INFECTIOUS STUNTING SYNDROME OF BROILER CHICKS I. CLINICAL SIGNS AND PATHOLOGICAL LESIONS

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Summary

This study represented an endeavor to observe clinical signs and pathological lesions in broiler chicks suffering from experimental Infectious stunting syndrome (ISS). One hundred and twenty day old broiler chicks were divided randomly into two equal groups i.e. control (A) and inoculated (B). At day one of age each chick of the groups (A and B) was dosed with one ml of either tryptose phosphate broth or prepared inoculum respectively. Chicks of both the groups were housed separately under similar standard management. Inoculation induced characteristic clinical changes in birds of treatment group like of brownish diarrhoea, lameness, feather developing problems and paleness of combs, wattles and shanks. By day-29 of the experiment all the stunted birds from group-B and an equal number of birds from group-A were slaughtered. These birds were examined thoroughly to record the gross changes in various structures and then the severely affected organs were processed for histopathological examination. The skeletons of affected birds were brittle, keel bones showed quite prominence while the muscles and subcutaneous tissues were almost devoid of fat. Grossly it was observed that pancreas, spleen and bursa of Fabricius were severely atrophied while the intestines were ballooned with undigested feed and gases. Histopathological examination of pancreas and spleen revealed a classical picture necessary for understanding the pathogenesis of the syndrome. The acinar cells of pancreas were atrophied and underwent vacuolation, degeneration and necrosis. The zymogen granules were almost absent from the acinar cells. A characteristic change was an inflammatory reaction in one or more pancreatic ducts where the epithelium and fibrous tissues occluded the lumen of the ducts and led to the obstruction in pancreatic drainage.

(**Key Words** : Stunting Syndrome, Clinical Signs, Pathological Signs)

Introduction

Infectious stunting syndrome (ISS) is a condition causing growth retardation in young broilers and more rarely in other strains of domestic fowl. It was first reported in Netherlands in 1978 (Kouwenhoven et al., 1978) and in U. K. in 1981 (Bracewell and Wyeth, 1981). By early 1980 it was well established as a cause of colossal economic loss to the poultry farmers in America (Martland, 1989). It was first seen in Pakistan in 1983 but now appears as a major problem in commercial broilers. Neither the prevalence nor its severity appears to be limited by climatic or geographical factors. It is likely that disease has a vertical transmission. The incidence of ISS in commercial flocks is 0.5-30%, so the disease is of high economic significance (Jaffery et al., 1990). Etiology of the syndrome is probably multifactorial however the following viruses have been isolated namely reoviruses, caliciviruses, corona virus like particles, enteroviruses, togavirus like particles and parvoviruses. Clinically the syndrome is characterized by high early mortality, stunted growth, poor feed conversion, poor feathering, leg weakness and high incidence of lameness due to femoral head necrosis (Kouwenhoven et al., 1978). Different clinical manifestations seen across the world have produced a variety of names for the syndrome, like "Runting and leg weakness" (Kouwenhoven et al., 1978), "Infectious stunting syndrome" (Bracewell and Randall, 1984), "Helicopter chick" "Pale bird syndrome" and "Malabsorption syndrome" (Page et al., 1980). Upon necropsy it has been observed that affected broilers exhibited dilated and pale intestines often containing undigested feed. The pancreases were thin, white and firm

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because of loss of exocrine tissues and replacement by fibrous tissues. Many birds had skeletal changes suggestive of rickets (Riddle and Derow, 1984). Severely stunted birds had atrophied lymphoid organs, particularly the thymus and bursa of Fabricius.

The pathogenesis of this devastating syndrome is poorly studied in Pakistan. This present study was designed to experimentally reproduce the syndrome under local conditions and to elucidate clinical signs and pathological alterations occurring in various visceral organs as result of the experimental infection.

