Basrah Journal of Veterinary Research 2026; 25(1):143-161.

Print ISSN: 1813-8497
Online ISSN: 2410-8456
https://bjvr.uobasrah.edu.iq/

A Comprehensive Review of Marek's Disease in Chickens

Article Info.

Author

Sara Salim Mohammed[1], Budoor Muhammad Lateif[2], Isam Azeez Khaleefah[2] , Muhammad Tahir Abdul Razzaq Abdulrasoola[2].

1-Department of Veterinary Public Health; College of Veterinary Medicine; University of Basrah, Basrah, Iraq.

2-Department of Pathology and Poultry Diseases, College of Veterinary Medicine, University of Basrah, Iraq.

Corresponding Author Email Address: isam.azeez@uobasrah.edu.iq

Article History

Received: Dec. 8, 2025
Accepted: Jan. 31, 2026
Published: March 31, 2026

Article type: **Review Article**

https://doi.org/10.23975/bjvr.2026.166408.1255

Abstract

Gallid alphaherpesvirus 2 (MDV-1) is the cause of the disease that causes T-cell lymphomas, nerve enlargement and visceral lesions in chickens. Despite the large-scale vaccination, the Marek diseases (MD) continue to cause devastating economic losses by lowering productivity, increasing mortality and the expensive control measures. The virus is known to have three serotypes: MDV-1 oncogenic serotype, and MDV-2 and MDV-3 non-pathogenic serotypes employed as vaccine strains. The virulence classifications in MDV-1 are mMDV, vMDV, vvMDV and vv+MDV. The Asian studies have discovered the existence of highly virulent field strains that have mutations in the Meq oncogene, which indicates that the viruses have evolved and are capable of escaping vaccines. The virus is not transmitted vertically, and the main way of transmission is inhalation of the feather dust of an infected bird. It is also very stable in the environment and continues to stay in poultry houses for a long time. Lesions can also present themselves in the nerves, liver, spleen, kidneys and eyes, usually resulting in paralysis or grey eye. Diagnosis is a combination of clinical, pathologic, and molecular diagnosis to include PCR of Meq, gL, and ICP4, as well as immunohistochemistry. Though vaccination has significantly lowered the impact of the diseases, there is the constant development of virulent strains that pose a threat to the effectiveness of the vaccines and the adverse effects of the vaccines on preventing the diseases. The report aims to discuss the latest scientific research on MDV in chickens. In conclusion, the continuous evolution of MDV-1, particularly the emergence of Meq variability associated with augmented virulence, continues to challenge the immunization proficiency of the current vaccination practices. The above advances, combined with the environment of the virus and its high capacity to cause horizontal infections, highlight. Also, the importance of applying better molecular surveillance and establishing more efficient vaccine platforms in order to effectively manage Marek's disease in chicken farms.

Keywords: MDV, Gallid alphaherpes virus, Epidemiology, Virus evolution.

Publisher: University of Basrah,
College of Veterinary Medicine, Basrah, Iraq

Introduction

Marek's disease (MD) is one of the most severe viral infections affecting chicken production worldwide. This contagious immuno-oncological disease costs the chicken industry. The disease is characterized by its ability to cause T-cell lymphomas and peripheral nerve hyperplasia, resulting in various neurological and neoplastic symptoms in infected birds (1). Estimated to surpass US$1 billion each year owing to increased mortality, reduced productivity, and costs related to vaccines and preventive measures (2). Since the 1970s, vaccination has been very helpful in managing the condition; however, the Marek's disease virus (MDV) is constantly mutating, making it more challenging to stop the disease from spreading (3). However, the virus has evolved under immunological pressure from repeated vaccination over several years (4). The development of hypervirulent MDV strains capable of circumventing the defences offered by conventional vaccines (5). The present study aims to review the most recent scientific data and studies on MDV in poultry, including the definition and etiology of the disease, clinical symptoms, transmission and epidemiology, diagnostic methods, prevention and control strategies, genetic evolution of the virus, immune escape mechanisms, research developments, and future recommendations for controlling this disease.

Viruses Classification and Serotypes in MD:

MDV has historically been categorized into three serotypes based on their antigenic properties. Currently known as Gallid alphaherpesvirus type 2 (GaAHV-2). Serotype 1 (MDV-1) is the only pathogenic and/or carcinogenic serotype for hens. Serotype 2 (MDV-2) is Gallid alphaherpesvirus type 3 (GaAHV-3). Also, non-pathogenic to hens. Serotype 3 (MDV-3) is also the Herpesvirus of turkeys (HVT) or Meleagrid alphaherpesvirus type 1 (MeAHV-1). Just serotype 1 (MDV-1) strains of the three serotypes are pathogenic and/or carcinogenic in hens (6). Based on their virulence and pathogenicity evaluated with HVT mono-vaccination or HVT+ SB-1 dual-vaccination, MDV-1 isolates have been split into four pathotypes: Low pathogenicity strains of the mildly pathogenic Marek's disease virus (mMDV).

