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Review on avian encephalomyelitis

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Abstract: Summary: This paper is mainly aimed to review the avian encephalomyelitis. Avian Encephalomyelitis (AE) is an economic concern to poultry industry as it causes economic loss by declining egg production in laying hens as well as egg hatchability . This disease is very common in commercial aviculture which is caused by genus *Tremovirus,* family of Picornavirridae. It is essentially an enteric infection which can be acquired horizontally between birds by oral ingestion and vertically from infected breeding females through the egg to progeny. It affects mainly young chicken and it is characterized by neurological signs like ataxia and rapid tremors of the head and neck giving rise to the former name of ''Epidemic Tremor '' but no gross lesions are seen in the brain of birds. Both morbidity and mortality can be 50% sometimes reach up to 60% and Its Vertical infection followed by horizontal infection causes a characteristic biphasic mortality pattern. Protection against this virus can be successfully achieved by using the appropriate vaccine strains. Vaccinated birds are life time immune and recovered do not spread the virus. Wild birds are not considered as reservoir of the virus which poses no threat to the human and this disease is not considered as zoonotic disease. Therefore it can be concluded that AE is economically important viral disease which can hinders the poultry production, so it is recommended to control the disease by proper vaccination of parent stock.
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1. Introduction

Avian encephalomyelitis virus (AEV) is a widespread infectious diseases with preference of central nervous system and other parenchymatous organs of birds. This disease is very common in poultry industry with significant economic impacts. It's huge economic lost was before introduction of vaccine in the 1960s (Cal neck, 2008). Over 200 species of birds are susceptible to the infection but it mainly affects chicken, turkeys, Japanese quail and pheasants (Sentıes *et al.,* 2015). Turkeys are less susceptible to natural infection and generally, develop a milder clinical disease than chicken. Duck and pigeon are susceptible to experimental infection and wild birds are not considered as reservoir of the virus which poses no threat to the human (Abdul Aziz, 2013).

Avian encephalomyelitis virus was first reported in New England region of the USA in 1930 in two weeks old chicken and this syndrome was termed as "Epidemic Tremor, New England disease and Star-gazing Syndrome''(Jones,1932). OIE classifies this disease as list C disease. It is essentially an enteric infection and it is transmitted horizontally between birds by oral ingestion and vertically from infected breeding females or parent stock through the egg to the chicken. Horizontally infected chicken usually show clinical signs at 2–4 weeks of age and vertically infected chicken commonly show clinical signs of avian encephalomyelitis during the first week (1-7 days) after hatching. Both morbidity and mortality may exceed 50%. Vertical infection followed by horizontal infection causes a characteristic biphasic mortality pattern (Freitas and Back, 2015).

Protection against this virus can be successfully achieved by using the appropriate vaccine strains (i.e. strains that have not been embryo adapted) and administration of protocols to the breeders so as to transfer maternal immunity to their progeny (Francisco, 2014). The presence of neutralizing antibodies in the serum of chicken has long been recognized as a major determinant of resistance to infection by avian encephalomyelitis virus (Tannock and Shafren, 1994). It was described that maternal antibodies in the chicken yolk sac are fully absorbed between the second and third week post-hatch (Szabo, 2012). Therefore, the detection of antibodies against AEV in a flock of chicken with 21 days of age or older indicates field infection. The best way to prevent and to control the disease is vaccinating breeder flocks during their grow-out period, preventing the infection

of breeders during the egg production period and thereby, and virus transmission to the progeny. Moreover, breeder and parent stock vaccination provides good immunization of progeny, ensuring protection against virus infection during the rest weeks of life, when they are most susceptible to the virus (Roy *et al,* 2009).

Hence, this review is under taken with the following objective:

- To review avian encephalomyelitis.

