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Clinico-pathological assessment of naturally occurring Newcastle disease in broiler chicken reared in northern Himalayas

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Abstract
The aim of the study was to examine the Pathological analysis of naturally occurring Newcastle disease in broiler chickens during the period of May to August, 2019 in commercial broiler farms at Ganderbal district of Kashmir. In this study, a total number of ten outbreaks were recorded in different poultry farms and dead birds that were brought to the Division of Veterinary pathology for detailed postmortem examination. The confirmation of cases was performed by detection of gross and histopathology of different organs. The gross pathology and histopathology changes were pneumonia, pericarditis, myocarditis, proventriculitis, enteritis, perihepatitis, inflamed Bursa of Fabricius, interstitial nephritis, splenitis and encephalitis. This disease remains an economical threat to the poultry industry and require regular monitoring in order to prevent the occurrence of disease in poultry farms.

Keywords: Broiler, newcastle disease, hematology, histopathology, outbreak, diagnosis

Introduction
Rural poultry production is an important agricultural activity in the Union Territory of Jammu and Kashmir. Poultry farming is the form of animal husbandry which raises domesticated birds such as chickens, ducks, turkeys and geese in order to produce meat and eggs for food. There are mainly two systems of poultry farming namely extensive and intensive system of poultry farming. In Extensive type of system of farming, poultry birds are permitted to roam freely for search of food instead of being confinement. While in case of intensive method of chicken rearing in which birds are kept indoors with more space and natural light that encourage foraging and perching. The mortality is a daily consideration for poultry farmers and the carcasses must be disposed of in order to limit the spread of disease and the prevalence of pests. There are different methods of poultry disposal in which burial, composting, incineration and rendering are considered as the best method of disposal. Burial is a common method of carcass disposal to manage mortalities but it may pose contamination threat to the groundwater. Composting is the method of disposal in which organic material is broken down and decomposed with the help of the bacteria in order to reduce it to stable humus. Incineration is a costly method of disposal in which the carcass is burned with the help of fuel energy. In the process of rendering, the carcasses are exposed to high temperatures of nearly about 130 C using pressurized steam to ensure destruction of most pathogens. The mortality records in poultry plays an important role in determining the prevalence of diseases and strategies in order to control the disease (Sanjit Kumar, 2018) [17].
There are many bacterial, viral and fungal poultry diseases in the Union Territory of Jammu and Kashmir in which Newcastle disease cause huge economic loses to poultry farmers. The Newcastle disease is a highly contagious viral disease that affects birds, particularly domestic poultry and wild birds. It is caused by the Newcastle disease virus (NDV), which belongs to the Avulavirus genus within the Paramyxoviridae family. The disease is named after the city of Newcastle in England, where it was first identified in 1926. The Newcastle disease affects various species of birds, including chickens, turkeys, pigeons, ducks, geese and wild birds such as cormorants and sparrows. The transmission of Newcastle disease occurs through direct contact with infected birds, their droppings and respiratory secretions. The wild birds can carry the virus without showing any symptoms, acting as carriers and spreading the disease to domestic flocks Newcastle disease is one the most serious diseases of poultry which leads to

100% morbidity and mortality. The severity of the disease can vary depending on the strain of the virus and the species of bird affected. Some strains cause mild respiratory symptoms, while others can lead to severe respiratory distress, nervous system disorders and high mortality rates. The gross lesions consists of haemorrhages in proventriculus, bilateral pneumonia, thickened air sacs, exudates in trachea, enteritis and atrophy of spleen. This disease has a worldwide distribution and is prevalent in many Asian countries, including in India.

Keeping in view the paucity of information regarding Newcastle disease in broiler chickens in Kashmir, this study was undertaken to evaluate the pathological alterations on the structure and functionality of different organs of infected broiler chickens.

Materials and Methods

Study area and study period

The study was conducted at Division of Veterinary Pathology, SKUAST-KASHMIR during the period of May to August, 2019.

Selection of the cases

Both organized and unorganized poultry farms were visited regularly during the study period in order to record the mortality. A total number of 100 death birds were brought to Division of Veterinary Pathology for postmortem examination as target study cases. The Confirmation of cases was performed by detection of gross and histopathology of the organs.

Hematological examinations of sick birds

The blood samples were randomly collected from the suspected sick birds to evaluate the hematological parameters during Newcastle disease. The Blood samples were kept in sterile vials with anticoagulant (EDTA). The blood samples collected with anticoagulant were analyzed for routine examination of blood within 24 hours as per Weiss and Wardrop.

Postmortem and Histological examination

The Post mortem examination was performed according to protocol described by Calnek (1997) [7] and lesions were recorded. Among the different cases, different samples from each of bursa of Fabricius, spleen, thymus, kidney and liver with gross typical lesions were taken for histopathology. The Collected samples were fixed in 10% formalin and the tissues were processed routinely and stained with Haematoxylin and Eosin as recommended by Lillie (1954) [15].

All the chemicals and reagents utilized in this study were obtained from Erba and glass wares used in this study were obtained from Borosil (India). The glass wares were properly cleaned and sterilized before use. All the plastic wares used in this study were obtained from Tarsons (India). The plastic wares were scientifically sterilized by autoclave prior to use.

Statistical analysis

All data are presented as mean (M) ± standard deviation (SD). T-Test was applied to determine the significant differences among different groups.

Results

Hematology

The results showed that the hemoglobin level, packed cell volume, Total red blood cell (RBCs) count and total white blood cells (WBCs) count differ significantly between the groups under this study as shown in Table 1 &Table 2.

Table 1 Hematological parameters in different groups of Broiler Chickens.

S. No	Group	Haemoglobin concentration (g/dl)	Packed cell volume (%)
1.	Non infected Group (Normal birds)	13.00±0.57 [a]	26.32±1.63 [c]
2.	Infected Group (Diseased birds)	7.00±0.41 [b]	22.21±1.32 [d]

Means of various Hematological parameters differ significantly

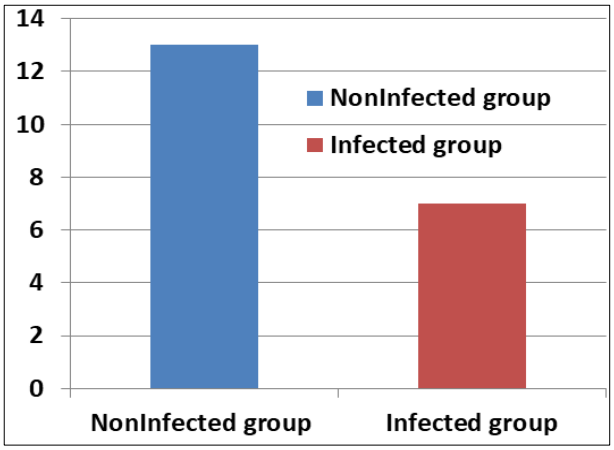

Fig 1: Haemoglobin alteration (Hb) in Newcastle disease in Broiler Chickens

The graph represents decreased level of haemoglobin in infected group as compared to other non-infected group

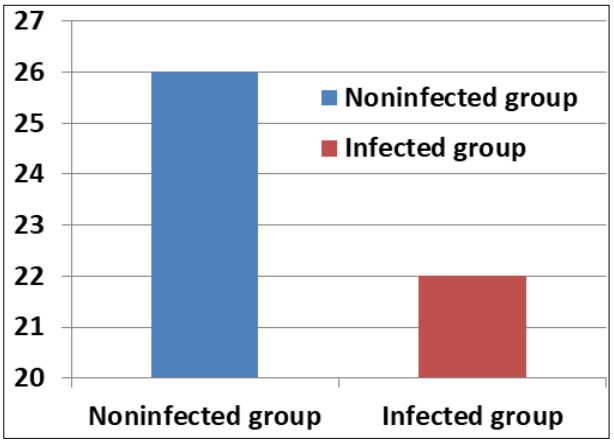

Fig 2: Packed cell volume alteration (PCV) in Newcastle disease in Broiler Chickens

The graph represents decreased level of Packed cell volume in infected group as compared to other non-infected group

Table 2: Hematological parameters in different groups of Broiler Chickens

S.NO	Group	Total erythrocyte count (M/mm^3)	Total leukocyte count (Th/mm^3)
1.	Non infected Group (Normal birds)	8.00±0.25^a	7.00±0.25^c
2.	Infected group (Diseased birds)	5.00±1.52^b	22.00±1.25^d

Means of various Hematological parameters differ significantly

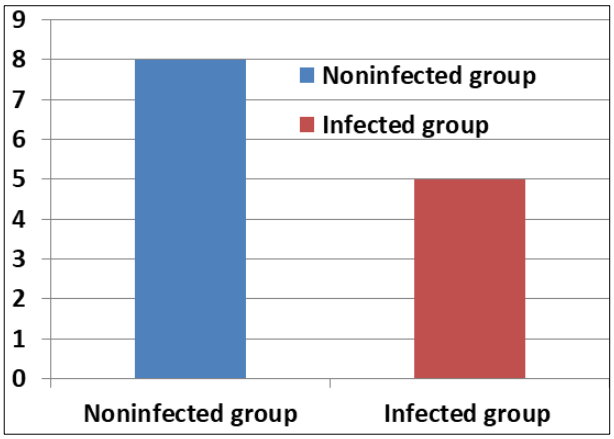

Fig 3: Total erythrocyte count (TEC) alterations in Newcastle disease in Broilers

The graph represents decreased level of Total erythrocyte count in infected group as compared to other non-infected group

