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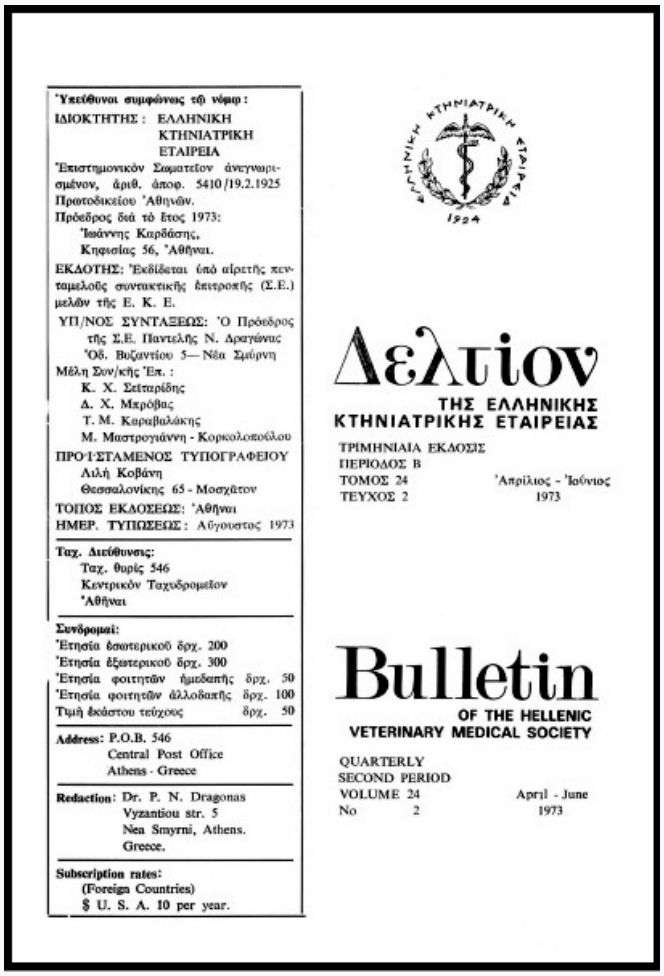

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B. C. HATZIOLOS

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Departments of Veterinary Science and Animal Health.
University of Maryland, College Park, Maryland 20740.

SYSTEMIC ASPERGILLOSIS IN A CALF*

By

BASIL C. HATZIOLOS

Mycotic infections have been known for a long time. They were first noted in birds (Corvus glandarius) in 1915,[10] in man in 1839,[19] and in mammals in 1868,[15]. A pneumonomycosis - producing aspergillus in cattle was first reported in 1884,[16].

Aspergillosis in cattle is relatively rare[1]. As in other animals, this infection in cattle may affect one or several organs and thus be manifested in various ways. Given the rarity of its occurrence in cattle, the pulmonary form is the most common and the gastro - intestinal, the rarest[23].

Pneumonomycosis is often seen in European countries[17], particularly in the U. S. S. R., where enzootic and systemic infections have also been observed[3,9]. In the U. S., the most frequently recognizable form in cattle is that associated with placental infection, causing abortion. Surveys have revealed asteroid structures - possibly indicating resistance - in the lungs of 66% of slaughtered cows in England[2], but this high incidence was not found in the cattle in the U. S.[14]. Prominence of aspergillosis was attributed to feeding on moldy sweet potatoes or to use of moldy litter[22].

Whereas the pulmonary form is encountered sporadically and the systemic infection is uncommon, the nervous form, to our knowledge, has not been reported in cattle. The present report is concerned with a clinically unsuspected case of systemic aspergillosis involving a 6 - month - old calf having lesions in the lungs, brain, liver, and abomasum.

CASE REPORT

A 6 - month - old purebred female Hereford, one of a group of 16 cal-

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Author' s address : Prof. Dr. Basil C. Hatziolos, Veterinary Science Department, University of Maryland, College of Agriculture, College Park, Md. 20740, U. S. A.

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ves bought directly from a farm in the Leesburg, Va. area, developed clinical signs of disease within two weeks after shipment to a Maryland farm.

The onset of disease was insidious and the clinical manifestations were vague. They consisted of slight grogginess, abnormal head postures (hanging head), rough coat, depression, and mild diarrhea. Body temperature was normal. Despite symptomatic therapy, the calf got worse and died 2 weeks after the onset of clinical signs of the disease.

