

Malignant Catarrhal Fever: Current insights into epidemiology, pathogenesis, diagnosis, and control strategies

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ARTICLE INFO

Received: 01 July 2026

Accepted: 29 July 2026

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Keywords:

AIHV-1, Malignant Catarrhal Fever, OvHV-2, Ruminants, Virus.

ABSTRACT

Rhadinoviruses in the *Herpesviridae* family, mainly Ovine herpesvirus 2 (OvHV-2) and Alcelaphine herpesvirus 1 (AIHV-1), are the cause of Malignant Catarrhal Fever (MCF), a systemic viral illness of ruminants. In susceptible hosts like cattle, deer, and certain wild ruminants, the disease is sporadic and has a high death rate. Sheep and wildebeest are examples of natural reservoirs that are the main source of infection without clinical signs. Virus-containing aerosols are the main means of transmission to the end host. A pathogenic immune response to latent T lymphocyte infection, which causes systemic lymphoproliferation and non-suppurative vasculitis in multiple organs, is the primary cause of MCF. The eyes, gastrointestinal tract, respiratory system, and central nervous system are all affected by this illness. Clinically, the illness manifests as eye lesions, stomatitis, diarrhea, and neurological abnormalities as it advances from acute to peracute. A thorough approach that incorporates clinical symptoms, histological alterations, and the identification of the causative agent using molecular techniques like PCR is necessary for the diagnosis of MCF. Since there is currently no viable vaccine or targeted treatment, illness control relies on prevention by separating susceptible hosts from reservoirs and enforcing stringent biosecurity. As a foundation for scientific knowledge to assist disease mitigation efforts in ruminant populations, this study provides a thorough synthesis of the origin, epidemiology, pathophysiology, clinical symptoms, diagnosis, and control options of MCF.

Introduction

Ruminant infectious illnesses continue to be a significant barrier to the growth of the world's cattle industry (Hussain *et al.*, 2017). Malignant Catarrhal Fever (MCF) is a prominent viral agent that affects this population since it is sporadic and has a high death rate in susceptible hosts (Zakharova *et al.*, 2020). Ovine herpesvirus 2 (OvHV-2) and Alcelaphine herpesvirus 1 (AIHV-1), two members of the Rhadinovirus genus in the *Herpesviridae* family, are the main etiologic agents of this illness, which mostly affects cattle, deer, and some wild ruminants (O'Toole and Li, 2014).

Because MCF does not sustain ongoing transmission between susceptible hosts, it has a distinct epidemiological pattern (Li *et al.*, 2011). Natural reservoirs that serve as asymptomatic sources of infection are crucial to the disease's survival (Alsaad and Atshan, 2026). The main reservoirs are sheep and wildebeest, which can carry the virus without exhibiting any symptoms but occasionally expel it through respiratory secretions (Damayanti, 2016). The ecological interaction between the reservoir and vulnerable animals determines the development of disease since aerosol transmission is the primary mode of transmission over short distances (Hamid *et al.*, 2026).

MCF is not endemic to any one area, yet it is found throughout the world. Rather, it is modeled after livestock management systems and reservoirs (Yohanes *et al.*, 2026). The risk of exposure is increased by mixed husbandry methods and the close proximity of domestic animals and wildlife (Lankester *et al.*, 2015a). Therefore, rather than internal cycles of transmission within populations of cattle or deer, disease incidence reflects local ecological factors (Mlilo *et al.*, 2015).

The pathophysiology of MCF is primarily immunopathological (Mei-

er-Trummer *et al.*, 2009). The virus does not significantly cause lytic replication in T cells after infection (Gong *et al.*, 2023). An increased immune response brought on by unchecked T cell activation results in non-suppurative vasculitis and systemic lymphoproliferation (Dewals and Vanderplasschen, 2011). The eyes, gastrointestinal tract, respiratory system, central nervous system, and other visceral organs are all harmed by this method (Zakharova *et al.*, 2020). Therefore, tissue damage in MCF is more likely to result from the host's immunological reaction than from the virus itself (Russell *et al.*, 2009).

MCF typically presents with acute to peracute clinical symptoms that worsen quickly (Larska *et al.*, 2026). Ocular problems, erosive stomatitis, diarrhea, and neurological symptoms follow the initial symptoms of fever, anorexia, and general decline (Li *et al.*, 2011). Despite its relatively modest occurrence, this disease has a substantial clinical impact due to its exceptionally high mortality rate, which frequently approaches 100% in susceptible species (Hamid *et al.*, 2026).

Because of its ambiguous symptoms and resemblance to other ruminant diseases, MCF diagnosis necessitates a thorough approach (Russell *et al.*, 2009). A combination of clinical observations, histological alterations such as lymphocytic vasculitis, and the identification of viral DNA using molecular methods like polymerase chain reaction (PCR) are typically used for confirmation (Kumar *et al.*, 2021). Early case control is frequently hampered by limited field diagnostic capabilities (Oliveira *et al.*, 2021).

There isn't currently a cure for MCF or an effective vaccine (Wambua *et al.*, 2016). The focus of disease control is mostly on preventative measures, such as managing livestock systems to reduce interspecies interaction, removing reservoirs from susceptible hosts, and putting biosecurity in place (Hussain *et al.*, 2017). The only strategy that has been shown to

lower the likelihood of illness occurring in the field is this one (Orono *et al.*, 2019).

Therefore, this review aims to provide a comprehensive scientific overview of Malignant Catarrhal Fever, covering aspects of etiology, epidemiology, pathogenesis, clinical manifestations, diagnosis, and control strategies, as a basis for developing an understanding and future mitigation efforts.

Etiology

MCF is brought on by infection with viruses belonging to the genus Rhadinovirus in the family *Herpesviridae* (Atshan and Alsaad, 2026). These viruses are distinguished by their capacity to remain latently in reservoir hosts while causing lethal illness in non-adapted hosts (Larska *et al.*, 2026). OvHV-2 and AIHV-1, which are linked to sheep and wildebeest, respectively, as natural reservoirs, are the main etiologic agents found (Sood *et al.*, 2013).

