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Pathological Investigation of Organ-Specific Lesions in Poultry Infected with Newcastle Disease

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Abstract

This study was aimed to evaluate the pathological features of naturally occurring Newcastle disease in broiler chickens reared in commercial broiler farms of Srinagar and Ganderbal District of Kashmir during the year 2025. A total number of eight outbreaks were recorded across different poultry units and carcasses of dead birds were submitted to the Division of Veterinary Pathology for post-mortem examination. The diagnosis was confirmed based on characteristic pathomorphological changes observed in visceral organs, including proventriculitis, pneumonia, hemorrhagic enteritis, bursitis, nephritis, splenitis, encephalitis and lymphoid depletion in lymphoid tissues of affected birds. These findings emphasize that Newcastle disease remains a significant economic concern for the poultry industry, underscoring the need for continuous surveillance and preventive measures in poultry farms.

Keywords: Broiler, Newcastle Disease, outbreak, Pathology, diagnosis

Introduction

Newcastle disease is a highly contagious and acute viral infection affecting a wide range of avian species, resulting in considerable morbidity and mortality in the poultry industry. In the Union Territory of Jammu and Kashmir, this disease is a major contributor to economic losses among poultry farmers, along with other bacterial, viral and fungal infections. It primarily affects both domestic and wild birds and is caused by the Newcastle disease virus (NDV), which belongs to the genus *Avulavirus* under the family *Paramyxoviridae*. The disease was first reported in Newcastle, England, in 1926, from which it derives its name. This disease affects multiple avian species including chickens, turkeys, pigeons, ducks, geese, as well as wild birds such as cormorants and sparrows. The transmission mainly occurs through direct contact with infected birds, although contaminated fences and respiratory secretions also serve as important sources of infection. Newcastle disease is considered one of the most devastating diseases of poultry, with the potential to cause up to 100% morbidity and mortality depending on the virulence of the strain and the susceptibility of the host species. The clinical severity ranges from mild respiratory signs to severe respiratory distress, neurological manifestations and high mortality. The common gross lesions include hemorrhages in the proventriculus, pneumonia, and thickening of air sacs, tracheal exudates, enteritis and splenitis. The disease has a worldwide distribution and is particularly prevalent in many Asian countries, including India. Due to limited available information on Newcastle disease in broiler chickens in Kashmir, this study was undertaken to evaluate the pathological alterations affecting the structure and function of various organs in infected broiler birds.

Materials and Methods

The study was carried out at the Division of Veterinary Pathology, SKUAST-Kashmir. During the study period, systematic visits were conducted to both organized and backyard poultry farms to monitor mortality patterns. A total of 100 carcasses of broiler birds were collected and submitted to the Division for detailed post-mortem examination. Diagnosis was

confirmed based on the evaluation of gross and histopathological lesions observed in various organs. Necropsies were performed in accordance with the standard procedures described by Calnek (1997) ^[4] and all pathological findings were carefully recorded. The tissue samples from different organs exhibiting characteristic lesions including the proventriculus, gizzard, bursa of Fabricius, spleen, thymus, kidney and liver were collected for microscopic examination. The collected samples were fixed in 10% formalin, processed by using routine histological techniques and stained with Hematoxylin and Eosin following the method outlined by Lillie (1954) ^[9].

Results

The pathological findings of this investigation are systematically described under clinical, gross and microscopic observations recorded in poultry infected with Newcastle disease.

