

Cerebellar malacia in seven-day old broiler chicks: suspected case of avian encephalomyelitis

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Abstract

Neurotropic viral infections in young chicks may cause rapid neurologic disease and high mortality in unvaccinated flocks. The present case report documented clinicopathologic and histopathologic features of an acute neurologic disease outbreak in seven-day-old broiler chicks, and evaluated the relative likelihood of its aetiologic agent being avian encephalomyelitis virus (AEV) versus Newcastle disease virus (NDV). Representative freshly dead chicks from a broiler flock (n = 100) presenting clinical signs of progressive ataxia, circling and 15 deaths over seven days were necropsied. Post-mortem examination revealed minimal gross changes, limited to pulmonary congestion, mild enteritis, retained yolk sac and exudates in the abdomen. The brain (cerebrum, cerebellum, and brainstem), proventriculus and sciatic nerve were dissected out, fixed in formalin, paraffin-embedded, and stained with haematoxylin and eosin for light microscopy. Results of histology showed severe, diffuse non-suppurative leptomeningoencephalitis with dense perivascular mononuclear cuffing, endothelial swelling, extensive gliosis, neuronal degeneration and neuronophagia. Notably, the cerebellum exhibited multi-focal to coalescing granular layer necrosis with liquefactive encephalomalacia. Marked ependymal hyperplasia and subependymal lymphocytic infiltration were also present. There was no convincing transmural fibrinoid necrosis of vessels or widespread visceral necrosis or haemorrhage. In birds of this age (seven-day-old), the lesion distribution (prominent cerebellar malacia and granular cell loss), epidemiologic history (early onset, possible vertical transmission), and lack of systemic haemorrhagic or necrotizing visceral lesions favour avian encephalomyelitis virus as the most likely cause; NDV remains an important differential. This case report emphasizes the critical role of histopathology in diagnosing central nervous system disorders in poultry, and highlights the importance of early vaccination and biosecurity in preventing severe neurologic diseases in broiler flocks.

Keywords: Neurologic disease; Broiler chickens; Histopathologic diagnosis; Cerebellar malacia; Avian encephalomyelitis.

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Introduction

Poultry production globally serves as a source of income, high-quality reasonably priced animal protein, and a significant opportunity for investment and employment (Attia *et al.*, 2022). Broiler chickens exhibit rapid growth, excellent feed efficiency and produce tender meat that is widely accepted and consumed globally. Broiler chicken production has expanded significantly in response to the increasing demand for affordable and high-quality poultry meat (Haque *et al.*, 2020). However, broiler production faces challenges of infectious disease outbreaks, poor management, and lack of or failure of vaccinations, especially in resource-limited settings. Among the infectious causes of disease, viruses and viral diseases of poultry, especially those affecting young chicks, are of particular concern due to their acute presentation and high case fatality rate.

Avian encephalitis (AE), commonly referred to as avian encephalomyelitis, is a viral infectious disease caused by the avian encephalomyelitis virus (AEV). AE is essentially an enteric infection, and it is transmitted between birds by oral ingestion, but it can also be transmitted vertically from infected breeding females through the egg to the chick, resulting in clinical signs at hatching (Calnek, 2008). It primarily affects the central nervous system of one- to four-week-old chicks, and causes significant economic losses worldwide to the poultry industry (Zhang *et al.*, 2023). In young birds, AE can lead to high morbidity and mortality, particularly in the absence of vaccination (Al-Mubarak *et al.*, 2023). The virus may infect a variety of birds, including pigeons, quail, turkeys, pheasants, and chickens (Saif *et al.*, 2020).

Typical neurological signs of AE in young chicks include ataxia, depression and sudden head and neck movements (Struthers *et al.*, 2024). Approximately 40 to 60% of chicks will exhibit signs of the disease, and about 25% of morbid

birds die on the average (Zhang *et al.*, 2023). Furthermore, the virus may infect chicks through the embryo, which is detrimental to the poultry business. Both horizontal and vertical routes of transmission can spread AEV in both healthy and diseased flocks (Kovacs *et al.*, 2025); the primary horizontal route is the faecal-oral route (Plain *et al.*, 2014) and vertical transmission results in larger losses since day-old chicks are affected. This occurs when breeders with no immunity become infected during the egg production stage and pass the virus to their offspring (Al-Hammadi and Al-Rasheed, 2024). Chicks infected vertically may exhibit clinical signs shortly after hatching.

