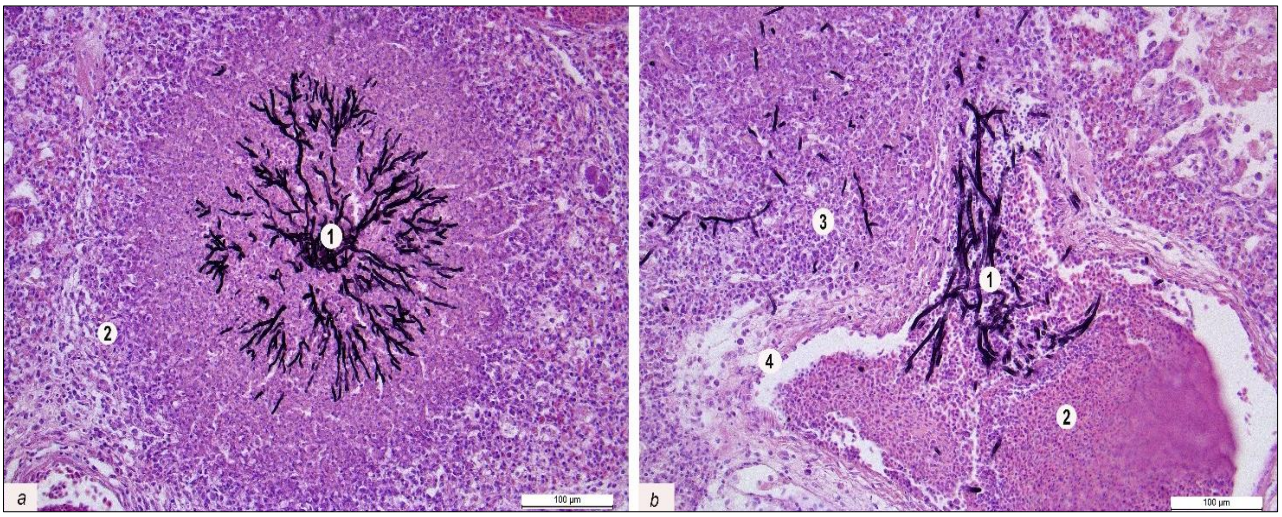

Fig. 5. Lungs of broiler chicks with aspergillosis: *a* – granuloma with septate, dichotomously branching hyphae of *Aspergillus* spp. (*1*) in the center and heterophilic cellular infiltration (*2*); *b* – hyphae (*1*) within the vascular lumen, erythrostasis (*2*), occasional hyphal fragments around cellular elements (*3*), edema with loosening of the vascular wall (*4*); hematoxylin and eosin + Grocott

