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Bovine Malignant Catarrhal Fever

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INTRODUCTION

Malignant catarrhal fever (MCF) or malignant head catarrh has been recognized for over 125 years as a fatal disease of cattle and some wild ruminants. Found worldwide, it is prevalent in Australia, the British Isles, Canada, the United States and Africa. The causative agent is believed to be a herpesvirus, but it is thought that tissue damage is the result of hypersensitivity to viral antigens and not direct effects of the virus.

In Africa, wildebeest are involved in harboring and transmitting the disease, and it is often referred to as “African or wildebeest-driven MCF.” Outside of Africa it is speculated that sheep are involved in transmission, and it is referred to as “sheep-associated MCF” or “American MCF.” U.S. cattle are susceptible to both American and African MCF. African MCF may be contracted from zoo animals if cattle graze near a zoological park. Clinically one cannot differentiate between American and African types of MCF.

Occurrence of MCF in cattle is usually sporadic, but explosive outbreaks have been documented in feedlot and dairy cattle in the United States. Although morbidity is considered low in cattle, mortality is greater than 95%. Typical signs and lesions are high fever, swollen lymph nodes, oral erosions, conjunctivitis, corneal opacity and diarrhea. There is no good treatment for MCF, but if cattle are in contact with sheep they should be separated immediately to prevent more cases from occurring.

ETIOLOGY

The causative agent was first isolated from outbreaks of African MCF.1,2 A herpesvirus recovered from clinically normal wildebeest caused fatal MCF when cattle were exposed to it. Morphologically it was similar to Herpes simplex B, pseudorabies and varicella. In thyroid monolayer cultures, researchers saw syncytial arrangement, intranuclear inclusion bodies and extracellular herpes-like particles. This virus is now identified as bovid herpesvirus 3.

The MCF virus is quite fragile and can be destroyed by freezing or freeze-drying, and the virus does not survive in cell-free cultures.3

Unlike the African MCF, the American variety has never been isolated from suspected reservoir animals such as sheep. Two cases of MCF were reported outside of Africa, one in Minnesota and one in the Netherlands, in which a herpes-like virus was isolated.4,5 In both cases, the tissues were tested serologically using antisera from African MCF, and the viruses were found to be immunologically related to the African MCF virus. In the case of the Minnesota outbreak, a killed vaccine from this virus was administered to eight susceptible cattle. These animals were protected from challenge by African MCF, while all control animals died from MCF.

EPIDEMIOLOGY

In the United States domestic animals that show clinically fatal MCF are beef and dairy cattle and bison.6,7,8 Wildlife species of buffalo, American and European bison, white-tailed and mule deer and sika deer in Canada also experience the fatal disease.5,9,10,11,12,13 At least three outbreaks have occurred in zoological parks in the United States14,15,16 and in one case a dairy farm near one of the parks.
experienced sporadic cases of MCF for two winters at about the same time of the clinical cases at the zoo.

Although MCF virus is infectious, each of the species above is considered a dead end host, and direct contact between susceptible cattle and other clinically infected animals (domestic or wildlife) will not spread the disease to susceptible cattle. But a few researchers believe there may be carriers within a susceptible group of animals. A study in 1972 reported on one cow experimentally exposed to MCF virus and giving birth to four infected calves up to 80 months after initial exposure. In another report from Colorado, thirty-one cases of MCF occurred in a cattle feedlot and the owner elected to sell the surviving animals. Afterwards forty-two more cases of MCF were reported from the original (infected) herd, yet the disease did not spread to the thousands of other exposed cattle in three salebarns and several feedlots.

MCF affects all ages, sexes and breeds of susceptible species. Morbidity is usually considered to be quite low, affecting only one or two individuals in a herd. But cases have occurred in the U.S. where epizootic outbreaks have killed 10–37% of a cattle herd. Mortality on the other hand is considered greater than 95%, and in the above cattle herds it was essentially 100% of all clinically affected animals.

Natural reservoirs that sustain the MCF organism are inapparently infected animals. In Africa, it has been proven that the wildebeest or gnu and hartebeest are the inapparent carriers. Outside Africa, sheep are the suspected reservoir although the MCF organism has never been isolated from sheep. Most cases of MCF in cattle in the U.S. show a close association of communal raising of cattle and sheep. A study in 1954 reported MCF in cattle that had been exposed to sheep taken from near previous outbreaks of MCF. However he could not continuously infect cattle year after year using the same sheep.

MCF also commony occurs in cattle during or shortly after the end of lambing season. MCF does not always occur around lambing ewes; at least one case of MCF occurred in cattle that were housed with rams. A few cases of MCF have occurred where there was never any contact between sheep and cattle, and no sheep had ever used the same facilities or grounds. This may add more support to the theory in which carrier cattle may be present.

