Pancreatitis Associated with Adenovirus Infection in Broilers

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ABSTRACT

Filed cases representative for (5) poultry farms of retarded growth and diarrhea, where examined for detection of the causative agents , Post mortem revealed congestion of the liver and nodular appearance of pancreas ,with pinpoint haemorrhage .

Samples from affected organs were collected and processed to be inoculated into embryonated SPF eggs via yolk sac. Infected embryos showed haemorrhages in the liver, stunted growth, liver discoloration. Yolk of infected eggs was titrated and inoculated into 50 chicks 10-day old, which kept under observation for 3 weeks.

Experimentally and naturally infected chicken exhibited symptoms in the form of depression, diarrhea, ruffled feather, pallor of the comb and wattles with stunted growth. . Gross lesions were in the form of congestion and liver discoloration, also nodular appearance of pancreas with haemorrhage, mortality rate recorded were 4%.

Histopathological changes were in the form of focal to confluent pancreatic necrosis and presence of intranuclear inclusion bodies in the pancreas, intracytoplasmic eosinophilic hyaline bodies in the proventricular mucosa and slight inflammatory reaction in the liver.

Screening of some biochemical parameters showed increase in liver enzyme (AST, ALT) also uric acid and creatinine were elevated, beside alteration in blood glucose level. Hence, our results confirmed the clear association of adenovirus infection with pancreatitis in broilers.

INTRODUCTION

Adenoviruses are common infectious agents in poultry. Most of the viruses replicate in healthy birds with little or no apparent signs of infection, Avian adenoviruses are divided into 3 antigenically different subgroups. The subgroup I, which contain 12 serotypes on the basis of serum neutralization, namely FAV 1-12 .In contrast with the clear association of subgroup II (turkey hemorrhagic enteritis and related viruses), and subgroup III (egg drop syndrome) adenoviruses with diseases, the role of most subgroup I avian adenoviruses as pathogens is not well defined .Many strains rapidly can exploit opportunities when the health of the bird is compromised (e.g., by co infection with other pathogens such as chicken infectious anemia [CIA] or infectious bursal disease [IBD]) *(1)*.

FAVI were isolated from different clinical cases as hydropericardium syndrome serotype 4. The transmission of disease occurs laterally by the feco-oral route. The livers of affected birds showed necrotic foci, and basophilic intranuclear inclusion bodies fill the entire enlarged nucleus *(2)*.

Digestive system signs and lesions often are observed in broiler chickens submitted for laboratory evaluation because of impaired weight gain and feed conversion, and watery faeces.

Various lesions have been identified in the proventriculus, gizzard, intestines, pancreas, and liver in the course of these investigations. Adenoviruses and reoviruses are commonly isolated from the affected viscera, particularly from the proventriculus and duodenum in birds with lesions in these organs. Marked pancreatic and ventricular lesion were recorded after experimental infection while these lesion varied from mild to moderate in proventriculus and bursa with no lesion at all in the intestine *(3)*.

Adenoviruses have been associated with spontaneous erosive ventriculitis (gizzard erosions) in Leghorn and meat-type chickens and in quail. *(4 ,5)* Gizzard erosions and ulcers

and necrotizing pancreatitis occurred in SPF chickens inoculated with a type 8 avian adenovirus *(6).*

Necrotizing pancreatitis was observed in adenovirus-inoculated chickens, and the intranuclear inclusion bodies indicated a causal relationship. This lesion was identified only in the occasional case of fully developed inclusion body hepatitis, as has been reported in association with hypoglycemia and high mortality *(7).*

Viral DNA replicates in nucleus of the cell, then the viron are assembled in the nucleus where they form crystalline aggregates which can be seen by light microscope as intra nuclear inclusion bodies , then the viron are released via cell lysis *(8).*

Variation in the pathogencity of group I different serotypes and also within the same serotype, this depends on both virus and the host (age at infection, maternal immunity ,concurrent infection and previous infection which affect susceptibility) *(1).*

Propagation of FAV groupI was successful in embryonating eggs via chorioallantoic, yolk sac route or cell culture. Yolk sac culture was superior to chorioallantoic in virus yield and is reliable alternative to cell culture to all type I adenoviruses. Inoculating embryonating eggs showed stunting, curling, hepatitis, splenomegaly, congestion, hemorrhages in different organs and urate formation in the ureters *(9).*

Symptoms and mortality rate and gross lesion are varied among serotypes of FAV group I, either under natural or experimental infection. Symptoms were in the form of pallor of the comb and wattles, ruffled feathers, depression of the body weight, tremors .While mortality may vary from 0% and may reach up to 60% *(10-13).* Gross lesions were in the form of haemorrhagic liver, enlarged pale kidneys, haemorrhage in skeletal muscle, atrophied spleen, hydropericardium, necrotizing pancreatitis, haemorrhage in pancreas *(3,10,13-15).*

Liver functions are greatly affected by the infection of FAV I which lead to increase in the level of either GPT , GOT and decrease in the level of albumin , total protein *(16,17)* , in the same time hypoglycemia were noticed *(4).*

The aim of this work was the isolation and identification of suspected viral etiology and its related pathological changes.