Two other calves of this group had died 4 weeks previously, with signs somewhat similar to those of the present calf. Neither necropsy nor presumptive diagnosis was made in those cases.

GROSS PATHOLOGIC FINDINGS

Necropsy, performed 10 hours after death, revealed good physical condition, including large amounts of adipose tissue deposits. The skin was moderately dehydrated. There was extensive hepatization of the diaphragmatic and cardiac lobes of the lungs, with purulent fluid and fibrin deposits on their surface. The pulmonary parenchyma was dark red, edematous, and studded with numerous superficial and deep nodules approximating 2 - 5 mm. in diameter. Emphysematous areas were localized in the dorsal parts of the lungs. The pleurea were thickened and opaque, having round, grayish plaques with a rough surface among hemorrhagic spots. Adhesions between the pulmonary lobes and parietal pleura were extensive. Fluid in the thoracic cavity was thick but scant. The trachea was reddened and filled with hemorrhagic foamy material.

Small necrotic areas were in the liver. The gall bladder was distended and contained jelly - like, dark green, bile.

Several ulcerated spots approximately 3×4 cm in diameter were in the rumen, as was denudation of larger areas in the abomasum where several *Haemonchus contortus* were found. Black nodular structures were scattered throughout the intestinal tract, extending to the tunica muscularis.

The heart had petechiae of the endocardium, discolored areas in the musculature of both ventricles, and nodular thickening of the valves. The kidneys had superficial dark red areas. The spleen was small and fibrotic.

Multiple punctate hemorrhagic spots and necrotic lesions were observed in almost the entire left occipital lobe of the cerebrum. involving both the leptomeninges and parenchyma. The rest of the cerebrum was moderately congested, and somewhat soft. The medulla oblongata and cerebellum appeared normal.

Microscopic fecal examination revealed eggs of *Nematodirus* spe-

cies. Results of chemical analysis for lead and arsenic were negative. Cultures of lung tissue for fungus isolation were made, but were lost due to a laboratory accident. Escherichia coli was recovered from practically all organs.

HISTOPATHOLOGIC FINDINGS

The nodules in the lungs varied in size. They were unevenly distributed, but circumscribed and sharply marked (Fig. 1).

The early stage of nodule formation was characterized by dilatation of the bronchiolo - alveolar apparatus, marked exudation in the terminal stru-

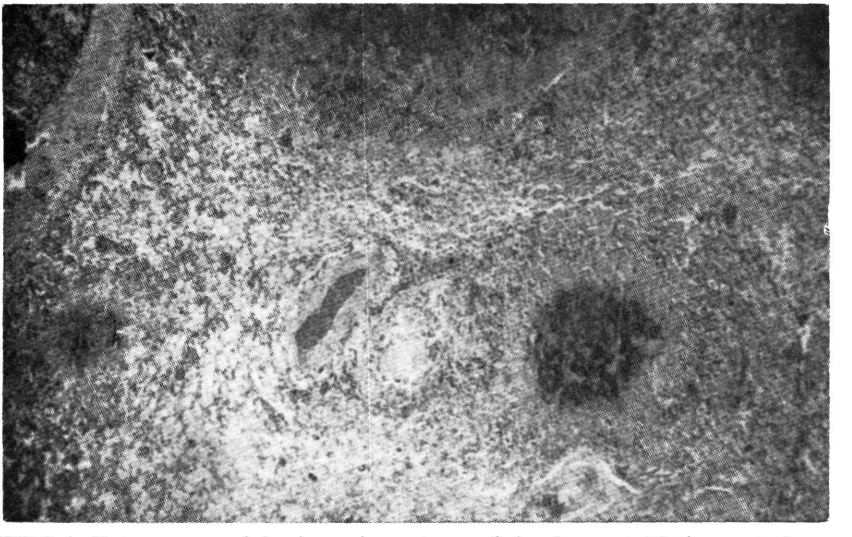

FIGURE 1. Pulmonary nodules in various stages of development. Notice central necrotic areas containing hyphae surroumded by clear zone and tendency toward encapsulation. Scattered giant cells, alveolar edema, and exudation effacing normal architecture of pulmonary parenchyma were distinct in higher magnification. E & H x 63.

cture, and accumulation of serofibrinous fluid in which the amount of fibrin increased with the stage of development. Subsequently, alveolitis with beginning necrosis of the alveolar exudates or a necrotic fusion of the figured elements caused effacement of surrounding structures, leaving recognizable traces at the periphery. Mycelial elements, resembling those of Aspergillus sp., appeared in the center of the faintly outlined young nodules. Necrosis around mycelial fragments attached to the bronchioles apparently followed the development of the fungus, thus accounting for enlargement of the nodule.