Strains with intermediate virulence are associated with the virus causing violent Marek's disease (vMDV). Very virulent Marek's disease virus (vvMDV) is a highly pathogenic strain. Strains with extremely high pathogenicity (7). The viruses that cause ultra-virulent Marek's disease (vv+). Furthermore, some virulent variants—especially hypervirulent Marek's disease virus (HV-MDV) strains isolated from clinical cases in vaccinated chicken flocks in China—have lately been identified as HV-MDV variants based on different genetic evolutionary pathways shown in genetic phylogenetic trees built using the main oncogene meq or whole viral genome sequences (8).

Virus virulence evolution:

The virus has contributed to the evolution of new strains with greater capacity to maintain their specificity, which remains persistent and reliant on traditional methods (9). In a recent study in Egypt, phylogenetic analysis of the Meq gene in sequenced samples revealed that all marker virus isolates were associated with the highly virulent European Gallid herpesvirus 2 ATE and PC12/30, with high amino acid identity (AAI) ranging from 99.2% to 100% (10). The study also revealed that Egyptian strains contain specific mutations, allowing them to be grouped into two subgroups (A and B). The research also reported mutations specific to distinct strains, which contributed to the development of highly virulent strains by analyzing mutations in the Meq gene (11). A recent study carried out in China has revealed that strains of HV-MDV that are highly virulent are common in chicken flocks, and some of them have tremendously interfered with the immunity that is offered by classical HV-MDV. The pathogenicity of seven HV-MDV strains isolated from tumor-ridden chickens in China was analyzed and found to be pathogenic to chickens. Four isolates (SDCW01, HNXZ05, HNSQ05, and HNSQ01) were identified as highly virulent HV-MDV strains (12). This ongoing evolution in the virus's virulence poses a known challenge to current control strategies. Consequently, there is an urgent need for new, more effective approaches to address this challenge. Extensions in developing virus strains or variants to control the disease (13).

Transmission and Epidemiology:

Transmission: The virus is primarily transmitted by inhaling airborne dander from contaminated feather follicles. The virus can remain alive in these skin dander particles for several years in the surrounding environment. Interestingly, Marek's illness cannot be passed vertically through eggs. Confirms that although the hens are infected, the chicks hatch clear of the virus as well. Figure 1 shows the critical point of MDV transmission (14).

Epidemiology:
Among the most common viral infections affecting poultry worldwide is Marek's disease. Regular MD outbreaks have occurred in vaccinated chicken flocks, suggesting the emergence of novel viral strains capable of overcoming the defence provided by traditional vaccination, with considerable geographical differences (15). calculated the seroprevalence of MDV antibodies in the indigenous Tilili chicken breed in Ethiopia to be 74.6%. Nakesha (57.5%) had the lowest rate, while Banja (93.8%) had the highest (16). Also, MD was recorded in vaccinated flocks in Belgium. Their vaccination program was shown to have multiple contributory risk factors, including poor biosecurity, inadequate immunization, limited bird space, and high population density (17). The prevalence of MD in both commercial and backyard flocks in Bangladesh ranged from 15% to

45%. Some studies found that rearing system, stocking density, and biosecurity policies were substantially associated with infection rates, as shown in Figure 2 (5).

