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Mortality in some domesticated pigeons (*Columba livia*) from Jammu, India

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Abstract: Although pigeons are reared for recreational purposes in Jammu region, disease occurrence is sparsely documented. Based on clinical signs, lesions and laboratory investigation, approximately 600 out of 4000 (15%) domestic pigeons were found to be disease affected. Necropsy of 60 birds was conducted to determine the cause of death. *Haemoproteus columbae* demonstrated within erythrocytes and various development stages in tissue and endothelial schizogony caused majority deaths due to pneumonia and hepatitis (45%) accompanied by severe vascular and tissue reaction. Newcastle disease (20%) confirmed by positive serum haemagglutination inhibition manifested in a severe neurological form with characteristic cerebral malacia and demyelination. Mycotoxicosis (13.33%) detectable in tissue and feed samples were associated with typical hepatic and renal degeneration. Aspergillosis (8.33%) was associated with severe granulomatous pneumonia and systemic affections. *Aspergillus fumigatus* could be isolated from deep tissues. Pigeon pox (3.33%) associated with severe cutaneous and diphtheritic form. Traumatic injury (6.67%), salmonellosis and colibacillosis (1.67% each) were other causes of mortality. The study summarizes pigeon mortality pattern of this region andhighlights the warranted health management of these birds. The potential for pathogen dissemination in the environment, to other susceptible hosts and possibly across International borders by free flying birds have been emphasized.

Key words: *Columba livia*, mortality, pathology, pigeon

1. Introduction

Pigeons are amongst the most prevalent and readily observable birds in all provinces of India. Besides constituting a small part of human food, they are used for ornamental or exhibiting purposes (fancy pigeons), pets, sport activities, and as bio-experimental models. Jammu region is traditionally home to many pigeon fanciers with a rich historical and anecdotal background. In spite of their charming qualities, pigeons are often regarded to be closely associated with filth and disease [1]. Pigeons are subject to contracting many serious diseases, produced by viruses, rickettsiae, bacteria, fungi, protozoa, helminths and arthropods. Besides, they themselves might be potential carriers of numerous harmful pathogens and thus constitute a major source of infection and transmission of diseases [2] some of which may contribute to serious public health hazards. Even then, pigeon fanciers around the world rarely seek veterinary advice, in part as a commercial decision or based on the value of a racing pigeon [3].

Although mortality amongst pigeons is common due to a variety of disease conditions, however, the disease status of the birds in this region is largely unknown and unreported. As no systemic study was previously carried out in these birds, it is thus envisaged that an investigation through the present study would identify various pathogens prevalent amongst the pigeon population. It is also appropriate and contextual considering the adjacent International Indo-Pakistan border where the potential and possible trans-boundary disease dissemination exists.

2. Materials and methods

In the present study, a total of approximately 4000 domestic pigeons were screened for disease affection based on flock history, clinical examination of signs and lesions, and laboratory investigation. The birds were reared for sports and recreational activity by their owners. The study encompassed twelve areas within Jammu district, out of which ten were in the RS Pura sector and two were within the Jammu city limits. The areas near RS Pura were very close to the International Indo-Pak border (approximately between 1 km and 10 km in distance), and birds were often found to fly across the international boundary. They

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were housed on rooftops/terraces in a wire mesh enclosed area consisting of a run and a small covered area housing wooden crates for laying and brooding. The top of the wired enclosure opened with a trap for birds to fly in or out.

The study was undertaken under approval of the Institutional Animal Ethics Committee (Approval No. 862/ac/04/CPCSEA Dated 16/12/2004). Clinical samples including blood and swabs were collected from suspected birds showing symptoms or lesions of specific disease for pathological, microbiological or parasitological examination. Sixty dead birds were collected for detailed necropsy examination in the laboratory from July 2015-June 2016. Reported mortality by owners was not accounted for, as the data was very arbitrary and vague. Mortality was therefore accounted only for birds that were accessed for necropsy examination. Tissues were collected in 10% formal saline solution for histopathology and paraffin embedded sections were prepared and stained with hematoxylin and eosin as per standard protocol.

Isolation and identification of coliform bacterial culture was followed using appropriate bacteriological culture media [4] and morphological characteristics of bacteria and fungi as per Cruickshank et al. [5]. The isolation of pigeonpox virus from clinical specimens was done in embryonated egg via chorioallantoic membrane (CAM) route as described by OIE[1]. Haemagglutination (HA) and haemagglutination inhibition (HI) assay was done according to OIE[2]. HI assay was done using 4HAU titre of NDV vaccine to detect circulating antibody titre in serum of affected/convalescent birds. Haemoprotozoa were identified according to Valkiunas [6]. Feed samples suspected for presence of mycotoxins were pooled and collected. Approximately 500 g of each feed sample, and approximately 200 g of fresh pooled tissues from kidneys, lungs and livers each were collected and sent in saturated salt solution (sodium chloride) to Pharmacovigilance Laboratory for Animal Feed and Food Safety, TANUVAS, Chennai for mycotoxin screening using high performance thin layer chromatography (HPTLC) and estimation using high performance liquid chromatography (HPLC).

