



Print ISSN: [1813-8497](#)

Online ISSN: [2410-8456](#)

<https://bjvr.uobasrah.edu.iq/>

Malignant Catarrhal Fever (A review)

Article Info.

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Article History

Received: Dec. 29, 2025

Accepted: Feb. 4, 2026

Published: March 31, 2026

Article type: Review Article

<https://doi.org/10.23975/bjvr.2026.168203.1270>

Abstract

Malignant Catarrhal Fever (MCF) is a severe, fatal viral disease in ruminants (such as cattle, bison, deer, buffalo) caused by Gamma-herpesviruses type 1, typically spread from asymptomatic carrier animals (wildebeest or sheep/goats). It's characterized by fever, severe inflammation, and ulceration of mucous membranes of the eyes, nose, and mouth, blindness, difficult breathing, diarrhea, and neurological signs, with a grave prognosis and no effective treatment or vaccine. The disease is nearly endemic in Iraq. (As the number of recorded clinical cases began to increase) and of significant clinical and epidemiological importance because of its adverse health effects on infected animals due to high infection rates and increased mortality among infected animals. The disease affects cattle and buffalo, especially those raised with sheep. The disease manifests in multiple forms, and due to its clinical significance, proper preventative measures must be taken to prevent its spread and minimize economic losses.

Keywords: MCF, Farm animals, Review.

Introduction

Malignant catarrhal fever (MCF) is a highly contagious and potentially fatal disease that infects domestic and wild animals, including cows, buffalo, deer, bison, exotic ruminants, and even pigs (1,2). The disease caused by ten different causative viruses belongs to the Rhadino-virus family (of the Herpesviridae family), where two of those viruses are carried by sheep. However, the other five viruses were found in those carrier animals (3,4).

The disease is present on most continents, including America, Africa, and Asia, and affects domestic and zoo animals. It is known by various synonyms, such as Malignant Head Catarrh, Malignant Catarrh, Catarrhal Fever, Snotsiekt, and Gangrenous coryza (1,5). Two essential and distinct groups of causative viruses have been documented, namely the Alcelaphinae–Hippotraginae group and the Caprine group. The Alcelaphine group of herpes virus 1 (A1HV-1) is globally endemic in wild animals and causes disease in those species. Moreover, the ovine type of herpesvirus 2 (OvHV-2) is endemic in sheep, and the caprine type of herpesvirus 2 (CpHV-2) infects goats and causes in utero infection (6,7,8). The disease can be transmitted directly between animals, and close contact is essential. Transmission also occurs when animals move away by one hundred meters or a little more. Aerosol infection has also been recorded (where diseased calves can release the causative agent via lacrimation and nasal discharges), in addition to in utero transmission (9,10). Nonetheless, Constable et al. (1) added that transmission through semen in rams is possible. On the contrary, the virus is rarely transmitted through colostrum and milk. Furthermore, transmission is more prevalent when sheep and cattle are housed or mixed, meaning when both susceptible and carrier animals are present.

It was shown that Cattle and buffalo of all ages were susceptible to the disease and could become infected. However, a high morbidity rate was registered in adults aged (11).

The disease is characterized by several forms (per acute, acute with alimentary tract form, acute with head and eye form, and mild form), each with distinct clinical manifestations. The per acute form is essential and may be the most common, presenting with high fever and rapid death, usually within 12-24 hours, which may be preceded by weakness and depression, as well as diarrhea that is always mixed with blood (1). Nevertheless, when the animal survives the per acute form, an acute form with clinical signs, including anorexia and mostly bilateral corneal problems (corneal opacity), is observed, with ocular mucus discharge that may progress to mucopurulent and then purulent. Moreover, the muzzles of diseased animals and the nasal tissue become encrusted. Diseased animals also show open-mouth breathing with dyspnea, as well as salivation due to stomatitis, characterized by hyperemic oral mucosa with necrotic foci (12).

Malignant catarrhal fever has been reported in Iraq (13). Therefore, reviewing and defining the disease are valid scientific methods for all scientists and those interested in developing plans to control and prevent this severe disease.