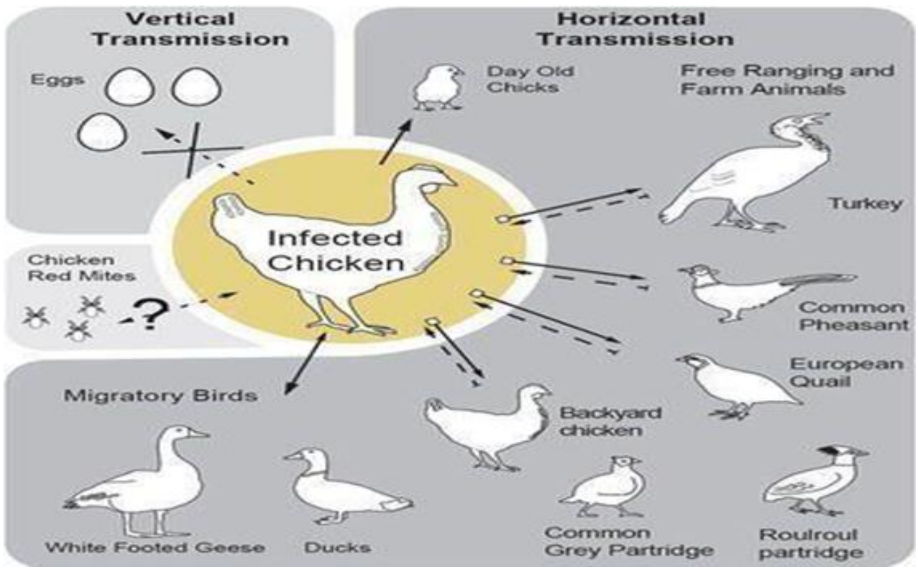

Figure (1): Vertical and horizontal transmission(14).

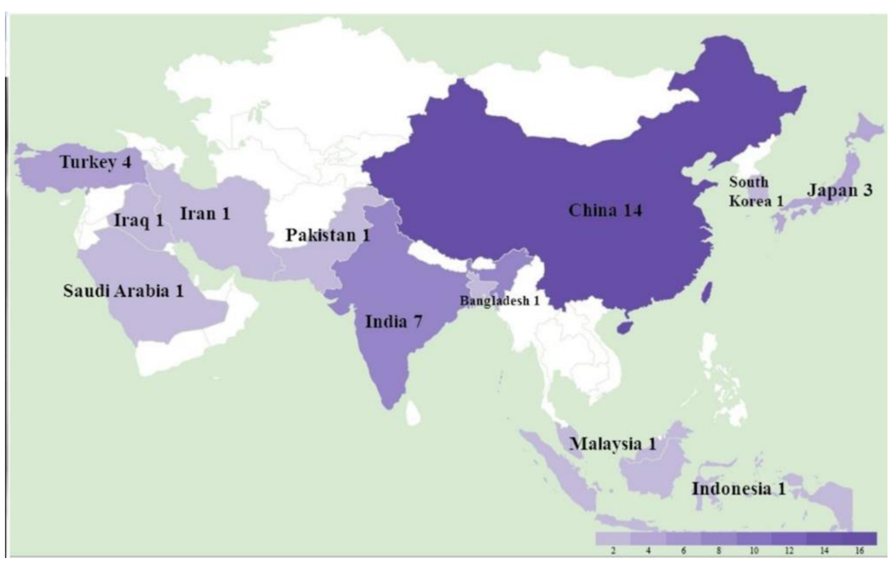

Figure 2. The study location and number of MD outbreaks in Asia between 2011 and 2021.(5)

Susceptibility and Resistance:

The immunological response induced by MDV varies among hosts, with different levels of resistance or susceptibility (18). Heterogeneity among chicken breeds is crucial for their susceptibility to the disease (19).

Previous research indicated that egg-laying hen breeds, especially Leghorn and light-colored breeds, are more vulnerable to the disease than other breeds (20). A genome-wide association study conducted by (21) to discover new genetic sites associated with MDV resistance. The data suggest that resistance to MD is polygenic, meaning it is controlled by multiple genes, each having a minor effect (22). Also (23) They discovered that immune cells from resistant chickens had elevated expression of key immune genes compared with those from susceptible hens, indicating an intrinsic immunological advantage.

Risk Factors Associated with Disease Spread:
Identified significant risk factors for MDV antibody positivity in indigenous chicken breeds in Ethiopia, including contact with other birds in the environment, sex, age group, and house hygiene. However, factors such as breed, production system, and farm type were not significantly associated with serological status (24). Identified risk factors for MDV transmission in chicken flocks in Nigeria, including open housing systems, lack of vaccination, contact with wild birds, and large flock sizes (25). The prevalence and characteristics of MD in native chickens in Ethiopia are influenced by age (older chickens are more susceptible to infection), season (a higher incidence during the dry season), and production system (mixed systems increase the risk) (26). Serological studies of MDV antibodies in native chickens in Tanzania showed that infection rates were associated with factors such as geographical location, housing system, and previous outbreak history in the area(27). Research conducted in the Basra province established the existence of Marek disease virus in both native and Brahma chickens that show paralysis and swelling of nerves and internal organs. Molecular identification of the virus was done by the detection of the Meq gene, and the infection rate of the native chicken population was recorded to be 25%(28). Genetic analysis of layer chickens in Basra found that the type of circulating MDV virus strains is very virulent (vvMDV). The virulence-related Meq gene was found to have mutations, which contribute to the explanation of the recurrent outbreaks of the disease despite the vaccination of the flocks (29). A research in Al-Najaf province recorded the initial report of an aggressive ocular tumor in a layer flock, where the condition was also reported to be highly promoted by the Marek disease virus infection. Clinical and histological results showed that the virus aggravated these distinctive and extreme lesions of the eye(30).