MATERIAL AND METHODS

Specimens: A total number of 5 broiler farms at 3-5 weeks old in Kafr elsheikh governorate were investigated during period extending from February to November 2009.This flocks had history of infectious bursal disease (IBD) infection, the birds exhibiting digestive signs and general signs of illness with few mortalities. Postmortem examination was conducted and recorded, pancreas from 5-7 freshly dead birds per farm was collected and processed under aseptic condition for viral isolation trials.

Virus isolation : SPF embryonated chicken eggs were obtained from Serum and Vaccine Research Institute , Abbasia, Egypt , they were used for virus isolation and propagation via yolk sac inoculation with suspected materials prepared from infected pancreas and antibiotic treated (pooled sample from each farm). Three blind egg passages were carried out before titration and identification of isolated virus. *(18)* . Virus titration was carried out *(19).*

Sterility test for bacterial contaminants :Small amount of collected supernatant fluids were inoculated into a set of media including: nutrient agar, MacConkey agar, blood agar and tryptic soy agar in order to exclude bacterial contamination.

Virus haemagglutination (HA) :HA has been previously reviewed *(20).*

FAV-1 virus haemagglutinates rat erythrocytes(1%). Optimal agglutination of erythrocytes occurs between pH 6 and 9 at temperatures between 20° and 45°C.

Experimental infection: It was carried out to study the pattern of infection with isolated adenovirus in 10 days age chicken *(3).* Fifity chicken (Hubbard) were injected intramuscularly with 0.5 ml of infective yolk containing 10^4

EID_{50} / 0.1ml. Chicken were kept under observation for 3 weeks. Twenty chicks were kept as control, control birds were injected with 0.5 ml of sterile yolk and kept in far place from the infected one. Chicken were kept under observation for 3 weeks. Feed and water were offered ad lib. Routine vaccination program was applied to all birds.

Biochemical parameters: Serum were collected from 5 birds of the infected and control group at 14 days post infection for measuring total protein *(21)*, albumin *(22)*,aspartate amino transferase (AST) and alanine amino transferase (ALT) *(23)*. Creatinine *(24)*, uric acid *(25)* ,and glucose *(26)* using diagnostic kits Bio-Merieux , France.

Histopathological examination:

Samples from pancreas, Proventriculus and liver were collected from natural and experimental infected chicken and fixed in 10% neutral buffered formalin. Then, the samples were dehydrated in ascending grades of alcohols, cleared in xylem, embedded in paraffin wax, sectioned at 5µm and stained with hematoxylin and eosin (H&E) and then examined by the light microscope *(27)*.

Data obtained in this study representing the different variables were statistically analyzed *(28)*.

RESULTS AND DISCUSSION

Adenovirus have a wide spread occurrence in many avian species as primary or secondary pathogens causing many disease problems and economic losses by poor feed conversion with reduced weight gains and condemnation of carcass. Moreover, it may be involved in immunosuppression through lymphoid depletion leading to increase susceptibility to various infectious agents and interfere with efficacy of vaccination programs *(29)*

Five broiler flocks with capacity varying from 5000 to 10000 chick aged 3-5 weeks had history of IBD were extensively examined , chicken exhibiting depression, inappetance, pale comb and wattles, ruffled feathers ,diarrhea and retarded growth with low mortalities (2-4 %) .

The most consistent post mortem lesions were confined to the pancreas ,which severely enlarged with nodular appearance, oedematous , pin point haemorrhages and many focal areas of pancreatitis (Fig. 1). Liver in most cases was enlarged, congested and friable. Spleen and kidneys were congested and swollen in majority of chicken. Catarrhal enteritis in many birds was recorded. Occasionally, minute clear straw coloured watery fluid in the pericardial sac was observed.

The inoculated SPF chicken embryonated eggs via yolk sac route , revealed deaths of the embryos at 5 days post inoculation with death rate at 20% , while, the most prominent lesions were stunting, curling, hepatitis, liver discoloration, splenomegaly, congestion and hemorrhage of internal organs, with urate accumulations in the kidneys(Fig.2).Similar lesions reported by *(9)*.

The isolated virus agglutinates rat washed RBCs(1%) within one minute at room temperature and the titer of isolated virus was 104Eid50/0.1ml.