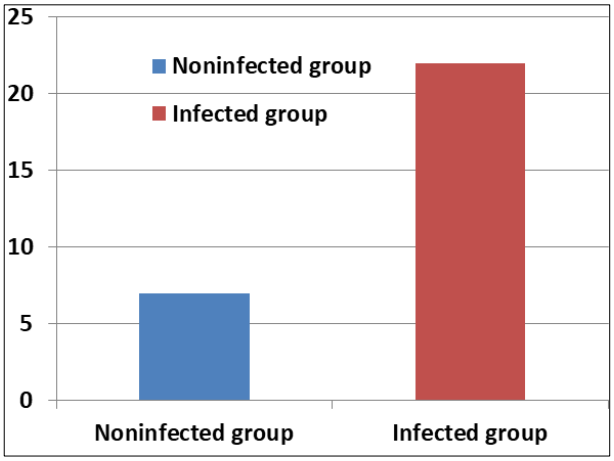

Fig 4: Total leukocyte count (TLC) alterations in Newcastle disease in Broilers

The graph represents increased level of Total leukocyte count in infected group as compared to other non-infected group

Clinical Signs

The affected birds appeared weak, depressed, loss of feather and excessive salivation. The diseased birds exhibited growth retardation, twisted necks, emaciation, dehydration and lameness. The affected birds also revealed depression, drowsiness, in appetence, huddling together, drooping of wings, laboured breathing, lowering of head, ruffiness of feathers and weakness. The diarrhoea was apparent in birds in early stage of disease and the droppings were yellow in colour. The respiratory movements were decreased and birds appeared to have difficulty in breathing. These diseased birds appeared ruffled with pale head and shrunken comb. There was a decline in body weight gain throughout the study period. The clinical signs observed in the affected birds may include sternal recumbency, nervous convulsion, lacrimation from eyes, rough feather coat, profuse salivation, restlessness and ataxia. The keel bone appeared sharp due to emaciation in the diseased birds. Some affected birds may also reveal increase in temperature and emaciation. The affected birds which survived for few days revealed retarded growth, watery droppings, muscle weakness, staggering gait and loss of response to stimulus prior to death. The diseased birds stood motionless for long period of time with the feathers puffed and both eyes closed. The affected birds showed repeated sneezing and fluid came from mouth which appeared to be clogged.

Gross lesions

Proventriculus

The proventriculus may appear congested and reddened due to increased blood flow to the affected area. This hyperemia is a result of the inflammatory response caused by the viral infection. The petechial hemorrhages may also be present on the surface or within the mucosal layer of the proventriculus (Fig: 1). These hemorrhages are a result of the damage to blood vessels caused by the virus and the associated inflammatory response. The small necrotic areas may be seen on the proventricular surface. This necrosis may manifest as areas of pale, yellowish discoloration or ulceration in the mucosa. The severe cases of this disease may show extensive necrotic lesions involving a large portion of the proventricular wall. The edema of the proventricular wall may be observed, leading to thickening of the tissue. This edema is caused by the inflammatory response and increased vascular permeability associated with the infection. In some cases, Fibrin may be deposited on the surface of the proventriculus which appeared as yellowish-white plaques or fibrinous exudates, indicating the presence of inflammation and tissue damage (Fig: 2).

Brain

The brain exhibits encephalitis which is characterized by redness and swelling. The brain may also revealed congestion, appearing reddened due to increased blood flow. The petechial hemorrhages may be observed on the surface of brain tissue which was suggestive of damage to blood vessels caused by the virus. The necrotic areas are also present within the brain tissue which can be visualized as yellow discolored region. In severe cases, meninges appear congested, thickened and have a fibrinous exudate. The ventricles of the brain may show the inflammatory changes known as ventriculitis. In severe cases, the ventricles may contain an excessive amount of inflammatory fluid and cellular debris. The Bleeding can also occur in the brain, leading to the formation of blood clots in certain regions of brain. There might be the loss of the protective myelin sheath surrounding nerve fibers in the brain which can impair nerve function and produce neurological signs. The specific locations and severity of brain lesions can vary depending on the strain of NDV and the individual bird's immune response.

Lungs: The lung tissue may exhibit congestion and hemorrhage which is cauterized presence of blood in the lung

tissue. These lesions are often observed in severe cases of Newcastle disease and can involve different lobes of the lungs. There might be the edema in the lung tissue, leading to swelling and a frothy appearance. This can affect the air exchange within the lungs and compromise respiratory function. In more advanced stages of the disease, the lung tissue may undergo consolidation and the affected areas may appear firm and discolored (Fig: 3). The consolidation was bilateral in only few cases i.e., in advanced stages while as it was unilateral in most of the cases. In some cases, necrotic spots are found on the surface of the lungs which may appear as pale coloured which can sometimes undergo liquefaction, forming caseous material that has a cheese-like consistency There may be the cloudy air sacs which can be manifested with thickening of the walls of air sac and the presence of caseous material.

Heart: Grossly, the heart may appear larger as compared to normal due to the accumulation of blood and known as cardiomegaly. This enlargement can involve one or more chambers of the heart. The blood vessels supplying the heart may appear dilated and engorged with blood due to increased blood volume. The chambers of the heart, including the atria and ventricles, may show signs of engorgement with distension. The congested blood within the heart can give a bluish discoloration to the affected cardiac tissues. The congestion can also lead to fluid accumulation within the heart tissue, resulting in edema. The affected areas may appear swollen and have a pale appearance. The heart of the affected birds also revealed hemorrhages within myocardium which may appear as small red spots on the surface of the heart and within the myocardium (Fig:4.). In severe cases of Newcastle disease, small necrotic foci can be observed on the heart muscle cells. The affected areas of the myocardium may appear pale, yellowish, or grayish and have a friable consistency. In some cases, the birds infected with Newcastle disease may reveal accumulation of fluid within the pericardial sac surrounding the heart. This condition is known as pericardial effusion and can be observed as an excessive amount of fluid surrounding the heart. The pericardium of the affected birds is thickened with a formation of a fibrinous exudate and a layer of fibrin on the pericardial surface.

Liver: Grossly, the liver of the affected birds revealed enlargement, swelling and congestion due to inflammation and cellular damage. The liver surface may show a mottled appearance with patches of red discoloration which might be caused due to hemorrhage and congestion (Fig: 5). The petechial hemorrhages may be observed on the liver surface and within the liver tissue. These indicate bleeding caused by damage to blood vessels. In severe cases of Newcastle Disease, there might be the necrotic foci observed in the liver parenchyma which may appear as pale, yellowish, or grayish in color and have a friable consistency. In some cases, the liver may appear yellow or icteric due to impaired liver function and accumulation of bile pigments. The liver may exhibit congestion due to increased blood flow caused by inflammation and tissue damage. The borders of liver were generally rounded and livers appeared spherical in shape. The livers were easy to cut and the cut surface appeared bulged, hazy and oily in appearance. The affected livers were swollen with rounded borders and in some cases liver presented severe swellings with granular appearance of the surface. The livers of the affected birds also contained numerous yellowish patches and reddish hemorrhagic foci which were distributed uniformly on their surfaces. In few cases, large irregular necrotic foci and hemorrhagic streaks were present on the surface of liver. The gross lesions in the liver can be either focal, affecting specific areas, or diffuse, involving the entire organ. The focal lesions may appear as distinct nodules and masses within the liver, while diffuse lesions affect a larger portion of the liver tissue.

Kidneys: Newcastle disease can lead to enlargement of the kidneys, also known as nephromegaly. The affected kidneys may appear swollen and larger than normal (Fig: 6).The kidneys may show congestion which is characterized by an increased blood supply giving them a reddish coloration. This congestion is caused by the inflammatory response and vascular changes associated with the disease. The Newcastle disease can cause severe hemorrhages within the kidney tissue. These hemorrhages may appear as small red or purple spots on the kidney surface or within the renal parenchyma. In severe cases of Newcastle disease, kidney may reveal necrotic foci on the surface. The affected renal tissue may appear pale, yellowish, or grayish in color and have a friable consistency. In some cases, Newcastle disease virus may result in alterations in the urine-producing capacity of the kidneys. This can lead to changes in the urine color, consistency and volume. The affected kidneys also exhibit mottled appearance due to changes in the renal tissue caused by renal tubular necrosis. The prolonged infection and renal damage can lead to thinning of the renal cortex which can be observed upon gross examination. The development of fibrous tissue in the kidneys can be observed in severe infections resulting in fibrosis and scarring. These areas of fibrosis may appear as white regions on the surface of the kidney tissue. The surface of the affected kidneys may appear rough and irregular due to the presence of fibrotic areas and scarring of tissues.

Spleen: Grossly, spleen was congested and enlarged in young birds. In older birds, white diffused and multiple small foci were observed on the surface of spleen. The spleen may appear swollen, engorged and may be mottled in nature (Fig: 7). The hemorrhagic lesions are observed on the surface of spleen, resulting in red discoloration and the formation of large blood clots. The spleen exhibit congestion, which give the spleen a dark red or purple discoloration. In severe cases of Newcastle disease, necrotic areas may be observed in the spleen which can result in the formation of areas with a pale appearance in the spleen. In some cases, Fibrin deposits may get accumulate in the spleen which can be appeared as yellowish-white nodules on the surface or within the spleen tissue. In this disease, the virus can target and destroy lymphocytes, leading to lymphoid depletion in the spleen. This depletion is characterized by a reduction in the number and size of lymphoid follicles and the overall loss of lymphocytes resulting sometimes severe atrophy of the splenic follicles and regression of lymphoid organ as it can be observed grossly.

Thymus: Grossly, thymic atrophy must be noticed in the affected birds. Thymic atrophy is a hallmark of this disease and reflects the destruction of lymphocytes in the thymic cortex. The thymus exhibits a pale and mottled appearance compared to its normal color. In severe cases of, hemorrhagic areas may be observed within the thymus as caused due to the destruction of blood vessels. In severe cases, hemorrhagic

areas may be observed in the thymus. The spleen may also exhibit congestion due to increased blood flow resulting from the inflammatory response to the virus. This can give the organ a darker coloration. The thymus appeared smaller in size as compared to a healthy thymus which might be due to lymphoid depletion and atrophy of the organ. The thymus exhibit swelling and edema which can be attributed to the inflammatory response and fluid accumulation within the organ. The thymus in few cases of this disease may become soft and fragile to the touch. This softening or friability is a result of tissue damage caused by the virus and the associated immune response. In some cases, areas of necrosis can be observed in the thymus which can be appeared as yellowish-white or grayish in colour.

