BOVINE MAMILLITIS
BOVINE ULSERATIF MAMILLITIS
PSEUDO LUMPYSKIN DISEASE

Bovine herpesvirus 2 (BHV-2) is the causative agent of two diseases.

I. The first is localized in the udder and called bovine herpes mammillitis.

II. The second is a generalized cutaneous form named Allerton disease or pseudo-lumpy skin disease, due to its similarity with the capripoxvirus infection causing lumpy skin disease.

Etiology

- Alphaherpesvirinae
- BHV-2
- It is serologically one type
- Antigenically close with human herpes viruses (herpes simplex 1 and 2).
- Virus forms roundness, lysis, and intranuclear inclusions in bovine cells.

- Cattle and buffalo are natural hosts of BHV-2. Sheep and goats can be experimentally infected and develop local lesions
- BHV-2 can occur sporadically or in outbreaks that often have a seasonal association with cold weather and may result in reduced milk production and increased susceptibility to bacterial mastitis.
- The generalized form is often seen in South Africa, summer and autumn. Arthropods are also thought to transmit the virus. However, vector identification was not performed.
- Mamillitis form, milking machines is the most important reason.
  - Arthropods have also been reported to be transmitted.
- Outbreaks are associated with the introduction of subclinically infected animals into the herd.

Clinical Signs

➢Bovine herpes mammillitis

- The incubation period is 4 to 10 days.
- This disease is usually observed in dairy cows during the second part of the year and usually heifers in 2 to 10 days after calving. It can also be observed in bulls.
- The virus is transmitted directly from animal to animal and indirectly by contaminated material.
- Biting flies could also play a role in virus transmission.
- The lesions are localized on the teats, and rarely on the udder and perinea.
- The skin is swollen and translucent and some vesicles may be visible. The lesions appear blue or purple.
- Teats are generally painful, and affected cows often resist milking, leading to development of mastitis.
- They evolve as ulcers and resolve without complications within 4 weeks.
- Suckling calves may become affected, showing the same lesions on the lips, the nose and in the mouth.

https://www.vetstream.com/treat/bovis/illustration/herpes-mammillitis-sloughing-teat-2

➢Pseudo-lumpy skin disease

- Pseudo-lumpy skin disease is a generalized and febrile disease.
- Circumscribed nodules suddenly appear on the skin of the whole body.
- These nodules are hard, palpable and circular.
- A slight depression is visible in their center.
- After a few days, necrosis follows, the nodules evolve as ulcers and are covered by scabs.
- After 2 weeks, the lesions resolve but temporarily leave hairless areas.

http://www.fao.org/docrep/003/t0756e/T0756E03.htm

Diagnosis

Clinical diagnosis

Bovine herpes mammillitis is suspected when the characteristic lesions appear on the teats, especially in late summer and early winter.

The disease must be differentiated from pseudo-cowpox (milker's nodule).

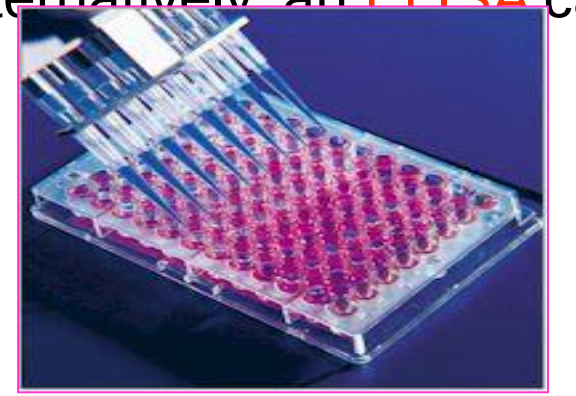
Pseudo-lumpy skin disease produces lesions very close to these induced by lumpy skin disease. However, the slight depression observed in the center of the nodules is characteristic of pseudo-lumpy skin disease.

Laboratory

The virus can be isolated from the lesions, especially from vesicles when they are present.

Serological diagnosis is achieved by seroneutralization.

Alternatively, an ELISA can be performed for the detection of specific BHV-2 antibodies.



Prevention and Control

- No commercial vaccine against BHV-2 is available.
- Control is only performed during BHV-2 outbreaks, preventive controls are not used.
- Affected cows should be isolated from the herd and milked separately.
- The milking machine should be disinfected with iodophores, and insecticides should be used to eliminate biting flies.

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Caprine Herpesvirus 1 (CpHV 1)

- CpHV-1
- Features of Herpesviridae
- Alphaherpesvirinae

- CpHV 1 is closely related to infectious bovine rhinotracheitis virus (BHV-1) of cattle and causes sporadic outbreaks of late-term abortions often unassociated with other clinical signs.

Figura 3. Balanopostitis pustular y ulcerativa de ligera a grave, multifocal coalescente, infectado por herpes virus. Semental Alpino Francés.