2. Etiology

Avian Encephalomyelitis (AE) is caused by *Avian encephalomyelitis-like virus 1* (AEV), which is a single-stranded RNA (ssRNA) virus belonging to the *Picornaviridae* family. It was formerly classified as an *Entero-virus. Avian encephalomyelitis-like virus 1* is not related antigenically to other avian *Picoronavirus s* that is why it has been referred to in the past as an *Enteroviruses or Enterovirus-like viruses.* Recent characterization of AEV genome or protein homologies indicates that it is more closely related to *Hepatitis A virus* than to *Enteroviruses* and it had been provisionally classified as a tentative species in the genus *Hepatovirus* (Van regenmortel *et al.,* 2000) in the family *Picornaviridae*. Recently AEV has now been reclassified as a unique virus and placed as the sole member of the *Tremorvirus* genus (King *et al.,* 2012). Recent characterization of AEV genome indicates that it is more closely related to *Hepatitis A virus* than to *Entero-virus* and it had been professionally classified as a tentative species in the genus *Hepatovirus* (Van regenmortel *et al.,* 2000) in the family Picornaviridae. Recently, AEV has now been reclassified as a unique virus and placed as the sole member of the *Tremorvirus* genus (King *et al.,* 2012).

The virus has a non-enveloped capsid with icosahedral symmetry and is approximately 26 nm in diameter and has buoyant density of 1.31 g/ml in caesium chloride and it is stable at pH 3.0 and has a buoyant density of 1.31 to 1.33 g cm^{-3} *(ICTV, 2019). This virus is resistant to chloroform, acid media, enzyme trypsin and pepsin as well as environmental condition. Magnesium* cation were shown to stabilize preparations of the virus against heat effect (56°C, 1 h), hence, this enables it to be resistant environmental condition. However, incubation with rib nuclease and treatment at high pH (9.0 to 10.0) reduced its titer by 1007 to 102 EID50/ml (Go sting *et al.,* 1980) and it can be inactivated by single fumigation cycle by formaldehyde (Francisco, 2014). The laboratory protype strain is called Van Roekele strain (Van Roekel, 1938).

3. Pathogenesis

The strains of avian encephalomyelitis virus (AEV) are antigenically uniform, but there is variation in tropism and virulence, which can be enterotropic with horizontal transmission or neurotropic with vertical transmission (Martins and Silva, 2009). The infection by the AEV occurs mainly through the fecal-oral route and its replication takes place in the intestinal epithelial cells. Following the oral ingestion of virus, it enters the bloodstream via Payer's patches and lymphatic vessels, reaching other organs, such as the pancreas, heart, proventriculus, gizzard and the central nervous system (Tannock and Shafren, 1994) resulting clinical signs and symptoms in susceptible chicken that usually aged between 1 and 30 days (Calnek *et al*., 1997).

4. Transmission

The transmission of the virus is both vertical and horizontal. Vertical transmission tends to cause higher losses because day-old chicken are affected. This happens when breeders with no immunity are infected during the egg production period and transmit the virus to progeny. This virus can also transmitted horizontally by fecal-oral route through ingestion of feed, litter, or contaminated water *(Back, 2010*). The virus can exist in droppings for more than 4 weeks in this case, the disease is less severe and tends to affect older chickens. Horizontally infected chicks usually show clinical signs at 2–4 weeks of age; thus, clinical disease progresses through the flock from the first few weeks, and the episode is usually over by the time the flock is ~4 weeks old. After oral ingestion the virus can be first detected in pancreas, liver and spleen within 3 days and then CNS and other enteric tissues such as proventriclus, gizzard, small intestine and caecum (Tannock, 1994).

Infected eggs laid during the period of viremia, usually lasts 1–2 week. After 4 weeks of age, chicken are resistant to disease but not to infection, an exception occurs occasionally in older chicken after vaccination with chick-embryo–propagated vaccines in which the vaccine virus inadvertently becomes embryo-adapted during production of the vaccine. Chick-embryo-adapted strains are highly neurotropic and can cause clinical disease after parenteral administration. Affected birds exhibit typical neurologic signs like those seen in younger chicken (Calnek, 2008).