The most frequent cause of MCF in cattle worldwide is OvHV-2 (Meier-Trummer *et al.*, 2009). Although this virus does not produce clinical illness in sheep, it is a major source of infection for vulnerable animals because it replicates in the respiratory epithelium and is expelled in nasal secretions (Wiyono *et al.*, 2021). AIHV-1, on the other hand, is more prevalent in African wildebeest herds and exhibits a similar infection pattern, with viral shedding mostly taking place early in the animal's life (Lanckester *et al.*, 2015b).

Rhadinoviruses are characterized by a double-stranded DNA genome and a preference for lymphoid cells, especially T lymphocytes (El-Tarabili *et al.*, 2008). The pathophysiology of the disease is caused by excessive immunological activation in the terminal host, whereas the virus maintains a latent infection in the reservoir host without causing a severe pathogenic immune response (Palmeira *et al.*, 2013).

Although there is genetic heterogeneity across viral isolates, strains linked to MCF do not significantly differ in their core pathogenic capabilities (Zakharova *et al.*, 2020). This implies that variations in the virulence of the virus itself have less of an impact on clinical manifestations than do viral-host interactions (Wiyono *et al.*, 2021).

Apart from OvHV-2 and AIHV-1, a number of additional Rhadinoviruses have been found in wild ruminant species; however, it is yet unknown how these viruses contribute to MCF in domestic animals (Zhu *et al.*, 2020). All of these etiologic agents, however, have the same capacity to linger latently and cause an immunopathological reaction in vulnerable hosts (Hamid *et al.*, 2026).

Epidemiology

The availability of natural reservoir hosts for rhadinoviruses, including OvHV-2 in sheep and AIHV-1 in wildebeest, has a significant impact on MCF's unequal global spread (Damayanti, 2016). Clinical cases are mostly seen in regions where domestic ruminants and virus-carrying wildlife interact ecologically. The disease's distribution pattern is irregular (Li *et al.*, 2011).

OvHV-2 is the main etiologic agent of MCF in cattle, which is most commonly documented in regions with dense sheep populations, such as Europe, North America, Australia, and New Zealand (Tomkins *et al.*, 1997; Taus *et al.*, 2014; Coradduzza *et al.*, 2022; Makoni *et al.*, 2024). Even though the sheep in these places do not exhibit clinical signs, transmission typically takes place in mixed livestock systems or in areas near sheep and cattle farms (Moré *et al.*, 2025).

In contrast, AIHV-1, which comes from wildebeests (*Connochaetes* spp.), is more commonly linked to MCF in East Africa and some parts of Southern Africa (Wambua *et al.*, 2016). The wildebeest migration season is typically linked to infection in domestic cattle in these areas, especially during the calving time when viral excretion through respiratory secretions increases dramatically (Sharma *et al.*, 2019).

Land-use patterns and animal production methods also have an impact on the spatial distribution of MCF (Shiba *et al.*, 2026). Compared to isolated, intensive systems, areas with large livestock systems close to wildlife habitats are more vulnerable (Miller *et al.*, 2013). Additionally, the frequency of contact between reservoirs and susceptible hosts may increase due to changes in land use and the spread of cattle into protected areas (Gale *et al.*, 2009).

The illness does not show a permanent endemic pattern in vulnerable hosts because of their need on reservoirs, despite the fact that case reports of MCF in domestic ruminants have been recorded throughout several continents (Damayanti, 2016). Therefore, rather than a constant cycle of transmission throughout cow populations, illness incidence is more likely to follow local ecological changes (Mlilo *et al.*, 2015).

Pathogenesis

Rhadinovirus infection (mostly OvHV-2 and AIHV-1) and an unchecked host immune response interact intricately to cause MCF (Meier-Trummer *et al.*, 2009). Instead of primarily causing lytic replication in vulnerable species like cattle, the virus causes immunological regulatory disturbance, which results in multisystem tissue damage (Russell *et al.*, 2009). Figure 1 illustrates the pathogenesis of MCF, beginning with aerosol transmission of Rhadinoviruses from asymptomatic reservoir hosts, primarily sheep or wildebeest, to susceptible, dead-end hosts such as cattle.

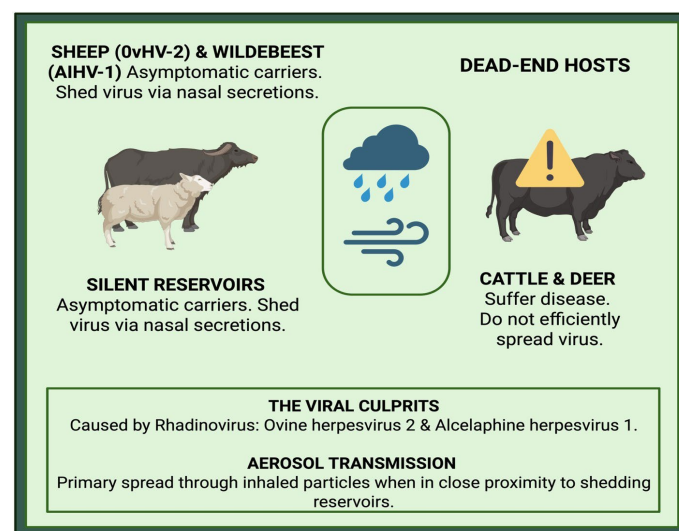


Figure 1. Malignant catarrhal fever spreads from asymptomatic sheep or wildebeest reservoirs to dead-end hosts through aerosol exposure.

Aerosols from natural sources are typically the source of infection. Once within the body, the virus creates a latent phase by infecting T cells (Dewals *et al.*, 2008). The virus can then move systemically through the circulation to a number of target organs, such as the vascular system, lymph nodes, gastrointestinal tract, eyes, and central nervous system, by activating and clonally expanding these infected lymphoid cells (Martins *et al.*, 2017).

Lymphoproliferation along with non-suppurative vasculitis is the primary characteristic of MCF pathophysiology (Sharma *et al.*, 2019). Endothelial damage, edema, and compromised vascular integrity result from lymphocyte infiltration into blood vessel walls brought on by excessive T cell activation (Saura-Martinez *et al.*, 2021). Widespread systemic lesions affecting different organs are caused by this procedure (Zhu *et al.*, 2018).

