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DIPHTHERITIC FORM OF POX IN CHICKENS

(With 6 Figures and 5 Tables)

By

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(Received at 20/7/1995)

صورة تنفسيه لجدري الطيور في الدجاج

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لوحظ ظهور أوبئة من جدري الطيور في خمسة قطعان دجاج بياض في صورة تنفسية بدون ظهور آفات على الجلد الخارجي ، وكانت سبباً في خسائر إقتصاديه كبيرة نتيجة لإنخفاض في معدل إنتاج البيض وزيادة معدلات النفوق. وكانت الأعراض عبارة عن صعوبة في التنفس والموت نتيجة الإختناق. وكانت الآفات التشريحيه عبارة عن مواد صفراء متجبنة على فتحة الحنجره وكذلك في تجويف القصبه الهوائيه. وقد أثبت أن المسبب هو فيروس جدري الدجاج بعد الفحص الباثولوجي ، حقن المستنبتات النسيجية ، مدى العدوى في السلالات المختلفة ، إختبار الترسيب في الأجار وإختبار التعادل. وقد أظهر الفحص الباثولوجي تغيرات هستولوجية في الطبقة المخاطية مع وجود خلوية أجسام داخل السيتوبلازم . أمكن تنمية الفيروس في بيض الدجاج المخصب والذي أدى إلى ظهور ندبات على أغشية الأجنة المحترنة وكذلك على المستنبتات النسيجية وأدى إلى تغيرات باثولوجية بها مع وجود الأجسام الخلوية داخل السيتوبلازم. وجد أن الفيروس المعزول ضاري بالنسبة للدجاج والرومي لكنه غير ضاري بالنسبة للحمام والبط. التحصين ضد جدري الدجاج أعطى حماية جزئية ضد العدوى عن طريق القصبة الهوائية لكنه أعطى حماية جيدة وفعالة ضد العدوى بالفيروس المعزول عن طريق الجلد والحقن في الوريد.

SUMMARY

Outbreaks of fowl pox were observed in five chicken layer flocks assuming a respiratory diphtheritic form without cutaneous lesions and was responsible for severe economic losses due to drop in egg production and increased mortalities. The clinical signs were mainly respiratory distress, rales, and death from suffocation. Lesions were localized on laryngeal opening and in trachea in the form of yellow caseous plugs that occluded the tracheal lumen. The isolated pox virus was characterized as fowl pox by histopathological examination, tissue culture inoculation, host range infection, agar gel precipitation (AGP) and neutralization tests. The histopathological findings revealed proliferative and degenerative altera-

tions of the tracheal mucosa with presence of eosinophilic intracytoplasmic inclusion bodies. The virus could be propagated in chicken embryos producing characteristic pock lesions on the chorioallantoic membrane (CAM), as well as in tissue culture producing a cytopathic effect. Plaques were formed on overlaid monolayer chicken embryo fibroblast cells with presence of intracytoplasmic inclusion bodies in tissue culture. The isolated virus was found to be pathogenic to chickens and turkeys but nonpathogenic for pigeons and ducks. Vaccination of chickens with fowl pox vaccine protected partially against intratracheal (I/T) inoculation with isolated pox virus, but good protection was obtained against intradermal (I/D) and intravenous (I/V) inoculations.

Keywords: *Diphtheritic form-pox-chickens*

INTRODUCTION

Fowl pox is an acute contagious disease caused by fowl pox virus. Outbreaks of the disease still affect the poultry industry in many countries *(BIGGS, 1982)*. Losses are mainly due to reduction in egg production in a slowly spreading natural fowl pox infection in unvaccinated chickens *(JONES and JORDAN, 1976)*.

Avian pox viruses constitute a subgroup of pox viruses which include fowl pox, pigeon pox, turkey pox, canary pox, sparrow pox, Junco pox and starling pox *(MATTHEWS, 1982)*.

All avian pox viruses have the same ultrastructural details and the differentiation between them has been attempted on the basis of host susceptibility, characte-ristic lesions on the dermal epithelium, growth on chorioallantoic membrane, cross-protection, cross-neutralization, complement-fixation, and agar-gel precipitation tests *(TRIPATHY et al., 1973)*.