AE is not considered as zoonotic disease (Gough, R.E. and Mcnulty, M.S. 2007). Recovered birds are live immune and do not spread the virus, where as in older birds infection is subclinical, resulting in declines in egg production in laying chicken, there is a sudden 5%–10% drop in egg production, which usually lasts for <2 weeks and hatchability may drop as much as 5% due to late embryonic mortality followed by a return to normal production and hatchability (Cheville, 1970). This viral disease can

produce no deterioration in egg shell quality (Calnek *et al.,* 1960)

5. Clinical findings

The main clinical signs of avian encephalomyelitis as described in the figure below are ataxia and leg weakness that varies from inclination to sit on hocks, falling on their backs and laying with one side to unilateral and bilateral paralysis. Fine tremors of the head and neck are evident in some birds and are characteristic of the disease, hence are responsible for the common name, Epidemic Tremors (Konstantinos, C. K, 2015).

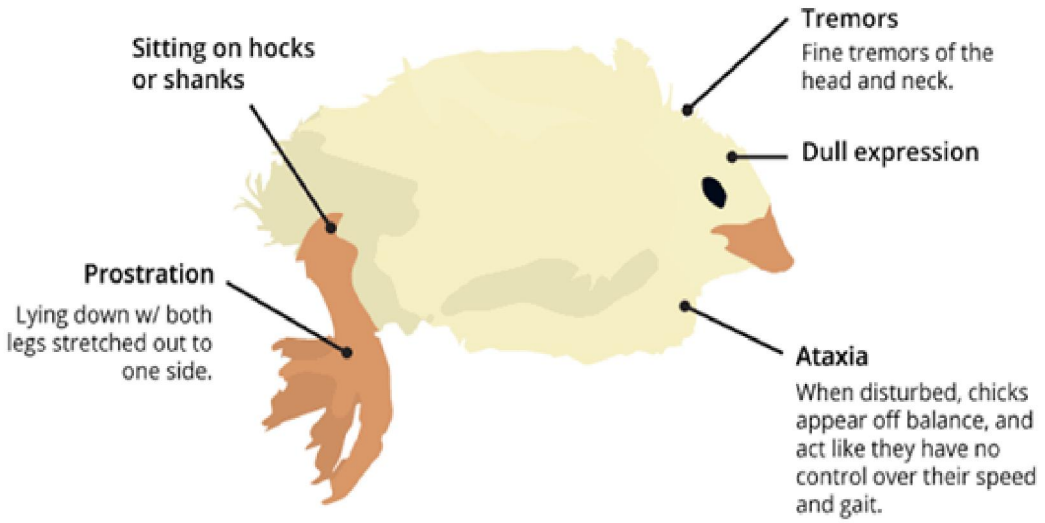

Figure 1: Diagramatic presentation of clinical sign of AE

Figure 2: Neurological sign in day old chicken affected through vertical transmitted AE

Figure 3: Two-week-old broiler presenting neurological signs of avian encephalomyelitis virus infection (Freitas and Back, 2015).

Figure 4: Neurological sign in old laying hens (Rocha, 2019).

Figure 5: Opacity of eye lenses (cataracts) in old chicken after infection (Rocha, 2019).

6. Necropsy findings

No gross lesions are seen in the brain of birds infected with avian encephalomyelitis. But gray to white foci may be visible on surfaces of the pancreases and hemorrhage in proventriclus, gizzard and crop can also be appreciated.

