

PHYCOMYCOSES IN ANIMALS IN THE TROPICS

by

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Emmons, Binford and Utz (1970) have defined the phycomycoses as mycoses caused by fungi traditionally placed in the class phycomycetes, but although *Rhinosporidium seeberi* is considered to be a phycomycete (Ashworth, 1923; Ciferri, 1932), Emmons (1964) stated that rhinosporidiosis should not be regarded as a phycomycosis. In 1968, Clark proposed that diseases caused by species of Entomophthorales be called entomophthoromycoses and those caused by Mucorales, mucormycoses. The name saprolegniosis is applied to infections of fish and amphibians by members of the order Saprolegniales.

A review of the literature on phycomycosis of animals in tropical countries has shown that there are very few reports of the occurrence of such diseases.

Saprolegniosis

Within the order Saprolegniales, species of *Saprolegnia* are the best known and most important pathogenic fungi of fish, fish eggs and amphibians. According to the Animal Health Year Book (1969) Saprolegniosis has been reported from North Africa, Taiwan and South America and is widespread in South Africa where it has been found in dams, rivers and fish hatcheries. Infection, usually following cold weather, has occurred in bream, yellow fish and mud fish. The mortality rate is relatively heavy.

Entomophthoromycoses

The name rhinoentomophthoromycosis (*syn.* phycomycosis entomophthorae, rhinophycomycosis entomophthorae) was suggested by Clark (1968) to distinguish the human disease caused by *Delacroixia coronata* (Constantin) Saccardo and Sydow (1899) from rhinomucormycosis and rhinophycomycosis. Although many human cases have been reported from Africa, the West Indies and South America, animal rhinoentomophthoromycosis has

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only been recognised from horses in Texas (Emmons and Bridges, 1961) and a mule in Brazil (Johnston, Soerensen, Saliba, Lacaz, Bella Neto and Cruz, 1967). That the causal organism *D. coronata*, previously known as *Entomophthora coronata*, could be a pathogen of vertebrates was first reported by Emmons and Bridges (1961) who obtained it in pure culture from lesions in 4 horses. A detailed description of these cases was given by Bridges, Romane and Emmons (1962).

The granulomatous lesions involved the nasal mucous membrane, the skin of the nostrils and the lips and continued to develop by extension to contiguous tissues. The infected tissue consisted mainly of highly vascular granulation tissue infiltrated with eosinophils and neutrophils. Numerous acidophilic areas of necrosis, irregular in shape and size, were observed and within these were found wide (4-8 μ diam.) hyphae with some large globular cells and an occasional septum. In one case the growth was so extensive that the horse was unable to eat and had to be destroyed. The other cases were treated by surgical excision of the lesions. A few small lesions were cauterized. The authors concluded that provided surgical treatment was carried out early enough it was adequate to limit the disease.

D. coronata is found in decaying vegetation and soil and is also a cause of disease in insects. No indication of the mode of infection of the horses was found but the authors suggested that infected insects might be possible vectors. For man, the postulated route of infection is inhalation of spores with subsequent implantation into the nasal mucosa through minor trauma or insect bites.

Bridges, Romane and Emmons (1962) also recorded the isolation of a *Conidiobolus* sp., another member of the Entomophthorales, from similar granulomatous lesions in the nostrils of 3 other horses but further details of these cases have not been given.

Rhinosporidiosis affects man, horses, mules, cattle, goats and dogs and as a result of infection soft, lobulated, highly vascular polypoid growths develop on the nasal mucosa. Differentiation of rhinosporidiosis and rhinoentomophthoromycosis of horses on clinical signs only might be difficult in some cases and it may be that, in areas in which rhinosporidiosis is endemic, cases of rhinoentomophthoromycosis are being mis-diagnosed. It is interesting that Nino and Freire (1957) reported 9 cases of nasal polyps in horses but found *R. seeberi* in only 8.

Basidiobolus ranarum Eidam (1886) is one of the species of *Basidiobolus* implicated in human subcutaneous phycomycosis which is widely distributed in tropical countries, especially Africa. This fungus was first reported as a pathogen of mammals in 1925 by van Overeem who isolated it from a mare which had leg lesions resembling those of «*hyphomycosis destruens equi*». *Basidiobolus* spp. are found in decaying vegetation and soil and also in the digestive tracts of lizards, frogs and toads. Infection of man and animals probably arises by penetration of wounds by the fungus.

