THE ROLE OF REOVIRUS IN RUNTING, STUNTING SYNDROME IN BROILER CHICKENS

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ABSTRACT

This study was conducted on a flock suffering from stunded growth, enteritis, diarrhea and lameness. Reovirus was isolated and identified by AGPT. For the reproducing of runting, stunting syndrome (RSS) in broiler chickens, day-old broiler chicks were used. Chicks were examined for the development of RSS, 4 weeks post infection. Chickens placed on litter on which chickens had previously suffered from RSS showed clinical aspects of the syndrome, induced growth inhibition, began significant decrease in body weights at 7 and 14 days of age and highly significance decrease was observed at days 21 and 28 of ages. Broilers inoculated per os with reovirus preparation, showed slight growth inhibition, but was of no significance, allover the experiment. Chicken exposed to tissue homogenates showed moderate growth inhibition with significant decrease in body weights at day 7 of age and highly significant decrease at days 21 and 28 ages. In this study, reovirus isolated from field outbreaks proved to be not capable of reproducing RSS experimentally in broiler chickens. However, obtained results using infected litter or tissue homogenate induced the disease. Therefore other infectious agent(s) were not identified could be considered in reproducing RSS in broilers.

INTRODUCTION

Stunted growth, enteritis accompanied with diarrhea and lameness are clinical manifestation described in association with the so-called, runting, stunting syndrome (RSS). Synonymous are stunting syndrome (SS): Madigestion syndrome (MDS).

Infectious runting and stunting syndrome (I-SS or I-RSS), pale-bird syndrome (PBS), osteoporiosis, brittle bone disease (BBD), femoral head necrosis (FHN), helicopter disease (HD), infectious proventriculitis, malnutrition syndrome, limited or no growth disease. Infectious stunting syndrome, and broiler runting syndrome, was first described by **Kouwenhoven *et al.*, (1978)** as a complex problem of young broiler chickens resulting from one or more infectious agents. This syndrome is characterized

by a varying incidence of birds with marked reduced live-weight (2 to 15%) and retard feathering in a flock at about 2 weeks of age.

Rosenberger, (1980) isolated reoviruses from joint swabbings, bone marrow, liver, spleen and tendons from young chickens suffering from stunting, feathering abnormalities, osteoporiosis, lack of skin pigmentation and arthritis, while **Villegas *et al.*, (1981)** isolated four reoviruses from tissue from broilers showing signs and lesions of proventricular hyperplasia.

Hieronymus *et al.*, (1983) isolated five reoviruses of three distinct serotypes from intestine of broilers with malabsorption syndrome.

Van der Heide *et al.*, (1981) were not able to reproduce diarrhoea but feathering abnormalities, femoral head fractures and osteoporiosis occurred in a small number of SPF birds infected orally with a reovirus isolated form a field case. The authors stated that avian reovirus is a possible aetiological agent of femoral head necrosis and brittle bone disease.

The syndrome was recorded in broiler breeds for the first time in Egypt by **Kheir El-Din and El-Sonousi, (1986) and Bekhit *et al.*, (1993)** described the disease in broiler chickens, as well as **Ghanam and Abd-Allah, (1998)** who reported the disease in native baladi chickens.

Although the RSS syndrome is described as early as 1978. Studies on the etiology extended over 20 years period, various agents were found associated with field outbreaks.

Therefore, the present study aimed to isolate and identify reovirus from a field cases and to study it's ability to produce the syndrome in broiler chickens experimentally.

MATERIAL AND METHODS

Flock history:

Four thousands and seven hundred chickens of Hubbard broiler breed aged 2 weeks old were present in a private farm at Sharkia Province. Parent flocks of these broilers were vaccinated with reovirus vaccine. The clinical signs observed were diarrhoea, stunded growth, lameness, ruffled feather and low vitality. Mortality rate among chickens was about 7%.

Reovirus isolation:

According to **Smart *et al.*, (1988)**.

a- Tissue homogenate inocula:

Homogenates were prepared from intestine and pancreas of diseased birds which subsequently developed RSS. The entire pancreas and small intestine were suspended in 20% (w/v) PBS containing 1000 IU/ml of penicillin and 1000 ug/ml streptomycin and gentamycin. The suspension was homogenized. The homogenate was then centrifugated at 2000 g for 10 min. and the supernatant was harvested.

b- Viral inocula:

The supernatant was filtered through a 450 nm filter. Bacteriological examination of these suspensions proved to be free from aerobic and anaerobic bacteria. The supernatant was then inoculated into the yolk sac of 5-6 days commercial embryonated chicken eggs and on the CAM of 9-10 days. Virus identification was accomplished by testing CAM in AGPT against the S-1133 strain, according to **Olson and Weiss, (1972)**. It was regarded negative after at lest five "blind" passages.