Disease progression is largely influenced by immune dysfunction (O'Toole and Li, 2014). A chronic inflammatory response results from an imbalance between immune regulatory function and effector T cell activation (Dewals *et al.*, 2008). Proinflammatory cytokines including interleukin-2, interferon- γ , and tumor necrosis factor- α are produced in greater

quantities, which increases immune cell recruitment to target tissues and exacerbates vascular and parenchymal damage (Galasso *et al.*, 2024).

Perivascular mononuclear cell infiltration, endothelial degradation, and tissue necrosis as a result of reduced perfusion are characteristics of pathogenesis at the tissue level (Zaki *et al.*, 2016). The lack of a dominating viral cytopathic pattern in these lesions suggests that immunopathological mechanisms, rather than direct impacts of viral replication, are responsible for the damage (Meier-Trummer *et al.*, 2009).

The central nervous system (non-suppurative encephalitis), the gastrointestinal tract (mucosal ulceration), and the eyes (keratitis and uveitis) are the main organs affected by the increasing multi-organ involvement (Zakharova *et al.*, 2020). The significant mortality rate in clinical cases of MCF is a result of both systemic inflammation and vascular problems (Larska *et al.*, 2026).

Immune response

Immunopathological reactions to Rhadinovirus infection, specifically AIHV-1 and OvHV-2, are the main source of tissue destruction in MCF, where the immune response plays a crucial role in defining the progression of disease pathogenesis (Hamid *et al.*, 2026). Instead of causing widespread lytic replication in terminal hosts like cattle and deer, the virus causes excessive and uncontrollable immune activation (Russell *et al.*, 2009).

The virus remains dormant in T cells, especially the CD4+ and CD8+ subsets, after the first infection (Saura-Martinez *et al.*, 2021). In addition to increased expression of surface activation markers and the generation of inflammatory mediators, these cells experience unchecked activation and proliferation (Dewals and Vanderplasschen, 2011). An imbalance between the activation and control of the adaptive immune response results from this syndrome, which is indicative of defective immunological regulation (Hamid *et al.*, 2026).

In MCF, the cellular immune response plays a major role (Meier-Trummer *et al.*, 2009). Pro-inflammatory cytokines like interferon- γ (IFN- γ), interleukin-2 (IL-2), and tumor necrosis factor- α (TNF- α) are produced in greater quantities when cytotoxic T cells are overactivated (Sasiain *et al.*, 1998). These cytokines help target tissues, especially the vascular system and parenchymal organs, by enhancing the inflammatory response

and attracting more immune cells (Zakharova *et al.*, 2020). This causes endothelial damage and widespread perivascular lymphocytic infiltration (Saura-Martinez *et al.*, 2021).

Although it plays a secondary function, humoral immune system engagement has also been identified in addition to the cellular immunological response (Anderson *et al.*, 2007). There is no effective defense against the advancement of the disease because the generation of particular antibodies against viral antigens is insufficient to eradicate latent infection (Cunha *et al.*, 2022). This weak antibody response implies that T cell modulation, rather than humoral responses, is more important for immunological control in MCF (Meier-Trummer *et al.*, 2009).

The loss of immunoregulatory systems, especially regulatory T cell (Treg) activity, is one of the main characteristics of the immune response in MCF (Damayanti, 2016). A chronic inflammatory response is the outcome of this malfunction, which causes a lack of control over effector T cell activation (Russell *et al.*, 2012). The primary pathological features of the illness, non-suppurative vasculitis and systemic lymphoproliferation, are exacerbated by this imbalance (Saura-Martinez *et al.*, 2021).

The unchecked immune response causes a chronic inflammatory response at the tissue level, which is typified by lymphocytes, macrophages, and plasma cells infiltrating the blood vessel walls (Russell *et al.*, 2009). Increased arterial permeability, endothelial damage, and ischemia-induced secondary tissue necrosis are the outcomes of this process (Hamid *et al.*, 2026). The systemic and extensive immune response is directly responsible for the clinical manifestations, which include eye lesions, mucosal abnormalities, and neurological symptoms (Gong *et al.*, 2023).

Pathology

Systemic lymphoproliferative lesions and non-suppurative vasculitis are pathological features of MCF that indicate a pathogenic immune response to rhadinovirus infection, mainly AIHV-1 and OvHV-2 (Luvizotto *et al.*, 2010). The vascular system, lymph nodes, gastrointestinal tract, eyes, and central nervous system are the organs where these lesions are most common (Headley *et al.*, 2020). Table 1 summarizes the main pathological changes in MCF based on the involvement of various organ systems.

Lymphocytic vasculitis affecting small to medium-sized blood vessels is the main pathogenic alteration in MCF (Saura-Martinez *et al.*, 2021).

Table 1. Pathological organ manifestations in MCF.

