Review Article

BASRAH JOURNAL OF VETERINARY RESEARCH, 2023, 22 (3):75- 87.
https://bjvr.uobasrah.edu.iq/

Recent aspects of metapneumovirus in broiler/article review

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DOI: 10.23975/bjvetr.2023.140592.1029

Received: 29 May 2023 Accepted: 9 September 2023.

Abstract

Avian metapneumovirus (AMPV) is a worldwide an acute, extremely contagious upper respiratory illness in Turkey. Chickens may also be affected with virus and cause Swollen Head Syndrome. Sometime it is associated with reproductive affection. The disease's first disorder appeared in late 1970 in South Africa, where 6 subtypes (A, B, C, and D) and 2 additional subtypes were found. The virus belongs to the family Paramyxoviridae, genus Metapneumovirus. . Both of systemic and local immunity may be developed but there is no relation of maternal immunity for produce production. The disease causes an important economic loss due to decrease in egg production and bad egg shell quality. The disease spread between birds rapidly and a wild birds may be infected and paly role in transmission of the disease Also in turkey there is no specific clinical signs and lesion and while the most effective methods to detection are Elisa and (RT-PCR) carry out the disease diagnosis. Biosecurity programs of vaccination are effective for controlling of disease. Both of alive attenuated and inactivated vaccines are useful for provide a good protection against the disease While, new vaccines were developed to avoid infection.

Keywords: virus, turkey, rhinotracheitis, respiratory tract.

Introduction

Avian metapneumovirus is an important multifactorial disease poultry and turkey upper respiratory tracts. Viruses can induce swelling head syndrome (1). and avian rhinotracheitis contagious in upper respiratory tract of turkey (2). The paramyxoviridae family's subfamily pneumovirinae (AMPV) includes four different subtypes of the virus have been recognized at

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the base of attachment (G) protein by nucleotide sequence analysis. (3,4). subtype A and subtype B are mainly found in Europe (5) subtype A and Middle Eastern and Asian nations have also encountered subtype B (6) While subtype C are found in USA (7) In France, subtype D has also been described (8).

Infraorbital sinus edema on both sides characterizes the syndrome's graphic manifestations incomplete in Broiler (10) Due to reproduction and respiratory infection and secondary bacterial infection. (11) Swollen sinuses are the typical signs that observed in outbreaks clinical signs in chickens or also turkey (12). Serous and watery discharge followed conjunctivitis in the late stage. Nasal discharge associated with sneezing, tracheal rales, coughing, ruffled feathers, anorexia and depression. Morbidity rate was 100 % and mortality 1 % to 30 % (13).

The gross lesions of the syndrome is characterized by swelling of infraorbital sinuses bilateral or unilateral and then nervous signs may be observed such as torticollis, disorientation and opisthotonos (14). Microscopical lesions including infiltration of inflammatory cell in the sub mucosa of bronchi (15). Horizontal spreading may occur within the flock either directly or by the use of infected objects(16). In experimental study the contaminated eggs post infection may have a small impact on vertical (17). The identification of AMPV antibodies in hens and turkeys might be done using serological testing (18). To avoid financial losses and because of a decline in egg production and mortality, biosecurity programs and vaccination with live attenuated vaccine and inactivated vaccine are crucial (19).

Synonyms:

AMPV virus is a respiratory pathogen effect Broiler. It causes acute and contagious infection in upper respiratory tract, The clinical disease of turkeys rhinotracheitis of Turkey (TRT). and chickens with avian rhinotracheitis (AR) or swollen head syndrome (SHS) (20).

Epidemiology

The virus was first discovered in turkeys in South Africa in the 1970s, It has since spread to other nations around the world. It is placed initially in the genus of pneumovirus (21). In 1980 is the disease was discovered in chickens later known as SHS (21). During 1990 s, alive attenuated vaccine was discovered, the subtype A and B were existed in 1994 and it is the first decided in turkey TRT in 1996 in the USA (22). The virus was detected in Italian broiler farm with high density from 2014 to 2016 (23).

In Iran, subgroup B were characterized during 2011 ,2013 ,2016 ,2017 ,2018 survey study (24). In last few years (2014- 2019) a fragment of the AMPV subtype B G gene was found in European nations. (25). A seroprevalence study of AMPV in Duck and chicken have an economic impact in Egypt. (26). Finally, the disease virus was recorded in Iraq utilizing primers tailored to the proportion of subtypes A, B, and C of the SHS virus in broiler flocks (27).