Clinical Forms of MD

Neurological Form: In the classic or neurological form of Marek's disease, the virus causes inflammation and the development of tumors in the nerves, spinal cord, and brainstem (31). These birds exhibit a range of behavioral abnormalities, most notably unilateral or bilateral limb weakness, which often causes the bird to lean to one side or lie on its back, rendering it unable to stand(32).

Other symptoms may include tremors in the head and neck, rapid breathing, and loss of balance (33). Birds with neurological diarrhea often die from starvation or dehydration due to a lack of access to food and water (34). They may also be trampled by other birds or suffer serious injuries to their bodies from lying down for too long. Recovery from this disease is sporadic (35), as shown in Figure 3 A&B (29).

Tumorous Form: The neoplastic form of Marek's disease is characterized by the appearance of lymphoid tumours in various internal organs, especially the liver, spleen, heart, kidneys, ovaries, testes, lungs, and skeletal muscle. These tumors appear as multiple white or grey nodular thickenings of varying sizes (36). Affected birds gradually lose weight, become emaciated, and may have respiratory or digestive problems, depending on the location of the tumour. Eventually, patients die from organ failure or extreme cachexia (37), as shown in Figure 5 A&B (29).

Ocular Form: MD can also cause changes in the eyes, especially the iris. These changes include discoloration or spots on the iris, anisocoria (unequal or irregular pupil sizes), and corneal clouding. Such changes can lead to visual impairment or complete blindness in affected birds, as shown in Figure 4 A(38).

Cutaneous Form: In some cases, MD can cause skin lesions and changes in the feather follicles. These often appear as enlargement and roughening of the feather follicles, causing the skin to appear raised or rough. Skin lesions may also occur as nodules or ulcers on various parts of the body, as shown in Figure 4 B(38).

Clinical and gross pathological findings:

Typically, MD begins with the observation of specific clinical signs, as in Figure 3, including leg or wing asymmetry, increased weight loss, emaciation, decreased egg production, and feather follicle growth. However, these symptoms are not sufficient to make a definitive diagnosis because they are similar to those of other diseases (39).

Necropsy of infected birds, as in figure 4, often reveals swelling of peripheral nerves, particularly the sciatic nerve and brachial plexus. These nerves differ from their normal bright white appearance, appearing grey or mottled. Internal organs, such as the liver, spleen, heart, kidneys, and ovaries, may also be enlarged and present with white-to-grey, tumour-like lesions of varying sizes (40). Changes to the iris may be observed in the ocular form of the disease, including uneven pigmentation, anisocoria (different pupil sizes), or corneal opacity. Feather follicles may appear larger and rougher in the skin, causing raised or rough skin (41).

Figure 3 Classical form of MD,(A); Unilateral paralysis of the legs and wings, (B) Complete spastic paralysis as a fan shape(29).

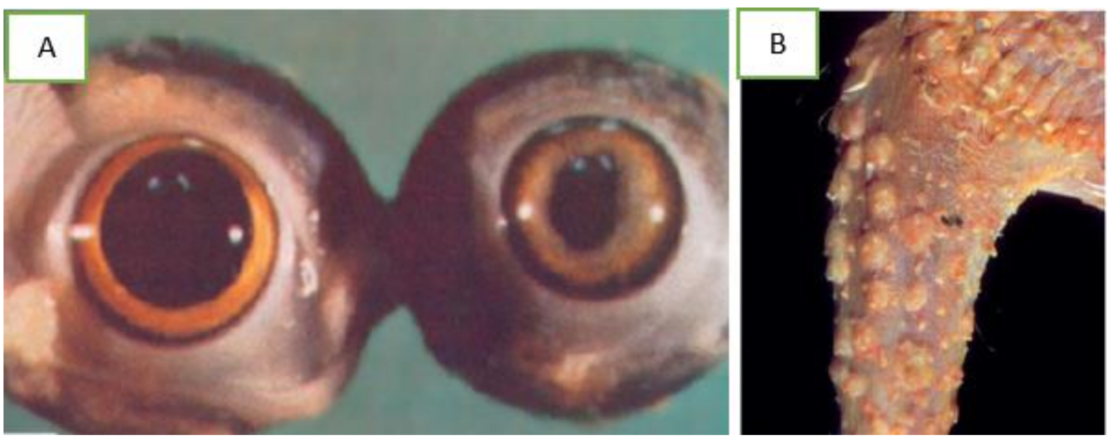

Figure 4 (A)Feather follicle leukotic tumors (skin lesion). (B) Ocular lesions of MD. It should be noted that the normal eye (left) has a well-defined pupil and pigmented iris. The right eye is infected because of the infiltration of mononuclear cells into the eye, which results in the discoloration of the iris and a highly irregular pupil(38).

