Fever pitch

By Alice Fraser

Outbreak patterns and predisposing factors of malignant catarrhal fever in New Zealand cattle and deer.

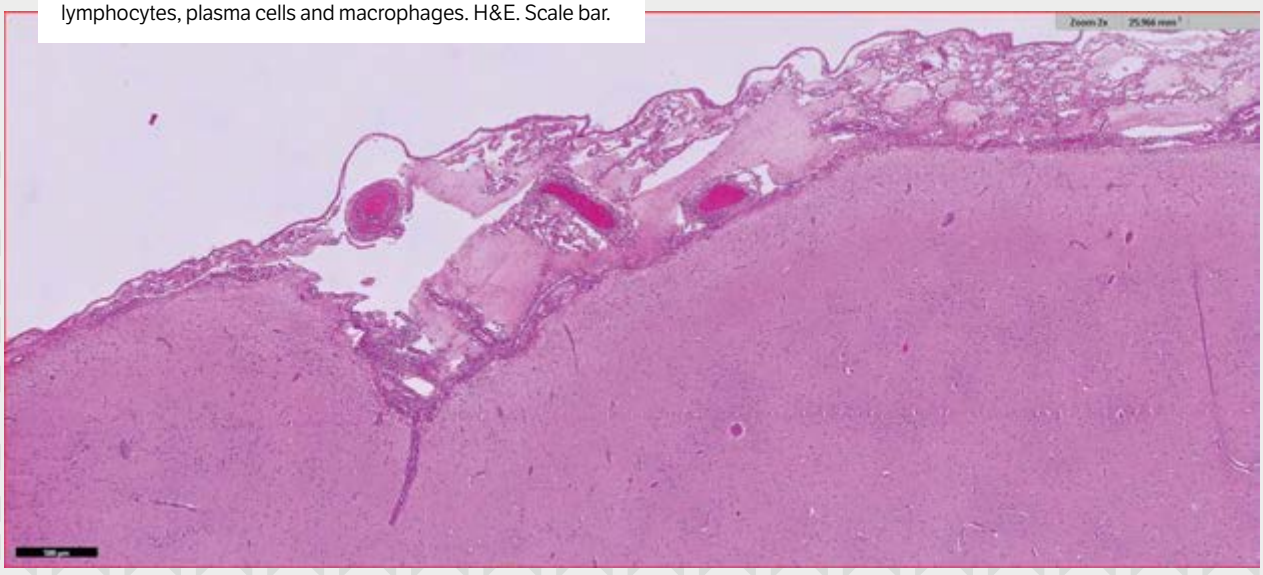
Background
Malignant catarrhal fever (MCF) is a usually fatal disease of cattle and deer (and other susceptible ruminants) caused by gammaherpesviruses. In New Zealand the disease occurs as the ovine-associated form, caused by ovine herpesvirus-2 (OvHV-2), carried asymptomatically by sheep, with particularly high levels of virus shed by sheep aged six to nine months (after the colostral-derived maternal antibody levels have waned), mostly via nasal secretions.

Transmission from sheep to susceptible species usually occurs sporadically; affected animals are considered dead-end hosts, as there is no evidence of onward transmission between cattle or deer. The pathogenesis of MCF involves a dysregulated T-cell-mediated immune response that produces lymphocytic vasculitis and epithelial necrosis (O'Toole and Li, 2014). This immunopathological process requires a mature adaptive immune system; hence the disease most commonly affects animals of one to two years of age and very rarely occurs in animals less than six months old. Periods of likely high exposure relating to farm management factors also play a role in the incidence of age affected.

Clinical presentation and diagnosis
Clinically, MCF in cattle and deer presents as an acute febrile illness characterised by a range of findings including depression, anorexia, ocular opacity, mucopurulent nasal discharge, oral and nasal erosions, skin lesions, dysentery, seizures and death. Peracute cases may present as a sudden death. Rarely, cutaneous forms occur with a predominance of skin lesions. In deer, sudden death without overt premonitory signs or severe dysentery followed by death within 12–24 hours are the typical presentations. Mortality is high, and recovery is rare.