Reference chicken reovirus antiserum (S-1133):

SPAFAS Comp. USA, was obtained from Animal Health Research Institute, Dokki, Egypt.

For experimental infections with reovirus, CAMs were harvested 3 to 5 days post-inoculation, homogenized and the suspension was clarified by centrifugation at 3000 rpm for 30 min. in cooling centrifuge. The supernatant was titrated on CAM of 10 day embryonated eggs. The virus titres were determined according to **Reed and Muench, (1938)**, and varied from $10^{3.2}$ to $10^{3.9}$ ELD_{50}/ml.

Samples for bacterial isolation:

Liver, heart, spleen, intestine were collected form diseased and freshly dead chickens.

Bacteriological media:

Tetrathionate broth and Selenite F-broth (DIFCO) Xylose-lysine-Desoxycholate agar (XLD) (DIFCO). Nutrient agar and MacConkey agar media were prepared principally according to **Cruickshank *et al.*, (1982)**, the media were mainly utilize to isolate other combining bacteria which may play a role in increasing the mortality among diseased chickens.

Chickens:

One hundred one-day-old meat strain (Hubbard) chickens obtained form commercial Hatchery were randomly divided into four equal groups:

- G1: chickens were placed on litter known to produce enteric disease in chickens (exposed group).
- G2: chickens were given 0.2 ml of an isolated reovirus per os with syringe (infected group).
- G3: chicken infected with tissue homogenate and administrated by oral route.
- G4: chicken (non-infected, non-exposed) act as control.

All groups were given food and water ad libitum. When required, suboptimal temperature exposure was achieved, the heater was turned off of the first week of the experiment. The desgin of the experiment was illustrated in Table (1).

Histopathology:

On day 7, 14, 21 and 28 after placement, five birds from each group were randomly collected, weighted, killed and necropsied. Samples of intestine and pancreas were taken for histopathology; organs were fixed in buffered formalin and stained with E & E after **Perry *et al.*, (1991).**

Data were statistically analysed according to **Snedecor and Cochran, (1967)**.T-test was used to compare two groups in Table (2).

RESULTS

Clinical signs:

Affected birds were lethargic, usually display clinical signs of stunded growth, malfeathering, diarrhoea low vitality, undigested feed in the faeces, poor pigmentation, raising of two wings upward taking the appearance of helicopter wings (see Fig. 1), lameness, the hock joint was immabililized with development of architic condition.

P.M. lesions included catarrhal enteritis and the entire intestinal tract was grossly distended and fluid filled and/or contained undigested feed, proventricular swelling, lameness associated with tenosynovitis, ricketly changes (swollen rib heads in stunded broilers), pale pancreas, caeca distended with frothy material (in some cases), atrophy of thymus (in few cases) and there was little or no compensatory growth in stunded birds following the course of the disease; therefore, birds remained stunded through the grow-out period to market age.

Virus isolation:

Following passages in embryonated eggs, embryonic mortality was observed 3-5 days post inoculation via yolk sac, while mortality was observed 7^{th} to 8^{th} day post inoculation on CAM. The inoculated embryos were stunded with marked s/c haemorrhages allover the body, the internal organs were congested. Slightly small white pock lesions were found on the CAM of some embryos. Embryos that survive until 21 days were, markedly haemorrhagic dwarfed and as compared with the control ones.

Five virus isolates were examined in AGPT against REO hyperimmune serum of which 3 proved to be reoviruses.

Results of bacteriological examination:

Salmonella species and E. coli, were isolated from, heart, liver, spleen and intestine of infected chickens, indicating a complex infection.

Results of experimental infection:

Clinical signs:

Immediately following infection, the birds were clinically ill, visibly depressed and decreased in weight.

Birds of group (1) developed, profuse yellow-orange mucoid diarrhoea with pasting around the vent at age of 2 weeks-old and lasted up to the end of the experiment, most birds stunted with helicopter wings and litter eating. The birds showed more pronounced growth inhibition with significant decrease in

body weight in days 7 and 14 of age and developed high significant decrease at days 21 and 28 of age of the affected birds as in Table (2).