Bursa: The infected bursa of Fabricious was edematous, enlarged and inflamed in most of the cases. The petechial and ecchymotic hemorrhages were seen on the mucosal surface of bursa. The birds from infected flocks had enlarged and hemorrhagic bursa having blood mixed with cheesy exudate in the lumen. Along with swollen and hemorrhagic bursa, the other prominent gross lesions from dead birds included mottled appearance of bursa with pale discoloration. In severely affected cases longitudinal striations on the mucosal surface of bursa were prominent and the mucosal surface of bursa of the affected birds was covered by gelatinous yellowish transudate. In addition to this, there may be swollen bursa and is friable in nature. The bursa of the affected birds may also have excess exudate with fibrin deposits in the bursal cavity. The affected birds may also have a watery discharge from the cloaca. In severe cases, the bursa may rupture, leading to peritonitis and death. Chickens that have recovered from this infection may have small, atrophied, cloacal bursas due to the destruction and lack of regeneration of the bursal follicles.

Crop: The crop is an organ located in the digestive system of poultry birds, where food is temporarily stored and partially moistened before entering the rest of the digestive tract. Grossly, there may be the development of ulcers and erosions on the inner lining of the crop observed in the Newcastle disease, which may vary in size and can lead to the formation of raw wounds. The crop walls may thicken due to inflammation caused by the viral infection. The hyperemia can also be observed, giving the crop a bluish appearance. The necrotic lesions can develop in the crop, causing the affected areas to turn dark brown in colour.The edema can also be observed in the crop, leading to swelling and an enlarged appearance. The hemorrhagic areas may also be present in the crop which gives reddish appearance to the crop. In some cases, there may be the formation of a fibrinonecrotic exudate, which is a combination of fibrin and necrotic tissue. In severe cases, the crop may develop a pseudomembrane, which is a thick and yellowish-gray membrane covering the inner surface of the crop as a result of accumulation of cellular debris.

Gizzard: The gizzard is a muscular organ responsible for grinding and crushing of food in birds. There may be the development of ulcers on the surface of the gizzard of affected birds, which can appear as irregular-shaped areas with loss of the epithelial lining. The gizzard may also show the erosions which are shallow defects in the gizzard lining that are less severe than ulcers. The affected gizzard may also exhibit congestion which gives a reddened appearance to the gizzrad. In some cases, the gizzard wall can exhibit abnormal thickening as a result of inflammation. In severe cases, there may be a presence of fibrinous exudate, which is a sticky and white material present on the gizzard's surface or within the lumen. The fibrinous exudate consists of fibrin and necrotic tissue and is often associated with severe inflammation. There may be visible areas of hemorrhages within the gizzard tissue, appearing as red patches. The Newcastle disease viral infection can lead to the formation of nodules in the gizzard which may be seen as small and raised structures within the gizzard tissue. These nodules are caused by the proliferation of lymphocytes in response to the viral infection.

Intestine: The affected intestine may exhibit increased vascularity, appearing redder than normal due to congestion of blood vessels. There might be the hemorrhages in the affected intestinal mucosa and submucosa which may appear as pinpoint spots and larger patches of blood within the intestinal wall. The inflammation caused by Newcastle Disease Viral infection can result in thickening of the intestinal wall and edema resulting in the swollen appearance of the intestine. In severe forms of Newcastle Disease, necrosis and ulceration can be observed in the intestinal mucosa (Fig: 8). These ulcers can be irregular in shape and may be associated with areas of necrotic tissue. Newcastle Disease Viral infection can cause excessive mucus production in the intestine. The presence of excessive mucus can be observed as a slimy coating on the intestinal surface. The pseudomembranes can be formed on the surface of the intestinal mucosa in certain infections of Newcastle Disease Viral infection. These membranes consist of fibrin, inflammatory cells and debris, which may appear as yellowish or grayish plaques. Sometimes narrowings of the intestinal lumen caused by fibrosis or scarring may also be observed in the affected intestine which can be resulted from inflammation and ulceration.

Microscopic Lesions

Proventriculus: The proventriculus may show varying degrees of inflammation, primarily characterized by infiltration of inflammatory cells such as lymphocytes, plasma cells and heterophils. The severity of inflammation can range from mild to severe, depending on the stage of the disease. The glandular epithelium of the proventriculus can exhibit degenerative changes, such as vacuolation, loss of cellular integrity and sloughing off proventicular epithelium (Fig: 9). The necrotic areas may appear as erosions and ulcerative in severe cases. The proventricular glands of the affected birds appeared atrophic and disrupted with loss of normal architecture. The microscopic examination of the affected tissue also reveal severe congestion (Fig: 10). In early infection, there may be focal areas of epithelial necrosis, characterized by loss of the superficial epithelial layer and necrosis (Fig: 11). This can progress to more extensive necrosis, ulceration and sloughing of the epithelium. The remaining epithelial cells may appear degenerated with vacuolation and hypertrophic in nature. The presence of the viral infection in the proventriculus can stimulate the activation and enlargement of lymphoid follicles in the lamina propria. The hemorrhagic changes were also observed in the proventriculus (Fig: 12). The Syncytial cells are present in the proventricular mucosa and is formed by the fusion of infected and neighboring cells. The microscopic changes further

revealed atrophy of the proventricular gland (Fig: 13) and loss of glandular architecture (Fig: 14). The severe cases of Newcastle disease can result in fibrosis of the proventriculus. The fibrotic changes occur due to the reparative process following tissue damage and inflammation. Fibrosis can lead to the loss of normal tissue architecture, impaired organ function and potential complications.

Brain: Newcastle disease infection in the brain can lead to an inflammatory response known as encephalitis. The brain tissue may show perivascular and parenchymal infiltration of inflammatory cells, including lymphocytes, plasma cells and macrophages. The extent and severity of the inflammation can vary depending on the stage and virulence of the virus. The microscopic examination of the affected brain tissue revealed satellitosis (Fig.15). Neuronal necrosis can also be observed in spinal cord and various regions of the brain, including the cerebrum, cerebellum, brainstem (Fig.16). The affected neurons may exhibit eosinophilic cytoplasm, pyknosis and karyorrhexis. In response to tissue damage and inflammation, there is reactive proliferation of glial cells which is known as gliosis. Astrocytes and microglial cells may undergo hypertrophy and hyperplasia, leading to an increase in their numbers. The gliosis can be observed in the affected areas of the brain and may contribute to the formation of glial nodules. The vasculitis is characterized by inflammation of the vessel walls, which is which characterized by infiltration of inflammatory cells and thickening of the wall of the blood vessel. This can result in vascular congestion, thrombosis and hemorrhage in the brain tissue. The Inflammatory cells, particularly lymphocytes, may accumulate around blood vessels in the brain forming perivascular cuffs. These cuffs can be observed in the meninges and brain parenchyma. The Perivascular cuffing is a characteristic finding in many viral infections, including Newcastle disease. In some cases, intranuclear inclusion bodies can be detected in infected brain cells. These inclusion bodies are aggregates of viral proteins or viral particles and can aid in the diagnosis of Newcastle disease.

Lungs: The microscopic examination of the lungs revealed bronchointerstitial pneumonia characterized by inflammation and damage to the bronchi, bronchioles, and surrounding lung tissue. The affected bronchi and bronchioles may exhibit epithelial necrosis, sloughing and infiltration of inflammatory cells, including lymphocytes, plasma cells and heterophils around the blood vessel (Fig:17). The interstitial spaces between the bronchi and blood vessels may also show inflammatory infiltrates. In severe cases of Newcastle disease, the lung tissue can undergo consolidation, atelectasis and hemorrhagic in nature (Fig: 18). Consolidation and atelectasis can lead to impaired gas exchange and respiratory dysfunction. In the lungs, heterophils may accumulate in response to infection, leading to heterophilic infiltration. Heterophils are often observed in the airspaces, interstitium, and around blood vessels. The microscopic examination of the affected lung tissue revealed congestion (Fig: 19) and necrosis (Fig: 20). The presence of edema can be observed in the affected lungs of the broiler chickens which can contribute to respiratory distress and compromise lung function. Newcastle disease infection can cause inflammation and damage to blood vessels in the lungs, leading to thrombosis, which is the formation of blood clots. Thrombi can occlude blood vessels and compromise blood flow, further contributing to tissue damage and impaired lung function. In chronic cases of Newcastle disease, fibrotic changes can occur in the lung tissue. Fibrosis refers to the excessive deposition of collagen fibers, leading to the formation of scar tissue. Lung fibrosis can result from the healing process following inflammation and tissue damage. It can disrupt the normal lung architecture and impair respiratory function. The bronchial epithelium may show necrosis, loss of cilia, and infiltration of inflammatory cells, such as lymphocytes, plasma cells, and heterophils. The lumen of the bronchi and bronchioles may contain cellular debris and exudates.

Spleen: The histopathological examination of the spleen revealed lymphoid depletion in the spleen which is characterized by a decreased number of lymphocytes in the white pulp of the spleen (Fig: 21). The depletion may result in the loss of normal splenic architecture and poor immune response against the antigen. Histologically, there may be an increased amount of blood in the spleen sinuses and red pulp. The hemorrhagic areas can also be observed in the affected spleen which is characterized by the extravasation of red blood cells. The microscopic examination of the spleen further revealed necrotic areas which are characterized by loss of cellular architecture and eosinophilic debris (Fig: 22). The Inflammatory changes are observed in the spleen during NDV infection which includes the infiltration of inflammatory cells, such as lymphocytes, macrophages and heterophils in the affected tissue. The germinal centers within the spleen revealed alterations in response to NDV infection. There may be disruption and destruction of the germinal centers, leading to changes in their size, shape and cellular composition. In some cases, NDV infection can lead to the formation of granulomas in the spleen. The granulomas are organized collections of immune cells, including macrophages, surrounded by a rim of lymphocytes. These structures may be observed in the splenic tissue.

