

Malignant catarrhal fever viruses

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Malignant catarrhal fever viruses

- Disease: **Malignant catarrhal fever (MCF)**
- Etiology: Group of viruses belonging to **family *Herpesviridae*, subfamily *Gammaherpesvirinae*, genus *Macavirus***
- The MCF subgroup of Macaviruses, called MCFV, contains at least 10 members, five of which are currently known to cause disease
- These viruses cause **inapparent infection in their reservoir hosts**; however, can cause serious illness in other species
- Almost invariably **fatal, generalized lympho-proliferative disease** of even-toed ungulates, including cattle, buffalo, deer, antelope, giraffe and swine

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- **Alcelaphine herpesvirus 1** and **ovine herpesvirus 2 (OvHV 2)** are the major (and best characterized) causative agents of MCF
- **Alcelaphine herpesvirus 1** : Wildebeest are natural host, infection restricted to Africa
- **Ovine herpesvirus 2** : Sheep are natural hosts and infection occurs worldwide in sheep and goats
- Viruses: stable between pH 5.5 and 8.5, inactivated by common disinfectants and sunlight
- **OvHV-2 has never been cultured in vitro**
- MCF viruses, like other herpesviruses, establish lifelong, latent infections in natural hosts, do not infect humans

Transmission

- Inhalation- primary means of transmission for all MCF viruses, although ingestion might also be possible

Alcelaphine herpesvirus 1

- All wildebeest calves are infected within the first few months of life by in utero, direct contact or aerosol routes
- Contamination of pastures and fomites may also contribute to transmission
- Virus is shed by wildebeest calves in nasal and ocular secretions

Transmission of Ovine herpesvirus 2

- Mainly by the respiratory route, probably in aerosols
- Shed intermittently in nasal and ocular secretions, particularly by 6- to 9-month old lambs
- virus also present in feces and semen
- Infection is acquired through contact with young lambs
- Cattle and other incidental hosts are thought to be dead end hosts, because they do not appear to transmit virus

Pathogenesis

- The pathogenesis of MCF is poorly understood
- It is presumed that virus enters the body through the upper respiratory tract
- A cell-associated viraemia occurs
- MCF is characterized by extensive pathological changes, however, virus is virtually absent from the sites of lesions
- It is thought that tissue changes in MCF have an immunopathological basis
- Cell-mediated reactions have been implicated in lesion development
- MCF is characterised by the accumulation of lymphocytes (predominantly CD8(+) T lymphocytes) in a variety of organs, often associated with tissue necrosis

Clinical symptoms

- **Peracute:** No clinical signs are detected, or **depression followed by diarrhoea and dysentery** may develop 12–24 hours prior to death
- In general: **high fever**, increased serous **ocular and nasal exudate** progressing to profuse mucopurulent discharge, **inappetance**, and **decreased milk yields**
- Progressive **bilateral corneal opacity**, starting at the periphery, is characteristic
- **Skin ulceration and necrosis** may be extensive or restricted to the udder and teats
- **Generalized lymphadenopathy, extensive mucosal erosions, and central nervous system signs** that are characteristic of the “head and eye” form of the disease
- **Erosions of the gastrointestinal mucosa** lead to hemorrhage and melena, as well as extensive ulceration throughout the oral cavity, including the tongue



Lesions

- Erosions and haemorrhages in gastrointestinal tract
- Enlarged lymph nodes
- Catarrhal exudate, erosions and diphtheritic membranes often observed in the respiratory tract
- Urinary bladder with characteristic ecchymotic haemorrhages of the epithelial lining, especially in bison
- Interstitial accumulation of lymphoid cells in nonlymphoid organs, particularly in renal cortex and periportal areas of liver, is typical
- Kidney with multiple raised white foci
- Brain- nonsuppurative meningoencephalitis

Diagnosis

- Clinical symptoms and lesions, ulceration of surface epithelia is a prominent feature of MCF
- AIHV-1 may be recovered from clinically affected animals using peripheral blood leukocytes or lymphoid cell suspensions
- Ovine herpesvirus 2 has not been propagated in cell culture
- Tissues for virus isolation: 10–20 ml anticoagulated blood in EDTA, spleen, lung, lymph nodes, and adrenal glands
- Tissues for PCR: anticoagulated blood, kidney, lymph nodes, intestinal wall, brain and other tissues
- Virus neutralisation
- Immunoblotting
- ELISA
- Immunofluorescence
- Viral DNA detection using PCR

Control

- No vaccine is available
- Control depends on the separation of susceptible species from reservoir hosts
- Identification and elimination of sheep harbouring OvHV-2 using the PCR assay in order to establish a virus-free flock
- Cattle should not graze pastures where wildebeest have grazed and given birth
- Co-housing of sheep and cattle should be avoided
- Bison, some deer and other highly susceptible species should not be allowed near sheep
- OvHV-2 free sheep can be produced by early weaning and isolation