Histologically, AE is characterized by two major lesion patterns: non-suppurative encephalomyelitis affecting the central nervous system, and lymphoid aggregates in visceral organs, particularly the proventriculus (Al-Hammadi and Al-Rasheed, 2024). Lesions predominantly affect the brainstem, cerebellum and spinal cord (Senties-Cué *et al.*, 2016). The most consistent microscopic findings include perivascular lymphocytic cuffing, diffuse or focal gliosis and glial nodule formation, reflecting a mononuclear inflammatory response typical of viral infections (Mohanty *et al.*, 1973; Senties-Cué *et al.*, 2016). Neuronal degeneration is a key feature, commonly presenting as central chromatolysis, neuronal shrinkage and necrosis, often accompanied by satellitosis and neuronophagia. These lesions correlate with the classical neurologic signs observed clinically, such as tremors, ataxia and paresis in young birds. Extra-neural involvement has been reported, most notably in the pancreas, where lymphocytic infiltration, acinar degeneration and focal necrosis may occur, resulting in non-suppurative pancreatitis (Toplu and Alcigir, 2004). Mild mononuclear infiltrates have also been described in other tissues, including skeletal and cardiac muscle, particularly in experimental infections (Toplu

and Alcigir, 2009). Overall, the characteristic combination of non-suppurative CNS inflammation, neuronal injury and gliosis constitutes the histopathologic hallmark of avian encephalomyelitis and remains central to its diagnosis and understanding of disease pathogenesis.

The present case report documents severe diffuse non-suppurative meningoencephalitis with vasculitis and cerebellar encephalomalacia in seven-day-old broiler chicks from an unvaccinated flock. The authors discuss clinicopathologic features, differential diagnoses, diagnostic limitations and practical control recommendations.

Case Presentation

On July 10, 2025, a client presented a case of neurologic signs and progressive mortality in a flock of 100 one-week-old broiler chicks. According to the client, the flock began showing signs of disease within a few days of arrival from the supplier/hatchery. Clinical signs included progressive ataxia, recumbency with extended hind limbs, dullness, reluctance to move, drooping and deviation of the head, circling, incoordination, lameness, dyspnea

(respiratory distress) and sudden death (Figure 1). Mortality occurred at a rate of 3 – 4 birds per day, resulting in 15 deaths over the course of one week. The birds had not received any vaccinations prior to the outbreak. The farmer reported that the previous flock had also shown similar signs. In response to the losses, the farmer administered Trypanor® (tylosin powder) empirically, but this intervention yielded no clinical improvement.

Representative numbers of the one-week-old chicks were submitted to the Veterinary Pathology Laboratory for necropsy and histopathological evaluation. Following submission to the laboratory, the live birds were examined clinically and humanely euthanised. The brain, proventriculus and sciatic nerve samples from the carcasses were submitted for histopathological and bacteriology evaluation. Samples were cultured on 5% sheep blood agar and MacConkey agar (Oxoid, Basingstoke, UK) and were incubated aerobically for 18 to 36 hours. Samples taken from the birds for histopathology were fixed in buffered formol saline, and sections of the tissues were prepared using conventional methods.