MAYR (1963) did extensive studies on differentiation of avian pox viruses. He found that the turkey pox virus could affect ducks and produced local pox lesions following cutaneous application and generalized pox lesions following intravenous inoculation. The fowl pox and pigeon pox viruses did not cause either local or generalized pox lesions in ducks. The pigeon pox virus has been found to cause mild local reaction, but no generalized pox lesions in chickens or turkeys. Generalization was induced only in pigeons.

BENGELSDORFF and SCHNEIDER (1963) demonstrated that vaccines prepared from fowl pox and pigeon pox virus strains propagated in chick embryo tissue culture were

suitable for practical use and protection against field exposure.

BAXENDALE (1971) found a detectable immunity against virulent fowl pox virus which was induced after vaccination with turkey pox virus vaccine administered by feather follicle scarification. He also found that the turkey pox virus vaccine was safe to use in chicks as young as two weeks old and did not spread to susceptible incontact fowls.

TRIPATHY and HANSON (1978) infected hens with fowl pox virus originally isolated from a natural mild infection and found that egg production started to drop at the 2nd week and continued to the 5th week postinfection. Birds challenged with fowl pox virus fifteen months after vaccination with pigeon pox vaccine were susceptible indicating limited duration of immunity following single vaccination.

In spite of vaccination against fowl pox, several outbreaks were observed in the last few years manifested mainly by respiratory distress without development of skin lesions. The present study was planned to investigate the type of pox virus involved in such outbreaks and its epidemiological features as well as the role of fowl pox vaccine in protecting chickens against challenge with isolated poxvirus.

MATERIAL and METHODS

Reference antisera:

Reference avian pox antisera (Fowl pox 1, pigeon pox, and turkey pox) were kindly supplied by ***Prof. Dr. S. Mousa, Dept. of Poultry Diseases, Fac. of Vet. Med., Assiut University***.

Fowl pox vaccine:

Fowl pox virus vaccine (**Ovo-Diphtherin Fort®**) containing at least 5000 EID_{50} per bird dose, provided by ***Intervet International B. V. Boxmeer Holland*** was used for vaccination of broiler chickens.

Embryonated chicken eggs:

Fertile eggs were obtained from the farm of Faculty of Agriculture, Assiut University, and incubated at 37 C until used at 10 days for virus isolation and titration.

Samples and virus isolation:

A total of 120 diseased and freshly dead birds of Lohman and Hy-line egg type varieties showing respiratory distress were obtained from outbreaks of fowl pox in Beni-Suef, El-Menia and Sohage governmental poultry farms and used for virus isolation. Specimens as exudates, tracheal scrapings, cheesy materials, larynxes and tracheas were collected, ground with sterile fine sand and suspended in saline. The suspension was centrifuged and antibiotics (penicillin and streptomycin) were added to the supernatant fluid and held at 4 C for 1/2 hour.

The bacteria free suspensions were inoculated on the chorioallantoic membrane of 10-day-old developing chicken embryos by the drop membrane method. The inoculated eggs were incubated at 37 C and candled daily for 6 days. The membranes were examined for presence of pock lesions and stored at -20 C until used.

Preparation of stock virus suspension:

Suspensions were inoculated onto CAM of embryonated chicken eggs and after 6 days of incubation the CAM s were harvested. A 10% suspension of CAM was prepared and titrated to contain about 3 X 10^8 EID_{50} per ml. Aliquots of suspensions were stored at -20 C till used for experimental infections as well as tissue culture inoculation *(MINBAY and KREEIER, 1973)*.

Virus titration:

The titration was conducted in 10-day-old chick embryos with the dropped CAM inoculation technique. Examination of CAM for lesions to determine the end point of infectivity was made on the 6th day postinoculation. Titration end points (EID_{50}) were calculated by the method of *REED and MUENCH (1938)*.