Pathology changes:

Histopathology characteristic lesions are typically lymphocytes that infiltrate visceral organs and peripheral nerves, and also contain abnormal lymphocytes that resemble tumor cells (42). The typical architecture of the affected nerves is disturbed, and lymphoid infiltrates are visible between nerve fibers. In damaged visceral organs, abnormal lymphocyte foci often replace standard tissue architecture (43). Immunohistochemical techniques can be used to detect viral antigens in infected tissues, which helps corroborate the diagnosis and differentiate MD from other conditions that may produce analogous lesions (6).

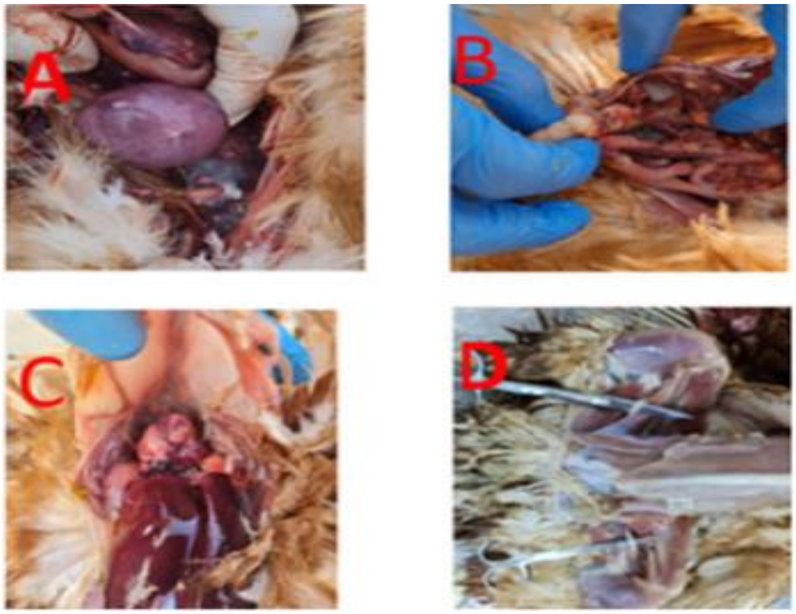

Figure 5: Macroscopic lesions in sick chickens (A) Splenomegaly with a nodular tumour. (B) Changes in the intestines, such as growth, thickness, and nodular tumours. (C) The heart had tumours in the cardiac muscle and was swollen. (D) The sciatic nerve is swollen, congested, and has oedema. (29).

Diagnosis of Marek's Disease in Poultry

To confirm infection and determine the viral burden in tissues, a combination of clinical, pathological, histological, and molecular approaches is used to diagnose Marek's disease (MD) in birds (40). Birds infected with MD may exhibit common clinical signs, such as uneven paralysis of the limbs or wings, gradual weight loss, and overall weakness. These signals are generally the first things that make people think they could have MD. The presence of lymphoid tumors in peripheral nerves and visceral organs, such as the liver, spleen, kidneys, and gonads, as observed during necropsy, substantially corroborates the clinical diagnosis (44). The condition can be confirmed by diagnostic veterinary laboratory tests (tissue biopsies), which normally show typical lymphocytic lesions, like pleomorphic lymphoid cells attacking nerves or organs. Along with the usual methods of diagnosis, molecular methods also play a vital role in the identification of the disease and the type or strain of the virus. The effective identification of MDV DNA through PCR in certain affected samples was carried out in a recent study cited in (45). In addition, the differentiation and virulence characterization of MDV strains was realized through the sequencing of Meq, gL and ICP4 genes since these genes are known to differentiate MDV strains and define their virulence. All these diagnostic methods, including gross and microscopic analyses, as well as the advanced molecular tests, are necessary to make a proper pathogenetic diagnosis and differentiate MD and other neoplastic or neurologic poultry disorders (4).

Differential Diagnosis

Marek's disease must be differentiated from several other diseases that present with similar clinical signs, such as:

Avian Leukosis (ALV):

Avian leukosis is another neoplastic disease in poultry caused by the avian leukosis virus (ALV). It can lead to tumour development in internal organs, which may resemble those caused by MD. However, ALV typically does not produce lesions in peripheral nerves, and symptoms usually appear at an older age than in Marek's disease (46).

