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بعض الدراسات عن كوليرا البط في الوادي الجديد
١- دراسات مورفولوجيه وميكرومورفولوجيه عن المرض الطبيعي والتجريبي

مختار طه ، بيخيت سالم ، * طلبه يونس *

تم دراسة التغيرات في الشكل والتركيب للأعضاء الداخلية لبط مصاب
بالعدوى الطبيعية لمرض الكوليرا . تم عزل وتصنيف ميكروب البستريلا ملتوسيدا
من الطيور المريضة . تم عمل العدوى التجريبيه للمرض بواسطة الحقن العضلي
لهذه العترة المعزولة في البط البكيني . تم عمل دراسات هستوباثولوجيه مقارنة
وكذلك تطور ونشؤ هذا المرض نوقشت النتائج .

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**SOME STUDIES ON DUCK PASTEURELLOSIS
IN THE NEW-VALLEY
II- MACRO- AND MICRO-MORPHOLOGICAL STUDIES ON THE
NATURALLY AND EXPERIMENTALLY INDUCED DISEASE
(With One Table and 7 Figures)**

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SUMMARY

Gross and histopathological examination of naturally occurring pasteurilla infection was carried out in clinically diseased ducks. Pasteurella multocida D was isolated and identified. Experimental induction of the disease was performed via intranasal and intramuscular routes of infection using the isolated new serotype in native and Pekin ducks. Comparative histomorphologic studies as well as the pathogenesis of infection were investigated and discussed.

INTRODUCTION

Enzootic pasteurilosis is an acute septicaemic disease causing high losses in poultry industry. In spite of its great importance, yet, little has been known about its histopathogenesis and routes of infections.

Surveying the available literatures, duck cholera was investigated in Egypt by MASOUD & KONSOH (1963), EL-MONGY (1977) and LOTFY *et al.* (1977). CARTER (1961) & HEDDLESTON (1962) isolated different pasteurilla multocida strains from living and dead birds.

Herein, the present work was designed to fulfil the following purposes:

- Gross and histopathologic studies on the naturally occurring disease in the New-Valley duck farm.
- Comparative histopathologic picture of the experimentally induced disease via intranasal and intramuscular routes of infection with the isolated new pasteurilla serotype.

MATERIALS and METHODS

I- Naturally infected cases :

20 dead and diseased adult ducks were randomly selected from the New-Valley duck farm. Isolation and identification of new Pasteurella multocida serotype from these cases was done by ABDEL-MOTELIB and SALEM (1986) in the first publication. The ducks were subjected to careful P. M. examination. Tissue specimens from the liver, kidney, heart, lung, spleen and intestine were collected, fixed in 10% neutral buffered formaline and were prepared for light microscopic examination.

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II- Experimental induction :

30 adult native and Pekin ducks of 6 - 8 weeks old were examined bacteriologically and parasitologically and proved to be free. The birds were divided into 2 groups, each contained 12 birds, in addition to 6 control ducks. The experimental groups were infected as shown in Table (1). During the experimental period, the ducks were observed and left until daily deaths occurred. P. M. examination was done shortly after death. Re-isolation of the inoculated strain was successful from all the dead birds till the end of the experiment. Specimens for histopathologic studies were taken from the lung, heart, liver, kidney, intestine and spleen. The samples were fixed in 10% neutral buffered formaline and were prepared for light microscopy. The control birds were sacrificed at the end and specimens were also collected in the same manner.

Table (1)

Exp. groups	Type of infection	Route and dose	Days post-infection	No. of dead birds
First (12 ducks)	P. multocida D ⁺	Intranasal & 30 x 10 ⁷ ++	12 hrs.	5
			3 days	3
			4 "	2
			7 "	2
Second (12 ducks)	P. multocida D ⁺	Intramuscular & 30 x 10 ⁷ ++	12 hrs.	7
			3 days	5
Third (6 ducks)	Non-infected control	--	--	Sacrificed at the end

+ : Abd El-Motelib and Salem (1986) ++ : Shahata (1977).