Tissue culture:

Primary chick embryo monolayer cells were prepared in petri dishes from 9-day-old embryonated hen's eggs. Growth medium was Earle's balanced salt solution supplemented with 0.5% lactoalbumin hydrolysate, 0.01% yeast extract, and 10% bovine serum. Monolayer cells were cultivated at 37 C in a CO_2 incubator for 2 days. The virus suspension was added (0.2 ml) and allowed to adsorb to cells at 37 C for one hour with intermittent agitation. For the plaque technique, 5 ml of overlay medium containing 1.0% Bacto-Agar (**Difco**) were added. After additional incubation, 4 ml of a second overlay medium with 1.0% agar and containing 0.01% neutral red were added. On the next day, plaques were counted and their diameters measured. Mean value was calculated from the diameters of 50 plaques *(MORITA, 1973)*.

Experimental birds:

1- One-day-old chicks:

They were obtained from the farm of Faculty of Agriculture, Assiut University, and used for intravenous inoculation with isolated pox virus.

2- Broilers:

Six-week-old broiler chickens were obtained from the same source and used for intradermal and intratracheal infection with isolated pox virus as well as for vaccination with fowl pox virus vaccine.

3- Pigeon squabs:

3-4-week-old pigeon squabs were used for intravenous in-

oculation with isolated pox virus.

4- Ducks:

12-week-old ducks were purchased from the local market and used for I/V inoculation with isolated pox virus.

5-Turkey poults:

10-week-old turkey poults were supplied by Beni-suef turkey governmental farm and were used for I/V inoculation with isolated pox virus.

Agar gel precipitation test:

The microtest was carried out using 1.0% Noble agar suspension in 8% sodium chloride solution. The gel was poured onto microscope slides and 6 wells were placed around a central well. Wells were filled and slides incubated in a moist chamber at 37 C for 24 to 72 hours *(SCHMIDT. 1969)*.

Neutralization test:

Ten-fold dilutions of isolated pox virus were mixed in equal volumes with specific antisera and incubated for two hours at 37 C. The mixtures were then titrated on the CAMs of embryonated chicken eggs using 0.1 ml of the mixtures per egg. The neutralization index was expressed as the log difference between the control virus titre and the titre of the virus after incubation with the specific antisera *(BAXENDALE, 1971)*.

Histopathological examination:

Impression smears from tracheas were prepared by gentle pressing the tracheas on glass slides, stained with Giemsa and haematoxylin and eosin, then examined microscopically. Specimens from larynx and tracheas of field cases and experimentally infected birds as well as CAMs of inoculated chicken eggs were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned, and stained with Giemsa and haematoxylin and eosin.

Routes of experimental infection:

1- Intradermal route:

This method was done by scarification of comb and rubbing with about 0.05 ml of stock virus.

2- Intravenous route:

The wing vein was used for inoculation. Each bird was inoculated with 0.5 ml of stock virus.

3- Intratracheal route:

Using a tuberculin syringe, 0.1 ml of virus suspension was injected directly into the trachea of each bird.

Determination of pathogenicity of isolated pox virus for chickens by different routes:

Three groups each of 10 chickens were used. The first group consisting of one-day-old chicks were inoculated intra-venously with 0.5 ml/bird of stock virus. The second and the third groups of 6-week-old chickens were used

for intradermal and intratracheal inoculations respec-tively. A fourth group of 5 chickens served as noninfected control. All groups were observed for 2 weeks and signs, lesions and mortalities were recorded.

Determination of host-range of isolated pox virus:

Four groups of birds were used in this experiment. The first group consisted of 10 broiler chicks, the second of 10 pigeon squabs, the third of 5 turkey poults and the fourth group of 5 ducks. All groups were inoculated with isolated fowl pox virus by the intravenous route. Birds were observed for 2 weeks for clinical signs, lesions and mortalities.

Evaluation of protection afforded by vaccination with fowl pox vaccine against challenge by different routes:

Four groups of broiler chicks (A, B, C and D) each contained 10 birds were used. All groups were vaccinated with fowl pox vaccine at 6 weeks of age by stick wing web method. Groups A, B and C were challenged with a field isolate of fowl pox virus two weeks postvaccination by I/D, I/T, and I/V routes respectively. Group, D was kept as non-vaccinated control. The challenged birds were observed daily for two weeks for clinical signs, lesions and mortalities.