Reticuloendotheliosis Virus (REV):

Another virus that can develop tumours in birds, resembling those observed in MD. Nonetheless, the location and histological features of tumours induced by REV are distinct from those associated with MDV (47).

Other Neurological Diseases:

Marek disease needs to be distinguished from other similar diseases in chicken, like Newcastle disease, avian encephalomyelitis, vitamin E deficiency, thiamine (vitamin B1) deficiency and lead or arsenic poisoning. Case history, clinical manifestations, and laboratory analysis can be combined to achieve differentiation (48).

Impact of the Virus on Immune Cells:

The four major stages in the virus life cycle were:

1. First Infection Phase: The virus enters the respiratory tract and infects the immune system cells, such as dendritic and macrophages.

2. Cellular Replication Stage: The virus multiplies in the immune cells, especially the B and T lymphocytes, killing them and causing immunosuppression.

3. Latency Stage: The virus becomes latent in T cells, whereby it inoculates its DNA into the host cell genome.

4. Oncogenic Stage: The virus induces the T lymphocytes to become malignant cells, which in turn become lymphomas in other organs and tissues (32).

At each of the stages, MDV escapes the immune cells in different ways:

• Killing of Immune Cells: The virus kills B and T lymphocytes, causing the suppression of humoral and cellular immunity.

• Immune Cell Function Modulation: MDV modifies the functions of the infected immune cells so that they are unable to respond to other pathogens.

. Tumorigenesis: MDV causes the transformation of T lymphocytes into cancer cells, which causes the development of lymphoma.

• Interferon Suppression: MDV suppresses interferon, which is a major protein in innate antiviral response (49).

Host Immune Responses:

The infection of MDV induces a full-fledged host immune response that comprises both new and adaptive immunity, including cytokine release, immune cell recruitment, and distorted immune-related gene expression (22). The degree of the immune reaction differs widely between hosts having diverse susceptibility/resistance to Marek disease. Transcriptomic results of bone marrow-derived macrophages and dendritic cells of susceptible and resistant chickens after MDV infection found a greater viral load and immune activation in susceptible birds (34). The immune genes were more expressed in resistant chicken innate immune cells versus the susceptible chickens, indicating that an inherent superiority in immunity exists (40).

Relationship Between Immunity and Genetic Resistance:

There is a close relationship between the host immune response and genetic resistance to Marek's disease. The susceptibility and clinical outcome of strains of chicken depend on differences in genetic variations in immune response. Light strains of egg-layers, such as Leghorn, are more vulnerable to MD compared to broiler strains. MD is very sensitive to silkies, in particular (2). Innate immune response to MD is associated with resistance to MD in the initial phases of infection. The resistant chickens have been observed to have increased expression of essential immune genes in the innate immune cells, which is a demonstration of an innate immune advantage (8). Although genetically different chickens are resistant, the development of genetically resistant lines of chicken has taken decades and was not completely achieved (16). The relationship between immunity and genetic resistance to MDV may be understood to aid in designing new prevention and control measures, including breeding of more resistant chicken strains and designing new vaccines that stimulate specific immunity response associated with resistance (27).

Preventive Strategies:

The MD prevention is based on a number of strategies, the most important of which is vaccination, as well as other prevention measures, including the use of biosecurity and genetic resistance breeding. Because of the absence of effective cures to MD, prevention is the major approach to managing MD in poultry flocks (45). The main measure of the prevention and control of MD is vaccination, with the additional measures of stringent hygiene to reduce or postpone exposure and genetic selection in favor of genetic resistance (5). The surest way of preventing clinical disease is to vaccinate newly hatched chicks and the birds have to be vaccinated before being exposed to the virus (48).

Along with the vaccination, other preventive measures are:

1. Biosecurity: It involves taking tough measures which will enforce biosecurity in the farm to ensure that viruses do not enter the farm and that they do not spread among flocks. Incorporates the cleaning and disinfection of poultry houses between production phases, the efforts of managing the movement of people and material, wearing special clothing and shoes, and the implementation of limited entry measures (41).

2. Isolation: It is used to isolate new chicks from older birds to avoid infection. The vaccination should be followed by a minimum of 47 days of complete isolation of the chicks before the vaccine works. A different caretaker should deal with them, and they should be separated from other birds (33).

3. Genetic resistance breeding: Some breeding programs are working to increase the genetic resistance of poultry to MD, even though breeding genetically resistant lines is a decades-long process that has not been entirely successful (45).

4. Environmental control: This is the enhancement of conditions in the rearing area to minimize stress and ensure the health of birds. Provides appropriate ventilation, minimizing congestion, ensuring clean litter, and eating a balanced diet (42).

