PREVALENCE AND ETIOPATHOLOGY OF VARIOUS PATHOLOGICAL CONDITIONS AFFECTING THE JAPANESE QUAIL *(Coturnix coturnix japonica)* in Punjab

Thesis

Submitted to the Punjab Agricultural University
in partial fulfilment of the requirement
for the degree of

MASTER OF VETERINARY SCIENCE
in
VETERINARY PATHOLOGY
(Minor Subject : Veterinary Bacteriology and Virology)

by

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1997

DEDICATED TO

MY PARENTS -

THE PHARAOH OF MY LIFE

CERTIFICATE I

This is to certify that the thesis entitled, **"Prevalence and etiopathology of various pathological conditions affecting the Japanese quail (*Coturnix coturnix japonica*) in Punjab"**, submitted to the Punjab Agricultural University, Ludhiana in partial fulfilment of the requirements for the degree of **Master of Veterinary Science** in the subject of **Veterinary Pathology** (Minor subject: **Veterinary Bacteriology and Virology**), is a bonafide research work carried out by **Mr. Harjit Singh (L-94-V-249-M)** under my supervision and that no part of this thesis has been submitted for any other degree.

The assistance and help received during the course of investigations have been fully acknowledged.

B Singh 12/12/08

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CERTIFICATE II

This is to certify that the thesis entitled, **"Prevalence and etiopathology of various pathological conditions affecting the Japanese quail (*Coturnix coturnix japonica*) in Punjab"**, submitted to the Punjab Agricultural University, Ludhiana in partial fulfilment of the requirements for the degree of **Master of Veterinary Science** in the subject of **Veterinary Pathology** (Minor subject: **Veterinary Bacteriology and Virology**), is a bonafide research work carried out by **Mr. Harjit Singh (L-94-V-249-M)** has been approved by the Student's Advisory Committee after an oral examination on the same, in collaboration with an External Examiner.

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CONTENTS

Chapter I

INTRODUCTION

Domestication and hunting of birds as game or for food dates back to ancient days of human civilization. With advancement in knowledgement some of game birds such as guinea fowl, pigeons, pheasants, quails and pea fowls etc. are added to the long list of domestication particularly as specialities.

At the turn of the century the Egyptians were exporting migratory quails over 2 million each year. The peak in trade reached 3 million in 1920. But in 1930's big flocks were gone as migrations occurred. In Eastern Asia in early 1920's quails were killed and marketed in millions as they gathered near Port Arthur to cross Yellow Sea. Domestication of quail in Japan dates back to the 16th century and there is a myth for quail eggs as antidote for tuberculosis. Quail farming in China and Taiwan is practiced since centuries back. In India quail is well known as sport bird for fighting purposes and also as a favourite delicacy.

The best known breed of quail is Japanese quail (*Coturnix coturnix japonica*). It is a multipurpose animal and source of meat, egg, sport and experimental animal. Observations by Wilson *et al.*, (1961) on physiology, development and morphology of Japanese quail suggest its use as an experimental model in poultry research. The rate of embryonic development and growth of young quail is more rapid than that of the domestic fowl. In USA alone more than 100 laboratories use quail as experimental animal.

In 1973, Japanese quail was introduced in India for commercial production and since then quail farming is gaining popularity. This is due to factors like minimal capital outlay, convenience of management due to lesser space (5-6 birds / sq. feet), feed (20-25 g / bird / day), prolific laying capability and early returns than that of chicken farming. Laying starts at 5-6 week ages and peaks at 9-10 weeks and average weight of an egg is 10 grams and production is more than 250 eggs. Under good environmental conditions quail produces double the egg mass per unit of body weight as compared to good laying strains of domestic fowl (Wilson *et al.*, 1961). For meat purpose quails are ready at about 5 weeks of age and weighs about 150 g.

Quails are considered to be more resistant to many diseases affecting poultry. However, intensive system of rearing led to the occurrence of several diseases which leads to mortality and lowered productivity. Severe outbreaks of some diseases viz. Marek's Disease and Inclusion Body Hepatitis have been recorded in the department of Vety. Pathology, Punjab Agricultural University, Ludhiana. In order to plan a systematic disease control programme it is necessary to know prevalence and etiopathology of diseases in quails. Thus, the present study was undertaken with following objectives: -

1. To study prevalence of pathological conditions affecting Japanese quail.
2. To study detailed pathological alterations in various organs.
3. To establish etiopathological agents/factors of various pathological conditions wherever possible.

Chapter II

REVIEW OF LITERATURE

Lymphoproliferative Diseases

Lymphoid leucosis and Marek's disease constituting lymphoproliferative disease group are important diseases affecting poultry birds. Wight (1963) described gross and histological lesions of naturally occurring lymphoid leucosis and fowl paralysis (Marek's disease) in the Japanese quail in 5 cases. Grossly, lesions in all 5 cases were similar having enlarged livers with yellowish-white foci, thickened walls of small intestine either diffusely or in patches. The case of fowl paralysis showed greatly enlarged spleen and greyish-red tumourous ovary. All major peripheral nerves were normal.

Histologically, cases of lymhoid leucosis showed invading masses of lymphoblasts having large nuclei, rather prominent nucleoli and a relatively large amount of basophilic cytoplasm; numerous mitotic figures in liver, intestines, spleen and sometimes kidneys. In fowl paralysis cases, lesions were of granulomatous type consisting of mature lymphocytes and plasma cells and less mitotic figures. The tumourous ovary consisted of lymphoid mass of mature lymphocytes mainly with some more primitive lymphoid elements and plasma cells. One brachial plexus showed lymphocytes and plasma cells infilteration. During a 5 year period, lymphoid leucosis and fowl paralysis were recorded in 2.2% and 25.7% of the cases of total necropsies, respectively.

Dutton *et al.* (1973) exposed 80 families of Japanese quail to CR 64 strain of acute Marek's disease virus (MDV) by placing in contact with

MDV paralysed Gamefowl. Gross and microscopic lesions of MD appeared when quails were 75 days of age (68 days post exposure). Greatly enlarged livers with white nodules or diffuse enlargement with yellowish "nutmeg" pattern, highly enlarged spleens with neoplastic nodules and subcapsular haemorrhages; normal to enlarged and diffusely whitish kidney or nodular distribution of lymphoid aggregates; focal white areas or diffused enlargement of proventriculus were the lesions observed. The duodenum had localised to diffuse white thickenings with or without enlarged caecal tonsils. Rarely enlarged fleshy ovaries and raised white nodular lesions on heart were seen.. Microscopically lymphoid cells with occasional plasma cells and infrequently mitotic figures were present in various organs. Lymphoid cells, although quite variable in size, were characterised by rounded nucleus and moderate rim of basophilic cytoplasm. Neural lesions consisted of interneuritic and perivascular accumulations of mononuclear cells and schwann cell hyperplasia in some cases. The overall incidence in all quail was found to be 35.8.%.

Fujimoto *et al.* (1975) described the lesions of Marek's disease in Japanese quail infected by JM strain as slight enlargement of liver with scattered or diffuse foci on its surface extending into the parenchyma, spleen and enlarged gonads and duodenal wall thickening.. Histological lesions were characterised by lymphoreticular cells proliferation in liver, spleen, kidney, lung, intestine and gonad tissue. Immunofluorescent antigen was demonstrated in epidermis and superficial epithelial skin cells. Clinical signs comprised of depression and diarrhoea but no nervous signs were evident.

Mikami *et al.* (1975), while studying the serology and pathology of infections by JM strain of Marek's disease Herpes virus in Japanese quail confirmed that Japanese quail were susceptible to the JM strain, in contrast to

report by Calnek and Witter (1972) that newly hatched quails are refractory to inoculation with JM strain. Precipitating antigen in the feather follicles of MD exposed quail was demonstrated frequently in the early stage of infection but the antibody was discovered less often and formed in the quail only after a long incubation period. The antigen disappeared from the feather follicle epithelium and the antibody appeared in some quails at 360 days of age differing from observations on chickens, in which the antigen persisted in the tissue as long as they survived. Microscopic lesions were similar as observed by others but proliferation of lymphoid cells in the peripheral nerves was less frequent.

Khare *et al.* (1975) showed susceptibility of Japanese quails to JM strain of Marek's disease virus by inoculating with chicken blood infected with MDV The clinical signs of depression, anorexia, gasping, swelling of eyelids and blindness were noted. Prominent gross lesions consisted of bilateral focal and proliferative lung lesions along with enlarged spleens, kidneys, proventriculus, duodenum and testis. Peripheral nerves and ovaries were normal grossly. Histologically lymphoid cells of various sizes and a few plasma cells in visceral organs were seen.

Schat *et al.* (1976) reported outbreaks of naturally occurring lymphoproliferative disease in three flocks of Japanese quail. Sixteen per cent of birds revealed gross tumor like lesions in livers and enlarged spleens with white to yellowish-white discrete to confluent foci ; heart and duodenum in some cases were also involved. Histopathology showed lymphoproliferative infilteration of pleomorphic cells. Blood vessels lumen was also infilterated with abnormal cells. Neither Marek's disease virus nor antibodies to this virus were detected. However, antibodies to Avian leucosis virus subgroup A and reticuloendotheliosis were present in some cases.

Pradhan *et al.* (1985) studied natural cases of Marek's disease in Japanese quails. Gross lesions were confined to liver and spleen which showed enlargement and granular appearance. The proventriculus, duodenum and ovary in some cases had granular or tumourous growths. In these cases brachial plexus was slightly thickened. Microscopically pleomorphic lymphoid cells infilteration in various organs i.e., proventriculus, duodenum and feather follicles with mild to moderate infilteration in epidermis and dermis were seen. In brachial plexuses and sciatic nerves, infilterating cells were seen in interstitial tissue (perineurium) but not in nerve fibres. The morphological picture of tumor cells in quails was similar to that in chickens with MD, but the site of predilection of these lesions varied. In chickens, gonads, liver, heart and skin are commonly involved in acute form of MD and peripheral nerves are affected in classical form. In quails, however, all these organs except liver are less commonly affected. Spleens, proventriculus, liver and duodenum were found to be target organs. It was found that tumor cells infilterated the lumen of feather follicles as compared to the aggregation of lymphoid cells around the feather follicles in chicken. Serum antibodies against MD were detected with highest incidence at 15 weeks in 39 birds out of 139 examined.

Nair *et al.* (1986) reported Marek's disease in quails with liver and spleen persistently involved. The heart, jejunum and ileum were also affected in some cases but nerves and brain had no gross lesions. Histologically lesions were extensive infilteration of pleomorphic lymphoid cells in various organs and local sprinkling of lymphocytes in sciatic nerves.

Similarly, Kobayashi *et al.* (1987) observed pathological changes specific for MD in a non-vaccinated flock. MD antigen was found in feather tips of some birds. Lesions were of diffuse infilteration or

proliferation of reticulocytes and lymphoid cells in various organs, but not in nervous system.

Imai *et al.* (1990) isolated MD virus from Japanese quails with lymphoproliferative disease resembling MD and found it to be of serotype 1 by indirect immunoflourescence antibody test.

Adenovirus Infections

Quail bronchitis was first reported by Olson (1950) in bobwhite quail as highly fatal respiratory disease with symptoms ranging from slight rales to coughing, sneezing and rattled breathing and nervous symptoms in few birds. The course was 1 to 3 weeks. The isolated filterable agent was not killed by streptomycin. The disease was reproduced in quails from which virus was reisolated.

DuBose *et al.* (1958) isolated a virus from bobwhite quails with acute, contagious respiratory disease causing high mortality at 2 to 3 weeks of age. The virus caused dwarfing, thickening of amniotic sac, and death when inoculated into allantoic cavity of chicken embryos. The virus was not neutralised by infectious bronchitis antiserum but neutralised by serum from quail that survived the disease.

Chew - Lim (1980) confirmed quail bronchitis in two flocks of adult Japanese quails with history of respiratory signs and 10-15% drop in egg production, with appearance of soft-shelled eggs and white eggs with no pigments on shells.. Lesions consisted of congested lungs and trachea, egg peritonitis , bile stasis in liver and enlarged spleen. Purulent sinusitis, tracheitis and necrotic foci on liver were also seen. Fowl adenovirus serotype 1 was recovered from soft-shelled and white eggs, brains, trachea lung suspensions.

Pradhan *et al.* (1980) reported isolation of quail bronchitis virus and pathological changes in Japanese quail showing respiratory distress having tracheal rales, sneezing and coughing along with nervous symptoms with twisting of neck in 6 weeks old quails. Grossly, mucus exudate in trachea and bronchi was present. Histopathologically marked hyperplasia of the epithelium and mucus secreting glands with infilteration of lymphocytes in trachea , mild to moderate hyperplasia of bronchial epithelium and peribronchial accumulation of lymphocytes were observed. No microscopic changes were observed in brain , gizzard, pancreas, heart and proventriculus.

Jack and Reed (1989, 1990) produced quail bronchitis in bobwhite quails experimentally with type I adenovirus. Clinical signs produced were minimal, but occasionally birds were ruffled, exhibited open mouth-breathing. Gross lesion comprised of necrotising tracheitis, proliferative and necrotising bronchitis and pneumonia, multifocal necrotising hepatitis, necrotising splenitis. Histopathologically hyperplasia of splenic mononuclear phagocytes, bursal lymphoid necrosis and bursal atrophy were usually seen. Large intranuclear inclusions, characteristic of adenovirus infection, were identified in trachea, lungs, liver and bursa.

Jack *et al.* (1994) studied pathogenesis of quail bronchitis and found viral inclusions in tracheal mucosa by day 2 PI by intratracheal route. Deciliations of tracheal epithelium formed irregular luminal border on day 3 PI. On days 4 and 5 PI tracheal epithelium was partially desquamated with minimal leukocytic infilteration. Bronchial epithelium had similar changes but with more intense leukocytic infilteration. Hyperplasia of splenic macrophages was found on day 2 PI and peaked by day 5 PI.

Two outbreaks of egg drop syndrome in quails maintained in association with chicken were detected resulting in egg production drop upto

50%. by Das and Paradhan (1992). Haemagglutination inhibition antibodies to EDS-76 were detected and virus was also detected in lining epithelium of glandular cells of uterus by FAT.

Mohapatra *et al.* (1994) isolated haemaggulutinating adenovirus similar to chicken EDS-76 virus causing sudden drop in egg production in quails. Experimental infection with isolate resulted in severe drop in egg production (27 to 30%) along with aberrant eggs. Grossly, no change in any organ was present, but microscopically hyperplasia of epithelium, mononuclear cell infilteration, atrophy of tubular glands and formation of lymphoid follicles was observed in uterus.Viral antigen was detected in blood leukocytes, uterus, spleen, vagina and nasopharynx. The highest HI antibody titre and ELISA titre were detected at 3 weeks PI.

Grewal *et al.* (1994) reported naturally occurring outbreak of Inclusion Body Hepatitis from which avian adenovirus was isolated. Multiple pale foci were seen throughout the liver with enlarged and mottled spleen. Histologically hepatic necrotic foci with infilteration of mononuclear cells and a few heterophils along with basophilic intranuclear inclusion bodies in most hepatic cells were evidenced.

Ranikhet Disease (RD)

Although Japanese quails are considered to be resistant to RD, however, outbreaks of RD have been reported. Higgins and Wong (1968) gave first report of RD outbreak with 40% mortality. The post-mortem findings like wasting of breast muscles, congestion of intestines, lungs and meninges were reported.

Lu *et al.* (1987) reported 369 outbreaks in Taiwan from 1970 to 1985 of which 344 (93%) were in chicken. Other species affected were quail, pigeon, pheasant, geese and turkeys.

In India disease was first reported by Kumanan *et al.* (1990) in organised quail farm (4 weeks old birds) with 10% mortality. Symptoms included anorexia, diarrhoea and nervous signs like drooping of wings torticollis, extended legs, paralysis and lateral recumbency. Post-mortem findings were wasting of breast muscles, congestion of intestines, lungs and meninges. Disease was confirmed by inhibition of haemagglutinating activity by known antiserum.

Mycotic Infections

Fungal infections of the lungs and air sacs are common in poultry. Olson (1969) reported epizootics of aspergillosis in Japanese quail with clinical signs of inappetence, depression, accelerated breathing and ataxia. Grossly lesions of yellow caseous nodules in lungs and air sacs and focal areas of malacia in infected brains were found. Microscopically, nodules consisted of central eosinophilic mass of dead heterophils surrounded by epithelioid cells. Radiating turf of fungal hyphae was found in centre of nodule with PAS stain.

Reece *et al.* (1986) reported cases of mycosis of lungs and air sacs by *Aspergillus fumigatus*, *Penicillium* sp., *Mucor* sp., *Puttularia* sp. and Zygomycetes in poorly ventilated and dust laden environment at quail farms. Along with infection of lungs and air sacs, most birds also showed kyphosis and paresis associated with mycotic spondylitis of the last cervical or first thoracic vertebrae. Birds with nervous signs had small abscesses in brain. Histological lesions were same as mentioned earlier.

Incidence of mycosis of other organs is also reported. Mycotic salpingitis was reported in a case from a flock with Marek's disease by Singh *et al.* (1994).

Chaudhary *et al.* (1988) inoculated *Aspergillus fumigatus* spores intratrachealy into 100 , day old quails and found lesions restricted to respiratory tract. Lesion in general included congestion and focal haemorrhages in first two days followed by development of greyish white nodules in lungs, air sacs and trachea. Microscopic changes comprised of congestion, haemorrhages and diffuse cellular infiltration in first two days followed by granulomatous reaction with well developed granulomas in lung, air sac and trachea.

Pandita *et al.* (1995) studied experimental infection using *A. flavus* and found less involvement of lung tissue and no involvement of trachea and air sacs. Grossly, only greyish white consolidations were present without discrete nodules.

Salmonellosis

Many species of Salmonella have been associated with disease conditions in quail. Cunningham (1943) reported first *Salmonella bredeney* infection in quail with highest mortality in 3 to 9 days old with enteritis, yellow friable liver, unabsorbed yolk sac lesions and showing symptoms of general depression, inappetence, diarrhoea and ruffled feathers. He was able to experimentally reproduce the disease with isolate.

Edgar *et al.* (1964) studied the susceptibility of coturnix quails to various poultry pathogens and found them to be susceptible also to *Salmonella pullorum*, *S. gallinarum* and *S. typhimurium* infection.

Bigland *et al.* (1965) isolated 5 species of Salmonella from Japanese quail as *S. anatum, S. give, S. infantis, S. london, S. kentucky* from tissues and contents of caeca and intestine.

Tanaami *et al.* (1977) reported a mixed infection of *Salmonella typhimurium, S. thompson* and Aspergillus fungus in quails and ducks, showing respiratory signs, diarrhoea and lowered hatchability. Salmonella organisms were isolated from dead in shell embryos, lungs, liver, yolk sacs of chicken as well as incubators. Aspergillus was isolated from lungs and liver having yellowish white nodules with greyish white spores in lungs and liver of infected ducks along with pericarditis and serositis.

Kapoor *et al.* (1980) reported the first outbreak of *Salmonella bareilly* infection in quails with chicks hatched in same incubator. Lesions consisted of necrotic foci on liver, distended gall bladder, enlarged and congested spleen and pericarditis. Congestion, haemorrhages, along with infiltration of heterophils, lymphocytes, a few plasma cells in mucosa and hyperplasia of reticuloendothelial cells in serosa of intestine were noticed. Focal hepatic necrosis, microgranulomae and subcapsular infarcts in liver due to thrombophlebitis and giant cells laden with organisms in pericarditis were seen.

Awaad *et al.* (1981) experimentally infected Japanese quails with *Salmonella gallinarum* (streptomycin-resistant) either orally or through egg dipping and observed depression, drooping of wings, huddling together, transient diarrhoea, lateral deviation of head and neck, hyperexcitability, paresis and backward movements as clinical signs. Lesions consisted of subcutaneous haemorrhages, congestion and haemorrhages in parenchymatous organs, misshapen, pedunculated, discoloured cystic egg follicles in those which survived infection. In oral infection, organism was shed in faeces from 80% and 40% quail chicks after 2 and 4 hours respectively, and no

faecal swab was positive later on. Eggs from orally infected adult birds yielded organisms in 2.3%, showing vertical transmission. Serodiagnosis using stained antigen, rapid whole blood agglutination and microagglutination test were unable to detect *S. gallinarum* carriers in quails, showing their test to be insufficient proof of flock being free of organism.

Sarma *et al.* (1988) reported natural outbreak of *S. gallinarum* (9, 12 : - : - serotype) in Japanese quails and its subsequent spread to chicken, causing heavy mortality (71%) in 1-3 day old quails with no typical symptoms. Lesions consisted of perihepatitis and necrotic foci on liver, congestion of lungs, slight enlargement of spleen, hydropericardium and acute haemorrhagic enteritis.

Mohapatra (1992), reviewed the Salmonella infections in quail and found that different Salmonella species viz. *S. anatum, S. give, S. london, S. infantis, S. kentucky, S. bareilly, S. gallinarum* and *S. stanley* have been isolated and disease process studied, but no report regarding natural outbreak of *S. pullorum* infection in quails is there. *Salmonella gallinarum* mortality might reach 70-90% and lesions were same as described by Sarma *et al.* (1988).

Fowl cholera

Fowl cholera, caused by *Pasteurella multocida* is an acute or chronic disease known to affect almost all species of fowl. Hinshaw and Emlen (1943) cited by Panigraphy and Glass (1982) reported one case of fowl cholera in California valley quail (*Lophortryx. californicus*).

Panigraphy and Glass (1982) reported three outbreaks of acute fowl cholera in quails in Texas causing high mortality, one in pharaoh Japanese quail (*Coturnix coturnix*) and two in bobwhite quail (*Colinus virginianus*).

Lesions in pharaoh quail comprised of enlarged and friable livers with focal areas of necrosis, mottled and hemorrhagic spleens, petechial haemorrhages on coronary fat and hemorrhagic intestinal mucosa. *Pasteurella multocida* was isolated from heart blood and livers.

Polero *et al.* (1988) encountered chronic endemic fowl cholera in breeding and egg producing flock of 5000 Japanese quail after introduction of new birds from another flock. About 15 birds died daily and no drug treatment was effective. Control was achieved with an oil adjuvant vaccine prepared with isolate from flock and with hygenic measures.

Myint and Carter (1988) described incidence of outbreaks of highly acute fowl cholera predominantly affecting birds of four weeks to four months causing high mortality characterised by generalised vascular congestion with petechial haemorrhages on serous surfaces of internal organs and focal necrotic hepatitis. Hemorrhagic enteritis was frequent with bloody accumulate in anterior portion of small intestine.

Glisson *et al.* (1989) reported severe clinical fowl cholera in Japanese quails causing acute high mortality in these flocks, beginning at 24 to 28 days of age with signs of severe depression, inappetance, ruffled feathers and prostration and death within a few hours of obvious clinical signs. Grossly, lesions were absent or were composed of either multifocal small pale areas on liver and spleen. The lungs were slightly darker in colour than normal. Histopathological lesions were septicaemic in nature i.e. multifocal areas of fibrinoid splenic necrosis with infilteration of heterophils in areas of necrosis and areas of reticuloendothelial cell hyperplasia involving cells of periarterial sheath. Liver showed similar lesions, lungs had multifocal interstitial pneumonia.