Mucormycoses

Within the order Mucorales, species of *Absidia*, *Mucor*, *Rhizopus* and *Mortierella* have been reported as pathogens of many kinds of animals (Ainsworth and Austwick, 1959) but, although mucoraceous fungi and human mucormycoses are world-wide in distribution, there are not many records of these diseases in animals in the tropics.

Horses

In South America, a cutaneous equine mycosis has been described by Ciprian, Mocoroa, Andreatta and Epstein (1965). Infected horses showed granulomatous lesions on the posterior aspect of the pasterns. A fungus identified as *Absidia corymbifera* was isolated from one case and experimental transmission was attempted but without success. Surgical and chemical treatments were not effective.

A mucormycosis which occurred in a series of horses from the Texas Gulf Coast was described by Bridges and Emmons (1961), who named the causal fungus *Hyphomyces destruens* but could not further identify it because sporing structures were not produced. They thought, however, that their isolates had some of the characteristics of a *Mortierella*. Localized granulomatous lesions were found, most frequently on the fetlocks but also on the neck, mammary glands, umbilical area and trachea. These lesions, which developed rapidly and could cause extreme debility within a few weeks, appeared as exuberant granulation tissue which might show fistulous cracks or as swollen ulcerated regions with fistulous openings. Although localized, many lesions were unsuitable for surgical treatment and did not respond to medical therapy. Within the granulation tissue there were neutrophils, eosinophils and occasionally multinucleated giant cells and many firm, irregular masses of necrotic tissue in which branching, occasionally septate hyphae measuring from 3-10 μ in diameter were found.

As most of the horses had a previous history of wounds at the site of infection there can be little doubt that *H. destruens* entered the body in this way. Many cases occurred during an unusually wet period and all the horses had access to ponds of water. Constant wetting would soften the skin and render it more liable to minor trauma and it is also possible that the fungus, which has not yet been isolated from a natural source, grows saprophytically in moist sites.

Bridges and Emmons noted a clinical resemblance between their cases and a number of other equine diseases associated with fungi. «*Hyphomycosis destruens equi*» was described from a series of horses in Indonesia by de Haan and Hoogkamer (1903). The causal fungus was not identified but according to Bridges and Emmons resembled *H. destruens*. Similar cases have been recorded from France (Drouin, 1886) and Germany (Frank-Speyer, 1890).

«Florida horse leeches», a disease endemic to marshy areas of the southern states of U. S. A. was, according to Moore (1913) and Bridges and Emmons (1961), believed by some workers to be mycotic in origin.

Hutyra, Marek and Manninger (1938) noted similarities between Florida horse leeches and bursattee a disease endemic to India, Burma and the Sunda Islands. Smith (1884) who studied fresh material from bursattee lesions mounted in Potassium hydroxide-glycerine observed fungal hyphae and he also reproduced the disease experimentally using material from infected horses. Other suggested causes of bursattee are an actinomycete (Datta, 1931) and cutaneous infection with larvae of the nematode *Habronema* (Datta, 1933).

The clinical signs of the disease caused by *H. destruens* also showed similarities to cutaneous habronemiasis. Lesions in 18 of the 41 horses examined by Bridges and Emmons were due to *Habronema* but in the 23 horses with mycotic lesions there was no parasitic infection. Another mycotic granulomatous dermatitis of horses caused by a fungus of the family Mortierellaceae has been described from Japan by Amemiya and Nishiyama (1968) and the clinical signs were reproduced experimentally by subcutaneous injection of the fungus into rabbits and horses (Amemiya, 1969). The equine lesions were characterized by granulation tissue, eosinophils and areas of necrosis. Of the 10 horses with mycotic lesions 2 were also infected with *Habronema* and a series of clinically similar cases in another locality were entirely due to habronemiasis.