System/Organ	Main pathological lesion	Characteristics of changes	Functional/clinical implications	References
Vascular system	Non-suppurative lymphocytic vasculitis	Infiltration of lymphocytes and macrophages into small-medium blood vessels, accompanied by endothelial degeneration, intimal thickening, and increased vascular permeability	Tissue edema, multifocal hemorrhage, thrombosis, and impaired organ perfusion	Saura-Martinez <i>et al.</i> (2021); Martins <i>et al.</i> (2017); Zakharova <i>et al.</i> (2020)
Lymphoid system (lymph nodes and spleen)	Reactive lymphoid hyperplasia	Excessive proliferation of lymphocytes due to viral antigenic stimulation without neoplastic transformation	Enlargement of lymph nodes and spleen, reflecting systemic immune activation	Schock and Reid (1996); Meier-Trummer <i>et al.</i> (2009); Makoni <i>et al.</i> (2024)
Gastrointestinal tract	Mucosal necrosis and ulceration	Damage to the oral cavity, esophageal, and intestinal mucosa accompanied by severe inflammatory infiltration and fibrin exudation	Diarrhea, anorexia, impaired nutrient absorption, and dehydration	Teankam <i>et al.</i> (2006); Swai <i>et al.</i> (2013); Simon <i>et al.</i> (2003)
Eye	Interstitial keratitis and uveitis	Ocular inflammation secondary to vasculitis, associated with corneal edema and optic tissue involvement	Photophobia, visual impairment, and progression to blindness	Teankam <i>et al.</i> (2006); Ricer (2015)
Central nervous system	Non-suppurative encephalitis	Perivascular lymphocytic infiltration, gliosis, and neuronal degeneration	Neurological dysfunction, ataxia, and behavioral abnormalities	Hierweger <i>et al.</i> (2021)
Liver	Interstitial hepatitis and focal hepatocellular necrosis	Perivascular lymphocytic infiltration with localized hepatocellular damage and parenchymal degeneration	Impaired hepatic function and reduced metabolic capacity	Pesavento <i>et al.</i> (2019); Russell <i>et al.</i> (2009)
Kidney	Interstitial nephritis	Inflammatory cell infiltration and renal parenchymal injury leading to compromised filtration	Reduced filtration function and disturbances in fluid balance	Moore <i>et al.</i> (2010); Russell <i>et al.</i> (2009)
General histopathological features	Lymphocytic vasculitis with mononuclear infiltration	Multifocal tissue damage characterized by perivascular mononuclear infiltration and absence of prominent intranuclear inclusions; lesions are predominantly immune-mediated	Supports an immunopathological mechanism rather than direct viral cytopathic effects	Martins <i>et al.</i> (2017); Hristov and Peshev (2016)

Endothelial degeneration, intimal thickening, and increased vascular permeability are the results of mononuclear cells, mainly lymphocytes and macrophages, infiltrating the vascular wall (Martins *et al.*, 2017). This illness frequently results in local thrombosis, multifocal bleeding, and tissue edema, all of which impede organ perfusion (Zakharova *et al.*, 2020). Figure 2 illustrates the pathological progression of MCF, highlighting the central role of immune-mediated lymphocytic vasculitis in the development of multisystemic lesions.

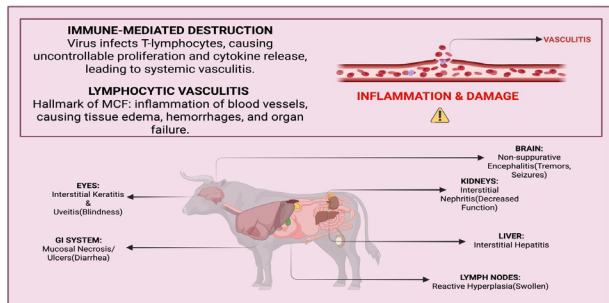


Figure 2. Malignant catarrhal fever causes immune-mediated vasculitis that damages multiple organs in infected cattle.

There is noticeable lymphoid hyperplasia in the lymphoid system, especially in the spleen and lymph nodes (Schock and Reid, 1996). Excessive systemic immunological activation is reflected in this unchecked lymphocyte multiplication (Meier-Trummer *et al.*, 2009). Nevertheless, this proliferation is reactive and linked to viral antigenic stimulation rather than primary neoplastic change, in contrast to neoplasia (Makoni *et al.*, 2024).

Mucosal necrosis and ulceration, especially in the oral cavity, esophagus, and intestines, are characteristics of gastrointestinal tract lesions (Teankam *et al.*, 2006). Severe inflammatory infiltration and fibrin exudation frequently accompany these alterations, impairing absorption and causing clinical symptoms including diarrhea and anorexia (Swai *et al.*, 2013). In more severe situations, substantial vascular injury may cause necrosis to spread to the submucosal layer (Simon *et al.*, 2003).

Interstitial keratitis, uveitis, and corneal edema brought on by vasculitis of the ocular tissues are common pathologies in the eye (Teankam *et al.*, 2006). Due to damage to the optic nerve, these lesions frequently cause photophobia and eventual blindness (Ricer, 2015). In the meantime, neurological damage can be caused by non-suppurative encephalitis with perivascular lymphocyte infiltration, gliosis, and neuronal degeneration in the central nervous system (Hierweger *et al.*, 2021).

Additionally, the liver and kidneys exhibit pathological alterations such as parenchymal deterioration and perivascular lymphocytic infiltration (Pesavento *et al.*, 2019). The liver exhibits focal hepatocellular necrosis, while the kidney experiences interstitial nephritis with compromised filtration function (Moore *et al.*, 2010). These alterations support the disease's systemic character, which affects several organs at once (Russell *et al.*, 2009).

Histopathologically, MCF typically manifests as a combination of target tissue degradation, perivascular mononuclear cell infiltration, and lymphocytic vasculitis without evident intranuclear inclusions (Martins *et al.*, 2017). The lack of prominent direct cytopathic lesions demonstrates that immunopathological processes, not the direct consequences of viral replication, are responsible for tissue destruction (Hristov and Peshev, 2016).

Clinical manifestations

MCF's clinical symptoms can be acute or peracute, and they often progress quickly (Zakharova *et al.*, 2020). Systemic vasculitis and a pathogenic immune response to Rhadinovirus infection are the main causes of the multisystem involvement that results in symptoms. As a result, early on, clinical symptoms are frequently ambiguous (Iván *et al.*, 2022).

Early on, infected animals show signs of a general loss in health, including fever, anorexia, sadness, and decreased productivity in animals that are productive (Li *et al.*, 2011). Serous to mucopurulent nasal and ocular discharge, along with conjunctivitis that may develop into keratitis, are typical symptoms that indicate mucosal and vascular involvement as the disease advances (Damayanti, 2016).

The most well-known clinical form is the head and eye variant, which frequently results in blindness and is characterized by swelling and inflammation of the head region, including periorbital edema, epiphora, and interstitial keratitis (Atshan and Alsaad, 2026). Additionally prevalent are oral lesions including erosive and ulcerative stomatitis, which are accompanied by hypersalivation and trouble eating (Pérez-Guiot *et al.*, 2023).