RESULTS

Field investigations:

The disease was observed in 5 laying chicken flocks of 20-25,000 birds each which had been vaccinated with fowl pox vaccine. Two flocks were in Beni-Suef Governorate and aged 40 and 45 weeks, two flocks in Sohag Governorate and aged 35 and 50 weeks, and one flock in El-Menia Governorate and aged 25 weeks. Data concerning these flocks are summarized in (Table, 1). The most important clinical signs observed were respiratory distress and deaths from suffocation. Drop of egg production (10-15 %) was recorded in the affected flocks. The mortality rate ranged 3-5 % over one month. Upon necropsy, lesions were localized in the respiratory tract without external skin lesions. Lesions were proliferative and observed at the laryngeal opening with a thick, yellow caseous plug adhered to the lesions. These lesions extended down and, in some cases, a yellow caseous pseudomembrane had occluded the lumen of the trachea (Fig.1).

Virus characterization:

On the second chicken embryo passage, the isolated viruses were found to produce characteristic pock lesions of a millet size on inoculated CAMs. Isolated viruses reacted positively in agar gel precipitation test with all tested

reference antisera; however, the reaction against fowl pox antiserum was visible with three lines of precipitation as compared to one line against pigeon and turkey pox antisera.

Results of virus neutralization are shown in table (5). It is clear that a complete neutralization was achieved against fowl pox reference antiserum, while weak incomplete neutralization was observed against pigeon and turkey reference antisera.

Histopathological findings:

Most of the examined cases revealed obstruction of the tracheal lumen with degenerated and necrosed haemorrhagic masses (Fig 2). The tracheal mucosa showed hypertrophy and hyperplasia of mucus-producing cells, and flattening of the epithelial cells with infiltration of lymphocytes, followed by proliferation and enlargement (Fig. 3). In many cases, loss of orientation and organization of epithelial cells were evident, and clusters of degenerated cells protracted toward the lumen. Most of enlarged cells in the affected area contained charac-teristic eosinophilic cytoplasmic inclusion bodies. The lamina propria showed more severe congestion and inflammatory cell infiltration.

The tracheal imprints showed sloughing of epithelial cells and presence of characteristic intracytoplasmic inclusions (Fig. 4).

Results of tissue culture inoculation:

The virus, in the form of CAM suspension, produced a cytopathic effect within 3-5 days postinoculation of chicken embryo cell cultures. Plaques started to be visible micro-scopically in monolayers within 4 to 5 days. Plaques could be seen by naked eye after 7 to 10 days postinoculation as clear unstained areas of 0.6 to 0.8 mm in dia-meter against deep red back-ground (Fig 5). Microscopic exa-mination of stained cultures revealed the presence of intracytoplasmic inclusion bodies (Fig. 6).

Pathogenicity for chickens:

The results of experimental infection of chickens by different routes are summarized in table (2). In case of intradermal inoculation, lesions of pox were restricted to the site of inoculation. Typical primary lesions were observed by the 4th day, papules by the 5th and 6th days and vesicular stage came next. Scab formation occured during the second week after inoculation. The intravenous in-oculation of one-day-old chicks resulted in generalized lesions of pox developed on many parts of the body including both sides of the comb and wattles, eyelids, beak, skin as well as on the feet. After

intratracheal inoculation lesions were localized in the larynx and the proximal part of the trachea in the form of caseous plugs.

Host-range:

The results of this experiment (Table 3) indicated that I/V inoculation of isolated pox virus resulted in generalized lesions in chickens and turkeys, while pigeon squabs and ducks were not affected.

Protection experiment:

Results summarized in (Table 4) indicated that a good pro-tection was induced by fowl pox vaccination against I/D and I/V challenge, while protection was low against I/T challenge.