The mode of transmission of the disease is unknown. In Africa it is common for cattle to contract the disease after grazing grasslands where wildebeest had recently calved. This would also fit the pattern in the United States where MCF in cattle is associated with ewes that are lambing. It has been speculated that insect vectors may be involved in transmission, based on the fact of slow spread and the incidence being higher during the warm spring months. However, there are other cases in which large numbers of animals became infected within a short period of time and during the winter months. Spread from lambing ewes supports the view that congenital infection may occur, especially in light of the case where a cow gave birth to infected calves. The virus has been detected in nasal and ocular secretions of wildebeest and it can be speculated that this might also be occurring in sheep.

The persistance of MCF on a particular farm from year to year where no other carrier animals (such as sheep) are present is unexplained. Sheep which run with cattle or have some contact are possible sources of infection. The virus is too fragile and is not maintained in cell-free fomites which suggests that transmission of the virus from reservoir animals to cattle is either by direct contact or insect vectors. The possibility also exists that there are inapparently infected carrier animals within a cattle herd which could account for carry-over infections.

**CLINICAL FINDINGS**

The incubation period in natural infection varies from 3 to 8 weeks, but in experimentally infected animals it ranged from 7 days to 10 weeks. Researchers found the American strain had a longer incubation period (average 32 days) than the African strain experimentally induced in cattle (average 13 days). But the course of the disease was much shorter for American MCF, averaging 4 days, while the African form averaged 12 days.

Although there are some differences between American and African MCF experimentally, it is almost impossible to distinguish the two in natural infections, and it should be emphasized that U.S. cattle are susceptible to both varieties.

Bovine MCF is described as occurring in
four forms: peracute, the eye and head form, the alimentary form and the mild form. Because one form grades into another, cases are classified on the predominant clinical signs seen. All forms may be produced from one viral strain in one outbreak.

In all forms the development of fever (>103°F) and enlarged lymph nodes, particularly the prescapular nodes, precedes all other signs. In the eye and head form (which is the most common) a sudden onset of depression, anorexia, profuse mucopurulent nasal and possibly ocular discharges, blepharospasm and photophobia are noticed. The buccal mucosa is reddened, and areas of necrosis are evident on the hard palate and gingiva. The muzzle may appear crusty, and papillae on the buccal areas may appear blunted, with the tips hemorrhagic.

Other signs which may appear are corneal opacity beginning at the periphery, hypopyon, conjunctivitis and reddening of the skin of the udder, coronary bands and interdigital spaces. Nervous signs, such as belligerence or a demented appearance, incoordination and stumbling and particularly weakness in one leg, may be apparent. In later stages, head pressing and convulsions may be evident. Fecal consistency varies from constipation to profuse diarrhea with dysentery.

In cases of longer duration, skin changes may occur, and an eczematous weeping of the loin and withers may be evident and result in a crusty formation and matting of the hair. Rarely horns and hooves are shed. The fever may persist or fluctuate. In some outbreaks an animal apparently on recovery will die 7 to 10 days later of acute encephalitis.

In the peracute form, the disease runs a short course of 1 to 3 days. The animal may display a high fever, dyspnea and acute gastroenteritis or may be found dead without having shown any signs of illness.

The alimentary form is similar to the eye and head form, but only minor conjunctivitis is noticed, while diarrhea is marked. The disease rapidly progresses to the final fulminating stages. A study in 1979 found the alimentary form to be present more often in American MCF, possibly causing some confusion in diagnosis of the disease by field veterinarians.

The mild form occurs most commonly in experimental animals. There is a transient fever, and a few erosions may appear on the oral and nasal mucosae, with recovery following.

**CLINICAL PATHOLOGY**

Both leukopenia and moderate leukocytosis have been described, but neither is important clinically. All other clinical pathological findings appear to be within the normal ranges.

**NECROPSY FINDINGS**

Besides these lesions already mentioned under clinical findings, erosions and linear ulcerations may be found in the nasal, oral and esophageal mucosae. The lungs are not grossly affected unless a secondary pneumonia is present. The mucosae of the forestomachs and intestines are not eroded, but some cases of edema of the intestines with prominent Peyer's patches have been recorded. The liver may be swollen, the adrenal glands focally hemorrhagic and the renal surface covered with whitish-tan, raised foci. A study in 1978 found many cases of reddening and edema of the bladder mucosa. All lymph nodes, tonsils and Peyer's patches appeared large, friable and moist, while occasionally the spleen had prominent lymphoid follicles. Joints commonly contain increased quantities of cloudy synovial fluid, and synovial membranes are swollen. The brain sometimes exhibits petechial hemorrhages and congestion or cloudiness of the meninges. Any animal dead from MCF may show all or very few of these lesions at necropsy.

**HISTOLOGY AND PATHOGENESIS**

Histologically, vascular fibrinoid necrosis and vasculitis without thrombosis can be found in virtually every organ that is affected. These are considered the pathognomonic lesions of MCF. Lymphoid hyperplasia is also characteristic for the disease. Because of these consistent histopathologic findings, it has been theorized that the virus for MCF may not be immediately responsible for the histologic lesions and suggests that the vascular damage might be a result of hypersensitivity to the virus or viral antigens in the vascular walls. Histologically, MCF bears a strong resemblance to a graft vs host reaction.