Thymus: The histopathological changes in the thymus of the Newcastle disease affected birds may include lymphoid depletion, lymphocytic necrosis and infiltration of inflammatory cells. This lymphocytic infiltration is often diffuse and can lead to enlargement of the thymus. In the early stages of Newcastle disease, lymphoid depletion can be seen in the thymus resulting in the decreased number of lymphocytes (Fig: 23). As the disease progresses, areas of necrosis may develop in the thymus. These necrotic changes can be observed as areas of cellular disintegration which results in the destruction of thymic tissue and affect its normal function (Fig: 24). In severe cases of Newcastle disease, hemorrhage may be observed in the thymus which might be due to damage to the blood vessels within the organ. The thymus gland may exhibit varying degrees of atrophy which is characterized by a reduction in size and weight of the organ. The atrophy is primarily due to the destruction of thymic epithelial cells and loss of lymphoid tissue architecture. In response to the viral infection, the inflammatory cells, such as lymphocytes, plasma cells and macrophages may migrate to the thymus as part of the immune response to the viral infection. NDV has the ability to induce syncytia formation, which are multinucleated cells formed due to the fusion of infected cells' plasma membranes.

Bursa: The bursa of Fabricius is a primary lymphoid organ in birds that plays a crucial role in the development and

maturation of B lymphocytes. The microscopic examination of the bursa of Fabricius revealed lymphoid depletion which is characterized by significant reduction in the number of lymphocytes within the bursal follicles leading to the loss of normal bursal architecture (Fig: 25). The lymphoid depletion may result to the bursal atrophy in which bursa may appear smaller in size as compared to a healthy bursa. This atrophy is caused by the destruction of bursal follicles and lymphoid tissue (Fig: 26). The Inflammatory changes can also be observed in the bursa during NDV infection. The infiltration of inflammatory cells, such as lymphocytes, macrophages and heterophils may be seen histologically which is a part of the immune reaction against the viral infection. The microscopic examination further revealed degenerative changes in lymphoid follicles, interstitial oedema along with the congestion. As the disease progresses, the bursa undergoes further degenerated, small and irregular in shape. The affected cells of the bursa may exhibit certain morphological changes such as nuclear pyknosis, fragmentation and necrosis which are characterized by the presence of loss of cellular architecture. The bursal epithelium becomes disrupted and the underlying lymphoid cells undergo programmed cell death. These changes lead to a significant reduction in the size of the bursal follicles and a loss of its immunological function. The microscopic examination of the affected tissue further revealed hemorrhagic areas in the bursa during NDV infection. These areas show extravasation of red blood cells and can be associated with congestion and vascular damage. In addition this, there may be the destruction of bursal follicles. The affected bursal follicles may appear disrupted and disorganized with loss of distinct follicular structure. This destruction affects the normal process of B-cell maturation and antibody production. The affected bursa further revealed syncytia formation under microscopic examination which is multinucleated cells formed due to the fusion of infected cells and plasma membranes. The prolonged and severe NDV infection can lead to fibrosis in the bursa which is characterized by excessive deposition of fibrous connective tissue, resulting in the distortion of normal tissue architecture. Fibrosis may occur as a reparative response to the damage caused by the virus. The severity of the lesions depends on the virulence of the virus strain, age of the bird and the immune status of the bird.

Liver: The microscopic examination of the affected liver tissue section revealed marked disorganization of hepatic architecture, congested central veins, dilated blood sinusoids and perivascular infiltration **(**Fig: 27). The liver sections also revealed lysis of hepatic cytoplasm with increased vacuolation and some cells are completely filled with large vacuoles presenting balloon like appearance. The microscopic examination of the affected revealed Portal triad of Liver revealing dilated portal vein with engorgement of blood (Fig: 28). Most of the liver section revealed proliferative changes which were characterized by infiltration of leukocytes predominantly mononuclear cell together with hetetrophils and proliferation of fibroblasts at various places in the liver parenchyma. These changes were accompanied by hepatic congestion, necrosis and cellular swelling with thickening of the wall of Blood vessel. The necrotic hepatocytes were characterized by presence of eosinophilic cytoplasm together with nuclear changes which included pyknosis, karyorhexis and karyolysis. The severe cases of Newcastle disease infection may result in the development of fibrosis within the liver tissue characterized by excessive accumulation of connective tissue, leading to the formation of scar tissue. In some cases, the histopathological examination of bile ducts in the liver may reveal inflammation, necrosis and degenerative changes in the epithelium of bile duct. Moreover, liver cord arrangement is highly disrupted and the hepatocytes could be distinguished only by the degenerated nuclei surrounded by small masses of cytoplasm. The most of the cases of this disease revealed swollen hepatocytes and the cytoplasm had a granular appearance with increased eosinophilia. NDV infection can lead to hemorrhage and congestion in the liver, which is characterized by an increase in blood vessels dilation and engorgement with blood. Sometimes the microscopic examination also reveals inclusion bodies within the hepatocytes which are formed by viral replication. Some liver sections also revealed presence of multiple necrotic foci throughout the parenchyma together with hepatocellular degeneration. The liver Parenchyma of the affected birds showed varying degrees of venous congestion, sinusoidal dilatation and haemorrhage. In few cases, the liver sections of the affected birds revealed severe capsular thickening, degenerated hepatocytes, elongated nuclei and leukocytic infiltration. The affected hepatocytes appeared swollen with rounding of corners and their cytoplasm appeared hazy. There was heavy infiltration of leukocytes predominantly by heterophils and lymphocytes around portal triads. The histopathological changes can vary depending on the strain and virulence of the Newcastle disease virus, as well as the stage of infection.

Kidney: The affected kidneys revealed glomerular atrophy, dilatation of renal tubules, fragmentation of glomeruli associated with nephritis. The renal medulla of the affected kidneys revealed degenerative changes in the epithelium of renal tubules, peritubular fibrosis, hemorrhage and interstitial nephritis (Fig: 29). In addition to this, the collecting tubules revealed necrosis of epithelial lining, interstitial hemorrhages and cortical tubular degeneration characterized by swelling. The affected kidneys also reveal severe renal tubular degeneration and glomerular nephritis. In most of the cases, the affected kidneys revealed destruction of both parietal and visceral layers of glomerulus with its atrophy (Fig: 30). A few birds in this study, revealed loss of nuclei and indistinct cell boundary of renal tubular lining of epithelial cells with nephrotic changes which were more severe in subcapsular regions. Moreover there was cellular infiltration in the glomerulus with increased Bowman's space observed in the affected kidneys. The blood vessels within the kidney may show congestion and hemorrhage due to increased permeability and inflammation. The microscopic examination further revealed inflammation of the interstitial tissue surrounding the renal tubules which is characterized by infiltration of inflammatory cells, such as lymphocytes and macrophages. The necrotic renal tubules may contain cellular debris and inflammatory cells. Depending on the severity of the infection, glomerular changes may also be observed, including congestion, proliferation of glomerular cells and infiltration of inflammatory cells. Some severe cases of Newcastle disease reveal expansion of the renal tubules with massive influx of inflammatory cells.

Heart: Myocarditis is a common feature of Newcastle disease which is characterized by infiltration of inflammatory cells, such as lymphocytes, heterophils and macrophages, within the

myocardial tissue. There may be degenerative changes in the myocardial cells also, such as vacuolation, swelling loss of striations and disruption of myocardial fibers (Fig: 31). Myocarditis can range from mild to severe, depending on the stage and severity of the disease. The microscopic examination further revealed necrotic changes in the heart, leading to the death of heart muscle cells (Fig: 32). The severe cases of Newcastle disease can lead to hemorrhage and necrosis of myocardial cells. This can be observed as areas of coagulative necrosis or loss of cellular architecture. Necrosis can be observed as areas of cellular disintegration or loss, often accompanied by infiltration of inflammatory cells. Newcastle disease can result in vascular damage within the heart, leading the leakage of blood into the cardiac tissue. The hemorrhagic lesions may be observed as areas of reddish discoloration within the heart. The edema in the myocardial tissue may also occur as a result of inflammation and vascular changes associated with Newcastle disease. In chronic cases of Newcastle disease, the heart may undergo fibrotic changes which is characterized by the deposition of excess connective tissue, leading to scarring and impaired heart function.

Crop: The crop may exhibit inflammation and degenerative changes in its muscle fibers which can range from mild to severe (Fig: 33). The inflammatory cell infiltration, including lymphocytes, plasma cells, macrophages, and heterophils, may be observed in the affected crop tissue. The crop's epithelial lining may show various alterations, including hyperplasia, hypertrophy and degenerative changes. These changes can disrupt the normal architecture of the crop epithelium. The severe cases of Newcastle disease can lead to necrotic changes in the crop (Fig: 34). This may result in the formation of ulcers or erosions on the surface of the crop. The inclusion bodies are present within the affected crop cells. The Edema and hemorrhage can also occur in the crop tissue, leading to swelling and discoloration. In early stages of Newcastle disease, the crop may show increased blood flow, resulting in congestion and dilation of blood vessels. This can be observed as red discoloration of the crop tissue. The presence of Newcastle disease virus can stimulate the immune response in the crop, leading to the enlargement and hyperplasia of lymphoid follicles. This can be observed as increased size and prominence of lymphoid follicles in the crop tissue. In some cases, heterophilic granulomas may be observed in the crop. These are aggregations of heterophils that form in response to the infection. Some severe cases of Newcastle disease can lead to ulceration of the crop lining which can manifest as areas of denuded epithelium, erosion and ulcer formation. The chronic cases of Newcastle disease can lead to fibrosis in the crop. Fibrosis is characterized by the deposition of collagen fibers in the affected tissue.