History of the disease

Malignant catarrhal fever (MCF) is a highly fatal, systemic lymphoproliferative disease that affects a wide range of ungulate species, including cattle, deer, buffalo, and, occasionally, horses (14). The causative agents belong to the genus *Macavirus* within the subfamily *Gamma-herpesvirinae*, with *Ovine herpesvirus 2* (OvHV-2) and *Alcelaphine herpesvirus 1* (AIHV-1) recognized as the principal etiologic agents. OvHV-2 causes sheep-associated MCF (SA-MCF), while AIHV-1 is linked to wildebeest-associated MCF (WA-MCF). Both viruses are typically asymptomatic in their natural reservoirs, sheep and wildebeest, but can induce severe, often fatal lymphoproliferative disease when transmitted to susceptible hosts (15,16).

Historically, MCF was first recognized in Africa in the late nineteenth century, when outbreaks occurred among cattle grazing alongside wildebeest during the calving season. Early European and African veterinarians described a highly febrile illness with corneal opacity, mucosal erosions, and ocular-nasal discharge decades before AIHV-1 was identified as the causative virus of WA-MCF (1,2). Throughout the twentieth century, researchers documented similar syndromes in cattle, deer, and other ruminants in contact with sheep in Europe, North America, and Asia. Molecular and serological studies subsequently validated *Ovis aries* as the natural host of OvHV-2, thereby designating SA-MCF as the principal global manifestation of the disease (15,17).

Recent advances in molecular epidemiology and genomic sequencing have significantly refined our understanding of *Macavirus* diversity, host range, and geographic distribution. MCF is now recognized as a multi-virus syndrome involving several genetically related *Macavirus* species capable of cross-species transmission. For example, a 2023 outbreak in Mexico provided the first molecular identification of OvHV-2 in horses and domestic artiodactyls, demonstrating susceptibility in these species and the ongoing risk of interspecies infection (14). Likewise, OvHV-2 DNA has been detected in apparently healthy cattle and buffalo in endemic regions, suggesting subclinical circulation and maintenance within livestock populations (18). Comparative molecular analyses across free-ranging cervids and zoo ungulates have revealed extensive genetic variation and viral recombination among *Macaviruses*, emphasizing their long-term co-evolution with ruminant hosts (19).

Clinically, MCF is characterized by mucogingival ulceration, hypersalivation, keratoconjunctivitis, anorexia, and progressive emaciation, typically culminating in death (1,2). Neuropathological manifestations ranging from meningoencephalitis to variable cerebral lesions have also been reported, highlighting the systemic and immune-mediated nature of the disease (20). Molecular diagnostics, including PCR targeting the ORF75 gene and sequence analysis, have revolutionized detection accuracy, demonstrating > 98% nucleotide identity with reference OvHV-2 strains during recent outbreaks (14). Surveillance in mixed-species zoological collections and wildlife reserves has revealed numerous previously uncharacterized *Macavirus* genotypes, reinforcing the complexity of viral ecology and transmission dynamics (21).

Regionally, SA-MCF remains the dominant form across Europe, Asia, and the Americas, particularly in mixed sheep and cattle farming systems. In contrast, WA-MCF persists in sub-

Saharan Africa, where cattle and wildebeest interact (19,22). Recent molecular evidence also indicates Macavirus infections in atypical hosts such as wild boars and cervids, and even cases of transplacental transmission in ruminants (4, 23). These discoveries underscore the evolutionary adaptability of macaviruses and the ecological complexity underlying their persistence and spread. Despite remarkable progress in molecular virology, there is still no licensed vaccine or effective antiviral therapy for MCF. Consequently, control strategies rely primarily on biosecurity and ecological management, especially preventing contact between reservoir and susceptible species during the lambing or calving periods (1,19). From its discovery in nineteenth-century Africa to the genomic era, the evolving scientific understanding of MCF illustrates a dynamic host–virus relationship that continues to challenge global veterinary health, epidemiology, and wildlife conservation (15, 24).

