A Canine Case of Profound Granulomatosis due to Paecilomyces Fungus

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ABSTRACT. A 5-year-old dog showed remarkable edematous swelling of the left hock with lameness and local cellulitis, and paecilomyces fungus was isolated from ulcerative lesion of the hock joint and mediastinum. At autopsy severe effusive pleuritis was shown and numerous necrotizing and granulomatous lesions with fungal elements were seen in the liver, pancreas, kidney and mediastinal lymph nodes. - KEY WORDS: canine mycosis, granulomatosis, paecilomyces.


A few canine cases have been reported on systemic or profound mycosis [6-8, 10, 12, 17]. The present note is to describe pathologic findings of a canine case showing effusive pleuritis and granulomatous lesion in the multiple organs due to fungal species of paecilomyces.

5-year-old female German shepherd weighing 28 kg was hospitalized on February 8, 1993, presenting remarkable edematous swelling of the hock joint with lameness persisting for one month. Erosive lesions were seen on the nasal mucosa. X-ray revealed the calcification at the left heel joint and periosteal hyperplasia on the proximal end of the left humerus as well as the left antrabrahial diaphysis.

A biopsy was made of the hyperplastic periostium on the left antrabrahial bone, revealing necrotizing myelitis in the presence of conidial organisms. On Sabouraud dextrose agar inoculated with samples from ulcerative lesions of the left hock joint, fungal organisms were almost purely grown at 25°C.

The isolates grew rather rapidly at 25°C on potato dextrose agar. The surface of the fungal colonies was velvety and white in color, and later became powdery and greenish brown. Medium stained red brown. By microscopy 3 to 6 phialides were arranged producing elliptic conidia in long chains. These findings indicated that the isolate was of paecilomyces fungi.

Anti-fungal fluocytosine and fluconasole were repeatedly prescribed for 7 weeks in association with dexamethasone, 0.1 mg/kg/day for 3 days, resulting in temporary disappearance of the heel swelling and lameness. However, recurrence soon came out and the patient was euthanized on July 15, 1993.

At autopsy, a walnut-sized hemorrhagic ulcer appeared on the left hock articulation, while no gross changes were recognized in the popliteal and lymph nodes. Fibrin clots were present in the pericardial cavity and the epicardium was partly calcified. The thoracic cavity contained bloody exudation about 600 ml in volume and rice grain- to soybean-sized abscesses were scattered on the pleura. The enlarged mediastinal lymph nodes were hen's egg-sized, from which the paecilomyces organism was isolated. A number of grayish white nodules of rice grain in size appeared on the surface of the whole left lung as well as the posterior lobe of the right lung. The liver was moderately enlarged and the spleen had several hemorrhagic infarct foci. The renal cortex had some small irregular-shaped white spots.

The main organs and the enlarged mediastinal and mesenteric lymph nodes as well as the diaphragm were fixed in 10% buffered formalin, and paraffin sections were made and stained with hematoxylin and eosin (HE), Masson trichrome, periodic acid-Schiff (PAS), Schwör, Grocott and Gram. For electron microscopy some tissues were reflexed with 1% osmium tetroxide and ultrathin sections were made and stained with uranyl acetate and lead citrate.

Histopathologically, necrotized and purulent foci varied in size were seen in the pleura and diaphragm, and they contained a number of fungal elements. Extensive fibrosis was developed around the necrotic foci. The mediastinal lymph nodes had multiple necrotic foci with fibrosis, containing filamentous and conidial fungal elements (Figs. 1-3). In the slightly enlarged mesenteric lymph nodes a large number of neutrophil leukocytes were accumulated in the lymphatic sinus and vascular lumens.

A number of calcified foci were seen in the myocardial interstitium. The lungs were severely congested with some partial atelectasis. The bronchial epithelium was partly degenerated and desquamated with infiltration of some lymphocytes, plasma cell and macrophages containing PAS- and Schmorl-positive granules. Some clusters of fine PAS- and Grocott-positive fungal filaments were seen within the bronchial arteries.

No lesions were detectable in the digestive tract and submaxillary glands. Some fungal granulomas were produced in the portal triangles of the liver with dilated sinusoids. Similar lesions were produced also in the pancreas. Splenic follicles were severely atrophied with fibrosis of the trabecules and red pulp.

The basement membrane of the Bowman’s capsule of the both kidneys was thickened and there was remarkable hyperplasia of mesangial cells. The renal cortex had numerous necrotizing and granulomatous foci, seemingly produced by the embolism of fungal elements in the glomerular capillaries (Fig. 4). Fungal elements including conidial forms (Fig. 5) were accumulated at the center of
in the cerebellum and medulla.

The present canine case of profound mycotic purulent granulomatosis probably resulted from repeated dissemination of mycotic elements from the superficial lesion of the left limbs, which were persistently infected with an opportunistically pathogenic fungal species of paecilomyces. From the superficial as well as profound necrotizing and granulomatous lesions, a common saprophyte of paecilomyces sp., which is widely distributed in the natural environment [11] was isolated.

Systemic or profound mycosis in canine and other species of animals has been reported to be mostly due to pathogenic fungi of candida, aspergillus or cryptococcus species [2-4, 6, 8, 9, 15, 16]. Opportunistic fatal infection with paecilomyces or penicillium species has been reported in human patients after thoracotomy or in those with endocarditis, acute leukemia or human immunodeficient virus (HIV) [1, 5, 14], suggesting that immunodeficiency might be predisposing condition for those fungal diseases. However, a limited dose of dexamethasone used in the present case seemed not to cause so severe immunosupression as seen in cases of long-term dosing of corticosteroids [1, 9, 12, 16].

REFERENCES

Fig. 4. Necrotizing and granulomatous lesions in the renal cortex. HE stain. Bar = 5 μm.

Fig. 5. Fungal colony within a necrotizing granulomatous lesion in a renal glomerulus, seemingly produced by embolism of fungal elements. Grocott-HE stain. Bar = 10 μm.

122: 81–85.