RESULTS**P. M. findings :**

In the experimentally infected groups, congestion of the visceral organs was a common finding. Petechial and ecchymotic haemorrhages were found on the intestinal and caecal mucosa, coronary fat, myocardium, trachea, lung and liver. In few cases hydropericardium was observed. In the intranasally infected group, differential from the intramuscular one, the liver showed obvious miliary whitish foci regularly distributed all over the parenchyma. In sporadic cases, there was lung fibrinous pericarditis.

The P. M. picture of the naturally collected cases did not differ significantly from the previous picture except lung consolidation and fibrinous pericarditis could not be observed.

DUCK PASTEURELLOSIS

Histopathologic findings :**Intranasal route****The Liver :**

After 12 hrs., 3 and 4 days, the liver showed severe congestion of the central veins, sinusoids and the vessels of the portal triads. Edema of the wall of some hepatic veins with lymphoid cell infiltration was observed. Slight edema of Disse spaces was found. The liver parenchyma manifested minute miliary necrobiotic foci with regular relation to the portal areas.

On post-inoculation day 7, edema became extensive and resulted in dissociation and atrophy of the hepatic cords (Fig. 2). Perivascular plasma cell accumulation was also prominent in the portal triads. The necrobiotic lytic foci were persistent and were accompanied with activation of Kupffer cells.

The lung :

The lungs examined 12 hrs. post-infection showed severe congestion of the interlobular and alveolar capillaries. In the interstitium, there was edema, erythrocytic extravasation and lymphoid cell infiltrate. The parabronchial lumina showed also few RBCs and lymphoid cell accumulation.

After 3 and 4 days, the lung showed fibrinous pneumonia in a stage of red hepatization. The alveoli were filled with fibrinous network, RBCs and few lymphoid cells (Fig. 3). On post-inoculation day 7, only the vascular changes of the lung were seen.

The kidney :

All the examined cases showed severe congestion of the intertubular and glomerular tuft of capillaries. Slight intertubular haemorrhages were also found. The renal parenchyma revealed tubulonephrosis which became prominent at the 7th day (Fig. 4). Moreover, interstitial edema and pericapillary plasma cell infiltration were evident in some cases examined after 1 week.

The heart :

After 12 hrs., the heart revealed edema and haemorrhages. On post-inoculation day 3, 4 and 7, there was fibrinous pericarditis. The fibrin network was deposited on the pericardium. The submesothelial connective tissue showed edema, congestion, slight haemorrhages and cellular reaction of lymphoid and plasma cells (Fig. 5).

The intestine :

The intestine of all the examined cases revealed severe congestion of the serosal and mucosal capillaries. The villar and glandular epithelium showed slight degeneration and desquamation.

The spleen :

The spleen of the cases examined after 4 and 7 days showed hyperplasia of the reticulo-endothelial cells of the ellipsoid. These cells were transformed into mobilized macrophages. The lymphoid tissue was reduced. Some cases showed severe congestion and foci of haemorrhages. The findings of the other cases were missed.

Intramuscular route

The liver of the examined cases showed similar vascular disturbances as well as necrobiotic changes. The necrobiotic changes were expressed by the lytic foci and fatty degeneration of the hepatocyte.

The examined lungs showed only congestion, edema and focal haemorrhages.

In the kidney, congestion of intertubular and glomerular tuft of capillaries as well as tubulonephrosis were seen.

The heart examined after 12 hrs. showed slight endocardial and myocardial haemorrhages. After 3 days, no microscopic changes could be detected.

The intestine revealed only congestion of the serosal and mucosal capillaries.

The splenic picture of the examined cases was also macrophagal reaction of the ellipsoid similar to the first group.

Natural infected cases

The liver of the naturally examined cases presented vascular changes expressed by congestion and edema associated with dissociation and atrophy of the hepatic cords. The hepatocellular changes were characterized by fatty degeneration and miliary necrobiotic lytic foci (Fig. 6).

The kidney showed focal haemorrhages, congestion and tubulonephrosis of the parenchyma.

The lungs revealed only congestion and extensive parabronchial and alveolar haemorrhages.

The heart showed no prominent alterations.

The intestine showed only serosal and mucosal congestion.

