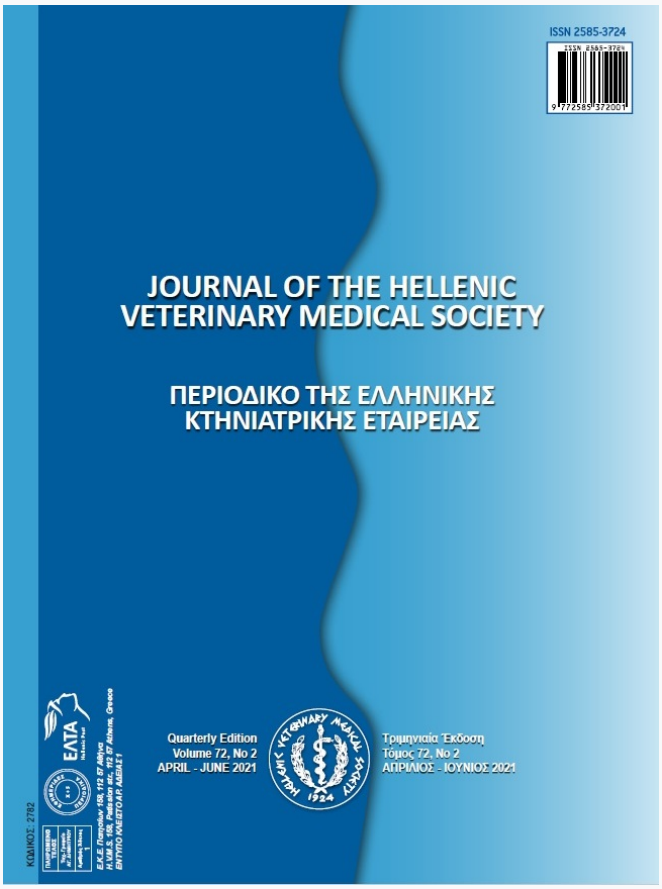
Journal of the Hellenic Veterinary Medical Society

Vol 72, No 2 (2021)



Concurrent Occurrence of Avian Encephalomyelitis and Vitamin A Deficiency In a Commercial Broiler Chicken Flock

BM ABBASABADI, S SEIFI, H GOLSHAHI

doi: 10.12681/jhvms.28027

To cite this article:

ABBASABADI B., SEIFI, S., & GOLSHAHI, H. (2021). Concurrent Occurrence of Avian Encephalomyelitis and Vitamin A Deficiency In a Commercial Broiler Chicken Flock. *Journal of the Hellenic Veterinary Medical Society*, *72*(2), 2989–2992. https://doi.org/10.12681/jhvms.28027

J HELLENIC VET MED SOC 2021, 72(2): 2989-2992
ΠΕΚΕ 2021, 72(2): 2989-2992

Case report
Κλινικό περιστατικό

Concurrent Occurrence of Avian Encephalomyelitis and Vitamin A Deficiency In a Commercial Broiler Chicken Flock

B.M. Abbasabadi[1*], S. Seifi[2], H. Golshahi[3]

[1] *Department of Basic Sciences, Faculty of Veterinary Medicine, Amol University of Special Modern Technologies, Amol, Iran*

[2] *Department of Clinical Sciences, Faculty of Veterinary Medicine, Amol University of Special Modern Technologies, Amol, Iran*

[3] *Nanobiotechnology Research Center, Avicenna Research Institute, ACECR, Tehran, Iran*