Animals may suffer from watery to hemorrhagic diarrhea in the digestive tract as a result of intestinal mucosal ulcers (Zakharova *et al.*, 2020). Rapid weight loss and dehydration are common side effects of this illness (Headley *et al.*, 2020). In contrast, fast systemic vascular injury is the main cause of death in the peracute type, which can happen without any overt clinical symptoms (Larska *et al.*, 2026).

Neurological symptoms like ataxia, tremors, hypersensitivity, and behavioral abnormalities can be brought on by involvement of the central nervous system (Martins *et al.*, 2017). Non-suppurative encephalitis, which gradually decreases brain function, is linked to these symptoms. Seizures have also happened prior to death in certain instances (Sánchez *et al.*, 2013).

The course of the illness is frequently accompanied by additional systemic symptoms such lymphadenopathy, a prolonged fever, and limb weakness (Li *et al.*, 2011). The majority of MCF cases result in death shortly after the start of clinical signs, and the mortality rate is extremely high, particularly in highly sensitive animals like cattle and deer (Zakharova *et al.*, 2020).

Diagnosis

Because the symptoms of MCF are nonspecific and can resemble a number of other systemic infectious diseases in ruminants, a complete approach that incorporates clinical observations, pathology, and laboratory confirmation is necessary for the diagnosis (Li *et al.*, 2011). As a result, molecular and histological techniques are typically used to make a conclusive diagnosis (Martins *et al.*, 2017).

Animals exhibiting severe fever, ocular diseases (keratitis and conjunctivitis), erosive stomatitis, and increasing neurological impairment are clinically suspected of having MCF (Wiyono *et al.*, 2021). However, additional validation is necessary because these symptoms can coexist with other illnesses such foot and mouth disease, infectious bovine rhinotracheitis, or bovine viral diarrhea (Headley *et al.*, 2020).

An essential technique in the diagnosis of MCF is histopathological investigation (O'Toole and Li, 2014). Non-suppurative vasculitis, perivascular lymphocytic infiltration, and epithelial necrosis in different organs are common findings (Sharma *et al.*, 2019). Because the disease is multi-systemic, these lesions are usually found throughout the gastrointestinal tract, central nervous system, lymph nodes, and vascular system (Alsaad and Atshan, 2026).

Molecular methods, such as polymerase chain reaction (PCR), which is used to identify AIHV-1 or OvHV-2 DNA in tissue or blood samples, can be utilized to detect the causative agent (Riaz *et al.*, 2021). PCR is the preferred technique for laboratory diagnostic confirmation due to its high sensitivity and specificity (Crawford *et al.*, 1999). Figure 3 illustrates the diagnostic approach and control strategy for MCF.

Furthermore, viral antigens in infected tissues, especially lymphoid cells, can be found using immunohistochemistry (IHC) techniques (Anderson *et al.*, 2007). Although the sensitivity of this method varies according on the stage of infection and sample quality, it aids in mapping the spread of the virus within pathological lesions (Makoni *et al.*, 2024).

Because latent infection frequently occurs in natural reservoirs without clinical signs, serological testing has limits when it comes to identifying MCF (Hamid *et al.*, 2026). As a result, the use of antibodies is more epidemiological than conclusive because their presence does not necessarily correspond with active disease in vulnerable individuals (Moré *et al.*, 2025).

To differentiate MCF from other illnesses with comparable symptoms, including as respiratory viral infections, systemic bacterial diseases, and neurological abnormalities in ruminants, a thorough differential diagnosis is necessary (Li *et al.*, 2011). The gold standard for accurately diagnosing MCF is the combination of clinical evidence, histology, and molecular confirmation (Russell *et al.*, 2025).

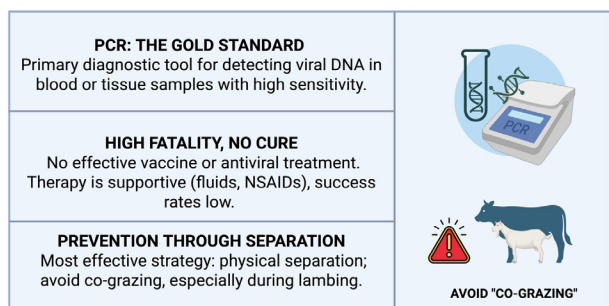


Figure 3. MCF diagnosis relies on PCR, while control depends on separation from reservoir hosts because no effective cure exists.

Differential diagnosis

Because MCF has non-specific, multisystem symptoms, especially in the early stages of infection, differential diagnosis is essential in clinical practice (Kumar *et al.*, 2021). A thorough differential screening is necessary to prevent misdiagnosis because symptoms like fever, eye lesions, stomatitis, respiratory distress, diarrhea, and neurological indications can mirror a number of other infectious diseases in ruminants (Martins *et al.*, 2017). Table 2 presents a comparison of diseases that share clinical manifestations with MCF in ruminants.

Bovine viral diarrhea (BVD) is one condition that is commonly employed in the differential diagnosis (Wang and Pang, 2024). Immunosuppression, diarrhea, and erosive mucosal lesions can be present in both conditions (Melkamsew *et al.*, 2025). However, BVD does not show the same severe systemic vasculitis as MCF and is typically associated with more significant leukopenia and a distinct pattern of infection (Bellino *et al.*, 2015).

Additionally, respiratory discomfort and ocular abnormalities are similar to infectious bovine rhinotracheitis (IBR) (Rimayanti *et al.*, 2024). In contrast to MCF, which is multisystemic and involves widespread vasculitis, IBR mostly causes respiratory symptoms such rhinitis and tracheitis with less systemic involvement (Petrini *et al.*, 2022).

Particularly when stomatitis and hypersalivation are present, foot and mouth disease (FMD) should be taken into consideration (Wong *et al.*, 2020). The primary distinctions are the development of distinctive vesicles in FMD and the extension of lesions to the udder and hooves, which are absent in MCF (Stenfeldt *et al.*, 2025). Additionally, FMD has a higher rate of transmission and epidemiological traits (Khairullah *et al.*, 2024).

In the acute stage, bacterial illnesses like pasteurellosis and septicemia can also resemble MCF, especially when there is a high fever and respiratory distress (Narsana and Farhat, 2015). But unlike MCF, bacterial infections typically show bacterial infection on culture and respond to antibiotic therapy (Giuliano *et al.*, 2019).

