بثالوجيا العدوى التجريبية للنيواسكارس فيتولورم
فى خنـازير غنيـا

للدكتور م . أ . الشرى والدكتور م . ع . توفيق

الملخص

أعدى تجريبيا ٢١ خنزير غينى بواسطة بيض النيو أسكارس فيتولورم • وقـد ذبحت ثلاثة خنازير فى أوقات مختلفة بعد العدوى ، بعد ثلاث ساعات و ١٨ ساعة وبعد يوم ، ويومين وثلاثة أيام وخمسة أيام وبعد أسبوع من العدوى •

فقس البيض بعد ثلاث ساعات من العدوى فى الأليم ووجدت اليرقات فى النجويف البروتونى والكبد بعد ١٨ ساعة من العدوى • وبعد يومين وجدت مسارات اليرقات المهاجرة فى الكبد والرئة والمخ والكليتين .

وقد وجـد أن مسـار هجرة اليرقات من الكبد الى الرئة كان دائـم الحـدوث فى كـل الخنازير الغينى . ولكن مسارات الهجرة العضوية خلال المخ والكليتين وجدت فى حالات مفردة . وكانت تغيرات الأنسجة هى التكسير الميكانيكى من هجرة اليرقات وكذلك درجات مختلفة من الاستحالات فى الأعضاء المختلفة . وقد كانت دفاعات الأنسجة ضد وجود الطفيل غير واضحة الا فى الرئتين وقد لوحظ استجابة من كرات الدم البيضاء وخصوصا الأيوزينوفيل .

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THE PATHOLOGY OF EXPERIMENTAL INFESTATION OF NEO ASCARIS VITULORUM IN GUINEA PIGS.

(9 Illustrations)

By

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SUMMARY

21 Guinea pigs were experimentally infested by eggs of Neo ascaris vitulorum. 3 Guinea pigs were sacrificed at different times: 3, 18 hours, 1, 2, 3, 5 and 7 days postinfestation. Hatching occurred in the ileum after 3 hours. 18 hours later, the larvae were found in the peritoneal cavity and liver. Within 2 days the migratory tracts were found in the liver, lungs, brain, and kindeys. Migration through liver to the lungs was a constant route in all guinea pigs. Somatic migration through the brain and kidneys was of sporadic incidence.

Tissue alterations were mechanical damage from larval migration and variable degrees of dystrophic changes in the parenchymatous organs. Reaction to the presence of parasites was not expressed except in the lungs. Leucocytic response was observed in which eosinophilia was predominating.

INTRODUCTION

In Egypt, the incidence of Neo ascaris vitulorum infestation was recorded to be 36.36% among buffaloes of various ages (TAWFIK, 1970). This incidence was specially high 74.4% in 40 days old buffalo calves (SELIM and TAWFIK 1966). The economical losses of parasitism lies in retardation of animal growth, lowering the state of its resistance, illness and actual death of animals.

Knowledge of the biology and pathogenesis of Neo ascaris vitulorum is fundamental for control of animal parasitism. Prenatal infestation was established by Neo vitulorum in buffaloes by SRIVASTAVA and MEHRA (1955) and TAWFIK (1970). Post-natal infestation failed to be established by HERLICH

and PORTER (1953, 1954) ; REFUERZO and ALBIS-JIMINEZ (1954). The post-natal infestation was established only in very young calves or animals under stress factor as pregnancy or old age (DAVTYAN, 1931 and TAWFIK, 1970). Embryonation of egg and factors influencing it, was studied in vitro by many authors ; REFUEROZ *et al.* (1952), LAPAGE (1962), ENEYNIHI (1969).

Studying the route of larval migration in the animal body necessitates a model experimental animal. Mice, rat, and guinea pigs were used. CASAROSA (1951, 1953), REFUEROZ (1952) and HERLIGH (1955) stated that the migration of Neo ascaris vitulorum larvae fellows the lung journey or the tracheal type of migration. LEE (1958) and SOULSBY (1965, 1968) criticized the forementioned results and indicated that Neo ascaris vitulorum larvae simulate Toxocara cains larvae and undergo the somatic type of migration.