Gizzard: The glandular epithelium lining the gizzard may show degenerative changes, such as vacuolation, loss of normal cellular architecture and cell death. This can result in the disruption of normal glandular function. The microscopic examination further of the affected crop revealed congestion with thickening of the wall of the blood vessel (Fig: 35). In response to the viral infection, an inflammatory reaction may occur in the gizzard. The Infiltration of inflammatory cells, such as lymphocytes, heterophils, and macrophages, may also be observed in the affected tissue. The gizzard may exhibit edema and hemorrhage due to the increased permeability of blood vessels caused by the viral infection. These changes can lead to the thickening and congestion of the gizzard wall. The microscopic examination also revealed hyperplasia of the gizzard epithelium, leading to the thickening of the mucosal lining. Additionally, ulceration, erosion and necrosis of the gizzard may also be observed under microscopic examination (Fig: 36). The lymphoid tissues such as the lamina propria and submucosa of the gizzard, may exhibit lymphoid hyperplasia and lymphocytic infiltration in response to the viral infection. The multinucleated gaint cells commonly known as Syncytial cell are also observed in the gizzard epithelium which are formed due to the fusion of infected cells and are characteristic of Newcastle disease. In severe cases, intranuclear inclusion bodies may be present within the affected cells. These inclusion bodies are aggregates of viral proteins and are indicative of Newcastle disease infection.

Intestine: The microscopic examination of the Intestines revealed marked degeneration, necrosis and denudation of mucosa. The affected villi revealing Villus blunting with Inflammatory infiltrates (Fig: 37). The submucosal oedema and mononuclear infiltration in lamina propria was also noted**.** The affected tissue also revealed necrosis of the intestinal lining, leading to the formation of ulcers. The affected areas may appear yellowish and have a fibrin purulent exudate. The microscopic examination further revealed villous atrophy leading to decrease in the surface area. The Inflammatory cells, such as lymphocytes and heterophils, can infiltrate the lamina propria of the intestine. This infiltration is often associated with the necrotic areas and ulcers. In response to the viral infection, there may be an increase in the size of intestinal glands which is a compensatory mechanism to repair the damaged epithelium. The presence of lymphocytes within the epithelial layer of the intestine is another characteristic feature. These lymphocytes may disrupt the normal architecture of the epithelium. In severe cases of Newcastle disease, hemorrhage may be observed in the intestinal mucosa which might be resulted from the damage to the blood vessels within the affected areas. The microscopic examination further revealed fragmentation and fusion of intestinal villi (Fig: 38). In addition to this, epithelial necrosis and sloughing of epithelium may also be observed in the affected intestines. In severe cases, fibrinonecrotic material may be present on the surface of the intestine, along with the formation of ulcers. These ulcers can disrupt the normal structure of the intestinal mucosa.

Gross lesions on Different Organs

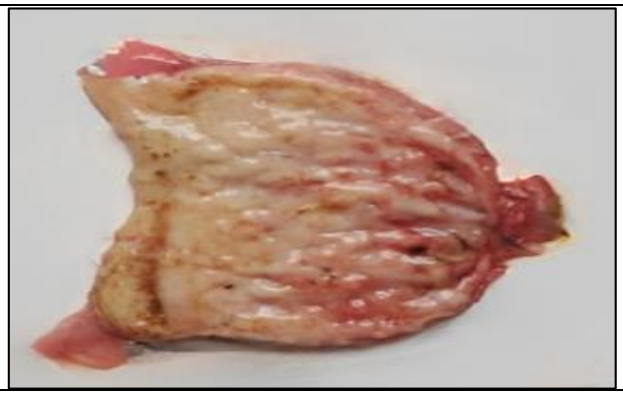

Fig 5: Photograph revealing hemorrhagic lesions found on the surface of the proventriculus

Fig 6: Photograph revealing fibrin deposition on the surface of the proventriculus

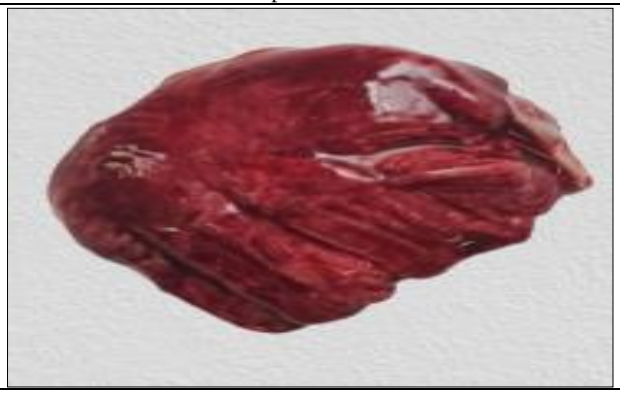

Fig 7: Photograph revealing Congested and Consolidated lungs

Fig 8: Photograph revealing cardiac enlargement and hemorrhagic areas on the surface of heart

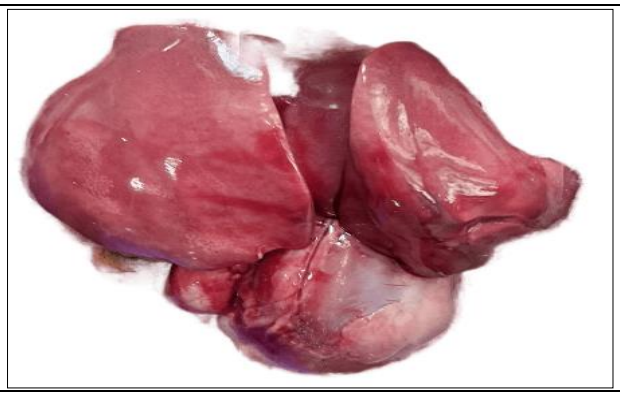

Fig 9: Photograph revealing dark appearance of the liver due to congestion

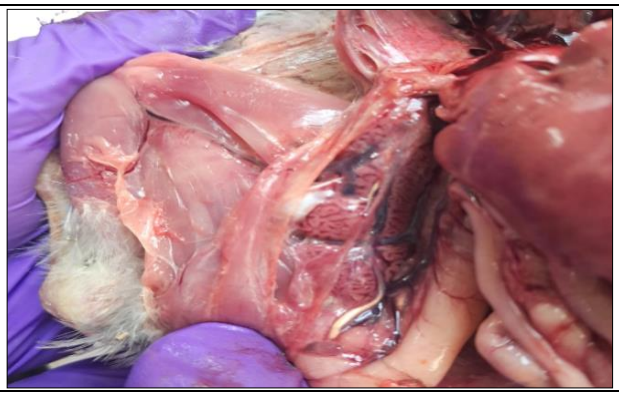

Fig 10: Photograph revealing swollen and enlarged Kidneys

Fig 11: Photograph revealing enlarged and mottled spleen

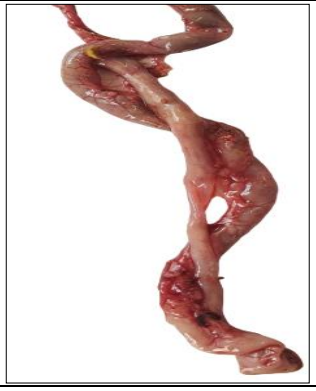

Fig 12: Photograph of intestine revealing haemorrhages and ulceration

Histopathology

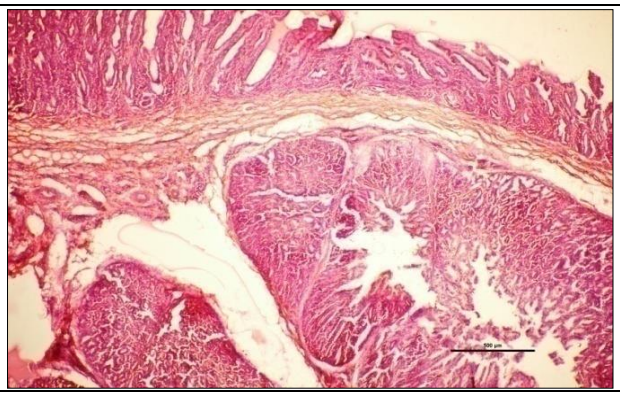

Fig 13: Photomicrograph of Proventriculus revealing erosions and sloughing off proventicular epithelium (H&E 10X)

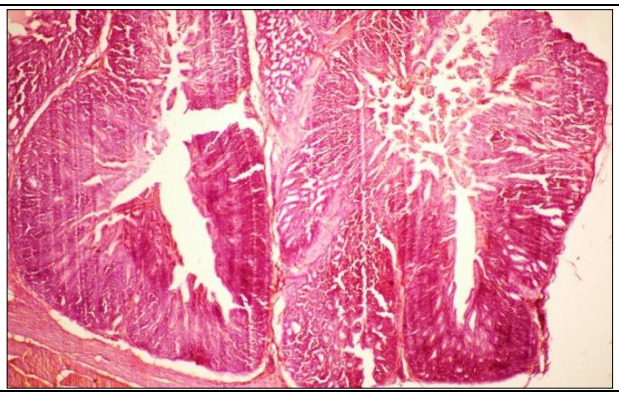

Fig 14: Photomicrograph of Proventriculus revealing congestion and degeneration in the affected tissue (H&E 10X)

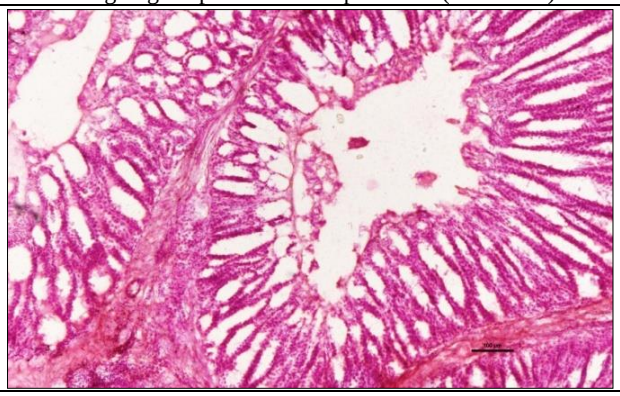

Fig 15: Photomicrograph revealing necrosis in the Proventricular Submucosal Gland (H&E 10X)