Materials and Methods

Inoculum preparation

Ten gastrointestinal tracts were collected from a flock of 11 days old commercial broilers diagnosed on the basis of clinical findings as having infectious stunting syndrome. Their gastrointestinal tracts along pancreases were blended with T. P. B. in 1:0.5 (wt:wt) ratio. The mixture was centrifuged at 5,000 RPM for 30-minutes and the resultant supernatant was collected in a sterilized flask for use as inoculum.

Experimental design and inoculation of birds

One hundred and twenty commercial day old broiler chicks were obtained from a commercial hatchery. Chicks were divided randomly into two equal groups i.e. control (A) and inoculated (B) and each chick of group A was dosed per os with one ml of T. P. B. and group B with inoculum. Birds were housed separately in two pens under similar standard management.

Clinical symptoms

During the experiment birds of both groups were observed keenly to record their respective clinical behavior.

Pathological examination

On day 29 of the experiment all the stunted birds from inoculated group and an equal number of normal birds from control group were slaughtered. Gross pathological lesions were searched in whole carcasses of both the groups. Small pieces of visceral organs-intestine "mid-duodenal segment" pancreas, spleen, liver and kidney of both groups were collected for histopathological examination. These tissues were preserved in 10% buffered formalin and later on stained with routine hematoxylin and eosin stain (Drury and Wallington, 1980).

The stained sections of different tissues from both groups were visualized under different magnifications of light microscope and prominent histopathological lesions were photographed.

Results

By 1-2 days post inoculation pasty vents/diarrhoea of yellow to brownish color was visible in affected birds. They were having a hunched appearance, ruffled feathers and drooping wings. There was an unevenness in the size of inoculated birds which was detectable apparently by 4th to 5th day of age. Almost by the end of first week birds of inoculated group were seemed to be active but variation in sizes was much more pronounced. The feed intake of experimental chicks was much reduced. During the second week of age it was evident that some of the underweight birds had developed feather problems and some of the stunted birds were lame with swollen hock joints. During the third week of experiment it was observed that these severely affected birds had few ruffled, short, broken shaft wing feathers which gave the chick a curious appearance. The characteristic paleness, a feature of ISS, was quite evident in the later phases of the experiment. During the present study 20% of the inoculated birds died, more in beginning which were decreased in the successive weeks of experiment.

During postmortem the following gross lesions were recorded: the keel bones of the affected carcasses were quite prominent, while the muscles and subcutaneous tissues were almost devoid of fat. The skeletons of the affected carcasses were brittle as during disarticulation mostly the heads and sometimes the shafts of the femur were broken. Upon opening the carcasses it was noticed that intestines in majority of the affected birds were swollen, filled with gases and ingesta and paler in color as compared to the intestines of birds of control group. The lymphoid organs bursa of Fabricius to a lesser extent while spleen and thymus were severely atrophied.

The gross and histopathological lesions observed in different visceral organs were as follows:

Gastrointestinal tract

Lesions of proventriculitis were observed in few affected birds. Paleness of the intestines having partially digested feed and catarrhal enteritis were frequently encountered features of ISS. Microscopically atrophied villi in the duodenal region with sloughing and necrosis of epithelium at their tips was quite evident. Edema and mononuclear cells infiltration in lamina propria and villi were observed in most of the affected cases as shown in figure 1.

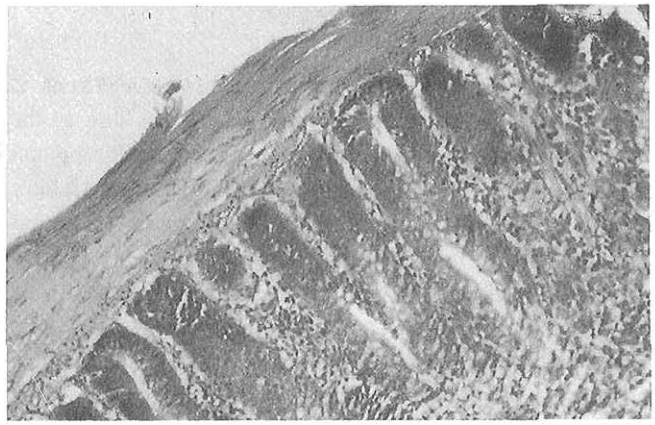

Figure 1. Intestine showing atrophied villi with sloughing at their tips, cellular infiltration, edema and increased intervillous space.