Data dealing with the Pathological effect of experimental infestation are still numerable.

In the light of these conflicting results, the aim of this work is to set up experimental infestation of Neo ascaris vitulorum in guinea pigs in order to explore the route of migration of the larvae in side the animal body, the type of tissues alterations and the reaction of the animal tissues to the presence of parasite.

MATERIAL AND METHODS

The experiment was carried on 21 guinea pigs of 4-6 months old. Infective eggs of Neo vitulorum were introduced orally through a plastic syrings with a needle to which a piece of rubber tubing was attached. The dose of eggs was 200,000 for each animal.

Ascaris eggs were obtained by dissection of the distal portion of an adult female Ascaris uteri, gathered at slaughter house. The eggs were incubated in equal part solution of 3% sodium hydroxide and 3% solution sodium hypochlorite for 24 hours to reach the infective stage.

Three guinea pigs were sacrificed at each time after the elapse of the following periods : 3 hours, 18 hours, 1, 2, 3, 5, and 7 days. Postmortem examination was carried. After removal of the alimentary tract, the stomach, duodenum, jejunum, ileum, caecum, colon and rectum were dissected separatly. Each part was cut open into suitable container and its content were examined to detect the presence of eggs, egg shells or larvae by means of compressorium.

The internal organs as the brain, lungs heart, liver, kidneys, spleen, and genital organs were removed and studied for histopathological changes. Tissue slices not more than 1.0 × 1.5 and 0.5 cm. were fixed in formalin and embeded in paraffin. Sections of 7 microns were stained by H. & E.

RESULTS

3 *hours post-infestation*

The parenchymatous organs were more or less of normal gross appearance. The stomach and caecum contained the majority of eggs. Hatched eggs were observed in the ileum. Few numbers of eggs were observed in the rest of intestine.

The normality of the parenchymatous organs was also confirmed by microscopic examination.

18 *hours post-infestation*

The brain was hyperaemic in one animal. The lungs showed areas of emphysema. The heart was normal. Larvae were detected in the peritoneal cavity with slight amount of peritoneal exudate. The larvae as well were detected in the liver parenchyma. The liver was slightly congested, showing haemorrhagic spots under the serosa. The kidneys were normal. Few eggs were present in the stomach, small intestine, caecum and colon.

Microscopically, in one animal the cerebrum and cerebellum showed slight degree of focal hyperaemia. Migratory tracts of parasitic larvae were observed in the white matter of the cerebrum. The tracts had a ragged borders and containing erythrocytes (Fig. 1). The midbrain showed foci of demylination. The rest of the animals were of normal brains.

The lung parenchyma demonstrated area of emphysema and atelectasis. The bronchiolar walls were hyperaemic. Some bronchioles showed epithelial desquamation and slight leucocytic infilteration.

The liver showed the migratory tracts, lined by necrosed and ragged liver cells and filled by erythrocytes. The parenchyma manifested slight degree of hydropic proteinous dystrophy.

Slight focal reaction in the wall of the caecum and colon was manifested by leucocytic infiltration of submucosa together with hyperaemia and extravasation of R.B.C. Mucous dystrophy was detected. The rest of the organs were normal.

One day post-infestation

The post-mortem picture was more or less the same as that of 18 hours post-infestation. The exceptions were that :

The brain was normal. The heart showed slight greyish discolouration. The kidneys showed in one case slight congestion and few dark haemorrhagic spots. The other kidneys showed only the greyish discolouration. The The testicles in two animals were soft and flabby.

Microscopically, the brain was normal. The findings in the lungs were similar to that detected 18 hours post-infestation. The myocardium showed slight degree of granular proteinous dystrophy and slight perivascular histocytic activity. The kidneys parencyma in all animals manifested mild degree of granular proteinous dystrophy. In one case, the migratory tracts of parasitic larvae were detected in the kidneys parenchyma (Fig. 2) with hyperaemia. Testicular degeneration as indicated by aspermatogenesis and degenerated spermatids was detected in two animals. Haemosiderosis was detected in the phagocytic cells of the spleen.