In well - developed nodules, 3 distinct zones were recognizable. Central-

ly, a deeply stained circular area measuring 200 - 300 μ in diameter was prominent. This area was formed by caseation necrosis and the accumulation of eosinophilic material, cellular debris, and mycelial elements.

In the succeeding zone, the cellular debris was less marked, but the serofibrinous material was abundant. In this area, the fungus was developed abundantly by arborization of mycelial branches. From the periphery of this area, branching septate hyphae 3 to 5 μ in width and varying in length radiated outward in an irregular manner (Fig. 2). In old nodules, the zone was broader and included widely radiating hyphae (Fig. 3).

An external, wide zone of granulation tissue tended to separate nodules from less affected tissues. Growing nodules compressed the surrounding alveoli and caused them to collapse. There were few scattered giant cells, an aberration from the usual cellular arrangement in avian aspergillic pul-

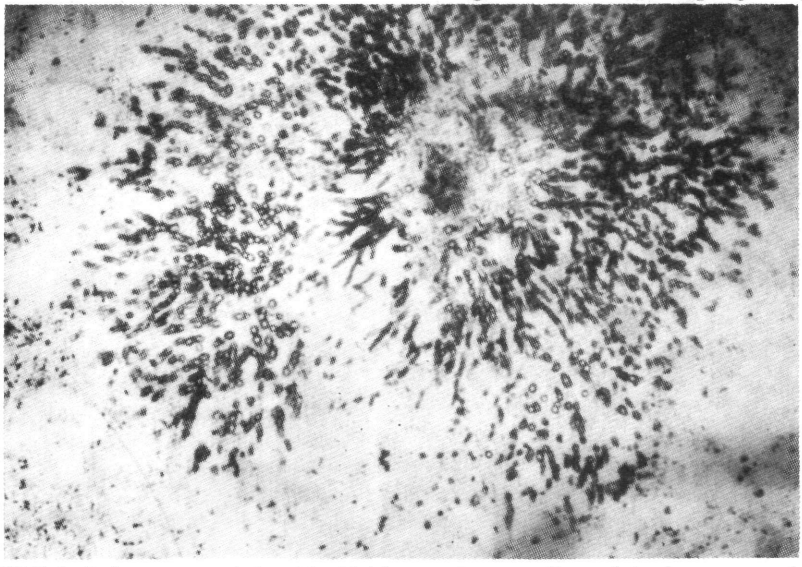

FIGURE 2. Pulmonary nodule with thickly growing mycelia and hyphae arranged in a radiating pattern. PAS x 160.

monary lesions. Edematous alveolar areas with fibrin deposits surrounded the affected nodular area. Edema in the intercellular spaces, exudation, and eosinophilic areas suggested fibrinoid degeneration or necrosis. The necrotic areas were usually round and, with the adjacent cellular reaction, resembled tubercles.

The afore mentioned zones were not distinct in all nodules. Some contained exceedingly numerous neutrophils; others were diffuse and unidentifiable. The remaining areas of the consolidated parenchyma were inten-

sely congested and had fibrin - hemorrhagic reaction and leukocytic infiltration. Most of the alveoli had thickened walls and were distended by the accumulation of sloughed epithelial cells, erythrocytes, macrophages, and fibrin deposits. A few areas of the pulmonary parenchyma were either left intact or had emphysematous alveoli. Asteroid structures were not found.