Birds of Group (2) their faeces were slightly wet from day 3 to day 8, showed a slight growth inhibition without significant decrease in body weight allover the experiment as in Table (2).

Birds of group (3) produced yellow mucoid faeces at 12 day of age and lasted up to the end of the experiment, some birds runted with helicopter wings, these birds showed moderate growth inhibition, with significant decrease in day 7 and high significant decrease in body weights in days 21 and 28 of age as in Table (2).

Lameness associated with tenosynovitis was manifested clinically in group 1 and 3 more than in group 2.

Reovirus was reisolated from fresh faeces from all exposed groups (No. 1, 2 and 3) at days 7-9 but not from the non-infected control birds of group (4).

Birds of group (4) were apparently healthy at all ages were in normal size, glisten uniform feathers and were active.

Gross findings:

Throughout the trial, exposed broilers were smaller and emaciated than the controls on days 7, 14, 21 and 28, the body weights of the exposed chickens have been reported (Table 2). On days 7 the entire intestinal tracts of the exposed chickens were thin and flaccid, grossly distended and fluid filled and/or contained undigested feed (Fig. 2). Proventriculus showed enlargement, with the presence of erosions and haemorrhage on their mucosal surface, filled with litter shavings in some cases. On day 7, 14 gall bladders were moderately (G 2) to markedly distended (G 1 and G 3). The pancreas was hard and white. The affected birds showed thinning of their pectoral muscles. Skeletal lesions were described as soft and pliable, Misshapen bone have also been observed in broilers specially in G 1 and G 3 than G 2, the shank lengths in the exposed birds were also significantly short in G 1, that recorded in birds of G 2 and G 3 than the lengths of the controls on days 7, 14 and 21, where bone lengths in the exposed birds at 21 days beings similar to those of the controls on day 14. All parenchymatous organs showed shrinkage. Bursa of fabricius was atrophid. While normal apparently healthy ones (G 4) showed normal viscera, bones and muscles.

Microscopic findings:

1- Intestinal lesions:

Included dilation of crypts of Lieberkuhn, necrosis of the crypts of epithelial cells and/or loss of crypts, these lesions were markedly noticed in G3 than G1 and was less in G2. These lesions appeaed on day 7 and were observed upto the day 21.

Lesions of pancreases were mainly confined to the exocrine pancreas, there were different stages of degeneration, atrophy fibrosis, vaculation of the cytoplasm of the acinar cells and few of these contain zymogen granules (Figs. 3 & 4).

The incidence of pancreatic atrophy assessed at 14, the results showed that group treated with tissue homogenate (G 3) showed pancreatic atrophy than (G 1) and (G 2).

2- Effect on mean live weight:

The effect of each treatment on mean live-weight at 7, 14, 21 and 28 days post inoculation was compared with an appropriate control group using analysis of variance. There were significant reduction in the mean live weight of the treated groups No. 1 and 3. Neither of the reovirus inoculated (G 2) and significant effect on mean live-weight as in Table (3). The difference in body weight between the control and exposed broilers become progressively more severe overtime.

DISCUSSION

Despite the preventive measures, the incidence of runting, stunting syndrome within commercial chicken flock remains high. In our study the infected chickens derived from vaccinated breeders with inactivated reovirus vaccine. This might be explained due to fact that, reovirus vaccine is not highly immunogenic and not induced high level of neutralizing antibodies in the vaccinated chickens. A further problem is the antigenic variation among avian reoviruses. Second vaccination of breeders with inactivated reoviruses, resulted in effective transfer of antibody to the offspring, but did not protect the offspring against malabsorption syndrome, **Kouwenhoven *et al.*, (1988)**. Third, the importance of reovirus antibody in establishing protection is not well understood since birds may become persistently infected in the presence of high level of circulating antibodies, **Jones and Nwajei, (1985)**. Fourth the reoviruses are highly immunosuppressive which retard protection against infection.