DISCUSSION

In the last few years, several outbreaks of the diphtheritic form of fowl pox were observed which were associated with respiratory distress, increased mortalities and drop in egg production. *TRIPATHY and HANSON (1978)* found drop of egg production after inoculation of laying hens with fowl pox virus. The clinical signs were manifested by respiratory distress, gasping and rales with lacrimation and conjunctivitis. Upon nec-ropsy, lesions involved larynx and trachea with presence of a thick yellow caseous pseudomembrane occluded the lumen of the trachea and laryngeal opening. Similar clinical signs and lesions were reported by *ELEAZER et al. (1983) and RIHTER-REICHHELM et al. (1985).*

The histopathological examination of the trachea revealed its partial or complete obstruction by degenerated and necrosed haemorrhagic deposits. In addition proliferative and degenerative alterations of the tracheal mucosa with presence of eosinophilic intracytoplasmic inclusion bodies were observed. These findings are similar to those observed by *TANIZAKI et al. (1986).*

The virus produced cytopathic effect within 3-5 days post-inoculation of tissue culture. Plaques started microscopically in monolayer chicken embryo fibroblast within 4 to 5 days. Plaques could be seen by naked eye 8 to 10 days postinoculation as clear unstained areas of 0.6 to 0.8 mm in diameter against deep red-background. Similar findings were discribed by *MAYR and KALCHER (1961), GAFFORD et al. (1969), and MORITA (1973).*

Results of pathogenicity studies of isolated virus in chickens revealed that lesions developed only at the site of inoculation after I/D inoculation 4 - 6 days post-inoculation. Similar results were obtained by *MINBAY and KREIER (1973),* while *TRIPATHY et al. (1973)* detected generalized lesions after cutaneous in-

oculation. Generalized lesions were produced 10 days after I/V inoculation in agreement with *MINBAY and KREIER (1973), MAYR and DANNER (1976) and TRIPATHY et al. (1973).* The I/T route of inoculation resulted in localized lesions typical to those observed in the field at the laryngeal opening and the proximal part of the trachea, 6-7 days post-inoculation. These results were similar to those reported by *ELEAZER et al. (1983) and RICHTER-REICHHELM et al. (1985).* On the other hand, *MINBAY and KREIER (1973)* failed to reproduce lesions in the trachea after I/T inoculation of fowl pox virus, which may be attributed to affinity differences among isolates of the virus.

Results of I/V inoculation to determine the host-range revealed that the isolated virus was pathogenic to chickens and turkeys but not to pigeons or ducks. Lesions in chickens and turkeys were generalized and involved many parts of the unfeathered areas. These results were considered important and dependable for differentiation of avian pox viruses as reported by *MAYR (1963).*

From the obtained results, it could be stated that the isolated viruses belonged to avian pox-viruses based on the histo-pathological findings, lesions on CAMs, and cytopathogenicity in tissue culture as reported by *MOUSA (1979); TANIZAKI et al. (1986), and TRIPATHY (1991).*

Moreover, the isolated poxvirus could be characterized as fowl pox virus according to the criteria reported by *MAYR (1963)* which included serological tests (AGP & VN), plaque shape and size, pathogenicity to chickens by different routes, generalization after I/V inoculation of chickens and turkeys, and failure of generalization after I/V in-oculation of ducks and pigeons.

Vaccination of broiler chickens with fowl pox virus vaccine induced good protection (90 and 80 % respectively) against I/D and I/V challenge with isolated virus but poor protection (40 %) against I/T challenge. These results may explain the field outbreaks in which the affected flocks were previously vaccina-ted against fowl pox but the disease assumed a respiratory diphtheritic form without skin lesions. Many factors play a role in the pathogenesis and immunity of fowl pox. Several investigators explained how the birds in the field got the wet form of pox at the tracheal opening with little or no evidence of external lesions on the unfeathered parts. *TRIPATHY (1991)* reported that in a con-taminated environment, the aerosol generated by feathers and dried scabs containing poxvirus

particles provides suitable condition for respiratory infection, especially because the cells of mucosa of the upper respiratory tract and mouth are highly susceptible to the virus. *ELEAZER et al. (1983)*, found that inoculation of chickens with fowl pox virus via eye drop and by laryngeal swabbing produced lesions of pox at larynx and trachea. They suggested that certain insects such as gnats are attracted to the eye of birds, presumably seeking moisture or to feed on ocular fluids, insects carrying the pox virus may also do this, thus depositing the virus in the eye of the bird which could travel down the lacrimal duct to the laryngeal region, where it finds cells for which it obviously has an affinity. *MOCKETT et al. (1990)* suggested that fowl pox virus could behave similar to other pox viruses known to spread via aerosol routes, e.g. ectromelia in mice and smallpox in man.