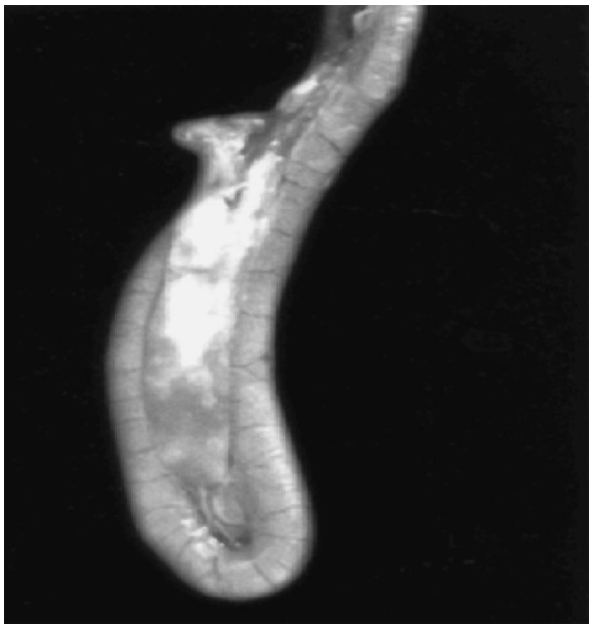

Figure 6: Large white–greyish foci extended across a lobe of the pancreas (Nihat, T. and Gunay, A., 2010).

7. Diagnosis

Presumptive diagnosis is based on clinical signs presented in chicks. However, the disease is only definitively confirmed by laboratory examination. Among the available techniques, histopathology is considered of essential, and provides the definitive diagnosis of the disease, as demonstrated by the pathognomonic lesions in various tissues, such as the central nervous system, pancreas, proventriculus, gizzard, and heart (Swayne, 2008). History-pathological findings observed in the central nervous system were characterized mainly by neuronal chromatolysis and necrosis, gliosis, neuronophagia and perivascular cuffs, indicating virus encephalitis, suggestive of AE, these lesions usually occur in the cerebellum, brainstem, spinal cord (Alexander, 2003). Central chromatolysis of neurons is characterized by rounding of the cell contour and displacement of the Nissl granules to the cell periphery. Dorsal root ganglia may present multifocal nodular collections of lymphocytes (Villarreal, 2009). Furthermore, serological tests may also contribute for the diagnosis of the disease, as well as virus isolation (Calnek, 2008). The presence of neutralizing antibodies in the serum of chicken has long been recognized as a major determinant of resistance to infection by avian encephalomyelitis virus (Tannock and Shafren, 1994). The maternal antibodies in the chick yolk sac are fully absorbed between the second and third week post-hatch therefore, the detection of antibodies against AEV in a flock of chickens with 21 days of age or older indicates field infection (Szabo, 2012).

8. Prevention and control

There is no treatment for avian encephalomyelitis (Silva, 2009). But it can be Controlled the disease through vaccination (Salma, 2015). To prevent infection of flock's hatcheries should only accept hatching eggs from immune breeder flocks. Life time immunity is acquired through vaccination or recovery from the disease. The presence of neutralizing antibodies in the serum of chickens has long been recognized as a major determinant of resistance to infection by avian encephalomyelitis virus (Tannock and Shafren, 1994).

Breeder pullets should be vaccinated between 8-15 weeks of age. It is also recommended for replacement egg layer pullets to be vaccinated at this age to prevent a temporary drop in egg production and to minimize the impact of the disease in an infected flock, remove all affected birds and provide good nursing, including fresh food and water, to the remaining birds. Affected birds should be killed and incinerated. Vaccination of meat and egg-type commercial breeders during the rearing phase is the most effective and economically viable tool to control the disease, since the humoral immunity transmitted to the progeny ensures the protection of the chicken against the challenges of the field (Martins and Silva, 2009).

AE live vaccine can be provided through drinking water and aerosol usually administered combined with fowl pox vaccine. Alternatively, it is given to chickens by wing-web. When using wing-web puncture method, it is recommended to verify that reaction occur at the site of inoculation 7-8 days post vaccination ''Take'' (Francisco, 2014). It was also worth noting that mixing of AE and FP viruses in a single preparation did not adversely affect or interfere with the immunogenic potential of any of the components of the vaccine preparation (Sarma, 2019).

9. Conclusion and recommendations

AE is an economically important viral disease of young birds which can imposes impacts on poultry production. Even though AE has no treatment it can be controlled through wise prevention measure. As the review and its conclusion reached accordingly the following recommendations are forwarded:

- Wise bio security measure should be practiced (i.e. fumigation of the house)

- Vaccinating parent stock
- Affected birds should be removed

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