Two days post-infestation

In all animals the brain was slightly congested. The lungs showed very small foci of hepatization. The heart, liver, and kidneys showed mild degree of dystrophy. Few eggs and egg-shells were present in the ileum and large intestine.

Microscopically, the brain showed no more than congestion. The lungs demonstrated hyperaemia of the bronchial walls, peribronchial and perivascular aggregation of neutrophiles, and eosinophiles. Lymphocytes were seen in follicles around some bronchioles. Scatered foci of few consolidated areas were filled by leucocytes, marcrophages, and lymphocytes.

The heart showed mild degree of proteinous dystrophy. The liver demonstrated the migratory tracts. The hepatic vessels were hyalinized. The vacuolar proteinous dystrophy of the parenchyma was zonal in some lobules and diffuse in others (Fig. 3). Small microfoci of liver cell necrosis were

detected together with haemosiderosis of kupffer cells. The vessels of the portal tract were degenerated. The kidneys showed mild degree of proteinous dystrophy, but no migratory tracts were detected. The spleen showed degeneration of the follicular arteries and exhaustion of the lymph follicles. (Fig. 4).

3 *days post-infestation*

The post-mortem picture was the same as that of 2 days post-infestation. Nearly all the parenchymatous organs were involved in pronounced dystrophic process. This was also confirmed by microscopic findings.

5 *days post- infestation*

The brain was hyperaemic and flabby in one animal. The heart, liver and kidneys showed pronounced dystrophic changes. The lungs demonstrated the picture of parasitic bronchopneumonia. Patches of greyish hepatization were scattered specially peribronchial. Foci of emphysema and atelectasis were also located.

Microscpically, in one case, the cerebrum and cerebellum was hyperaemic and migratory tracts were detected with area of necrosis in the granular layer of cerebellum (Fig. 5).

In the lungs, the epithelial desquamation of the bronchiolar wall was severe. There were peribronchial rich aggregations of eosinophiles and mononuclear cells. The bronchial arterioles were degenerated. The media showed vacuolar dystrophy of the smooth muscles. The adventitia was largely oedematous and infilterated by eosinophiles and few neutrophiles. The alveolar parenchyma showed large areas of consolidation, specially peribronchial, in which the alveoli were filled by neutrophiles, eosinophiles and septal cells. The rest of the alveoli showed atelectasis and emphysema (Fig. 6).

The proteinous dystrophy of the myocardium was expressed with vacuolation of the muscles. The degree of liver hydropic dystrophy was severe and diffusely manifested. The kidneys showed blood vessels degeneration, beside pronounced granular dystrophy. The dystrophic changes in the wall of the follicular arteries and in the lymph follicles of the spleen were prominent.

7 days post- infestation

The post-mortem picture was similar to that of 5 days post-infestation. Most of the larvae were found in the lungs and few in the liver. The degree of dystrophy was more pronounced in the parenchymatous organs.

Microscopically, the brain hyperaemia, migratory tracts, and granular necrosis were also detected. In the lung, the areas of alveolar consolidation were larger with eosinophilic abscesses formation (Fig. 7). The vessels degeneration was prominent.

The heart showed areas of myocardial necrosis (Fig. 9). The kidneys showed the migratory tracts in one case. The proteinous dystrophy and degeneration of the vassels wall were also prominent features in the other cases. The spleen showed follicular exhaustion and vessels degeneration.

DISSCUSSION

Oral adminstration of Neo ascaris vitulorum eggs to guinea pigs had established the picture of parasitism in them.

Guinea pigs can be regarded as representative of compound stomach animals. So the picture of their parasitism may be compared to that in sheep, cattle and buffaloes, as N.A. vitulorum infestation could not be experimentally established in the latter animals, by HERLICH and PORTER (1953, 1954) ; REFUERZO and ALBIS — JIMINEZ (1954).

3 hours post-infestation, hatching of eggs occurred in the ileum. That ileum is the common seat of hatching was also confirmed by the work of SPRENT (1952) and BHOWMICK (1964).

