Animal Health Research Institute
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CLINICOPATHOLOGICAL STUDY ON AN OUTBREAK OF INCLUSION BODY HEPATITIS IN BROILER CHICKEN

(With 15 Figures)

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دراسة إكلينيكية وباثو لوجيه على الالتهاب الكبدي الوبائي في دجاج التسمين

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أوضحت هذه الدراسة الباثولوجية الإصابة بالالتهاب الكبدي الوبائي في بعض مزارع دجاج التسمين في محافظة أسيوط. وكانت التغيرات المرضية في الكبد واضحة ومميزة لهذا المرض. إلي جانب التغيرات الباثولوجية التي أثبتت الإصابة بالالتهاب الكبدي كانت الصفة المميزة لهذا المرض هو ظهور الأجسام المحتواة داخل النواة في خلايا الكبد والبنكرياس في حوالى ٨٠% في الحالات التي تم فحصها.

SUMMARY

Outbreaks of inclusion body hepatitis were diagnosed and proved histopathologically in broilers at Assuit Governorate. The hepatopathic changes were the constant findings. In addition, the angiopathic alteration were prominent in most cases. The pathognomonic intranuclear inclusions could be seen in 80% of the examined cases.

***Key words:** Inclusion body, Hepatitis, Broiler chicken.*

INTRODUCTION

Inclusion body hepatitis was described and aetiologically diagnosed in chicken by Helmboldt and Frazier (1963) and McFerran *et al.* (1977). It has been reported by several authors in a wide range of birds (Sileo *et al.*, 1983; Coussement *et al.*, 1984; Saik *et al.*, 1986; Pass, 1987; Goryo *et al.*, 1988; and Capua *et al.*, 1995). The disease was also described at El-Waady El-Gadeed Governorate by Mousa *et al.* (1984).

Adenoviruses were isolated and characterized from chickens, pigeons and psittacins birds (Rosenberger *et al.*, 1975; Gomez-Villamandos *et al.*, 1992 and Capua *et al.*, 1995).

The disease usually affect immunosuppressed birds. It was reported in previously infected birds with infectious bursal diseases and or infectious aplastic anemia (Fadly *et al.*, 1976; and Toro *et al.*, 1999).

The gross and histopathologic findings in naturally affected as well as experimentally infected birds included hepatopathic changes associated with intranuclar inclusion bodies (Itakura *et al.*, 1977; Mousa *et al.*, 1984; Pass, 1987 and Weissenbock and Fuchs 1995 *et al.*, 1995 and Tsai *et al.*, 1998).

The aim of the present study is to describe the characteristic morphological changes of naturally affected broilers with Suspected inclusion body hepatitis.

MATERIALS and METHODS

Dead broiler chicks ranging from 3-5 weeks of age were brought to the animal health institute, Assuit regional Laboratory. Examination of the sick birds in three private farms (6000-9000 chicks each) was carried out. The morbidity and mortality rate were calculated and careful post-mortum technique was done on both freshly dead and scarified birds.

For histopathological Examination, samples from liver, kidney, heart, spleen, lung, pancreas and bursa were taken. Fixed in 10% neutral buffered formalin prepared for routine hematoxylin eosin staining and examined.

RESULTS

Clinical symptoms:

In the three examined farms the morbidity rate reached 28% and the mortality rate was up to 2%. The sick birds revealed restlessness, depression, dyspenia, yellowish green liquid diarrhea, twisted necks and great lendency for sitting on their hocks and huddle together.

Post mortem examination:

The most morphological changes of the dead cases revealed diffuse and severe muscular haemarrhages.

The other gross lesion was mainly restricted to livers. They were enlarged showing focal or diffuse pale yellowish white discoloration with rounded border. The cut section of the liver was either with blood

oozing or dry, Ecchymosis could be distinctly seen one some liver surfaces.

Histopathological findings:

The microscopic examination of the liver revealed the same principal alternative changes associated with acute inflammatory reactions in all examined cases. Although, some variation in the degree of severity were noticed.