Although it is assumed that all FAV groups 1 multiply in the embryonated eggs, not all chicken isolates cause recognizable lesions. The chorioallantoic membrane route of inoculation was found to be more sensitive for virus isolation than the allantoic cavity *(30)*. Most adenovirus isolates made in eggs have been typed as FAV-1 or 5, *(31)*. However, inoculation into the yolk sac, and to a lesser degree into the chorioallantoic membrane, supported the growth of 11 recognized serotypes *(9)*. In the present work the suspected adenovirus grows well in yolk sac .Yolk from inoculated eggs was capable to agglutinate rat RBCs and not agglutinate chicken RBCs. Similar result was previously recorded *(20)*.

Symptoms in experimentally infected chicken at 2 weeks post infection were in form of retarded growth, off food, ruffled feathers, pallor of the comb and wattles , tendency to recumbent (Fig.3). The same symptoms were observed *(32)*. While the mortality recorded were 4 % (2 out of 50 chicks) and are restricted to the 1st week post infection . Several workers

reported different mortality rate varied from 0 % to 60 % based on immune status of infected flock *(10-13).*

Post mortem lesions was in the form of foamy caecal contents, some chickens had diffusely pale green livers with scattered small irregular yellow foci, haemorrhagic liver, enlarged pale kidneys, atrophied spleen , haemorrhagic and focal pancreatitis, yellow necrotic foci occurred in the mucosa of the Proventriculus (Fig.4) .The gizzard lining was rough and sloughed ,more or less similar gross lesions were recorded by other workers *(3,10,13,14,15).* Liver and pancreatic lesions have been successful induced by other workers following parenteral inoculation in young chick *(6,33,34).*

Histopathology:

Marked to severe histological lesions occurred in the gizzard and pancreas, but only mild lesions occurred in the proventriculus.

Pancreas: mild multifocal to massive confluent intrapancrestic necrosis was observed in the pancreas of the affected birds. Mild pancreatic necrosis in the form of disappearance of small areas of pancreatic parenchyma with the presence of remnants of pancreatic acinii invaded by slight inflammatory cell infiltrate and surrounded with sound and intact acini was noticed (Fig. 5) while there were complete absence of pancreatic acini which in some cases involved a whole lobe (Fig. 6-8). The massive pancreatic necrosis had acinar cell degeneration; interstitial tissues were expanded by dense infilrates of histocytes, lymphocytes, and plasma cells and necrosis in association with intranuclear inclusion (Fig. 9&10) *(35).* Degenerative changes were represented by mild vacuolar degeneration of the acinar cell cytoplasm (Fig. 11). Some inflammatory reactions were observed in the affected pancreas; majority of them were perivascular mononuclear infiltration (Fig. 12).

Proventriculus: The proventriculus some area chronic inflammatory reaction with lymphoplasmacytic infiltrates in the lamina propria and submucosa, and hyperplasia in the mucosal lining of the gland (Fig. 13). Eosinophilic intracytoplasmic hyaline bidies were observed in the epithelial cells (Fig. 14). Multifocal mucosal epithelium hyperplasia and microcyst formation in the mucosal lining epithelium of the Proventriculus were noticed (Fig. 15). *(36).*

Liver: Only few inflammatory reactions were noticed in the liver in the form of perivascular mononuclear cell infiltrates (Fig. 16) or focal granuloma like reaction (Fig. 17) *(35).*

Biochemical parameters:

Biochemical parameters measured in the serum revealed an increase in the level of AST which appear to be significant either in experimentally or naturally infected chicken in comparison to control. While ALT show significance between naturally infected group and each of experimentally infected and control one (Table ,1), 12.8±0.73 vs 10.6 ± 0.6 and 8.8±0.58 respectively, In the same time the albumin production by the liver was impaired where there was significant difference between control group and infected groups either naturally or experimentally. Also total protein and renal function showing significant differences between infected groups and control one which may be attributed to the affection on the kidney or decreased feed intake as documented during experimental infection , all these findings are more or less agree with the findings reported during previous studies *(16,17,37).*

Serum glucose in the present work was significantly increased in naturally infected group than that recorded in each of experimental and control one, 281.4±3.7 vs 221.8±2.2 and 224.2±4.8 respectively this may be attributed to degradation of the acinar lining cell of pancreas or hepatic insufficiency result from hepatic pathology ,*(7,38).*

Table 1. Biochemical parameters in naturally and experimentally infected chicks with adenovirus ±SE

group	AST	ALT	Glu	T.P	ALB	U.A	Crn
Nat.infection	85.2±1.4^{c}	12.8±0.73^{b}	281.4±3.7^{b}	2.95±0.22^{b}	1.71±0.03^{b}	12.65±0.33^{c}	1.37±0.04^{b}
Exp.infection	70.2±1.7^{b}	10.6±0.6^{a}	221.8±2.2^{a}	2.47±0.15^{c}	1.65±0.07^{b}	6.98±0.8^{b}	1.2±0.08^{b}
control	44.6±1.2^{a}	8.8±0.58^{a}	224.2±4.8^{a}	3.39±0.11^{a}	1.94±0.04^{a}	5.05±0.57^{a}	0.99±0.06^{a}

In the same column means followed by the same letter are not significantly different at 0.01 level of significance.