It could be concluded that the respiratory diphtheritic form of fowl pox infection is common among laying flocks and is responsible for severe economic losses. The present paper has also thrown some light on the importance of impression smears in the rapid diagnosis of avian pox. Finally, proper vaccination against fowl pox must be adopted and a pooster dose of vaccination seems to be important for inducing high rate of protection especially in the endemic areas.

ACKNOWLEDGEMENT

Thanks are expressed to ***Prof. Dr. A. A. Bayomi***, Professor of Vet. Pathology, Dept. of Vet. Pathology, Fac. of Vet. Med., Assiut University for his help in the histopathological examination in this work.

REFERENCES

Baxendale, W. (1971): Studies on three avian pox viruses and the development of an improved fowlpox vaccine. Vet. Rec., 88: 5-9.

Bengelsdorff, H. J. and Schneider, B. (1963): Comparative experimental studies of the development of immunity in chickens following the use of pigeon and fowl pox virus vaccines. Deutsch. Tierarztl. Wschft., 70: 322-326.

Biggs, P.M. (1982): The world of poultry disease. Avian Pathol., 11: 281-300.

Eleazer, T.H.; Harrell, J.S. and Gaffney, H.B.(1983): Transmission studies involving a wet fowl pox isolate. Avian Dis., 27: 542-544.

Gafford, L. G.; Sinclair, F. and Randall, C. C. (1969): Growth cycle of fowlpox viruses and change in plaque morphology and cytopathology by contaminating Mycoplasma. Virology, 37: 464-472

Jones, R. C. and Jordan, F. T. W. (1976): Atypical fowl pox in chickens. Vet. Rec., 99: 56-57.

Matthews, R. E. F. (1982): Classification and nomenclature of viruses. Intervirology, 17: 42-46.

Mayr, A. (1963): Neue Verfahren für die Differenzierung der Geflügelpockenviren. Berl. Münch. Tierärztl. Wschr., 76: 316.

Mayr, A. and Danner, K. (1976): Oral immunization against pox. Studies on fowlpox as a model. Developments in Biological Standardization, 33: 249-259.

Mayr, A. and Kalcher, K. (1961): Plaque-bildung bei den Geflügelpockenviren. Arch. ges. Virusforsch., 11: 307-325.

Minbay, A. and Kreier, J. P. (1973): An experimental study of the pathogenesis of pox infection in chickens. Avian Dis., 17: 532-539.

Mockett, A. P. A.; Deuter, A. and Southee, J. (1990): Fowlpox vaccination: Routes of inoculation and pathological effects. Avian Pathol., 19: 613-625.

Morita, C. (1973): Studies on fowlpox viruses. I. Plaque formation of fowlpox virus on chick embryo cell culture. Avian Dis., 17: 87-92.

Mousa, S. (1979): Die Identifizierung und Charakterisierug eines Falkenpocken-und Psittacidenpockenvirus unter Verwendung von Zellkulturmethoden. Dissertation, Ludwig-Maximilians-Universität München, Germany.

Reed, J. L., and Muench, H. (1938): A simple method of estimating fifty percent end-points. Am. J. Hyg., 27: 493-497.

Richter-Reichhelm, H. B.; Boning, W. and Althoff, J. (1985). Neoplasms. Response to carcinogens of respiratory epithelium, syrian golden hamster (Mesocricetus auratus). In: Respiratory system. Jones, T. C.; Mohr,U. and Hunt, R. D., eds. Springer-Verlag, Berlin, Heidelberg. pp.33-47.