The alternative changes included hepatocytic degenerative changes and vacuolation such changes reached up to coagulative necrosis (Fig. 1, 2 and 3). The hepatocytes were relatively swollen with increase acidophelia of the cytoplasm and pyknotic or even karyolytic nuclei (Fig. 4 and 5).

In the most examined cases the liver revealed marked infiltration and displacement of hepatic parenchyma by a of lymphoid cells infiltration with peripheral hetrophilic infiltration in the portal area. (Fig. 5 and 6).

Examination of the hepatic cells revealed two forms of intranuclear inclusions. The majority of the inclusions internuclear bodies appeared as distinct eosinophilic varying in shape and size, they are round, angular or rod shape. They were scattered throughout the karyoplasm. They appeared as a totally homogenous mass with a hallow zone surrounded all of these type of inclusions (Fig.7 and 8).

The second form of the inclusion bodies were basophilic which disseminated randomly throughout the hepatic cells. They occupied the entire hepatocyte and cytoplasm seemed to be completely absent in many cases.

Histopathological examination of the pancreas showed damage with loss of pancreatic acini. In some cases, acinar epithelium showed cytoplasmic cell vaculation (Fig.11 and 12).

Numerous focal acinar necrosis infiltrated with pleomorphic inflammatory cells, were seen (Fig. 13).

The epithelium of the pancreatic duct epithelium showed hyperplasia and subepithelial eodema (Fig. 14).

Inclusion bodies were observed in the pancreatic acinar epithelium. They varied in shape, size and degree of eosinophilia (Fig.15 a,b,c and d).

DISCUSSION

Inclusion body hepatitis disease appears to have become more important since 1992 and its spread is indicated by the occurrence of some new outbreaks, sudden death increased mortality and presence of diffuse and sever muscular heamorhges are the elements which usually suggest the field occurrence of this disease (Massi *et al.*, 1995). The present study was done to describe the characteristic morphological changes of naturally affected broiler. In our finding in addition to sever muscular hemorrhage, ecchymotic area of hemorrhage could be distinctly seen on liver surface.

As it is reported by (Goodwin *et al.*, 1996). In this study the outbreak among chicks with inclusion body hepatitis is unusual in young chicks. In the three examined farms mortality rate was up to 2% but mortality rate reached 28%. Similar result reported by Hunter *et al.*, 1979; Lowinstin and Fry, 1985; Mori *et al.*, 1989; El-Attrache and Villegas, 2001, which stated that adenovirus inclusion bodies are often found in healthy birds, and appear to be less significant as a cause of illness or death. On the other hand, Tasai *et al.*, (1994) observed a high mortality rate of inclusion body hepatities.

Histopathology is valuable for making a definitive diagnosis of many viral disease of the liver. This is particularly true of those in which viral inclusions are a diagnostic feature such as herpes virus adenovirus paravirus and (Weissenbock and Fuchs, 1995). In our result, histopathological lesion observed in the liver chicken revealed the same principal alternative changes associated with acute inflammatory reaction. The alternative changes included hepatocytic degenerative changes and vacuolation. Such changes reach up to coagulative nccrosis. Similar findings were recorded by Massi *et al.*, 1995, who observed that the extensive hepatic necrosis is an important characteristic features of the disease.

Histopathological lesions observed in the liver of chicks which died from fatal necroses were similar to those described by Vindevogel and Pastroet, (1981) and Toro *et al.* (1999) in racing pigeons infected with adenovirus.

The presence of nuclear inclusion bodies in hepatocyte due to adenovirus in pigeon was described by (Goryo *et al.*, 1988) who found that eosinophilic and basophilic bodies surrounded by a halo. Goodwin and Davis (1992) and Ketterer *et al.* (1992) described the inclusions as

large basophilic bodies occupying the entire nucleus where Coussement *et al.* (1984) found only eosinophilic inclusion, in our investigation revealed large eosinophilic inclusion bodies and basophilic inclusion bodies were detected. These were agreement with those described by Weissenbock and Fuchs (1995) observed all type similar to those of IBH avian species (McFerrer and Adair, 1977; Winterfield 1984; Goryo *et al.*, 1988).