3. Results

Approximately 600 birds (15%) were found to be affected with some clinical form of disease or other. A detailed necropsy was conducted for 60 pigeons in the place of study from July 2015-June 2016. The causes of death as diagnosed by necropsy examination and confirmed by laboratory methods are represented in Table.

3.1. Protozoal pneumonia and hepatitis

In a severe disease outbreak, high mortality was reported amongst pigeons. Haemoprotozoans were detected in the blood smears of 8 out of 30 sick or apparently normal birds within RBCs, with elongated and crescent shaped intracorpuscular gametocytes morphologically consistent with *Haemoproteus columbae*. Clinically, signs of severe respiratory distress, anorexia, and malaise, loss in body weight, dehydration, stupor and heavy mortality (Figure 1A) were noticed. A total of 27 bird carcasses were necropsied where all had severe and consistent hepatic and pneumonic lesions. The liver appeared congested, with miliary pinpoint grey-white foci throughout the parenchyma (Figure 1B). Hydropericardium was noticed in three birds (Figure 1C). Lungs were sometimes haemorrhagic (Figure 1D) or severely congested (Figure 1E). Sometimes, subcutaneous haemorrhage was noted beneath the skin of the cervical region (Figure 1F). The spleen was enlarged and the kidneys were pale with visible distension by urates.

Microscopically, lung tissue of the infected pigeons revealed various stages of protozoan parasite development. Severe congestion was seen in the lung tissue (Figure 1G). Pulmonary blood vessels which were dilated and congested and schizonts could be detected within the endothelial lining (Figure 1H). Areas of complete destruction of the lung parenchyma or marked dilatation of the pulmonary airspaces were observed. There was severe infiltration by mononuclear cells in the interstitium. Typical round cell granuloma was seen around blood vessels.

Histopathological examination of the liver showed multifocal and coalescing lesions of subacute hepatic necrosis with marked infiltrates of macrophages, lymphocytes, fewer plasma cells and, there were encountered scanty heterophils concentrated in the perivascular spaces around protozoal development stages in the portal area (Figure 1I), sinusoids (Figures 1J and 1K) and central vein (Figure 1L). The predominantly infiltrative round cell was characteristic. Parasitic schizonts were also seen invading the portal blood vessels, central vein and the endothelial lining of the hepatic sinusoids. Schizonts were seen to invade through the endothelial lining and into the liver parenchyma. Areas of hepatocyte degeneration with vacuolated cytoplasm and pyknotic nuclei were frequently detected. Small round cell granulomas have also been observed especially near areas of massive hepatocyte degeneration. In some cases fragmented and round forms of cellular debris was observe

[1] World Organisation for Animal Health (2018). Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2019. Fowl pox, Chapter 3.3.10. [online]. Website https://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/3.03.10_FOWLPOX.pdf [accessed 04 March 2019].

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Table. Cause of mortality in some pigeons due to various pathological conditions.

S.No.	Pathological condition	Number of dead birds	Per cent (%)
1.	Haemoproteosis	27	45.00
2.	Newcastle disease	12	20.00
3.	Mycotoxicosis	8	13.33
4.	Aspergillosis	5	8.33
5.	Traumatic injury	4	6.67
6.	Pigeon pox	2	3.33
7.	Salmonellosis	1	1.67
8.	Colibacillosis	1	1.67
Total		60	100.00

3.2. Newcastle disease

Sporadic outbreaks of suspected Newcastle disease (ND) infection have been occasionally presented. Clinical signs and symptoms observed in the flocks were predominantly neurological. They included depression, mild to severe tremors, torticollis, incoordination, unilateral or bilateral paresis with paralysis of wings or limbs (Figure 2A), head pressing, circling and terminal coma. Sick birds were unthrifty and anorexic, often emaciation and dehydrated. Some birds were often found to void greenish watery excreta.

Necropsy examination of twelve birds variously showed petechial haemorrhages on proventricular mucosa with ulcerations (Figure 2B), serosa and mucosa of the ventriculus (Figure 2C), on the crop, catarrhal to haemorrhagic enteritis (Figure 2D), congestion and haemorrhages on the trachea and lungs, pancreas, epicardium (Figure 2E), congestion of meninges and brain (Figure 2F). Ulceration was also visible on the crop, gizzard and intestines. In a severe case of NCD affection, a large haematoma was seen in the left cerebral hemisphere (Figure 2G).

Microscopically, congestion, oedema, haemorrhage in trachea and lungs were seen. In the intestines, haemorrhagic and ulcerative lesions were seen. The submucosa was filled with infiltrating cells. Necrosis of lymphoid follicles in the intestine was also seen.

In the nervous system, nonsuppurative encephalitis was observed in all cases affected with neuronal signs. The predominant microscopic lesion was congestion and haemorrhages in the meninges and parenchyma (Figure 2H). Most parts of the neuronal tissue appeared degenerated. Meningeal oedema was also sometimes observed. There was also extensive malacia (Figure 2I) and demyelination (Figures 2J and 2K). In malacic lesions, there was frank destruction of neurons and the surrounding neuropil was oedematous. Demyelinating lesions seen mainly in the midbrain and brain stem were characterized by small to medium sized foci of vacuolation giving a spongy appearance. Marked multifocal perivascular cuffing with lymphocytes, plasma cells and microglia-like cells were observed. Oedema and perivascular rarefication could also be discerned. Satellitosis, microgliosis and gitter cell infiltration were also seen (Figure 2L).