Taxonomy and Classification:

AMPV below family paramyxoviridae and subfamily pneumoviridae and 2 genera belong it . It consists of the avian and human species of the orthopneumovirus and the metapneumoviru(28). The virus was divided by subtypes A, B, C, and D (29). The virus is an enveloped RNA un segmented single strand. The virus shows pleomorphic particles under an electron microscope that are typically spherical and 80–200 nm in diameter. They also have a surface projection (13–14) nm in length and a helical capsid (14 nm) in diameter (30). The virus is inactive at 56 ℃ for 30 min, stable at PH 3-9, and sensitive to lipid solvent (20). Figure (1). The use of disinfectant such as ethanol ammonia phenol, iodophor and sodium hypochlorite induced the virus's viability

decreased, and it shown resistance to PH. between 5 and 9, indicating that 50 ℃ is an efficient way to end the virus' viability. (31).

Economic significance:

Despite vaccination programs, the virus still one of the most important problems especially in turkey farms in decrease in egg production in layer and breeder and It causes a problem for many years especially in Midwest and united states, mainly in breeder especially the drop egg production. The subtype C cause economic losses in egg production in chickens, pheasant and ducks in Asia and Europe (32,33).

Transmission of AMPV:

The disease was transmitted horizontally through direct contact between birds. Aerosol spread also play a role in infection between farms and flocks. The disease also can spread via contaminated water, food and other materials found in chickens houses (34). The movement of people, equipment, and vehicles contributes to the spread of the illness. The likelihood of infection rises with the introduction of new birds and wild birds. There is no proof that the disease is spread vertically (35).

Pathogenesis and Pathogenicity of the AMPV:

Despite the significant morbidity and fatality rates which rise in the field, it is challenging to determine the pathogenicity in a laboratory setting because of the differences across virus strains. Experimentally infected birds show milder signs of rhinotracheitis than those in the field. The most signs which are seen in nasal mucus (36). Longitudinal study suggest AMPV infection in the field and its prevalence are directly related with colibacillosis infection and these correlations are responsible for mortality (37). So the pathogenicity in laboratory and field depends upon the condition which birds are kept (38).

The epithelial cell of respiratory tract is the target of AMPV and reproductive tract due to replication of the virus induce ciliostasis and loss of cilia (39). This is allowing secondary bacterial infection to invade the host which is caused immunosuppressive affection and reducing the responses for vaccines (40). The genome of virus not only detected in respiratory including the trachea, nasal turbinate, lung, harderian gland, but also in spleen and cloaca. The virus is spread by macrophages from the site of replication to the surrounding tissue. (41,42).

Immunological check point against AMPV:

Activated immunity

The immune system's cell-mediated response (CMI) demonstrated resistance to AMPV infection. (43). Alive attenuated vaccine when given in one day old, still unable to give resistance to virulent AMPV (44). T lymphocyte response in turkey infection showed a slow recovery from the disease in compared to T-cell intact birds (43). Maternal immunity derived due to vaccination give Turkish researchers found that the harderian gland and tracheal mucosa were both strongly stimulated by CD8+. While in other birds vaccination stimulate CD4+ T-cell mainly locally (45) .

Humoral immunity:

There is no indicator for humeral immunity to provide defense against AMPV infection, but a particular antibody prevents the virus from replicating. (46). B-cell were accumulated locally as a population in the nasal turbinate and release of IgA into nasal secretion (47). Lacrimal fluid, bile, and tracheal washes all contained specific IgA (48).

Passive immunity (vaccination program):

The circulating antibodies which released AMPV infection in hens pass to through way of the egg yolk, their offspring. experimentally the high level of antibodies which were found in circulation and in tracheal fluid did not give a protection against the virulent AMPV (49).

Clinical symptoms, microscopic lesions, and perspective of view:

The virus causes an acute infection of upper respiratory tract in turkey and it is highly contagious. The incubation period is 3-7 days (50), But in severe cases of turkeys occurred in birds between the ages of 3 and 12 weeks, with symptoms including dyspnea, rales, sneezing, coughing, nasal discharge, enlargement and edema of the infraorbital sinuses (50). Figure (2,3).

In laying turkeys, the symptoms are less severe, but the virus's re-infection of the oviduct epithelium results in a 10–40% reduction in egg production and changes to the egg shell's quality. (51) recovery takes up to 3 wks. (52). Secondary infection with bacteria in turkey exhibit sever clinical signs (53) a Newcastle disease virus infection resulting in morbidity and infraorbital sinusitis. (54).

The presence of mucoid exudate in the nasal turbinate in rhinitis and catarrhal irritation of the upper respiratory tract are signs of the gross lesion of TRT., tracheitis and laryngitis due to secondary complications induces more severe lesions such as pneumonia and airsacculates, osteomyelitis (55) figure (4,5) and subcutaneous. Reproductive abnormalities, such as egg peritonitis, a folded shell membrane in the oviduct, and an abnormally shaped egg (32).