ABSTRACT: Avian encephalomyelitis (AE) is accounted for a common infectious disease in poultry, which particularly targets the central nervous system and various parenchymatous organs of chickens.Vitamin A supplementation is considered as a viable option to strengthen the immune system in neonates. Vitamin A deficiency in broilers decreases the ability to synthesize specific antibodies and weakens the lymphocyte proliferation response leading to epithelial metaplasia. In the present study, the effect of vitamin A deficiency on the occurrence of AE in a day old commercial broiler chicken flock was evaluated.Twenty, Two-Day-old paralyzed chickens, had been referred to the clinic of Veterinary School.The chicks were selected from a broiler flock with 13,000 birds purchased from a broiler breeder flock, all vaccinated against the AE virus. After examination, ataxia, trembling, lateral recumbency, and incoordination were observed in the chickens. The chickens were euthanized, and the tissue samples from their brain, heart, and proventriculus were then collected for microscopic evaluation. According to microscopic studies, non-supportive encephalomyelitis was noted in the cerebellum and cerebrum. In the myocardium, fiber degeneration and lymphocytic aggregates between the muscle fibers were evidenced.The proventriculus in chickens revealed hyperplastic and thickened mucosa, and there was squamous metaplasia for some of the mucosal glands. Furthermore, some multifocal aggregations of lymphocytes were observed in the tunica muscularis layer of the proventriculus.Our findings showed that vitamin A deficiency in a broiler breeder farm might be had a significant effect on the occurrence of AE in their hatched chicks. This study, to the best of the authors' knowledge, is the first report of concurrent occurrence of avian encephalomyelitis and vitamin A deficiency in a Two-Day-old commercial broiler chicken flock in north of Iran.

***Keywords*:** Ataxia, brain, metaplasia, neuronal degeneration, proventriculus

Corresponding Author:
Behrokh Marzban Abbasabadi, Department of Basic Sciences, Faculty of Veterinary Medicine, Amol University of Special Modern Technologies, P. O. Box: 46168-49767, Amol. Iran
E-mail address: behrokh_ma@yahoo.com

Date of initial submission: 09-06-2020
Date of revised submission: 23-10-2020
Date of acceptance: 20-11-2020

CASE HISTORY

Avian encephalomyelitis (AE), is recognized as a worldwide and economically important viral disease affecting mainly chickens, turkeys, quail, and pheasants caused by *Tremovirus A* of the *Picornaviridae* family (Sentíes-Cué et al., 2016).The outbreaks of the disease were first reported in the New England region of the USA in 1930. It is known as "epidemic tremor" since there are unusual tremors and vibrations of the head or neck as result of the disease (Koutoulis et al., 2015). Although the virus targets chickens of all ages, the nervous symptoms are only manifested in young chicks, between one to five weeks of age (Sentíes-Cué et al., 2016). In older chickens, infection is usually subclinical, resulting in a decline in egg production and hatchability (Sentíes-Cué et al., 2016). The virus may be spread by the horizontal (oral-faecal) and vertical routes. Vertically transmitted virus to the offspring of a breeder flock infected during egg production leading to an outbreak occurrence (Back, 2015).

Vitamin A is considered as an essential micronutrient throughout the life cycle of broilers. The deficiency of this vitamin decreases the ability to synthesize specific antibodies and weakens their lymphocyte proliferation response. The immune system of neonates can be strengthened by supplementing them with vitamin A and β-carotene (Fan et al., 2015). Vitamin A deficiency decreases the antibody titer and bile Immunoglobulins IgA concentration after vaccination (Çevİk, 2018). Furthermore, vitamin A and its derivatives contribute in the regulation of the growth and differentiation of gastrointestinal epithelial cells (Amit-Romach etal., 2009).

A clinico-pathological study was performed to understanding of the influence of the vitamin A deficiency on the occurrence of the AE in newly hatched chicks.

Twenty, Two-Day-old paralyzed chickens, had been referred to the clinic of Veterinary School. The chickens were taken from a broiler flock with 13,000 birds. Day-old chicks were purchased from a local hatchery from a broiler breeder flock vaccinated against avian encephalomyelitis virus (AEV). Clinical signs, including ataxia, trembling, lateral recumbency, and incoordination, were observed in the chickens. Although the majority of the affected chicks attempted to eat and drink, they were not able to compete for food and water due to the paralysis of their extremities and wings. According to the owner's answer, 5% of the birds were clinically affected on the first day, but the number ofthe affected cases increased by the second day.

According to the gross evaluations, haemorrhagicspots were observed under the occipital bone, andaround the cerebellum. Moreover, in some cases, the hearts were larger than normal size (Figure 1).