In the present study, it could be concluded that, the clinical signs, characteristic lesions of pancreas of either naturally or experimentally infected broilers, isolation of the viral agent in embryonated SPF eggs, pathogencity tests and histopathological findings particularly basophilic intranuclear inclusion bodies in pancreatic acinii, confirm the clear association of adenovirus infection with pancreatitis in broilers .

FIGURE LEGEND

Fig. 1.Photomicrograph of pancreas showing nodular appearance with focal haemorrhage.

Fig. 2.Photomicrograph of embryonated chick showing discolored liver with stunted growth.

Fig.3Photomicrograph of stunted growth in the experimentally infected chicks (central) comparing with the normal control (right).

Fig. 4.Photomicrograph of proventriculus showing erosins.

Fig. 5.Photomicrograph of pancreas showing focal necrosis. Disappearance of small areas of pancreatic parenchyma with the presence of remnants of pancreatic acini invaded by slight inflammatory cell infiltrate and surrounded with sound and intact acini (H&E; X100)

Fig.6. Photomicrograph of pancreas showing massive necrosis involving a whole lobe (H&E; X25)

Fig.7. Photomicrograph of pancreas showing massive necrosis involving a whole lobe (H&E; X50)

Fig.8. Photomicrograph of pancreas showing massive necrosis leaving remnants of cytoplasm with infiltration of inflammatory cells (H&E; X100).

Fig.9.Photomicrograph of pancreas showing intranuclear inclusion bodies (H&E; X400).

Fig.10.Photomicrograph of pancreas showing intranuclear inclusion bodies (H&E; X400)..

Fig.11.Photomicrograph of pancreas showing degenerative changes were represented by mild vacuolar degeneration of the acinar cell cytoplasm (H&E; X100).

Fig.12.Photomicrograph of pancreas showing inflammatory reactions. Perivascular mononuclear cell infiltration (H&E, X200).

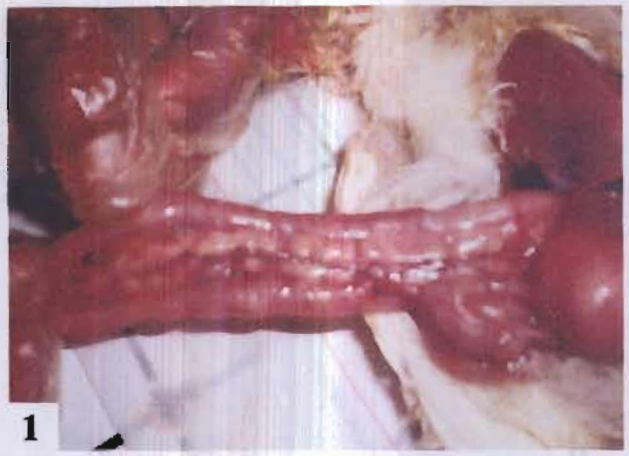
Fig.13.Photomicrograph of proventriculus showing chronic inflammatory reaction with lymphoplasmacytic infiltrates in the lamina propria and submucosa, and hyperplasia in the mucosal lining of the gland (H&E, X200).

Fig.14.Photomicrograph of proventriculus showing eosinophilic intracytoplasmic hyaline bodies in the epithelial cells (H&E, X400).

Fig.15.Photomicrograph of liver showing perivascular mononuclear cell infiltrates (H&E, X100).

Fig.16.Photomicrograph of liver showing perivascular mononuclear cell infiltrates (H&E, X100).

Fig.17.Photomicrograph of liver showing focal granuloma like reaction (H&E, X100).