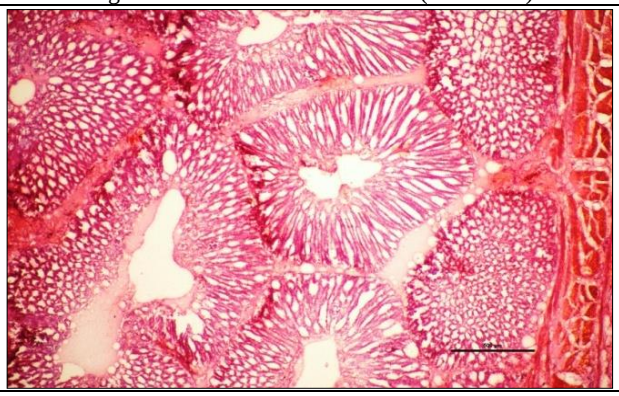

Fig 16: Photomicrograph of Proventriculus revealing hemorrhage indicating vascular damage (H&E 10X)

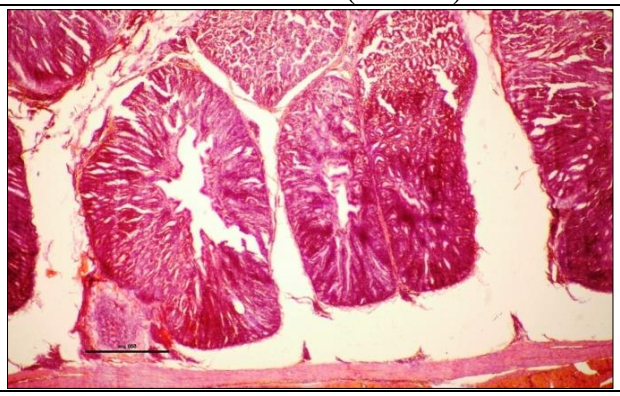

Fig 17: Photomicrograph of Proventriculus revealing atrophy of proventricular gland (H&E 10X)

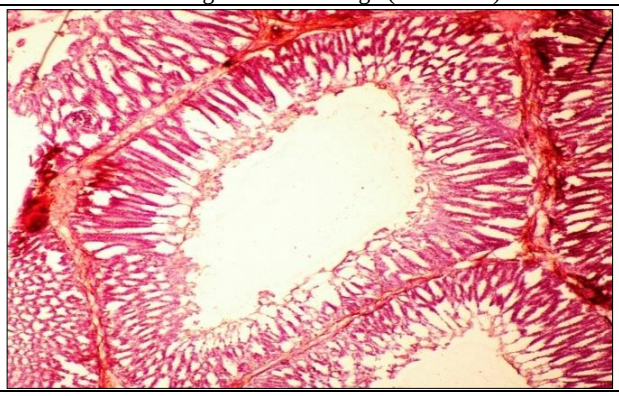

Fig 18: Photomicrograph of Proventriculus revealing loss of glandular architecture with hemorrhage (H&E 10X)

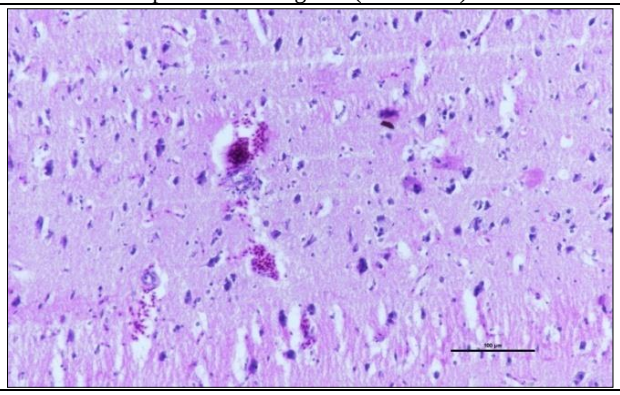

Fig 19: Photomicrograph revealing Neuronal *satellitosis in the brain* (H&E 10X)

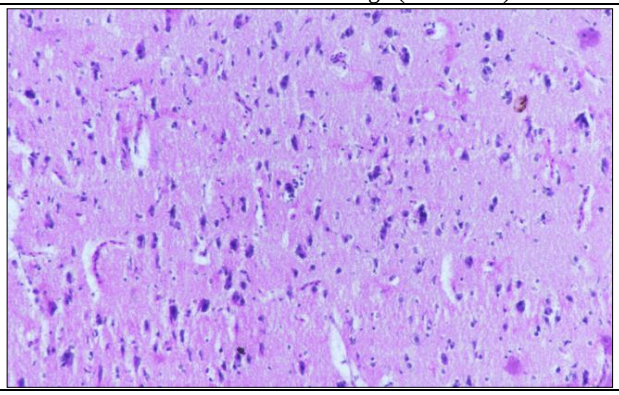

Fig 20: Photomicrograph of Brain revealing neuronal necrosis (H&E 10X)

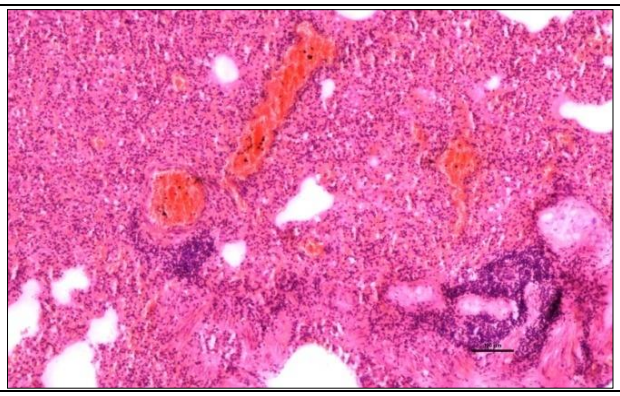

Fig 21: Photomicrograph of Lung revealing severe perivascular heterophilic infiltration (H&E 10X)

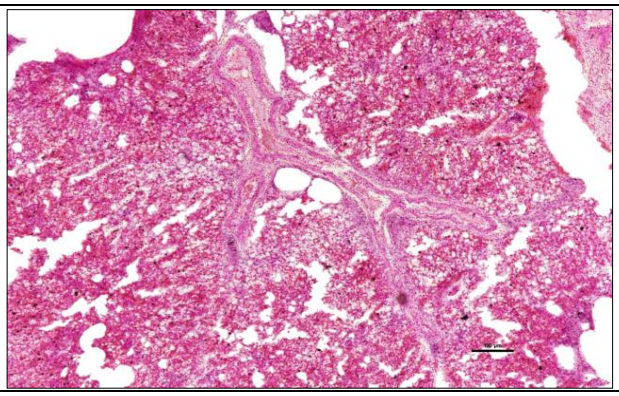

Fig 22: Photomicrograph of Lung revealing hemorrhage and severe degeneration in the affected tissue section (H&E 10X)

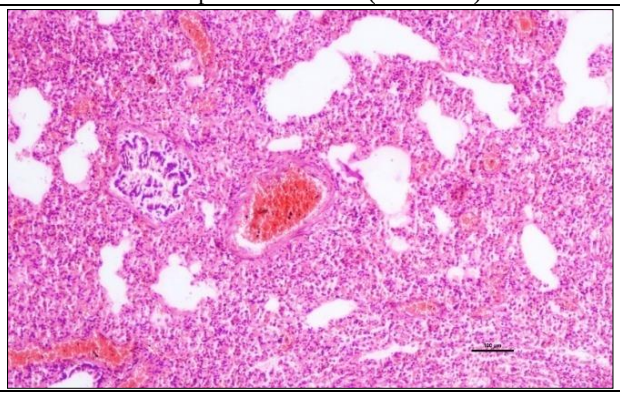

Fig 23: Photomicrograph of Lung revealing congestion with mild cellular infiltration (H&E 10X)

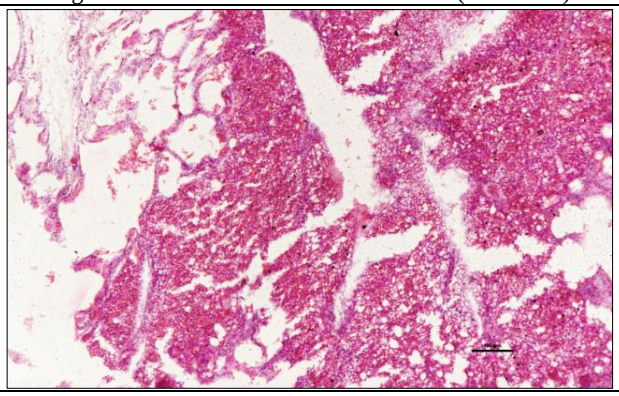

Fig 24: Photomicrograph of Lung revealing necrotic changes with hemorrhage (H&E 10X)

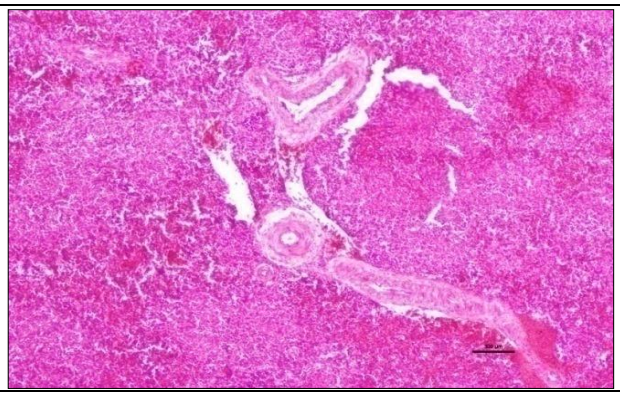

Fig 25: Photomicrograph revealing lymphoid depletion within the spleen (H&E 10X)

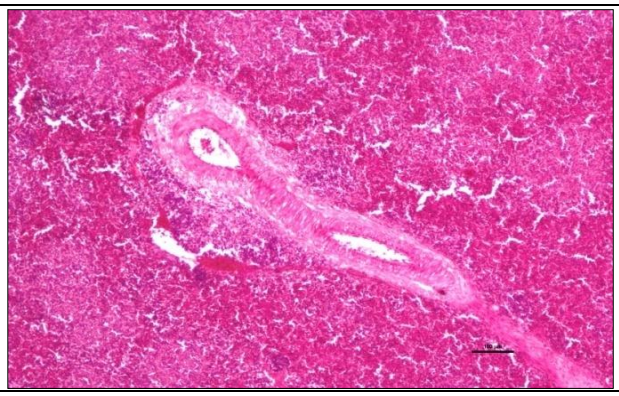

Fig 26: Photomicrograph revealing Necrotic areas in the spleen (H&E 10X)