As it is reported by Gallina *et al.* (1973), necroses of hepatocyte, and exocrine pancreatic acinar cells was accompanied by nuclear enlargement with margination chromatin.

The adenovirus caused massive destruction of the exocrine part of the pancreas (Goodwin *et al.*, 1996). Necrotizing pancreatitis and gizzard erosion have been reported in chickens associated with adenovirus infection in which large basophilic IBH were seen in the necrotic pancreatic acinar cells and necrotic epithelial cell of gizzard (Okud *et al.*, 2001) but Tsai *et al.* (1998) observed that the affected glandular epithelium of the gizzard without any associated changes in the pancreas. In the present study. The affected chicks has inclusion bodies in the pancreatic acinar epithelial cells.

CONCLUSION

Inclusion body hepatitis may occur as a secondary infection to other disease, and may cause immunosuppression, because adenoviruses are widely spread in nature. Through cleaning, disinfection of farms, equipments hatcheries are of high importance in control spread of inclusion body hepatitis.

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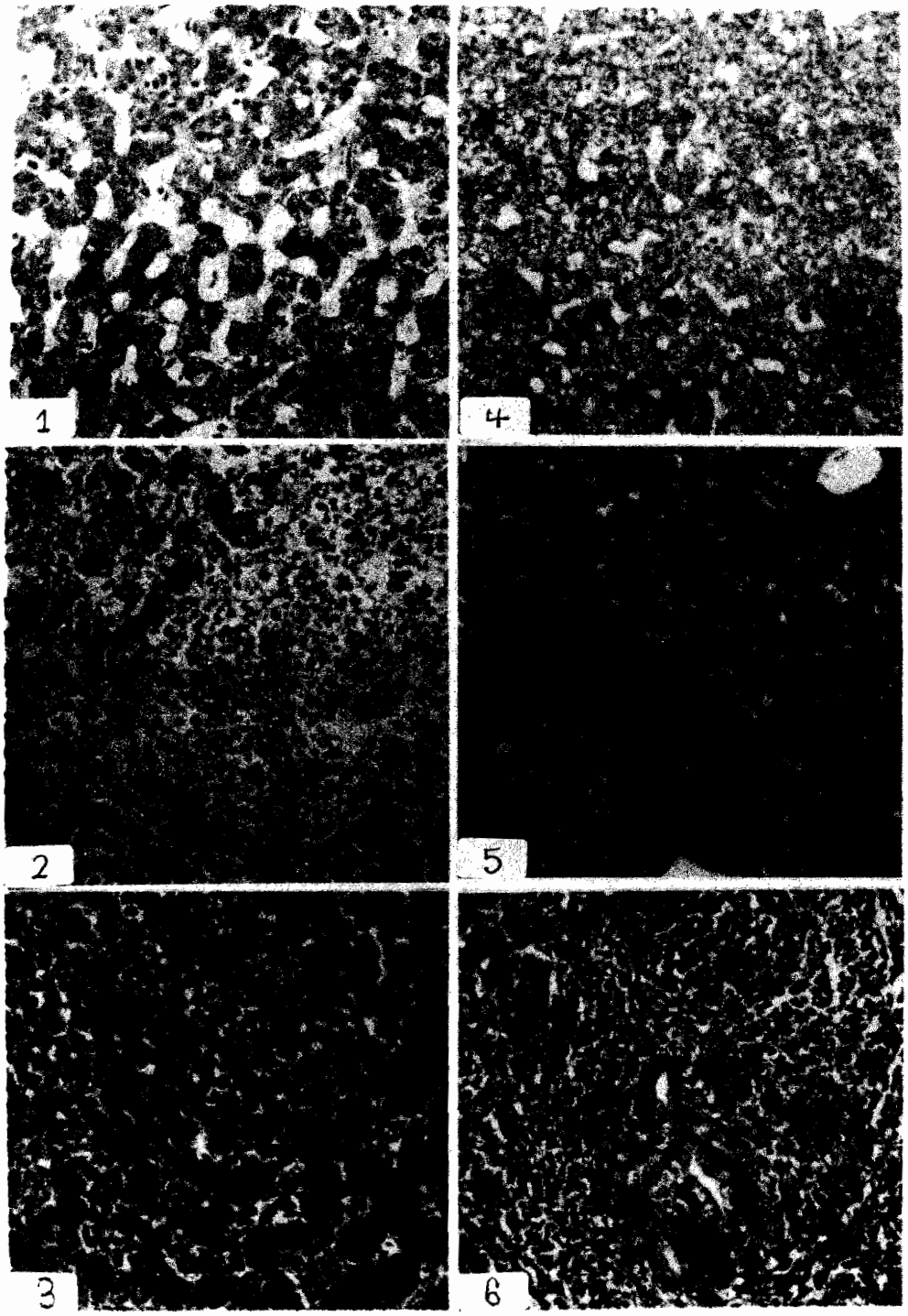