The prominent splenic reaction of all examined cases, was characterized by mobilization and transformation of the reticulo-endothelial cells of the ellipsoid into free macrophages (Fig. 7). This macrophagal reaction involved a wide area in the splenic pulps on the expense of the lymphoid elements. Congestion and repopulation of the spleen were observed in few cases.

DISCUSSION

In the present work, the P.M. findings of the naturally and experimentally diseased ducks revealed vascular lesions as well as degenerative and necrobiotic changes in the parenchymatous organs. This P. M. picture was in agreement with EL-MONGY (1977), SHARMA *et al.* (1974) and SHAHATA (1977). Moreover, lung consolidation and fibrinous pericarditis in the intranasally infected group as well as the necrobiotic foci of the liver presented the typical lesions of acute pasteurellosis (HEDDLESTON, 1985). The mortality rate in both routes was 100% and this indicated that this locally isolated strain was highly pathogenic for ducks.

DUCK PASTEURELLOSIS

Histopathologically, in all natural and experimentally examined groups, the parenchymatous organs manifested vascular disturbance in the form of vasculitis of some vessels, congestion, edema, petichelial and ecchymotic haemorrhages. These changes could be attributed to pasteurilla toxin and referred to the acute septicaemic nature of the disease (MASOUD and KONSOH, 1963 and SIRBU *et al.*, 1971).

The liver of all examined cases showed miliary necrotic foci which had a regular relation to the portal triads. These hepatic lesions could be attributed to the direct cytotoxic effect of the organism through its arterial dissemination. Similar lesions were recorded by DJAKOW (1960), KLUKAS and LOCKE (1970) and Sharma *et al.* (1974).

Tubulonephrosis as well as the intestinal changes of the examined cases were manifestation of septicaemia (TOMASEC, 1935 and MURATA *et al.*, 1964).

Splenic reaction in duck cholera seems to be deficient in the literatures. In our data, both in natural and experimentally examined cases, the splenic reaction was mainly macrophagal through the reticulo-endothelial cell hyperplasia of the ellipsoid for phagocytosis of the causative agent. However perivascular plasma cell infiltrate in the parenchymatous organs could be seen in the late stages of the disease.

The evaluation of the different routes of infecting duck with pasteurellosis has not been already studied. In our findings, in the intranasally infected group, heart and lung lesions could be correlated to the local effect of the micro-organism. However, the disease assumed a protracted course and some ducks survived till the 7th day. In the intramuscularly infected group, the ducks suffered from acute septicaemic infection and the disease had a short course and rapid deaths occurred till the 3rd day. On the contrary, WEBSTER (1927), HUGHES and PRITCHETT (1930), RHOADES (1964) and EL-MONGY (1977) considered the intranasal route of infection is the most efficient route for producing infection in ducks followed by the intramuscular one.

We could conclude that pasteurilla multocida D is highly virulent for producing the classic form of duck pasteurellosis in both native and Pekin ducks. It is also important to mention that both the intranasal and intramuscular routes of infection induced duck septicaemia while in the intramuscular route the disease exhibited more rapid and acute picture. Further experiments will be done to study the pathogenicity of the isolated serotype in other avian species.

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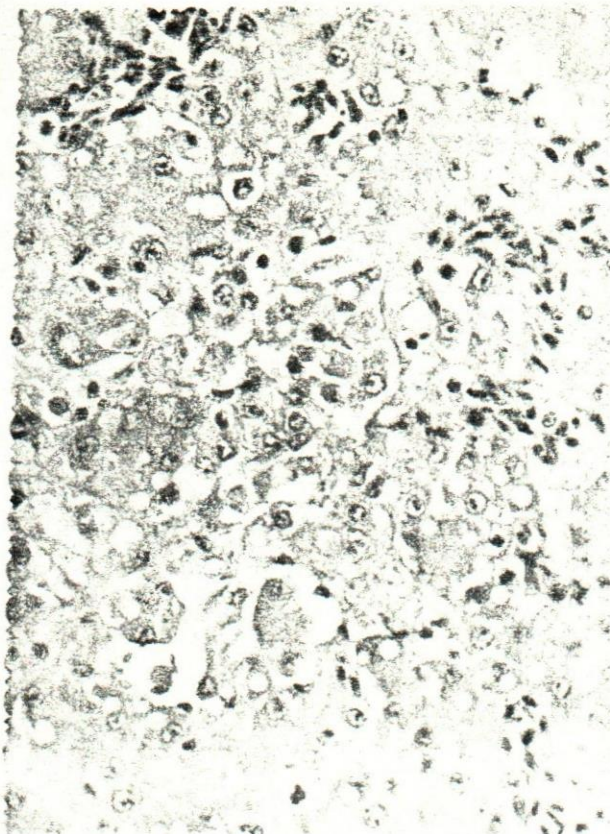