Diagnosis is via a PCR test for OvHV-2. In live animals the required sample is EDTA whole blood (to test for viral DNA in white blood cells in the buffy coat). In dead animals, the PCR can be done on heart blood or fresh tissue (particularly lymphoid tissue). In addition, histologic evaluation of a full range of postmortem tissues, including lymphoid tissues and brain (and any tissues with gross changes), reveals typical lesions of MCF, which should assist in differentiation from other diseases causing similar presenting signs or sudden death.

FIGURE 1: Brain, lymphohistiocytic meningoencephalitis and vasculitis. Blood vessel walls are infiltrated by small and large lymphocytes, plasma cells and macrophages. H&E. Scale bar.



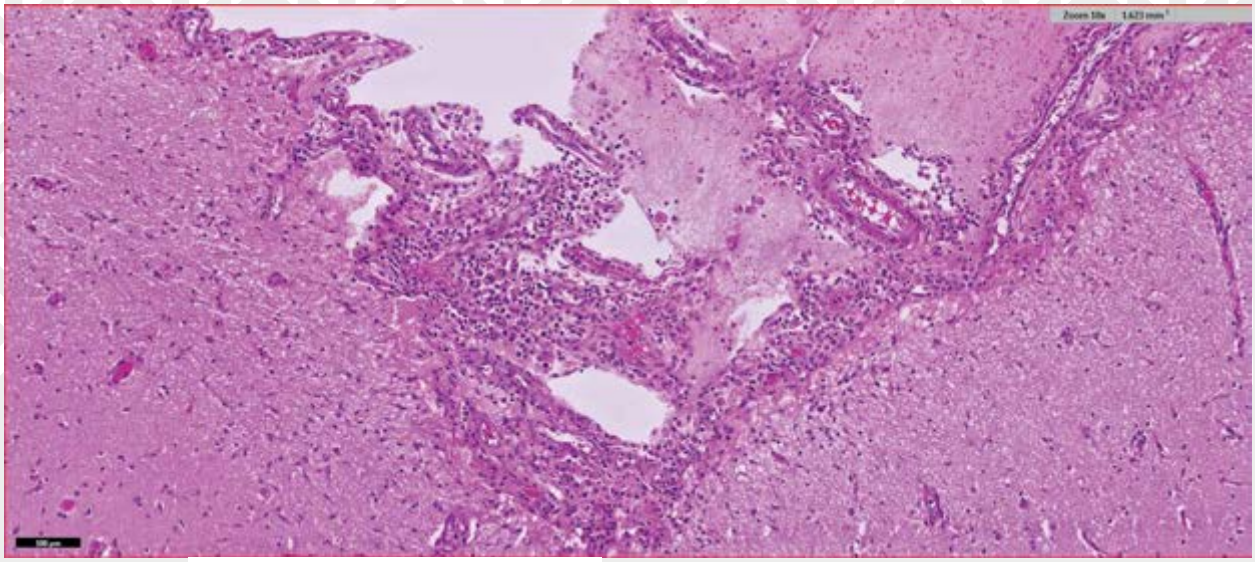


FIGURE 2: Higher power. H&E. Scale bar.

The virus establishes a latent, lifelong infection in its natural host species without causing disease.

Histopathological lesions

The histopathological features of MCF are distinctive and largely consistent across susceptible ruminant species. The principal lesion is a lymphocytic vasculitis, characterised by infiltration of medium- and small-calibre vessel walls by dense cuffs of lymphocytes, plasma cells and macrophages, frequently accompanied by endothelial swelling, fibrinoid necrosis, and vascular thrombosis (O'Toole and Li, 2014; Løken et al., 2009). These vascular changes underlie much of the multisystemic tissue necrosis observed at necropsy.