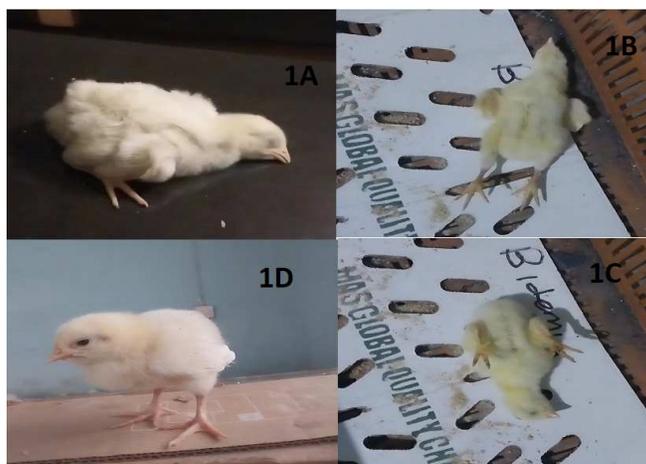


Figure 1. Clinical signs presented by broiler chicks suspected of having avian encephalomyelitis: 1A – Chick exhibiting recumbency and lateral head deviation with eyes closed; 1B and 1C – Chicks found recumbent on their back with extended legs; 1D – Chick standing, exhibiting dullness, reluctance to move, and mild head droop.

Results

At necropsy, the birds showed congested lungs and mild enteritis, while some exhibited a retained yolk sac, suggesting delayed yolk absorption. Additionally, caseous exudate was present in the abdominal cavity of the birds. Some showed no gross abnormalities.

Bacterial culture of tissues yielded light growths of coliforms, enterococci and staphylococci, but no single organism predominated, and no consistent bacterial isolates were recovered from the brain of any of the chicks.

Microscopic examination of the brain showed marked leptomeningeal thickening due to proliferation of leptomeningeal cells (Figure 2A) and dense infiltration by mononuclear inflammatory cells, predominantly lymphocytes and plasma cells. There was extensive perivascular cuffing and widespread non-suppurative vasculitis affecting the cerebral vessels (Figure 2B), accompanied by endothelial swelling and capillary congestion. Within the cerebrum, cerebellum, and brainstem, there was extensive gliosis characterized by microgliosis and astrocytosis (Figure 3A and 3B). Neuronal necrosis was evident, particularly in the cortex, and was associated with multi-focal neuronophagia (Figure 3A and 3B). Affected neurons displayed classical signs of degeneration, including

nuclear pyknosis, cytoplasmic eosinophilia and chromatolysis. These changes were multifocal and, in some areas, coalesced to form larger zones of necrosis. The lateral ventricles exhibited pronounced ependymal cell hyperplasia. Sub-ependymal areas showed lymphocytic infiltration and associated gliosis, predominantly involving oligodendrocytes and microglia (Figure 4A and 4B). These findings indicate a severe, diffuse non-suppurative meningoencephalitis with prominent involvement of the cerebellar granular layer and multi-focal encephalomalacia (Figure 5A and 5B).

Taken together, avian encephalomyelitis virus (AEV) was the most likely aetiology considering the very young age at onset, prominent cerebellar lesion distribution, uniform severe CNS involvement with minimal systemic gross lesions and a history compatible with possible vertical transmission. The observed perivascular lymphocytic cuffing and endothelial swelling are common findings associated with AE and other neurotropic viruses. A true necrotizing fibrinoid vasculitis with extensive haemorrhage, which is commonly observed in association with velogenic NDV infection, was not observed. Though Newcastle disease virus (NDV) remains an important differential diagnosis, ND is less consistent with the young age of onset.

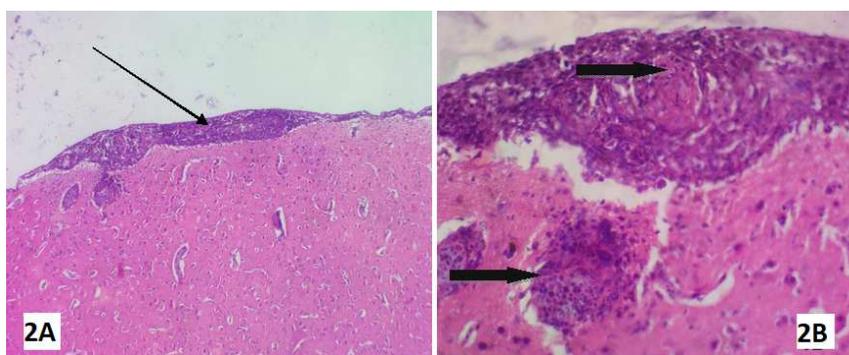


Figure 2. Photomicrograph of the meninges and cerebral cortex showing a marked thickening of the leptomeninges (arrowed) by mononuclear cellular infiltration (lymphocytic) and severe congestion (2A; H & E, 100×). The higher magnification (2B; H & E, 400×) shows capillary congestion, perivascularitis and vasculitis (arrow head).