Serum samples collected from 10 out of 12 affected pigeons were found positive by HI test by detection of high titres (1/32 to 1/64) of circulating antibodies greater than 2^4 or Log_2 4 against 4 HAU of paramyxovirus antigen.

3.3. Pigeon pox

The affected birds were emaciated and anorectic. Easily recognizable scabby lesions were found on the unfeathered parts of the body especially the beak and eyelids in the cutaneous form of the disease amongst many pigeons. Only two mortalities amongst young birds were observed with both cutaneous and a severe diphtheritic form of the disease (Figure 3A).

Grossly, on the mucous membrane of the mouth and oesophagus, a yellow, cheesy, necrotic and diphtheritic membrane was observed. Peeling the membrane exposed a raw bleeding surface. In one of the squabs, cutaneous scab lesions below the eye have also been observed.

Histological changes found in the skin surrounding the cutaneous lesions consisted of varying degrees of proliferative epithelium under a crust consisting of keratin and proteinaceous fluid debris and surface bacteria was present in most lesions (Figure 3B). The typical lesion was characterized by ballooning degeneration of keratinocytes containing pale vacuolated cytoplasm (Figure 3C). Numerous keratinocytes were enlarged, rounded and, frequently, contained eosinophilic A-type intracytoplasmic inclusions with central unstained zones distending the cytoplasm (Figure 3D). All proliferative changes were found in the epidermis, although some cells from the granular layer were ruptured, creating small