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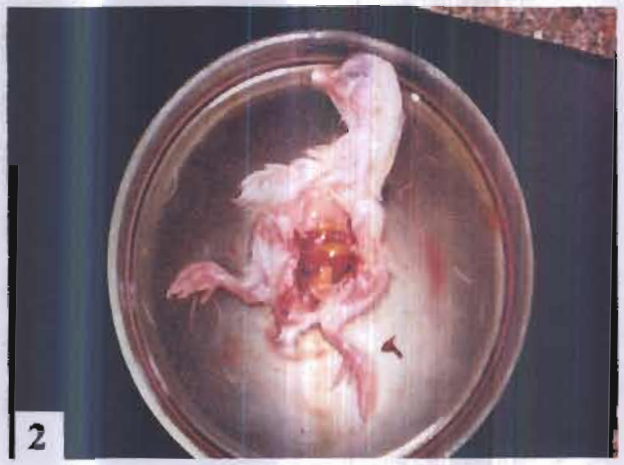

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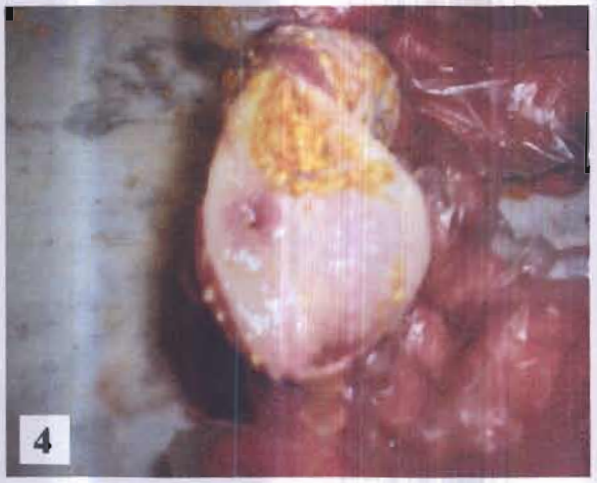

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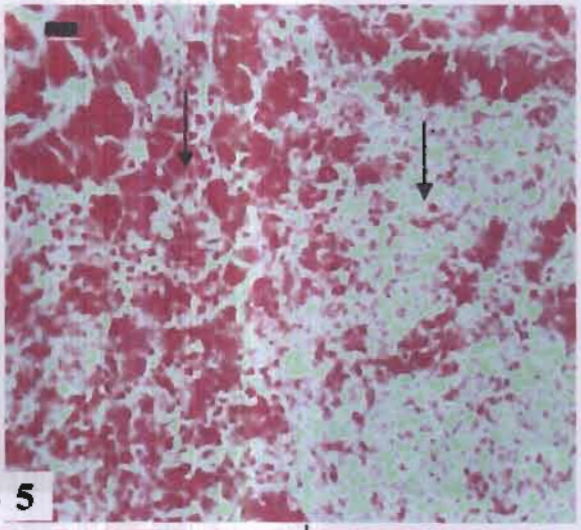

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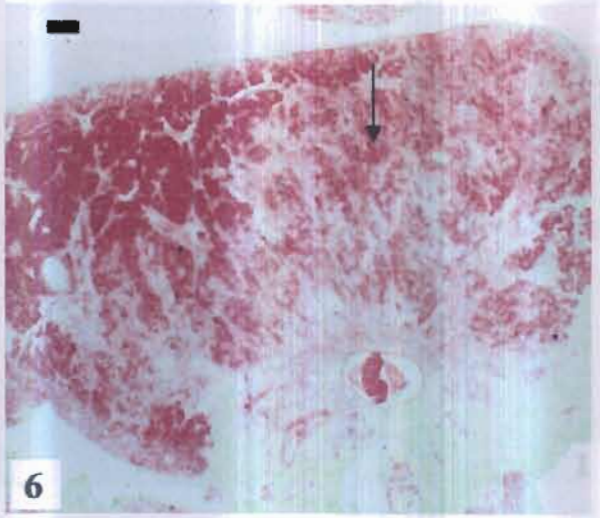

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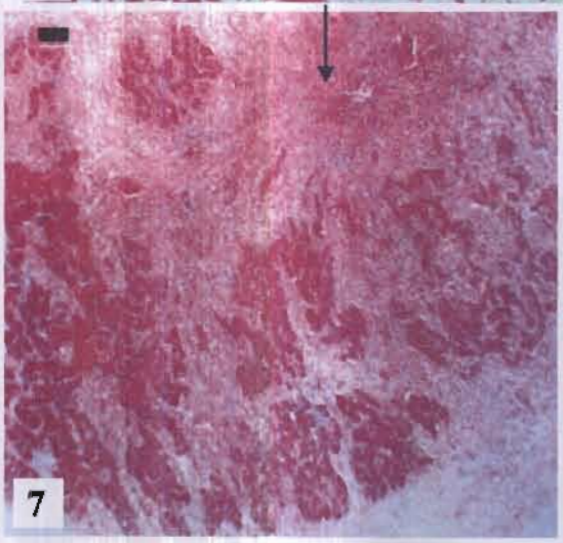