Three reoviruses isolates could be isolated from the intestine and pancreas of affected birds that were suffering clinical and PM lesions of RSS. Similar observation showed that the reovirus was assoaciated with MAS (**Rosenberger, 1980; Van der Heide *et al.*, 1981; Villegas *et al.*, 1981; Hieronymus *et al.*, 1983 and Kouwenhoven *et al.*, 1988**). This investigator isolated viruses from cases with clinical signs of MAS, but were not able to reproduced MAS experimentally. The role of these agents could not be defined

Salmonella and E. coli infections were demonstrated in this study. Mortality 7% in this study could not be attributed to reovirus infection alone,

but due to the reo and/or isolated complicated bacteria, this result accords with **Kouwenhoven *et al.*, (1986 a, b).**

Experimentally infected chickens in G1 showed decrease in body weight stunded growth. These seem to be a feature of reovirus infection, **Rosenberger *et al.*, (1989)**. The mechanism through which avian reovirus reduce the body weight is not yet clear but it has been hypothesized that when reovirus induced enteritis it interferes with the digestion and/or intestinal absorption, resulting in malabsorption syndrome, **Knowenhoven *et al.*, (1978); Rosenberger, (1980) and Van Der Heide *et al.*, (1981)**. On the other hand pancreatic inflammation and degeneration has also been incriminated by **Van Der Heide *et al.*, (1981)** who suggested that lack of secretion of digestive enzymes due to pancreatic degeneration would lead to improper absorption of nutrients and presence of undigested feed in the intestinal lumen. Other hypothesis were reported by **Rossler and Rosenberger, (1989)** who suggested that reovirus cause significant physiological and biochemical alterations that can be reflected in growth inhibition of proteins and mineral metabolism. While, **Reece and Frazier, (1990)** mentioned that infectious stunting syndrome caused significant intestinal pathology, the balance between the damage of the intestines and the compensatory mechanisms probably affects the final outcome of the disease by reducing the growth rate.

In this study diarrhea was manifested in naturally and experimentally infected chickens, primary malabsorption or maldigestion may be involved. Malabsorption is caused by intestinal dysfunction, lack of bile salts or lymphatic obstruction. Maldigestion occurs as the result of defecience of pancreatic enzymes, **Moon, (1978), Riddell and Derow, (1985) and Martland and Farmer, (1986),** and/or at least three components contribute to diarrhea that develops in nutrient malabsorptive state. First, water and electrolytes are pulled into the intestinal lumen by the osmotic activity of unabsorbed solutes, secondary, villos atrophy and crypt hypertrophy alter the balance of absorption and secretion. Finally, growth of small intestinal or clonic bacteria may deconjugate bile acids and hydroxylase fatty acids. These molecules in turn are more amphophilic and insert into the lipid phase of plasma membranes, resulting in increased permeability and active secretion as described by **Field *et al.*, (1989).**

Skeletal lesions that occurred in broilers in this study were described also by **Riddell, (1981) and Van der Heide *et al.*, (1981) and Perry *et al.*, (1991)** and explained by runting, stunting syndromeassociated with hypocalcemia, which progresses into architic lesions associated with vitamin D delplestion and hypophosphatemia.

The different signs presented by birds affected by RSS in our study may be due to the different nutritional and management practices in different countries. Notably, intestinal homogenates which had activity in our model were also found to be active when tested by **Kouwenhoven *et al.*, (1988).**

This suggests that the active agent of RSS, but not necessarily the clinical signs, may be common in different parts of the world.

Failure to reproduce pathological features of RSS (in the experimental work) using reovirus isolate from active intestinal homogenates, has lead to the suggestion that other unknown agents (viral, bacterial, environmental, nutritional, managemental, hygienic, etc.) may be involved in a secondary, or associated infection. Reported observation and experimental findings carried out in this study have provided evidence that this is an infectious disease, similar to observation of **Kouwenhoven et al. (1978), Angel *et al.*, (1990) and Sell *et al.*, (1992),** although the involvement of non infectious agents has not been completely ruled out according to **Bracewell and Randall, (1984)**, who found that about 60 agent(s) predispose, cause and/or associated with RSS. Several different viral agents have been identified as reovirus, rotavirus, togavirus, calicivirus, parvovirus, FEW virus, corona like particle and adeno virus, several reports indicate that bacteria may play a role as E. coli, Salmonella, Proteus,...etc. Other as dehydration, over heating, irregular temperature during first days of incubation, unbalanced ration, poor sound hygiene measures and immunosuppressive agents in the flock. Because no recognized entropathogen(s) has been consistently incriminated as the etiologic agent for this syndrome, investigators have been searching for viruses and other agents that are associated with this condition of numerous viral agents, **Reynolds, (1997).**

In conclusion, this study indicating that reovirus isolated from field outbreaks is not capable to reproduce RSS experimentally in broiler chickens, but obtained results using infected litter or tissue homogenate could produce the disease condition.