Schmidt, U.(1969): Die Agar Gel-prazipicationsreaktion in der praktischen Diagnostik der Geflugelpocken. Arch. exp. Vet. Med., 23: 1085.

Tanizaki, E.; Kotani, T. and Odagiri, Y. (1986): Pathological changes of tracheal mucosa in chickens infected with fowl pox virus. Avian Dis., 31:169-175.

Tripathy, D.N. (1991): Avian pox. In: *Diseases of Poultry*, Calneck, B. W.; Barnes, H.J.; Beard, C. W.; Reid, W. M. and Yoder, H. W. (Eds), 9th edn, pp. 198-212, Ames, Iowa State University Press.

Tripathy, D. N. and Hanson, L. E. (1978): Pathogenesis of fowlpox in laying hens. Avian Dis., 22: 259-265.

Tripathy, D. N.; Hanson, L. E. and Killinger, A. H. (1973): Studies on differentiation of avian pox viruses. Avian Dis., 17: 325-333.

LEGENDS

1): Yellow caseous plugs at the laryngeal opening (Left side) and in the tracheal lumen (Right side).

Fig. (2): Trachea showing luminal obstruction. H & E x 40.

3): Trachea showing proliferative and metaplastic epithelial changes. H & E x 250.

4): Tracheal impression smear showing acidophilic intracytoplasmic inclusion globules. H & E x 100.

Fig. (5): Plaque in secondary chicken embryo fibroblasts overlay 7 days postinoculation.

6): Intracytoplasmic inclusion bodies and lysis of chicken embryo fibroblasts infected with fowl pox isolate. H &E x10.

Table (1):Field data of outbreaks of the diphtheritic form of pox in laying chickens.

Flock	Age in weeks	Total No.	Vacc. history	% drop in egg prod.	Lesions observed	% Mortality *
I	40	22,000	+	10 %	Resp. (diphtheritic)	5 %
II	45	22,000	+	15 %	Resp. (diphtheritic)	5 %
III	35	20,000	+	12 %	Resp. (diphtheritic)	4.5 %
IV	50	21,000	+	14 %	Resp. (diphtheritic)	3.6 %
V	25	25,000	+	10 %	Resp. (diphtheritic)	3 %

* Mortalities over one month.

Table (2): Results of experimental infection of chickens by different routes with fowl pox isolate.

Birds	Age	No.	Route	Lesions observed	No.Dis. birds
Chicks	one day	10	I/V	Generalized skin lesions	10
Broilers	6 weeks	10	I/D	Localized skin lesions	10
Broilers	6 weeks	10	I/T	Localized diphtheritic	10
Broilers	6 weeks	5	None	None	-

Table (3): Host-range of isolated fowl pox virus after I/V inoculation.

Species	No. of birds	Age in weeks	No. of Dis. birds	Type of lesions
Chickens	10	6	10	Generalized
Pigeons	10	4	0	-
Turkeys	5	10	6	Generalized
Ducks	5	12	0	-

Table (4): Protective immunity in chickens vaccinated against fowl pox and challenged by different routes at two weeks postvaccination.

Group	Age in weeks	No. of birds	Route of infection	No. of Diseased	Lesions observed	% Protection
A	6	10	I/D	1	Localized cutaneous	90 %
B	6	10	I/T	6	Localized diphtheritic	40 %
C	6	10	I/V	2	Generalized	80 %
D	6	10	None	0	-	-

Table (5): Results of virus neutralization of isolated pox viruses with reference antisera.