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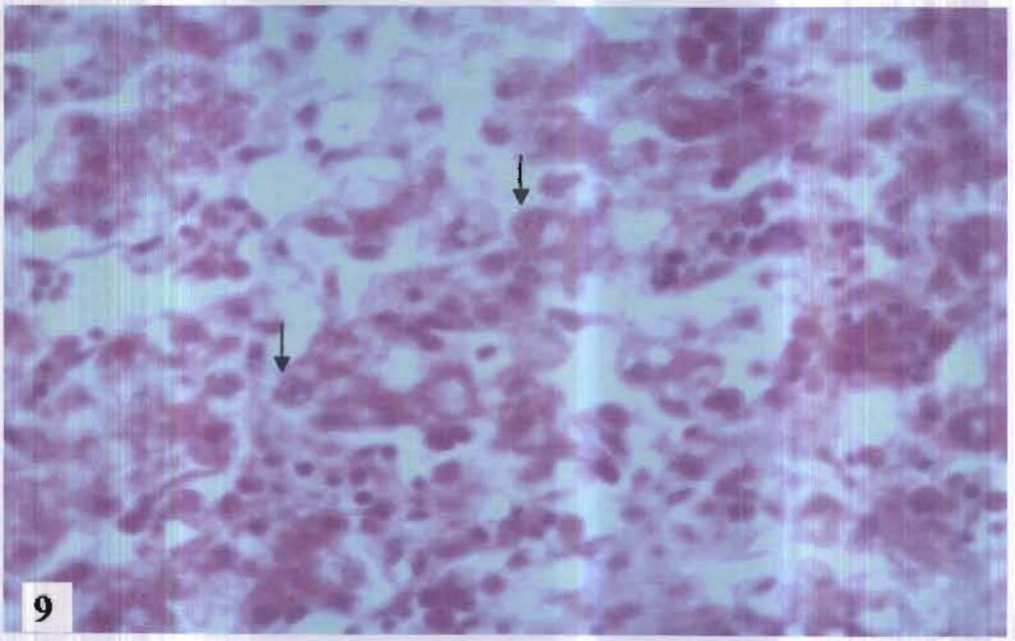

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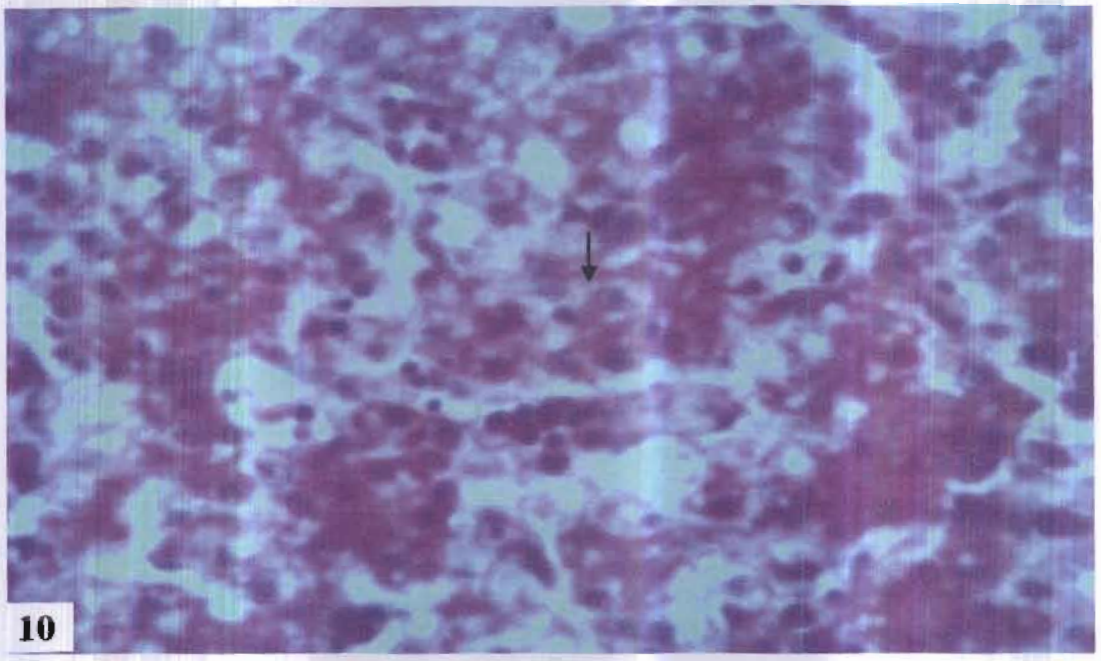