Thus, the lesions of cutaneous habronemiasis and a number of mycotic diseases in horses are alike in the type of lesions produced, in the parts of the body affected and in causing intense itching. Histologically they are characterized by granulomatous tissue containing necrotic foci and the presence of numerous eosinophils, cells more frequently associated with parasitic infection than with mycoses. According to Bridges and Emmons, although the lesions caused by *H. destruens* tended to be slightly larger than those caused by *Habronema* and did not regress in cooler weather, differential diagnosis could not be made on clinical examination only. These authors found that it was not difficult to demonstrate the presence of larvae by microscopy but fungal foci might be missed if only one biopsy were examined.

It is not very surprising, or with regard to treatment very significant that cutaneous lesions caused by various species of mucoraceous fungi should show similar clinical signs. However, that mycotic lesions resemble those of cutaneous habronemiasis is of the utmost importance with regard to control and treatment. Moore (1913) discussing the possibility that Florida horse leeches and bursattee were the same disease stated « the question of their identity ought to be held in abeyance until a more thorough investigation can be made », a statement which is still valid 50 years later.

Cattle

The few reports of bovine mucormycosis from tropical countries include a case of generalized mucormycosis in a calf described from South America by da Silva and Mackado (1963). Mucoraceous hyphae were observed in miliary lesions in the lungs, in ulcers of the tongue, tonsils and reticulum and in necrotic areas in the liver. Two cases of mucormycosis, believed to be secondary infections, have been recorded in South

Africa by du Plessis, Cameron and Langen (1967). Mucoraceous hyphae were found in lesions in the brain of one calf and in the rumen of the other. In India, mucormycosis was diagnosed in a 1 1/2 year old buffalo calf which died after a brief history of severe diarrhoea (Tewari, 1963).

Abortion associated with mycotic placentitis, probably the most frequently occurring mucormycosis of cattle in temperate zones, has been noted infrequently from tropical countries. One case caused by an unidentified species of *Mucor* has been recorded from India (*Baruah and Ahmed*, 1963) and 3 cases caused by *M. pusillus* from Southern Rhodesia by Shone, Phillips, Roberts and Christie (1958). A very high incidence of mycotic abortion has been reported from Hong Kong by Turner (1964, 1965, 1965b) who investigated 34 cases of abortion and diagnosed fungal infection in 29 per cent. Of these, 70 per cent were caused by Phycomycetes and species of *Mucor*, *Rhizopus* and *Syncephalastrum* were obtained in culture. A possible explanation for the high incidence of mycotic abortion in Hong Kong is that the cattle are housed throughout the year, although they are allowed out daily into concrete enclosures.

From these few reports it would appear that in tropical countries Phycomycetes are the most frequently encountered aetiological agents of mycotic abortion. This is in contrast to temperate zones in which species of *Aspergillus* predominate (Hugh-Jones and Austwick, 1967). These authors, however, noted that, in England in years in which the summers were warmer and drier than average, higher than average incidence of mucoraceous infections of the placenta were encountered. As a possible explanation they suggested that hay made in warm, dry weather would, because of its higher content of soluble carbohydrate, provide a better substrate for mucoraceous fungi.

Other domestic animals

Mucormycoses of other domestic species occur infrequently. Two cases of ruminal infection in goats have been reported from Ceylon by Mostafa, Cerna and Cerny (1966). Hyphae were present in necrotic foci in the rumen and in a thrombosed blood vessel in the lungs of one goat and in necrotic lesions in the rumen, lungs, liver and abomasum of the other. The ruminal lesions were considered to be primary with haematogenous spread to the other organs. One case of spontaneous mucormycosis of a rabbit has been reported from South America (Lopes, 1970) and a single case of avian mucormycosis, a much rarer disease of birds than aspergillosis, has been noted in Brazil by Reis and Norbrega (1955).

Wild life

There are no reports from tropical countries of mucormycoses in wild life. However, that animals native to these countries are susceptible to infection is shown by the occurrence of this type of disease in captive animals. Combined aspergillosis and mucormycosis has been described in young okapi calves (Hewer, 1964; Campbell, 1967). In each case the

aspergillotic lesions were confined to the lungs. Mucoraceous hyphae were recognised histologically in heart, liver, kidneys, abomasum and lymph nodes of one calf and in kidney lesions in the other. *A. corymbifera* was isolated from the latter case. Ruminal mucormycosis of a gazelle has been reported by Marcato and Dimon (1962) and generalized mucormycosis in a roe kid by Kretschmar (1954).