Fig. (1): Liver 3 days post-intranasal infection showing necrobiotic lytic focus. (H. E., X 400).

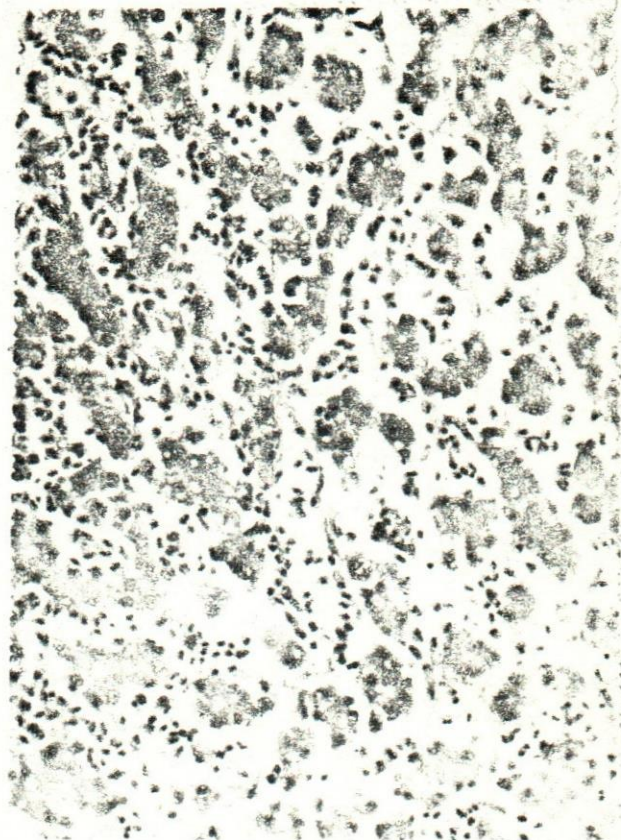


Fig. (2): Liver 7 days post-intranasal infection showing severe congestion, edema, dissociation and atrophy of the hepatic cords. (H.E., X 250).

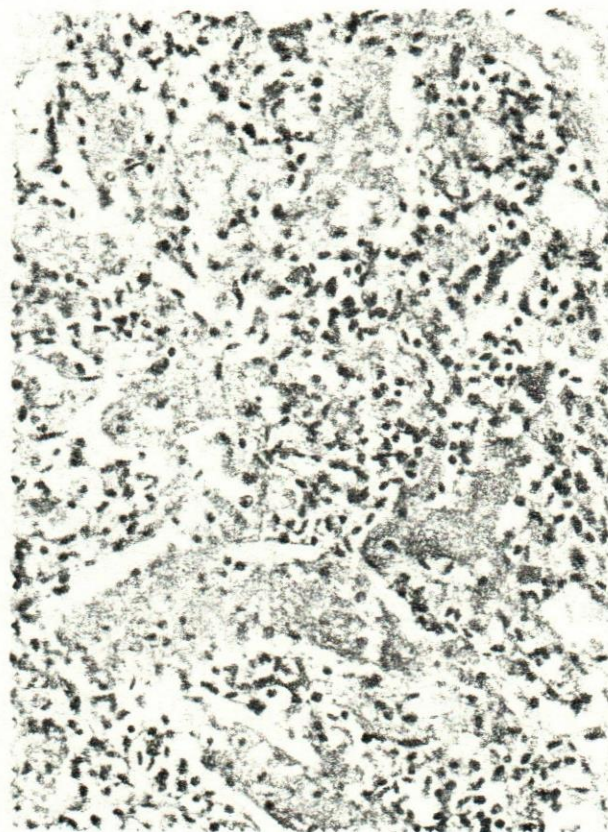


Fig. (3): Lung 4 days post-intranasal infection showing fibrinous pneumonia. (H.E., X 160).