Microscopical evaluation in the brain revealed non-supportive encephalomyelitis characterized by multifocal foci of neuronal degeneration and necrosis, central chromatolysis of Nissl substance, multifocal lymphoid infiltration with perivascular cuffing (Figure 2 (A)), hyperemia, microglial proliferation, both in nodular and diffuse form and demyelination. In the myocardium, fiber degeneration and lymphocytic aggregates between the muscle fibers of the myocardium were observed. In the proventriculus, noticeable rete pegs and acanthosis were noted, indicating hyperplastic and thickened mucosa. Squamous metaplasia of glandular epithelium was also seen by mucosal glands. Furthermore, the accumulation of necrotic debris, admixed with inflammatory cells, and desquamated epithelial cells were found in the lumens of glands. On the other hand, in lamina propria and surrounding glands, the accumulation of granulocytes was demonstrated. Surprisingly, some multifocal aggregations of lymphocytes were observed in the layer of the tunica muscularis of the proventriculus (Figure 2).

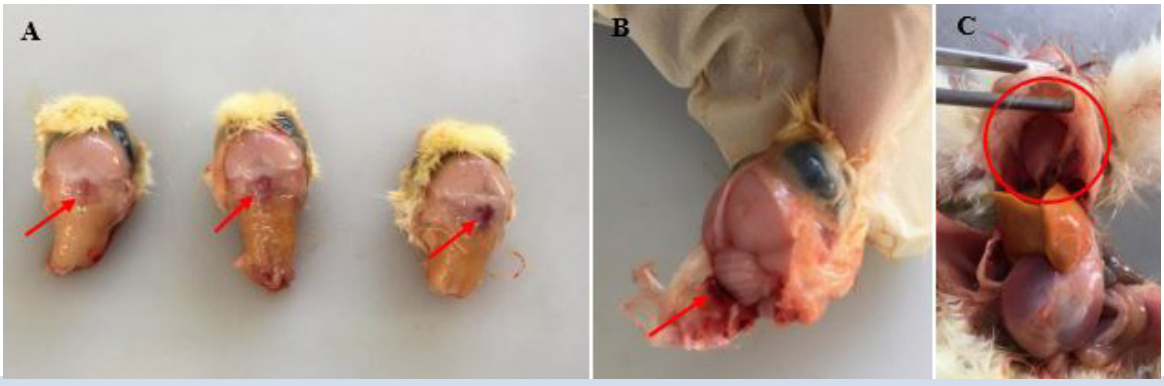


Figure 1. (A & B): The haemorrhagic spots under the occipital bone, around the cerebellum, **(C):** Cardiac enlargement in some cases