The bronchial and bronchiolar walls were markedly altered and were filled with mucopurulent material formed by desquamated epithelial cells, many granulocytes, and, occasionally, free erythrocytes. Fungal elements

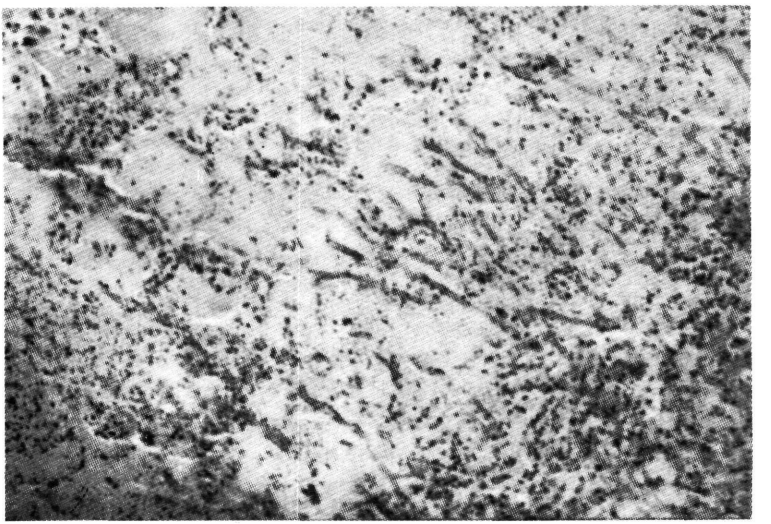

FIGURE 3. Alveolar edema, exudation, and accumulation of detritus in pulmonary parenchyma; numerous hyphae arranged in a radiating pattern at the periphery of an old nodule. H & E x 160.

were not found in these areas. The trachea had sloughing of the epithelium, hemorrhages, and interstitial edema in the submucosa and underlying tissues. Bacterial colonies, mostly cocci, were occasionally present.

The hepatic parenchyma was congested and studded with minute hemorrhages; some hepatic vessels were thickened. Scattered fatty changes and necrotic diffuse inflammatory reaction had occured. The growth of the fungus there was less abundant than in the lung (Fig. 4).

The heart was congested and there was interstitial edema, muscular atrophy, scattered nuclear pyknosis, thickened endocardium, and, occasionally, foci of leukocytic infiltration.

In the kidney, changes were limited to albumin precipitates in the convoluted tubules.

The spleen had partial lymphocytic depletion of the malpighian corpuscles, reduced red pulp, and large amounts of hemosiderin. The capsule was thickened and extended prominent trabeculae.

The rumen had necrotic areas and ulceration of the mucosa, with focal inflammatory reaction and bacterial cells, similar to that noted in the trachea. Several hemorrhagic spots, necrosis of mucosa, shalow ulcer formation with focal inflammatory reaction, and fibrin deposits were encountered in

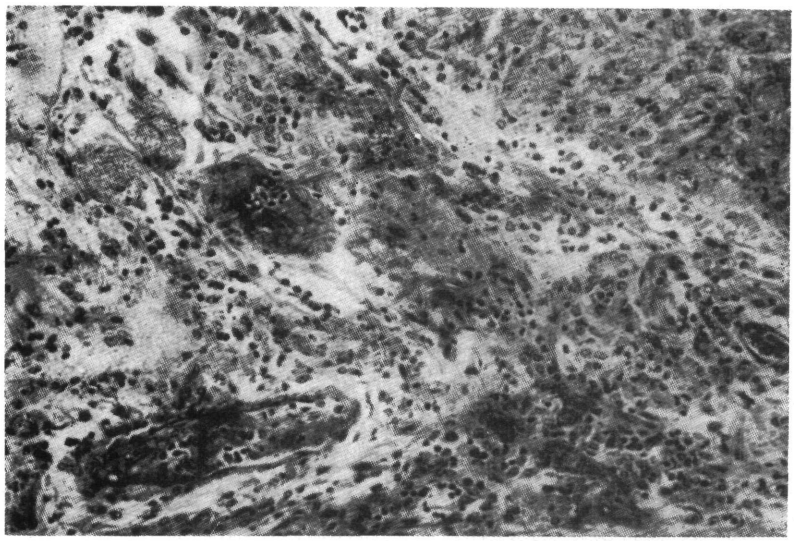

FIGURE 4. Necrotic area of liver with exudation, granulocytic reaction, and scattered small groups of hyphae within or outside of the vessels. PAS x 160.

the abomasum. A few septate hyphae were in the edges of some of the ulcers.