After hatching, the larvae penetrated the wall of the intestine to be found in the peritoneal cavity and liver 18 hours post-infestation. Within the group of 18 hours, one, and two days post-infestation, migratory tracts were observed in the liver, lungs and (although not in all cases) in the brain and kidneys.

The presence of the free larvae in the peritoneal cavity points to the mechanical route of penetration to the liver. That ascaris larvae must follow the portal circulation to the liver as stated by HOPPLI (1923) and ASADA (1926) is not necessary. BHOWMICK (1964) stated that the blood

route to the liver for ascaris larvae was considered an atypical one. In our opinion the migration of the larvae to the liver takes both ways : the mechanical penetration through the intestinal wall to the peritoneal cavity to the liver parenchyma and through blood portal ciculation to the liver. Further migration throught blood venous circulation takes place to the lungs, then the heart and from the arterial circulation from the heart to the brain and kidneys. This was indicated by the presence of the larval migratory tracts and vessels degeneration in these organs.

The migration of the larvae to the liver (observed 18 hours post-infestation) and to the lung (observed 2days post-infestation) was a constant finding in all guinea pigs. Migration to the brain and kidneys was of sporadic incidence. This results indicated that, the liver lung — tracheal journey described by CASAROSA (1951-1953), REFUERZO *et al.* (1952) and HERLICH (1953) was the common route of migration.

The larvae penetrated to the liver, go to the right side of the heart through the venous circulation and reach the lung. In the lung they travel along the trachea and will be swallowed again in the stomach and intestine.

In the present experiment, migratory tracts in the brain and kidneys indicated that somatic migration occurred in guinea pigs. This result coincides wth the opinion of LEE (1958) and SOULSBY (1965, 1968). The fact that prenatal infestation of Neo ascaris vitulorum was observed in calves by SRIVASTAVA and MEHRA (1955) and TAWFIK (1970) also proves the somatic migration. But the sporadic occurrance of migratory tracts in the brain and kidneys, in the present materials pointed that, this route of migration is not the usual one.

Tissues alterations in guinea pigs were mechanical damage from larval migration and variable degrees of dystrophic changes in the paraenchymatous organs were a secondary result.

Migratory tracts in the liver and lungs were present in all guinea pigs. The liver parenchyma manifested granular proteinous dystrophy 18 hours post-infestation. 2 days after infestation the process proceeded to severe hydropic dystrophy and ended to coagulative necrosis after 7 days.

MARCARIANZ (1933) and GRIGER (1934) described human liver lesions which was caused by migration of the adult ascaris worm to the bile duct. Such migration during pregnancy was followed by abortion.

OLDHAM and WHITE (1944) and ZENDUIKA (1960) showed experimentally in pigs and TAFFS (1965) in guinea pigs and rabbits that focal interstitial chronic eosinophilic hepatitis was associated with migration of Ascaris suum larvea. KAZIOWICZ (1972), described a reactive inflammation of the naturally infected liver.

In the lungs, frank areas of consolidation appeared only after two days of infestation. The typical picture of parasitic bronchopneumonia was established 5 days from infestation. 7 days later, eosinophilic abscess formation was observed. Data on tissue alterations through experimental infestation of Neo ascaris vitulorum are not richly available. CASAROSA (1951 & 1953) described the occurrence of greenish spots in the liver and lungs of guinea pigs, which correspond to parasitic nodules. ALLEN (1962), MARROW (1968), had described pneumonia in calves and heifers caused by ascaris lumbericoides larvae. Histopathologically diffuse interstitial pneumonia was demonestrated together with the presence of larvea. MCRAW and LAUTENSLAGER, (1971) had described pneumonia in calves associated with migratory scaris suum larvae.

Larvae were found in the bronchioles and alveoli. The walls of the alveoli were thickened and their luminae contained a proteinous fluid, mononuclear cells and few eosinophiles. These changes were accompanied by areas of diffuse haemorrhage in the alveoli.