Pancreas

The pancreas was the most severely affected organ amongst the inoculated birds. In majority of the birds it was severely atrophied presenting a shrunken fibrous band like structure in the convoluted duodenal loop. The color of these pancreases was white or off-white to pink and were firm in consistency. Microscopically it was observed that acinar cells were atrophied and underwent vacuolation, degeneration and necrosis. The zymogen granules were almost absent from the acinar cells. Heterophil, leukocytic and lymphocytic infiltration were frequently encountered in affected cases. A characteristic change was an inflammatory reaction in one or more pancreatic ducts where the epithelium and fibrous tissues occluded the lumen of the ducts and led to the obstruction in pancreatic drainage. These microscopic changes are shown in figures 2 and 3.

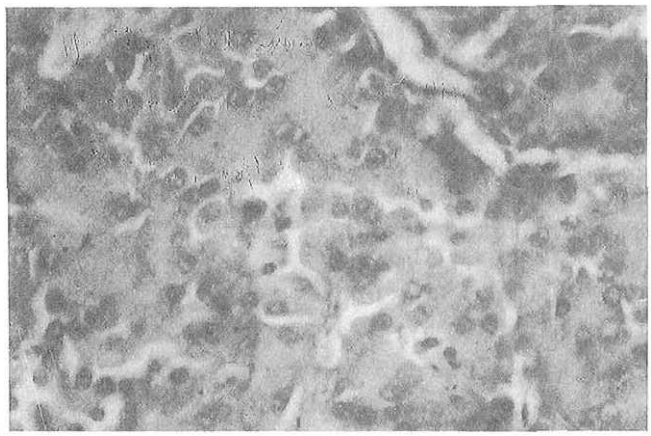

Figure 2. Pancreas showing heterophil and leukocytic infiltration, vacuolation, necrosis and absence of zymogen granules.

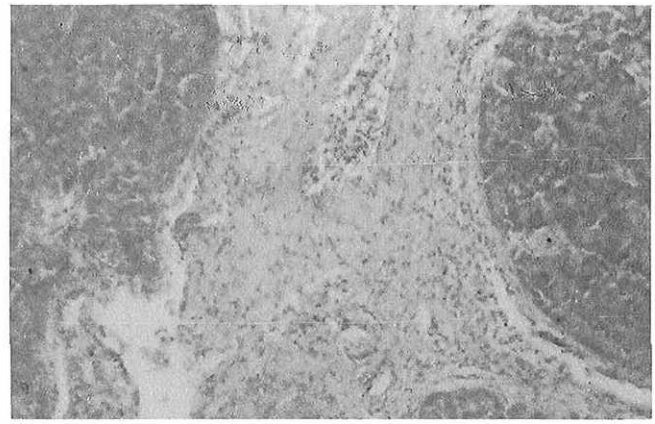

Figure 3. Pancreas showing obstruction of pancreatic duct, atrophied acinar cells and necrosis.

Liver

In most of the affected birds the liver was pale and necrosed however in some cases congested livers showing the lesions of perihepatitis were also noticed. Microscopically cloudy swellings and severe necrosis were seen in the affected livers. Mononuclear and plasma cells infiltration were also quite evident. These changes are elaborated in figure 4.

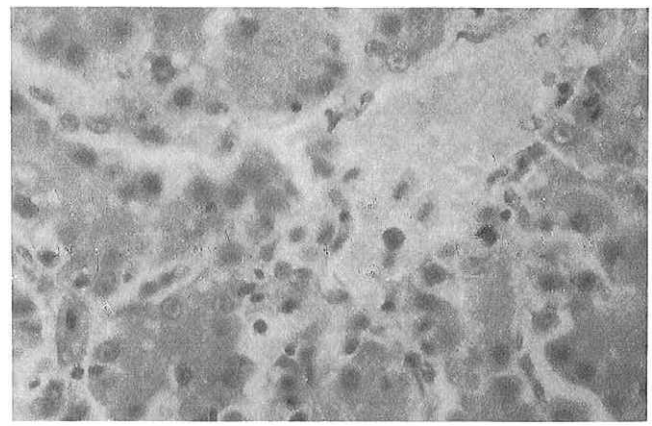

Figure 4. Liver showing mononuclears and plasma cells infiltration, cloudy swelling and necrosis.