E. coli **infections**

Silva *et al.* (1989) reported occurrence of coligranulomatosis in quail flock in Brazil causing 85% drop in egg production and 15% mortality. Gross and microscopic lesions found were similar to those described in chicken and turkeys. Whitish grey nodules of 3 to 15 mm diameters, with smooth surface and firm consisterncy were present on mesentry, intestines, gizzard, heart, uterus, ovaries and liver. Microscopically nodules were found adhering to serosa of organs and sometimes penetrating the parenchyma. Cells involved were macrophages, epitheloid cells, mononuclear cells, plasma cells and scarce heterophils. Small areas of caseous necrosis, haemorrhage and proliferation of fibrinous connective tissue characterised the granuloma. Bacterologically pathogenic *E. coli* was isolated.

Colibacillosis occurring in quails ranging from 21 to 330 days and characterised by hepatomegaly, thickening of liver capsule and pericardium, necrosis of hepatocytes and fibrinous pericarditis was mentioned by Ito *et al.* (1990).

Reddy and Koteeswaran (1994) studied relative susceptibility of Japanese quail to serotypes (019, 060, 068, 0101, 0109) of pathogenic *E. coli* isolated from chicken. Both 7 day old and 4 week old quail chicks were susceptible. Infection was found to be more acute in nature in 7 day old chicks with rapid death. Four week old quail chicks, showed lesions typical of avian colibacillosis showing airsacculitis, fibrinous pericarditis and perihepatitis.

Proteus Infections

Association of *Proteus mirabilis* with septicemic disease in Japanese quail was reported by Sah *et al.* (1983) causing 70-90% mortality in

successive breeds of 3 to 7 day old quail chicks showing tremors, progressive weakness, prostration, coma, followed by death within 24 hours of appearance of signs. Lungs, liver, spleen, heart and kidneys were congested. The yolk sac remained usually unabsorbed. Histopathologically, lesions comprised of congestion, haemorrhage, serofibrinous exudation into peribronchiolar spaces, hypertrophy of bronchiolar and tracheal mucous glands and mild hyperplasia. Lungs and liver sections stained with MacCallum-Goodpastures stain revealed slender-gram negative rods and Proteus was isolated from heart blood and lungs.

Myint (1987) reported septicemic Proteus infection in blood of Japanese quail chicks. Disease was characterised by high mortality rate in quail chicks under 1 week of age with anorexia, frothy diarrhoea, dark enlarged spleen, frothy intestinal contents, unabsorbed yolk sac along with congested heart, liver, lung and other organs. Among breeders mortality was 5-8 birds per day with enlarged and black spleen.

Ulcerative Enteritis (Quails disease)

Bass (1939) studied the ulcerative enteritis condition in quails and found lentil shaped ulcers in lower third of ileum and caecum. Gram negative anaerobic bacillus was isolated.

Durant and Doll (1941) reviewed ulcerative enteritis as infectious and contagious disease of game birds, chiefly quail and birds upto 2 weeks of age. When artificially reared, the severe infection takes place.

Bendela (1976) experimentally found *Clostridium colinum* causing denuding of brush border of intestinal epithelial cells. Well developed ulcers were produced as bacteria penetrated deep into mucosa. Focal coagulative necrosis of hepatocytes occurred in chronic cases. Abundant bacteria were

seen by indirect immunoflourescent technique in intestinal lumen necrotic villi of infected quail intestines.

Patro et al (1992) recorded necrotic foci on liver, splenomegaly , haemorrhagic and necrotic lesion throught the intestines with isolation of Clostridial organisms from intestinal contents and demonstration of organisms from necrotic livers and intestines .

Naveen and Arun (1992) reviewed ulcerative enteritis as highly infectious and fast spreading disease affecting young quails. Acute form is characterised by sudden and high mortality especially of heavily muscled quail and lesions were haemorrhagic enteritis in upper part of intestine. Birds surviving longer might show ulceration in intestine, congested, enlarged and haemorrhagic spleen, liver may show light yellow mottling to large irregular areas of necrosis along the edges.

Infectious Coryza and Sinusitis

Reece *et al.* (1981) reported natural infection in flock of Japanese quails with *Haemophilus paragallinarum* with signs of mucoid sinusitis, nasal exudation and conjunctivitis, opacity or ulceration of cornea. In chronic cases abscesses in infraorbital sinuses were found.

Tiong (1978) isolated *Mycoplasma gallisepticum* from a flock of 20,000 birds with symptoms of contagious sinusitis. The clinical signs consisted of respiratory distress, paralysis of limbs and soft shelled eggs. Necropsy lesions showed swelling of infraorbital sinuses with caseous or mucoid gelatinous exudate.

Nascimento and Nascimento (1986) isolated *Mycoplasma gallisepticum* from sinusitis of Japanese quails showing swollen infraorbital sinuses. Grossly, gizzard had mild hemorrhagic changes; lungs and brain had diffuse

haemorrhagic lesions and masses of caseous exudate in sinuses. Cloudy to caseous air sacculitis was also observed.

Miscellaneous conditions

Other miscellaneous diseases of quails reported are visceral gout by Das *et al.* (1992a) characterised by abundance of urate crystals. Das *et al.* (1992b) also reported a case of bumble foot characterised by typical clinical signs of disease in footpad and thickening of synovial membrane of tarsal joint. Hyperplastic changes were observed in histological preparations and *E. coli* was isolated from lesions.

Mortality pattern and conditions

Srinivasan *et al.* (1980) studied the mortality pattern during the years 1976 to 1977 at HAU farm and attributed mortality rate in 0-6 week age group to pneumonia and omphalitis mainly. In female adult the major cause of mortality was egg bound/peritonitis where as in males the mortality was caused by different conditions such as hepatitis, enteritis, pneumonia, internal haemorrhage, colisepticemia and aspergillosis.

Suneja *et al.* (1983) studied the mortality pattern in quail and found that maximum deaths occured during first week of life, followed by those in 1-6 weeks. In birds between 1-6 weeks of age, the highest mortality (30.65%) was observed in July, followed by October, February, June and May. After 6 weeks, mortality was found to be comparatively higher during the period from June to October. No significant differences were observed in the pattern of mortality in two sexes. Pneumonia was found to be the most prevalent cause of mortality in quails between 1-6 weeks of age. Incidence was higher during November and February. Omphalitis/unabsorbed yolk was

the principal lesion in birds of age 0-1 week age and it was most frequently encountered in January. Enteritis, egg bound condition, drowning death in chicks and internal haemorrhage were other conditions noticed in quails. Gross and histopathological changes of lymphoid leukosis were observed in few quails.

Sharma and Kaushik (1986) carried out surveillance of disease in Japanese quail based on post-mortem examination and recorded following disease conditions-Pneumonia (40.4%), ulcerative enteritis (19.7%), yolk retention (16.0%), colibacillosis (10.6%), egg peritonitis (6.9%), hepatitis (4.9%), avian leucosis complex (1.4%), aspergillosis (0.2%). Cases were of birds aged 0-5 weeks or adult birds. Maximum mortality was observed during winter and lowest during rainy season.

Gangadharan *et al.* (1989) analysed mortality in quails at Kerala Agricultural University Poultry Farm. Mortality was severe in age group of 0-3 weeks. Mortality was lowest in rainy season while it was more in dry season of the year. Omphalitis was most important cause of death in young stock (41.08%) followed by hepatitis (21.99%) and then pulmonary congestion and oedema (18.37%). In adult quails, hepatitis (46.38%), enteritis (19.97%), Marek's disease (4.35%), coccidiosis (0.92%) and Ranikhet disease (0.24%) were the common diseases.

Patro *et al.* (1992) studied aetiopathology of quail disease in Orissa. They attributed causes of mortality in descending order to - lymphoid leucosis, enteritis, cannibalism, egg bound, egg peritonitis, necrotic hepatitis, fatty degeneration of liver, pneumonia, visceral gout, salmonellosis, staphylococcal infection, hemangioendothelioma, leiomyoma with dermoid cysts, pulmonary aspergillosis and ascariasis.

Ravindran *et al.* (1994) made investigations of 5 years mortality record of quails at Poultry Research Station, Madras. Mortality was higher among age groups of 0-1 and 2-6 weeks. The higher mortality was recorded during winter and monsoon seasons upto 6 weeks of age. The mortality trend had significant influence on high brooding losses. Conditions commonly observed were drowning, paralysis, colibacillosis, egg bound and egg peritonitis etc.

Chapter III

MATERIALS AND METHODS

Prevalence

In the present study, dead and sick Japanese quails brought to Poultry Diagnostic Lab., Deptt. of Vety. Pathology from University Poultry farm and various Govt. poultry farms in the state of Punjab and Chandigarh were necropsied. Visits were also made to these farms to collect the dead quails. Detailed gross lesions, age, sex etc. were recorded. Post-mortem records of last six years starting from January, 1991 to December, 1996 were screened for studying the prevalance of different conditions affecting the Japanese quail.

Collection of samples

The postmortem examination of 2000 cases was conducted from August, 1995 to May, 1997. The detailed gross lesions were recorded. The tissues from 166 cases comprising of pieces of liver, spleen, kidneys, heart, brain, ovary, intestine, oviduct were preserved in neutral buffered formalin for histopathological studies. Cases of gout were taken in absolute alcohol. Tissues were processed by routine paraffin embedding technique and 5μ thick sections were cut and stained with haematoxylin and eosin. Wherever necessary special stains were used. For microbial isolation suspected organs were sent to department of Vety. Bacteriology and Virology in sterilized petriplates for bacterial or mycological isolations.

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Serotyping of S. gallinarum isolated from quails

The isolates of *S. gallinarum* were got serotyped from National Salmonella and Escherichia Centre, CRI, Kasauli, India and a strain from these isolates was further used for experimental infection of quails.

Passaging

Single passaging of isolated strain was done in one-day old Japanese quail to test its pathogenicity. Japanese quails were procured from Punjab Agricultural University, Ludhiana, hatchery and they were tested by rectal swabs culture for Salmonella gallinarum free status.

Procurement and maintenance of experimental birds

One hundred and eighty, day-old quail chicks were obtained from Central Poultry Breeding Farm, Chandigarh in two batches for experimental studies. They were kept in hygienic conditions with proper breeding temperature and lighting. They were fed on quail feed free from antibiotics. All birds were subjected to rectal swab cultures to check their Salmonella gallinarium free status.

Computation of LD_{50}

A pilot experiment was conducted to find out LD_{50} of *S. gallinarum*. Ten fold serial dilutions from 10^{-2} upto 10^{-16} of 18 hours broth culture of *S. gallinarum* were made in sterile normal saline solution and groups of 4 quail chicks (7 day old) each were inoculated intraperitoneally with 0.1ml of different dilutions. Deaths and survivals among infected quail chicks were noted for a period of 10 days for calculating LD_{50} as per Reed and

Muench (1938). The heart blood, liver, spleen from inoculated birds were cultured for re-isolation.

Experimental design

Twenty-five birds (7 days old) were inoculated intraperitonealy with 0.1 ml of *S. gallinarium* containing 6.75 x 10^8 organism per ml - the dose calculated as the LD_{50}. Twenty-five control birds were inoculated intraperitonealy with normal saline solution. Natural mortality was recorded daily upto 10 days post inoculum (DPI), thereafter three birds were sacrificed daily upto 15 DPI. Three control birds were also sacrificed daily. Liver and spleen from dead and sacrificed birds were taken in sterilized petri plates for isolation.

Clinical signs and gross lesions were noted in detail. Various tissues viz. liver, spleen, lungs, kidney, bursa, brain, trachea, intestines were collected in neutral buffered formalin. Tissues were processed by paraffin embedding techniques. Sections of 5μ thickness were cut and stained with haematoxylin and eosin.

Chapter IV

RESULTS

To study the mortality pattern in Japanese quail, the data pertaining to population, mortality, age and months for the period of 1991 to 1996 was obtained from the Punjab Agricultural University Poultry Farm. To assess the causes of mortality, data of postmortem records of Department of Veterinary Pathology was used. The disease encountered were classified and agewise, seasonwise and diseasewise distribution were analysed.

The data showing number of dead birds, total number of birds and calculated percent mortality arranged agewise during different months of the period 1991 to 1996 have been set out in Tables 1 & 2.

Yearwise and Agewise Mortality Rate

The mortality rate worked out for different years and age groups are presented in Table 3 (Figure 1). Analysis of variance carried out with arcsine $\sqrt{P}$ transformation suggested that mortality rate between different years and between different age groups differed non-significantly.

Month/Seasonal Incidence

The mortality rate and monthly seasonal variation indices for different age groups have been shown in Table 4 & 5. The highest incidence of mortality in general was observed in July (6.00%) followed by May (5.05%), June (4.58%) and January (4.45%). In 0-1 week age group, months in descending order of mortality rate were July (12.33%), January (10.94%),

Table 1: Monthwise and Yearwise & Agewise mortality pattern of Quails (1991-1996)

Year	Age Group	Jan.	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
	0 - 1 Wk	-	-	-	-	15 (400)	-	182 (2053)	101 (1186)	0 (330)	-	-	256 (2378)
1991	1 - 6 Wk	17 (1068)	-	-	-	4 (385)	8 (381)	118 (1384)	22 (1436)	23 (1230)	-	-	109 (1011)
	> 6 Wk	12 (1050)	18 (1037)	18 (1017)	20 (999)	74 (979)	22 (1278)	43 (1256)	43 (2387)	233 (2675)	103 (2055)	107 (1728)	73 (1608)
	0 - 1 Wk	276 (1783)	31 (804)	-	-	300 (4084)	255 (5653)	255 (1490)	-	-	-	44 (3565)	60 (2600)
1992	1 - 6 Wk	267 (2384)	230 (1312)	53 (379)	-	252 (1655)	467 (5175)	418 (1376)	36 (1232)	0 (1196)	-	57 (4890)	21 (2537)
	> 6 Wk	209 (2021)	26 (2899)	41 (2815)	30 (2408)	42 (1726)	16 (2360)	119 (6522)	156 (1902)	141 (4862)	75 (4047)	65 (2537)	82 (5338)
	0 - 1 Wk	11 (840)	-	-	14 (2484)	45 (2704)	35 (607)	-	-	-	240 (3976)	89 (1852)	0 (373)
1993	1 - 6 Wk	8 (829)	3 (366)	0 (363)	0 (1334)	63 (3790)	7 (1224)	4 (262)	-	-	82 (3736)	173 (3084)	36 (1699)
	> 6 Wk	47 (4527)	106 (4573)	207 (4115)	217 (3852)	168 (1712)	109 (3694)	98 (3909)	107 (3851)	86 (3427)	69 (3484)	63 (4007)	85 (4210)
	0 - 1 Wk	-	-	-	121 (3951)	7 (1478)	-	-	4 (555)	-	54 (3307)	-	-
1994	1 - 6 Wk	0 (337)	-	-	83 (2352)	130 (1522)	0 (1391)	-	-	22 (551)	162 (3782)	103 (1342)	-
	> 6 Wk	94 (5685)	151 (3383)	91 (3149)	119 (2667)	52 (3970)	115 (3999)	41 (3539)	74 (1916)	54 (1418)	49 (1363)	19 (1439)	44 (2038)
	0 - 1 Wk	-	-	34 (1800)	34 (3551)	12 (1260)	-	-	-	122 (1950)	50 (1486)	33 (890)	-
1995	1 - 6 Wk	-	-	-	92 (3517)	38 (1238)	0 (1196)	-	-	27 (1828)	110 (1991)	56 (412)	0 (398)
	> 6 Wk	49 (1648)	76 (1493)	70 (1190)	77 (759)	95 (2382)	244 (1604)	40 (594)	38 (499)	59 (341)	135 (960)	146 (1492)	148 (1181)
	0 - 1 Wk	-	13 (1458)	21 (2622)	-	161 (1913)	-	-	-	-	-	-	-
1996	1 - 6 Wk	-	54 (1445)	83 (3992)	13 (2524)	140 (1752)	25 (700)	-	-	-	-	-	-
	> 6 Wk	52 (1169)	58 (1047)	78 (1731)	163 (3081)	133 (1292)	86 (1051)	109 (1360)	50 (814)	25 (694)	9 (592)	24 (498)	21 (454)

Figures out of parenthesis indicate mortality

Figures in parenthesis indicate total number of quails during a particular period and of specified age group

- Indicate that quails were not kept during that period

Table 2: Agewise occurrence of mortality (in percent) in quails during different months of the years (1991-1996)

Year	Age Group	Jan.	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
	0 - 1 Wk	-	-	-	-	3.75	-	8.86	8.52	0.00	-	-	10.76
1991	1 - 6 Wk	1.59	-	-	-	1.04	2.10	8.53	1.53	1.87	-	-	10.78
	> 6 Wk	1.14	1.74	1.77	2.00	7.55	1.72	3.42	1.80	8.71	5.01	6.19	4.53
	0 - 1 Wk	15.48	3.86	-	-	7.34	4.51	17.11	-	-	-	1.23	2.30
1992	1 - 6 Wk	11.20	17.53	13.98	-	15.23	9.02	30.28	2.92	0.00	-	1.13	0.82
	> 6 Wk	10.34	0.89	1.46	1.24	2.43	0.68	1.82	8.20	2.90	1.85	2.56	1.54
	0 - 1 Wk	1.30	-	-	0.56	1.66	5.76	-	-	-	6.03	4.80	0.00
1993	1 - 6 Wk	0.96	0.82	0.00	0.00	1.66	0.57	1.53	-	-	2.19	5.60	2.19
	> 6 Wk	1.03	2.31	5.03	5.63	9.81	2.95	2.50	2.77	2.50	1.98	1.57	2.02
	0 - 1 Wk	-	-	-	3.06	0.47	-	-	0.72	-	1.63	-	-
1994	1 - 6 Wk	0.00	-	-	3.52	8.54	0.00	-	-	3.99	4.28	7.67	-
	> 6 Wk	1.65	4.46	2.89	4.46	1.30	2.88	1.51	3.86	3.80	3.59	1.32	2.16
	0 - 1 Wk	-	-	1.88	0.95	0.95	-	-	-	6.25	3.36	3.70	-
1995	1 - 6 Wk	-	-	-	2.61	3.06	0.00	-	-	1.47	5.52	13.59	0.00
	> 6 Wk	2.97	5.09	5.88	10.14	3.98	15.21	6.73	7.61	17.30	14.06	9.78	12.53
	0 - 1 Wk	-	0.89	0.80	-	-	-	-	-	-	-	-	-
1996	1 - 6 Wk	-	3.74	2.07	0.51	7.99	3.57	-	-	-	-	-	-
	> 6 Wk	4.46	5.54	4.50	5.29	10.29	8.18	8.01	6.14	3.60	1.52	4.82	4.62

- Indicate that quails were not kept during that period

Table 3: Agewise mortality rate in quails

Year	Age Group			Overall
	0 - 1 Week	1 - 6 Weeks	Above 6 Weeks	
1991	8.72 (554/6347)	4.49 (301/6895)	4.23 (766/18069)	5.17 (1621/31311)
1992	6.11 (1221/19979)	8.13 (1801/22136)	2.54 (1002/39437)	4.93 (4024/81552)
1993	3.38 (434/12836)	2.25 (376/16687)	3.00 (1362/45361)	2.90 (2172/74884)
1994	2.00 (186/9291)	4.43 (500/11277)	2.61 (903/34561)	2.88 (1589/55129)
1995	2.60 (285/10937)	3.05 (323/10580)	8.33 (1177/14143)	5.00 (1785/35660)
1996	3.25 (195/5993)	3.02 (315/10413)	5.86 (808/13783)	4.36 (30216)
Overall	5.01 (2875/57375)	4.63 (3616/77988)	3.63 (6018/165354)	

Figures out of parenthesis indicate percent mortality

Figures in parenthesis indicate number of quails died out of total number of quails

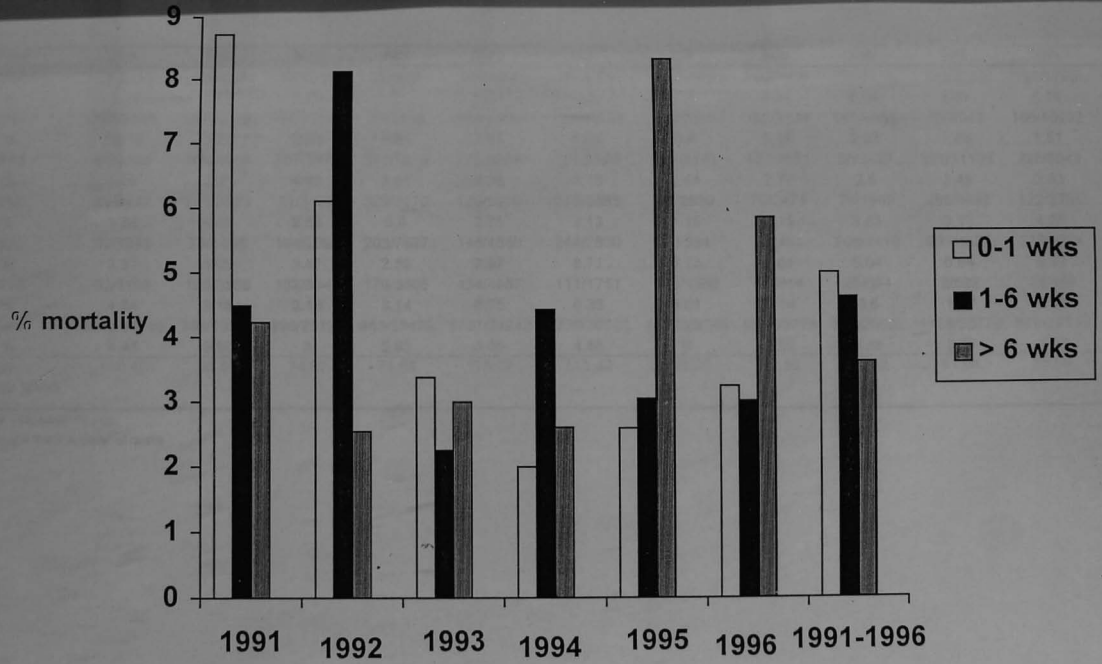

Figure 1 : AGE - WISE MORTALITY RATE IN QUAILS FROM 1991 TO 1996

Table 4: Overall mortality pattern in different months

Year	Jan.	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1991	29/2118	18/1037	18/1017	20/999	93/1764	30/1659	343/4693	166/5009	256/4235	103/2055	107/1728	438/4997
%	1.36	1.73	1.76	2	5.27	1.8	7.3	3.31	6.04	5.01	6.19	8.76
1992	752/6188	287/5015	94/3194	30/2408	594/7465	738/13188	792/9388	192/3134	141/6058	75/4047	166/10992	163/10475
%	12.15	5.72	2.94	1.24	7.95	5.59	8.4	6.12	2.32	1.85	1.51	1.55
1993	66/6196	109/4939	207/4478	231/7670	276/8206	151/5525	102/4171	107/3851	86/3427	391/11196	325/8943	121/6282
%	1.06	2.2	4.62	3.01	3.36	2.73	2.44	2.77	2.5	3.49	3.63	1.92
1994	94/6022	151/3383	91/3149	323/8970	189/6970	115/5385	41/3539	78/2471	76/1969	265/8452	122/2781	44/2038
%	1.56	4.46	2.88	3.6	2.71	2.13	1.15	3.15	3.85	3.13	4.38	2.15
1995	49/1648	76/1493	104/2990	203/7827	145/4880	244/2800	40/594	38/491	208/4119	295/4437	235/2794	148/1579
%	2.97	5.09	3.47	2.59	2.97	8.71	6.73	7.61	5.04	6.64	8.41	9.37
1996	52/1196	125/3950	182/8345	176/5605	434/4957	111/1751	109/1360	50/814	25/694	9/592	24/498	21/454
%	4.34	3.16	2.18	3.14	8.75	6.33	8.01	6.14	3.6	1.52	4.81	4.62
Total	1042/23368	766/19817	696/23173	983/33479	1731/34242	1389/30308	1427/23745	631/15778	792/20502	1138/30779	979/27736	9.35/25825
%	4.45	3.86	3	2.93	5.05	4.58	6	3.99	3.86	3.69	3.52	3.62
Seasonal Variation/ Week	110.40	95.52	74.62	71.64	125.37	113.43	149.25	98.50	95.52	91.04	86.56	89.55