The causative agent:

Malignant Catarrhal Fever (MCF) is caused by two principal gamma-herpesviruses in the genus *Macavirus* of the subfamily *Gamma-herpesvirinae* within the family *Herpesviridae*: Alcelaphine herpesvirus 1 (AIHV-1) and Ovine herpesvirus 2 (OvHV-2). These viruses are clinically and pathologically indistinguishable in their susceptible ruminant hosts but differ significantly in their ecological reservoirs, transmission routes, and epidemiological patterns. Alcelaphine herpesvirus 1 (AIHV-1) is maintained asymptotically in its natural reservoir, the blue wildebeest (*Connochaetes taurinus*), and causes the wildebeest-associated form of MCF (WA-MCF) when transmitted to cattle, buffalo, or other susceptible species. Recent immunogenomic investigations have identified a clonal expansion of CD8⁺ T-cells with an activation/exhaustion phenotype in cattle infected with AIHV-1, suggesting that viral latency in T-cells plays a central role in disease pathogenesis (16). Ovine herpesvirus 2 (OvHV-2) is endemic in domestic sheep (*Ovis aries*), where infection remains subclinical throughout life. Transmission to susceptible hosts occurs mainly via aerosolized nasal secretions, resulting in sheep-associated MCF (SA-MCF). A recent seroepidemiologic survey in Brazil demonstrated widespread circulation of OvHV-2 antibodies in dairy herds, confirming the ubiquity of sheep-borne exposure even in the absence of clinical disease (4). The complete genome of an OvHV-2 strain, published, provided refined phylogenetic insights into Macavirus diversity and evolution (25). Neither AIHV-1 nor OvHV-2 exhibits efficient horizontal transmission among susceptible hosts (e.g., cattle-to-cattle), emphasizing that control relies primarily on separating reservoir and target species rather than inter-herd biosecurity alone. Moreover, macaviruses have also been detected in free-ranging cervids and small ruminants, such as goats and deer, expanding the recognized host and reservoir range (23). Molecular and phylogenetic analyses consistently confirm that OvHV-2 and AIHV-1 are the principal causative agents of MCF outbreaks worldwide, showing high nucleotide identity among isolates from various regions and host species when transmitted to susceptible animals particularly cattle, buffalo, deer, bison, and occasionally horses these viruses induce a severe lymphoproliferative and vasculitis syndrome, characterized by uncontrolled immune activation and high fatality (14,16).

**Epidemiology of Malignant Catarrhal Fever:
Occurrence, Prevalence, and Transmission:**

The disease arises following cross-species transmission to incidental hosts (4). Two principal epidemiological forms of MCF are recognized worldwide. The sheep-associated form (SA-MCF), caused by OvHV-2, is prevalent in regions where sheep cohabit or graze near susceptible species. The wildebeest-associated form (WA-MCF), caused by AIHV-1, occurs mainly in East and Southern Africa and coincides with wildebeest calving periods when viral shedding is highest (2,17). Cross-species transmission is a constant risk in places where different species live together, such as zoos. A large-scale surveillance study of 715 ruminants representing 96 species detected gamma-herpesvirus DNA in 22.5% of samples. Still, only 6.8% of these PCR-positive animals exhibited clinical MCF, indicating that infection is common, yet clinical disease remains infrequent. Moreover, 44 distinct viral genotypes were identified, underscoring extensive genetic diversity and the potential for interspecies transmission (1,21).

Outbreaks of MCF have been documented globally in both domestic and captive wildlife populations. A notable 2023 outbreak in Mexico demonstrated OvHV-2 infection across several artiodactyl species and horses, with 26.2% of tested animals PCR-positive and clinical cases confirmed in multiple species. This event provided the first confirmed detection of OvHV-2 in horses in the Americas (14). In endemic settings, infection among reservoir hosts is widespread, but only a small proportion of exposed susceptible animals develop disease. Molecular surveys in Pakistan identified OvHV-2 DNA in asymptomatic cattle and water buffalo, revealing subclinical transmission within mixed ruminant populations (26). Similarly, serological studies in South American dairy herds confirmed broad exposure to OvHV-2, supporting the global distribution of SA-MCF (4,5).