- **Clinical signs:** Newcastle disease in broiler chickens is a highly contagious viral infection that primarily affects the respiratory, digestive and nervous systems, often leading to severe economic losses due to high morbidity and mortality. In broilers, respiratory signs are usually the earliest and most prominent, including gasping, coughing, sneezing, rales and nasal discharge, along with open-mouth breathing due to tracheal involvement. The affected birds commonly show marked depression, reduced feed intake, ruffled feathers and huddling behaviour, indicating systemic illness. Digestive disturbances such as greenish, watery diarrhoea are frequently observed, leading to dehydration and weakness. In more virulent infections, nervous signs may develop, including tremors, paralysis, torticollis and incoordination, although these may be less pronounced compared to layers. Since broilers are raised for meat production, egg-related signs are absent, but the disease significantly impacts growth rate, feed conversion efficiency and flock performance. The sudden onset of illness with rapid spread through the flock is typical. In severe cases, especially with velogenic strains, mortality can be very high, often occurring within a short period, making early detection and control critical.
- **Gross Lesions:** Newcastle disease in affected birds is characterized by pronounced, multisystemic gross pathological alterations observed during post-mortem examination. The carcasses are typically emaciated and dehydrated, exhibiting ruffled feathers and a generally poor body condition. The detailed observations of gross lesions across different organs are outlined below.
- **Proventriculus:** The proventriculus consistently exhibits marked congestion and hyperemia, reflecting an intense inflammatory response. The mucosal surface often contains multiple petechial hemorrhages resulting from vascular damage and increased capillary fragility. There may be small necrotic foci, appearing as pale yellow spots, which are commonly observed and may coalesce in severe infections to form larger areas of necrosis. Sometimes Oedematous changes lead to thickening of the proventricular wall, altering its normal structure. In advanced cases, fibrinous plaques or diphtheritic membranes may cover the mucosal surface, indicating severe tissue injury and inflammation.
- **Gizzard:** The gizzard shows varying degrees of epithelial damage, including erosions and ulcerations that appear as irregular disruptions of the koilin layer. Congestion is commonly present, imparting a reddish discoloration to the organ. The inflammatory processes often result in thickening of the muscular wall. Hemorrhages may be visible as red patches within the mucosa or submucosa. In severe cases, fibrinous exudates may accumulate either on the surface or within the lumen. Additionally, the presence of lymphoid nodules, formed due to lymphocyte proliferation, indicates an active immune response to viral infection.
- **Brain: Grossly,** the brain is revealing congestion, edema and swelling. Petechial hemorrhages are frequently seen on the brain surface and within the parenchyma. Necrotic areas may develop due to neuronal damage. In advanced stages, the meninges often show thickening and fibrinous exudation, suggesting severe inflammation. Sometimes ventriculitis may be observed in the affected birds as characterized by fluid accumulation within the Brian ventricles, along with demyelination of nerve fibers, contributing to neurological dysfunction.
- **Lungs:** Pulmonary lesions include marked congestion and haemorrhage, often accompanied by edema that leads to swelling and impaired respiratory function. In severe infections, areas of consolidation may develop, indicating loss of normal lung architecture. The necrotic foci may also be present within the lung tissue. The air sacs frequently appear thickened and may contain caseous or fibrinous material, indicative of airsacculitis and chronic inflammatory processes.
- **Heart:** The heart may exhibit cardiomegaly due to vascular congestion and accumulation of blood. Hemorrhages are commonly observed within the myocardium and focal areas of necrosis may develop as a result of tissue damage. In some cases, pericardial effusion is evident, characterized by the accumulation of serous or fibrinous fluid within the pericardial sac. These changes may impair cardiac function and contribute to systemic circulatory disturbances.
- **Liver:** The liver is often enlarged and congested, displaying a characteristic mottled appearance with areas of red discoloration due to haemorrhage. The multiple necrotic foci may be scattered throughout the parenchyma, giving the organ a friable consistency. In some cases, the liver appears swollen and rounded with granular surface changes. These alterations reflect hepatocellular injury, vascular disturbances and inflammatory responses associated with the infection.
- **Kidneys:** Renal lesions are characterized by enlargement and marked congestion, with visible hemorrhages on the surface. Necrotic areas may be present within the renal tissue, indicating cellular damage. In chronic cases, fibrosis may develop, resulting in a rough, irregular and scarred appearance of the kidneys. These structural changes can significantly impair renal function.
- **Spleen:** The spleen typically shows enlargement, congestion and a mottled appearance. Hemorrhages and necrotic lesions are frequently observed. Fibrin deposits may form nodular structures on the surface of the spleen. In severe infections, lymphoid depletion occurs,

leading to splenic atrophy and reduced immunological function, making the birds more susceptible to secondary infections.