**DIAGNOSIS**

Definitive diagnosis of MCF is difficult because virus isolation from infected species is very difficult. Often large quantities of fresh
blood and tissue are sent immediately to the lab and inoculated into test animals or onto bovine calf thyroid cell cultures. In many cases the infective agent is lost. Laboratory tests such as fluorescent antibody, complement fixation and serum neutralization have been perfected but are not in use in any diagnostic laboratory except those doing research on the disease.

To make a clinical diagnosis a complete history is needed, which includes clinical signs and lesions, plus a necropsy of the dead animal. Confirmation of a presumptive diagnosis can be made by histopathologic examination. Tissues that should be sent for histopathologic evaluation include brain, liver, kidney, heart and lymph nodes. Histological findings of perivasculitis and vasculitis without thrombosis, and mononuclear cell aggregations within the affected tissues can be accepted as confirmatory evidence.

Symptoms of fever, diarrhea and lacrimation along with oral lesions and linear erosions may be suggestive of BVD-MD and Rinderpest. But BVD usually exhibits discrete, rounded and sharply defined oral lesions versus the diffuse lesions and mucous membrane hyperemia of MCF. Rinderpest is exotic to this country and if introduced would spread rapidly with high morbidity and mortality whereas MCF is very sporadic and morbidity is very low.

The clinical reactions of MCF probably most closely resemble Bluetongue in cattle, especially the oral and muzzle lesions. Vesicular diseases (e.g. Foot-and-Mouth disease, Vesicular stomatitis) might also be a differential diagnosis of MCF, but can easily be excluded on the grounds that these diseases elicit vesicles on the oral mucosa, teats and coronary bands.

All of the above mentioned diseases do not exhibit the severe ocular and conjunctival lesions associated with MCF. Occasionally BVD exhibits corneal edema with slight opacity. The corneal opacity of MCF begins peripherally and moves centrally and should not be confused with infectious bovine keratoconjunctivitis ("pinkeye") which begins centrally and moves outward. Infectious Bovine Rhinotracheitis (IBR) may show a similar corneal lesion but infection is limited to the upper respiratory tract and is usually not fatal.

Chronic unresponsive diphtheria and bovine respiratory disease complex ("shipping fever") may resemble MCF, but there are no ocular or oral lesions. One may also consider various encephalitides, especially Sporadic Bovine Encephalomyelitis, except oral lesions are not found. Mycotic dermatitis and photosensitive dermatitis may appear similar to skin lesions in a case of MCF of long duration.

Other diseases which must be considered especially with the alimentary form are salmonellosis, coccidiosis and acute fatal indigestion.

**TREATMENT AND CONTROL**

MCF is nearly always fatal and usually does not respond to treatment. Considering the hypersensitive immunopathologic reaction, large doses of corticosteroids (dexamethasone at 55 mg per 50 kg) may help in symptomatic treatment of the disease. Antibiotics and/or sulfonamides should be administered simultaneously. In one case a yearling bison bull affected with MCF was treated daily with antibiotics and *Lactobacillus acidophilus* orally and survived for one month but died when treatment was withdrawn.

Isolation of affected cattle is usually recommended, but its value has been questioned due to the slow spread and unknown mode of transmission. If sheep are present, it is highly recommended that they be separated from cattle at once. This will usually result in the disappearance of any further cases. Recovered animals are immune to further infection for 4 to 8 months.

Attempts to produce a vaccine, either killed or modified live, have met only with limited success. Since MCF does not usually affect large numbers of animals and is not a continuous threat to cattle, a vaccination program would be of benefit only in the face of a large outbreak. The best preventative is to restrict contact between cattle and sheep.

**CONCLUSION**

There is enough evidence to suggest that the American variety of MCF is probably a different strain of the same virus that causes African MCF (bovid herpesvirus 3) and not an enterovirus or a bovine syncytial-forming virus as earlier thought by researchers.

MCF is typically thought of as sporadic in nature affecting only one or two individuals at a time. One report points out that MCF in the
U.S. is also emerging as a disease of explosive outbreaks that affect large numbers of animals in a short period of time; this can lead to serious economic loss.\textsuperscript{13}

Also of economic consideration is a misdiagnosis of MCF. An animal that is misdiagnosed as having bluetongue or BVD may lead the owner or veterinarian to institute a vaccination program that is not needed. This could well be the situation in the field when we consider that in one study 69\% of experimental cases of American MCF presented as the alimentary form with insidious diarrhea.\textsuperscript{22} Cases may continue to appear because sheep were left to co-mingle with the herd and not separated.

The point is that a definitive diagnosis needs to be made in order that appropriate steps can be taken to stop further losses and prevent implementation of time consuming and costly programs that are of no value.

\textbf{REFERENCES}