Transmission of MCF primarily occurs through direct or indirect contact with nasal and ocular secretions in reservoir hosts. Sheep and wildebeest shed large amounts of virus during the lambing and calving seasons, particularly from young or stressed animals (1,19). Aerosolized spread is also possible in dense or mixed-species environments, such as zoological collections and communal farms, where animals share confined airspace. At the molecular level, the AIHV-1 genes A7 and A8 are essential for efficient viral propagation: A7 mediates cell-to-cell spread, whereas A8 facilitates cell-free transmission. Absence or disruption of either gene impairs the virus's capacity to activate CD8⁺ T lymphocytes, which are central to MCF immunopathogenesis (16). The detection of viral DNA in both clinically healthy and diseased animals of the same species highlights the complex relationship among host immunity, viral genetics, and environmental conditions (6,7).

Relevance to cows, Buffalo, and Under-Reported Regions:

Cows and water buffalo are highly susceptible to OvHV-2 infection. Molecular evidence from South Asia and Europe confirms spillover in cows and buffalo herds (19). Despite this, virus surveillance data from the Middle East remain scarce, even though mixed buffalo-sheep or cow-sheep farming, shared watering points, and seasonal lambing create ideal conditions for viral transmission. This gap underscores the importance of regional sero-molecular studies, especially

in Iraq, where cow and buffalo farming is extensive and epidemiological data on MCF are virtually absent. MCF is a globally distributed disease characterized by high viral prevalence and low clinical incidence. Cross-species transmission from asymptomatic reservoirs, coupled with environmental and management risk factors, drives its sporadic outbreaks. Direct and aerosol exposure to secretions from sheep and wildebeest remain the primary routes of infection, while molecular determinants, such as the AIHV-1 A7 and A8 genes, enhance viral dissemination (13,16). To stop the spread of MCF and protect at-risk livestock populations worldwide, it is vital to strengthen surveillance, improve diagnostics, and enforce strict biosecurity measures in environments with multiple species.

Morbidity and Mortality Rates of Malignant Catarrhal Fever (MCF):

Malignant Catarrhal Fever (MCF) is widely recognized as one of the most lethal viral diseases affecting domestic and wild ruminants, resulting in an exceptionally high fatality rate in clinically affected animals. Transmission primarily occurs from asymptomatic reservoir hosts, particularly sheep carrying ovine herpesvirus-2 (OvHV-2) and wildebeest carrying alcelaphine herpesvirus-1 (AIHV-1), to susceptible species such as cattle, water buffalo, bison, and various cervids (15,20). Because the virus establishes latent infection in reservoir hosts without inducing disease, susceptible species are exposed to a pathogen against which they have no natural immunity. Once clinical signs appear, the disease progresses rapidly and severely, with most studies reporting a case fatality rate near 100%. This makes MCF a significant economic and veterinary problem worldwide (1). The disease is one of the deadliest for susceptible ruminants because it is almost always fatal once symptoms appear. A thorough examination of MCF outbreaks in Brazil from 1956 to 2018 revealed that the case fatality rate in most outbreaks ranged from 90% to 100% across species, including cattle, water buffalo, swine, and deer (14,29). In highly susceptible species such as bison, outcomes are particularly devastating. One well-documented outbreak in a bison feedlot resulted in a 51.2% mortality rate in the entire herd following 19 days of exposure to sheep (9). This highlights that while the case fatality rate for individual animals is almost absolute, the impact at the herd level can be catastrophic. In contrast to its consistently high fatality rate, the morbidity rate of MCF, the proportion of a herd that becomes sick, is highly variable. It is influenced by several factors, including:

Host Species: Bison and deer are more likely to get sick than cattle.

Virus Strain: Viruses transmitted from wildebeest (AIHV-1) may differ in virulence from the sheep-associated virus (OvHV-2).

Exposure Dynamics: The proximity, duration, and intensity of contact with reservoir hosts directly impact the rate of transmission.

Generally, the disease is described as sporadic in cattle, affecting only a few animals within a herd at any given time. However, under specific epidemiological conditions, significant outbreaks can occur. Studies in East African pastoralist communities report that annual losses from MCF-induced fatalities range from 3% to 12% (24).

Malignant Catarrhal Fever remains a formidable disease, with a case fatality rate approaching 100% in clinically affected animals. In contrast, its morbidity rate is highly variable, ranging from low-level sporadic cases (under 1%) to devastating outbreaks (exceeding 80%), particularly in highly susceptible species such as bison and deer, or under conditions of intense exposure to reservoir hosts. Given the absence of an effective treatment, control strategies that prevent transmission, such as separating susceptible species from reservoir hosts and developing effective vaccines, remain the only viable approaches to mitigating the significant economic losses caused by this disease.