The mesenteric lymph nodes were congested, edematous, and infiltrated with plasmacytes.

In the cerebrum, the meningeal inflammatory involvement was marked. Vascular thrombosis and fungal elements were in the inflamed areas and occasionally within the veins of the leptomeninges. Most of the vessels there contained fibrin threads simulating hyphae in the coagulated blood.

In a large infarcted area of the occipital lobe, hemorrhages, extensive necrosis of the parenchyma, liquefaction, thrombosis, and destruction of the vascular walls were common (Fig. 5). Some of the vessels were collapsed or compressed and others, dilated by erythrocytes and PAS - positive material. Many vessels contained hyphae (Fig. 6).

The fungal material seemed to be less abundant in the arteries than in the veins, which were distended by blood. Mycelia were growing in the brain

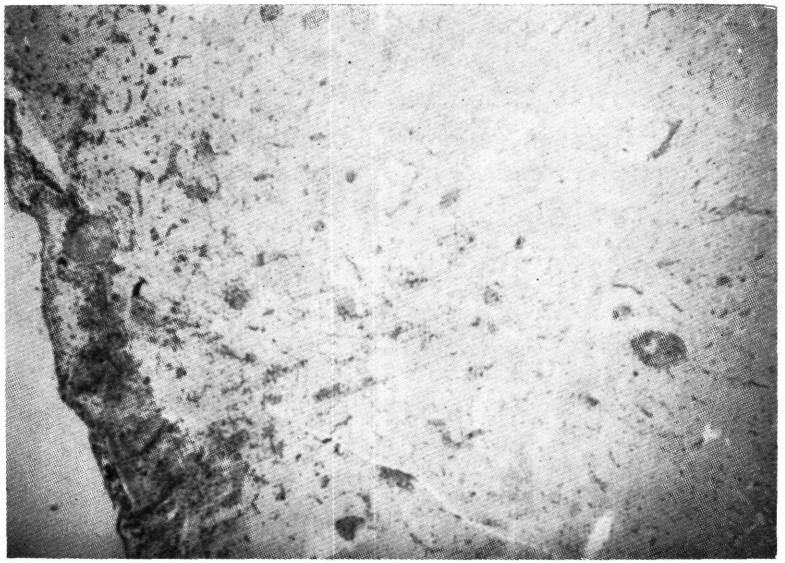

FIGURE 5. Large necrotic (infarcted) area in the occipital lobe; dilated or damaged vascular walls; hemorrhages and gliosis. H & E x 25.

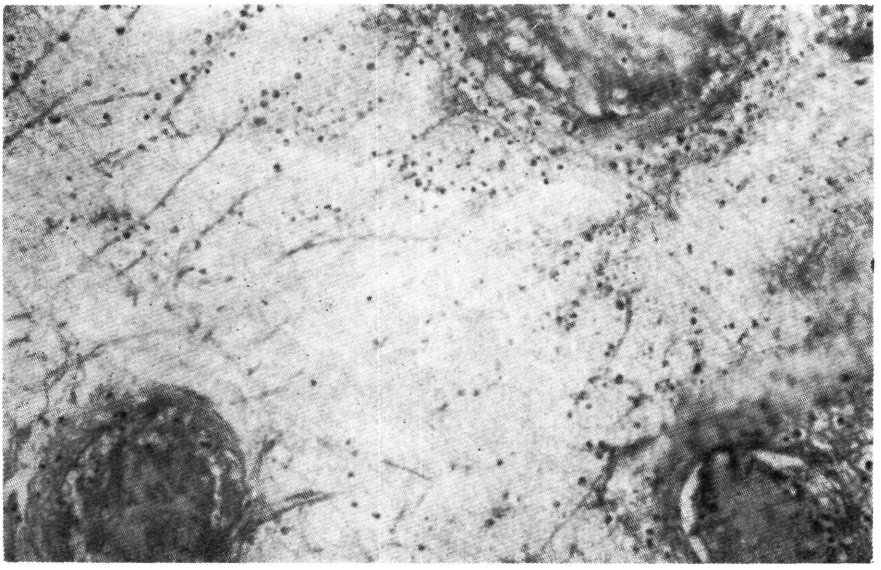

FIGURE 6. Necrosis of intervascular areas of occipital lobe of cerebrum with numerous hyphae and free erythrocytes. necrosis of vascular walls, and thrombosis. Vessels containig hyphae in the lumen. PAS x 160.

parenchyma, around the vessels containing the fungus; however, the fungus was confined within the vascular walls in many instances. Small vessels and

rarely capillaries contained fungus in some areas (Fig. 7), in contrast to some large ones that did not contain any fungal elements. Inflammatory reactions appeared in linear or circular arrangement around vessels and in perivascular regions. In the white matter of the less affected areas, axons were swollen or fragmented and myelin sheaths were ballooned.