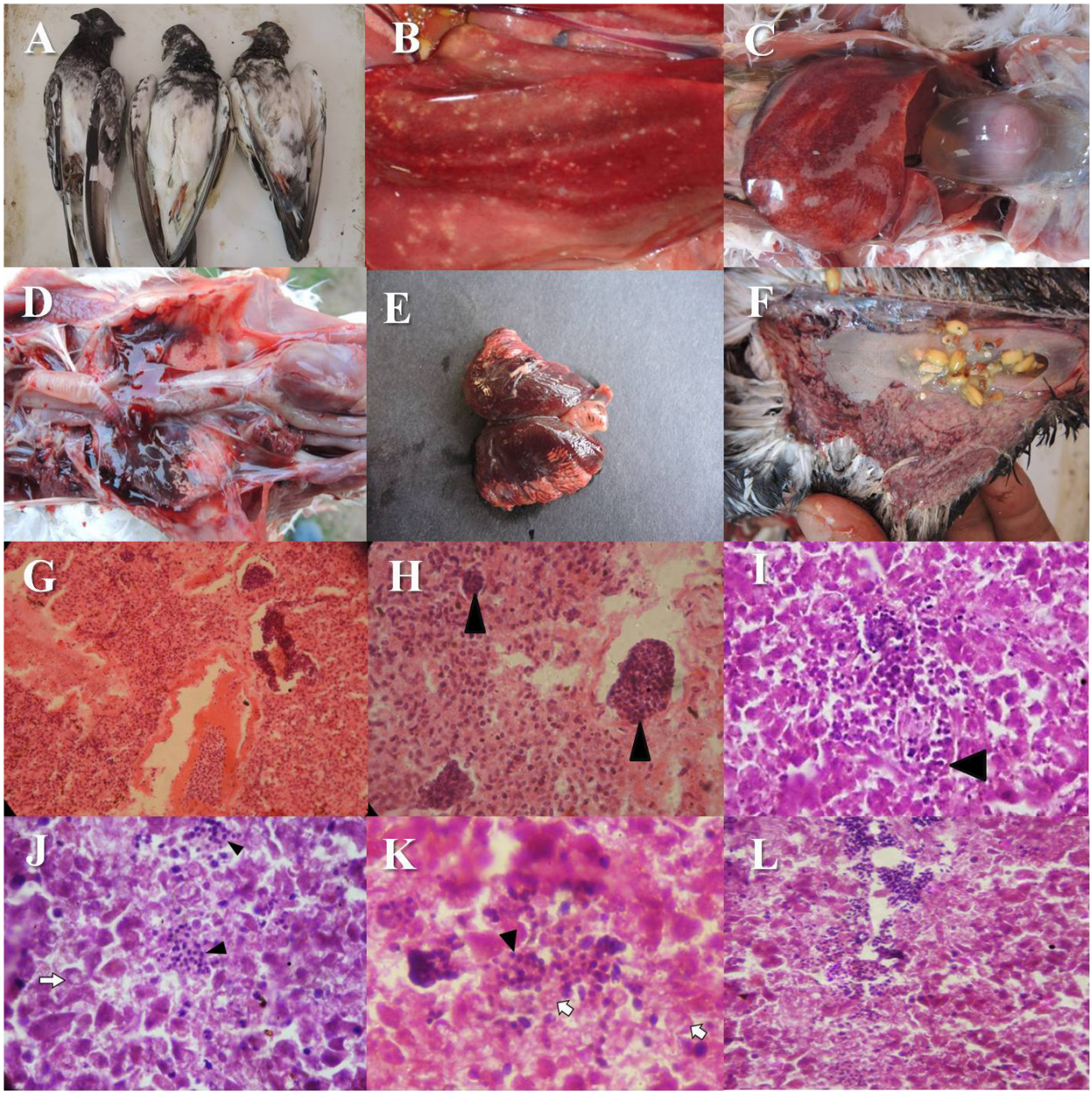

Figure 1. Tissue pathology of *Haemoproteus columbae* infection in pigeons (*Columba livia*). (A) Dehydrated and emaciated carcasses suspected for protozoal infection; (B) Congested liver, with miliary pinpoint grey-white foci in the parenchyma; (C) Congested liver and hydropericardium; (D) Severe haemorrhages in lungs; (E) Congestion and consolidation in lungs; (F) Haemorrhages beneath the skin in the cervical region; (G) Severe congestion was seen in the lung tissue. H&E, 10×; (H) Pulmonary blood vessels were dilated and schizonts could be detected within the endothelial lining (black arrowheads). H&E, 40×; (I) Developing schizonts in the portal vascular endothelium of liver (black arrowhead). H&E, 40×; (J) Developing schizonts (black arrowhead) in hepatic sinusoids. Adjacent hepatocytes are vacuolated (white arrow). H&E, 40×; (K) Developing schizonts (black arrowhead) in hepatic sinusoids. Adjacent hepatocytes are vacuolated (white arrow). H&E, 40×; (L) Developing schizonts in endothelium of central vein of liver. H&E, 40×.

vesicles. Moderate dermal oedema and congestion, with superficial infiltrates of lymphocytes, were observed. Infiltrates extended to underlying dermis, forming focal accumulation of lymphoid cells around the dermal blood vessels and connective tissue of the dermis. Histologically, lymphocytes were the predominant inflammatory cell, with scattered heterophils seen occasionally.

In the oesophagus, severe sloughing of the mucosa and covered with fibrinous debris admixed with necrotic cells and erythrocytes were observed.