Economic Importance of Malignant Catarrhal Fever (MCF):

Malignant catarrhal fever can cause substantial economic losses in livestock systems, although its perceived importance varies across regions and production settings. MCF, a highly fatal viral disease, remains a significant constraint in areas where domestic livestock and wildlife interact, particularly in East Africa, where wildebeest and sheep serve as primary reservoirs. Severe clinical outcomes, near-100% case fatality in susceptible hosts, and the absence of effective curative treatment make MCF one of the most economically consequential diseases at the wildlife-livestock interface (1,27). Direct economic losses stem from the sudden death of valuable cattle, buffalo, and small ruminants, leading to immediate reductions in herd size, milk production, meat yield, and long-term genetic potential. In pastoral and agro-pastoral systems, annual losses from MCF-associated mortality have been reported to range from 3% to 12% in affected communities, reflecting a substantial burden on household income and livestock productivity (2,28). Additional direct costs include veterinary interventions, diagnostic testing, and the replacement of valuable breeding stock.

Indirect and broader socioeconomic impacts further amplify the burden of MCF. The disease can disrupt local and national livestock markets, reduce household resilience, and threaten food security among pastoralist populations highly dependent on cattle productivity. MCF outbreaks may also trigger land-use conflicts at the wildlife–livestock interface, increase costs for preventive management, and cause long-term loss of genetic resources in breeding herds (19). Despite these significant consequences, MCF is not always prioritized by livestock keepers over more frequent and familiar diseases such as foot-and-mouth disease or contagious bovine pleuropneumonia. The sporadic nature of MCF outbreaks, coupled with challenges in field diagnosis and limited awareness among pastoralists, contributes to underestimating its actual economic impact and may reduce investment in preventive measures (1,2). Recent progress in control strategies, particularly field vaccination trials, has shown promising outcomes. Early results demonstrate strong immunogenicity and minimal adverse reactions, indicating that vaccination could substantially reduce both direct losses and the broader socioeconomic impacts of the disease in pastoralist communities (5,10). However, commercial availability and widespread implementation of such vaccines remain developmental challenges. Overall, MCF represents a significant economic threat in regions characterized by frequent wildlife-livestock interactions. Although direct and indirect losses can be considerable, the recognition of the disease’s importance leads to gaps in control

efforts. Continued investment in awareness, diagnostic capacity, and the development of effective vaccines is essential to reducing the economic burden of MCF and strengthening the resilience of livestock-dependent communities (12,17).

Zoonotic impact:

Malignant catarrhal fever is not considered a zoonotic disease; there is no evidence that these viruses infect humans or pose any risk to human health (14,24). Surveillance studies and outbreak investigations have consistently shown that MCF is restricted to animal hosts, with no documented human infections (19,28). In general, while MCF has a significant impact on animal health and agricultural productivity because it can cross species barriers among ruminants, it does not pose a zoonotic threat, as humans are not susceptible to infection by the causative viruses (1,14).

Diagnosis of Malignant Catarrhal Fever in Field Animals:

Clinical and Pathological Diagnosis in Field Animals

Field diagnosis begins with recognition of characteristic clinical signs, including mucogingival ulcers, hypersalivation, corneal opacity, reduced food intake, and weight loss. These signs, while suggestive, are not definitive and must be supported by pathological findings. Histopathology typically reveals lymphoproliferative inflammation and vasculitis in affected organs, findings that strongly indicate the disease in field cases (5,23).

Laboratory and Molecular Diagnostics

PCR Testing: Detection of OvHV-2 or AIHV-1 DNA in blood (especially the buffy coat) or tissues using endpoint or quantitative PCR is the gold standard for confirming MCF in field animals. PCR is highly sensitive and specific, and positive results can be further validated by sequencing viral gene fragments (13,16).

Immunohistochemistry and Immunofluorescence: These methods detect viral antigens in tissue sections or cultured cells, providing additional confirmation of infection (14).