Types of Vaccines Used:

Several vaccine types are available to control MD, each with varying efficacy and application methods. The most commonly used vaccines include:

1. **Turkey Herpesvirus (HVT)**: A live vaccine derived from the FC-126 strain of *Meleagrid alphaherpesvirus 1*, a naturally non-pathogenic virus. It is widely used due to its ease of production and application, but its efficacy is limited against highly virulent and hypervirulent Marek's disease virus strains (49).

2. **SB-1 or 301B/1**: Live vaccines derived from naturally non-pathogenic strains of *Gallid alphaherpesvirus 3*. These are often used in bivalently combined with HVT for enhanced protection against virulent MDV strains (10).
3. **CVI988/Rispens**: A live attenuated vaccine from the CVI988 strain of *Gallid alphaherpesvirus 2*. It is considered the most effective vaccine against very virulent and hypervirulent strains and is widely used in laying and breeder flocks (20).
4. **814**: Another serotype one vaccine used in countries like China (1).
5. **Recombinant vaccines**: HVT is now commonly used as a vector, incorporating genes from other poultry viruses such as Newcastle disease virus, infectious bursal disease virus, H5 avian influenza virus, and infectious laryngotracheitis virus. These protect against both MD and the inserted virus (13).
6. **Bivalent vaccines**: These combine HVT with either SB-1 or 301B/1 to offer additional protection against virulent MDV challenges (19).

Vaccination Methods and Timing:

Marek's disease vaccines are administered either at hatch or in Ovo to embryos on the 18th day of incubation. In-Ovo vaccination is now widely used in commercial broilers due to lower labor costs and greater accuracy (10). The preferred vaccination method is for hatcheries to vaccinate the chicks at the hatchery level. If vaccinated on-site, the vaccine must be handled strictly according to the label instructions—this includes the thawing rate and temperature, as well as the requirement to use the vaccine within 1–2 hours after reconstitution (30). The vaccine must be thrown away after two hours once it has been reconstituted. It is not something that can be kept in a store because it becomes ineffective. Its usage requires it to be injected (usually subcutaneously). The problem with petite chicks is that it is beneficial to have one person holding the chicks and another person to inject them (48). The other important action to take to achieve maximum protection of the vaccine is that of cleaning and disinfecting the brooder and putting down chicks. Also, any feather dander and debris of older birds should be removed in the vicinity of chicks at least one week (48).

Vaccine Effectiveness:

The effectiveness of the Marek disease vaccines is determined by a number of factors that include the nature of the vaccine, the mode of administration, the time of the vaccination, the virulence of the infecting viral strain and the immunity of the birds. Cell-associated vaccines tend to be more effective than cell-free vaccines since they are not readily inactivated by maternal antibodies. In most circumstances, the vaccine efficacy is more than 90 per cent (49). But the development of highly virulent strains of MDV has somewhat diminished the effectiveness of traditional vaccines. A Chinese study conducted recently assessed the protective immunity of four commercial vaccines: CVI988, HVT, CVI988+HVT and 814 against the hypervirulent strain, SDCW01. Surprisingly, the levels of protection indexes (PIs) 67 days after the challenge were just 46.2, 38.5, 50 and 28 percent, respectively. Moreover, the tumour incidence of birds vaccinated against CVI988 or HVT with a one-dose vaccine reached cumulative incidences of 7.7% and 11.5 primarily, respectively (11). Such findings are important to note that hypervirulent MDV strains that exist in China can easily circumvent the immune protection of the classical MD vaccines, highlighting the fact that it is high time to come up with more effective vaccines that can be used to combat hypervirulent MDV strains to ensure long-term management of the disease (50).

Other Preventive Measures:

Besides vaccination, other steps would contribute to the decrease of the Marek disease spread in poultry flocks:

1. All-in/All-out system: This is a system whereby birds of similar age are kept in a single house, and all the birds are cleared off, and a new batch of birds is introduced after cleaning and disinfecting the house (50).

2. Stress alleviation: Stress has the potential to undermine the immune system of the birds, leaving them more vulnerable to infection. Poor ventilation, overcrowding, abrupt changes in temperature and noise are examples of stressors that should be minimised (43).

3. Proper nutrition: Feeding of a balanced diet with all the necessary nutrients to increase the health of the birds and to boost the immune system (23).

4. Insect control: Insects can be controlled, such as litter beetles, which can spread the virus between flocks (29).

5. Culling of infected birds: Disposing of birds with clinical symptoms of Marek disease to limit particles of infection within the flock (11).

6. Sanitization of the environment: Cleaning and disinfection of poultry houses and equipment regularly to decrease the amount of viruses in the feather dander and surfaces (34).