Numerator = Mortality figures

Denominator = Total number of qualis

Table 5: Mortality pattern in different months in different age groups

	0 - 1 Week		Seasonal Variation Index	1 - 6 Weeks		Seasonal Variation Index	> 6 Weeks		Seasonal Variation Index
Jan	287/2623	= 10.94	216.60	292/4618	= 6.32	119.40	463/16100	= 2.87	79.72
Feb	44/2262	= 1.94	38.09	287/3123	= 9.18	173.50	435/14432	= 3.01	83.61
Mar	55/4422	= 1.24	23.80	136/4734	= 2.87	54.25	505/14017	= 3.60	100.00
Apr	169/9986	= 1.69	33.33	188/9727	= 1.93	36.48	626/13766	= 4.54	126.11
May	540/11831	= 4.56	90.47	627/10342	= 6.06	114.55	564/12061	= 4.67	129.72
Jun	290/6260	= 4.63	91.66	507/10067	= 5.03	95.08	592/13981	= 4.23	117.50
Jul	437/3543	= 12.33	244.04	540/3022	= 17.86	337.60	450/17180	= 2.61	72.50
Aug	105/1741	= 6.03	119.04	58/2668	= 2.17	41.02	468/11369	= 4.11	114.16
Sep	122/2280	= 5.35	105.95	72/4805	= 1.49	28.16	598/13417	= 4.45	123.61
Oct	344/8764	= 3.92	77.38	354/9509	= 3.72	70.32	440/12501	= 3.51	97.50
Nov	166/6307	= 2.63	51.19	389/9728	= 3.99	75.42	424/11701	= 3.62	100.55
Dec	316/5351	= 5.90	116.66	166/5645	= 2.94	55.57	453/14829	= 3.05	84.72

August (6.03%), December (5.90%), September (5.35%). In the group of 1-6 weeks age, July had maximum mortality (17.86%) followed by February (9.18%), January (6.32%) and May (6.06%). Adult birds (above 6 week) had maximum mortality in May (4.67%), followed by April (4.54%), September (4.45%), June(4.23%), August (4.11%) and November (3.62%).

Graph plotted for seasonal variation index showed two seasons of increased mortality in young (0-1 week) and grower (1-6 week) stock. Seasonal variation did not have marked affect on mortality pattern of adult birds (Figures 2 & 3).

Disease Profile

The data on prevalence of various disease in different age groups is shown in Table 6, 8 & 10 (Figures 4, 5 & 6). In 0-1 week age group, yolk sac infection and omphalitis, was the most common cause of death (52.25%). Hepatosis/hepatitis constituted the next important condition (23.16%) followed by pneumonia (4.89%) and nephrosis/nephritis (4.89%) condition.

In 1-6 weeks age group, hepatosis/hepatitis had 27.89% incidence. Next important disease condition were mycotic pneumonia (16.55%), nephrosis/nephritis condition (10.12%), coryza (8.76%), enteritis (6.27%). In 21 cases nephrosis/nephritis was the concurrent lesion alongwith hepatosis/hepatitis. In 10 cases, coryza was present alongwith pneumonic lungs. 10 cases of enteritis had nephrosis/nephritis condition alongwith. Similarly 5 cases of Gangrenous dermatitis had coryza as concurrent lesion (Table 9).

In adult birds (above 6 weeks of age) oophoritis was the most frequently diagnosed condition (17.05%). Next important conditions in

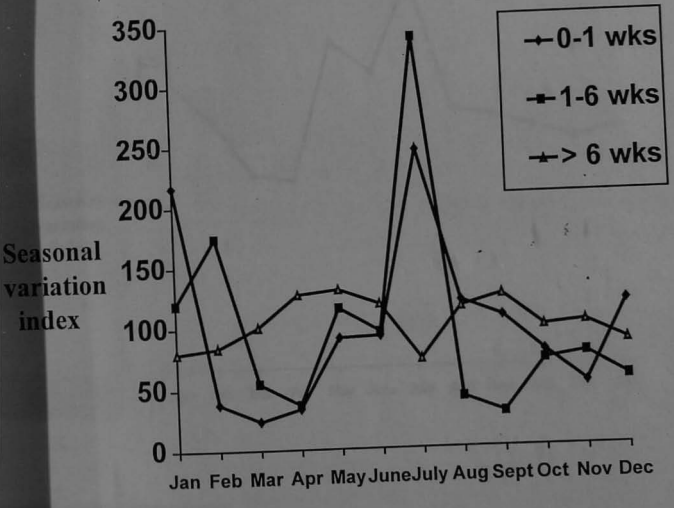

Figure 2 : Mortality pattern during different months of yea

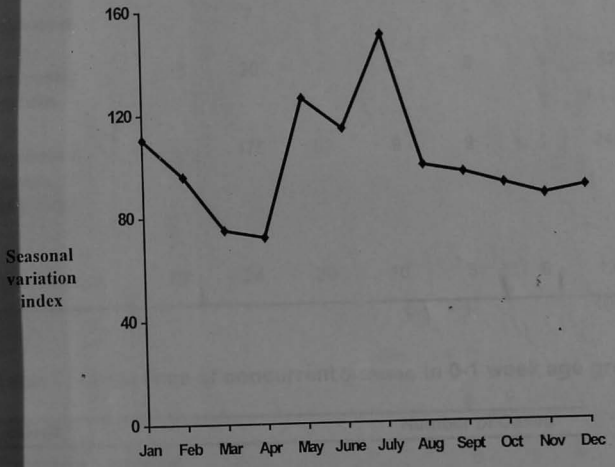

Figure 3 : Over all mortality pattern in quails throughout a year

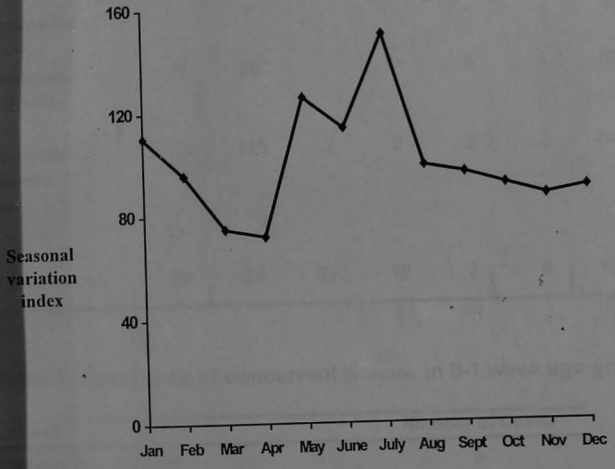

Figure 3 : Over all mortality pattern in quails throughout a year

Table 6: Prevalence of various diseases in 0 - 1 Week age group of Quails

Condition	Year						Total	Percentage
	1991	1992	1993	1994	1995	1996		
Coryza	1	6	1	2	3	1	14	1.31
Pneumonia	12	-	24	-	15	1	52	4.89
Yolk Sac Infection	159	191	29	113	36	27	555	52.25
Septicemia	-	7	-	-	-	-	7	0.65
Nephrosis / Nephrilis	25	20	-	1	5	1	52	4.89
Hepatosis / Hepatitis / Fatty Liver	-	175	52	9	9	1	246	23.16
Miscellaneous	62	24	29	10	3	8	136	12.80
							1062	

Table 7: Occurence of concurrent diseases in 0-1 week age group

Lesions	Number of Cases
Pneumonia + Hepatitis	6
Yolk Sac Infection + Hepatitis	1
Nephrosis Nephritis Syndrome + Hepatitis	17
Nephrosis Nephritis Syndrome + Pneumonia	5

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Septicemia	-	7	-	-	-	-	7	0.65
Nephrosis / Nephrilis	25	20	-	1	5	1	52	4.89
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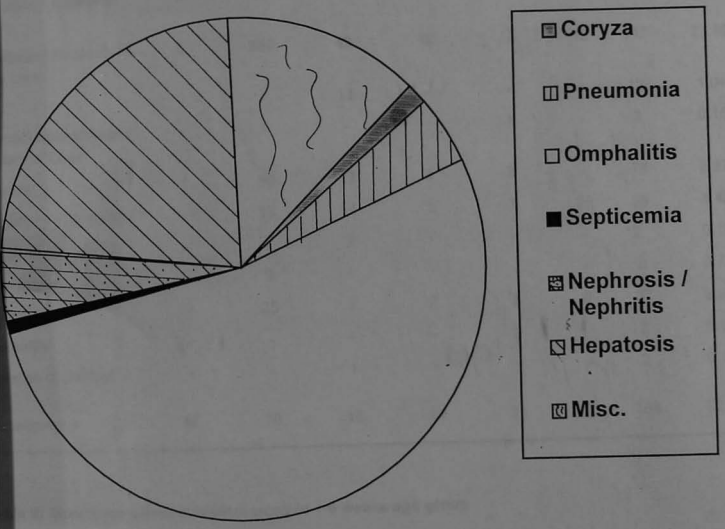


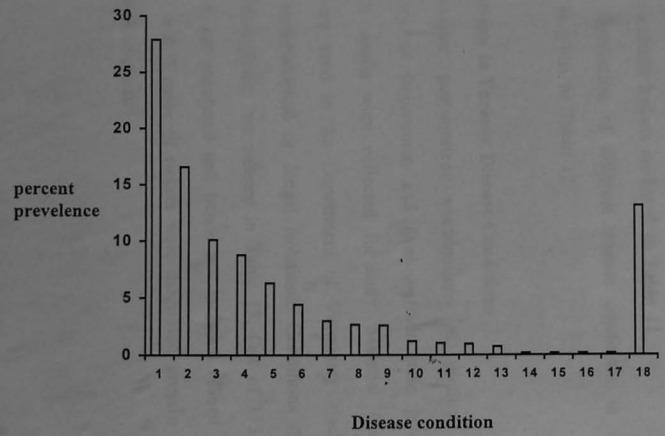
gure 4 : Prevalence of various conditions in 0-1 week age group

Table 8: Prevalence of various disease in 1 - 6 Weeks age group of quails

Condition	Year						Total	Percentage
	1991	1992	1993	1994	1995	1996		
Air sacculitis and CRD	-	14	-	-	-	1	15	1.20
Coryza	9	26	22	21	15	16	109	8.76
Pneumonia	1	12	1	6	4	9	33	2.65
Mycotic Pneumonia	-	192	10	4	-	-	206	16.55
Yolk Sac Infection	18	7	-	4	3	-	32	2.57
Septicemia	-	10	1	-	1	-	12	0.96
Nephrosis / Nephrilis/ Gout	6	83	17	4	8	8	126	10.12
Hepatosis / Hepatitis / Fatty Liver	8	259	42	12	14	12	347	27.89
IBH	-	-	13	-	-	-	13	1.04
Internal Haemorrhage / Rupture of Liver	-	1	-	-	1	-	2	0.16
Enteritis	1	50	11	8	8	-	78	6.27
Gangrenous Dermatitis	-	13	11	1	1	29	55	4.42
Egg bound	-	1	1	-	-	-	2	0.16
Heat Stroke	-	9	-	-	-	-	9	0.72
Oophoritis	-	23	3	2	1	8	37	2.97
Peritonitis	-	-	-	-	2	-	2	0.16
Prolapse of Oviduct	-	-	1	1	-	-	2	0.16
Miscellaneous	47	70	15	14	11	7	164	13.18

Table 9: Occurence of concurrent diseases in 1-6 weeks age group

Lesions	Number of Cases
Pneumonia + Coryza	10
Nephrosis Nephritis Syndrome + Coryza	5
Nephrosis Nephritis Syndrome + Hepatitis	21
Nephrosis Nephritis Syndrome + Enteritis	10
Gangrenous Dermatitis + Coryza	5



1. Hepatosis/Hepatitis
2. Mycotic Pneumonia
3. Nephrosis Nephritis Syndrome
4. Coryza
5. Enteritis
6. Gangrenous Dermatitis
7. Oophoritis
8. Pneumonia
9. Yolk Sac Infection
10. Air Sacculitis/CRD
11. IBH
12. Septicemia
13. Heat Stroke
14. Internal Haemorrhages/ Rupture of Liver
15. Peritonitis
16. Prolapse of Oviduct
17. Egg bound
18. Miscellaneous.

Figure 5 : Prevalence of various conditions in 1-6 week age group

descending order of prevalence were hepatosis/hepatitis condition (13.00%) gangrenous dermatitis/cannibalism (10.15%), coryza (7.74%), enteritis (6.96%), Marek's disease (6.18%), septicemia (4.04%), eggbound/salpingitis (3.94%), heat stroke (3.80%), nephrosis/nephritis/gout (3.58%), peritonitis (3.44%), air-sacculitis/CRD (1.77%), prolapse of oviduct (1.38%). Other conditions were Inclusion Body Hepatitis (0.63%), mycotic pneumonia (0.67%) and Lymphoid leukosis (0.31%). Oophoritis and coryza were concurrent in 23 cases. Nephrosis nephritis syndrome along with hepatitis/hepatosis was present in 15 cases. Oophoritis and enteritis occurred concurrently in 14 cases. Hepatosis/hepatitis condition occurred with enteritis in 13 cases. Oophoritis occurred with aplastic aneamia and dermatitis in 12 cases each. Other concurrent lesions are listed in Table 11.

The detailed distribution of different disease conditions in different months of the year is given in Table 12.

Pathological Alterations in Various Disease Conditions

From the routine post-mortem examinations at poultry Disease Diagnostic Laboratory of Department and from various state government farms, 175 Japanese quails were collected for study. Material from fresh necropsied cases were send to the Department of Veterinary Bacteriology and Virology for bacteriological or fungal isolations. Conditions diagnosed grossly and histopathologically are enlisted in Table 13. Out of 175 cases, 9 were found to have got autolysed and hence not included. Three cases of cystic right oviduct and 7 cases of rickets were diagnosed grossly but were not examined histopathologically.

Table 10: Prevalence of various disease in adult quails (over 6 Weeks age group)

Condition	Year						Total	Percentage
	1991	1992	1993	1994	1995	1996		
Air sacculitis and CRD	19	7	6	2	7	9	50	1.77
Coryza	11	83	23	34	34	33	218	7.74
Pneumonia	3	2	13	1	2	2	23	0.81
Mycotic Pneumonia	-	5	1	4	6	3	19	0.67
Aplastic / Hypoplastic Anemia	-	-	-	2	7	7	16	0.56
Septicemia	5	21	77	2	4	5	114	4.04
Nephrosis / Nephritis/ Gout	20	11	21	13	19	18	102	3.58
Hepatosis / Hepatitis / Fatty Liver	-	104	139	39	59	25	366	13.00
IBH	5	2	1	5	4	1	18	0.63
Internal Haemorrhage / Rupture of Liver	6	10	1	4	6	1	28	0.99
Enteritis	22	37	69	35	20	13	196	6.96
Dermatitis / Cannibalism	5	56	47	21	57	100	286	10.15
Egg bound / Salpingitis / Egg peritonitis	13	5	74	10	6	3	111	3.94
Heat Stroke	1	1	-	6	89	10	107	3.80
Oophoritis	52	65	76	94	92	101	480	17.05
MD	-	-	24	104	28	18	174	6.18
LL	-		-	9	-	-	9	0.31
Peritonitis	-	5	8	2	79	3	97	3.44
Prolapse of Oviduct	1	4	11	6	6	9	39	1.38
Miscellaneous	65	71	64	71	60	32	363	12.89

Table 11: Occurence of concurrent diseases in adult quails

Lesions	Number of Cases
Oophoritis + Coryza	23
Nephrosis Nephritis Syndrome + Hepatitis	15
Oophoritis + Enteritis	14
Hepatosis/Hepatitis + Enteritis	13
Aplastic Anaemia + Oophoritis	12
Gangrenous Dermatitis + Oophoritis	12
Gangrenous Dermatitis + Coryza	7
Nephrosis Nephritis Syndrome + Oophoritis	5
Salpingitis + Oophoritis	3
Gangrenous Dermatitis + Aplastic Anaemia	3

Table 10: Prevalence of various disease in adult quails (over 6 Weeks age group)

Condition	Year						Total	Percentage
	1991	1992	1993	1994	1995	1996		
Air sacculitis and CRD	19	7	6	2	7	9	50	1.77
Coryza	11	83	23	34	34	33	218	7.74
Pneumonia	3	2	13	1	2	2	23	0.81
Mycotic Pneumonia	-	5	1	4	6	3	19	0.67
Aplastic / Hypoplastic Anemia	-	-	-	2	7	7	16	0.56
Septicemia	5	21	77	2	4	5	114	4.04
Nephrosis / Nephritis/ Gout	20	11	21	13	19	18	102	3.58
Hepatosis / Hepatitis / Fatty Liver	-	104	139	39	59	25	366	13.00
IBH	5	2	1	5	4	1	18	0.63
Internal Haemorrhage / Rupture of Liver	6	10	1	4	6	1	28	0.99
Enteritis	22	37	69	35	20	13	196	6.96
Dermatitis / Cannibalism	5	56	47	21	57	100	286	10.15
Egg bound / Salpingitis / Egg peritonitis	13	5	74	10	6	3	111	3.94
Heat Stroke	1	1	-	6	89	10	107	3.80
Oophoritis	52	65	76	94	92	101	480	17.05
MD	-	-	24	104	28	18	174	6.18
LL	-		-	9	-	-	9	0.31
Peritonitis	-	5	8	2	79	3	97	3.44
Prolapse of Oviduct	1	4	11	6	6	9	39	1.38
Miscellaneous	65	71	64	71	60	32	363	12.89

Table 11: Occurence of concurrent diseases in adult quails

Lesions	Number of Cases
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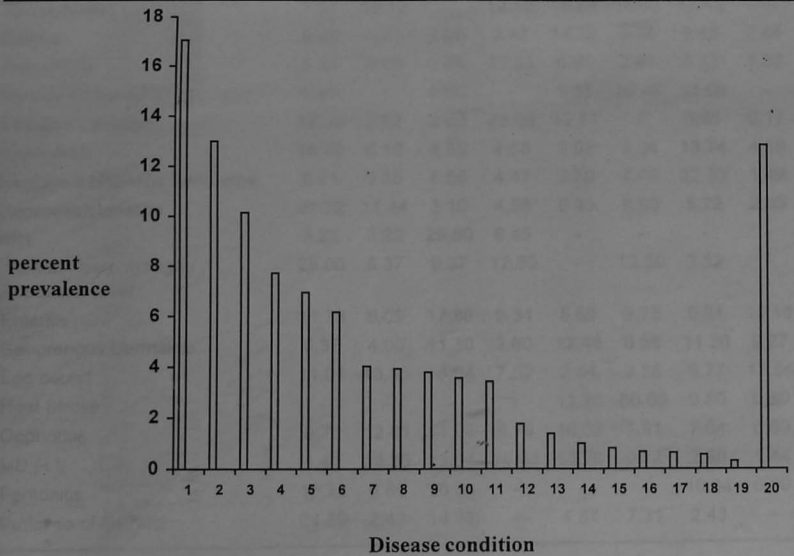
Nephrosis Nephritis Syndrome + Hepatitis	15
Oophoritis + Enteritis	14
Hepatosis/Hepatitis + Enteritis	13
Aplastic Anaemia + Oophoritis	12
Gangrenous Dermatitis + Oophoritis	12
Gangrenous Dermatitis + Coryza	7
Nephrosis Nephritis Syndrome + Oophoritis	5
Salpingitis + Oophoritis	3
Gangrenous Dermatitis + Aplastic Anaemia	3



1. Oophoritis
2. Hepatosis/Hepatitsa
3. Gangrenous Dermatitis
4. Coryza
5. Enteritis
6. Marek's Disease
7. Septicemia
8. Egg bound/Salpingitis
9. Heat Stroke
10. Nephrosis Nephritis Syndrome
11. Peritonitis
12. Air Sacculitis/CRD
13. Prolapse of Oviduct
14. Internal Haemorrhages/ Rupture of Liver
15. Pneumonia
16. Mycotic Pneumonia
17. IBH
18. Aplastic/ Hypoplastic Anaemia
19. LL
20. Miscellaneous.