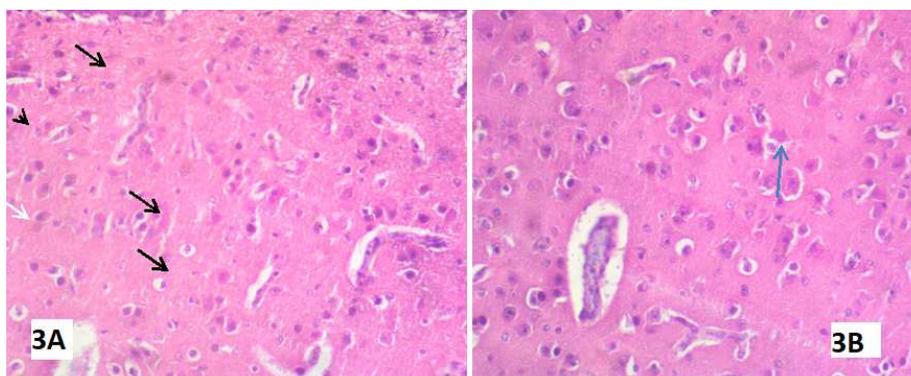


Figure 3. Photomicrograph of cerebral cortex showing various neuropathological changes including marked gliosis, neuronal necrosis (black arrow) with chromatolysis (white arrow) and neuronophagia (blue arrow), indicative of a non-suppurative encephalitis. [H & E, 400×]

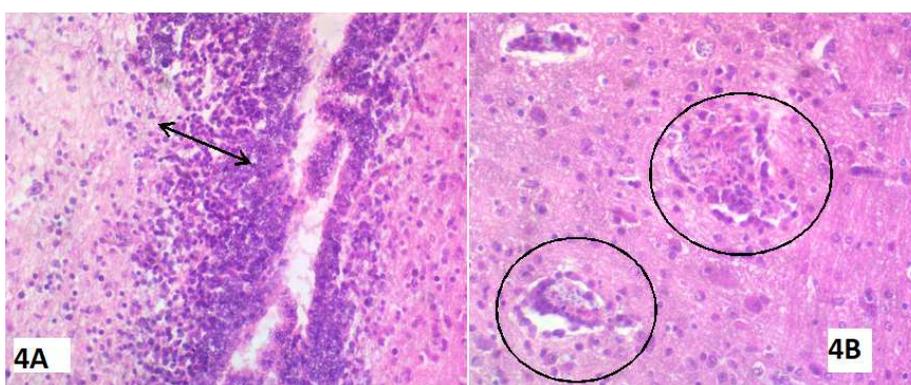


Figure 4. High magnification of the lateral ventricles showing marked thickening by severe ependymal cell hyperplasia and lymphocytic infiltration (4A) [H & E, 100×]. The oligodendrocytes in the periventricular areas are more pronounced in response to neuronal injury and inflammation. The picture on the right (4B) shows marked perivascular cuffing with lymphocytes and glial cells in two of the capillaries in the cerebral cortex (encircled) [H & E, 400×].

