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INFECTIOUS STUNTING SYNDROME OF BROILER CHICKS INCIDENCE, FEED CONSUMPTION AND GROWTH TRAITS

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SUMMARY

120, day old commercial broiler chicks were obtained and divided into two equal groups i.e. control (A) and treatment (B). Each chick of both groups was weighed and then dosed with one ml of either tryptose phosphate broth (A) or prepared inocula (B) at the same day. Chicks of both groups were housed separately under similar standard and managemental conditions. In the subsequent period of the experiment, it was observed that these severely effected birds had few ruffled, short broken shaft, mature wing feathers and in the later stages of experiment, these birds had pale combs, wattles and shanks. During 28 days experiment, 20% of the inoculated birds died while none of the bird died amongst control group. To determine the "Feed Conversion Ratio" FCR sufficient amount of feed was provided to each group and the remaining feed in feeding troughs was calculated at the same weekly intervals. It was statistically concluded that Feed Conversion Ratio was severely impaired in birds of group B which remained significantly lighter in the weight at all the stages of the experiment. Hence we can estimate a successful induction of the syndrome by a crude intestinal homogenate prepared of the affected birds under local conditions.

Key words: *Infectious stunting syndrome, broiler chicks, feed consumption.*

INTRODUCTION

Infectious stunting syndrome (ISS) is a condition causing growth retardation in young broiler chickens and more rarely in other strains of domestic fowl. It was first seen in Pakistan in 1983 in a few flocks, but now appears wide spread in the commercial broiler chickens in the whole of the country. The disease is of high economic significance and

losses increase with age since the stunted birds eat well but do not gain weight (Jaffery *et al*, 1990.

Clinically the syndrome is characterized by high early mortality, stunted growth, poor feed conversion, poor featherings, leg weakness and high incidence of lameness due to femoral head necrosis. Different clinical manifestations seem across the world have produced a variety names for the syndrome, like due to disturbance of growth and feathering it is called, "Runting and Stunting syndrome or Infectious stunting syndrome (Brace well *et al* 1984).

The microscopic lesions during infectious stunting syndrome of broiler chicks consisted of lymphocytic renal and pancreatic interstitial infiltrates, dilated or cystic duodenal and jejunal crypts of lieberkuhn, increased crypt depth and increased cellularity in the intestinal lamina propria (Montgomery *et al.*, 1997)

The pathogenesis of this divasting syndrome of poultry is poorly studied in Pakistan. That is why the present project was designed to elucidate metabolic derangement and pathological changes is visceral organs of broilers suffering from experimentally induced infections stunting syndrome.

MATERIALS and METHODS

120, one day old commercial broiler chicks were obtained from a commercial hatchery. Chicks were divided randomly into two equal groups, one as a control group (A) and the other as an inoculated group (B). Chicks at the same day of age were weighed and dosed per os one ml of either tryptose phosphate broth (Group A) or inoculum prepared from a crude intestinal homogenate of diseased birds (Group B). Birds were housed separately in two pens under similar standard managemental conditions. Feed and water were provided.

Feed consumption of both the groups were determined at the end of every seven days, starting from day 1-28 of the experiment. A measured amount of sufficient feed was put to each group and the remaining feed in the feeders was weighed at the end of each week. Feed consumption = total given feed – remaining feed in feeders. To demonstrate the weight gain, each chick of both groups was weighed individually at the end of every seven days. Thus, the correlation between the feed consumption and weight gain of both groups was determined separately throughout the period of investigation.

The tissues were processed for histopathology, prior to processing, the bones were incubated at 37^{o} C in 10 % formic acid for 2 hours for decalcification (Bancroft and Stevens, 1983 and Bancroft and Gamble, 2002).

The data collected was analyzed statistically by applying students unpaired t-test- (Steel and Torrie 1980; Wayne and Daniel 1995, and Roger *et al*., 2003)

RESULTS and DISCUSSION

During the present study it was observed that a higher number of birds died in the beginning i.e. post inoculation which gradually decreased in the subsequent weeks of the experiment. The mortality which took place in the present experiment was 20%. Generally, mortality is a feature of early acute phase of infectious stunting syndrome (Kouwenhoven *et al,* 1978 and Vertomen *et al,* 1980).

Martland, 1989 stated that between 7 and 10 days, variation in size becomes more pronounced and at around 10 days of age mortality may increase again. From approximately 14 days of age the syndrome enters a chronic phase which usually persists until the birds are slaughtered but results of our study differ from the report of Martland (1989).

Stunting syndrome was induced successfully as judged by severely depressed weight gain, reduced consumption of feed and impaired feed conversion in inoculated poults (Angel *et al* 1989). Sell, 1991 observed that inoculation with the infective material had a dramatic impact on poults growth within a very short time.

It was also observed that weight gain was reduced by infection to a much greater extent rather than feed intake.

Consequently feed to gain ratio which was much larger for the infected poults than for the control, illustrating that utilization of nutrients was impaired by the infection. During the present study, feed conversion ratio of both groups was also recorded by putting sufficient feed and calculating the remaining feed in the feeder at the end of every week. The difference in feed consumption to weight gain ratio increased through the experiment probably due to maldigestion and malabsorption. It was also observed that the overall weight of gastrointestinal tract of birds in control group was more, however, the percentage weight of gastrointestinal tracts of birds in inoculated group was significantly increased (Brace Well and Randall, 1984). Tang *et al.* (1987) observed

that stunted birds exhibited severe weakness and had pale breast muscles. It was observed at necropsy that bird with swollen hock joint had transparent fluid in their synovial cavities and lesions of tenosynovitis were also observed. These findings are in agreement with earlier workers (Gouvea and Schnitizer, 1982, Dhillon *et al,*. 1986 and Tang *et al,*. 1987).

The clinical and pathological observations were also alarming. Pancreas was of major significance as it presented a classical picture for understanding the pathogenesis of the syndrome. The affected pancreas was white or white to pink in colour and firm in consistency (Brace Well and Randall, 1984, Reece *et al,*. 1984, Derow, 1984 and Martland and Farmer, 1986). Much of work is needed to be done locally to understand the syndrome especially from the microbiological aspect. So, as to isolate the prevalent pathogens in our local environment to develop the control measures.

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