Figure 6 : Prevalence of various conditions in adult quails (Over 6 weeks age)

Table 12: Prevalence of different disease conditions in different months of year 1991-1996

Condition	Jan.	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Total	%
Air sacculitis / CRD	7.57	12.12	-	12.12	10.60	13.63	12.12	7.57	6.06	18.18	-	-	66	1.51
Coryza	9.45	1.71	2.86	3.43	14.32	5.44	9.45	7.44	18.62	11.46	11.17	4.58	349	8.01
Pneumonia	5.33	4.00	2.66	17.33	6.66	2.66	5.33	1.33	6.66	8.00	21.33	18.66	75	1.72
Mycotic Pneumonia	0.45	-	0.90	-	1.35	36.46	53.60	-	4.05	0.90	2.25	-	222	5.09
Yolk Sac Infection	22.48	5.62	2.89	23.50	12.77	-	0.85	0.17	-	2.04	0.17	29.47	587	13.47
Septicemia	16.79	6.10	4.58	4.58	9.92	5.34	13.74	4.58	3.81	3.81	5.34	21.37	131	3.00
Nephrosis Nephritis Syndrome	6.71	3.35	5.59	4.47	9.70	7.08	32.83	1.49	2.61	8.95	12.31	4.85	268	6.15
Hepatosis/Hepatitis	21.72	11.44	3.10	4.55	6.49	9.90	5.72	2.03	1.64	13.19	8.82	11.25	1031	23.66
IBH	3.22	3.22	25.80	6.45	-	-	-	-	9.67	16.12	25.80	9.67	31	0.71
Internal Haemorrhage / Rupture of Liver	25.00	9.37	9.37	12.50	-	12.50	3.12	-	3.12	3.12	6.25	15.62	32	0.73
Enteritis	11.78	6.09	17.88	9.34	5.69	9.75	0.81	10.16	4.06	4.47	10.56	9.34	246	5.64
Gangrenous Dermatitis	6.37	4.05	11.30	2.60	12.46	6.66	11.30	9.27	8.11	5.79	16.52	5.50	345	7.91
Egg bound	11.01	13.55	16.94	7.62	2.54	3.38	6.77	13.55	7.62	5.93	7.62	3.38	118	2.70
Heat Stroke	-	-	-	-	11.20	80.00	0.80	0.80	-	-	-	-	125	2.86
Oophoritis	9.75	12.61	11.08	8.79	10.32	1.91	7.64	6.69	8.03	5.16	10.51	7.45	523	12.00
MD / LL	1.44	19.56	13.04	24.63	13.76	0.72	2.89	1.44	5.07	8.69	8.69	-	138	3.16
Peritonitis	17.24	6.89	10.34	-	-	-	10.34	6.89	10.34	-	17.24	20.68	29	0.66
Prolapse of Oviduct	24.39	2.43	14.63	-	4.87	7.31	2.43	-	4.87	-	12.19	26.82	41	0.94

Table 13: Details of different disease

Sr. No.	Disease	No. of Cases	Remarks
1	Marek's Disease	21	Infilteration of pleomorphic lymphocytes in various organs **(Table 14)**
2	Lymphoid Leukosis	1	Uniform sized lymphoblasts in various organs.
3	Mycotic Pneumonia	14	Granulomatous lesions in lungs and air sacs. Fungal hyphae demonstrated by Grocott's stain.
4	Fowl Typhoid	4	*Salmonella gallinarum* isolated from liver with necrotic foci, bronze colour, mottled spleen. Ova having long stalks and oily contents.
5	Colibacillosis	7	*Escherichia coli* isolated from various cases.
6	Staphylococcal Infection	3	Staphylococcus isolated from necrotic patches on liver.
7	Gangrenous Dermatitis	17	Gram positive cocci and spore bearing gram positive rods in necrosed areas of skin.
8	Septicemia	4	Epicardial & serosal petechiation. Fibrinoid splenic necrosis. No attempt to isolate organisms.
9	Infectious Coryza	6	Mucopurulant exudate from sinuses. Mononuclear cells infilteration and hyperplasia of lining of nasal sinuses.
10	Chronic Respiratory Disease	1	Lymphoid follicles in trachea and tubuloalveolar hyperplasia of tracheal glands. Thickened mucosa of nasal sinuses by infilterating mononuclear cells.
11	Nephrosis / Nephritis Syndrome	19	Kidneys enlarged with distended tubules. Three cases of visceral gout had chalky white deposits on various organs.

(contd.)

Table 13 (contd.): Details of different disease

Sr. No.	Disease	No. of Cases	Remarks
12	Yeast Infection	1	Granulomatous lesion in kidney and budding yeast cells.
13	Non-specific Lesions and Concurrent conditions	45	Microscopic lesions of toxic hepatosis and nephrosis. Aflatoxin levels high in feed.
14	Traumatic Ventriculitis	1	Sharp objects pierced through gizzard.
15	Vent Gleet	1	*Aspergillus fumigatus* isolated from vent and whitish thick deposits on surface of vent. Heavy fungus growth, necrosis and cellular infilterations, giant cells.
16	Haemorrhagic Syndrome	3	Irregular patches of haemorrhage in breast and thigh muscles, internal organs. Hypoplastic anemia, emaciation also present.
17	Suppurative Myositis	5	Heterophils infilterations and haemorrhages in muscles around broken legs.
18	Cystic Right Oviduct	3	
19	Bony-Metaplasis in lungs	1	Lung tissue with small bony structures.
20	Deficiency of Vitamin E / Selenium	2	Muscular dystrophy and haemorrhages in cerebellum
21	Rickets	7	Beading of ribs in young birds.

Marek's Disease : It was diagnosed in 21 cases.

Gross Lesions : Enlargement of liver and spleen with presence of whitish areas was consistent finding in most of the cases (Figure 7). The duodenum portion of intestine looked highly enlarged and thickened. Focal or diffuse white thickenings of wall of small intestine especially of duodenum was present in all cases (Figure 8). Ulcerations were sometimes observed in mucosa of intestine and proventriculus. Congestion in liver and subcapsular haemorrhages in spleen were sometimes observed. Out of 10 female cases, oophoritis was grossly seen in 6.

Microscopic Lesions

Liver : Infilteration of pleomorphic lymphocytes was present which ranged from a few lymphocytes, small focal aggregates of lymphocytes to extensive infilteration disrupting and even replacing the liver parenchyma (Figure 9). In severe cases, the disintegrated islets of hepatic cells were discernible. Sometimes lymphocytes were diffusely distributed with relatively mild damage to liver parenchyma. Other features were presence of some plasma cells and mitotic figures. Hepatocytes changes ranged from granular degeneration, vacuolar degeneration and fatty changes. In some cases, liver was not infilterated by neoplastic cells.

Congestion and haemorrhages were invariably present.

Intestine : Intestinal lesions were present in all cases. Involvement of intestine ranged from moderate to extensive infilteration of pleomorphic cells resulting in the replacement of all the layers of intestine to varying degrees (Figure 10). Low to heavy infilterations on serosal surface were also present. In severe cases, muscular layer was disintegrated. The presence of a few intestinal glands at base of villi helped in histological identification

Figure 7 : Marek's Disease : Enlarged and whitish spleen (A) alongwith white spots on liver (B).

Figure 8 : Marek's Disease : Highly enlarged spleen with subcapsular haemorrhages (A), whitish areas on liver (B) alongwith white thickenings of the intestine (C).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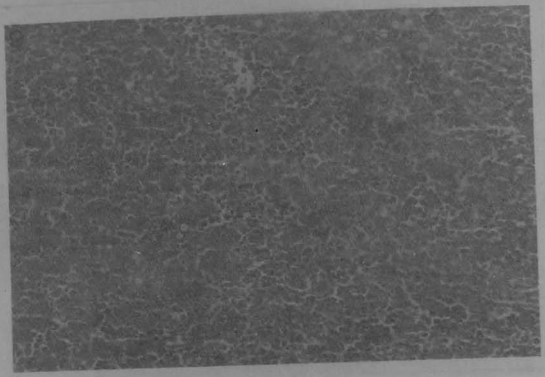
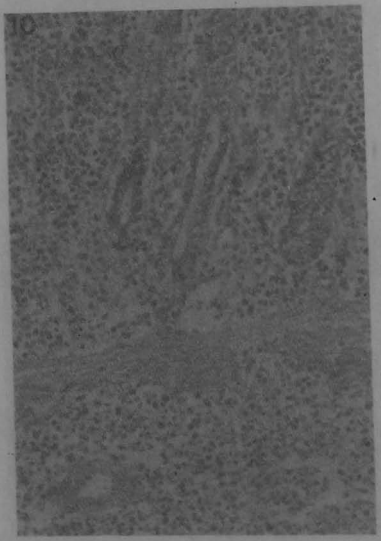
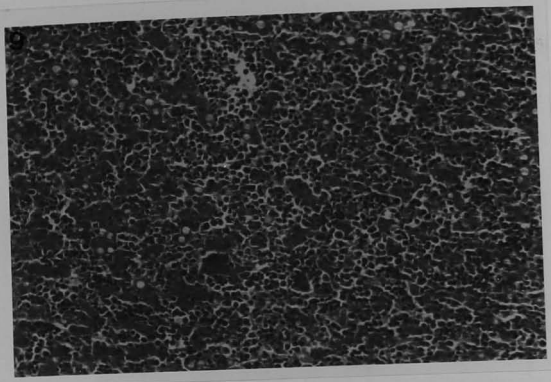
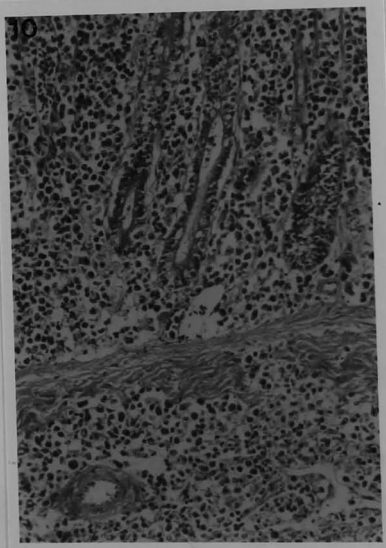








of intestine. Few plasma cells and mitotic figures were also present. Necrosis of infilterated villi at places was seen in two cases.

Proventriculus : Proventriculus was involved in 12 out of 21 cases. Mucosa and sub-mucosa were the frequently involved parts, serosa was least involved (Figure 11). In severe cases, massive accumulation of neoplastic cells was also noticed in glandular areas alongwith mucosa, serosa and muscular layer.

Gizzard : In 6 cases there was no involvement of gizzard. Only mild infilterations were seen in either mucosa or serosa or both in the rest of cases.

Spleen : Either no infilterations or moderate to marked infilterations were present. Haemorrhage was seen in one case.

Kidney : Congestion, haemorrhages, degenerations and necrosis of tubular epithelium were frequently present. Sometimes lumen of tubules were narrowed or obliterated. Infilterations were either absent or few cells to moderate infilterations.

Ovary : Varying degrees of infilterations by pleomorphic lymphoid cells were present in 10 out of 12 cases (Figure 12). One case also had presence of severe congestion and haemorrhage.

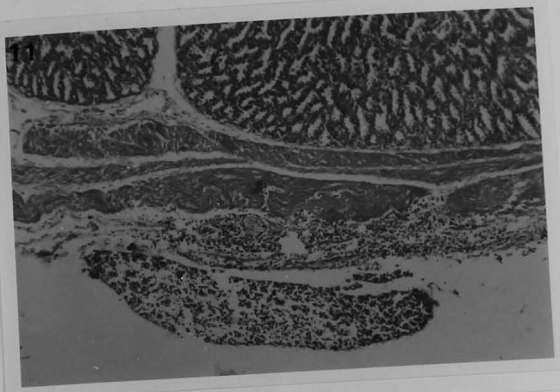
Brain : Congestion in brain and meninges was frequent In one case infilteration of pleomorphic lymphocytes in meninges and slight extension into brain parenchyma was seen.

Lungs and Trachea : Congestion of blood vessels and haemorrhages into parenchyma were present in all cases. Pleomorphic lymphocytic foci were seen in three cases. Mycotic granuloma and granuloma with bacterial colonies were the concurrent lesions in 2 cases.

Figure 11 : Marek's Disease : Infilteration of pleomorphic lymphocytes in serosa and muscular layer of proventriculus. H&E x 80.

Figure 12 : Marek's Disease : Heavy infilteration of pleomorphic lymphocytes in ovarian tissue. H&E x 325.

Figure 13 : Marek's Disease : Nerve - Edema alongwith moderate infilteration of lymphoid cells in perineurium. H&E x 160.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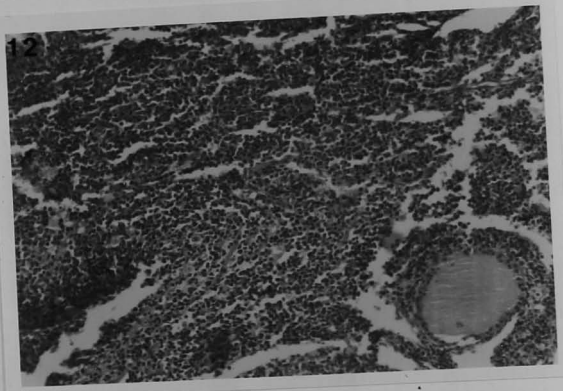
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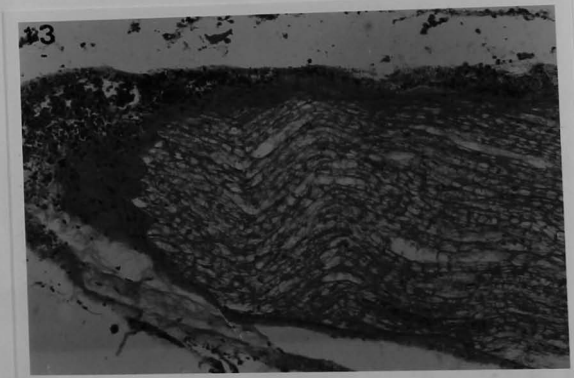
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In submucosa of trachea a few pleomorphic cells were present in one case having moderate infilteration of tumor cells in lung alongwith bacterial pneumonia. Few mononuclear cells and epithelial hyperplasia of tracheal epithelium was present in case showing mucus discharge from nostrils.

Nerves : No lesions were observed in peripheral nerves except in two cases, one of which had a few cells in the interneural space and the other had moderate infilteration in perineurium alongwith oedema in between the nerve fibrils (Figure 13).

No lymphocytic infilteration was seen in brain. Other organs did not have specific lesions of MD. However, congestion, haemorrhage and varying degrees of degeneration was observed.

The detailed distribution of lesions in different organs in MD cases is given in Table 14.

Lymphoid Leukosis

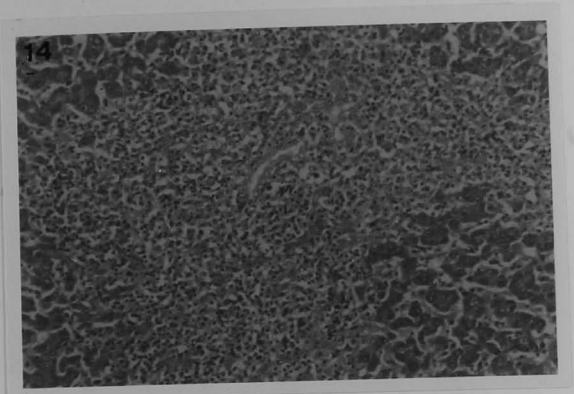
A case grossly diagnosed as of nephritis showed lesions of lymphoid leukosis microscopically. Liver had granular degeneration and focal infilteration by uniform sized lymphoblasts (Figure 14). Similar cells were present in ovary, mucosa of gizzard, mucosa of proventriculus resulting in replacement of the parenchyma (Figure 15). The parenchymal organs also had granular degeneration in liver, congestion, haemorrhage and necrosis in kidneys, congestion in brain, congestion and haemorrhage in lungs. No lesions were present in heart.

Mycotic Pneumonia : It was diagnosed in 14 cases.

Gross-Lesions : The lesions consisted of small whitish consolidated areas to well-developed nodules on the surface of the lungs and deep in the

Figure 14 : Lymphoid Leukosis : Infilteration of uniform sized lymphoblasts in liver parenchyma. H&E x 160.

Figure 15 : Lymphoid Leukosis : Uniform sized lymphoblasts infilterating the ovary. H&E x 160.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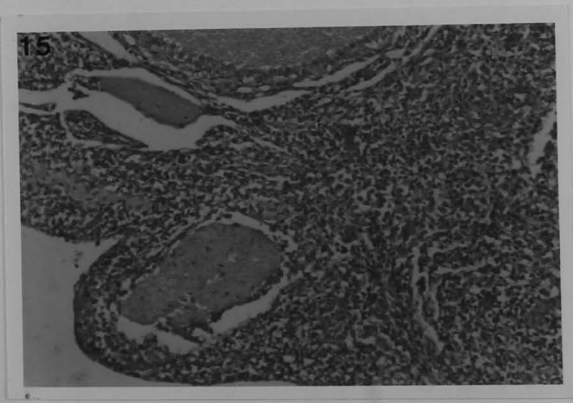
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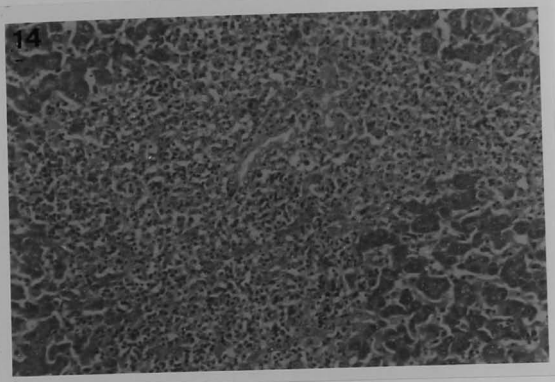
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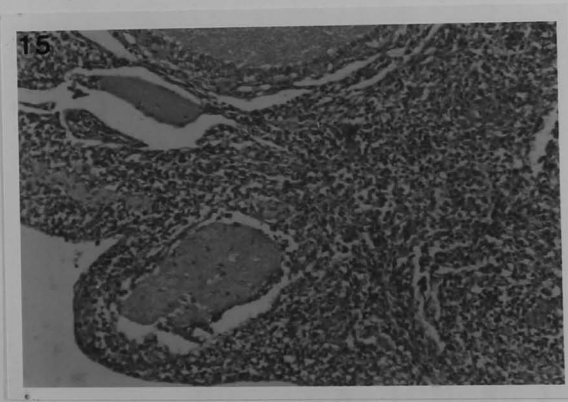
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Table 14: Histopathological Details of Distribution of Lesions of MD in Different Organs

Case No.	Age	Sex	Organwise distribution											
			Liver	Intestine	Kidney	Spleen	Ovary/Testes	Proventriculus	Gizzard	Lungs	Brain	Oviduct	Heart	Nerve
3324	Adult	F	+	+++	-	x	++	-	-	-	-	-	-	-
5730	Adult	F	++	++++	++	++++	+++	+	+	-	-	-	-	-
3836	Adult	M	++++	+++	x	++	-	+++	-	-	-	-	-	-
5178	Adult	M	++++	++	++	x	-	+	+	-	-	-	-	-
2174	Adult	F	++	+++	+	++	++	+++	++	-	-	-	-	-
5073	Adult	F	-	+++	-	-	-	++	-	+	-	-	-	-
5366	Adult	F	++	+++	-	x	+++	-	-	-	-	-	-	-
5327	Adult	F	+++	+++	+	++	++	++	+	++	-	-	-	-
3285	Adult	F	++	++	-	-	-	++	-	-	-	-	-	-
5421	Adult	F	-	++	-	-	+	-	+	-	-	-	-	-
5653	Adult	F	+++	+++	+	+++	+++	++	++	++	+	-	-	+

- = No infiltration; + = Mild infiltration; ++ = Moderate infiltration; +++ = Severe infiltration; ++++ = Extensive infiltration

Table 14 (cont.): Histopathological Details of Distribution of Lesions of MD in Different Organs

Case No.	Microscopic Lesions									
	Liver	Intestine	Kidney	Spleen	Ovary/Testes	Proventiculus	Lungs	Brain	Heart	Nerve
Q1	++++	++	x	x	++	++	x	x	x	x
Q2	++	+++	-	++	x	++	-	x	x	-
Q3	+	+++	x	x	x	x	x	x	x	x
Q4	++	++	-	+++	x	-	-	-	x	-
Q5	++	+++	x	x	-	++	x	x	x	x
Q6	++	x	+++	+	+++	x	x	-	-	++
Q7	+++	++	x	-	-	++	-	-	-	x
Q11	-	+++	x	+	-	x	x	-	x	-
Q12	++	++++	+	x	-	x	+	-	-	x
Q17	+++	++++	x	-	+++	++	+	x	x	-

- = No infiltration; + = Mild infiltration; ++ = Moderate infiltration; +++ = Severe infiltration; ++++ = Extensive infiltration

x = No histopathological study conducted

parenchyma (Figure 16). In some cases, there was severe involvement of lungs with extension of lesions into thoracic and abdominal air-sacs. Lungs were enlarged and hard with discrete white nodules. In two cases, mycotic lesions were present concurrent with gross lesions of lymphoproliferative disease. (MD and LL).

Isolations : Seven cases were sent for fungal isolation. *Aspergillus fumigatus*, *A. flavus* and *A. flavus* alongwith *A. ochraceus* were isolated from 5, 1 and 1 cases respectively.

Microscopic lesions

Lungs : Congestion and haemorrhage was present in all cases. Lung parenchyma had varying sized granulomatous lesions. The granulomatous lesions consisted of varying sized central necrotic area with fungal hyphae surrounded by a rim of inflammatory cells predominantly macrophages, lymphocytes, a few giant cells and heterophils (Figure 17 & 18). In severe cases, lung parenchyma was fully disrupted, just having fibrinous exudate filling the ruptured air spaces in between granulomas. The cases from which *A. flavus* was isolated had small sized granulomas with more cellularity and less necrosis. The cases from *A. fumigatus* was isolated had extensive lung involvement and large foci. The lesions consisted of large central caseous necrotic eosinophilic mass containing fungal hyphae (Figure 19 & 20), surrounded by zone of degenerating infilterated cells and then a rim of macrophages, giant cells and fibroblasts. Bronchioles sometimes were seen filled with mucus exudate, stained fibrin and nuclear fragments. In one case a large granuloma with central empty space filled with numerous hyphae and spores was seen.

Figure 16 : Mycotic Pneumonia : Varying sized white to red areas on the surface of lung.

Figure 17 : Mycotic Pneumonia : Granulomas with central eosinophilic necrotic mass surrounded by mononuclear cells. Lung parenchyma disrupted and compressed. H&E x 80.

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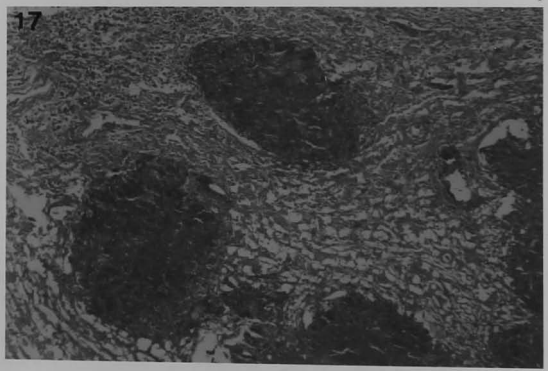
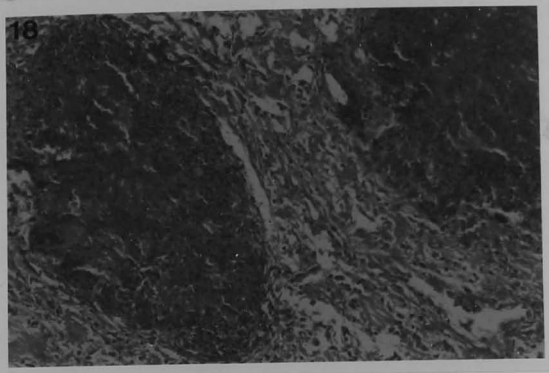


Figure 18 : Mycotic Pneumonia : Figure 17 at higher magnification. H&E x 160.

Figure 19 : Mycotic Pneumonia : Lung showing septate hyphae in granulomatous lesions Grocott's stain. x 160.

