

Clinico-Pathological Studies on Avian Encephalomyelitis in Shiraz, Iran

Keramat ASASI¹, Amjad FARZINPOUR^{2,*}, Azizollah Khodakaram TAFTI³

¹Department of Clinical Science, School of Veterinary Medicine, Shiraz University, Shiraz - IRAN

²Department of Animal Science, College of Agriculture, Kurdistan University, Sanandaj - IRAN

³Department of Pathobiology, School of Veterinary Medicine, Shiraz University, Shiraz - IRAN

Received: 10.10.2006

Abstract: Avian encephalomyelitis virus (AEV) is an important pathogen of poultry and is classified as a member of picornaviridae. Nearly all chicken flocks eventually become infected with the virus, but the incidence of clinical disease is very low unless a breeder flock is not vaccinated and becomes infected after the commencement of egg production. This report describes an outbreak of avian encephalomyelitis (AE) in broiler chicken farms around Shiraz, Iran. This survey included 60,000 birds (18-23 days of age), with 5% mortality. Characteristic clinical symptoms including ataxia, tremor of the head, and stretching of the legs were seen. Pathologically, lesions in the central nervous system (CNS) consisted of gliosis, lymphocytic perivascular infiltration, and neuronal degeneration. Lesions in other tissues involved foci of infiltrating leukocytes in the muscle layer of the gizzard, and lymphoid aggregations in the submucosal muscle layer of the proventriculus. There was an increase in serum encephalomyelitis virus antibody titer in affected flocks. These findings showed an AE infection in broiler flocks around Shiraz, Iran.

Key Words: Avian encephalomyelitis, neuronal demyelination, infiltrating lymphocyte

Introduction

Avian encephalomyelitis (AE) is an enteroviral infection primarily affecting young chickens and is characterized by ataxia and tremors, especially of the head and neck. The present study describes a clinico-pathological study of AE affecting 6 commercial broiler flocks around Shiraz, Iran.

Case History

A total of 60,000 birds (18-23 days of age) from several commercial broiler flocks around Shiraz, Iran, were suspected of having avian encephalomyelitis disease. About 15% of birds showed clinical signs. Mortality was about 5% in all farms. Specimens of organs like the brain, spinal cord, gizzard, and proventriculus were taken from dead birds and were fixed in 10% buffered neutral formalin for pathological examination as described by Ono et al. (1). Serum samples were collected for

serological diagnosis as Smart et al. suggested (2). ELISA was used to diagnosis active infections with AEV with sequential serum samples.

Results and Discussion

The clinical signs included depression, ataxia, tremor of the head, stretching of legs, lateral deviation of the head, and lameness. The ataxia varied from slight incoordination to sitting on the hocks, and lateral recumbency. Postmortem examinations of affected flocks were performed and no marked gross lesions were found in the dead birds. Histopathologically, a glial focus was seen in the molecular layer of cerebellum as nodular aggregates (Figure 1). Perivascular infiltration, gliosis, and demyelination were seen in the cerebellum. Central chromatolysis of neurons in the nuclei of the brain without an attending cellular reaction and neuronal

* E-mail: amjadfarzinpour@yahoo.com

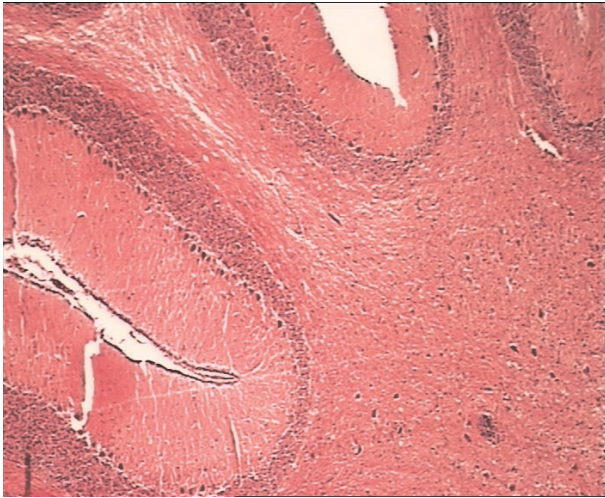


Figure 1. A glial focus was seen in the molecular layer of cerebellum as nodular aggregates. H & E, $\times 160$.

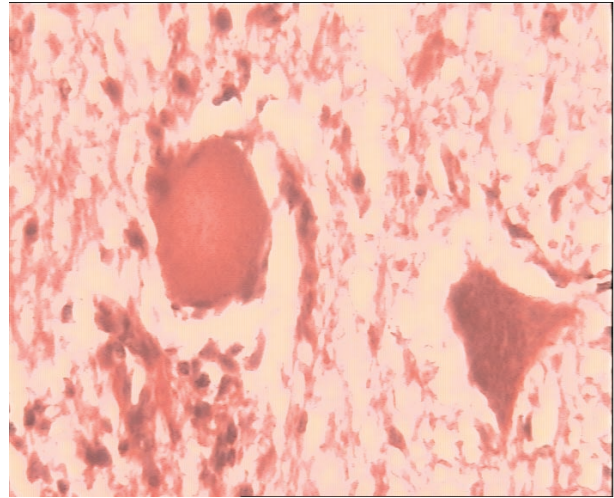


Figure 2. Central chromatolysis of a neuron (left neuron) in the brain of an affected chicken. H & E, $\times 250$.