The microscopic lesions are represented by loss of cilia multifocal deciliation of epithelium hyperemia with mononuclear infiltration in submucosa found in the turbinate, tracheal and conjunctival epithelial damage, as well as intracytoplasmic inclusion bodies. (6).

Direct Diagnostic Methods:

Isolating and identifying the virus: The tracheal organ culture (TOC) method of virus isolation, which involves incubating the virus in chicken or turkey embryos, is not appropriate for the isolation of subtypes of viruses because the isolated virus did not generate ciliasis. (57).

1- Culture in embryonated eggs that are six to eight days old, via the yolk sac method, is required for incubated embryo turkey and chicken eggs. The original subtype C was isolated using the method (58).

2- Utilising negative electron microscopy to identify viruses. Identification can be aided by the physical and chemical characteristics. (59).

3-Direct detection of viral antigens: It has been demonstrated that AMPV N protein, monoclonal, and polyclonal antibodies cross-react when utilised to detect an antibody's reaction at a nucleoprotein (N). Immunofluorescent (IF) labelling was used to find viral antigen in both unfixed and fixed tissues and smears. (60).

4-Serology: Elisa is the most common serological method for diagnosis of the virus and commercial kits for detecting antibodies.

Zoonosis:

There is no history of human disease from AMPV (34).

Differential diagnosis:

Since the clinical symptoms and gross lesions are not unique to TRT in turkey, it is possible that they will be mistaken for other respiratory conditions, including as paramyxoviruses, infectious bronchitis, and influenza, which can interfere with the production of eggs in chicken and turkey. The condition is comparable to other respiratory illnesses that can be brought on by bacteria and mycoplasma (35).

Biosecurity

The prevention of economic losses owing to a decline in egg production and mortality must be the foundation of biosecurity programs in the region, which has a significant number of poultry species (49). To minimize the severity of the disease caused by AMPV infections, there is

no effective treatment. To treat the bacterial disease that follows, it must be taking antibiotics (62).

Conclusion

This paper explains the significant AMPV research investigation.

Conflict of interest

The paper's content was not improperly influenced by any personal or financial relationships the author may have had with other people or organizations.

Fig..1. Electron microscopy of avian metapneumovirus revealing irregularly shaped particles with a fringe of spikes. (Courtesy of R.C. Jones.)

Figure2: Infection with the avian metapneumovirus (AMPV) in a chicken. Foaming eyes and nasal discharge are symptoms of an experimental infection (red arrow). *Source:* S. Rautenschlein

Figure3: SHS in chickens can be associated with aMPV infection (Red arrow). (Author's own image.)

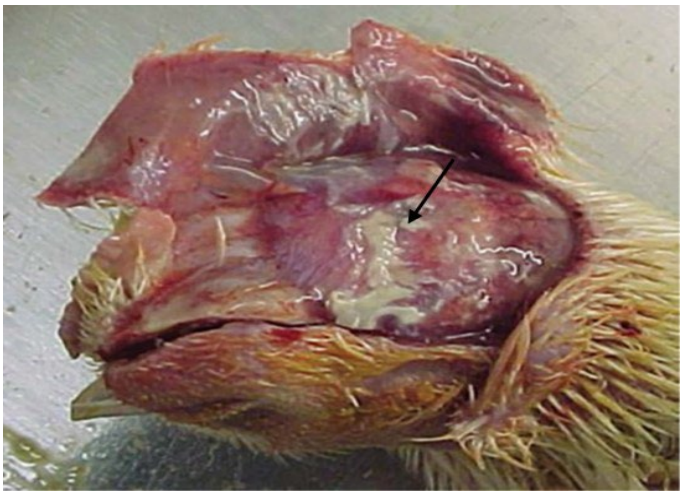

Figure4: Gelatinous caseous exudation of an enlarged skull under the skin (black arrow). (Author's own image.)

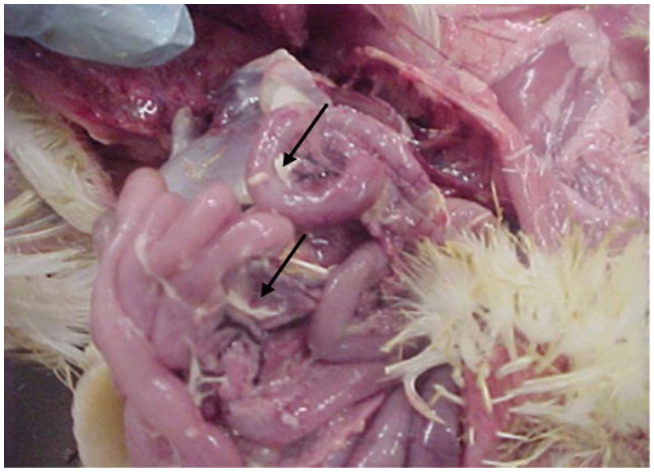

Figure5: An egg-laying hen with an infectious peritonitis.(Author's own image.)