Figure 20 : Mycotic Pneumonia : Higher magnification of Figure 19 Grocott's stain. x 325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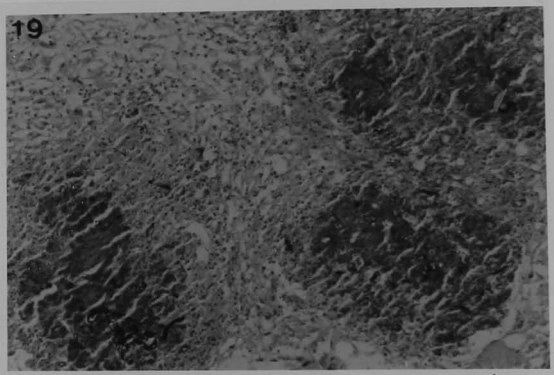
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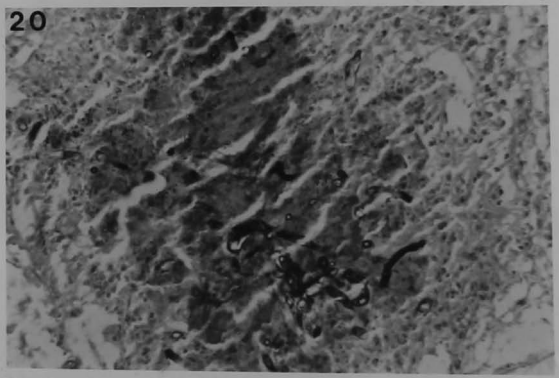
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In addition to mycotic lesions, one case had pleomorphic lymphocytic infilteration in lungs indicative of Marek's Disease and in the other case with lymphoproliferative lesions seen as infilteration of uniform sized lymphoblasts in lungs indicating lymphoid leukosis.

Air sacs : In cases of air sac involvement, lesions consisted of well developed granulomas in the air sac walls, which were thickened by fibrosis similar to those of lungs (Figure 21 & 22). The hyphae with fruiting bodies of *A. fumigatus* were present in granulomas of air sac in two cases.

Skeletal muscle in two cases at site of attachment to bone showed infilteration of mononuclear cells and fibrinous material but fungus was not found in special stain.

No lesions were seen in trachea except in one case which had mononuclear cell infilteration and epithelial hyperplasia of tracheal mucosa.

Chronic non-suppurative inflammation was encountered in ovary characterized by mononuclear cell infilteration in two cases.

No changes were present in brain in most of cases. Congestion in a few, mild meningitis, satellitosis alongwith neuronophagia was observed in one case each.

Various degrees of congestion, haemorrhage and degenerative changes were seen in other organs viz. kidneys, heart, liver.

Fowl Typhoid : Fowl typhoid were seen in 4 cases.

Gross Lesions : The gross lesions recorded in natural outbreak in adult birds comprised of necrotic foci on liver alongwith bronze discolourisation, mottled spleens, cyanosed and congested lungs, congestion of other visceral organs, oophoritis with long stalked ova having oily contents. Coryza was present in one case.

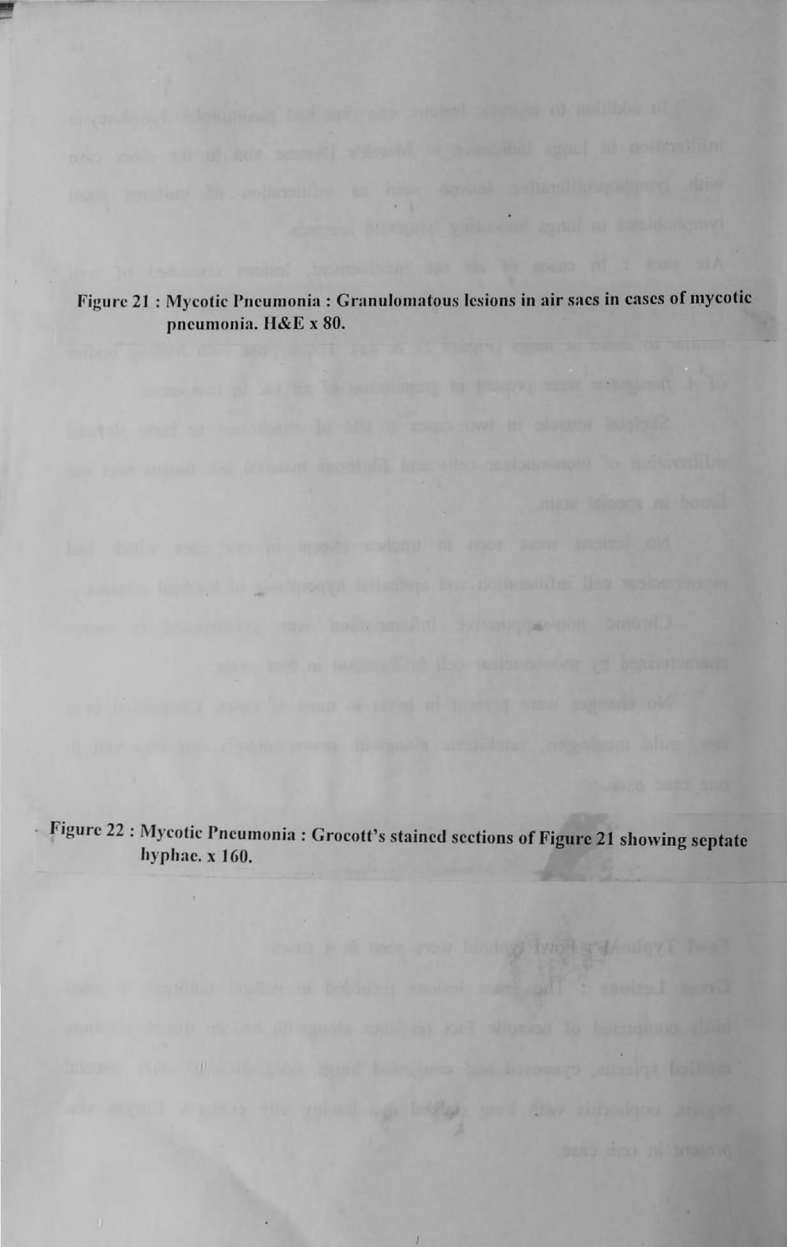

Figure 21 : Mycotic Pneumonia : Granulomatous lesions in air sacs in cases of mycotic pneumonia. H&E x 80.

Figure 22 : Mycotic Pneumonia : Grocott's stained sections of Figure 21 showing septate hyphae. x 160.


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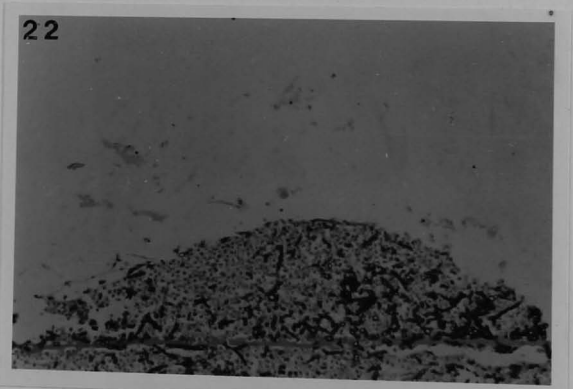
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Isolation : Salmonella gallinarum was isolated from liver and spleen of these birds. These were got serotyped from CRI, Kasauli and found to be (9 : 12 :-: -) type.

Microscopic lesions

Microscopic lesions in liver comprised of fatty changes, coagulative necrosis alongwith congestion and haemorrhages. Focal acellular areas of fibrinoid matrix with karyorrhectic material of hepatic cell nuclei and a few mononuclear cells indicating the initial stage of typhoid granuloma formation were present. Fully formed typhoid granulomas with mononuclear cell infilteration into these necrosed acellular areas were also seen (Figure 23).

Severe congestion and haemorrhage, exudations into secondary bronchi and air spaces marked the changes in lungs. Sometimes mild mononuclear cell infilteration in the interstitial tissue were present. Microangiopathy was observed in one case with severe lung lesions as degeneration of smooth muscle cells and elastic fibres of tunica media causing microaneurysm (Figure 24). Thickening of tunica intima was also present.

Kidney lesions consisted of congestion, haemorrhage, coagulative necrosis in tubular epithelium. Myocardium showed congestion and granular degenerative changes.

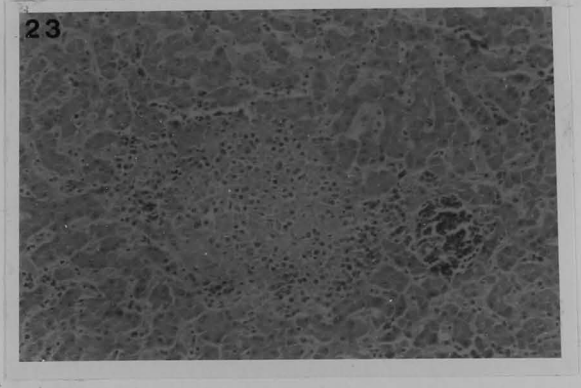
In spleen mild reticular cell proliferation was present. Fibrinoid necrosis characterized by presence of acellular fibrinoid areas and decreased cellularity in spleen was most frequently observed (Figure 25).

Severe congestion, haemorrhage and infilteration of mononuclear cells in the ovary were the usual lesions (Figure 26). In intestines only congestion in serosa was observed. However, in one case mild mononuclear cell infilteration could be observed. Proventriculus and gizzard had only congestion in serosa.

Figure 23 : Fowl Typhoid : Liver showing congestion and typhoid granuloma characterized by necrosed area having karyorrhectic material alongwith of infilteration of mononuclear cells. H&E x 160.

Figure 24 : Fowl Typhoid : Microaneurysn observed in lungs in the cases having degeneration of smooth muscle cells and elastic fibres of tunica media of pulmonary blood vessel. H&E x 160.

Figure 25 : Fowl Typhoid : Fibrinoid acellular areas in spleen in natural outbreak. H&E x 160.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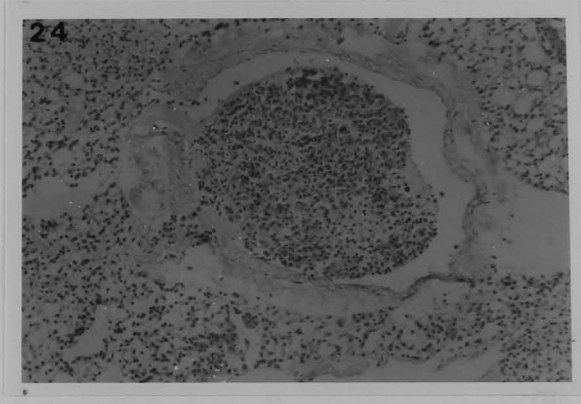
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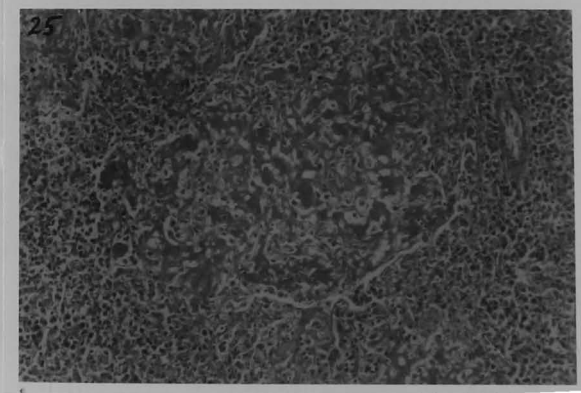
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Colibacillosis

Escherichia coli was isolated from 7 cases with lesions of oophoritis, mottled liver, egg bound, congested intestines, prolapse of oviduct, alongwith coryza. Lesions typical of avian colibacillosis i.e. perihepatitis, pericarditis, peritonitis seen in two cases also yielded *E. coli* on isolation.

Microscopically, congestion, haemorrhages in liver alongwith varying degree of degeneration particularly granular or vacuolar degenerations were noticed. In case of perihepatitis, thickened liver capsule with fibrinous exudate and mononuclear cells was present (Figure 27).

Myocardium had congestion, haemorrhages alongwith granular degenerations. Pericarditis was characterized by fibrinous material and mononuclear cells in epicardial sac.

The lesions in lungs comprised of congestion with extensive haemorrhages in the tissue.

Intestines had congestion of serosa, presence of fibrinous exudate and infilteration of mononuclear cells in serosa indicating peritonitis was observed.

Proventriculus was mostly without any changes. Only congestion, haemorrhage and few mononuclear cells in serosa were occasionally seen.

Infilteration of mononuclear cells in serosa of gizzard alongwith congestion and haemorrhage was commonly observed.

In other organs, severe congestion and haemorrhage were observed alone or alongwith infilteration of mononuclear cells. Oviduct showed severe congestion and haemorrhage in serosa and mucosal surface.

Mild reticular cell hyperplasia was occasionally observed in spleen. In a few cases, brain had congestion with or without gliosis.

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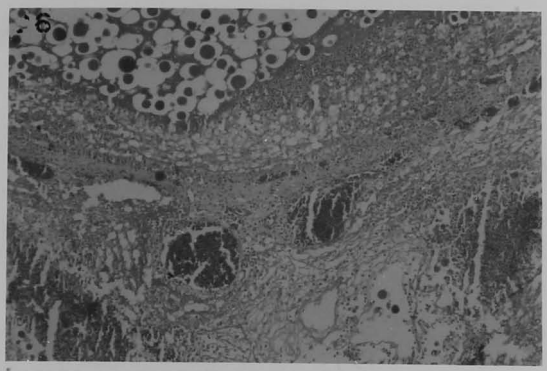
Mild reticular cell hyperplasia was occasionally observed in spleen. In a few cases, brain had congestion with or without gliosis.

Figure 26 : Fowl Typhoid : Acute oophoritis characterized by severe congestion, haemorrhages and mononuclear cells in ovary. H&E x 80.

Figure 27 : Colibacillosis : Adhesions between liver and pericardium by fibrinous material interspersed by large number of mononuclear cells indicating perihepatitis and pericarditis. H&E x 80.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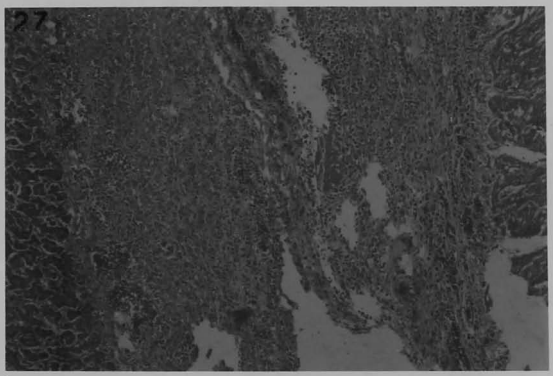
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Kidney lesions consisted of congestion, haemorrhage, extensive necrosis and degeneration in parenchyma. In one case some foci of a few lymphoid cells were seen in kidney.

Staphylococcal Infection

Staphylococcus was isolated from three cases from the necrosed areas of liver. Congestion was seen in other visceral organs without any gross abnormality. Microscopically, lesions consisted of congestion, haemorrhage, vacuolar degeneration and necrosis in liver. Severe congestion and haemorrhage were noticed in the lungs. The blood vessels in the lungs had edematous tunica adventitia. Extensive congestion and haemorrhages in kidneys, granular degeneration in myocardium, congestion and haemorrhages in the intestinal mucosa and fibrinous exudate on intestinal serosa, congestion and meningitis in brain, edema in submucosa of trachea were seen.

Gangrenous Dermatitis

It was recorded in 17 cases. Grossly, the lesions were present on head, back, wings and legs. Head was more commonly involved. The wings of birds showed lesions due to injury by tightening of wing bands (Figure 28). Lesion comprised of gangrene, necrosis and haemorrhages in skin of affected area. Congestion of visceral organs, oophoritis, mottling of spleen and liver were commonly found. Liver in one case had large necrotic patch on its surface while another case had whitish granular areas of few millimeter size on a lobe of liver. In some cases, mucus exudate in nasal sinuses was also present.

Microscopic lesions

In epidermis and dermis, haemorrhage, extensive necrosis and gangrence alongwith infilteration of mononuclear cells in dermis was present (Figure 29). Epidermis was eroded at places where only necrotic and gangrenous mass was present. Large number of bacterial colonies were present in necrosed areas. MacCallum Good Pasteur's stain revealed these bacteria to be predominantly gram positive cocci (Figure 30), alongwith some spore bearing gram positive bacilli and some gram negative bacilli. Chronic non-supportive dermatitis with healing tissue was found in one case (Figure 31).

Congestion and haemorrhages in lungs, congestion and haemorrhages in liver alongwith varying degrees of degenerations, granular degeneration in myocardium, congestion, haemorrhages, degeneration and necrosis changes in kidneys were the frequent lesions. Extensive reticular cell hyperplasia in spleen was present in two cases. Changes in brain consists of congestion alone or alongwith gliosis, mild neuronophagia and mild meningitis. In one case, mononuclear cells were seen around choroid villi alongwith severe congestion in meninges and brain. Oophoritis characterized by presence of mononuclear cells was found in two cases.

In three cases, lungs had lesions of pneumonia, characterized by presence of inflammatory cells and haemorrhages in perivascular area in one (Figure 32) case and giant cell granulomas in other two cases. In former case, colonies of gram positive coccobacilli were demonstrated in the lesions (Figure 33) and necrotic mucosa of intestine had colonies of gram positive bacilli with terminal spores and some gram positive cocci at its surface. Spleen of this case showed trapped gram positive coccal organisms by MacCallum Good Pasteur stain. In the latter two cases, granuloma consisted

Figure 28 : Gangrenous Dermatitis and Mycotic Pneumonia : Dry gangrene of the wing (A) caused by tightened wing band and presence of whitish pin point nodules in lung (B).

Figure 29 : Gangrenous Dermatitis : Extensive necrosis in epidermis and dermis alongwith haemorrhages in dermis. H&E x 80.

Figure 30 : Gangrenous Dermatitis : MacCallum Good Pasteur's stained skin section of Figure 29 showing colonies of Gram positive cocci. H&E x 325.

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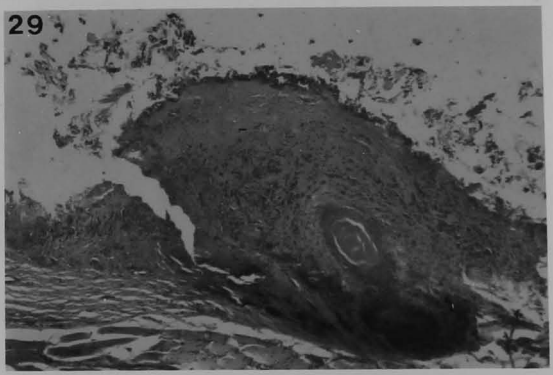

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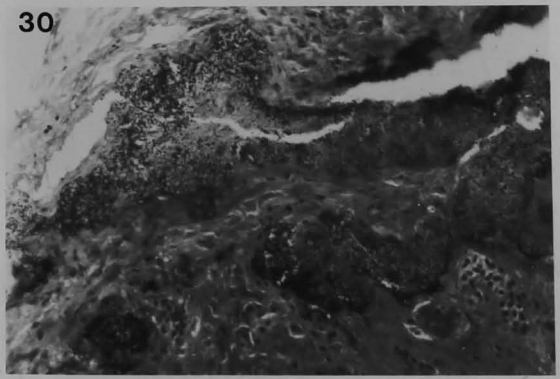
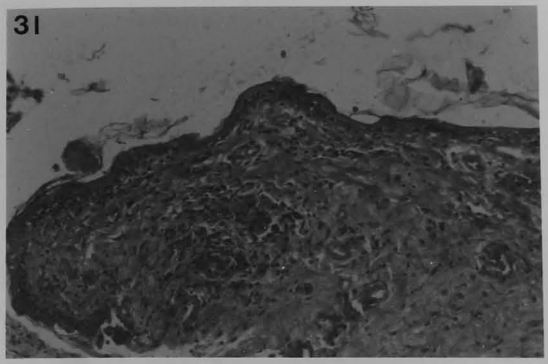


Figure 31 : Dermatitis : Chronic non-suppurative dermatitis with healing tissue. H&E x 160.

Figure 32 : Pneumonia : Haemorrhages, inflammatory cells and bacterial colonies in lung in a case of gangrenous dermatitis. H&E x 160.

Figure 33 : Pneumonia : MacCallum Good Pasteur's stained lung section depicted in Figure 32. Bacterial colonies noticed as of Gram positive cocci. x 325.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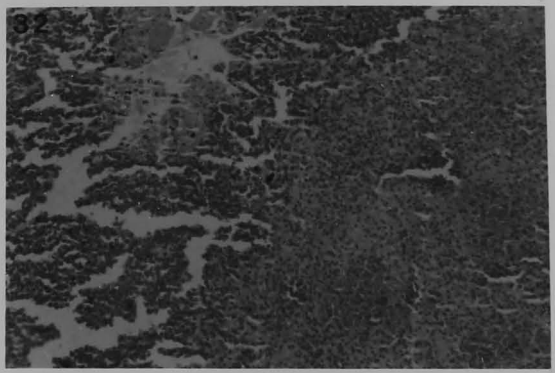
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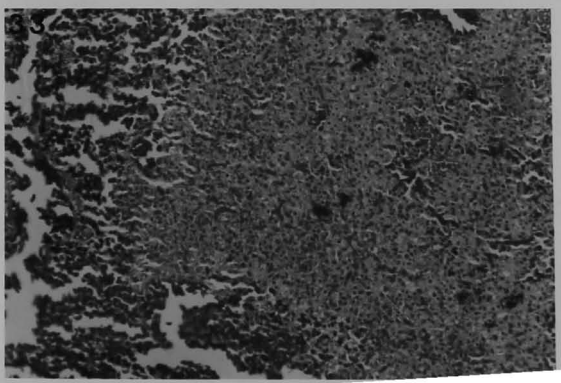
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of caseous necrotic centre surrounded by giant cells (Figure 34 & 35). Gram positive cocci were demonstrated in necrosed part of granuloma by MacCallum Good Pasteur's stain on one of these cases. Liver in one of these two cases had large area of infarction comprising of coagulative necrosis having reaction zone of inflammatory cells at periphery (Figure 36). Case having gross white granular appearance of liver showed large number of abscesses having caseous mass (Figure 37).

Subacute nephritis and hepatitis was present in one case. Few mononuclear cell aggregates were present in these organs.

Septicemia

Septicemic conditions were noticed in 4 cases. Gross lesions comprised of congestion and petechial haemorrhages on serous and mucous surfaces. No isolations were attempted from them. Lesions microscopically, comprised of fibrinoid necrosis in spleen characterized by presence of focal areas of acellular fibrinoid material and decreased cellularity of spleen. Livers showed severe degenerations and necrosis alongwith congestion and haemorrhage. Similar changes appeared in kidneys. Extensive haemorrhages and severe congestion in lungs, congestion and granular degeneration in heart, and epicardial haemorrhages in some cases were present. Mild serositis was found in proventriculus of one case. Brain had no lesions except gliosis in one case.