Generalized mucormycosis was described in a mandril by Lucke and Linton (1965) and gastric mucormycosis in a rhesus monkey following an intensive course of orally administered antibiotics by Hessler, Woodard, Beattie and Moreland (1967). Two cases of intestinal mucormycosis in macaca monkeys were diagnosed by Gisler and Pitcock (1962) who found extensive ulcers in the caecum and ascending colon and foci of necrosis in the liver of one monkey and ulcerative colitis of the large intestine in the other.

In conclusion, although the causal fungi are the same for man and animals, reports of human phycomycoses in tropical countries far outnumber those of animal phycomycoses. As pointed out by Austwick (1968) however, the distribution of mycoses still largely follows that of interested workers. There are few veterinary mycologists and these tend to be concentrated in the temperate zones. It is obvious therefore that until many more persons become interested in this field, a true incidence of mycotic disease in animals cannot be established.

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DISCUSSION

V. S. Pandey : About the treatment with formalin and potassium iodide, I believe, it is effective in rhino-sporidiosis in horses. Do you think that the same treatment will be good in other animals and how can you explain the effect of formalin in responding to this therapy ?

C. O. Dawson : I think the treatment should be equally effective in other animals. As I am not a pharmacologist I cannot explain the mode of action of formalin.

R. Vanbreuseghem : Although with a slightly mulish resistance quite typical for a vet, you have demonstrated quite clearly that I was right asking you to develop this subject; it is extremely broad, it proves only our ignorance, but may I ask you why you did include rhinosporidiosis in your cases of phycomycosis ?

C. O. Dawson : *Rhinosporidium* has been classified as a phycomycete closely allied to the chytridiales and also, as rhinosporidiosis and endomphthoromycosis of horses show similar clinical manifestations, differential diagnosis is important.

R. Vanbreuseghem : You only believe this ?

C. O. Dawson : Until *Rhinosporidium* has been successfully cultured and studied one cannot be certain of its taxonomic position.

E. Chick : In experimental animals to produce an infection with the phycomyces we have to use various drugs such as the corticosteroids, cytotoxins, and others. In your series of animals was there any evidence of underlying disease or were these a primary infection as far as you can determine ?

C. O. Dawson : We have found abomasal ulceration in apparently healthy calves fed on milk substitute diet. As yet there is no proof whether the infection is primary or secondary. Again, much more work is required in the field.

N. Nolard-Tintigner : Vous avez montré un poisson atteint de Saprolegniose. Je voudrais savoir si vous avez observé ce poisson post-mortem ou ante-mortem et d'autre part si vous êtes tout à fait certaine qu'il s'agit là d'une vraie Saprolegniose, c'est-à-dire une Saprolegniose avec envahissement des tissus internes et pas d'une surimposition d'un tissu en nécrose par un Saprolegnia.

C. O. Dawson : The fish was alive when captured. We do consider it to be a case of saprolegniosis, although the infection was probably secondary to lesions of ulcerative dermal necrosis (salmon disease). We have rarely seen cases in which deep penetration occurred. In the case in the photograph, although the fungus grew into mouth and nostrils it remained superficial.

R. Vanbreuseghem : I think that Mrs Nolard would like to tell you that her experience with saprolegniosis demonstrates clearly that the internal tissues are invaded to the deepest medulla, the nerves and the big vessels.

C. O. Dawson : This is very interesting — ours are most superficial.

N. Nolard-Tintigner : Au sujet de la répartition dans les tropiques : avez-vous connaissance de cas de Saprolegniose en Afrique ?

C. O. Dawson : No, I was quoting from records in the Animal Health Year Book. It is possible that different species or strains may be involved in infections in tropical and temperate countries because, for one thing, the optimal growth temperatures will differ. We frequently isolate *Saprolegnia* by putting inoculated bottles in the refrigerator at about 4 °C and obtain good growth.