Spleen

In almost all the cases the spleen was pale to brownish in color. It was severely atrophied and harder in consistency. Microscopically it was observed that lymphocytic infiltration was a common feature in affected spleens. A characteristic change observed during the present study was the reduced number of lymphoid follicles having less number of lymphocytes per microscopic field. These lymphoid follicles were atrophied and surrounded by fibrous tissue capsules. Haemorrhages

in the interstitial tissues were also noted, these microscopic changes are shown in figure 5.

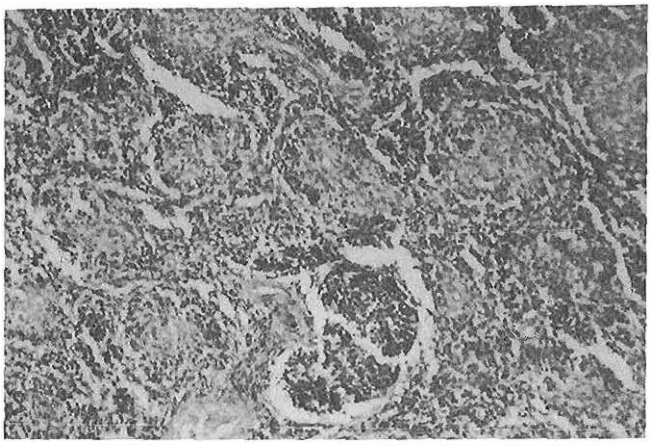

Figure 5. Spleen showing atrophied lymphoid follicles with reduced number of lymphocytes, lymphocytic infiltration and haemorrhages in interstitial spaces.

Kidneys

Majority of the affected birds showed that the kidneys were inflamed and congested. Urates depositions and pinpoint haemorrhages were also noted in few cases. Microscopically leukocytic infiltration in the interstitial spaces and glomerular region were quite evident. The dilated blood vassels and haemorrhages in tissue spaces were observed. In some of the severely affected birds the glomeruli were enlarged and were less in number as compared to that of normal birds. Lesions of necrosis were also seen in some of the affected kidneys as shown in figure 6.

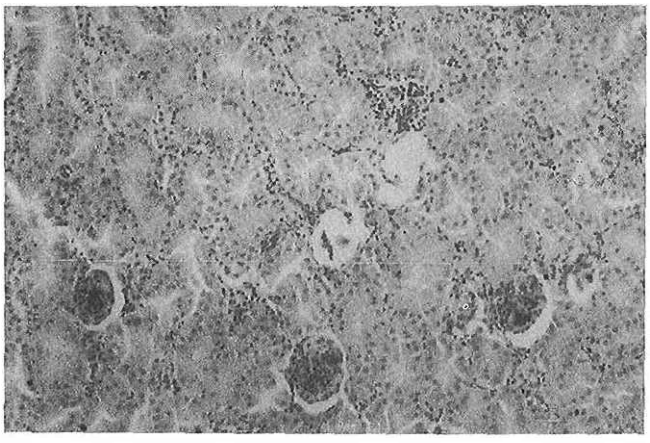

Figure 6. Kidney elucidating reduced number of enlarged glomeruli, leukocytic infiltration, necrosis and haemorrhages in interstitial spaces.