Fig. (4): Kidney 7 days post-intranasal infection showing tubulonephrosis. (H. E., 160).

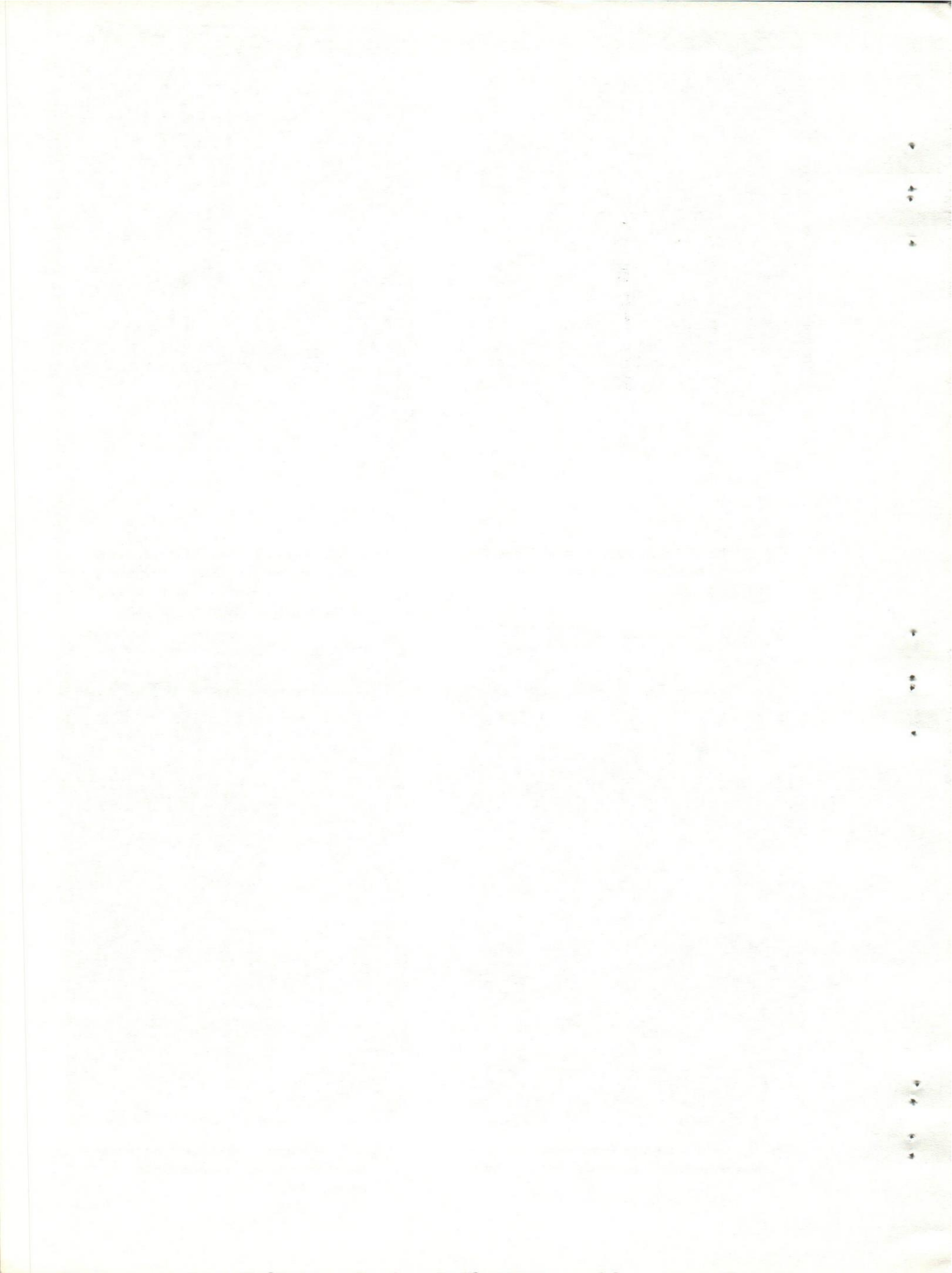




Fig. (5): Heart 3 days post-intranasal infection showing fibrinous pericarditis. (H.E., 160).