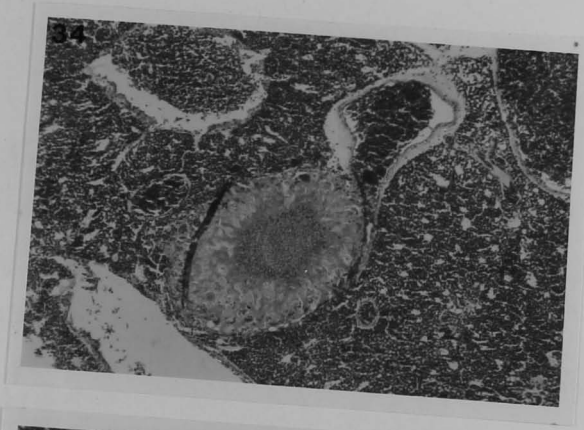
Infectious Coryza

It was observed in 6 cases. The diagnosis was based on presence of mucopurulent discharge from nostrils on pressing the nasal sinuses. Microscopically, nasal sinuses showed congestion and catarrhal exudation in

Figure 34 : Giant Cell Granuloma in Lung : Giant cell granuloma in lung noted in cases of gangrenous dermatitis characterized by central necrotic mass with karryohectic debris and giant cells at periphery. Extensive congestion and haemorrhages in lung parenchyma. H&E x 80.

Figure 35 : Giant Cell Granuloma in Lung : Higher magnification of Figure 34 H&E x 160.

Figure 36 : Infarction in Liver : Infarcted area of liver surrounded by zone of reaction in a case of gangrenous dermatitis. H&E x 80.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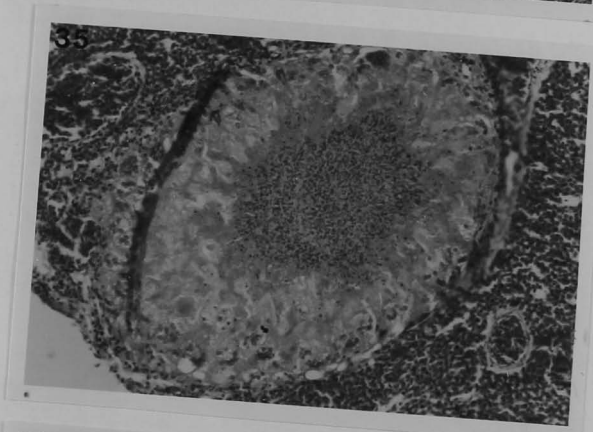
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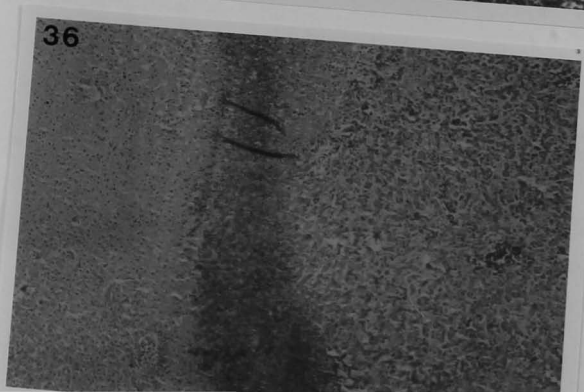
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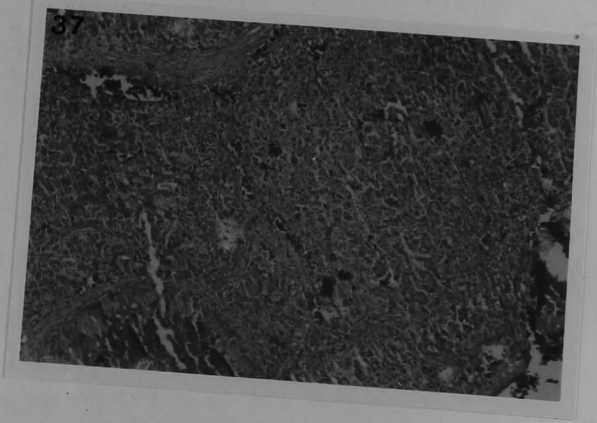
the mucosal lining (Figure 38). Mononuclear cell infilteration in the mucosa of sinuses was present in all cases. Hyperplasia of lining epithelium of sinuses was present in some cases. In tracheal mucosa mild mononuclear cell infilteration was frequently seen .Congestion and haemorrhages were seen in lungs. Liver showed congestion, haemorrhages and degenerations. Usual changes of congestion, haemorrhage, necrosis and degenerations were seen in kidneys. Brain had satellitosis, gliosis, neuronophagia in some of the cases. Rest of visceral organs were without any important lesions.

Chronic Respiratory Disease

A case of coryza diagnosed clinically had lesions indicative of chronic respiratory disease microscopically. Trachea had tubuloalveolar elongation of tracheal glands with lymphoid follicle formation in submucosa interspersed with mononuclear cells (Figure 39). Mucosa of nasal sinuses was replaced by cellular infilterations causing its thickening (Figure 40). At places infilteration was restricted to submucosa only. Intestine had a granuloma of about 1.5 cm size having caseous necrosis and degenerating nuclei in centre surrounded by epithelioid cells, macrophages, fibroblasts and fibrous tissues. Changes in kidney were of glomerular atrophy, extensive coagulative necrosis, desquamation of tubular epithelium, congestion with extensive haemorrhages. Liver had congestion, haemorrhage and vacuolar degeneration. Granular degeneration in myocardium, congestion and haemorrhages in lungs with edema in tunica adventition of pulmonary blood vessels were the lesion in these organs. No lesions were seen in other organs.

Figure 37 : Multiple Abscesses in Liver : Liver showing areas of necrotic mass encapsulated by pyogenic membrane in a case of gangrenous dermatitis. H&E x 80.

Figure 38 : Infectious Coryza : Nasal sinus having severe congestion, haemorrhages and mononuclear cell infilteration in mucosa. H&E x 80.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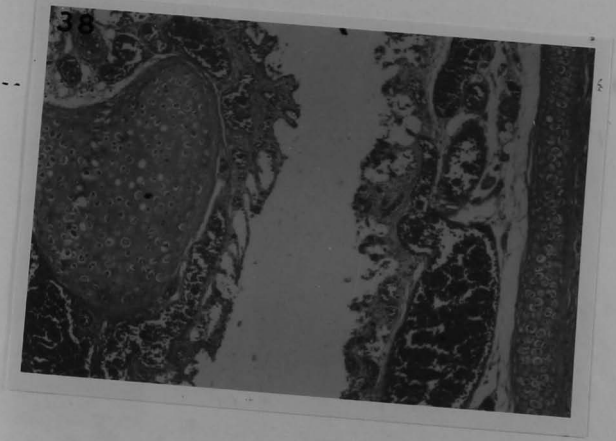
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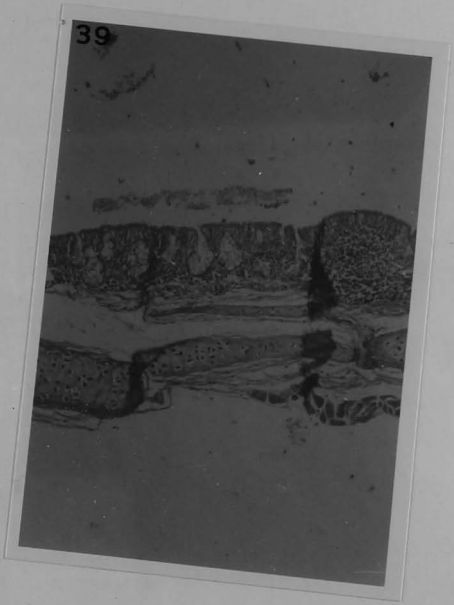
Figure 39 : Chronic Respiratory Disease : Lymphoid follicle in sub-mucosa alongwith tubuloalveolar elongation of tracheal glands and a few mononuclear cells. H&E x 80.

Figure 40 : Chronic Respiratory Disease : Thickened mucosa of nasal sinus by mononuclear cells infilteration. H&E x 80.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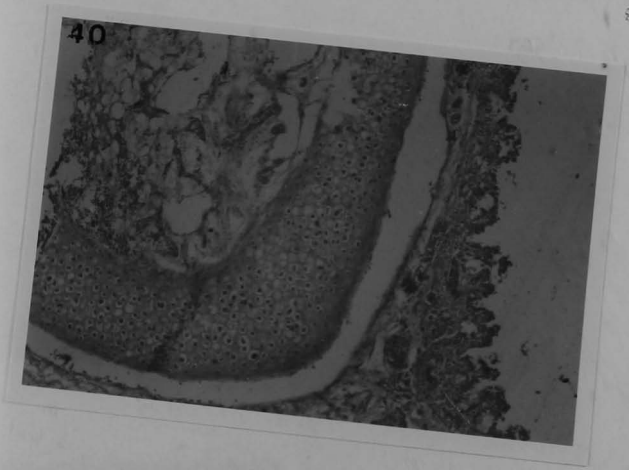
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Nephrosis/Nephritis Syndrome

Ninteen cases were categorized as of nephrosis/nephritis syndrome based on gross and histopathlogical findings. In nephrosis/nephritis syndrome, grossly the kidneys were enlarged and bulged out of renal fossae. Distended tubules were visible on the surface of the kidneys. Rest of the organs usually had no changes. Visceral gout was present in 3 out of 19 cases characterized by distended tubules with urates and chalky white deposits on other visceral surfaces (Figure 41). Microscopically, (Figure 42) varying degrees degenerative changes even upto necrosis alongwith congestion and haemorrhages were detected. The desquamation of tubular epithelium at places leading to narrowing of lumen of tubules and individualization of tubular cells were also seen. Glomeruli were usually contracted. Mild focal infilteration of inflammatory cells were seen in four cases. Severe necrosis accompanied by dystrophic calcification (confirmed by Van Kossa stain) in kidney was found in two cases. Changes in other organs consisted of congestion and haemorrhages in lungs, granular degeneration in liver, congestion in myocardium and brain. In visceral gout deposition of uric acid crystals on the surface of liver and heart were noticed. At places urate tophi were also seen which were surrounded by necrotic areas without any inflammatory response. Kidney showed dilated collecting tubules with inflammatory cells within the tubules. Multifocal tubular necrosis, cellular casts indicating interstitial nephritis was seen in all cases of urolithiasis.

Granulomatous Nephritis

A case of infection of kidney by budding yeast was encountered on histopathology. Grossly a small yellowish-white lobulated mass was present on the apical lobe of right kidney. Haemorrhages were present in

Figure 41 : Visceral Gout : White chalky depositions on pericardium and su.face of liver.

Figure 42 : Nephrosis Nephritis Syndrome : Kidney showing degeneration, desquamation, elongation, individualization of tubular epithelial cells alongwith disappearance of some of glomeruli and increased cellularity in others. H&E x 160.

Figure 41 : Visceral Gout : White chalky depositions on pericardium and su.face of liver.

Figure 42 : Nephrosis Nephritis Syndrome : Kidney showing degeneration, desquamation, elongation, individualization of tubular epithelial cells alongwith disappearance of some of glomeruli and increased cellularity in others. H&E x 160.

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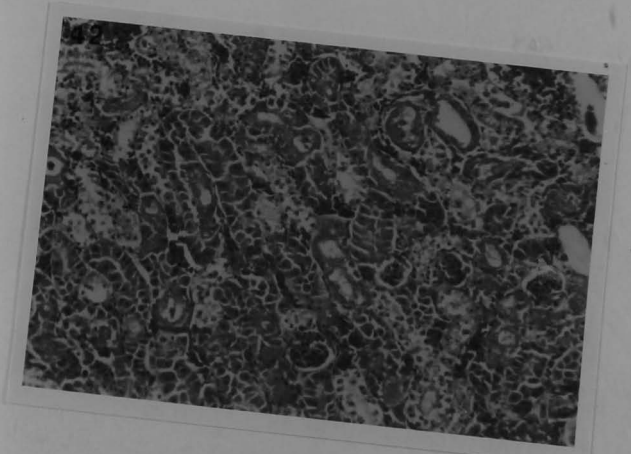

epicardium and endocardium. Ovary had yellowish opaque ova. Microscopically, the lesion present in kidney showed central empty zone filled with yeast like rounded budding cells, then caseous necrotic mass surrounded by macrophages, giant cells and fibrous tissue (Figure 43 & 44). Congestion and haemorrhage were also present. Rest tissue of kidney had varying degrees of degenerative changes alongwith severe congestion and haemorrhage. Liver had congestion, haemorrhage and vacuolar degenerations. Lungs revealed severe and extensive congestion and haemorrhage, fibrinous eosinophilic exudate with nuclear fragments in secondary bronchi. Varying degree of congestion and degenerative changes were seen in other organs.

Non-specific and Concurrent Conditions

Most of the cases (45) during study were found to be having non-specific lesions. Histopathologically and grossly the lesions were categorized as nephrosis and hepatosis (26 cases); nephrosis, hepatosis and oophoritis (9 cases); nephrosis, hepatosis with serositis of proventriculus or gizzard (6 cases) or epicarditis (2 cases) and 2 cases of acute hepatitis.

In cases (26 cases) of nephrosis and hepatosis, kidney lesions were similar to those of nephrosis/nephritis syndrome. Liver had degenerative and necrotic changes with varying degrees of congestion and haemorrhages. Vacuolar degenerations was more frequently seen. However, fatty changes were encounterd in some cases.

Nine cases had additional lesions of oophoritis besides nephrosis and hepatosis. In these cases severe congestion and haemorrhages in ovary and mild to moderate mononuclear cells infilterations were seen.

Those with additional lesions of serositis of proventriculus/gizzard (6 cases) or epicardium showed congestion in the serosa of the organs

alongwith fibrinous exudate and mild to moderate mononuclear cell infilterations.

Two cases of acute suppurative hepatitis had focal areas of necrosis in liver with heterophils infilteration alongwith congestions and haemorrhages.

In all of the above cases, lungs had congestion and haemorrhages in all cases. In most cases tunica adventitia of pulmonary vessels had edema causing its loosening due to oozing of plasma. Brain had lesions in five cases which comprised of congestion, gliosis, satellitosis and neuronophagia. Spleen had haemorrhages in two cases with mild reticular cell hyperplasia in one case only.

Traumatic Ventriculitis

In this case, a sharp pointed object had caused perforation through the gizzard. Intestinal contents where blackish. Congestion in brains and haemorrhages in chest muscles were present. Microscopically, the gizzard had congestion, haemorrhages in musculature and serosal surface with a presence of inflammatory cells. Skeletal muscles showed hyaline degeneration and presence of bacterial colonies. Lesions in other organs consisted of congestion and granular degeneration in liver, congestion, haemorrhage, degeneration and necrosis in kidneys, congestion and gliosis in brain.

Vent Gleet/Cloacitis

It was noticed in one case. Whitish thick deposit present on the mucosal surface of vent and grossly visible fungus growth were observed in this case. Liver had mottled appearance. *Aspergillus fumigatus* was isolated from the vent. Microscopically, the affected tissue was necrosed and large

alongwith fibrinous exudate and mild to moderate mononuclear cell infilterations.

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number of mononuclear cells and giant cells demarcated it from the normal underneath tissue. Grocott's stain demonstrated heavy growth of septate hyphae and presence of spores (Figure 45). Other organs were without lesions except degenerative and necrotic changes in liver and kidney.

Haemorrhagic Syndrome

Based on gross and histopathological findings three cases were grouped as of "haemorrhagic syndrome". Gross post-mortem lesions consisted of irregular patches of haemorrhages in breast and thigh muscles. Haemorrhages were present grossly in kidneys in one case and lungs in two cases. Emaciation, hypoplastic anaemia, dark blackened mucosa of gizzard were observed in another case. Microscopically, skeletal muscles had haemorrhages and fibrinous exudate in two cases, heterophilis, mononuclear cells infilteration, congestion, haemorrhage and fibrosis in the third case. Liver showed congestion, haemorrhage, degeneration and focal coagulative necrosis. Kidneys had severe haemorrhages in one case alongwith necrosis, general congestion, haemorrhage and necrosis in other two cases. Severe congestion in intestinal and gizzard serosa alongwith mononuclear and fibrinous exudate was present in two cases. Lungs had severe and extensive congestion and haemorrhages. Thickened tunica intima causing narrowing of lumen, duplication and fragmentation of internal elastic membrane of pulmonary blood vessels were observed in emaciated case. Myocardium in same case had haemorrhages, narrowed lumen of coronary artery branches with thickening of tunica media.

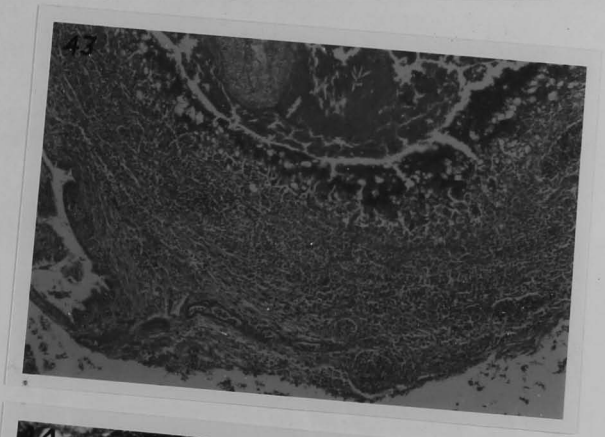
Suppurative Myositis

This condition was seen in 5 birds. It was usually found in young birds and sometimes adults also because of fractures or injuries in the legs by entangling in the wired floor of the cages. Grossly, lesions consisted of

Figure 43 : Granulomatous Nephritis : Central necrotic area surrounded by zone of macrophages and giant cells encapsulated by fibrous tissue interspersed with large number of mononuclear cells. H&E x 80.

Figure 44 : Granulomatous Nephritis : Grocott's stained kidney section depicted in Figure 43 showing rounded budding yeast cells in central empty zone. x 160.

Figure 45 : Fungal Cloacitis : Vent having heavy growth of hyphae and spores at the mucosal surface. Grocott's stain x 160.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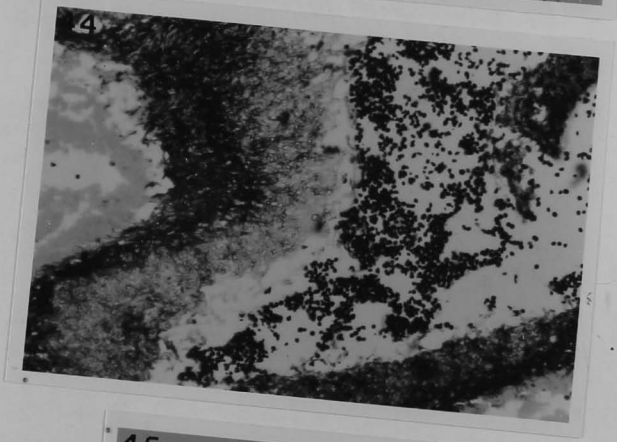
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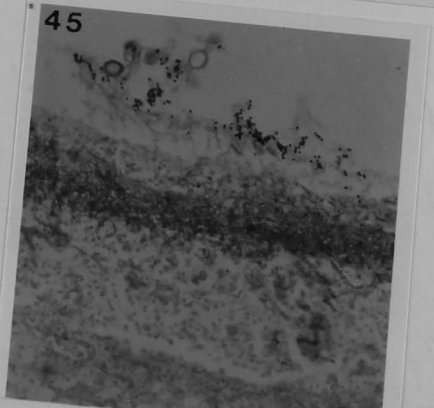
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inflammation of involved part, alongwith haemorrhage in adjoining muscular area. Rest of viscera usually appeared normal. Microscopic lesions included haemorrhages, edema, heterophilic and mononuclear cell infilterations in the muscles of affected part (Figure 46). No lesions were found in other organs except mild meningitis. Lungs, heart and liver had congestion alongwith haemorrhages in liver.

Cystic Right Oviduct

Though right ovary and oviduct in adult birds are immature and non-functional, cyst of about one inch size containing clear watery fluid were found in three cases during the study.

Bony-Metoplasia in Lungs

Metoplasia of lung tissue into small bony structure (Figure 47) was observed in one case having no specific gross lesions. Congestion, haemorrhage, degeneration were the changes in liver and kidneys. Rest organs did not have any lesions.

Lesions Suggestive of Vitamin E/Selenium deficiency

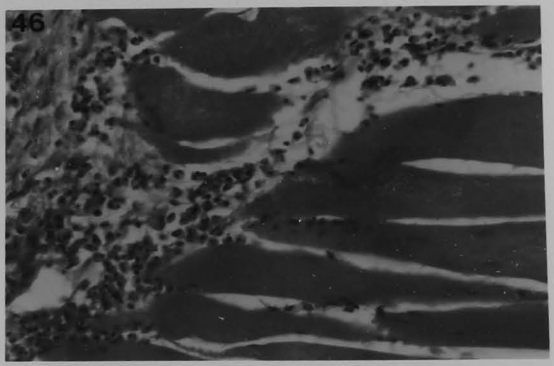
Two cases, one showing gross and microscopic lesions of muscular dystrophy and other with haemorrhagic lesions in the cerebellum and muscles suggested deficiency of Vitamin E or Selenium.

In first case, the breast muscles had light coloured streaks which distinguished them from normal pinkish muscle bundles. Microscopically hyaline degeneration of skeletal muscles, few erthrocytes and small number of infilterating cells were present between the seperated muscle fibres. In heart mononuclear cell infilteration with congestion and haemorrhages in epicardium were present. Lungs had congestion and haemorrhages and kidneys with congestion, haemorrhage, degeneration and necrosis.

Figure 46 : Suppurative Myositis : Infilteration of heterophils and haemorrhages between degenerating muscle fibres. H&E x 325.

Figure 47 : Bony Metaplasia : Metaplasia of lung tissue into bony structure. Also present is severe congestion and haemorrhage in lung. H&E x 160.

Figure 48 : Encephalomalacia : Extensive haemorrhages in cerebellum suggestive of Vitamin E deficiency. H&E x 80.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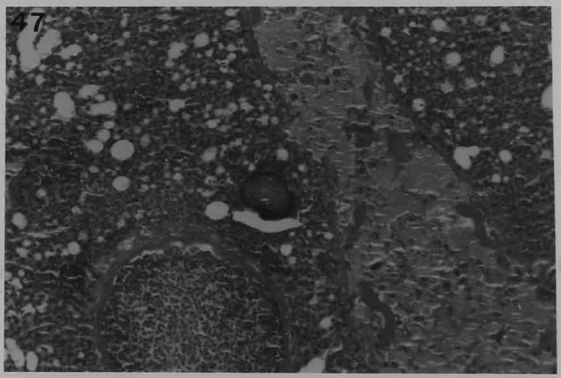
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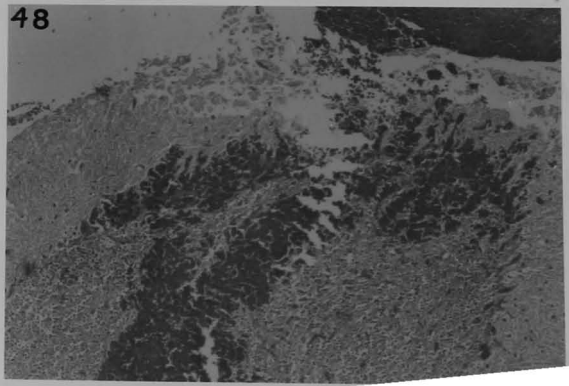
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Gross lesions in second case were : congestion in cerebrum and cerebellum, pale breast muscles, epicardial haemorrhages, congested lungs. Microscopically, congestion and severe haemorrhages in cerebellum (Figure 48), congestion in meninges of cerebrum, granular degeneration in skeletal muscles, severe congestion and haemorrhages in lungs, congestion haemorrhage and vacuolar degeneration in liver, congestion, haemorrhage and degenerations in kidney, mild mononuclear cell infilteration in glands of proventriculus were present.