The medulla oblongata had dilated vessels, perivascular edema, and thinned out spots in the area superior to the lateral cuneatus. Thrombosis or fungal growth was not found in the vessels or in the parenchyma. There were

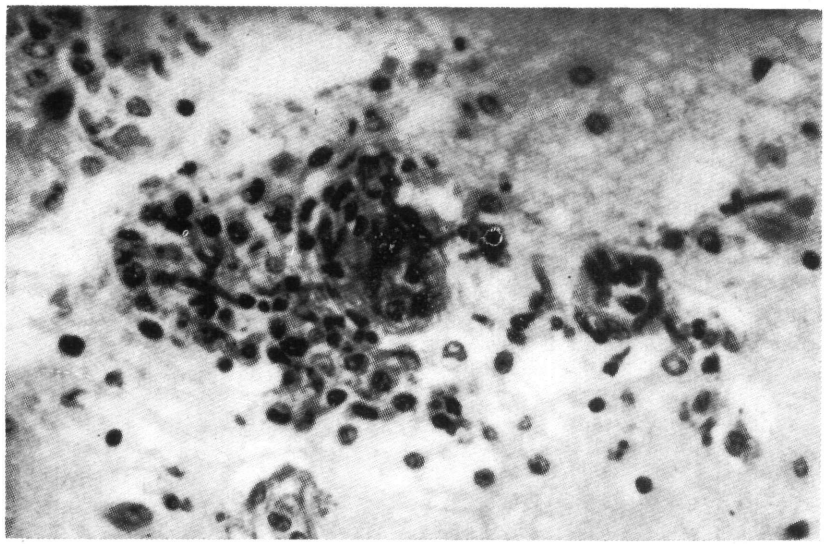

FIGURE 7. Perivascular cellular reaction including aspergillic elements in areas in proximity to infarction. PAS x 400.

edematous nerve tracts, dilated periaxonal sheaths, and arteries with thickened walls. The cerebellum was only moderately congested, suggesting that the aforementioned changes were not all the result of postmortem autolysis. The choroid plexus was slightly edematous, with increased cellularity in the interstitium.

DISCUSSION

Pulmonary aspergillosis in cattle, either in its acute form, which brings about death within 48 to 72 hours from acute pulmonary emphysema, or in its chronic form, which is characterized macroscopically by the development of multiple discrete granulomatous nodules simulating tuberculosis, is seldom encountered. Such cases occur among cattle remaining permanently in barns, where exposure to fungus spores from moldy feed or litter

might be extensive and continuous. This observation suggests that the bovine species naturally possesses a certain resistance to this kind of fungus infection.

On the basis of this concept, it is broadly accepted that the fungi cannot develop in the tissues of live animals unless determinant local factors favoring the growth of altered microflora exist, or unless the animal' s resistance is already markedly weakened by parasitism, debilitating diseases, or serious metabolic disturbances. Also, aspergillus infection often follows prolonged medication with antibiotics, cortisone[8, 20, 21], and other chemical compounds that stabilize the lysosomes of macrophages[6, 12, 24, 25].

However, serious consideration should be given to the development of an aggressive status of the Aspergillus, resulting in infection by continuous massive exposure to spores in association with some predisposing factors.

The scarcity of reports on systemic aspergillosis might be due in part to (1) cases that have beer overlooked or neglected, taking into consideration that the exact diagnosis is based on cultural and histologic examinations, (2) the widespread opinion of a saprophytic role of this fungus, (3) the erroneous belief that mycosis develops only in countries with warm climate, and (4) the frequent development of complications that tend to overshadow the basic cause.