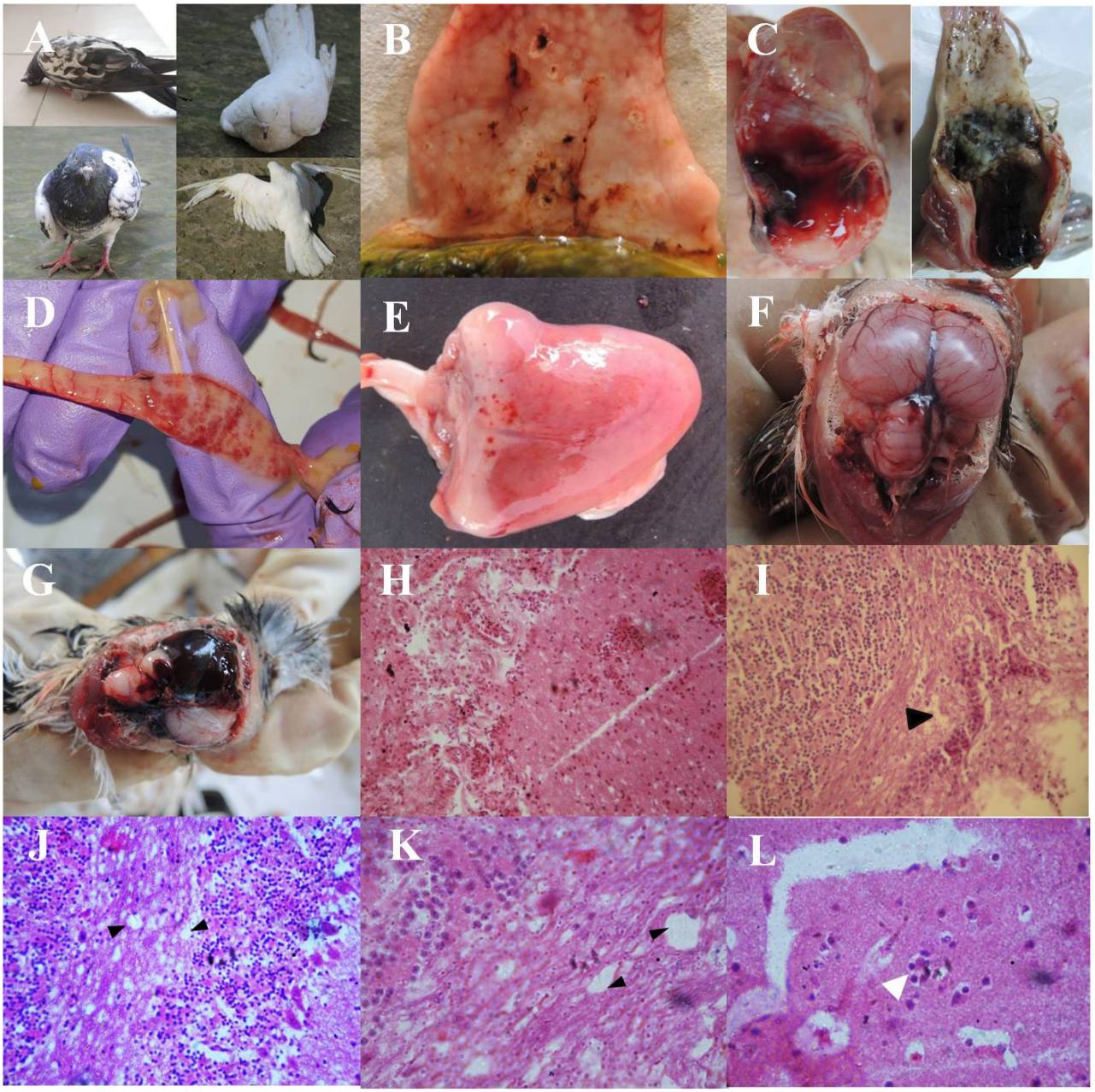

Figure 2. Signs and lesions of Newcastle disease in affected pigeons (*Columba livia*). (A) Neurological signs manifested include- severe torticollis, leg and wing paralysis in pigeon; (B) Petechial heamorrhages and ulcers on proventriculus; (C) Petechial haemorrhages on proventriculus and massive haemorrhage in the gizzard serosa (left) and mucosa (right); (D) Petechial and paint brush haemorrhages on small intestinal mucosa; (E) Petechial haemorrhages on epicardium.; (F) Congestion of blood vessels in meninges and brain; (G) Unilateral hematoma in left cerebral hemisphere; (H) Meningeal congestion and haemorrhage in brain. H&E, 100×; (I) Encephalomalacia with loss of the neurofibrillary architecture of the white matter. H&E, 100×; (J) Demyelination of cerebellar nerve tracts resembling spongiosis. H&E, 100×; (K) Demyelination of cerebellar nerve tracts in brain stem resembling vacuolation. H&E, 400×; (L) Satellitosis and neuronophagia (arrowhead). H&E, 400×.

Isolation of *Pigeon pox* virus attempted on 14th day developing chicken embryo revealed pock lesions on the CAM characterized by small white raised foci (Figures 3E and 3F).

3.4. Colibacillosis

One pigeon that had died due to severe diarrhoea had gross lesions showing severe enteritis with presence of petechial haemorrhages on the intestinal mucosa,