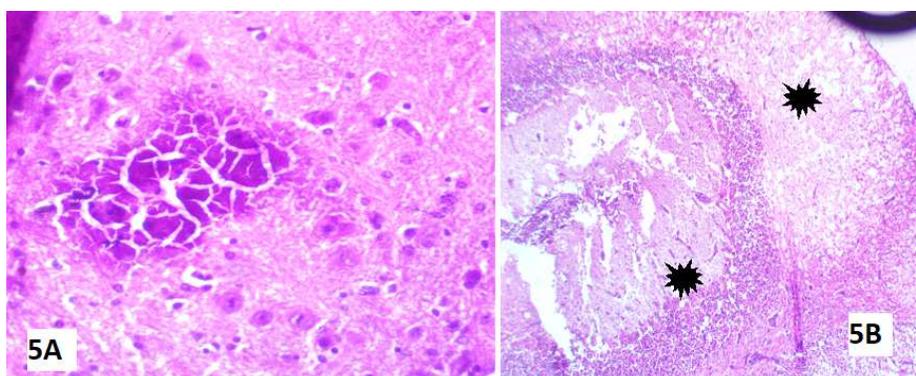


Figure 5. 5A – High magnification showing glial nodules comprising activated swollen astrocytes present in the brainstem of the chicken [H & E, 400×]; 5B – Low magnification showing marked multi-focal locally extensive areas of cerebellar malacia characterized by marked neuronal and glial cell necrosis (asterisk) [H & E, 100×].

Morphological Diagnosis [Joint Pathology Centre (JPC) system]

Meninges: Severe leptomeningeal hyperplasia with non-suppurative vasculitis and perivascular mononuclear cuffing without transmural fibrinoid necrosis.

Parenchyma: Multifocal to coalescing gliosis, neuronal necrosis with neuronophagia in cerebrum, cerebellum and brainstem.

Ventricular System: Ependymal hyperplasia with sub-ependymal gliosis and lymphocytic infiltration.

Cerebellum: Granular layer necrosis with multifocal encephalomalacia.

Brainstem: Focal eosinophilic glial nodules.

A final histopathological diagnosis of severe non-suppurative meningoencephalitis and cerebellar encephalomalacia consistent with avian viral encephalitis, likely of avian encephalitis virus aetiology, was made.

Discussion

Neurologic disease of neonatal and juvenile poultry remains a significant cause of mortality in unvaccinated flocks. The present outbreak involved seven-day-old broiler chicks with progressive ataxia, circling and high early mortality. Histopathology revealed a consistent, severe non-suppurative meningoencephalitis with striking cerebellar granular cell necrosis and multi-focal encephalomalacia. Taken together with the young age of onset and minimal visceral pathology, these features strongly support avian encephalomyelitis virus (AEV) as the likely aetiology in this outbreak.

While NDV and other neurotropic viruses can produce overlapping lesions comparable to that recorded for AE in this case report, certain features classically help distinguish them (Table 1): AEV preferentially targets the cerebellum (Purkinje/granular layers) of very young chicks and is commonly associated with vertical transmission, whereas velogenic NDV

frequently causes systemic necrosis, haemorrhage and a more diffuse necrotizing encephalitis.

When compared to previously documented cases, certain similarities and differences are worth noting. For instance, Welchman *et al.* (2009) reported an outbreak of neurological disease in pheasant chicks with tremors, incoordination and late-onset ocular abnormalities. Histopathology revealed moderate acute encephalomyelitis in only some of the affected birds. Unlike the Welchman case, our report involved uniform and severe histological lesions in both birds examined, with early onset (within a week of hatching) and no ocular involvement, suggesting a more virulent or differently acting neurotropic pathogen. Similarly, in a report by Temeeyasen *et al.* (2024), birds exhibited tremors, torticollis and wing drop by three weeks of age. Histologically, the CNS showed multi-focal lymphoplasmacytic perivascular cuffing, consistent with viral encephalitis. However, gross lesions were absent. This aligns with our findings in terms of histopathology but differs in age of onset and severity. The present case showed more widespread neuropathology, including ependymal hyperplasia and cerebellar malacia, indicative of a more aggressive course. Al-Hammadi and Al-Rasheed (2024) also reported encephalomyelitis in chicks with neurologic signs and no gross lesions. Their histological findings were localized to the cerebral molecular layer, while our case revealed extensive and multi-focal involvement of the cerebrum, cerebellum, brainstem and ventricular system. These differences may be due to variations in virus strain virulence or host susceptibility. Gabriel *et al.* (2016) reported signs such as lethargy, tremors, recumbency and increased mortality in commercial layers. Histology showed perivascular lymphocytic infiltration, central chromatolysis and gliosis in the cerebellum lesions similar to those found in the present

case. However, the birds reported on in the present case report were broilers, not layers, and the onset was significantly earlier. Additionally, the presence of glial nodules and ependymal hyperplasia in the present case report suggests more advanced or systemic CNS inflammation. Toplu and Alcigir (2004) documented encephalomyelitis in pigeons showing paresis, paralysis and torticollis. Histopathology revealed non-suppurative inflammation particularly in the cerebellum similar to our present findings of cerebellar encephalomalacia. However, the present case involved broiler chickens, and the breadth of lesions extended to include vasculitis and widespread gliosis, making the pathology more comprehensive.