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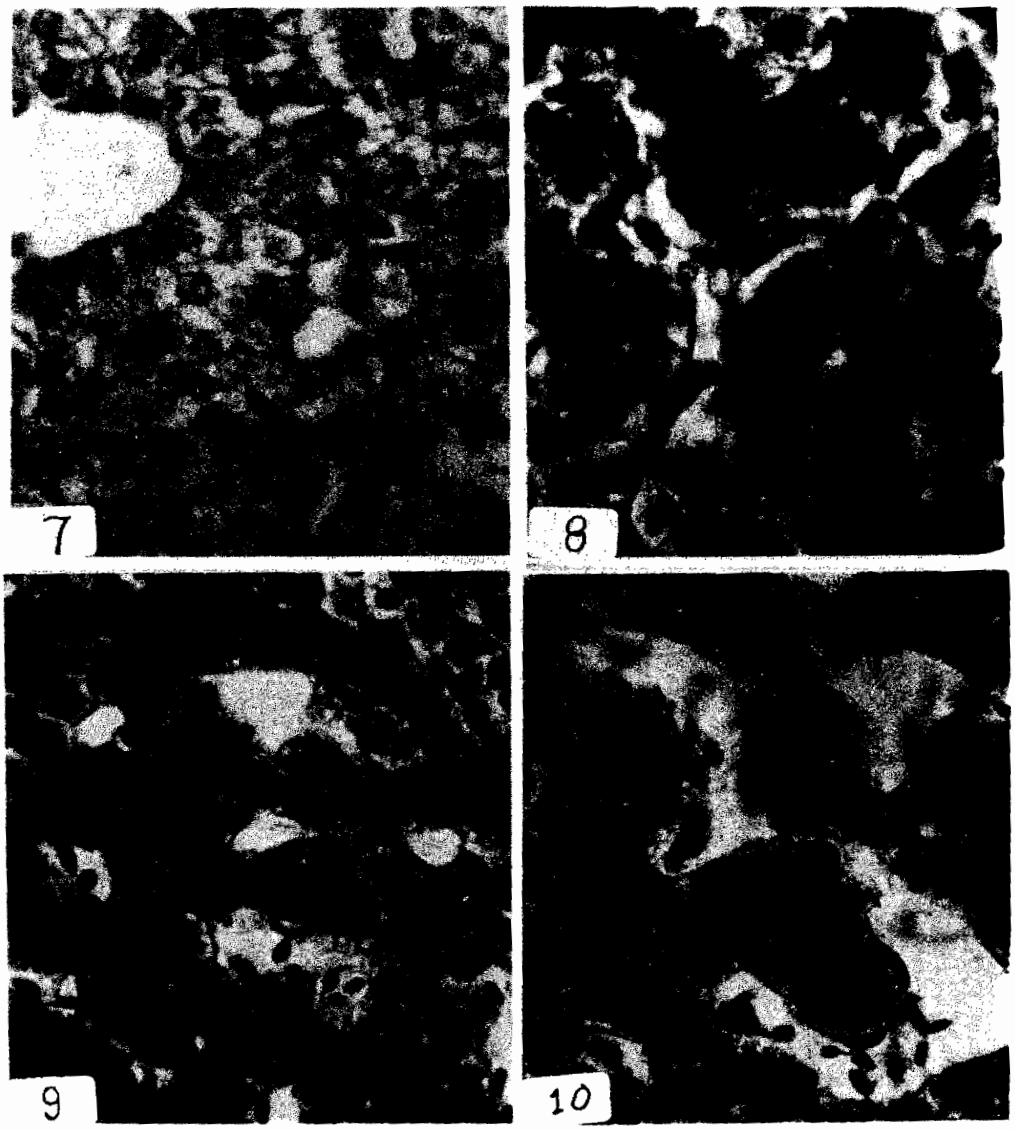
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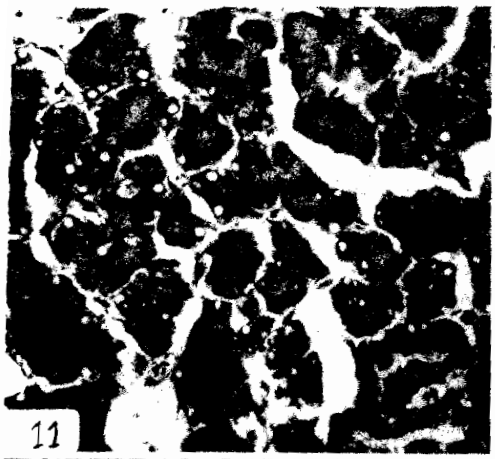
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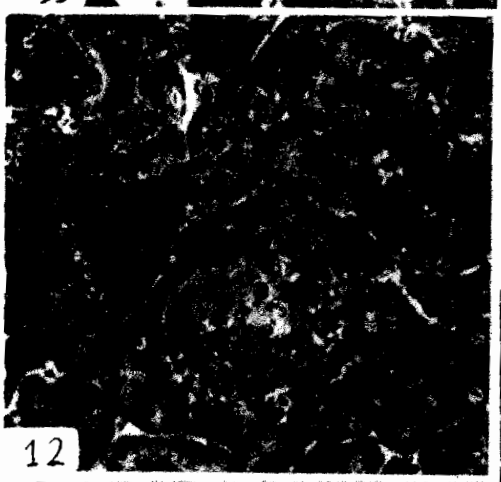
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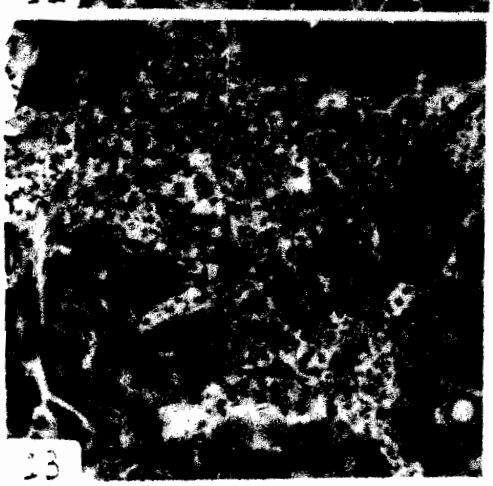
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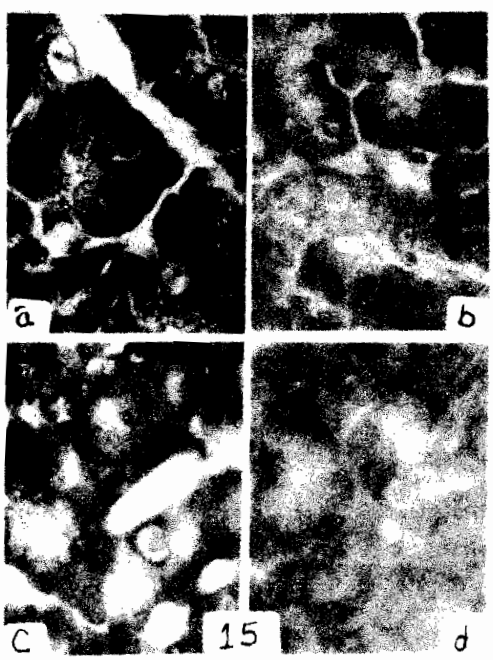
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