Reference antisera	Neutralizing index				
	Isolates				
	1	2	3	4	5
Fowl pox	2.1	1.8	2.2	1.9	2.4
Pigeon pox	0.6	0.7	0.5	0.9	0.9
Turkey pox	1.1	1.0	0.8	0.6	0.9

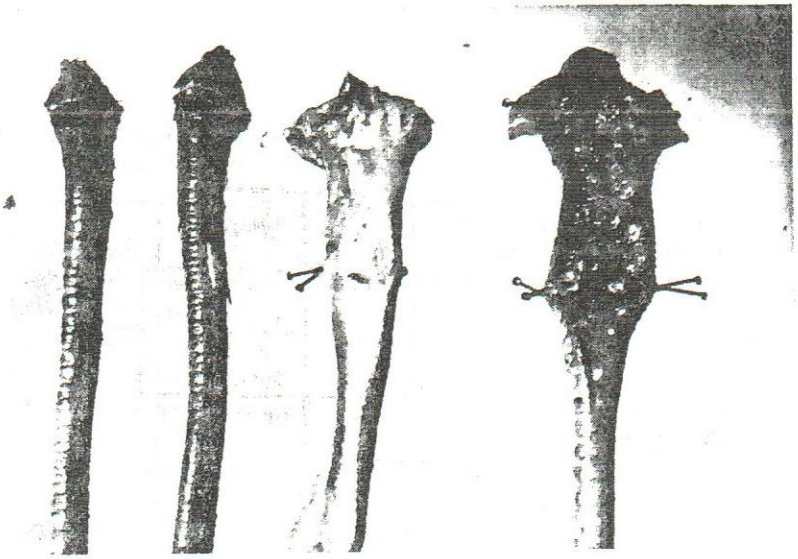

Fig. (1): Yellow caseous plugs at the laryngeal opening (Left side) and in the tracheal lumen (Right side).

Fig. (2): Trachea showing luminal obstruction. H & E x 40.

Fig. (3): Trachea showing proliferative and metaplastic epithelial changes. H & E x 250.

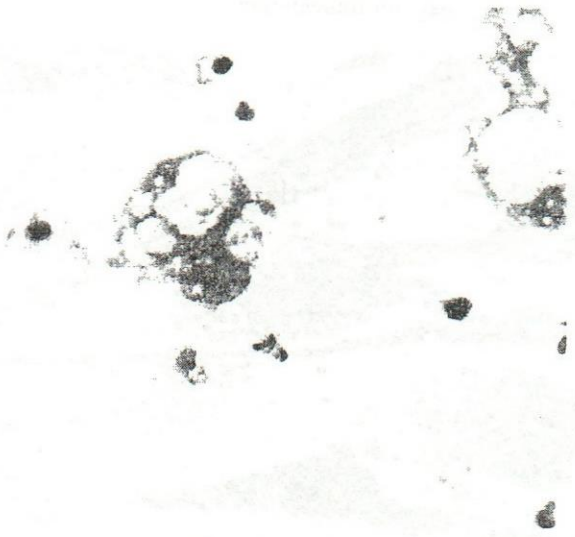

Fig. (4): Tracheal impression smear showing acidophilic intracytoplasmic inclusion globules. H & E x 100.

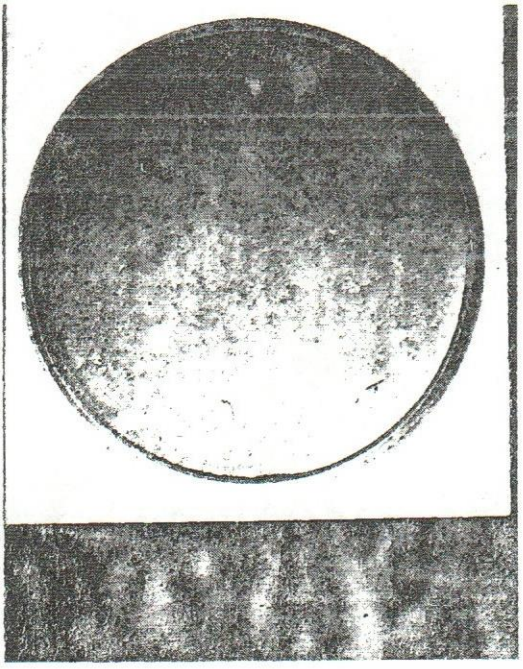

Fig.(5): Plaque in secondary chicken embryo fibroblasts overlay 7 days postinoculation.

Fig. (6): Intracytoplasmic inclusion bodies and lysis of chicken embryo fibroblasts infected with fowl pox isolate. H &E x 100