Secondary lesions reflect this immune-mediated vasculopathy and are most prominent in epithelial and lymphoid tissues. Common findings include necrotising and erosive inflammation of the upper respiratory and alimentary mucosa, corneal oedema and keratitis, and meningoencephalitis with perivascular lymphoid cuffing and gliosis (O'Toole and Li, 2014). Lymphoid hyperplasia, particularly of the spleen and lymph nodes, is a consistent feature. In deer, lesions are often most striking in the gastrointestinal and respiratory tracts, whereas in cattle, ocular and neurological involvement is more frequently emphasised. These patterns reflect the systemic nature of OvHV-2 infection and its pathogenesis as a T-cell-driven lymphoproliferative vasculitis rather than a direct cytopathic viral effect.

Outbreaks

The disease is more commonly of a sporadic nature, and outbreaks are often less expected. However, in the early days of deer farming in New Zealand, during the late 1970s and 1980s, outbreaks of MCF were common, mostly attributed to management practices in which sheep and deer were mixed. Once awareness of the disease and the influencing factors in farming deer developed, management changes were implemented to separate deer from sheep, particularly around lambing and fawning times; as a result, the incidence of these outbreaks was greatly reduced through the 1990s and 2000s. More recently, there have been occasional outbreaks incurring significant losses in cattle or farmed deer herds and triggering renewed interest in the environmental and management factors that may predispose to infection and influence disease expression under New Zealand farming conditions.

Outbreaks are most likely to occur when susceptible hosts are exposed to high levels of virus shedding from sheep and are under environmental and management conditions that favour transmission and stress. Cases most commonly occur in winter and early spring.

This pattern coincides with the period of increased virus shedding by sheep and higher stocking densities on mixed farms. Deer appear particularly susceptible, with mortality rates of up to 10% reported in outbreak situations. Cattle outbreaks, although less frequent, can also result in significant losses within affected mobs.

Other contributing factors may include climatic stress, nutritional status, animal age and breed susceptibility, and management practices such as sequential or mixed grazing of sheep and susceptible stock. The incubation period can be long, up to several months, so that investigation of the period of exposure and susceptibility is often retrospective.

The virus establishes a latent, lifelong infection in its natural host species without causing disease. In sheep, infection typically occurs early in life, and periods of virus shedding are most pronounced in younger sheep, around lambing and during stress events such as transportation or cold weather. Transmission to susceptible hosts occurs primarily via aerosolised nasal secretions.

Predisposing and risk factors for outbreaks

New Zealand outbreak reports reveal consistent epidemiological and management factors increasing the likelihood of multi-animal infection:

1. Proximity to sheep, especially lambing ewes and hoggets, on farm boundaries.
2. Mixed farming practices, especially shared paddocks or sequential grazing.
3. Season and climate, with winter-spring predominance.
4. Stress and stocking density, especially under feed restrictions or adverse weather.
5. Species and breed susceptibility, with deer particularly vulnerable.
6. Environmental conditions: the virus survives in the environment for short periods only, favouring cool, humid conditions.

Implications for prevention

Because no vaccine exists, management remains the cornerstone of prevention. Recommendations include physical and temporal separation between sheep and susceptible species during lambing, delaying grazing of lambed paddocks, and reducing stress through adequate nutrition and shelter. (The strategy of stock separation of cattle and sheep has to be carefully balanced with the worm-controlling advantages of mixing cattle and sheep, by taking into account factors such as stock age, season, other viral susceptibility factors, and worm lifecycle.)

Conclusions and implications for New Zealand livestock management

MCF remains a sporadic but significant cause of mortality in New Zealand cattle and deer. Outbreaks can occur when intense exposure to OvHV-2 coincides with environmental or physiological stress. Mixed livestock systems together with winter-spring climatic or nutritional stress factors can create predictable periods of elevated risk. VS

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