- **Thymus:** The thymus often undergoes atrophy, appearing pale and mottled in appearance. Hemorrhagic areas may be present, indicating vascular damage. Edema contributes to swelling of the organ, while necrosis may develop in advanced stages of the disease. These changes compromise the thymus's role in T-cell production, thereby weakening the immune response.
- **Bursa of Fabricius:** The bursa commonly exhibits edema, congestion and a mottled appearance. Hemorrhages are frequently present and in severe cases, mucosal striations and fibrin deposits may be observed. Gelatinous or serous exudate may accumulate within the lumen. In extreme cases, rupture of the bursa can occur, potentially leading to peritonitis due to leakage of infectious material into the abdominal cavity.

- **Crop:** The crop often shows ulceration and erosion of the mucosal lining, indicating significant epithelial damage. The pathological changes like congestion, edema and necrosis are common post-mortem findings. In severe cases, fibrinonecrotic exudates or pseudomembranous may cover the inner surface, further compromising the organ's function in food storage and initial digestion.
- **Intestine:** The intestines typically exhibit congestion, haemorrhage and edema, often accompanied by thickening of the intestinal wall due to inflammation. In severe cases, necrosis and ulceration of the mucosa occur, leading to disruption of the epithelial lining. The excessive mucus production and pseudo membrane formation may further interfere with normal digestive processes. The chronic changes such as fibrosis may result in narrowing of the intestinal lumen, significantly impairing nutrient absorption and gut function.

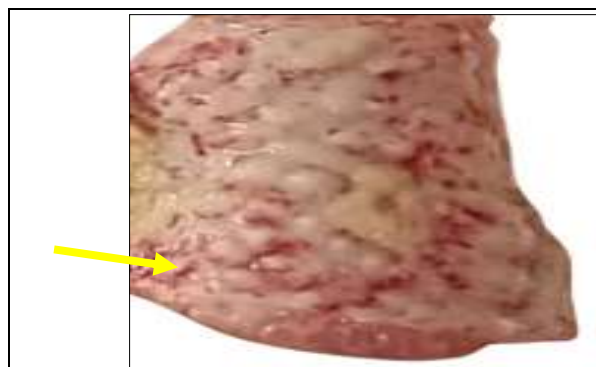


Fig 1A: Photograph of the proventriculus revealing hemorrhages at the tips of the periventricular glands

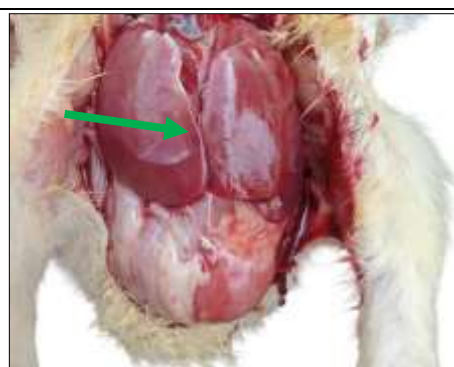


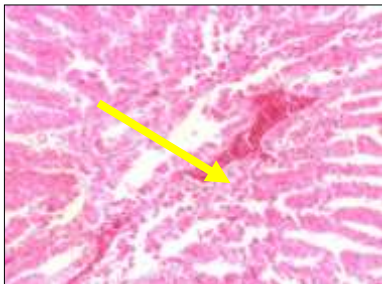
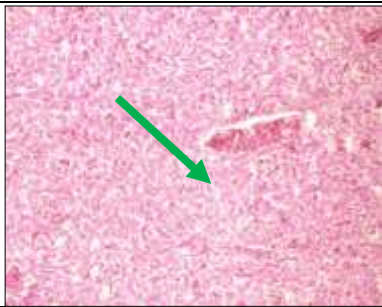
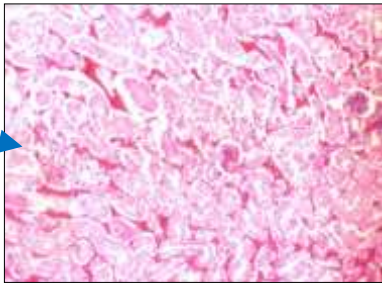
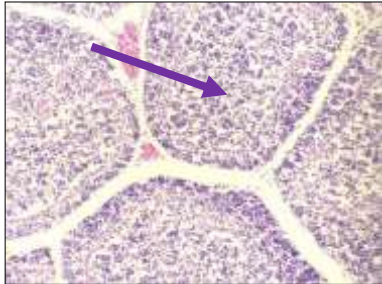
Fig 1B: Photograph of the liver revealing marked congestion, with enlargement of the organ