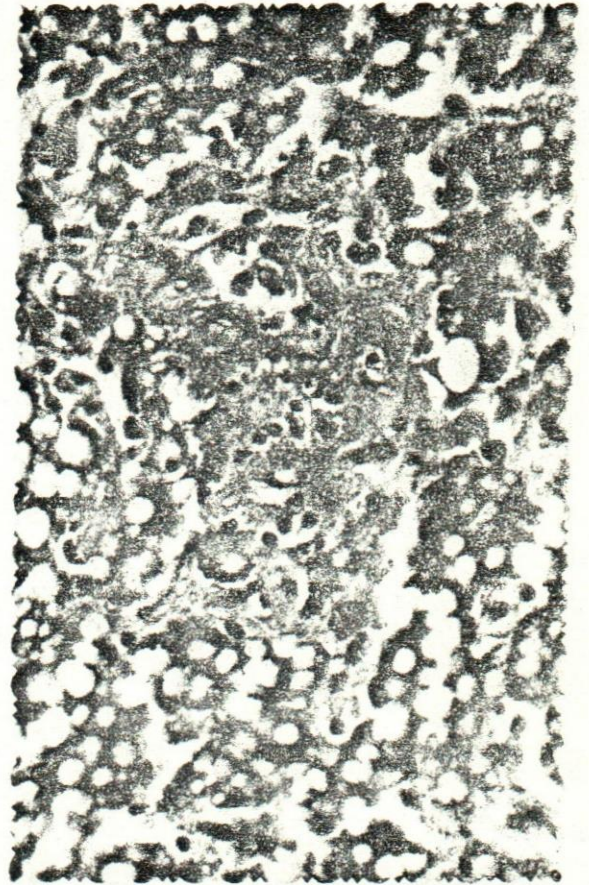


Fig. (6): Liver of natural cases showing necrotic lytic focus and fatty degeneration. (H.E., X 250).

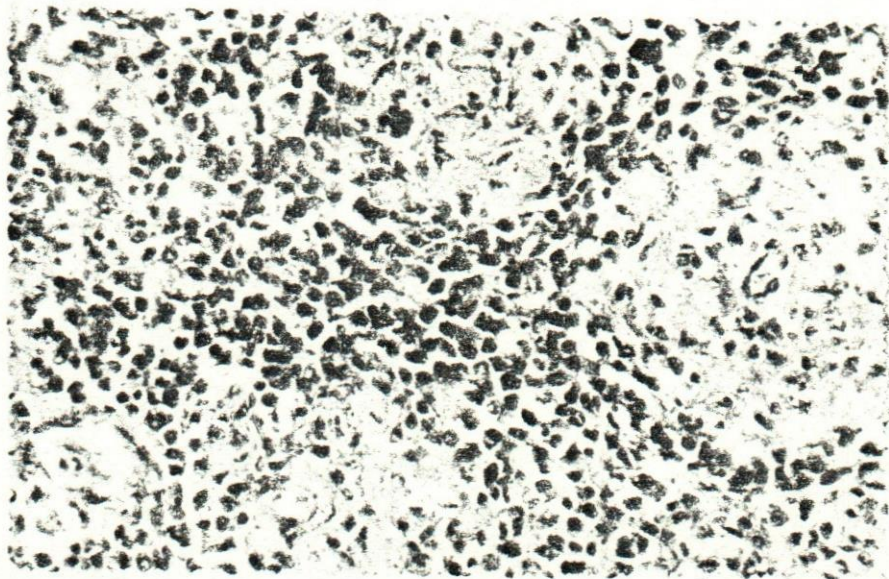


Fig. (7)

Spleen of natural cases showing : acrophagal reaction of the ellipsoid
(H. E., X 250)