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Table (1): the design of the experimental infection.

Group	Treatment
G 1	Chicken exposed (to contaminated litter)
G 2	Chicken exposed (reovirus infection)
G 3	Chicken infected with (tissue homogenate)
G 4	Control (non treated)

Table (2): Body weight (mean ± S.E.) at 7, 14, 21 and 28 day-old broilers after experimental inoculation.

Group No.	Weights days post inoculation/ gm				Weight loss/gm
	7	14	21	28	
1 X± SE	91.76± 10.1*	254.28± 20.3*	542.31± 28.6***	754.31± 16.7***	484.71
2 X± SE	116.47± 9.8	297.97± 30.5	607.03± 83.1	883.82± 81.6	222.08
3 X± SE	98.13± 8.3*	274.81± 31.6	583.59± 27.3**	803.64± 39.8**	367.20
4 X± SE	124.32± 0.3	312.74± 1.3	694.87± 0.8	995.44± 0.7	-

*: Significant at (P<0.05)
** : Highly significant at (P<0.01)
*** : Very highly significant (P<0.001)

Table (3): Reduction in live body weights.

Group	Reduction in the live weights (X) at days				Comm-ulative mean (X)
	7	14	21	28	
1	26.19%	18.69%	21.95%	24.22%	22.76%
2	6.31%	4.72%	12.64%	11.21%	8.72%
3	21.06%	12.12%	16.01%	19.26%	17.11%

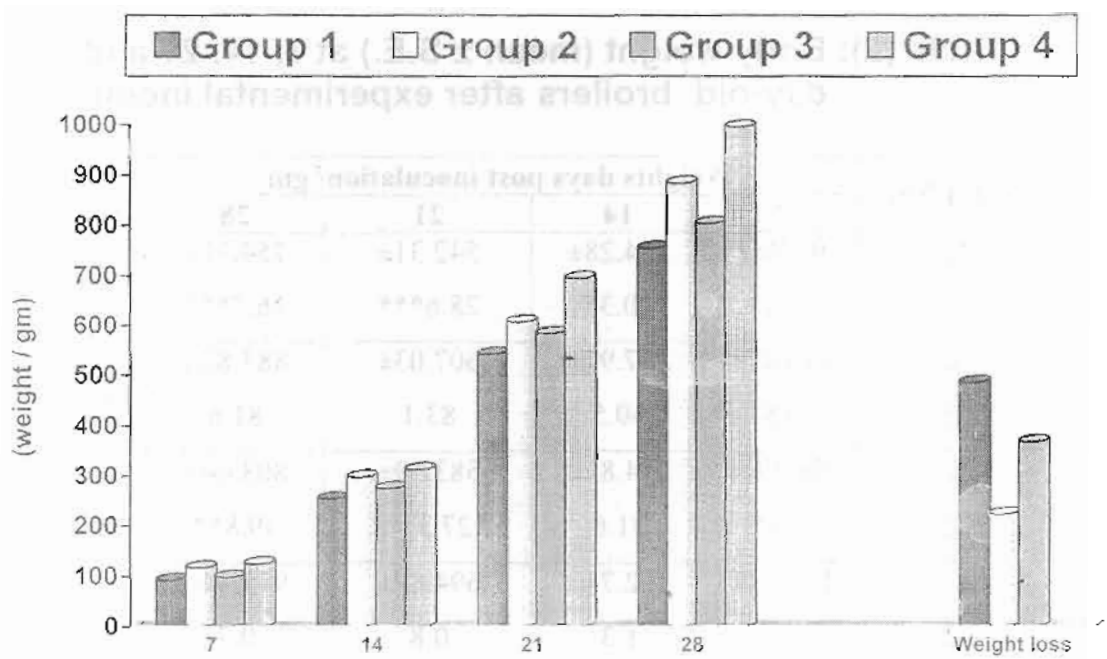

Fig. (1): Body weights at 7, 14, 21 and 28 day-old broilers after experimental inoculation.

Fig. (1): Abnormalities of the primary wing feathers result in a helicopter appearance.

Fig. (2): Intestine is pale and dilated, undigested food is present in the lower bowel.