- **Histopathology:** Newcastle disease in affected birds is characterized by extensive microscopic alterations involving multiple organ systems. Histopathological examination reveals varying degrees of cellular degeneration, necrosis, vascular changes and inflammatory cell infiltration, reflecting the systemic nature of the infection. The microscopic lesions in different organs are comprehensively described below.
- **Proventriculus:** The proventriculus exhibits varying degrees of inflammatory response, characterized by dense infiltration of lymphocytes, plasma cells and heterophils within the mucosa and submucosa. The glandular epithelium frequently undergoes degenerative changes such as cytoplasmic vacuolation, desquamation and disruption of cellular boundaries, reflecting direct viral injury. In more advanced cases, the glands may become distorted or completely destroyed, leading to ulceration of the mucosal surface. Chronic progression often results in fibrotic replacement of normal tissue, thereby causing a marked loss of the organ's structural integrity and secretory function.
- **Gizzard:** Microscopically, the gizzard shows extensive epithelial degeneration, particularly affecting the koilin layer and underlying mucosa. The inflammatory cell infiltration is prominent and is often accompanied by vascular congestion, interstitial edema and hemorrhages. Hyperplastic changes in the epithelium may occur as a regenerative response, while focal necrosis contributes to mucosal damage. Lymphoid hyperplasia may be observed in response to antigenic

stimulation. The presence of multinucleated syncytial cells and intracytoplasmic or intranuclear viral inclusion bodies further confirms active viral replication and involvement.

- **Brain:** The brain demonstrates classical features of encephalitis, with widespread infiltration of inflammatory cells in both perivascular spaces and neural tissue. Neuronal degeneration and necrosis are prominent, often associated with gliosis and the formation of glial nodules. Perivascular cuffing, consisting of concentric layers of inflammatory cells around blood vessels, is a hallmark finding. Vasculitis and endothelial damage further contribute to vascular leakage and haemorrhage. The presence of intranuclear inclusion bodies is indicative of viral infection and these changes collectively result in severe neurological impairment.
- **Lungs:** The pulmonary tissue shows lesions consistent with bronchointerstitial pneumonia, characterized by infiltration of inflammatory cells within the interstitium and around bronchioles. Necrosis of alveolar and bronchial epithelium is frequently observed, along with congestion and haemorrhage. The oedematous fluid may accumulate within alveolar spaces, impairing gaseous exchange. Thrombus formation in pulmonary vessels may also be seen in some cases. In chronic conditions, fibrotic changes develop within the interstitium, leading to reduced lung elasticity and long-term compromise of respiratory function.