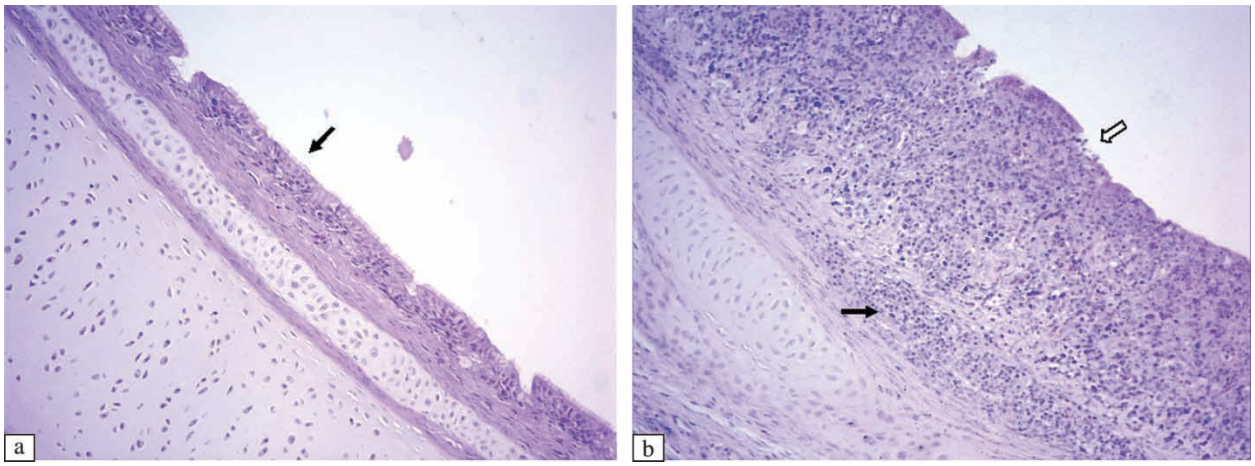

Figure6: Tracheal lesions with histopathology at 6 days post-injury (H & E stain, 200x magnification). Control broiler's intact ciliated epithelium (black arrow) in figure 6a. Trachea of a grill inoculated with the aMPV subtype-A showing focal exfoliation (white arrow), thickening brought on by oedema, and infiltration of inflammatory cells in the epithelium (black arrow) in figure 6b. comparable lesions in the tracheae of broilers immunised with an MPV subtype.

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الجوانب الحديثة من فيروس الميتانيموفيروس في دجاج التسمين

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الخلاصة

تعد الإصابة بفايروس Metapneumovirus من الإصابات الحادة شديدة العدوى وهو واسع الانتشار في العالم يصيب الجهاز التنفسي العلوي في طائر الرومي ويسبب (التهاب القصبات والانف في الديك الرومي) وقد تتأثر الأنواع الأخرى من الطيور مثل الدجاج وتسبب متلازمة انتفاخ الرأس بالاشتراك مع لإصابة بالجهاز التناسلي .اول حدوث للمرض كان أواخر عام 1970 في جنوب افريقيا اذ تم تحديد 6 أنواع فرعية A,B.C,Dونوعان جديدان تحت النوع .ينتمي الفايروس الى جنس Metapneumovirusتحت عائلة paramyxovirus وتتطور كلا النوعين من المناعة الجهازية والموضعية نتيجة الإصابة ولاتوجد علاقة للمناعة الامية في احداث تحصين لدى الافراخ ويتسبب المرض في حدوث خسائر اقتصادية مهمة بسبب انخفاض انتاج البيض وسوء جودة القشرة .ينتشر المرض بسرعة بين الطيور وقد تصاب الطيور البرية وتلعب دورا مهما في انتقال المرض لا توجد علامات سريرية محددة في طائر الرومي ولكن يمكن ملاحظة الافة العيانية المتمثلة بتورم الوجه. تساعد الاختبارات (الاليزا وRT-PCR) في تشخيص المرض وتعد برامج الامن الحيوي والتطعيم فعالة في السيطرة على المرض وتوفير حماية جيدة ضده كما تم تطوير لقاحات جديدة لتجنب المرض.

الكلمات المفتاحية: فايروس، التهاب القصبات والانف, الديك الرومي، الجهاز التنفسي.