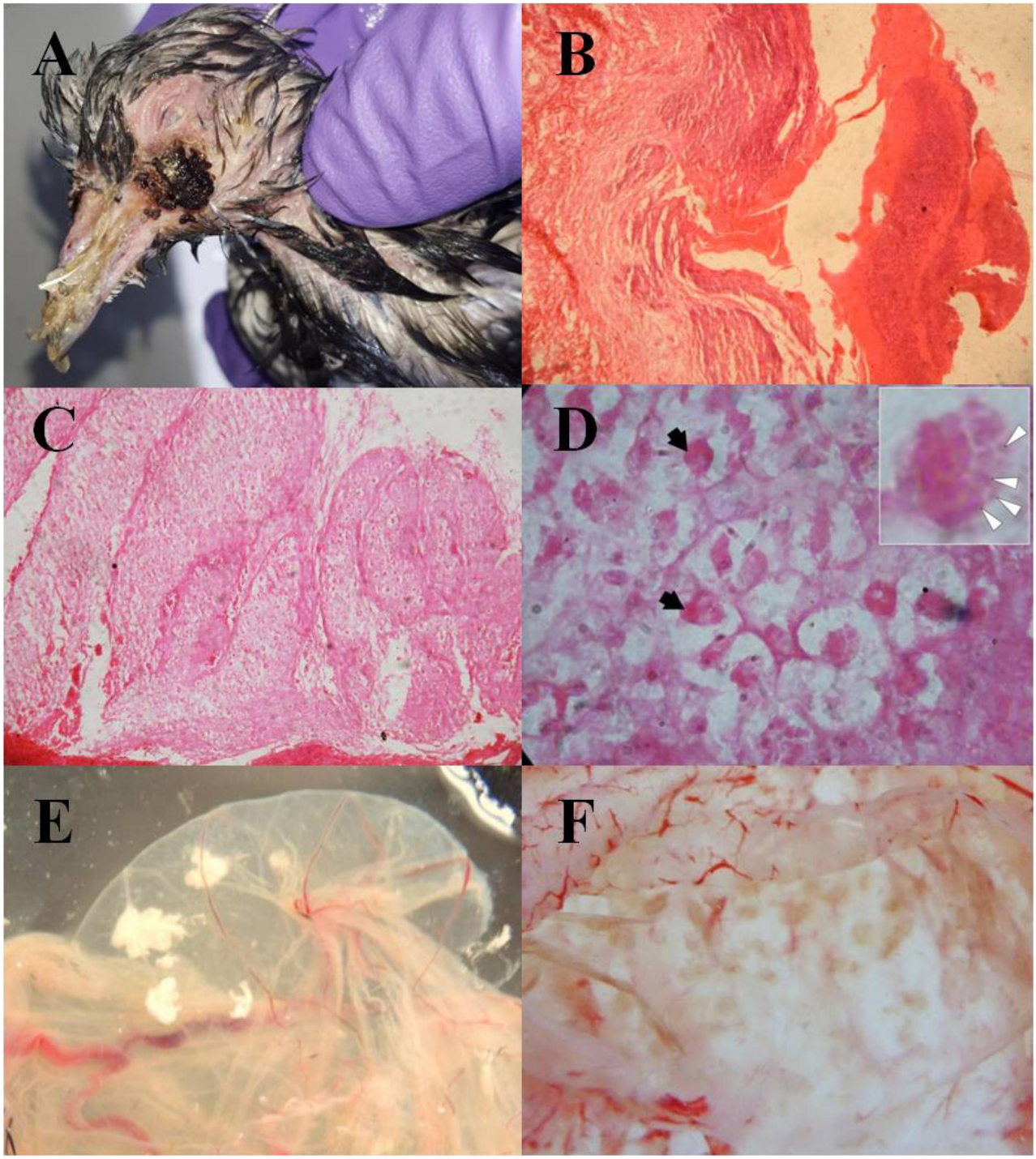

Figure 3. Pigeonpox lesions in affected pigeons (*Columba livia*). (A) Cutaneous and diphtheritic pigeon pox; (B) Cutaneous pock lesion of pigeon pox showing vacuolated prickle cells and shedding of necrotic scab containing dead, degenerated cells and often containing bacteria. H&E, 10×; (C) Cutaneous pock lesion of pigeon pox showing vacuolated prickle cells and intracytoplasmic eosinophilic inclusions. H&E, 100×; (D) Vacuolated prickle cells and intracytoplasmic eosinophilic inclusions or Bollinger bodies (black arrow) containing Borell bodies (inset- white arrowhead) of pigeon pox (H&E, 40×; inset 1000×); (E, F) Pock lesions on CAM of 19 day developing chicken embryo inoculated with pigeon pox tissue homogenate.

including petechiae on the pancreatic surface. Air sacs were thickened and cloudy, the lungs were severely congested, and fibrinous pericarditis was also seen. All visceral organs were congested. Liver was enlarged with mild fibrinous deposits on the surface.

Microscopically, liver showed degeneration with occasional dilatation of hepatic sinusoids. Fatty changes and cellular swelling with extensive engorgement and disintegration of sinusoids also observed. Focal heterophilic infiltration was seen in the liver parenchyma. Severe desquamation was also observed in the intestinal mucosa and congested submucosa. The capillaries in the lungs were congested and the air spaces were filled with fluid.

Liver impression smears and microbial culture from heart blood and liver revealed typical Gram negative bacilli. Based on culture, morphological characteristics and biochemical confirmation, the organismswere identified as *Escherichia coli*.

3.5. Salmonellosis (paratyphoid)

A pigeon was presented with severe diarrhoea, weight loss, anorexia and emaciation. The bird exhibited weakness, lameness and considerable dyspnoea. After sometime, the bird succumbed with terminal shivering and exudation of serous fluid from its mouth. Necropsy examination revealed pneumonia and severe congestion of lungs, catarrhal and haemorrhagic enteritis. The air sacs appeared cloudy and mild fibrinous pericarditis was apparent. Liver was congested with grey-white focal areas of degeneration. The ovaries appeared small, misshapen and haemorrhagic and associated with catarrhal salpingitis.

Microscopically, severe congestion and haemorrhages caused disruption of hepatic parenchyma and distortion of hepatocytes and sinusoids. Focal aggregation of mononuclear cells and varying degenerative changes of the hepatocytes were observed. Congestion and haemorrhages in the serosa of intestines with degeneration and desquamation of enterocytes were also marked. Leucocytic infiltration could be observed in the submucosa.

Liver impression smears and microbial isolation from the heart blood and liver showed Gram negative bacillus. Confirmation of the culture grown on brilliant green agar (BGA) media and xylose lysine deoxycholate (XLD) media with biochemical tests and morphological characteristics confirmed the isolates as *Salmonella* sp.

3.6. Aspergillosis

In an outbreak of aspergillosis affecting both young and old pigeons in one loft, clinical signs discernible were gasping and accelerated breathing, inappetance, emaciation, depression, rattling, dyskinesia and considerable mortality particularly in young birds.

Postmortem examination revealed that aspergillosis caused both pulmonary and systemic affections. In the uncomplicated pulmonary form lungs appeared congested and oedematous. In severe cases the lungs revealed a granulomatous pneumonia with white to yellowish opaque raised plaques. Air sacs were cloudy and thickened often showing plaque-like yellowish nodules. In a very severe form of pulmonary affection, sporulated forms of the fungal organism, appearing as a dark greenish-greyish growth with white tufts of mycelium were observed (Figure 4A). Considerable thickening of the membranes and adhesions to viscera and thoracic wall were also observed. Mucopurulent exudation was seen in the upper respiratory tract of one case. In the systemic form, yellowish-white nodules were seen attached to the viscera including, the ribcage, intestinal serosa and kidneys.

Microscopically, a lymphohistiocytic granulomatous pneumonia was observed. Granulomas had a necrotic centre containing inflammatory and cellular debris and surrounded by epithelioid macrophages and lymphocytes, and fibrous tissue proliferation at the periphery of the nodules (Figure 4B). Nonviable septate hyphae were observed in necrotic areas and in the vicinity of epithelioid cells. Sporulation was seen in the bronchial mucosa and lumen of the airways (Figures 4C and 4D). The granulomatous tissue expanded and compressed the adjacent alveoli. Air sacs also showed a granulomatous airsacculitis with massive thickening and infiltration with heterophils, macrophages and lymphocytes. Germinating conidia were also sometimes observed. These fungal bodies were found to invade through the parenchymatous tissue. In other organs, a similar granulomatous reaction was observed. Long branching septate fungal elements were readily discernible with Grocott's methenamine silver stain**,** and were found to be indistinguishable from *Aspergillus fumigatus*.