- **Heart:** The myocardium is affected by inflammatory changes consistent with myocarditis, where infiltration of inflammatory cells leads to degeneration of cardiac muscle fibers. Necrosis of myocytes is commonly observed, often accompanied by interstitial haemorrhage. As the disease progresses, fibrotic tissue may replace damaged myocardial fibers, resulting in impaired contractility and compromised cardiac output. These structural alterations can significantly affect the normal functioning of the heart.
- **Liver:** The liver exhibits marked histopathological alterations, including disruption of normal hepatic architecture. Hepatocytes frequently show degenerative changes such as vacuolation and ballooning, along with focal to widespread necrosis. The sinusoidal congestion and vascular damage are common, contributing to impaired blood flow. The inflammatory cell infiltration is often evident within portal areas and hepatic parenchyma. In advanced stages, fibrotic changes may develop, further compromising the liver's metabolic and detoxification functions.
- **Kidneys:** Renal lesions include degeneration and necrosis of tubular epithelial cells, along with glomerular atrophy and shrinkage. Interstitial nephritis is characterized by infiltration of inflammatory cells within the renal interstitium. Congestion and haemorrhage are frequently observed, indicating vascular involvement. In chronic stages, fibrosis may replace normal renal tissue, leading to permanent structural damage and reduced functional capacity of the kidneys.
- **Spleen:** The spleen shows pronounced lymphoid depletion, particularly affecting the white pulp, which is critical for immune function. Hemorrhage and necrosis are commonly observed within the splenic tissue.
- **Granuloma formation** may occur as part of the chronic inflammatory response. In severe cases, destruction of germinal centers leads to impaired lymphocyte production and fibrosis may further reduce the organ's immunological efficiency, rendering the host more susceptible to secondary infections.
- **Thymus:** The affected thymus revealed severe lymphoid depletion and necrosis, leading to significant atrophy of the organ. The inflammatory cell infiltration is evident and the presence of syncytial cells reflects viral-induced cellular fusion. The loss of thymic architecture and reduction in lymphocyte population impair T-cell maturation. In advanced cases, extensive tissue damage results in profound immunosuppression.
- **Bursa of Fabricius:** The bursa shows marked lymphoid depletion within the follicles, leading to their shrinkage and eventual atrophy. The inflammatory changes, including infiltration of immune cells, are commonly present in the affected bursal tissue section. Necrosis and fibrosis may occur in severe cases, further damaging the structural framework of the organ. Syncytial cell formation is frequently observed and serves as an indicator of viral activity. The destruction of bursal follicles significantly disrupts B-cell development and antibody production, weakening the immune response.
- **Crop:** The crop exhibits significant inflammatory changes, with congestion, edema and infiltration of inflammatory cells within the mucosa. Necrosis of epithelial cells is commonly observed and in severe cases, ulceration of the mucosal lining occurs. Chronic cases may show fibrotic changes, leading to thickening and reduced elasticity of the crop wall. These alterations impair the organ's functional capacity in food storage.

Histopathology	
	
Fig2: Photomicrograph of the proventriculus revealing degenerative changes accompanied by haemorrhages	Fig 3: Photomicrograph of the liver revealing necrosis along with congestion of the central vein
	
Fig 4: Photomicrograph of the kidney revealing marked interstitial congestion.	Fig 5: Photomicrograph of the bursa revealing lymphoid depletion resulting in marked immunosuppression.

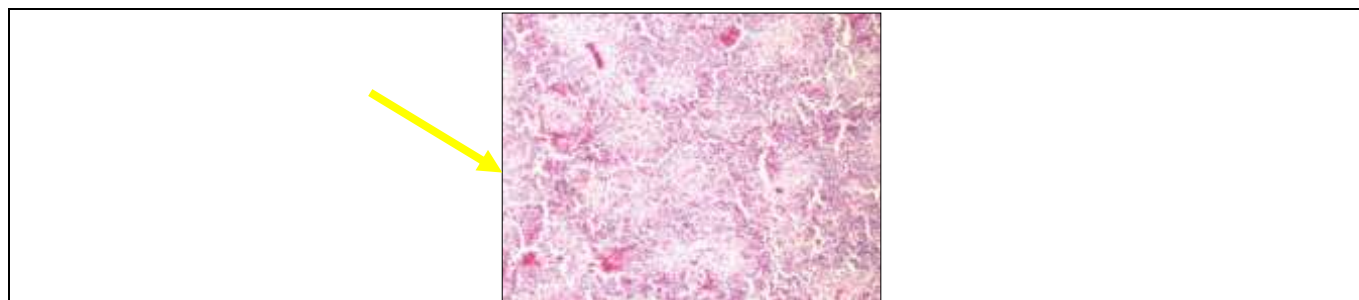


Fig 6: Photomicrograph of the spleen revealing pronounced lymphoid depletion, as evident by the presence of empty spaces within the splenic lymphoid follicles, indicative of marked immunosuppression.

Discussion

Newcastle disease, commonly referred to as Ranikhet disease, is a highly infectious viral condition that affects both organized and backyard poultry systems. It is caused by virulent strains of avian avulavirus-1, also known as Newcastle disease virus (NDV) or avian paramyxovirus-1 (APMV-1). The disease is characterized by high rates of illness and death, particularly in unvaccinated flocks. Apart from domestic birds, numerous wild and captive avian species are also susceptible and can serve as reservoirs of infection. The transmission between infected chickens and pigeons, both domestic and wild, has also been documented (Alexander *et al.*, 2005) ^[3].