degeneration in the brain stem and mid-brain were present (Figure 2). There was hyperplasia of the lymphocytic aggregates that were certainly pathognomonic in the tunica muscularis of the gizzard (Figure 3). There was a lymphocytic focus in the submucosal muscle layer of the proventriculus (Figure 4). The results of serological examinations (ELISA) showed an increase in serum avian encephalomyelitis virus antibody titer with sequential serum samples that they were collected at the end of the period. Similar clinical signs in AE were also reported by Chauhan and Roy (3), and Jana et al. (4). The ataxia varied from slight

incoordination to sitting on the hocks and lateral recumbency, which was also reported by Jana et al. (4). The histopathological results obtained in the present study were greatly correlated with those in the studies by Jana et al. (4), Butterfield et al. (5), and Calnek et al. (6), who reported neuronal degeneration, perivascular cuffing, and gliosis of the brain and spinal cord in AE. In the present study, AE was differentially diagnosed from Newcastle disease in which gliosis and central chromatolysis are not characteristic pathognomonic changes. Peripheral nerve involvement like enlargement of the sciatic nerve suggestive of Marek's disease was not

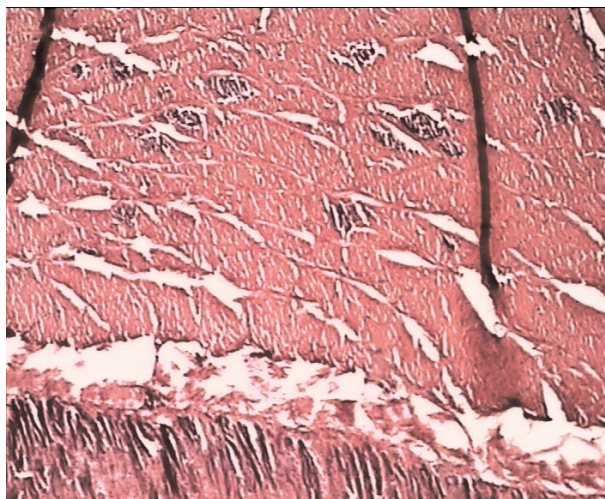


Figure 3. Dense lymphocytic foci were seen in the muscular wall of the gizzard. H & E, $\times 80$.

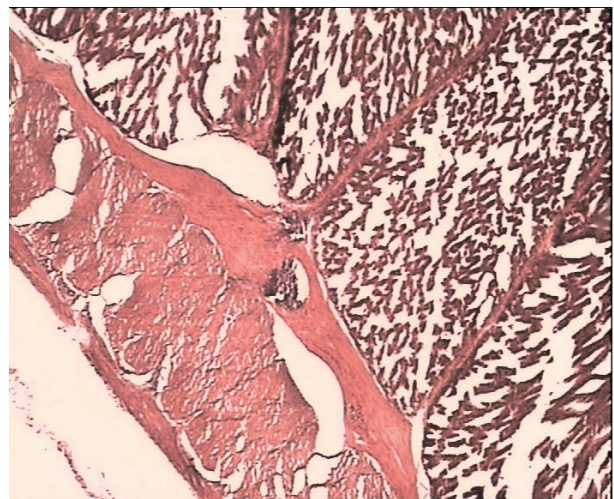


Figure 4. There was a lymphocytic focus in the submucosal muscle layer of the proventriculus. H & E, $\times 80$.

present in the affected birds in the present study. The negative response to vitamin B1 therapy of nutritional encephalomalacia excluded the possible presence of these diseases in the present study. Histopathological evidence

of gliosis, lymphocytic perivascular infiltration, neuronal degeneration in the CNS and serological findings could be considered as the basis for a positive diagnosis of AE (5,6).

References

1. Ono, M., Okuda, Y., Yazawa, S., Imai, Y., Shibata, I., Sato, S., Okada, K.: Adenoviral gizzard erosion in commercial broiler chickens. *Vet. Pathol.*, 2003; 40: 294-303.
2. Smart, I.J., Grix, D.C., Barr, D.A.: The application of the ELISA to the diagnosis and control of avian encephalomyelitis. *Aust. Vet. J.*, 1986; 63: 297-299.
3. Chauhan, H.V.S., Roy, S.: *Poultry Diseases, Diagnosis and Treatment*, 2nd edn., New Age International (P) Limited Publishers. New Delhi. 2001; 106-108.
4. Jana, P.S., Baksi, S., Kundu, S.K., Guha, C., Biswas, U.: Clinico-pathological studies on avian encephalomyelitis. *Indian Vet. J.*, 2005; 82: 1037-1039.
5. Butterfield, W.K., Helmboldt, C.F., Luginbuhl, R.E.: Studies on avian encephalomyelitis. IV. Early incidence and longevity of histopathological lesions in chickens. *Avian Dis.*, 1969; 13: 53-57.
6. Calnek, B.W., Barnes, J.H., Beard, C.W., Reid, W.M., Yoder, H.W.: *Diseases of Poultry*. 9th edn., Affiliated East-West Press and Iowa State University Press, Ames, Iowa, 1991; 250-254.