Migration of Neo ascaris vitulorum larvae through the brain and kidneys was of sporadic incidence. In the brain, the migratory tracts were accompained by hyperaemia. In one case it was accompanied by necrosis in the cerebellum.

TIMER (1953) in his experimental infestation of mice by : A. columnaris, T. canis, A. leovis found that in mice killed 10 days post-infestation, that the larvae were frequently present in the cerebrum, cerebellum as well as the medulla and other parts of the brain. This fact of brain larval infestation may throw light on the cause of neurological convulsions described in calves as well as in man with nematodal infestation.

JUBB and KENNEDY (1963) wrote that as there was no evidence of the presence of parasites or larvae in the nervous system, this parasitic nervous signs are explained as an allergic reaction developing in host which has been sensitized to previous antigen absorbed from the intestine.

MATOFF and KAMANDRAEV (1965) in his experimental infestation of mice and guinea pigs showed that larvae of T. transfuga reached the brain, while those of T. leonina did not. In the brain the larvae did not encyst, but continued to grow.

In the kidneys, the proteinous parenchymatous dystrophy was a constant finding among guinea pigs.

No migration was observed in the heart and the spleen of guinea pigs. Slight myocardial dystrophy was observed 2 days after infestation. 7 days post-infestation myocardial necrosis was observed. The spleen showed degeneration of the follicular arteries and lymph follicles which was prominent 7days post-infestation.

Tissue reaction to the presence of the parasites and larvae was not expressed except in the lungs. The lungs showed exudative leucocytic response in which the eosinophilea was predominating. The heart, kidneys, liver, spleen and brain. These organs no more than not well expressed hyperaemia. The liver in some cases manifested slight histocytic reaction of the portal tracts. These are a reaction of universal significance for any injury, tissue damage or may be allergic (JUBB AND KENNEDY, 1963) and are not specific for the presence of parasites. In the present materials, Neo ascaris vitulorum infestation did not initiate any reaction in guinea pigs, up to 7 days post-infestation except exudatve leucocytosis in the lungs with eosinophilia.

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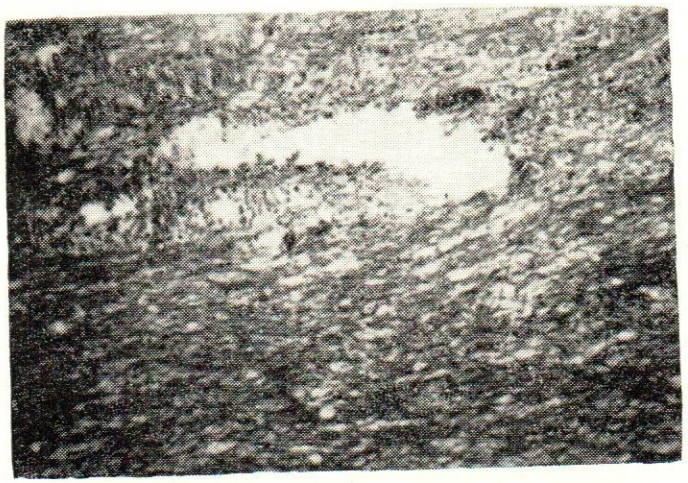

Fig. 1.—Larval migratory tract in white matter of cerebrum. 18 Hours post-infestation. H. and E. (10 × 12.5).

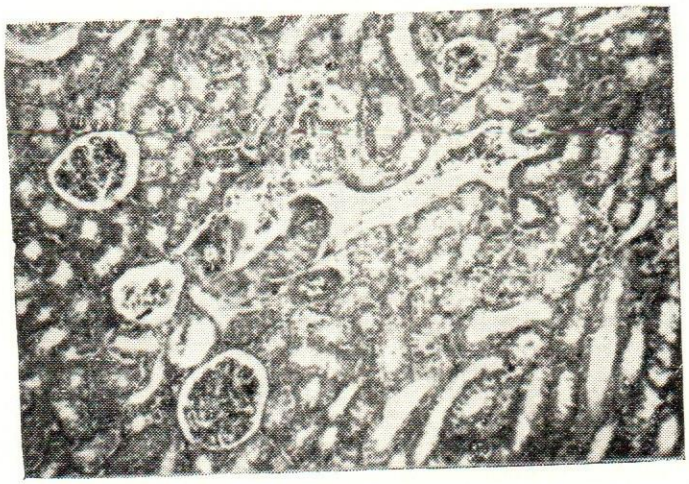

Fig. 2.—Larval migratory tract in the kidney. One day post-infestation. H. and E. (10 × 12.5).