The neurological signs observed in affected birds are mainly due to viral multiplication within the brain, resulting in neuronal damage and vascular injury. This triggers an inflammatory response marked by macrophage infiltration around blood vessels, which may extend to astrocytes and microglial cells. Similar observations have been reported in earlier studies, where encephalitis was consistently identified in infected birds. Clinically, affected birds show depression, lethargy, reduced feed intake, clustering behaviour, drooping wings, respiratory distress, lowered head carriage, ruffled feathers and weakness. In the early phase, yellowish diarrhoea is commonly seen, while surviving birds often display poor growth, reduced activity and a characteristic posture with closed eyes and fluffed feathers (Yadav *et al.* 2024 & Zachary *et al.*, 2012) ^[16, 21].

The proventriculus, which serves as the glandular stomach in poultry, is frequently involved in Newcastle disease, particularly in infections caused by highly virulent strains. Grossly, lesions include congestion and hemorrhages of the mucosa, with erosions and ulcerations in severe cases (Saidu *et al.*, 2009 & Rafiq *et al.* (2023) ^[11, 14]. Microscopically, there is significant infiltration of inflammatory cells, especially lymphocytes, along with necrosis of the glandular epithelium (King *et al.*, 2009 & Naikoo *et al.* (2025) ^[8, 17]. Although less commonly affected, the gizzard may exhibit hemorrhages, erosions and necrotic lesions, with mononuclear inflammatory cell infiltration evident around these areas. In the central nervous system, lesions such as perivascular cuffing, neuronal degeneration, gliosis and microglial proliferation are frequently observed (Susta *et al.*, 2011) ^[18], correlating with neurological signs like tremors, incoordination and paralysis. Pulmonary involvement typically includes congestion and haemorrhage, while histological examination reveals severe interstitial pneumonia with infiltration of lymphocytes, plasma cells and macrophages (Rafiq *et al.* (2024) ^[15]. Advanced cases may also demonstrate necrosis and haemorrhage of the respiratory epithelium (Miller *et al.*, 2011) ^[18].

Cardiac involvement is characterized by myocarditis, with hemorrhages affecting both the epicardial and myocardial layers. Histologically, infiltration of mononuclear cells and

multifocal necrosis of cardiac muscle fibers are commonly observed, which may compromise cardiac function and contribute to mortality (El-Tayeb *et al.*, 2012) ^[6]. The liver often appears mottled due to congestion and haemorrhage and microscopically shows disrupted architecture, dilated sinusoids, hepatocellular degeneration and inflammatory infiltration (Wang *et al.*, 2012) ^[19].

Renal lesions typically include enlargement, congestion and occasional hemorrhages. Histopathological findings such as tubular degeneration, necrosis and interstitial lymphocytic infiltration may impair kidney function, clinically reflected by dehydration and increased uric acid levels (Kim *et al.*, 2007 & Shafi *et al.* 2023). ^[7, 12] Lymphoid organs including the caecal tonsils, spleen, thymus, and bursa of Fabricius are markedly affected, exhibiting lymphoid depletion that leads to immunosuppression (Miller & Koch, 2013) ^[10].

Lesions in the crop are characterized by erosions and ulcerations of varying severity, sometimes forming raw surfaces, while the gizzard may show superficial erosions (Alexander *et al.*, 2016) ^[1]. The intestines frequently exhibit severe hemorrhages in both mucosal and submucosal layers. Histologically, submucosal edema, mononuclear cell infiltration and necrosis of the intestinal lining are evident, often resulting in ulcer formation (Dimitrov *et al.*, 2016 & Shafi *et al.* 2023 ^[5, 12]. These observations are in agreement with previous studies and reinforce the multisystemic nature of Newcastle disease, emphasizing its profound impact on poultry health and production.

Conclusion

Newcastle disease remains a significant viral infection of poultry, producing diverse pathological changes that vary with disease severity and the organs involved, ultimately leading to substantial economic losses, particularly for small-scale farmers. A clear understanding of its pathological features is essential for accurate diagnosis, effective control and prevention in avian populations. Therefore, strict implementation of appropriate biosecurity measures is essential to reduce the overall impact of this disease on the poultry industry.

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