7. Misconception correction: Do not practice mixing turkeys and chicks to prevent MD, not vaccinating birds to stimulate natural resistance, and vaccinating part of a flock in the hope that the rest of the flock would be covered by the vaccine spreading- this is ineffective and counter-productive (13). Since the practice of vaccination started, the MD losses have decreased greatly in the broiler and the layer flocks. Nevertheless, the disease remains a severe issue in single flocks or in a particular geographic zone. Among numerous postulated reasons that could be used to explain such consistent losses, exposure to very virulent strains at an early age seems to have played a major role (51).

Conclusion

The continuous evolution of MDV-1, particularly the emergence of Meq variability associated with augmented virulence, continues to challenge the immunization proficiency of the current vaccination practices. The above advances, combined with the environment of the virus and its high capacity to cause horizontal infections, highlight. Also, the importance of applying better molecular surveillance and establishing more efficient vaccine platforms in order to effectively manage Marek's disease in chicken farms.

Conflicts of interest

The authors declare that there is no conflict of interest.

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مراجعة شاملة لمرض ماريك في الدواجن

سارة سالم محمد[1]، بدور محمد لطيف[2]، عصام عزيز خليفة[2]، محمد طاهر عبد الرزاق عبد الرسول[2].

1-فرع الصحة العامة البيطرية; كلية الطب البيطري، جامعة البصرة، البصرة، العراق .
2-فرع الأمراض وأمراض الدواجن، كلية الطب البيطري، جامعة البصرة، العراق.

الخلاصة

يُعدّ فيروس الهربس ألفا للدجاج 2 (MDV-1) المسـبب لمرض ماريك، الذي يُسـبب أورام الغدد الليمفاوية التائية، وتضخم الأعصـاب، وآفات الأحشاء في الدجاج. وعلى الرغم من حملات التطعيم واسعة النطاق، لا يزال مرض ماريك يُسـبب خسـائر اقتصـادية فادحة نتيجة انخفاض الإنتاجية، وزيادة معدل النفوق، وارتفاع تكلفة إجراءات المكافحة. من المعروف أن للفيروس ثلاثة أنماط مصـلية: النمط المصـلي المسـبب للسـرطانMDV-1 ، والنمطان المصـليان غير الممرضـين MDV-2 وMDV-3، واللذان يُستخدمان كسـلالات لقاح. تُصـنّف ضـراوة مرض مارك الى منخفضـة ومتوسطة وشديدة الضراوة .وقد كشفت الدراسات الآسيوية عن وجود سلالات ميدانية شديدة الضراوة تحمل طفرات في جين Meq المسبب للسرطان، مما يُشير إلى أن الفيروسات قد تطورت وأصبحت قادرة على الإفلات من اللقاحات. لا ينتقل الفيروس عموديًا، والطريقة الرئيسية لانتقاله هي استنشاق غبار ريش الطائر المصـاب. يتميز هذا الفيروس بثباته العالي في البيئة، حيث يبقى في حظائر الدواجن لفترات طويلة. وقد تظهر آفاته في الأعصـاب والكبد والطحال والكليتين والعينين، مما يؤدي عادةً إلى الشلل أو ما يُعرف بالعين الرمادية. يعتمد التشخيص على مزيج من الفحوصات السريرية والباثولوجية والجزيئية، بما في ذلك تفاعل البوليميراز المتسلسل (PCR) لجينات Meq و gL و ICP4، بالإضافة إلى الفحص المناعي النسيجي. ورغم أن التطعيم قد خفّض بشكل ملحوظ من تأثير المرض، إلا أن ظهور سلالات ضـارية باسـتمرار يُهدد فعالية اللقاحات وآثارها الجانبية في الوقاية من المرض. يهدف هذا البحث إلى مناقشـة أحدث الأبحاث العلمية حول فيروس ماريك في الدجاج. وخلاصـةً، فإن التطور المستمر لفيروس ماريك-1، ولا سيما ظهور تنوع جين Meqالمرتبط بزيادة الضـراوة، يُشـكل تحديًا مسـتمرًا لكفاءة التحصـين في ممارسـات التطعيم الحالية. وتُبرز هذه التطورات، إلى جانب بيئة الفيروس وقدرته العالية على إحداث عدوى أفقية، أهمية هذا الفيروس. كما أن من المهم تطبيق مراقبة جزيئية أفضـل وإنشـاء منصـات لقاح أكثر كفاءة من أجل الإدارة الفعالة طويلة الأجل لمرض ماريك في مزارع الدجاج.

الكلمات المفتاحية: مرض مارك، فيروس ألفا هربس الدجاج، علم الأوبئة، تطور الفيروس.