Impression smears from the mouldy growth stained with lactophenol cotton blue revealed characteristic conidial heads radiating in columns (Figure 4E).

Tracheal and lung swabs from affected birds collected and grown on potato dextrose agar (PDA) revealed fungal growth after 7 days of culture showing characteristics morphological features. Luxuriant growth was observed in PDA (Figure 4F). Culture on Aspergillus differential agar (ADA) showed absence of intense yellow orange colour at the base of the colonies thus differentiating it from *A. flavus*.

3.7. Mycotoxicosis

Two outbreaks of mycotoxicoses were suspected amongst pigeons, showing nonspecific clinical signs such as emaciation, anorexia and greenish diarrhoea. The birds appeared unthrifty with considerable mortality reported by the owners.

Necropsy examination of eight pigeons from these two lofts revealed consistent yellow-greenish discoloration

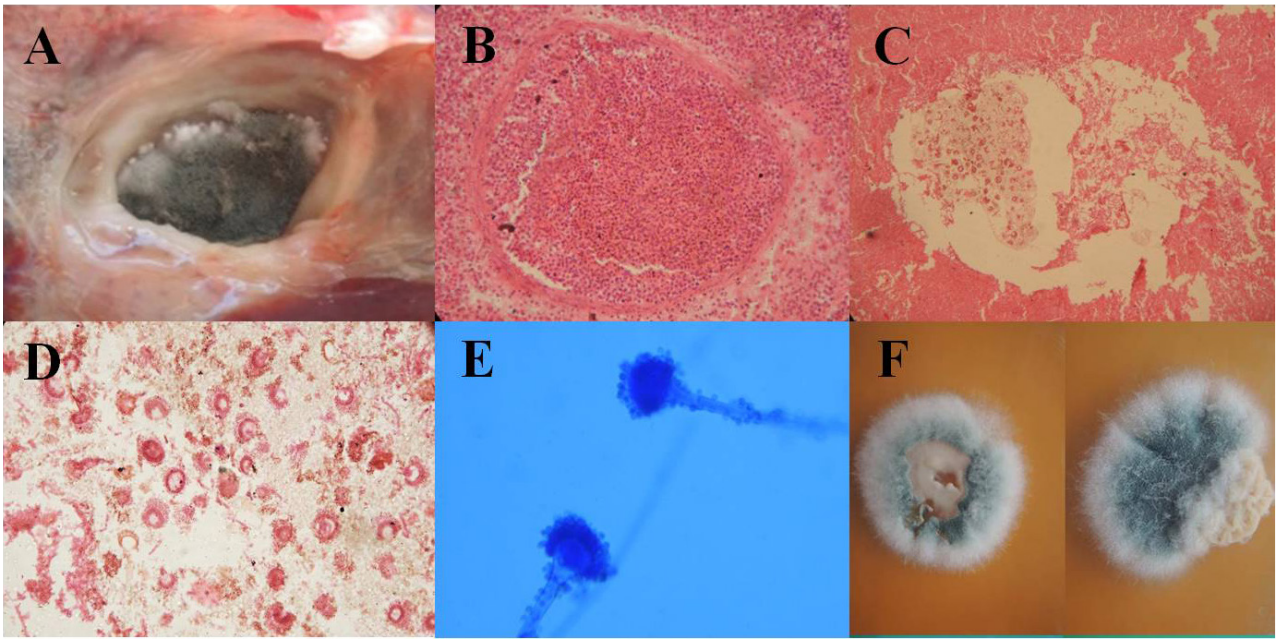

Figure 4. Lesions of aspergillosis caused by *Aspergillus fumigatus* in affected pigeons (*Columba livia*). (A) Fungal nodules in lungs with white tufts of mycelium; (B) Granulomas had a necrotic centre containing inflammatory and cellular debris and surrounded fibrous tissue proliferation at the periphery of the nodules. H&E, 100×. (C) Nongranuloma infiltrative lesions caused by invasive fungal elements in lungs tissue. H&E, 100×; (D) Fungal elements, consisting of germinating conidia in lumen of the parabronchi.H&E, 400×; (E) Typical conidial heads of *Aspergillus fumigatus* in fresh smear preparations. Lactophenol cotton blue, 400×; (F) Luxuriant growth of *Aspergillus fumigatus* in culture on potato dextrose agar (PDA).

of the liver with visible fatty change and multiple haemorrhages in the parenchyma. Concurrently, renal degeneration and haemorrhages were also sometimes observed. Affected kidneys were pale and enlarged and together with the ureters showed distinct urate deposits. Other concomitant lesions were mild air sacculitis, hydropericardium, massive haemorrhages in lungs with emphysematous patches and sloughing of gizzard lining.

Microscopic examination revealed cytoplasmic vacuolation of the hepatocytes indicating fatty change. In some instances there was proliferation of the bile ducts with mild infiltration by heterophils in the triad areas. In the kidneys, tubular epithelial degeneration and necrosis was observed. In the collecting tubules prominent casts were seen.