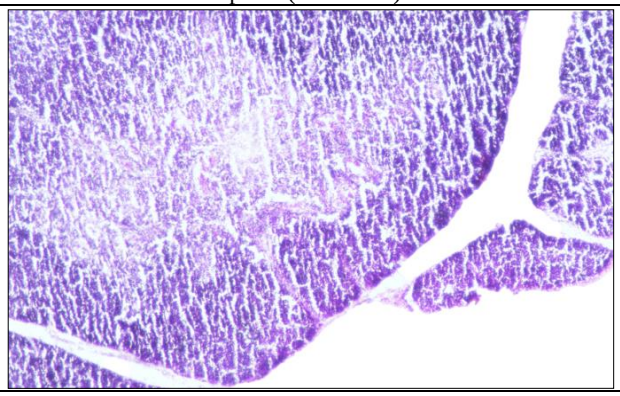

Fig 27: Photomicrograph revealing hypocellularity of the lymphoid follicles of Thymus (H&E 10X)

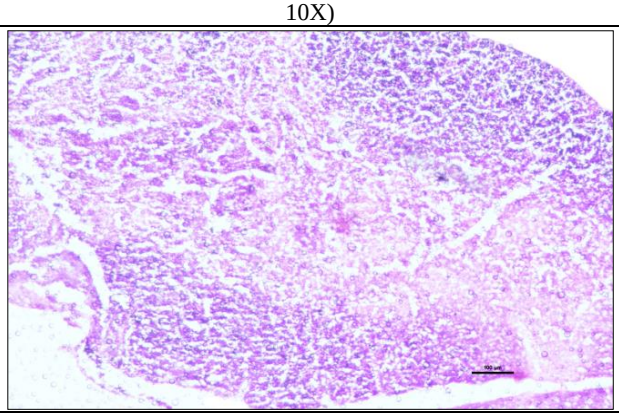

Fig 28: Photomicrograph of Thymus revealing necrosis with severe lymphoid depletion from thymic follicles (H&E 10X)

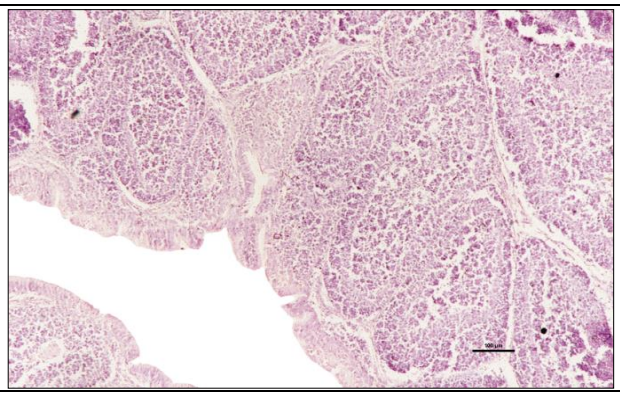

Fig 29: Photomicrograph revealing depletion of lymphoid tissue from the lymphoid follicles of Bursa of Fabricius (H&E 10X)

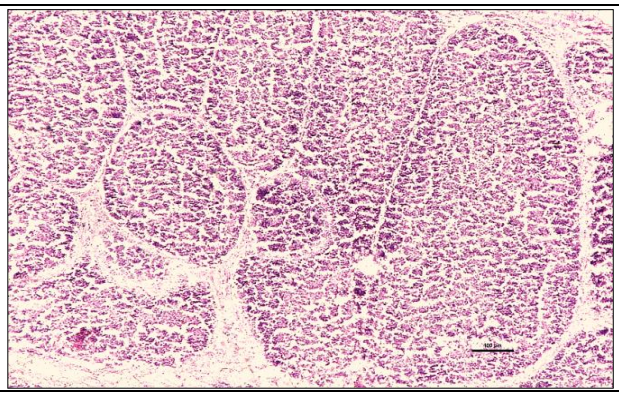

Fig 30: Photomicrograph revealing reduction in the size of the bursal follicles (H&E 10X)

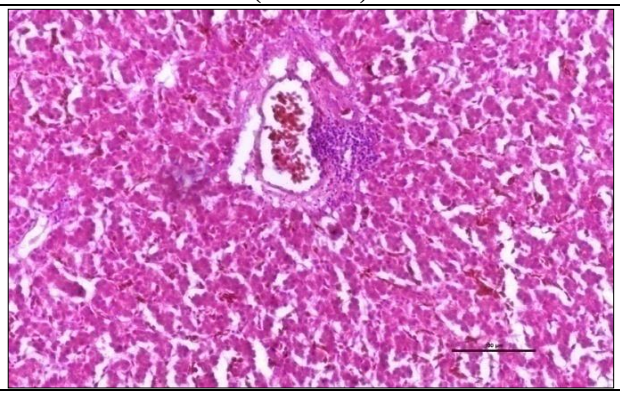

Fig 31: Photomicrograph of Liver revealing engorged and dilated central vein with blood and perivascular infiltration (H&E 10X)

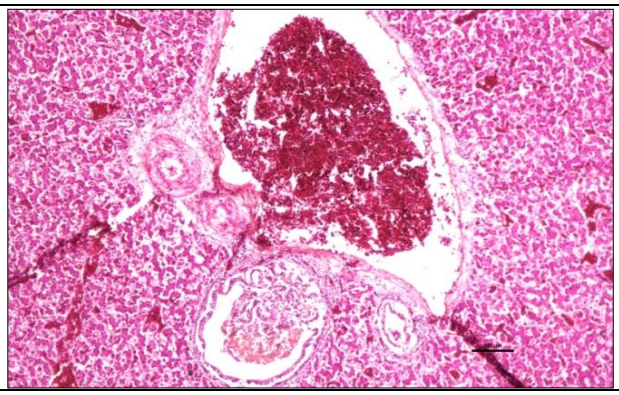

Fig 32: Photomicrograph of *Portal triad of* Liver revealing dilated portal vein with engorgement of blood (H&E 10X)

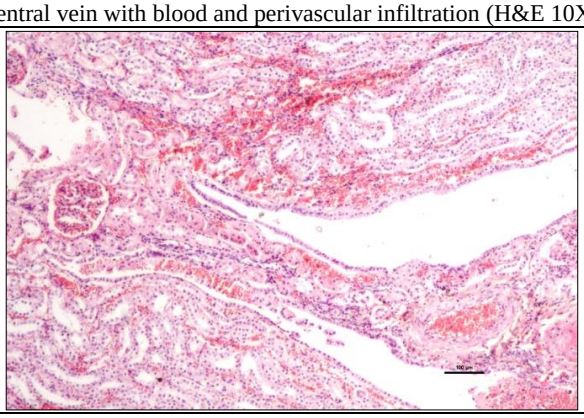

Fig 33: Photomicrograph of Kidney revealing hemorrhage with severe cellular infiltration in the interstitium (H&E 10X)

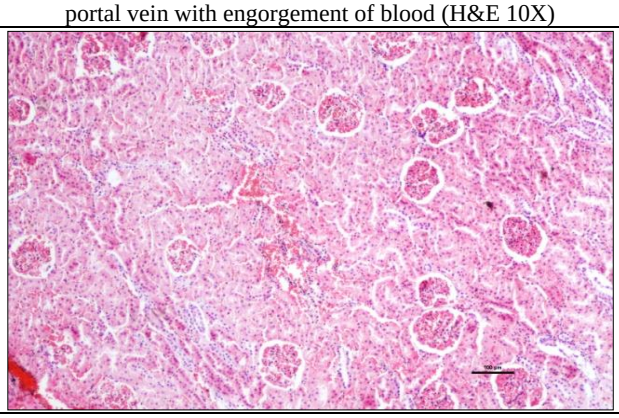

Fig 34: Photomicrograph of Kidney revealing glomerular atrophy as evidenced with increased Bowman's space (H&E 10X)

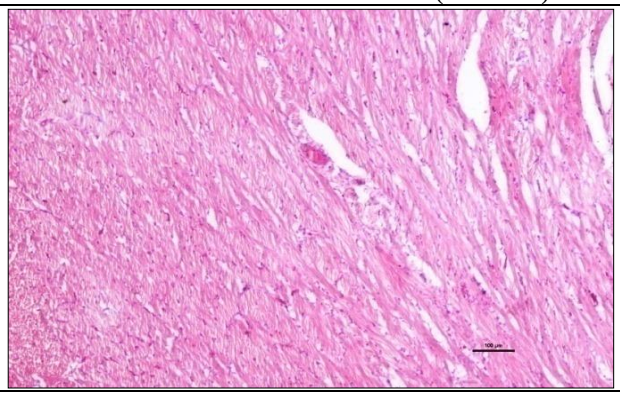

Fig 35: Photomicrograph of Heart revealing separation and disruption of myocardial fibers (H&E 10X)

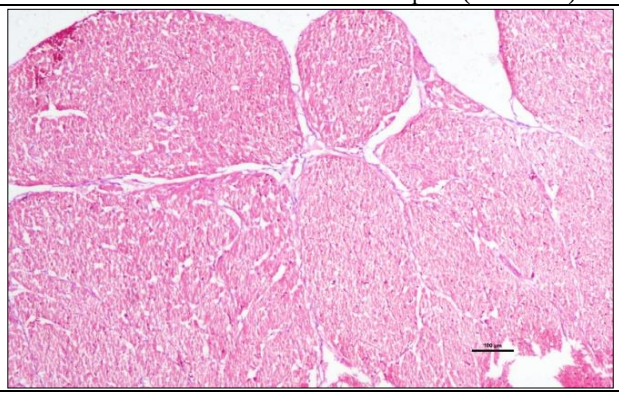

Fig 36: Photomicrograph revealing necrotic changes within the muscle fibers of heart (H&E 10X)