Additionally, neurological conditions like listeriosis (*Listeria monocytogenes*) are crucial differential diagnosis, particularly when ataxia and encephalitis are present (Pino and Delgado, 2025). The primary distinction is the histological pattern of lesions, MCF is characterized by non-suppurative encephalitis with perivascular lymphocytic infiltration, whereas listeriosis manifests as microabscesses in the brainstem (Coradduzza *et al.*, 2022).

Additionally, although they typically lack the systemic vasculitic lesions typical of MCF, parasitic or toxic disorders that affect the central nervous system should also be taken into consideration (Amara *et al.*, 2011).

Transmission

Rhadinovirus-carrying natural reservoir animals, mainly wildebeest for AIHV-1 and sheep for OvHV-2, are strongly associated with the spread of MCF (Gong *et al.*, 2023). Although the virus usually replicates subclinically in the respiratory epithelium of these reservoir species without producing illness, it is nevertheless released into the environment and acts as a major source of infection for vulnerable hosts (Russell *et al.*, 2009).

Aerosols containing viral particles from the respiratory secretions of the reservoir are the main way that MCF is spread (Yohanes *et al.*, 2026). Droplets produced when breathing, coughing, or other activities that stimulate the production of mucous secretions might release viruses (Zakharova *et al.*, 2020). The most effective mode of transmission, especially in mixed livestock systems involving domestic and wild ruminants, is close inhalation exposure (Li *et al.*, 2011).

Table 2. Differential diagnosis of MCF in ruminants.

Disease	Clinical similarities with MCF	Main distinguishing features	Diagnostic distinguishing markers	References
Bovine Viral Diarrhea (BVD)	Erosive mucosal lesions, diarrhea, and immunosuppression	Absence of severe systemic vasculitis characteristic of MCF	Marked leukopenia, distinct infection pattern, and absence of extensive vasculitis	Wang and Pang (2024); Melkamsew <i>et al.</i> (2025); Bellino <i>et al.</i> (2015)
Infectious Bovine Rhinotracheitis (IBR)	Ocular lesions and respiratory distress	Clinical manifestations are predominantly restricted to the respiratory system rather than multisystemic involvement	Rhinitis and tracheitis are dominant, with limited systemic lesions	Rimayanti <i>et al.</i> (2024); Petrini <i>et al.</i> (2022)
Foot and Mouth Disease (FMD)	Stomatitis and hypersalivation	Presence of characteristic vesicular lesions involving the mouth, hooves, and udder; more rapid transmission	Vesicular lesions and high transmission rate with distinct epidemiological characteristics	Wong <i>et al.</i> (2020); Stenfeldt <i>et al.</i> (2025); Khairullah <i>et al.</i> (2024)
Pasteurellosis and bacterial septicemia	High fever and respiratory distress	Bacterial etiology with responsiveness to antibiotic treatment	Positive bacterial culture and absence of immune-mediated vasculitis	Narsana and Farhat (2015); Giuliano <i>et al.</i> (2019)
Listeriosis (<i>Listeria monocytogenes</i>)	Neurological manifestations including ataxia and encephalitis	Lesions are primarily confined to the brainstem rather than multisystemic involvement	Brainstem microabscesses rather than non-suppurative encephalitis with lymphocytic infiltration	Pino and Delgado (2025); Coradduzza <i>et al.</i> (2022)
Parasitic or toxic disorders affecting the central nervous system	Neurological manifestations	No involvement of systemic vasculitis	Absence of characteristic vasculitic lesions associated with MCF	Amara <i>et al.</i> (2011)

Although the virus is very labile outside the host, environmental contamination via nasal and oral secretions contributes to the spread of the virus in addition to aerosol transmission (Mushi and Rurangirwa, 1981). Since terminal hosts typically do not excrete large amounts of virus to sustain the infection cycle, direct contact between susceptible animals is not thought to be a primary route of transmission (Abuelzein, 2021).

High population density, mixed farming methods (co-grazing), and the close proximity of domestic ruminants to natural reservoirs are epidemiological factors that raise the likelihood of transmission (Moore *et al.*, 2010). The likelihood of being exposed to infectious aerosols might also be increased by environmental factors including inadequate ventilation and animal stress (Zakharova *et al.*, 2020).

The natural reservoir's active viral shedding phase, which is frequently linked to the animal's reproductive cycle or particular physiological parameters, is the most crucial time for transmission (Russell *et al.*, 2009). The risk of infection in susceptible hosts rises during this period due to a notable increase in the rate of viral shedding (Headley *et al.*, 2020).

Risk factors

Numerous risk variables associated with livestock management, host-reservoir dynamics, and ecological interactions affect the incidence of MCF (Mlilo *et al.*, 2015). Exposure to natural reservoir animals, which are the main source of Rhadinovirus, such as sheep (*OvHV-2*) and wildebeest (*AIHV-1*), is a major factor in the disease's incidence because it does not transfer effectively between susceptible animals (Gong *et al.*, 2023). Table 3 summarizes the various risk factors contributing to the incidence of MCF in ruminants.

A mixed husbandry system that uses both domestic ruminants and natural reservoirs is the main risk factor (Larska *et al.*, 2026). Cattle are more likely to inhale virus-containing aerosols when they co-graze or are geographically close to sheep or wildlife (Yohanes *et al.*, 2026). The most important epidemiological factors influencing the emergence of MCF cases in different livestock regions are these parameters (Zakharova *et al.*, 2020).

The density of animal populations contributes to the increased risk of transmission (Decker *et al.*, 2021). Particularly in enclosed pens with inadequate ventilation, high-density settings raise the airborne concentration of infectious aerosol particles (Hamid *et al.*, 2026). In susceptible hosts, these circumstances lengthen exposure times and raise the risk of infection (Li *et al.*, 2011).

Another risk factor that may affect an animal's susceptibility to infection is stress (Yohanes *et al.*, 2026). The immune system's effectiveness can be lowered by stress from travel, environmental changes, hunger, or comorbidities, which raises the risk of getting a clinical illness following viral exposure (Shiba *et al.*, 2026).