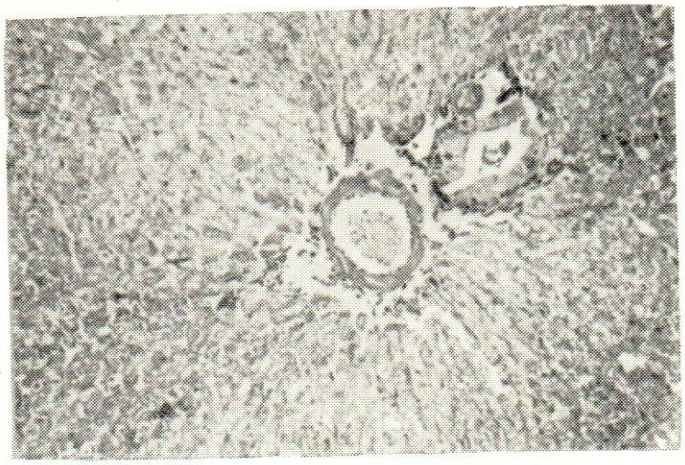

Fig. 3.—Hydropic dystrophy and degeneration of the hepatic vessels. 2 day post-infestation. H. and E. (10 × 12.5).

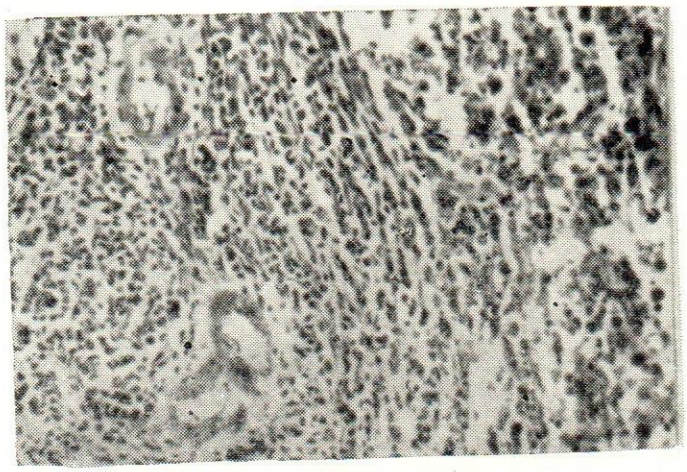

Fig. 4.—Degeneration of the follicular arteries and exhaustion of the lymph follicles in the spleen. 2 day post-infestion. H. and E. (20 × 12.5).

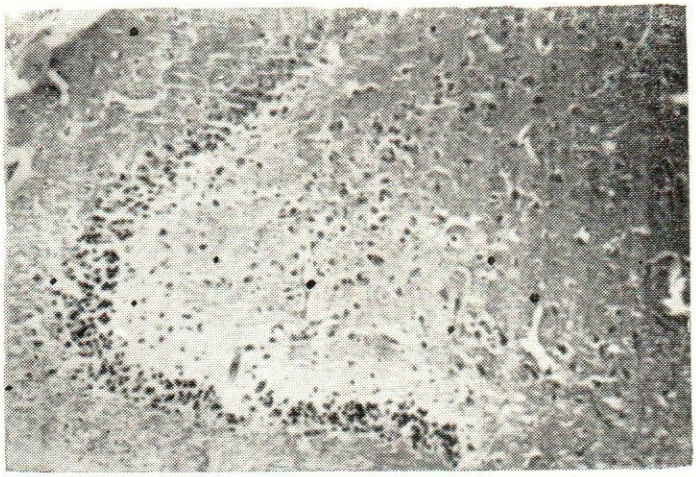

Fig. 5.—Necrosis of the granular cell layer of the cerebellum. 5 days post-infestation. H. and E. (10 × 12.5).