Experimental Salmonellosis

Salmonella gallinarum isolated from outbreak at Government Poultry Farm. Malerkotla was used for this study. It was got serotyped from CRI, Kasauli.

Determination of LD_{50} : Ten fold serial dilutions of S. gallinarum were made in sterilized normal saline and groups of 4 quail chicks (7 day old) were each inoculated with 0.1 ml of dilutions from 10^{-2} to 10^{-16} intraperitoneally. Deaths and survival were noted upto 10 days post inoculation (DPI). Results are summarized in Table 15.

All the birds upto 10^{-6} dilution died. In 10^{-7} dilution two died and two survived. From 10^{-8} dilution 1 died and three survived. With subsequent dilutions no mortality was found except a single bird that died from 10^{-13th} dilution. This bird was weak from starting and succumbed to higher dilution. LD_{50} was worked out according to Reed and Muench (1938) formula and was found to be $10^{-7.44}$. Cultures were taken from liver and spleen of all dead birds. *S. gallinarum* was isolated from birds inoculated with dilutions upto 10^{-8}. Thereafter no isolation could be made.

Experimental trail : Twenty five birds were inoculated with LD_{50} S. gallinarum and twenty five were kept as control. Clinical signs and gross

Table 15: Determination of LD_{50} of *S. gallinarum*

Dose (ml)	Dilution of broth culture (18 hrs)	Mortality Ratio	Number of birds		Accumulated Values			% Age Mortality (D/D+S) x 100
			Died	Survived	Died (D)	Survived (S)	Mortality Ratio	
0.1	10^{-2}	4 / 4	4	0	24	0	24 / 24	100
0.1	10^{-3}	4 / 4	4	0	20	0	20 / 20	100
0.1	10^{-4}	4 / 4	4	0	16	0	16 / 16	100
0.1	10^{-5}	4 / 4	4	0	12	0	12 / 12	100
0.1	10^{-6}	4 / 4	4	0	8	0	8 / 8	100
0.1	10^{-7}	2 / 4	2	2	4	2	4 / 6	66.66
0.1	10^{-8}	1 / 4	1	3	2	5	2 / 7	28.57
0.1	10^{-9}	0 / 4	0	4	1	9	1 / 10	10.40
0.1	10^{-10}	0 / 4	0	4	1	13	1 / 14	7.14
0.1	10^{-11}	0 / 4	0	4	1	17	1 / 18	5.55
0.1	10^{-12}	0 / 4	0	4	1	21	1 / 22	4.54
0.1	10^{-13}	1 / 4	1	3	1	24	1 / 25	4.00
0.1	10^{-14}	0 / 4	0	4	0	28	0 / 28	0.00
0.1	10^{-15}	0 / 4	0	4	0	32	0 / 32	0.00
0.1	10^{-16}	0 / 4	0	4	0	36	0 / 36	0.00

lesions noted daily. Gross lesions were first recorded at 6 DPI. These increased in severity upto 9 DPI and disappeared after 11 DPI. These comprised of congestion in almost all internal organs, haemorrhages on epicardium, liver, kidneys, spleen, lungs, mottling and bronze discolouration of liver, mottling of liver, pericarditis etc. (Figures 49, 50 & 51). Sequencewise gross lesions are given in Table 16.

Histopathology

Liver : Congestion of hepatic vessels and sinusoids with RBC's was prominent alongwith extensive haemorrhages. Degenerative changes in hepatocytes were of granular degeneration upto 8 DPI followed by mixed type of changes i.e. granular degeneration, vacuolar degeneration and fatty changes upto 9 DPI (Figure 52). Extensive vacuolar degenerations occurred later on after 9 DPI. Focal areas of necrosis and sometimes diffuse necrotic areas were seen. From 11 DPI onwards focal areas of mononuclear cell infilteration forming microgranulomae were seen till the end of experiment (Figure 53).

Kidneys : Engorged blood vessels of parenchyma of kidneys and haemorrhages were seen. Degenerative changes were seen in tubular epithelium. changes were of all type ranging from granular degeneration to extensive necrosis. No infilteration of mononuclear cells was found.

Heart : Congestion of blood vessels of myocardium, endocardium and epicardium was present., Haemorrhages in myocardium were also noticed. Myocardium showed extensive granular degeneration, vacuolar degeneration also appeared in some cases especially on 8 DPI. Pericarditis was seen from 7 DPI characterized by mononuclear cells in epicardium and fibrinous

Table 16: Table showing clinical signs and gross lesions in experiment

DPI	Clinical Signs	No. of birds died / killed	Gross Lesions	Isolations	
				Liver	Spleen
6 DPI	Ruffled feathers. Huddling together.	1	Liver : Enlarged, mottled with congestion. Spleen : Enlarged and haemorrhagic treaks. Lungs : Slightly brownish than normal. Heart : Haemorrhages in epicardium. Kidneys : Congested and harmorrhagic.	+	+
7 DPI	Ruffled feathers. Huddling together. Backward movement in one. Prostratrations, paddling and death.	3	Liver : Enlarged and mottled Spleen : Enlarged, mottled, dark in colour with necrotic foci, congestion and haemorrhages. Lungs : Dark brown. Kidneys : Congested and haemorrhages. Breast Muscles : Haemorrhages.	+	+
8 DPI	Prostration, paddling, torticollis	5	Liver : Enlarged and mottled Spleen : Enlarged, congested. Lungs : Dark brown. Kidneys : Congested and haemorrhage. Breast Muscles : Haemorrhages	+	+
9 DPI	No major sign. Ruffled feathers and huddling.	3	Liver : Bronze discolouration in two, mottled and perihepatitis in one. Spleen : Enlarged and mottled. Lungs : Dark brownish Heart : Pericarditis, Haemorrhage in pericardium. Brain : Congestion.	+	+
10 DPI	No signs.	3 sacrificed	Liver : Mottled, bronze. Spleen : Slight enlargement, mottled. Heart : Haemorrhage in pericardium. Brain : Congestion	+	+
11 DPI	No signs.	2 sacrificed	Liver : Pale. Spleen : Normal. Heart : Haemorrhage in pericardium. Brain : Congestion	+	+
12 DPI	No signs.	2 sacrificed	No major gross lesion.	-	-
13 DPI	No signs.	2 sacrificed	No gross lesion.	-	-
14 DPI	No signs.	2 sacrificed	No gross lesion.	-	-
15 DPI	No signs.	2 sacrificed	No gross lesion.	-	-

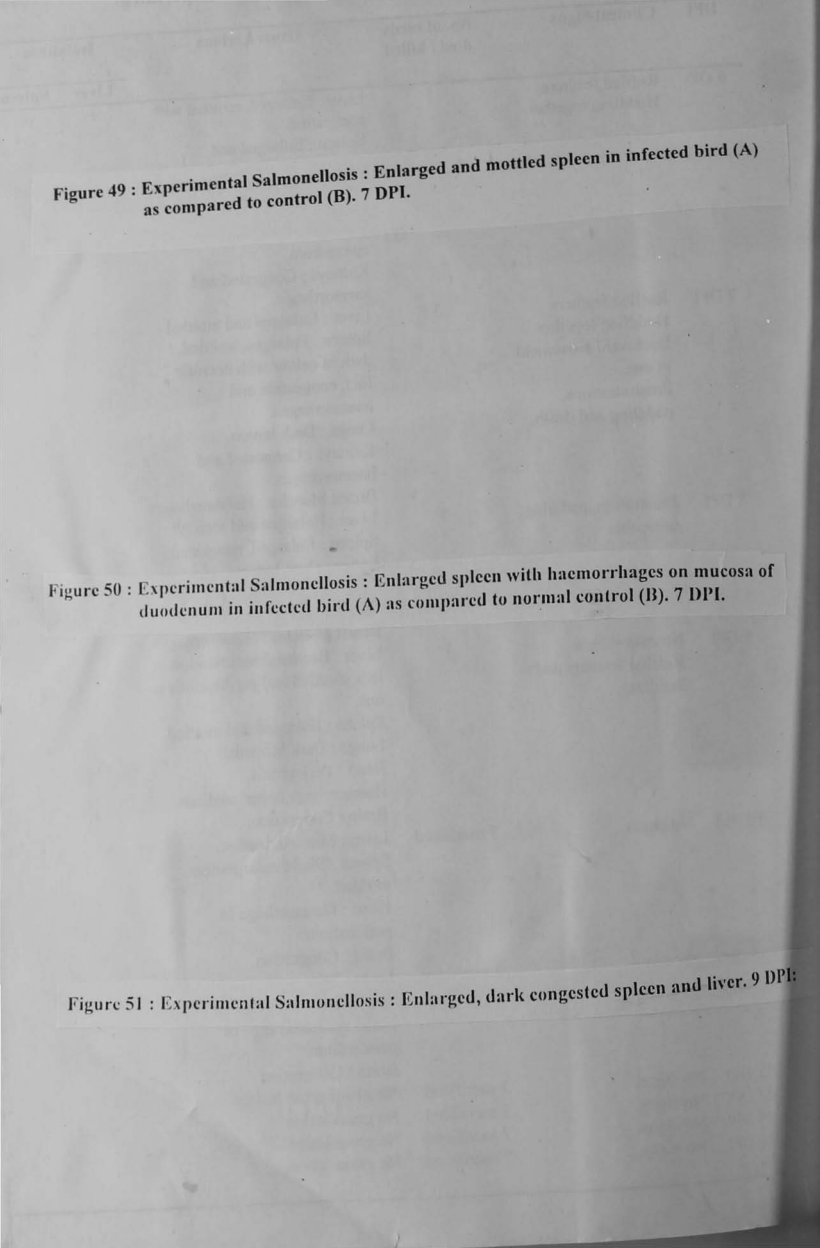
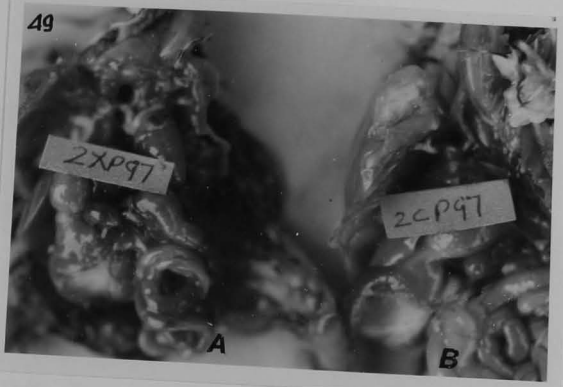


Figure 49 : Experimental Salmonellosis : Enlarged and mottled spleen in infected bird (A) as compared to control (B). 7 DPI.

Figure 50 : Experimental Salmonellosis : Enlarged spleen with haemorrhages on mucosa of duodenum in infected bird (A) as compared to normal control (B). 7 DPI.

Figure 51 : Experimental Salmonellosis : Enlarged, dark congested spleen and liver. 9 DPI.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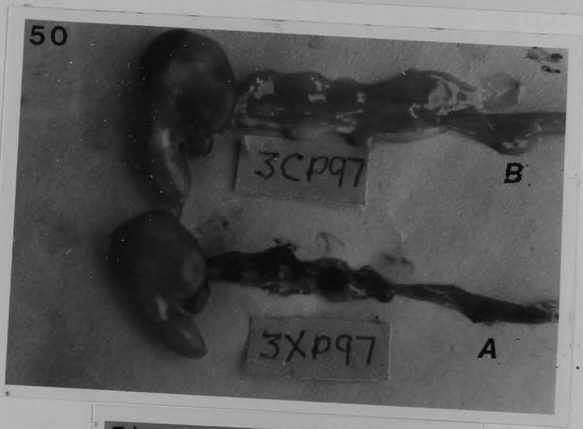
2XP97
2CP97
A
B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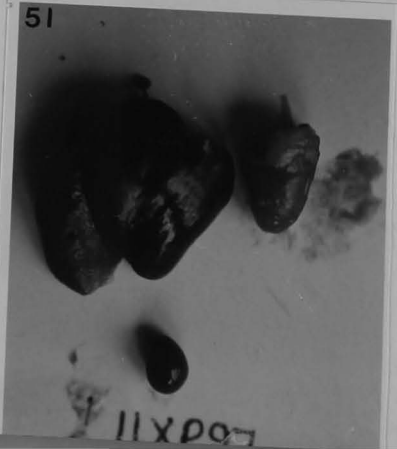
3CP97
B
3XP97
A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11XP97

exudate upto 11 DPI. Focal infilteration of mononuclear cells and heterophilis in myocardium occurred in some cases.

Lungs : Changes in lungs were of severe congestion and haemorrhages upto 10 DPI. In sacrificed birds no changes were noticed in lung parenchyma.

Spleen : The changes in spleen consisted of decreased cellularity of parenchyma and presence of acellular eosinophilic fibrinous matrix (Figure 54). Mild reticular cell hyperplasia was observed in some cases. No changes appeared in sacrificed birds.

Intestine : No significant lesions appeared in intestine except for congestion of serosal blood vessels and mild serositis characterized by presence of mild mononuclear cell infilteration in serosal layer. Degeneration was seen in external muscular layer of intestine in one case alongwith mononuclear cells in serosa. In one case, granulomatous lesion characterized by fibrosis presence of mononuclear cells, fibroblasts and giant cells surrounding necrosed area was present (13 DPI) (Figure 55).

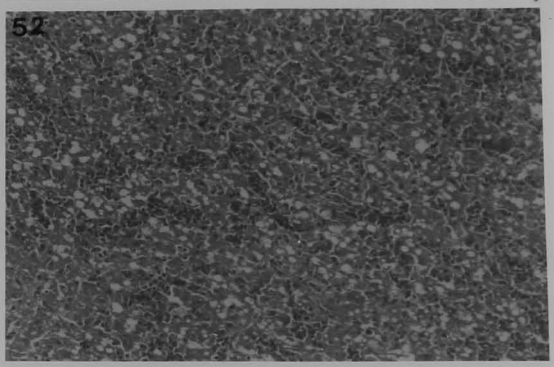
Proventriculus : No characteristic changes appeared in proventriculus except for serosal congestion and mild serositis in few cases.

Gizzard : Serositis was well marked in gizzard and was present in all birds that died during the experiment. No changes were observed in sacrificed birds except mild serositis in one case. The serositis was characterized by congestion of serosal blood vessels and mild to moderate infilteration of mononuclear cells in the serosa. Focal infilteration of mononuclear cells in muscular layer of gizzard was also observed (Figure 56).

Figure 52 : Experimental Salmonellosis : Congestion of hepatic blood vessels and sinusoids alongwith mixed type of changes (granular, vacuolar and fatty changes) observed on 9 DPI. H&E x 160.

Figure 53 : Experimental Salmonellosis : Microgranuloma alongwith haemorrhages, degenerative and necrotic changes observed on 12 DPI. H&E x 325.

Figure 54 : Experimental Salmonellosis : Acellular eosinophilic areas and decreased cellularity of spleen. H&E x 160.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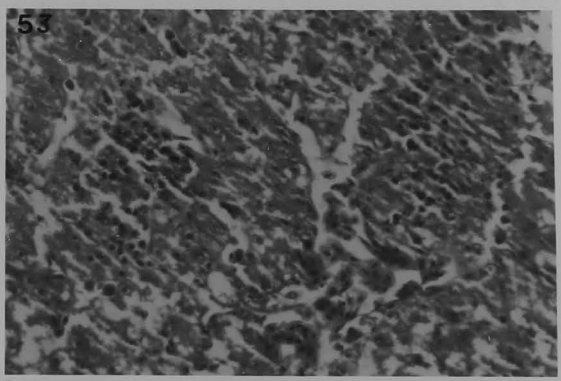
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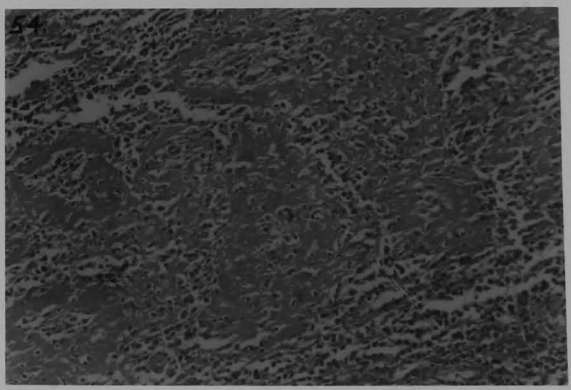
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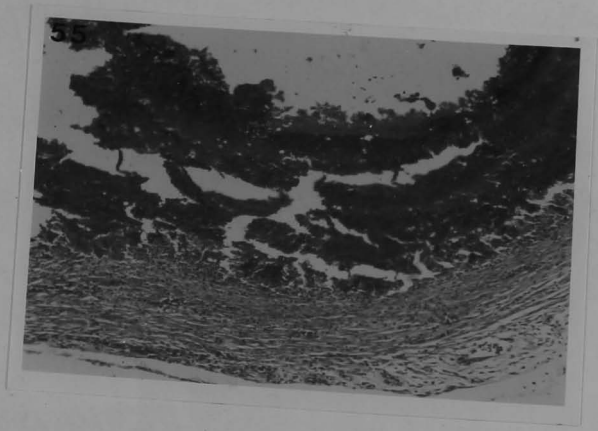
Figure 55 : Experimental Salmonellosis : Large necrotic area replacing mucosa of intestine alongwith fibroblastic proliferation and mononuclear cell infilteration in the wall of intestine. H&E x 80.

Figure 56 : Experimental Salmonellosis : Focal infilteration of mononuclear cells observed in muscular layer of gizzard. H&E x 160.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Brain : No important changes were seen in brain. Congestion and haemorrhages in cerebrum and cerebellum were noticed on 7 DPI and 8 DPI alongwith mild meningitis. Later on no changes were present.

Bursa of Fabricius & Trachea : No changes noticed.

Chapter V

DISCUSSION

Quail farming is becoming popular in Punjab. With commercialisation, it faces challenges from various diseases due to the intensive rearing. So in order to plan systematic control programme, it was necessary to study the prevalence and etiopathology of various conditions affecting the Japanese quail. To study the prevalence of various conditions and mortality pattern in Japanese quail, the data of 6 years available in the department (1991 to 1996) was analysed.

The mortality pattern shows that though mortality rate in 0-1 week age group is higher followed by 1-6 weeks age group and low in adults (over 6 weeks age), but this difference was found to be insignificant statistically. This insignificant difference may be due to the fact that mortality was recorded for one week duration, 5 weeks duration and duration variable upto 14 weeks, respectively for the age groups of 0-1 week, 1-6 weeks and 6 weeks onwards as quail farm was maintaining the records under these age groups. Srinivasan *et al.* (1980) during two year study observed the difference in the mortality rates in two different age groups (0-6 weeks and above 6 weeks) to be statistically insignificant. Suneja *et al.* (1983) found that mortality was maximum during first week of life, followed by those in 1-6 weeks and then above 6 weeks age, but his conclusions were based on observations of single year. Similar observations were also recorded during the year 1991 of our study. Gangadharan *et al.*(1989) documented higher mortality in age group of 0-3

weeks, but their observations were based on post-mortem records showing details in particular age group out of total birds necropsied. Ravindran *et al.* (1994), recorded high brooding losses in age groups of 0-1 and 2-6 weeks.

In general, for the whole population, high mortality in the descending order was seen in the months of July, May, June and January. Mortality graph for different months shows increased mortality in hot summers and cold dry winter months of the year in young (0-1 week age) and grower stock (1-6 weeks age). Pre-summer and rainy season had less mortality. Seasonal variations do not have marked affect on mortality pattern of adult birds (above 6 weeks age group), comparatively high mortality was observed from April to September. Similar observations were recorded by Suneja *et al.* (1983). Sharma and Kaushik (1986) also observed high mortality in winter and lowest during rainy season. Similarly Ganghadharn *et al.* (1989) observed lowest mortality in rainy season and more in dry season in both young and adult quails, the results indicate that special measures are necessary to keep the temperature in quail house at appropriate levels during summer and winter months.

The important disease condition affecting the Japanese quail in descending order of frequency were hepatosis/hepatitis, yolk sac infection, oophoritis, coryza, gangrenous dermatitis, nephrosis/nephritis syndrome, enteritis, mycotic pneumonia, lymphoproliferative diseases (mainly MD), septicemia etc. Yolk sac infection was important disorder in 0-1 week age group followed by hepatosis/hepatitis, pneumonia and nephrosis/nephritis conditions. Srinivasan *et al.* (1980) attributed mortality rate in 0-6 weeks age group primarily due to pneumonia and omphalitis. Suneja *et al.* (1983) and Sharma and Kaushik (1986) also observed omphalitis and pneumonia as major causes of mortality in young stock. Gangadharan *et al.* (1989) had

recorded omphalitis, hepatic disorders, pulmonary congestion and edema as frequent disorders.

In 1-6 weeks age group, hepatic disorders were most frequently encountered conditions, followed by mycotic pneumonia, nephrosis/nephritis syndrome, coryza, enteritis, gangrenous dermatitis etc. In adult birds (above 6 weeks age), disease condition recorded in descending order were oophoritis, hepatic disorders, dermatitis, coryza, enteritis, Marek's disease, septicemia, egg bound/salpingitis, heat stroke, nephrosis/nephritis syndrome etc. Suneja *et al.* (1983) had not recorded any incidence of hepatic infections, gangrenous dermatitis, mycotic pneumonia, coryza, Marek's disease, nephrosis/nephritis syndrome, air - sacculitis/CRD, in quails of 1 week onwards during his study on one year. On the other hand, Gangadharan *et al.* (1989) also recorded hepatosis, enteritis, pneumonia, Marek's disease as major conditions in birds above 3 weeks age group. They had recorded occurrence of coccidiosis and Ranikhet disease. However, these authors had not recorded conditions like mycotic pneumonia, nephrosis/nephritis syndrome, gangrenous dermatitis, heat stroke, air - sacculitis/CRD, Inclusion body hepatitis, lymphoid leukosis which have been encountered during our study. However, we have not come across any outbreaks of coccidiosis and Ranikhet disease. Patro *et al.* (1992) recorded high incidence of lymphoid leukosis, enteritis and cannibalism. They had also encountered neoplasms (haemangioendothelioma, leiomyoma), ascariasis, ulcerative enteritis out of a total of 90 cases. In the present study, the presence of conditions like gangrenous dermatitis and absence of incidence of coccidiosis, intestinal worms, ulcerative enteritis may be due to housing of quails in cages.