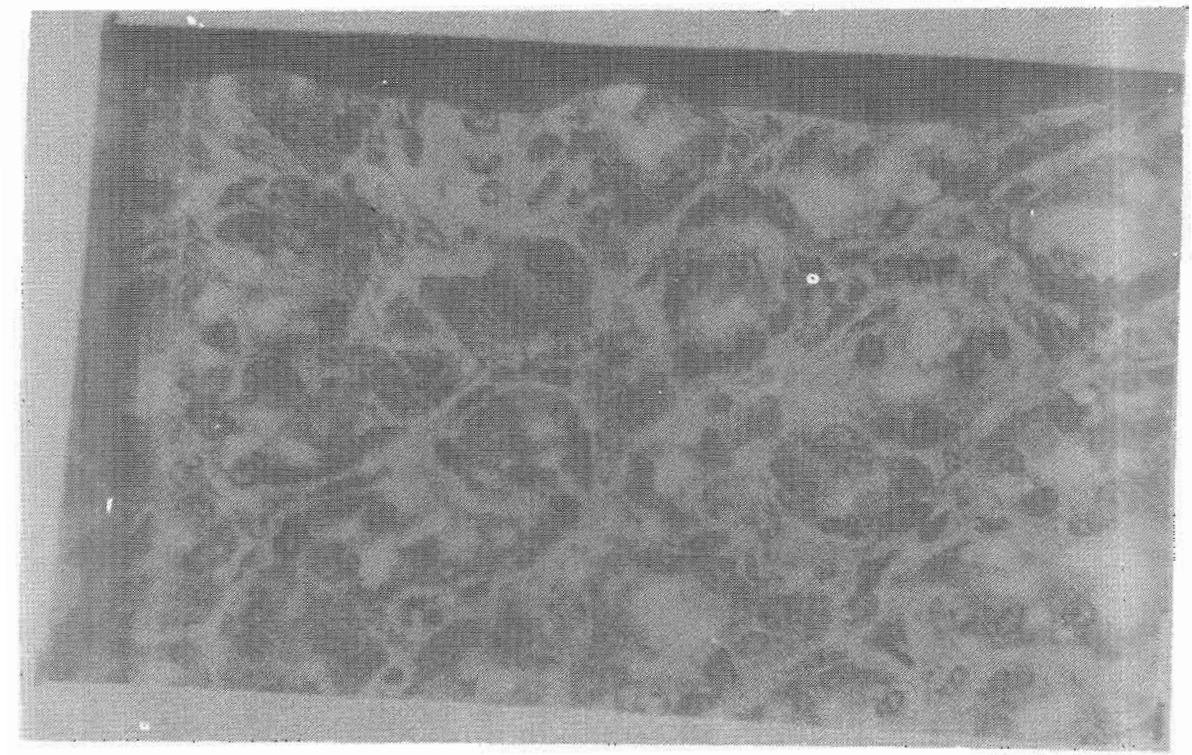

Fig. (3): Histopathological examination of pancreas of RSS infected broiler chicken. There is vaculation of the cytoplasm the acinar cells and few of them contain granules of zymogen (H & E.,).