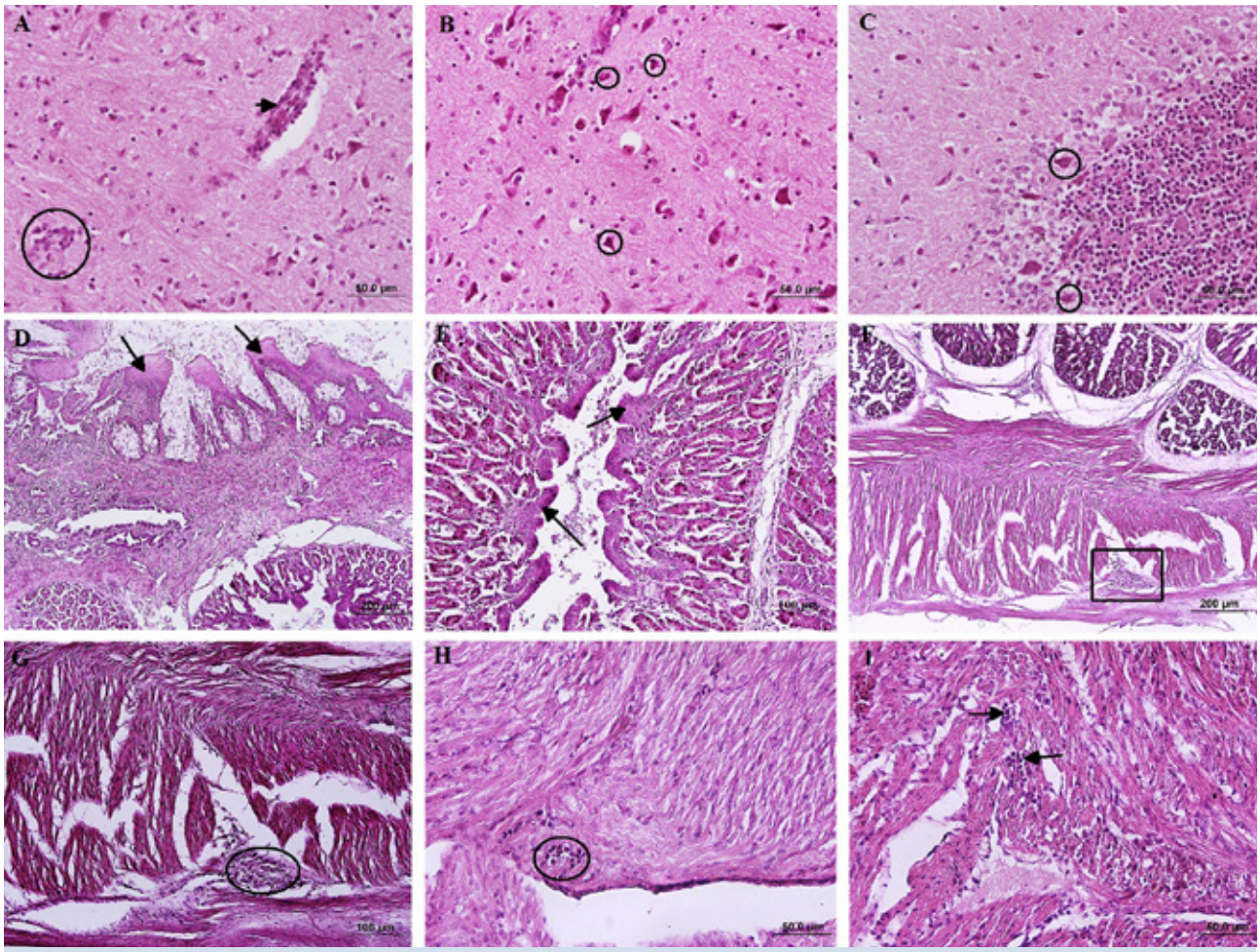


Figure 2. (A): Note tthe lymphocytic perivascular cuffing (arrow) and gliosis (circle), **(B):** Note the necrotic neurons (circle), **(C):** Necrotic and degenerative Purkinje cells (circle), **(D):** Note the squamous metaplasia and acanthosis in mucosal tunic of proventriculus (arrow), **(E):** Squamous metaplastic changes in glandular epithelium (arrow), **(F):** Note the aggregation of lymphocytes in muscularis tunic of proventriclous (rectangle), **(G):** Higher magnification of pervious slide, note thr lymphocytic aggregation (circle), **(H & I):** Foci of lymphocytic aggregates are seen in myocardium (circle and arrow respectively), (H & E, scale bar: A,B,C, G, H, and I: 50μm, D: 200 μm, E: 100 μm)

DISCUSSION

In the present study, the clinico-pathological features of a concurrent occurrence of AE and vitamin A deficiency in a two-day old commercial broiler chicken flock were described.

The major clinical symptoms of AE in adult chickens, are usually subclinical, detected by drop in egg production and the appearance of infected progeny. The clinical symptoms in young chickens are ataxia, and leg weakness from sitting on hocks to paresis that may lead to paralysis and recumbency (Marvil et al., 1999;Jana et al., 2005; Asasi et. al, 2008). In the initial stages of infection, lesions in chickens are frequently observed in the central nervous system than in other organs. Neuron degeneration may be a sign of initial changes of neural tissues, mainly in the medulla and the anterior horn cells of the spinal cord. The gradual disappearance of Nissl substance and the infiltration of perivascular by lymphoid cells were followed by progressing neuronal degeneration. This infiltration occurs mainly in the cortex, cerebellum, and medulla, while the grey matter of the spinal cord was usually unaffected. Although no significant gross changes were usually observed in other organs, there is a possibility of the histopathological lesions occurrence. Lesions in the parenchymous organs and body viscera are characterized by increased peri- and/or paravascular infiltration of lymphocytes and focal lymphoid hyperplasia. Eosinophilic swelling, necrosis, fragmentation, loss of striation in fibres, and heterophil infiltration were the fine changes in muscular structure (Tannock and Shafren, 1994).In the current study, in addition to clinical signs, some microscopic lesions including: multifocal foci of neuronal degeneration, necrosis in the cerebellum and cerebrum, fiber

degeneration and lymphocytic aggregates between the muscle fibers of the proventriculus were observed.