High incidence of mycotic pneumonia was observed in June and July months of 1992, only sporadic incidences were recorded during the rest of the period. In the same year, maximum cases of hapatosis and hepatitis appeared. So high contamination of environment/feed/droppings by fungus along with lowered resistance might have resulted in these outbreaks.

Maximum incidence of various conditions were observed during the years 1992 and 1993 of our study. This might be due to more number of birds kept during these years. An outbreak of MD was recorded in 1994.

Coryza, air sacculitis/CRD and pneumonia had maximum occurrence in September, October and November respectively. This is in accordance to more susceptibility of individuals to respiratory infections during the change of seasons and cold months. Yolk sac infection was most frequent in December, April and January indicating hatchery hygienic conditions. Heat stroke caused maximum mortality in June and nephrosis/nephritis syndrome was more frequent in July. Both conditions are attributable to high temperatures in these months. Hepatosis/hepatitis condition though present throughout the year was more frequent in January.

Detailed gross and histopathological study of 166 cases was conducted to know the pathology of various conditions in Japanese quails.

Gross and histopathological study revealed lesions of Marek's disease in 21 cases. The gross lesions primarily comprised enlarged intestines patricularly the duodenum. Diffuse or focal whitish thickening of the wall of small intestine particularly duodenum was the most frequently encountered lesion. Liver and spleen had white nodular masses. Thickening of brachial or sciatic nerves or tumour of ovaries could not be seen. Microscopically, pleomorphic lymphocytic infilterations were seen in different organs. The maximum infilterations were seen in the wall of duodenum, resulting in

replacement of normal tissue of intestinal wall. The frequency and extent of involvement of various organs in descending order were recorded as intestine, liver, proventriculus, spleen, ovary, gizzard, kidney, lungs. Brain and nerves had lesions in only one and two cases, respectively out of 21 cases. No infilterations were seen in myocardium. Other workers had reported similar lesions in experimental and natural cases. (Dutton *et al.* (1973), Fujimoto *et al.* (1975), Mikami *et al.* (1975), Khare *et al.* (1975), Pradhan *et al.* (1985) and Nair *et al.* (1986)). But most of them found liver and spleen as most frequently affected organs rather than small intestine. In quails we found that small intestine mainly duodenum, liver, proventriculus and spleen were the target organs. Presence of concurrent mycotic infections in some cases in adults may have resulted due to immunosuppression caused by Marek's disease.

Single case of lymphoid leukosis had uniform sized lymphoblasts in liver, ovary, proventriculus, gizzard and kidney. Wight (1963) and Patro *et al.*, (1992) described similar lesions affecting liver, spleen, intestine and proventriculus and occasional involvement of pancreas, kidney and myocardium.

Mycotic pneumonia was observed in 14 cases. In mycotic pneumonia *Aspergillus fumigatus*, *A. flavus* and *A ochraceus* were the fungi isolated. Lesions involved the lungs and extension to thoracic and abdominal air sacs in severe cases. *A. fumigatus* caused more severe lesions having large central necrotic areas with fungal hyphae and macrophages, giant cells and fibroblasts at periphery. On the other hand *A. flavus* had caused small sized granulomas with small necrotic masses but large areas of cellular infiltrations. Experimental infection by *A. fumigatus* causing well developed granulomas in lungs, air sacs and trachea had been reported by Chaudhary

et al. (1988). Similarly Pandita *et al.* (1995) reported that experimental infection by *A. flavus* resulted in less developed granulomas in lungs but with no extension to air sacs and trachea. Granulomas formed were similar to those reported in our study of natural cases i.e. less necrosis and more cellularity of the lesions.

The occurrence of infection of kidney by any budding yeast in Japanese quail could not be traced in the available literature. The probable route for infection by yeast may be through ureters as no systemic involvement of any other organs suggesting oral or haematological routes was there. The lesions were of granulomatous type with budding yeast cells.

Gross changes observed in typhoid outbreaks (*S. gallinarum* infection) in adult quails were necrotic foci and bronze discolourisation of liver, mottled spleens, cyanosed and congested lungs, oophoritis with long stalked ova having oily contents. Microscopically, typhoid granulomas and focal coagulative necrosis in liver, congestion, haemorrhage, and coagulative necrosis in renal tubules and oophoritis were similar to those found in chickens. The enteritis in quails was not pronounced and fibrinoid necrosis of spleen which was observed in quails is not the prominent feature in fowl typhoid in chickens. Natural outbreaks of *S. gallinarum* infection in 1-3 day old quail chicks reported by Sarma *et al.* (1988) had caused perihepatitis and necrotic foci on liver, congestion of lungs, slight enlargement of spleen, hydropericardium and acute haemorrhagic enteritis.

Frequent isolation of E. coli from various conditions i.e. oophoritis, salpingitis, prolapse of oviduct, coryza, septicaemia and polyserositis in quails revealed it to be of etiologic significance for such conditions. Lesions typical of avian colibacillosis recorded in two cases, comprised of thickened liver capsule with fibrinous exudate and mononuclear cells, fibrinous

pericarditis and peritonitis. Similar lesions have been described by Ito *et al.* (1990) in 21 to 330 days old quails.

Gangrenous dermatitis was observed in 17 cases. The gangrenous dermatitis was most frequently encountered on the head region in grower and adult quails. Quails take off from the ground with great thrust under the stress of fear and strike directly into the roof of cages, causing injury under the force of impact. The initial injury results in necrosis followed by entry of common inhabitant microbes viz. staphylococci. Partial anaerobiosis produced by necrosis becomes more exacting by the use of oxygen by aerobic bacteria. Thus, the entry and proliferation of clostridial organisms is promoted resulting in gangrene. Associated lesions in internal organs comprised of pneumonia in three cases, hepatic infarction in one case, degenerative lesions, congestion and haemorrhages in liver in all cases, extensive reticular cell hyperplasia in two cases, oophoritis in two cases, congestion, haemorrhages, degenerative and necrotic changes in kidneys in all cases. These lesions appear to be correlated with toxaemia produced by clostridial infection and superimposed by other bacterial infections.

Septicemic lesions comprising of severe congestion in all organs along with petechiae on serosal and mucosal surfaces were seen in 4 cases. Microscopically, the fibrinoid splenic necrosis, congestion and haemorrhages in various organs along with degeneration in parenchymatous organs was noticed. Glisson *et al.* (1989) noted such lesions of septicemic nature in fowl cholera in Japanese quails. In one case fibrinoid splenic necrosis, typhoid granulomas in the liver indicative of septicemic salmonellosis were seen during present study.

Infectious coryza was characterized by mucopurulent exudate from nasal chambers. Histologically, mononuclear infilterations in mucosa of nasal

chambers and sometimes along with hyperplasia of lining epithelium and frequent mononuclear cells infilterations in trachea were seen. Reece *et al.* (1981) had reported such lesions of mucoid sinusitis, nasal exudation and eye involvement with *Haemophillus paragallinarum* infection. No natural case of infectious coryza by *H. gallinarum* in Japanese quails appeared to have been recorded on the available literature. Cundy (1965) investigated susceptibility of Japanese quail to experimental infection with *H. gallinarum* and they developed sinusitis and conjunctivitis.

A case of chronic respiratory disease was diagnosed based on histopathological lesions which were characterized by lymphoid follicles in submucosa of trachea along with tubo-alveolar hyperplasia of tracheal glands. The sinuses had thickening of mucosa by mononuclear-cell infilteration. Concurrent granuloma in intestine was also present. Tiong (1978) and Nascimento and Nascimento (1986) observed caseous sinusitis and cloudy to caseous air-sacculitis as main lesions in Japanese quails and isolated *Mycoplasma gallisepticum* from them, but histopathological study was not done by them.

Nephrosis/Nephritis syndrome was important condition in different age groups of Japanese quails. Kidneys had varying degrees of degeneration, desquamation, individualisation and necrosis of tubular epithelium. Glomeruli were usually contracted. Oophoritis characterized by congestion, haemorrhage and mononuclear cells infilteration was observed in 9 cases. Proventriculitis, ventriculitis or epicarditis and granular degeneration in the liver were the accompnying lesions. Lungs in all cases had congestion and haemorrhage. The cases with varying degree of degenerative lesions without infiltration of inflammatory cells could be categorized as of nephrosis. Four cases having infilterations by mononuclear cells have been classified under nephritis. Two

cases had dystrophic calcification in kidney. Renal gout and visceral gout diagnosed in three cases indicated end stage of renal disease. Lesions of Nephrosis/Nephritis in domestic fowl have been reported to be caused by a variety of agents/factors viz. viruses (Infectious brochitis virus, adenoviruses, infectious bursal disease virus), degraded proteins, excess of calcium in diet, deficency of Vitamin-A in the diet, myctoxicosis particularly ochratoxicosis, water deprivation etc. (Chandra, 1982; Bokari, 1965; Rendall, 1991). However, no such information is available in quails. Since the information of the occurrence of one or more of the above factors in the above referred nephrosis/nephritis cases is not available, it is difficult to ascribe any specific etiological relationship to these cases.

Nephrosis and hepatosis was recorded in 26 cases. The hepatosis is primarily caused by aflatoxicosis. Jassar (1986) and Singh (1991) recorded concurrent occurrence of nephrosis and hepatosis due to aflatoxicosis in chicken. The feed used at the PAU poultry farm (which was source of majority of cases under study) was found to have aflatoxin levels above permissible limits. However, hepatic degenerative changes can also be consequence of general venous congestion which might have occured due to degenerative changes in other parenchymatous organs viz. kidney, lungs, mycardium etc.

The occurrence of lesions of serositis of proventriculus, gizzard, epicardium as the associated lesions can also not be assigned the causative relationship. Oophoritis has been recorded as associated lesion in nine cases. In domestic fowl, oophoritis has been ascribed to certain viruses viz. infectious bronchitis virus, egg drop syndrome virus and other adenoviruses. The viral damage in the ovary has been reported to be superimposed by

secondary mycoplasmal, bacterial infections (Singh, 1988). Similar etiological relationship can be expected in oophoritis of quails.

Traumatic ventriculitis was seen in one case which was caused by swallowing of cage wire which ultimately pierced through the wall of gizzard. Fungal cloacitis could be seen due to infection by fungus which might have occured due to damage possibly by picking or prolapse.

Gross lesions indicative of haemorrhagic syndrome were characterized by haemorrhages in breast muscles, thigh muscles, internal organs, hypoplastic anemia and emaciation etc. Microscopic lesions consisted of haemorrhages, fibrinous exudate and sometimes cellular infilterations in skeletal muscles of breast and thigh. Severe congestion and haemorragic conditions of liver, kidneys and lungs with degenerative and necrotic lesions of varying degrees in liver and kidneys were seen. Peckham (1972) had discussed haemorrhagic syndrome in chickens with similar lesions. He had discussed the possible role of toxic fungi, coccidiostats like sulphaquinoxaline and infectious agents particularly adenoviruses as possible etiological agents.

Persistent and cystic right oviduct was recorded in three cases in present study. Similar observations of persistent and cystic right oviduct were earlier recorded by Peckham (1972) in domestic fowls.

Bony metaplasia in lungs was seen in one case. Similar ossification in lung tissue had been recorded in mammals which could not be related with any specific etiological factor(s) (Jones and Hunt, 1983).

The lesions of muscular dystrophy in one case and of haemorrhages in the cerebellum and pale breast muscles in another case suggested deficiency of Vitamin E or Selenium. Scott and Krook (1972) had described Vitamin E being the cause of encephalomalacia, exudative diathesis and

muscular dystrophy in chicks, dystrophy of gizzard musculature and enlarged hocks in turkey and muscular dystrophy in ducks. They also recorded the role of selenium in such conditions.

In experiment conducted to study the pathology of *S. gallinarum* infection in quails, LD_{50} dose which was calculated out to be 6.75×10^7 organisms was administered intraperitoneally.

Mortality started on 6 DPI and continued upto 9 DPI which showed that incubation period of disease was 6 days and the course of disease was 4 days by the intraperitoneal route. No report from available literature regarding incubation period and course of disease in Japanese quail from *S. gallinarum* infection in experimental or natural outbreaks was found. Buxton (1957); Das *et al.* (1959) and Pomeroy (1972) reported incubation period to vary from 4-5 days and course of disease about 5 days in chicks. This again supports comparatively less susceptibility of Japanese quail to *S. gallinarum* as compared to chickens.

Clinical signs recorded were ruffled feathers, huddling together, backward movement, torticollis, prostration and paddling before death. Awaad *et al.* (1981) reported similar signs in experimental infection in quail by *S. gallinarum* by oral route or egg dipping. Sarma *et al.* (1988) were unable to report any symptoms from natural outbreak having peracute form of disease.

Gross lesions present in experimental infection were enlarged and mottled liver with congestion, haemorrhages and necrotic foci, enlarged and mottled spleens, dark bluish lungs, congested and haemorrhagic kidneys, haemorrhages in breast muscles, haemorrhages in heart. Bronze discoloration of liver, perihepatitis and pericarditis were also noted in some birds. Similar

lesions were reported by Sarma *et al.* (1988) in natural outbreaks in addition to hydropericardium.

In present study *S. gallinarum* could be isolated from liver and spleen of experimental birds showing these to be preferred organs for isolation.

Histopathologic lesions comprised of congestion, haemorrhage, varying degrees of degenerative lesions in parenchymatous organs viz. liver, kidneys, myocardium etc. In liver focal and sometimes diffuse necrotic and microgranuloma formation could be seen. Pericarditis and myocarditis with focal infilteration of cells besides congestion and haemorrhage were seen. Congestion and haemorrhages in lungs were persistent findings. Spleen had typical fibrinoid necrosis in most of the cases with mild reticular cell hyperplasia. Intestine showed mild chronic serositis characterized by mesothelial cell and reticular cell proliferation and mild fibrosis. The mononuclear cells and giant cells were present in one case on 13 DPI. Gizzard had pronounced serositis which may be extension of peritonitis. Cellular infilteration in muscular layer of gizzard was also observed. Mild meningitis was observed on 7 DPI and 8 DPI.

Similar but more severe lesions have been reported in experimental *S. gallinarum* infection in chickens (Bali, 1995). However, fibrinoid necrotic splenitis observed in Japanese quails in the present study was not observed in chickens.

Chapter VI

SUMMARY AND CONCLUSIONS

The present work was envisaged to study the prevalence of various pathological conditions affecting the Japanese quail in Punjab, pathological changes in different organs by these conditions and attempt to establish etiological factor(s)/agents for these conditions.

The study revealed that the mortality rate in different age groups of 0-1 week age, 1-6 week age and in age above 6 weeks differed insignificantly. Mortality rate was high during the hot summer and cold winter months in 0-1 week age and 1-6 week age groups and in adult birds (above 6 weeks age group) seasonal variations had not caused marked change in mortality pattern.

The important disease conditions recorded in Japanese quails were hepatosis/hepatitis condition (23.66%), yolk sac infection (13.47%), oophoritis (12.00%), coryza (8.01%), gangrenous dermatitis (7.91%), nephrosis-nephritis syndrome (6.15%), enteritis (5.64%), mycotic pneumonia (5.09%), lymphoproliferative disease (3.16%) etc. in the descending order. Yolk sac infection (52.25%) was important disorder in the 0-1 week age group. Important disorders in 1-6 week age group were hepatic disorder (27.89%), mycotic pneumonia (16.55%), nephrosis-nephritis condition (10.12%), coryza (8.76%), enteritis (6.27%) and gangrenous dermatitis (4.42%), etc. In adult birds (above 6 week age group) oophoritis (17.05%), hepatic disorders (13.00%), gangrenous dermatitis (10.15%), coryza (7.74%), enteritis (6.96%), Marek's disease (6.18%), Septicemia (4.04%), egg bound (3.94%), heat

stroke (3.80%), nephrosis-nephritis syndrome (3.58%), etc. were the important disease conditions in descending order. Cases of Inclusion body hepatitis, chronic respiratory disease and lymphoid leukosis were also recorded. The presence of conditions like gangrenous dermatitis and absence of coccidiosis, intestinal worms and ulcerative enteritis in our study may be attributed to housing of quails in cages at the university poultry farm.

An outbreak form of mycotic pneumonia was observed in June and July months of 1992 and an outbreak of Marek's disease had occurred in 1994.

To study the pathology of various conditions, gross and histopathological study of 166 cases was conducted. Lesions of Marek's disease (21 cases) mainly involved small intestine, liver, proventriculus and spleen as prime target organs. Brain and nerves were least involved and no lesions of Marek's disease were seen in heart, oviduct and bursa of Fabricius. A case of lymphoid leukosis studied had infiltration of uniformed sized lymphoblasts in ovary, liver, mucosa of gizzard and proventriculus and few cells in kidney.

From the cases of mycotic pneumonia (14 cases) *Aspergillus fumigatus*, *A. flavus* and *A. ochraceus* were the isolates. The cases involving *Aspergillus fumigatus* had more severity of lesions in lungs and involvement of air sacs, whereas, *A. flavus* cases had less severity of lung involvement, small sized granulomas with more cellularity and less necrosis and no extension to air sacs. Infection of kidneys by budding yeast like cells was present in one case.

Natural cases of fowl typhoid (4 cases) had necrotic foci and bronze discoloration of liver, mottled spleen, cyanosed and congested lungs, long stalked ova with oily contents. Microscopic lesions comprised of typhoid

granulomas, focal coagulative necrosis in liver, congestion haemorrhage and coagulative necrosis in renal tubules. In quails enteritis was not pronounced and fibrinoid necrosis of spleen was prominent feature.

Escherichia coil was isolated from 7 cases having conditions like oophoritis, salpingitis, prolapse of oviduct, coryza and septicemias. Perihepatitis, pericarditis, peritonitis typical of avian colibacillosis were also encountered from which *E. coli* could be isolated. Staphylococcal infection was recorded in three cases by isolation of staphylococcal organisms from necrosed parts of livers.

Gangrenous dermatitis mostly noted on head probably caused by thrashing against the roof of cages had extensive haemorrhagic and necrotic lesions in epidermis and dermis along with infilteration of inflammatory cells in dermis. Bacterial colonies mainly comprising of Gram positive cocci and some spore bearing Gram positive bacilli were present. Giant cell pneumonia in two cases, perivascular focal pneumonic lesions with bacterial colonies in one case and hepatic infarction in one case were the additional lesions in some cases of gangrenous dermatitis.

Lesions of septicemia seen in four cases comprised of congestion and petechiation on serosal and mucosal surfaces. Spleen in such cases had fibrinoid necrosis with congestion, haemorrhage. The degenerative and necrotic lesions were noted in other organs.

Infectious coryza seen in 6 cases was grossly characterized by mucopurulent exudate from nasal chambers and microscopically had mononuclear cell infiltration in mucosa of nasal chambers. Hyperplasia of lining epithelium of nasal chambers and mononuclear cell infiltration in trachea were also seen. In chronic respiratory disease, tuboalveolar hyperplasia of tracheal glands with lymphoid

follicles in submucosa were seen. Nasal sinuses had thickened mucosa with mononuclear cell infiltration.

Sixteen cases of nephrosis/nephritis syndrome and three cases of visceral gout were diagnosed grossly and microscopically. Changes consisted of degenerative and necrotic lesions with congestion and haemorrhages. Mild focal infiltration of inflammatory cells were seen in four cases. Two cases had dystrophic calcification in kidney. Visceral gout had white chalky deposits on various organs grossly, and microscopically, needle shaped uric acid crystals were seen in necrotic areas but without inflammatory response.

Non-specific cases of nephrosis-hepatosis had indicated toxic lesions attributable to aflatoxicosis or to degenerative changes in other parenchymatous organs.

Experiment was conducted using LD_{50} of *Salmonella gallinarum* (9:12:-:-) isolate from field outbreak to study the pathogenesis in experimental quails. Clinical signs of ruffled feathers, huddling together, backward movement, torticollis, prostration and paddling before death were recorded. Gross lesions comprised of enlarged, mottled and bronze discoloured livers, enlarged and mottled spleens, congested and haemorrhagic kidneys, haemorrhages on heart and skeletal muscles etc. The lesions were similar to those found in fowls. Liver and spleen were preferred site for isolation of organisms. Histopathological findings comprised of degenerative changes, focal or diffused necrotic changes, congestion and haemorrhages in liver parenchyma alongwith occasional occurrence of typhoid granulomas. Spleen had fibrinoid necrotic splenitis as an important feature. Pericarditis, myocarditis and mild serositis of intestine were present. Gizzard had pronounced serositis and mononuclear aggregates in muscular layer. Kidneys

had congestion, haemorrhages, degenerative and necrotic changes. Congestion and haemorrhages were present in lungs.

Conclusions

- Pre-summer and rainy seasons had lowered mortality.
- Yolk sac infection/omphalitis was most important condition in first week of age.
- Hepatic disorders were important conditions in 1-6 weeks age group.
- Oophoritis, hepatic disorders remained important conditions in adult birds.
- Caging of quails while checking some conditions gave emergence to other conditions like gangrenous dermatitis and fractured legs.
- Small intestine mainly duodenum, liver, proventriculus and spleen were target organs of Marek's disease in Japanese quail.
- Nerves were least involved in MD in Japanese quail as compared to nerve lesions being the major lesion in MD of domestic fowl.
- Japanese quail were susceptible to lymphoid leukosis.
- *Aspergillus fumigatus* was more pathogenic as compared to *A. flavus* and caused extensive and severe lesions in lungs.
- Fibrinoid necrosis of spleen was important pathological change in septicemic conditions in Japanese quail.
- Factors for nephrosis-nephritis condition in Japanese quail needed to be ascertained.
- Gross and histopathological changes in fowl typhoid in Japanese quail were quite similar to those in domestic fowl but differed in intensity.

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ABSTRACT

The present work aimed to study the prevalence and pathological alterations in various disease conditions of the Japanese quail in Punjab. Attempts to establish etiological factor(s)/agents were also made. It was found that pre-summer and rainy season had lowered mortality in 0-1 week and 1-6 weeks age groups and no marked change in mortality pattern occurred in adult birds (over 6 week of age) by change of season. In first week yolk sac infection was important disease condition (52.25%) and in 1-6 weeks age group hepatic disorders were important (27.89%). Oophoritis (17.05%) and hepatic disorders (13.00%) were main condition in adults. Changing the housing conditions of birds to cages resulted in fractured legs and gangrenous dermatitis. Histopathology of 21 cases of Marek's Disease revealed duodenum, liver, proventriculus and spleen as target organs in Japanese quail, however, nerves were least involved. Japanese quails were found susceptible to lymphoid leukosis. In mycotic pneumonia, *Aspergillus fumigatus* had more pathogenic lesions as compared to *A. flavus*. In septicaemic conditions, fibrinoid splenitis was important change. Histopathological changes produced by experimental *Salmonella gallinarum* infection in Japanese quail were similar to these found in chicken, but were of less intensity.

Signature of Major Advisor

Signature of the student