Transmission patterns are further influenced by environmental and seasonal factors (Larska *et al.*, 2026). There may be a brief increase in contact between domestic animals and natural reservoirs due to seasonal variations that impact population numbers and migration patterns (Atshan and Alsaad, 2026). Furthermore, the concentration of viral aerosols near cattle populations might rise in environments with poor air circulation (Abuelzein, 2021).

The likelihood of MCF outbreaks is increased by managerial variables such as inadequate animal health monitoring, poor biosecurity, and a lack of isolation between vulnerable species and reservoirs (Headley *et al.*, 2020). The prevalence of this disease is also significantly influenced by extensive livestock farming systems that lack distinct borders between domestic livestock and wildlife (Mlilo *et al.*, 2015).

Economic impact

The ruminant livestock industry, especially cattle and deer, is severely impacted economically by MCF because of high death rates and a dearth of efficient targeted treatments (Shiba *et al.*, 2026). Animal death is not the primary cause of economic losses; other factors include decreased productivity, control expenses, and disruptions to the cattle trade (Lankester *et al.*, 2015a).

Before mortality, MCF infection at the production level results in a sharp drop in productivity, including decreased milk output, weight loss, and reproductive issues in female animals (Zakharova *et al.*, 2020). This situation directly affects feed conversion ratios and business efficiency in intensive livestock systems, raising operating expenses per unit of production (Lankester *et al.*, 2015a).

In vulnerable species, acute and often usually lethal animal death causes the biggest losses (Li *et al.*, 2011). Livestock farmers are directly impacted financially by the loss of valuable animals, such as dairy or beef cattle, particularly in small and medium-sized businesses with little risk diversification (Oliveira *et al.*, 2021).

Additionally, efforts to diagnose and control diseases result in increased expenses (Iván *et al.*, 2022). Significant resources are needed for laboratory testing including PCR, histopathology, and epidemiological

Table 3. Risk factors that are associated with the incidence of MCF.

Risk factor category	Description of factor	Impact on MCF incidence	References
Host-reservoir interaction	Contact between domestic ruminants and natural reservoirs, particularly sheep (<i>OvHV-2</i>) and wildebeest (<i>AIHV-1</i>)	Increases exposure to Rhadinovirus through aerosol transmission from reservoir hosts	Mlilo <i>et al.</i> (2015); Gong <i>et al.</i> (2023)
Mixed husbandry system (co-grazing)	Shared grazing areas or close spatial proximity between cattle and sheep or wildlife	Major risk factor facilitating inhalation of virus-containing aerosols and increasing transmission opportunities	Larska <i>et al.</i> (2026); Yohanes <i>et al.</i> (2026); Zakharova <i>et al.</i> (2020)
Livestock population density	High animal density within confined areas, particularly enclosed housing systems	Increases the concentration of infectious aerosols and probability of exposure	Decker <i>et al.</i> (2021); Hamid <i>et al.</i> (2026); Li <i>et al.</i> (2011)
Poor environmental ventilation	Limited air circulation within housing facilities or livestock environments	Prolongs persistence of airborne viral particles and increases exposure duration	Hamid <i>et al.</i> (2026); Abuelzein (2021)
Animal stress	Transportation, malnutrition, environmental changes, or concurrent diseases	Reduces immune competence and increases susceptibility to clinical infection	Yohanes <i>et al.</i> (2026); Shiba <i>et al.</i> (2026)
Seasonal factors	Seasonal changes influencing reservoir migration patterns and population dynamics	Increases the frequency of contact between reservoirs and susceptible hosts	Larska <i>et al.</i> (2026); Atshan and Alsaad (2026)
Environmental conditions	Enclosed environments or areas with limited air circulation surrounding livestock populations	Increases local viral aerosol concentration and potential transmission	Abuelzein (2021); Hamid <i>et al.</i> (2026)
Poor biosecurity	Limited access control, inadequate sanitation, insufficient animal health monitoring, and lack of species separation	Facilitates viral exposure and spread among susceptible animals	Headley <i>et al.</i> (2020)
Extensive livestock farming system	Lack of clear boundaries between wildlife habitats and domestic livestock areas	Increases the likelihood of spillover events from natural reservoirs	Mlilo <i>et al.</i> (2015)

studies (Müller-Doblies *et al.*, 1998). Operating expenses for cattle are also increased by the implementation of biosecurity measures, such as species separation and reservoir population monitoring (Manuja *et al.*, 2014).

The commerce of animals and animal goods is also indirectly impacted by MCF (Lankester *et al.*, 2015a). Restrictions on animal movement and a decline in market trust in livestock goods from endemic areas can result from the presence of cases in a region (Zakharova *et al.*, 2020). This could cause supply chain disruptions and lower the regional livestock commodities' economic worth (Shiba *et al.*, 2026).

More generally, in regions where domestic ruminants and wildlife interact often, the existence of MCF may have an effect on the growth of the livestock sector (Russell *et al.*, 2009). Planning for the growth of the animal-based agriculture industry is impacted by the risk of this disease, which restricts livestock intensification in some locations (Lankester *et al.*, 2015a).

Vaccination

There is currently no commercially available MCF vaccine that consistently provides protection (Decker *et al.*, 2021). The biological intricacy of the causative organisms, rhadinoviruses like AIHV-1 and OvHV-2, which are dormant in reservoir hosts but produce immunopathological illness in vulnerable hosts, is the primary barrier to vaccine development (Li *et al.*, 2011).

Immunopathological processes that rely on T cell dysregulation and systemic inflammatory reactions rather than just active virus replication dominate the immune response in MCF (O'Toole and Li, 2014). These features make it more difficult to create a vaccine that can offer sterile protection because the main objective is to prevent the pathological immune activation that causes tissue harm in addition to eliminating the virus (Bainbridge *et al.*, 2025).

Attenuated viruses, inactivated vaccines, and viral protein subunit-based vaccines are only a few of the vaccine strategies that have been investigated (Yilmaz *et al.*, 2022). Research findings, however, show that the ensuing protective response is still insufficient to totally shield exposed animals from clinical illness (Lankester *et al.*, 2016a).