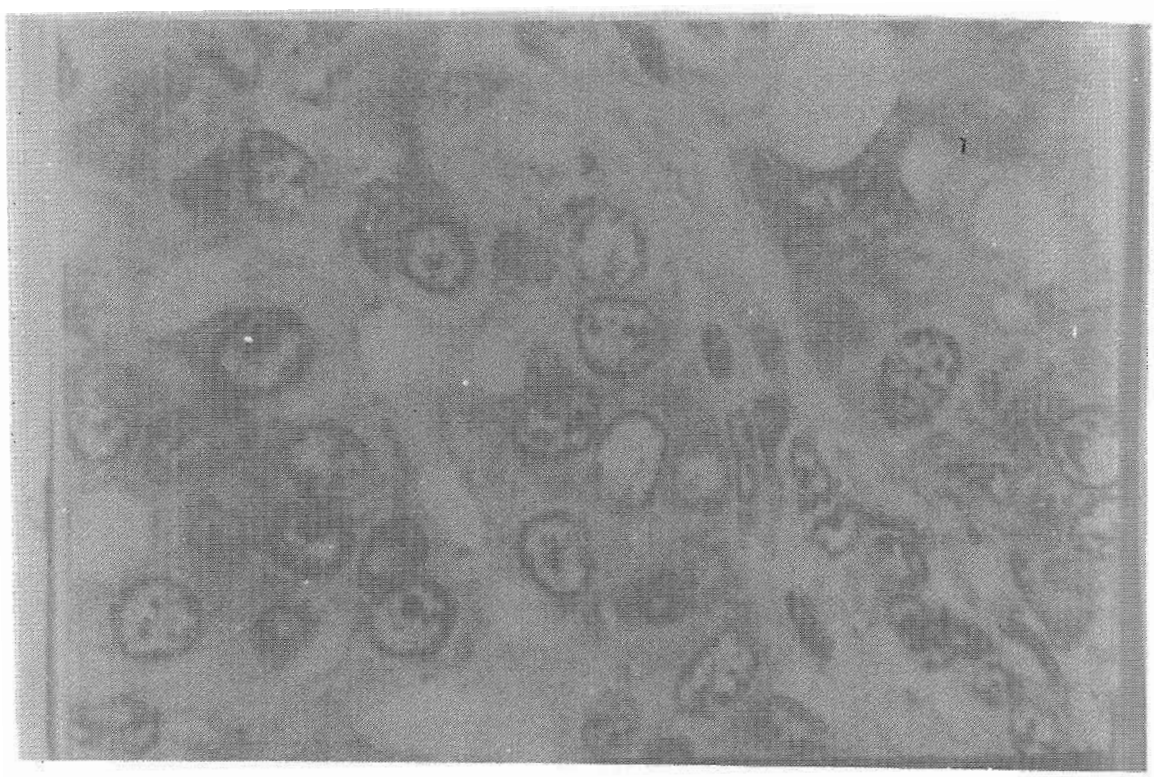

Fig. (4): Histopathological examination of pancreas of RSS infected broiler chicken. The acinar cells of the affected pancreas lose of zymogen granules (H & E).

الملخص العربي

دور فيروس الريو في متلازمة التقزم في دجاج التسمين

عاطف على أبو زيد
وحدة أمراض الدواجن – معمل بحوث صحة الحيوان – الزقازيق

أجرى هذا البحث على قطيع يعانى من التقزم والتهاب بالأمعاء وإسهال وعرج وتم عزل فيروس الريو من هذا القطيع وتصنيفه باستخدام الترسيب فى الأجار وأجريت العدوى الصناعية لبيان أثر فيروس الريو فى احداث هذه المتلازمة وذلك من خلال ثلاث مجموعات كتاكيت عمر يوم واحد ووضعت تحت الملاحظة لمدة ٤ أسابيع.

وجد أن المجموعة الأولى التى وضعت على الفرشة المأخوذة من القطيع المصاب طبيعيا بمتلازمة التقزم أظهرت كافة الأعراض لهذه المتلازمة وأحدثت انخفاض واضح في النمو وكان هذا الانخفاض معنوى فى عمر ٧، ١٤ يوم وازداد هذا الانخفاض ليصبح معنويا وعالي جداً عند ٢١ ، و ٢٨ يوم.

أما المجموعة الثانية التى تم فيها العدوى عن طريق الفم بفيروس الريو (المعزول من القطيع المصاب طبيعيا) فأظهرت تثبيط بسيط وغُير معنوى للنمو على مدار أيام التجربة.

بينما المجموعة الثالثة والتي تعرضت فيها الكتاكيت للعدوى بمحلول نسيج الأمعاء والبنكرياس حدث بها تثبيط متوسط فى معدل النمو والأوزان وكان هذا الانخفاض معنوى عند عمر ٧ يوم ومعنويا وعالي عند عمر ٢١ و ٢٨ يوم.

ومن خلال هذه الدراسة تم عزل فيروس الريو من الدجاج المصاب بمتلازمة التقزم تحت ظروف العدوى الطبيعية الإيجابية. ولم نتمكن من إحداث هذه المتلازمة عند عدوى الدجاج صناعيا بفيروس الريو ولكن النتائج التي حصلنا عليها باستخدام الفرشة أو محلول نسيج الأمعاء والبنكرياس للقطيع المصاب طبيعيا أظهرت أن هناك مسبب أو أسباب معدية أخرى لم نتمكن من معرفتها ولابد من وجودها لظهور متلازمة التقزم المعدى كاملة في الدجاج.