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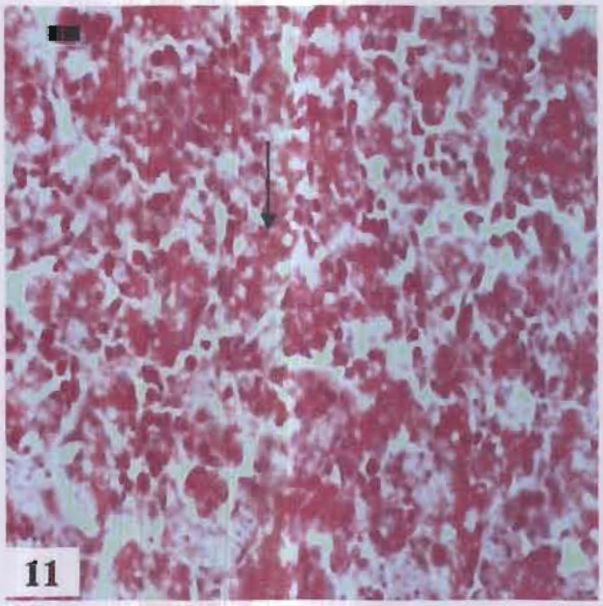

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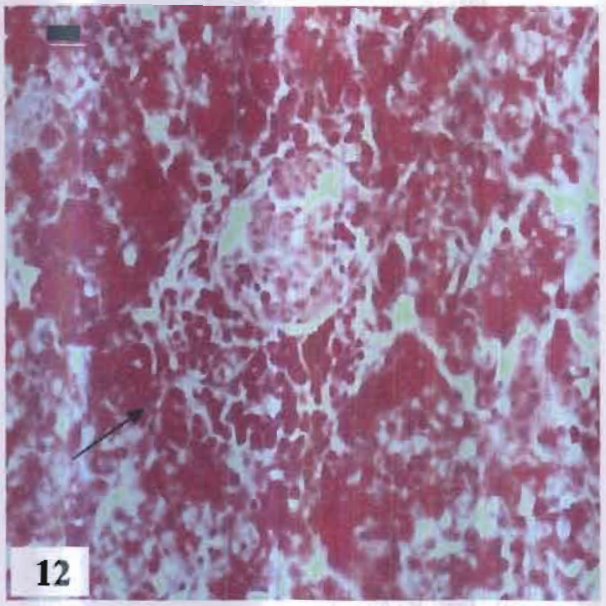

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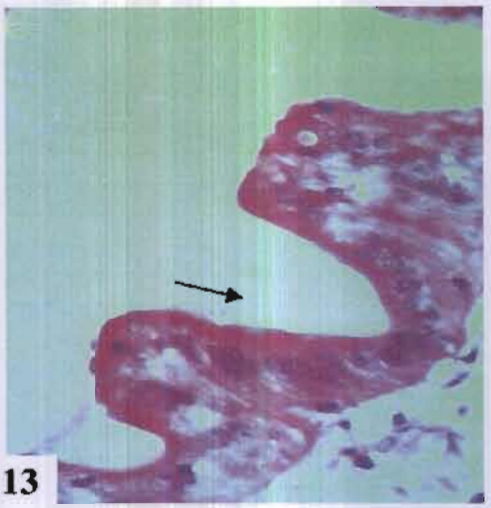

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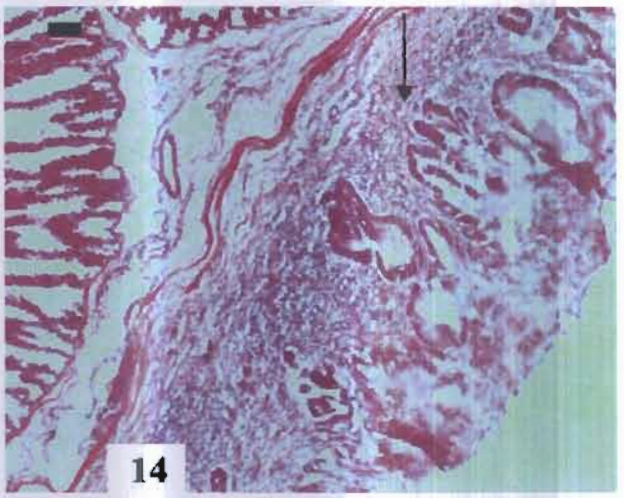