Cell Culture: Inoculation of primary cell cultures (e.g., rabbit testis cells) with field samples can reveal viral cytopathic effects, such as cytomegalic cells and intranuclear inclusion bodies (18).

Sequencing: Sequence alignment of PCR products with reference strains confirms the viral identity and can reveal epidemiological links (13,14).

Diagnostic Methods for MCF in Field Animals

Effective diagnosis of MCF in field animals requires integrating clinical observations, characteristic pathological findings, and confirmation by molecular and immunological laboratory tests. PCR and immunohistochemistry are central to confirming the causative viruses, while sequencing provides definitive identification and epidemiological insight (14,26).

Treatment and Control of Malignant Catarrhal Fever

Currently, there is no effective antiviral or curative treatment for MCF. Management of affected animals is limited to supportive care, including fluid therapy, nutritional support, and symptomatic relief; however, these measures do not alter the course of the disease or improve survival rates (1,19).

Recent research has focused on preventive strategies, particularly vaccination. A field trial in some countries demonstrated that an attenuated AIHV-1 vaccine induced virus-specific and virus-neutralizing antibodies in cattle and reduced AIHV-1 infection rates by 56% among vaccinated animals exposed to wildebeest. However, the vaccine's ability to prevent fatal MCF could not be conclusively determined because the number of deadly cases observed in the study was low (16). This suggests that while vaccination can reduce infection rates, its effectiveness in preventing clinical disease requires further investigation. In addition to vaccination, control of MCF relies heavily on management practices aimed at minimizing contact between susceptible livestock and reservoir hosts, such as wildebeest and sheep, especially during periods of high risk (e.g., wildebeest calving season) (2,19). These biosecurity measures remain the cornerstone of MCF control in endemic regions.

Advances in understanding the molecular biology of AIHV-1 have identified viral genes essential for disease development, such as A7 and A8, which are involved in viral spread and pathogenesis. Targeting these genes may offer new avenues for future vaccine development and disease intervention, although such strategies are still under investigation and not yet available for field application (5,16). Nevertheless, the current approach to MCF generally involves supportive care for affected animals, vaccination to reduce infection rates, and strict management practices to prevent exposure to reservoir hosts. There is an ongoing need for improved vaccines and therapeutic interventions to control and treat this disease effectively (1,2,19).

Conclusion

It had been concluded that, MCF represents a significant challenge to veterinary health and livestock productivity, necessitating continued research into effective vaccines and control strategies. Moreover, strengthening surveillance and diagnostic capabilities, along with raising awareness among livestock keepers, is crucial for mitigating the economic burden of MCF and protecting livestock populations.

Conflicts of interest

The authors declare that there is no conflict of interest.

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أحمه النزلية الخبيثة (مراجعة بحثية)

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الحمى النزلة الخبيثة (MCF) مرض فيروسي حاد، غالباً ما يكون مميتاً، يصيب المجترات (مثل الأبقار والبيسون والغزلان والجاموس) وينتقل عادةً من حيوانات حاملة للمرض لا تظهر عليها أعراض (مثل حيوانات النو أو الأغنام/الماعز). يتميز هذا المرض بالحمى والالتهاب الشديد وتقرح الأغشية المخاطية في العينين والأنف والفم، مما يؤدي إلى العمى وصعوبة التنفس والإسهال وأعراض عصبية، مع صعوبة التكهن بمستقبل المرض وعدم وجود علاج أو لقاح فعال. يكاد المرض ان يكون متوطناً في العراق (وذلك لتزايد تسجل الحالات السريرية) وذات أهمية سريرية وبائية كبيرة لما لها من تأثيرات صحية سلبية على الحيوانات المصابة بسبب نسب الإصابات المرتفعة فضلاً عن زيادة نسبة هلاك الحيوانات المريضة. حيث يصيب المرض الأبقار والجاموس، وبخاصة الحيوانات التي تربي مع الأغنام معاً. ويظهر المرض بأشكال متعددة، ولأهمية المرض سريرياً، يجب اتخاذ التدابير الوقائية الصحيحة لمنع انتشاره وتقليل الخسائر الاقتصادية.

الكلمات المفتاحية: الحمى النزلية الخبيثة، حيوانات المزرعة، مراجعة بحثية .