One of the unique features recorded in the present case was the severe vascular involvement, with widespread vasculitis and perivasculitis, which is less commonly emphasized in the earlier reports that were compared with the present one. Moreover, the ependymal cell hyperplasia and sub-ependymal gliosis recorded in the present case have not been widely described in the previous reports; this possibly represents a continuum of damage due to high viral load or severe inflammatory response. Although gross lesions were minimal, the histological pattern is compatible with severe neurotropic viral encephalitis.

Table 1. Neuropathology: Differentiating avian encephalomyelitis from Newcastle disease.

| Feature | Avian Encephalomyelitis | Newcastle Disease | References |
|------------------------------|---|--|---|
| Type of lesion | Non-suppurative lymphocytic encephalomyelitis. | Non-suppurative encephalitis with necrosis and vasculitis. | Shehata <i>et al.</i> , 2020; Bhaiyat <i>et al.</i> , 1994. |
| Primary localization | Cerebellum (Purkinje and molecular layers), brainstem, spinal cord grey matter. | Diffuse: cerebral cortex, optic lobes, medulla, spinal cord. | Sentías-Cué <i>et al.</i> , 2016; Toplu <i>et al.</i> , 2004. |
| Neuronal changes | Degeneration and loss of Purkinje cells, motor neuron degeneration. | Severe neuronal necrosis, degeneration, and neuronophagia. | Toplu <i>et al.</i> , 2004; Ecco <i>et al.</i> , 2011. |
| Vascular lesions | Rare/minimal. | Prominent: endothelial necrosis, perivascular cuffing, vasculitis, hemorrhage. | Shafi <i>et al.</i> , 2023. |
| Inflammatory response | Perivascular lymphocytic cuffing, gliosis. | Perivascular lymphocytic cuffing, macrophage infiltration, gliosis. | Mohammed <i>et al.</i> , 2019; Asasi <i>et al.</i> , 2008. |
| Systemic pathology | Usually absent outside CNS. | Marked systemic lesions: tracheitis, Rhinitis, enteritis, lymphoid depletion, necrosis in viscera. | El-Morshidy <i>et al.</i> , 2021. |

On the balance of evidence of age of birds (seven days), pronounced cerebellar malacia and granular cell loss, uniform CNS changes, minimal visceral pathology and a history suggestive of vertical transmission, AEV is the leading presumptive diagnosis in this present case. The noted ependymal hyperplasia and glial nodules reflect an intense ventriculitis and parenchymal response typical of severe neuroinvasion. Velogenic NDV can cause necrotizing encephalitis with vascular necrosis and systemic visceral lesions (Mariappan *et al.*, 2018); these features were not prominent in the present case.

Conclusion and Recommendations: The combination of (1) early onset in seven-day-old chicks, (2) severe granular cell layer necrosis with multi-focal cerebellar malacia, (3) uniform severe CNS inflammation, and (4) absence of extensive visceral necrosis or haemorrhage, supports avian encephalomyelitis as the leading diagnosis in present case. NDV remains an important differential and cannot be excluded without targeted testing. We therefore recommend prioritized diagnostic sampling for future outbreaks: fresh cerebellum and yolk sac for avian encephalomyelitis virus reverse transcription polymerase chain reaction (RT-PCR) and immunohistochemistry (IHC), Newcastle disease virus (NDV) matrix/fusion gene RT-PCR on brain and swabs, virus isolation in embryonated eggs, and serology of the breeder flock to assess vertical transmission.

Conflict of interest

The authors declare that this case report was conducted in the absence of any commercial or financial association that could be construed as a potential conflict of interest.

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