Feed and tissue samples send for mycotoxin detection and quantification revealed that all samples were positive, and aflatoxin B1 was detected in both feed (25 ppb) and lung/liver/kidney tissue samples (5 ppb), while as citrinin and ochratoxin was also found in feed samples (40 ppb and 60 ppb, respectively).

3.8. Traumatic injury

Amongst those necropsied, four birds were found to have died due to fatal traumatic and haemorrhagic injury caused probably by infighting.

4. Discussion

The present study to record the various pathological conditions affecting the domestic pigeon (*Columba livia*) in Jammu region is probably the first attempt in the region to realize such a survey. Although the exact population figures could not be ascertained, it was always a calculated guess of the owners when keeping a daily record of their flock size which is therefore always variable. This is due to the nature of the rearing of the pigeons, where the birds are let loose and lured back, and find their way to each other's lofts as part of the competitive sporting activity. The population figure therefore represents an average number of birds during the study period. Apart from these limitations, keeping track of affected birds also becomes difficult. Except for very sick and resident brooding birds, the flock is largely variable. Owner reported mortality figures were very arbitrary and vague and has not been represented. Mortality was accounted only for birds that were accessed for necropsy examination.

Evidently, protozoal pneumonia with hepatitis, Newcastle disease and mycotoxicoses are the three main etiological causes of mortality, accounting for 78.00% deaths amongst the birds.

Haemoproteus schizonts in the studied tissues were morphologically similar to descriptions in the literature

[7]. The principal histopathological changes were marked by vascular erosion and extravascular cellular reaction, much akin to observations of earlier workers [7, 8]. The intracorpuscular forms of the gametocytic stages of the parasite have been reported before [9].

Newcastle disease is a serious problem in pigeons caused by avian paramyxovirus-1 (APMV-1). Fairly high morbidity and mortality rate due to its infection in pigeons was observed within the lofts. The predominant indications of the disease were typically nervous manifestations as reported [10,11] attributable to degenerative and inflammatory lesions in the peripheral nerves and a nonpurulent meningoencephalitis. Lesions such as neuron degeneration, neuronophagia, gliosis, endothelial cell proliferation and severe degeneration of Purkinje cells [12] and malacic/demyelinating lesions [13] have also been recorded. Characterized by a high morbidity, it causes severe economic losses to the fanciers. Because the pigeon and chicken strains are related and similar, both belonging to APMV-1, cross-transmission between the species is a possibility. Pigeons as a reservoir of infection for ND viral transmission and dissemination are therefore a matter of serious concern.

Diagnosis of pigeon pox was fairly conclusive based on gross lesions, histopathological characteristics and inclusions. Pock lesions on chicken embryo CAM were similar to those described by Bwala et al. [14].

The gross and microscopic lesions observed were similar to those observed in colibacilloses [15, 16] and salmonelloses [15, 17] in pigeons. The zoonotic concern for *Salmonella* and *E. coli* isolates may be highlighted, while also disseminating the agents in the spread of colibacilloses and paratyphoid infection in domestic poultry. Because of their close proximity to human habitations, certain public health concerns are raised for being potential reservoirs of several pathogenic microorganisms including *Chlamydia* sp., *Salmonella* sp., *E. coli*, *Mycobacterium* sp. [18] and *Nocardia* sp. [19].

The signs and lesions of aspergillosis observed in the study were similar to reports of Tokarzewski et al. [20]. As observed in the present study, sporulation reportedly may occur in the lungs and air sacs [21] which may penetrate the air sacs, causing serositis and superficial necrosis in the adjacent organs [22]. These could be a basis disseminated mycosis and direct extension of the infection through the air sac wall.

The presence of molds on feed grains has been commonly observed. The fanciers feed the birds on poor quality and stale grains in the economical perspective, increasingly leading to the possibility of mycotoxicoses. Aflatoxins have been known to cause severe hepatic damage, including fatty degeneration, bile duct hyperplasia and massive necrosis [23], including tubular degenerative lesions in the kidneys [24], haemorrhages in lungs with emphysematous patches, air sacculitis and sloughing of gizzard lining [25, 26] all of which were amply demonstrated in the present study. The tissue concentration of mycotoxins in the present study were probably first reports in pigeons. Although, toxicological analyses of the feed and tissue samples have indicated the presence of toxins, their exact prevalence and exposure in the birds could vary because the samples were pooled. The presence of B1 in both feed and tissues is a concern and indicates that pigeons are susceptible.

The present study summarizes the leading mortality causes in pigeons and a glimpse of various diseases prevalent within the resident population. It may be emphasized that there could be many other affections, possibly life-threatening, amongst the domestic pigeon which however did not represent within the sample size accessed at necropsy. The objective of the study was to identify major aetiologies of mortality rather than a comprehensive and exhaustive survey. Even then, it is evident that pigeons are susceptible to various disease conditions and may harbour many infectious agents that can be disseminated in the environment and extend disease to susceptible hosts, spread zoonotic agents, including possible trans-boundary spread of pathogens. The vicinity of the international border for pathogen dissemination is a concern for adopting efficient health managerial strategies. More insights in the disease dynamics are therefore warranted.

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