The creation of a universal vaccination is further hampered by antigenic diversity and variations in virus strains between reservoir and susceptible hosts (Bianchessi *et al.*, 2022). Immunization effectiveness may be diminished by mismatches between vaccine antigens and field viruses, particularly in natural exposure environments with high infection burdens (Cunha *et al.*, 2022).

The latent nature of infection in natural reservoirs, where animals like sheep and wildebeest continue to operate as sources of transmission despite not exhibiting clinical signs, is another complicated element (Reid *et al.*, 1984). Because of this, vaccination programs in susceptible hosts are not enough to end the disease's epidemiological cycle (Lankester *et al.*, 2016b).

Non-vaccination tactics, like keeping susceptible animals apart from reservoir species, enforcing stringent biosecurity protocols, and controlling livestock systems to reduce direct contact, have been the mainstay of MCF management initiatives up to this point (Zakharova *et al.*, 2020). As a result, vaccination is still in the experimental research stage and has not yet been incorporated into regular field control initiatives (Bainbridge *et al.*, 2025).

Treatment

The treatment strategy for MCF is supportive and palliative since there is currently no viable etiotropic medication (Li *et al.*, 2011). This is because, unlike viral replication, which can be directly targeted by traditional antiviral medications, the disease is predominantly mediated by a disordered immunological response to Rhadinovirus infection (Russell *et al.*, 2009).

et al., 2009).

The goal of supportive therapy is to lessen the intensity of clinical symptoms while preserving the patient's physiological state (Zakharova *et al.*, 2020). While electrolyte balance correction is required to preserve metabolic stability, intravenous fluid infusion is utilized to treat dehydration brought on by diarrhea or reduced feed intake (Hamid *et al.*, 2026). Another crucial element in maintaining anorexic animals' overall health is supplemental nourishment (Zakharova *et al.*, 2020).

Although they are not very successful at stopping the progression of the condition, non-steroidal anti-inflammatory drugs (NSAIDs) can be used to lower fever and systemic inflammation (Martins *et al.*, 2017). Local treatments like lubricants or topical anti-inflammatory drugs can be used to lessen discomfort in cases with severe ocular involvement, but they cannot stop more tissue damage (Russell *et al.*, 2009).

In order to prevent subsequent infections brought on by mucosal damage and weakened immunity, broad-spectrum antibiotics are occasionally administered as supplementary therapy (Damayanti, 2016). However, as MCF is not brought on by a bacterial pathogen, their use has little effect on the disease's fundamental course (Russell *et al.*, 2009).

Corticosteroids have been used in certain studies in an effort to reduce the overreaction of the immune system that underlies the pathophysiology of MCF (O'Toole and Li, 2014). However, because immunosuppression might exacerbate the illness at specific stages of the disease or raise the risk of subsequent infections, the outcomes are inconsistent and contentious (Martins *et al.*, 2017).

Because the disease progresses quickly and is nearly always deadly in vulnerable animals, the success rate of therapy is typically very low (Li *et al.*, 2011). Therefore, rather of curing MCF, treatment focuses more on enhancing animal wellbeing and reducing suffering (Zakharova *et al.*, 2020).

Control

Since there are now no viable treatments or vaccinations available, MCF control depends on interfering with the epidemiological interaction between the natural reservoir of Rhadinovirus and susceptible hosts (Damayanti, 2016). To reduce the chance of aerosol transfer from reservoirs like sheep (OvHV-2) and wildebeest (AIHV-1) to domestic ruminants, control techniques are preventive and risk-management focused (Li *et al.*, 2004).

Physical and ecological species isolation is the main management strategy. The best way to decrease viral exposure is to keep cattle away from sheep or wild animals (Shiba *et al.*, 2026). Avoiding co-grazing livestock systems is advised, particularly in regions with a history of MCF or large reservoir populations (Zakharova *et al.*, 2020).

A key element of the control method is strict biosecurity (Yohanes *et al.*, 2026). This entails limiting access to cages and feeding locations, managing animal movement, and cleaning facilities (Russell *et al.*, 2009). To lower the concentration of possible virus-containing aerosols in enclosed spaces, pens must be properly ventilated (O'Toole and Li, 2014).

Early case detection and the identification of high-risk locations are facilitated by epidemiological and disease surveillance (Headley *et al.*, 2020). Regular livestock health monitoring lowers the possibility of additional exposure within the population by enabling the quick isolation of animals exhibiting clinical symptoms (Shiba *et al.*, 2026).

Another essential component of MCF control is the management of natural reservoir populations (Larska *et al.*, 2026). In certain places, habitat zoning or limiting access to shared grazing areas are used to control interactions between domestic livestock and wildlife (Zakharova *et al.*, 2020). The livestock and wildlife conservation industries must work together to implement this strategy (Shiba *et al.*, 2026).

Effective control is also aided by teaching livestock owners about the dangers of MCF and safe husbandry techniques (Lankester *et al.*, 2015a). Farm-level disease incidence can be considerably decreased by raising awareness of the significance of species separation and biosecurity pro-

cedures (Yohanes et al., 2026).

Conclusion

The Rhadinovirus that causes MCF in ruminants has epidemiological traits based on natural reservoirs and aerosol transmission to vulnerable hosts. Clinical manifestations are mostly dependent on ecological interactions between domestic cattle and reservoirs like sheep and wildebeest because the disease does not show effective transmission between affected animals. In terms of pathogenicity, MCF is mostly caused by immunopathological processes such increased T-cell activation, which results in multisystem damage and systemic vasculitis without significant viral cytopathic involvement.

The diagnosis needs to be confirmed in the lab using histology and molecular techniques because the clinical presentations are severe and have a high death rate. Since there is currently no viable vaccination or targeted treatment, illness prevention depends on preventative measures like species isolation and biosecurity. Because of its substantial clinical and financial impact and the scarcity of current intervention strategies, MCF continues to pose a serious threat to animal health.

Acknowledgement

The authors thank Universitas Airlangga for the funding support with grant number 397/UN3.14/PT/2020.

Conflict of interest

The authors declare that there is no conflict of interest.

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