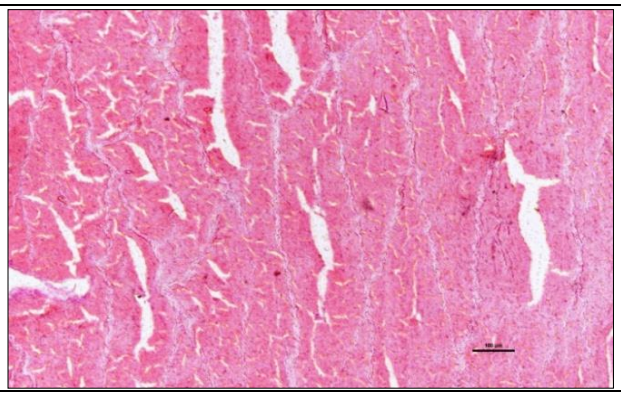

Fig 37: Photomicrograph of Crop revealing degenerative changes in the muscle fibers (H&E 10X)

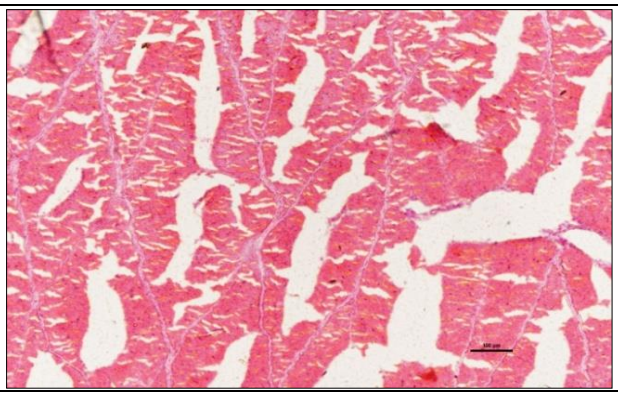

Fig 38: Photomicrograph of Crop exhibit necrotic areas within the muscle fibers (H&E 10X)

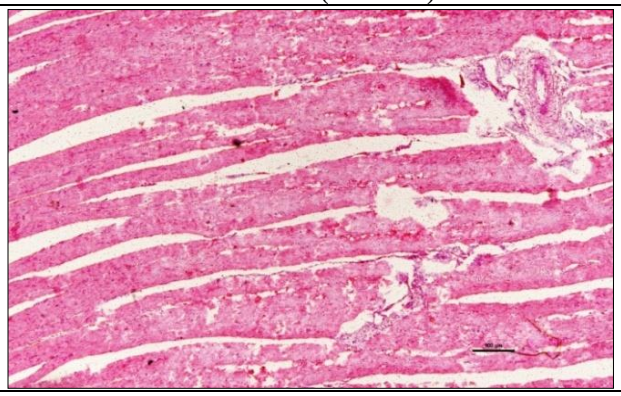

Fig 39: Photomicrograph of Gizzard revealing congestion with thickening of the wall of the blood vessel (H&E 10X)

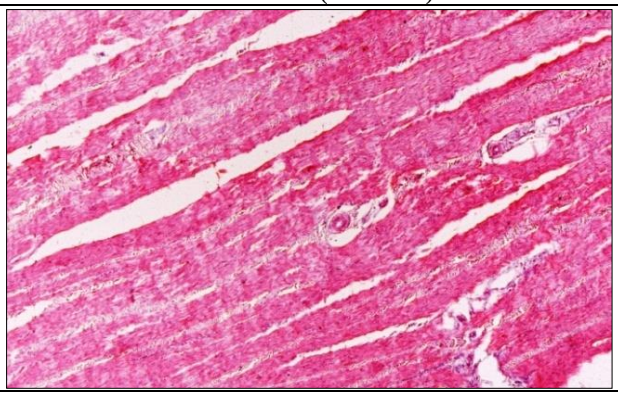

Fig 40: Photomicrograph of Gizzard revealing necrosis and disruption of muscle fibers (H&E 10X)

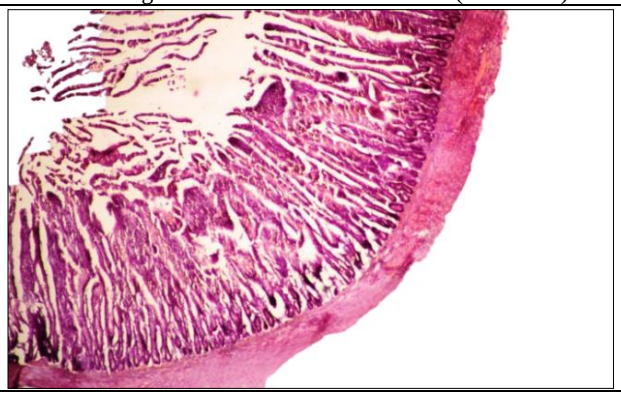

Fig 41: Photomicrograph of Intestine revealing Villus blunting with Inflammatory infiltrates (H&E 10X)

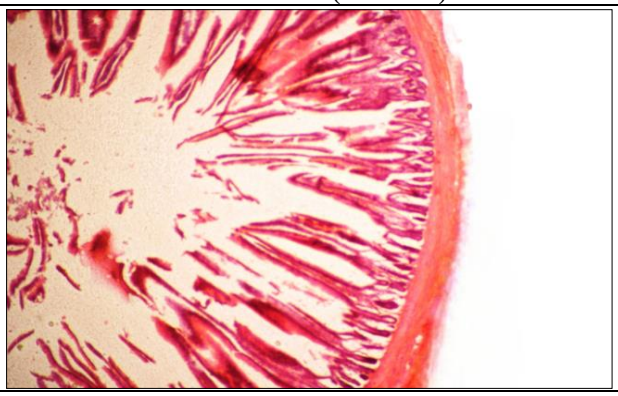

Fig 42: Photomicrograph of Intestine revealing fragmentation and fusion of intestinal villi (H&E 10X)

Discussion

Newcastle disease is also known as Ranikhet disease and is highly contagious in nature affecting many commercial and backyard poultry farms. The disease is caused by infections with virulent avian avulavirus 1, commonly known as Newcastle disease virus (NDV) and designated as avian paramyxovirus-1 (APMV-1). It causes high morbidity and mortality in non-vaccinated birds. Apart from commercial and backyard poultry, a wide range of captive and free living birds are also susceptible, that can sometimes act as primary source of Newcastle disease infection to chicken. The disease has also been reported in pigeons that occurs due to the spread from diseased chicken flocks, and can occur vice versa from domesticated or feral pigeons to poultry (Alexander *et al*., 1984) [20]. The clinical symptoms observable originating from the nervous system was caused by nerve cell destruction in the brain from infection and NDV replication. The presence of NDV within brain can cause vascular and neuron damage which will result in an inflammatory response. Inflammatory response begun by macrophrage spread in perivascular cuffing from which then spread to surrounding astrocytes and microgial (Zachary *et al*., 2012) [19]. NDV distribution in nervous organ system was identical with a previous report (Ecco *et al*., 2011) [9] which stated that encephalitis was found in infected chicken brain. The Hb, PCV and TEC in the affected broiler chickens were low as compared to normal broiler chickens in this study (Adeyemo *et al*., 2013; Chekwube *et al*., 2014; Calderon *et al*., 2005) [3, 5, 6]. In this study, Total Leukocyte Count Values were found significantly higher than the normal healthy chickens (Calderon *et al*. 2005**,** Harrison *et al*., 2011) [6, 12].

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inappetence, huddling together, drooping of wings, laboured breathing, lowering of head, ruffiness of feathers and weakness. The diarrhoea was apparent in birds in early stage of disease and the droppings were yellow in colour The affected birds which survived for few days revealed retarded growth stood motionless for long period of time with the feathers puffed and both eyes closed.

The pathological alterations like multifocal haemorrhages around proventricular glands and ulcers were observed throughout the intestine of the affected birds (Saidu *et al* 2009) [16]. The petechial hemorrhages and necrosis present within the mucosal layer of the proventriculus might be caused due to damage to the blood vessels caused by the virus and the associated inflammatory response. The brain exhibits redness swelling, encephalitis, satellitosis and neuronophagia. The heart may appear larger as compared to normal due to the accumulation of blood and known as cardiomegaly. The lungs revealed consolidation and pneumonia (Akamura *et al*. 2008) [2]. The liver surface revealed mottled appearance with patches of red discoloration which might be caused due to hemorrhage and congestion. The microscopic examination of the affected liver tissue section revealed marked disorganization of hepatic architecture, congested central veins, dilated blood sinusoids and perivascular infiltration (Wang *et al*. 2012) [18] The affected kidneys revealed congestion, glomerular atrophy, dilatation of renal tubules, fragmentation of glomeruli associated with nephritis (Kim *et al* 2007) [14]. The gross and histopathological lesions observed lymphoid organs lymphoid organs like caecal tonsils, spleen, bursa, thymus and bursa of fabricius (Hu *et al*. 2012) [13]. The outbreaks of this disease immunosuppression in the poultry birds (Ezema *et al*. 2009) [10]. There may be the development of ulcers and erosions on the inner lining of the crop observed in the Newcastle disease, which may vary in size and can lead to the formation of raw wounds. The gizzard may also show the erosions which are shallow defects in the gizzard lining that are less severe than ulcers (Alexander *et al*. 2000) [1]. The severe hemorrhages were observed in the affected intestinal mucosa and submucosa which may appear as pinpoint spots and larger patches of blood within the intestinal wall. The microscopic examination further revealed submucosal oedema and mononuclear infiltration in lamina propria was also noted. The affected tissue also revealed necrosis of the intestinal lining, leading to the formation of ulcer (Dimitrov *et al*. 2016) [8]

Conclusion

In conclusion, Newcastle disease is a significant viral disease of poultry birds with varying forms of pathology depending on the severity and organs affected. Understanding the pathology of Newcastle disease is essential for its diagnosis, control and prevention in affected bird populations. The effective biosecurity measures, vaccination and proper management practices are key to minimizing the impact of this disease on the poultry industry.

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