

# Malignant Catarrhal Fever

by  
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This paper was written not because of the common occurrence of malignant catarrhal fever but because of its close resemblance to the more common diseases IBR and BVD. It is imperative, therefore, that the practitioner keep malignant catarrhal fever in the back of his mind as a differential diagnosis when considering IBR or BVD. If this is brought to the attention of just a few practitioners, the purpose of this paper will be accomplished.

## Summary

Malignant catarrhal fever is an infectious, usually fatal, disease of cattle caused by a virus and characterized by a catarrhal, mucopurulent inflammation of the upper respiratory tract, keratoconjunctivitis, encephalitis, and enlargement of the superficial lymph nodes. A tentative diagnosis can be made on clinical signs concurrent with a persistent high temperature but must be confirmed by histopathology. There is no treatment and mortality is 100% in most cases.

## Case History

This summer I was working at the Sumner Veterinary Clinic. On Saturday, June 29, we were called out to a client's farm because he had a cow off feed. The cow was a four year old Holstein and had been fresh for about five months. Physical examination revealed slight lung congestion and slight dyspnea along with a rectal temperature of 105.5°F. Rectal exam revealed she was about 60 days pregnant but nothing else unusual.

On Sunday, June 30, the dyspnea was more severe and a catarrhal stringy exudate was coming from her nostrils. Rectal temperature was 105.5°F. and the eyes were becoming cloudy at the corneoscleral junction but sight was still evident in both eyes. A blood sample was drawn and the differential blood count revealed a leucopenia. A tentative diagnosis of IBR was made.

On Monday, July 1, there was severe dyspnea and a temperature of 105°F. The cloudiness in the eyes, which appeared to be pus, in the anterior chamber, was increasing to the point where sight was lost in the right eye. The scleral vessels were injected and the catarrhal exudate from the nostrils revealed necrotic tissue present.

On Tuesday, July 2, the dyspnea was not as severe and the temperature was 104.5°F. The cloudiness in the eyes was progressing and there was a mucopurulent nasal discharge. By this time it appeared that this cow had something more severe than IBR. Upon further questioning of the client, we found that he had housed his sheep in the barn with the cows last winter.

On Wednesday, July 3, both eyes appeared blind, the nasal mucosa was eroding away and there was a mucopurulent nasal discharge. The cow died later that day and a postmortem was done that evening. Gross lesions were suggestive of malignant catarrhal fever and formalinized samples of brain, liver, heart, kidney, and the eyes were sent to the Iowa State Diagnostic Lab. The diagnosis of malignant catarrhal fever was confirmed by the diagnostic lab by histopathology on July 19.

Both sexes and all breeds of cattle are susceptible. Incidence is highest in cattle

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six months to four years of age with the disease rarely occurring in cattle less than six months of age. Sheep do not develop the disease but appear to be a carrier of the virus. Deer can also contract the disease and show clinical signs similar to cattle as well as serve as a wild reservoir of the virus. Malignant catarrh can occur any time of year but is most common in fall and spring and occurs in most countries of the world. The disease has a very low morbidity and a very high mortality.

### *Etiology and Pathogenesis*

The cause of malignant catarrh is a virus that is classified in the herpesvirus group. It is very difficult to isolate from the blood because it is closely associated with mononuclear leukocytes as well as lymphoid tissue cells of nodes and spleen but is not found in secretions or excretions. The route of entry of the virus into the bovine is not known but experimentally when it is administered parenterally it first attacks lymphoid tissue and replicates here. Many small lymphocytes are destroyed and germinal centers depleted resulting in a leukopenia. This leukopenia occurs at the same time that there is a rise in body temperature. The virus also attacks the vascular adventitia causing necrosis of the vessels which account for the gross lesions.

### *Clinical Signs and Post-Mortem Lesions*

The incubation period in natural infection varies from three weeks to five months or even longer in some cases. The course of the disease is usually between four and 14 days. There are four forms of malignant head catarrh—peracute, alimentary form, head-and-eye form, and the mild form, with the head-and-eye form being the most common. In this form, onset is usually sudden with anorexia, agalactia, high fever (106–107°F.), rapid pulse (100–120/min.), a profuse mucopurulent nasal discharge, blepharospasm and congestion of scleral vessels.

Superficial necrosis is evident in the anterior nasal mucosa and on the buccal mucosa. The skin of the muzzle is usually

involved beginning with areas of necrosis around the nostrils which soon coalesce causing tenacious scabs over the entire muzzle. Similar lesions may be seen at the coronary band and between the toes. In acute cases, skin on the teats may slough and some animals develop a dermatitis. Superficial lymph nodes are usually enlarged.

The consistency of the feces varies from constipation to severe diarrhea and occasionally melena. Corneal opacity is always present to some degree. It starts as a narrow gray ring at the corneoscleral junction and spreads centripetally. Persistent fever (greater than 103°F.) is characteristic of malignant catarrhal fever. Some animals develop an encephalitis and may show abrupt temperament changes.

Most of the gross lesions are caused by a necrotizing vasculitis which is due to the virus attacking the adventitia of the blood vessels. Gross lesions involve:

**EYE:** conjunctivitis and keratitis.

**MUZZLE:** encrusted and eroded

**SKIN:** exanthema—exuded lymph gives appearance of sweating.

**RESPIRATORY SYSTEM:**

1. Nasal cavity—serous to mucopurulent inflammation with ulcers and pseudomembranes.

2. Trachea—hyperemia to petechia and ulcers.

**DIGESTIVE SYSTEM:**

1. Oral cavity and esophagus—erosions and ulcers.

2. Abomasum—hemorrhage and ulcers.

3. Intestines—erosions, petechiae and excess mucus.

4. Liver—mottled with less than 1mm white foci.

**URINARY SYSTEM:**

1. Kidney—2–4mm white foci.

**LYMPH NODES:** retropharyngeal nodes are consistently enlarged.

Microscopic lesions are a generalized vasculitis with cellular accumulations in the adventitia.

### *Diagnosis*

A presumptive diagnosis of malignant catarrhal fever can be made when the

nasal, oral, and ocular lesions are observed along with a persistent high temperature, enlargement of peripheral lymph nodes, and terminal encephalitis. The diagnosis can be confirmed histologically by demonstrating perivascular, mononuclear cell aggregations in brain, liver, heart, and kidney tissues.

The differential diagnosis should include BVD-MD, Rinderpest, Infectious Stomatitides, IBR, and Pneumonic Pasteurellosis. BVD-MD, Rinderpest, and the Infectious Stomatitides are not accompanied by the typical ocular lesions, lymph node enlargement, or encephalitis and they each have a distinctive histopathology. IBR is not usually fatal, recovery is rapid, the lesions are restricted to the upper respiratory tract and the disease is readily transmitted. Pneumonic Pasteurellosis is not accompanied by oral, nasal, or ocular lesions and responds well to treatment.

### ***Treatment and Control***

Because malignant catarrhal fever is caused by a virus, treatment is of little or no value.

Because association with sheep is usually in the history, separation of cattle and sheep herds is recommended and has resulted in the disappearance of the disease in some cases. The introduction of sheep from areas where the disease has occurred should be avoided. Very few animals survive, but those that recover are immune to further infection for four to eight months.

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# **History as a Diagnostic Aid in Canine Epilepsy**

by  
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## ***Summary***

Diagnosis of the etiology of an epileptic dog involves prudent history acquisition

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coupled with clinical signs, neurological examination, blood chemistry, cerebrospinal fluid evaluation, and urinalysis.

The histories of five clinical cases are presented to show the variations in the amnesia and the significance these variations might have.