Fig. 6.—Parasitic broncho-penumonia. 5 days post infestation H. and E. (2.5 × 12.5).

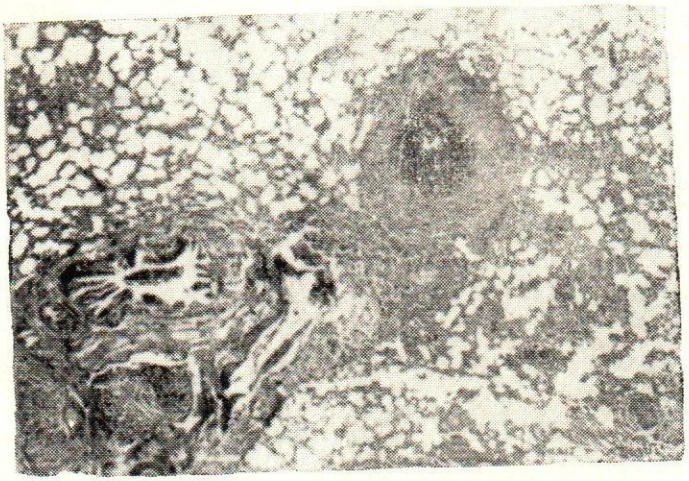

Fig. 7.—Parasitic broncho-pneumonia with eosinophilic abscess formation. 7 days post-infestation. H. and E. (2.5 × 12.5).

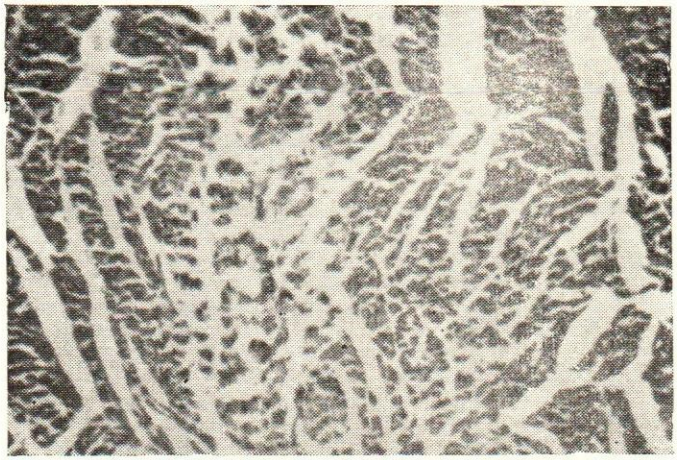

Fig. 8.—Myocardial necrosis. 7 day post-infestation. H. and E. (10 × 12.5)

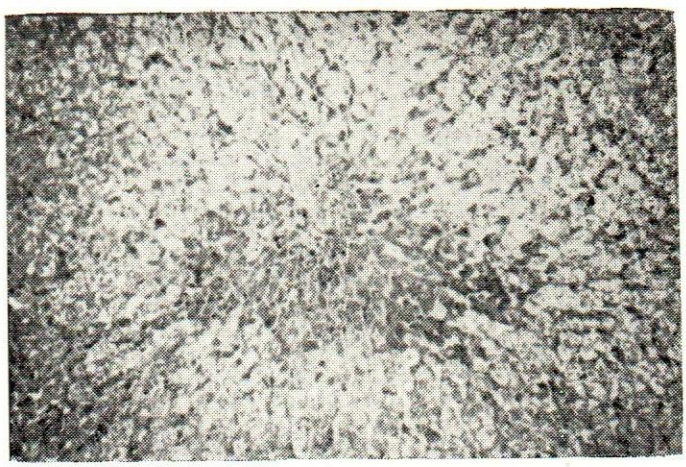

Fig. 9.—Liver coagulative necrosis and hydropic dystrophy of the parenchyma. 7 days post-infestation. H. and E. (10 × 12.5).