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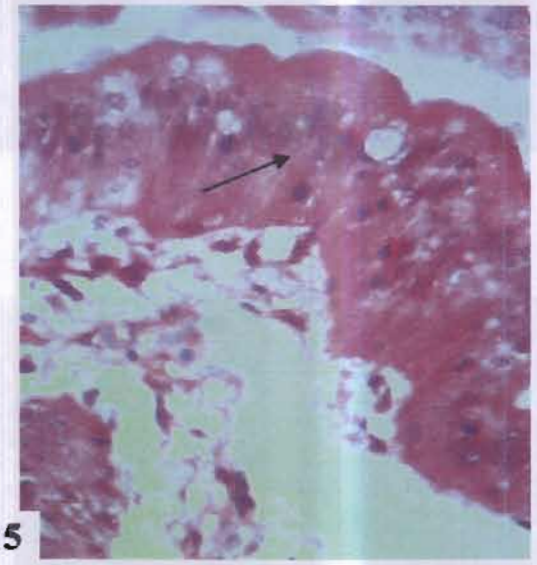

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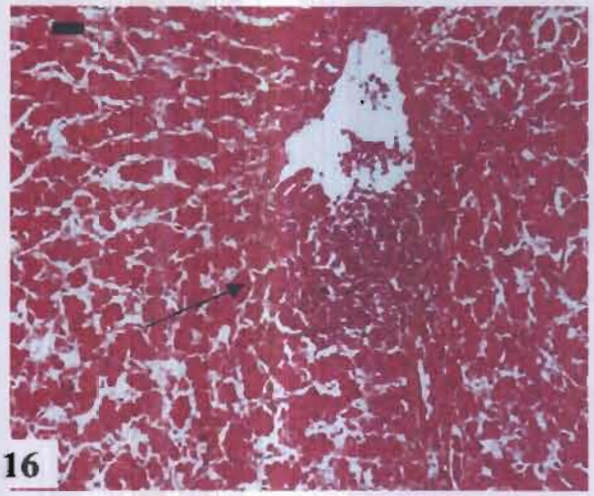

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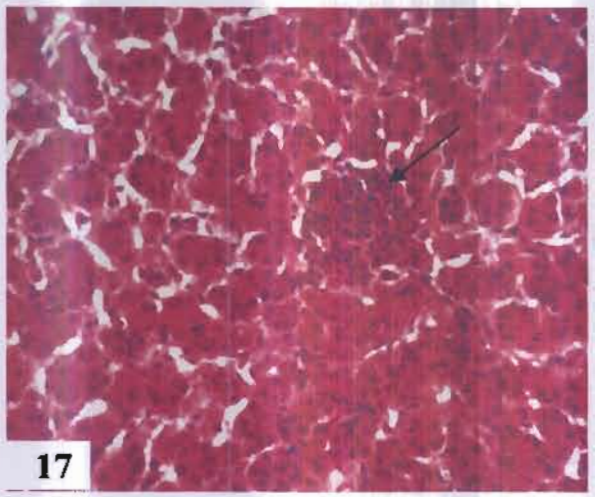

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الملخـــص العربـــي

التهاب البنكرياس المصاحب لعدوى فيروس الأدينو في دجاج التسمين

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ظهرت بعض الحالات الحقلية التي تعاني من انخفاض في الوزن و إسهال و انتفاش الريش و تم فحص هذه الحالات لتحديد المسبب . و قد أظهرت الصفة التشريحية احتقان في الكبد مع وجود بعض الأنزفة و احتقان في الطحال و أنزفه و احتقان و تدرن عقدي بالبنكرياس و قد أظهر العزل البكتيريولجي من هذه الأعضاء خلوها من البكتريا ٠ تم تجميع عينات من الأعضاء المصابة للحقن في البيض عن طريق كيس المح في البيض المخصب . الأجنة المحقونة أظهرت تأخر في النمو (تقزم) , أنزفه في الكبد مع تغير اللون.

السوائل التي تم تجميعها كانت قادرة علي تلازن ١ % من كرات الدم الحمراء المحضرة من الفئران الصفار المعدي تم معايرته و تحديد EID_{50} ٠

تم حقن ٥٠ كتكوت عمر ١٠ أيام بواسطة المح المحتوي علي الفيروس بعياريه 10^{-4} بجرعة ٥, مللي لكل كتكوت وظهرت الأعراض علي الكتاكيت في خلال ٤٨ ساعة و كانت في شكل إسهالات و انتفاش في الريش ثم انخفاض في الأوزان و كانت الصفة التشريحية في شكل احتقان و تغير في لون الكبد و أنزفه و تدرن عقدي في البنكرياس ٠ و كان معدل الوفيات ٤ % ٠

ظهرت تغيرات هيستو باثولوجية مطابقة لعدوى فيروس الأدينو و بإجراء بعض القياسات البيو كيميائية في مصل الدم لوحظ ارتفاع في مستوي إنزيمات الكبد و حامض البوليك و الكرياتينين إلي جانب مستوي جلوكوز الدم ٠