The clinical manifestations of the calf in this report, though in some ways similar to heavy metal poisoning, were nonspecific and misleading. This emphasizes the difficulty in making a diagnosis of aspergillosis in live animals[4, 26, 27]. Even at necropsy, difficulties may be encountered in diagnosis because the gross pulmonary changes are similar to those of other respiratory diseases[16].

Histologic examination of the lungs, revealing characteristic nodular formation with 3 distinct zones and abundant fungal material, facilitates an accurate diagnosis. Histogenetically, the nodular formation arises from a necrotic fusion, in circumscribed foci, of the exudate and the parenchyma within the center of the bronchiolar - alveolar inflammation, which results from localization of fungal elements in the bronchioli. Consequently, the fungal elements in their development penetrate the bronchial walls and settle in the central cavity of the nodule. Pulmonary infection seems to be achieved through an aerogenous route, but a hematogenous one seems feasible.

In the cerebrum, vascular thrombosis and hemorrhagic infarction were prominent lesions. Vessels and perivascular regions were often the major sites of both hyphae development and inflammatory reaction. These changes, although localized in the occipital lobe, matched the pattern of Aspergillus lesions reported in man[11, 13, 18, 28] and in mice[7]. The lack of a granu-

lomatous reaction was probably due to the early stage of the cerebral infection. Also, the presence of fungal elements in the dilated vessels of the cerebrum suggest transport by the hematogenous route to various organs; however the meningeal vascular system did not seem to be involved in dissemination of the fungus.

The rarity of Aspergillus infection in the central nervous system may be explained by the possible existence of a mechanism impairing spores from germinating and proliferating or by barriers to metastasizing fungal elements.

SUMMARY

A 6 - month - old Hereford female calf with vague clinical manifestations was found at necropsy to be affected with systemic fungal infection extending to the lungs, brain, liver, and abomasum. Microscopically, the fungal elements in the lesions resembled Aspergillus sp. Nodules in the lungs had necrotic centers with numerous mycelial elements surrounded by a granulomatous reaction.

In the cerebrum, vascular thrombosis and multiple spots of hemorrhagic infarction and necrosis were prominent. Numerous fungal elements were found within and around affected vessels. In the liver and abomasum, lesions and fungal elements were scant. This is the first such case to be reported in the veterinary literature.

ΣΥΣΤΗΜΑΤΙΚΗ ΑΣΠΕΡΓΙΛΛΩΣΙΣ ΕΙΣ ΜΟΣΧΟΝ

Ὑπὸ

Β. ΧΑΤΖΗΟΛΟΥ

ΠΕΡΙΛΗΨΙΣ

Μοσχὶς 6 μηνῶν, φυλῆς Hereford, παρουσιάσασα ἀκαθόριστα κλινικὰ συμπτώματα εὑρέθη κατὰ τὴν νεκροψίαν προσβεβλημένη ὑπὸ συστηματικῆς μυκητιακῆς λοιμώξεως ἐνδιαφερούσης τοὺς πνεύμονας, ἐγκέφαλον, ἦπαρ καὶ 3ον στόμαχον. Μικροσκοπικῶς τὰ μυκητιακὰ στοιχεῖα ἐντὸς τῶν ἀλλοιώσεων προσωμοίαζον πρὸς τὸ Aspergillus sp. Τὰ ὀζίδια εἰς τοὺς πνεύμονας ἐνεφάνιζον νεκρωτικὰ κέντρα μετὰ πολυαρίθμων μυκητιακῶν στοιχείων, περιβαλλομένων ὑπὸ κοκκιωματώδους ἀντιδράσεως.

Εἰς τὸν ἐγκέφαλον προεῖχον ἡ θρόμβωσις τῶν ἀγγείων, πολυάριθμα σημεῖα αἱμορραγικῆς ἐμφράξεως καὶ νεκρώσεως. Πολυάριθμα μυκητιακὰ στοιχεῖα ἀνευρέθησαν ἐντὸς καὶ πέριξ τῶν προσβεβλημένων ἀγγείων. Εἰς τὸ ἦπαρ καὶ τὸν 3ον στόμαχον αἱ ἀλλοιώσεις καὶ τὰ μυκητιακὰ στοιχεῖα ἦσαν σπάνια.

Πρόκειται περὶ τοῦ πρώτου τοιούτου περιστατικοῦ εἰς τὴν Κτηνιατρικὴν βιβλιογραφίαν.

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