Discussion

Stunting syndrome in commercial broiler chickens is an emerging problem. Heavy economic losses due to the disease are inflicted to the broiler producers in Europe and America (Farmer, 1985) and now also in Pakistan (Jaffery et al., 1990). During 2nd week of experiment lameness and lesions of tenosynovitis were observed in severely affected chicks, which are in agreement with the observations of Jones and Gunerante (1984). In later stages of the present study it was observed that typical stunted birds had pale shanks, combs and wattles. This observation correlates with the pathogenesis of paleness described by Heide et al., (1981) and Page et al. (1980) that affected birds fail to absorb dietary carotenoid pigments and Vit. E necessary for normal skin coloration. Maldigestion would also prevent the plant pigment being released from maize, which would account for the paleness of birds. Hence the syndrome is also named as "Pale bird syndrome" and "Malabsorption syndrome". During the present study it was observed that a high number of birds died in the beginning i.e. P. I. which gradually decreased in the subsequent weeks of experiment. The mortality pattern was suggestive of age related resistance. Kouwenhoven et al. (1978) and Vertomen et al. (1980) also stated that mortality is generally a feature of early acute phase of ISS.

During postmortem it was observed that affected birds were severely emaciated and their keel bones were too prominent. Their flesh and sub-cutis were almost devoid of fat. Tang et al. (1987) observed that stunted birds exhibited severe weakness and had pale breast muscles. The skeletons of birds during the present study were fragile and brittle. The bone changes in severely affected birds are assumed to be caused by the decrease in vitamin D absorption and exacerbated by the likelihood of intestinal calcium being chelated to lipid and lost in the faeces. The lymphoid organs were severely atrophied. The changes in lymphoid organs may not be due to a primary effect of ISS. The gastrointestinal tracts were distended with poorly digested feed and gases. Such observations have also been recorded by Ruff (1982). Intestines were paler in color and catarrhal enteritis was a common lesion amongst the affected birds. This manifestation was previously reported by Page et al. (1980). Microscopically the vili of the duodenal region were atrophied, the epithelium at their tips was necrosed and some were sloughed off. Edema and mononuclear cells infiltration were also noted in majority of the affected intestines. These histopathological observations are in agreement with Elmubarak et al. (1990). Pancreas was the most

severely affected organ. Such finding was also reported by Reece et al. (1984). An inflammatory reaction was observed in the pancreatic ducts where the epithelium and fibrous tissues led to the obstruction of the pancreatic drainage by occluding the lumen. In this respect Martland and Farmer (1986) have elucidated the pathogenesis of ISS where the cause of pancreatic atrophy was described as the pressure caused by obstruction of pancreatic drainage. Thus pancreatic enzymes were prevented from entering the intestines, with consequent maldigestion of primary nutrients, so that fat soluble vitamins will not be properly absorbed. This in turn to the retardation in growth and feathering and to reduce absorption of nutrients including carotenoid pigment. Randall et al. (1981) postulated that the pancreatic lesions due to ISS were similar in some respects to that induced by selenium deficiency and the possibility that some interference with digestion or absorption resulted in poor growth, poor feathering, rickets and pancreatic lesions can not be ignored.

Spleen in almost all the affected birds was pale to brownish in coloration, it was harder in consistency and was severely atrophied as observed by Hieronymus et al. (1982). Microscopically lymphocytic infiltration was quite obvious. A characteristic change noted during the present study was the reduced number of lymphoid follicles having less number of lymphocytes per microscopic field. Probably it was an indication of immunosuppression as described by Jaffery et al. (1990). Griffiths et al. (1985) suggested that changes in lymphoid organs could be due to the nutritional effects associated with poor nutrient utilization.

Kidneys in majority of the affected birds were inflamed and congested. Urates deposition was also noted in few cases. These findings coincide with the observations of Elmubarak et al. (1990). Microscopically leukocytic infiltration in the interstitial tissues was quite evident. The blood vessels were dilated and hemorrhages in affected kidneys were also noticed. In some of the severely affected kidneys glomeruli were less in number and were enlarged. These histopathologic observations were previously reported by Elmubarak et al. (1990).

Pancreas and spleen were of major significance as they presented a classical picture necessary for understanding the pathogenesis of the syndrome. The pancreas may be the primary organ affected leading to a subsequent interference with normal digestion.

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