In addition, the squamous metaplasia of the glandular epithelium of mucosal glands was observed in the proventriculus. In a study conducted by Amit- Romach (2009), the role of vitamin A in regulating the growth and differentiation of gastrointestinal epithelial cells was described. Vitamin A deficiency leads to epithelial squamous metaplasia and loss, which affects its integrity and density, thereby allowing the pathogen to easily invade and infect the organism (Yuan et al., 2014; Fan et al., 2015).

In conclusion, since the symptoms of fat-soluble vitamin deficiency are exhibited with by delay,it is assumed that the studied case were selected from a breeder flock suffering from vitamin A deficiency leading to the formation of the eggs with metaplastic changes in the epithelium of the proventriculus. It is also assumed that the AEVin the one-day chickens has been transmitted by an AE infected breeder. The primary cause of the AE outbreak in the breeder flock is not clear. However, since the whole breeder flock was vaccinated against AEV, vitamin A deficiency may decrease the antibody titer concentration after vaccination leading to the occurrence of the disease in the progeny. This study, to the best of the authors' knowledge, is the first report on a broiler farm in north of Iran.

ACKNOWLEDGEMENTS

This research work has been supported by a research grant from the Amol University of Special Modern Technologies, Amol, Iran.

CONFLICT OF INTEREST

The authors declare that there was no conflict of interest.

REFERENCES

Amit-Romach E, Uni Z, Cheled S, Berkovich Z, Reifen R (2009) Bacterial population and innate immunity-related genes in rat gastrointestinal tract are altered by vitamin A-deficient diet. JNutrBiochem 20(1): 70-77.

Asasi K, Farzinpour A, Tafti AK (2008) Clinico-Pathological Studies on Avian Encephalomyelitis in Shiraz, Iran. Turk J Vet Anim Sci 3(3):229-231.

Back A (2015) New Occurance of Avian Encephalomyelitis in Broiler-Is this an Emerging Disease?. Braz J Poultry Sci 17(3): 399-404.

Çevİk A (2018) Pathological findings in broiler chicks with vitamin A deficiency. Harran Univ Vet Fak Derg 7(2): 219-221.

Fan X, Liu S, LiuG, Zhao J, Jiao H, Wang X, Song Z, Lin H (2015) Vitamin a deficiency impairs mucin expression and suppresses the mucosal immune function of the respiratory tract in chicks. PloS one 10(9).

Jana PS, Baksi S, Kundu SK, Guha C, Biswas U (2005) Clinicopathological studies on avian encephalomyelitis. Indian J Vet Pathol 82(10): 1037-1039.

Koutoulis KC, Horvath-Papp I, Tontis D, Papaioannou N, Evangelou K (2015) An outbreak of Avian Encephalomyelitis in broilers in Greece. J Hell Vet Med Soc 66(2): 93-100.

Marvil P, Knowles NJ, Mockett AP, Britton P, Brown TD, Cavanagh D (1999) Avian encephalomyelitis virus is a picornavirus and is most closely related to hepatitis A virus. J Gen Virol 80(3): 653-662.

Sentíes-Cué CG, Gallardo RA, Reimers N, Bickford AA, Charlton BR, Shivaprasad HL (2016) Avian encephalomyelitis in layer pullets associated with vaccination. Avian Dis60(2):511-515.

Tannock GA, Shafren DR (1994) Avian encephalomyelitis: A review, Avian Pathol 23:603-620.

Yuan J, Roshdy AR, Guo Y, Wang Y, Guo S (2014). Effect of dietary vitamin A on reproductive performance and immune response of broiler breeders. PloS one 9(8).