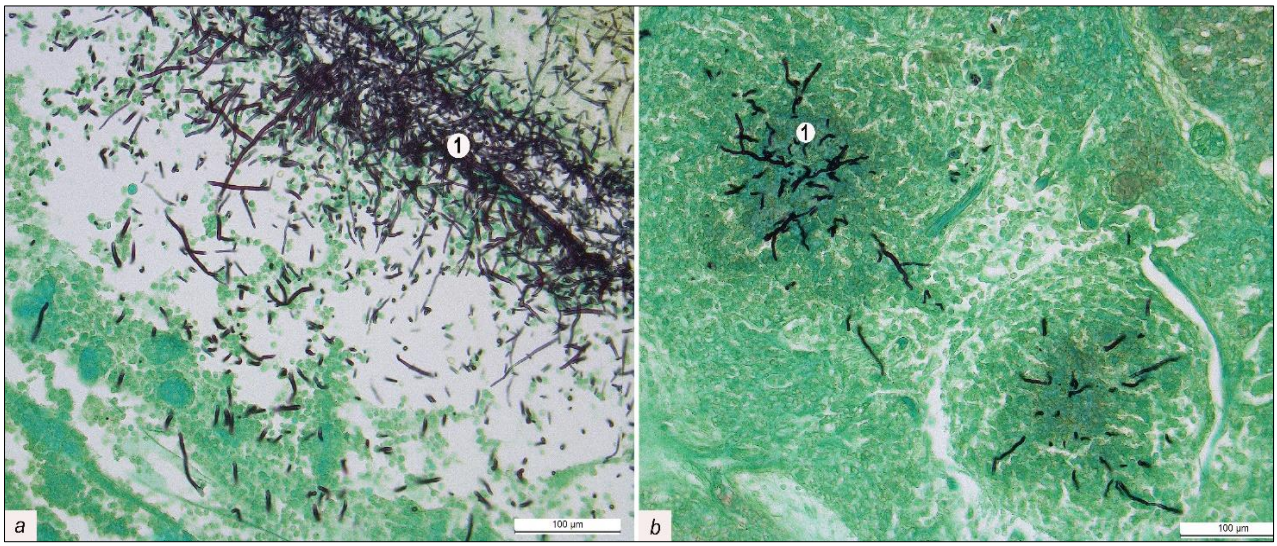

Fig. 6. Histological changes in the air sacs and lungs of broiler chicks with aspergillosis: *a* – dense conglomerates of septate, dichotomously branching *Aspergillus* spp. hyphae (*1*) within the air sac lumen; *b* – pulmonary parenchyma with formed granulomas, in the centers of which black-stained hyphae (*1*) are visible; Grocott methenamine silver stain

Discussion

Aspergillosis is a respiratory disease of chickens, humans, mammals, and wild birds caused by the globally distributed fungus *Aspergillus*; it is associated with high mortality, particularly in chickens. Several factors play an important role in the development of the disease, namely poor hygiene in poultry premises, litter or grain contaminated with fungi, and inadequate ventilation; in combination with other husbandry factors, these increase the likelihood of disease occurrence and spread (Cacciuttolo et al., 2009; Hauck et al., 2020).

In the outbreak described, the primary lesions were localized in the air sacs and lungs, which is consistent with the typical route of

spore entry into the avian host and the development of early *Aspergillus* infection. The macroscopic and histopathological findings reflected a classic granulomatous pattern of lesions similar to that described by Sawale et al. (2012), characterized by the formation of dense caseous granulomas, extensive mycotic colonies, and marked heterophilic-macrophagic infiltration. Our pathohistological examination confirmed invasion by septate hyphae with dichotomous branching that extended into the walls of bronchioles, the air sacs, and the surrounding pulmonary parenchyma, resulting in coagulative necrosis and granuloma formation.

As reported by Ahmed et al. (2017), infection develops after inhalation of a large number of spores that invade the respiratory epithelium and initiate mycelial growth. The lesions identified in our study likewise support the invasive nature of the infection, with diffuse involvement of the air sacs followed by lung damage, consistent with the pathogenesis described by Richard et al. (1983) and Martin et al. (2007). Pulmonary lesions were more localized than the extensive granulomas formed in the air sacs, which may indicate secondary involvement of the pulmonary parenchyma.

According to Richard & Thurston (1983), dissemination of the fungus to parenchymal organs, such as the kidneys and liver, and to the serosal membranes of intestinal loops is usually attributable to hematogenous spread of the agent. In our case, lesions of parenchymal organs were not observed. Kshetrimayum (2025) noted that an immunosuppressive state in birds, with atrophy of the bursa of Fabricius and thymus, typically contributes to the development of a severe visceral form of aspergillosis, but primarily in adult birds. Similar observations were reported by Martin et al. (2007) and Arné et al. (2011), who detected caseous foci in the kidneys and liver of laying hens.

When establishing a diagnosis of a fungal disease, verification is required, for example differentiation of *Aspergillus* from *Penicillium*, which can be readily distinguished by their reproductive structures. In *Aspergillus*, these are represented by a conidial head consisting of a vesicle with phialides borne on it and chains of conidia formed at their tips. In *Penicillium*, reproductive structures form a characteristic "brush", without vesicles. The conidiophores are branched, lack a vesicle, and bear multi-tiered phialides, from which conidia are produced in chains.

Thus, the combination of macroscopic and histological changes in the respiratory organs of broiler chicks allowed us to diagnose this case as a systemic pulmonary form of aspergillosis. The high mortality and frequent occurrence of respiratory disease in young broiler chicks indicate high susceptibility to aspergillosis and underscore the importance of early diagnosis, microclimate control, and management of other risk factors, with subsequent monitoring of the immune status of young birds. In this study, we modified the Grocott method by omitting the Light Green solution and subsequently counterstaining with hematoxylin and eosin, which, in our view, enabled detailed differentiation of cellular composition and identification of histopathological features of fungus-host interaction.

Conclusions

Fungi of the genus *Aspergillus* have a high capacity for growth under diverse environmental conditions, which accounts for their ubiquitous distribution. Following entry of spores into the avian host, severe lesions typically develop in the respiratory organs, leading to high mortality. Establishing a diagnosis of aspergillosis is complex and requires a comprehensive approach using multiple investigative methods.

None of the authors have any personal or financial relationships with individuals or organizations that could inappropriately influence the content of this publication.

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