Figure 3. Mild to severe coalescent multifocal pustular and ulcerative balanoposthitis, infected with herpes virus. French Alpine male.

1) The virus causes vulvovaginitis, balanoposthitis, and respiratory disease in adult goats
2) Enteric and systemic diseases in neonatal goats.

Fetuses can be fresh or autolyzed and do not contain diagnostic gross lesions. Presumptive diagnosis is by microscopic identification of necrosis with the presence of intranuclear inclusion bodies in the liver, lungs, and other organs

Transmission

- All nasal, conjunctival, genital exudates, semen

Diagnosis

- Definitive diagnosis is by identification of CpHV 1 by isolation, PCR, or immunologic staining methods.
- Not all fetuses contain lesions or virus, so multiple fetuses should be submitted.

Infected goats can become latently infected and can shed the virus during times of stress.

Vaccines are not commercially available.

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- https://www.msdvetmanual.com/reproductive-system/abortion-in-large-animals/abortion-in-goats

CORYZA GANGRENOSA BOVUM

- Malignant Catarrhal Fever

- Malignant catarrhal fever (MCF) is an infectious systemic disease that presents as a variable complex of lesions affecting mainly ruminants and rarely pigs.
- It is mainly a disease of domestic cattle, water buffalo, Bali cattle, American bison, and deer.
- In some species, such as bison and some deer, MCF is acute and highly fatal and can affect large numbers of animals.
- With occasional exceptions, in cattle the disease is normally seen sporadic and affects single animals.

Etiology

- MCF results from infection by one of several members of a group of closely related ruminant gammaherpesviruses of the *Rhadinovirus* genus.
- The MCF subgroup of viruses, called MCFV, contains at least 10 members, five of which are currently known to cause disease.

- Wildebeest-associated MCF: alcelaphine herpes virus 1 (AlHV-1). Endemic in wildebeest populations worldwide.
- Sheep-associated MCF: ovine herpesvirus 2 (OvHV-2). Endemic in most sheep populations worldwide.
- Caprine-associated MCF: caprine herpesvirus 2 (CpHV-2): Endemic in most domesticated goat populations worldwide, and causes MCF in cervids.
- Unknown origin: causes MCF in white-tailed deer (MCFV-WTD).
- Roan antelope origin: Hippotragine herpesvirus-1 was used to experimentally induce MCF in rabbits.

Transmission

AlHV-1

- Transmission of AlHV-1 within free-living wildebeest populations is very efficient: all wildebeest calves are infected within the first few months of life by in utero, direct contact or aerosol routes, and remain carriers for life.
- Virus is shed by wildebeest calves in nasal and ocular secretions, mainly in the cell-free form
- Most transmission by wildebeest calves occurs at 1–2 months of age – transmission after six months of age is rare
- Transmission to susceptible hosts occurs only from wildebeest; there is no definitive evidence that MCF-affected animals transmit the disease horizontally to others
- Most cases of wildebeest-associated MCF occur when susceptible animals are exposed to parturient wildebeest or young calves, or pasture contaminated by them.

OvHV-2

- Transmitted mainly by the respiratory route, probably in aerosols
- Shed intermittently in nasal and ocular secretions mainly, particularly by 6- to 9-month-old lambs, but also reported in feces and semen (OvHV-2 DNA detected in semen of domestic rams)
- Some lambs are infected in utero, while with most lambs are infected perinatally, though infection may not occur in some situations till after 3 months of age.
- Close contact with sheep by susceptible species is usually required, but cases have been reported when sheep and cattle were separated by 70 meters, and in bison herds up to 5 km from a lamb feedlot
- Transmission is only from sheep to susceptible hosts, not horizontally between infected hosts


https://tr.wikipedia.org/wiki/Koyun

- AlHV-1, OvHV-2 and CpHV-2 seem to be carried in many or all individuals of the reservoir species, and the virus spreads readily within infected herds.
- The case fatality rate from MCF viruses is 80-90% in symptomatic cattle, and approaches 100% in symptomatic bison, deer and water buffalo.
- Residual corneal opacity is often seen in recovered cattle, but complete recovery is also possible.

Clinical Signs

The clinical signs of MCF are highly variable and range from peracute to chronic.

- In general, the most obvious signs develop in the more long-term cases.
- Peracute: either no clinical signs are detected, or depression followed by diarrhea and dysentery may develop 12–24 hours prior to death
- In general: high fever, increased serous lachrymation and nasal exudate progressing to profuse mucopurulent discharge, inappetence, 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

Bovine, muzzle. Multiple shallow erosions are filled with dried nasal exudate.

Bovine, muzzle. The muzzle is hyperemic, multifocally covered by adherent mucopurulent exudate, and contains many shallow erosions.

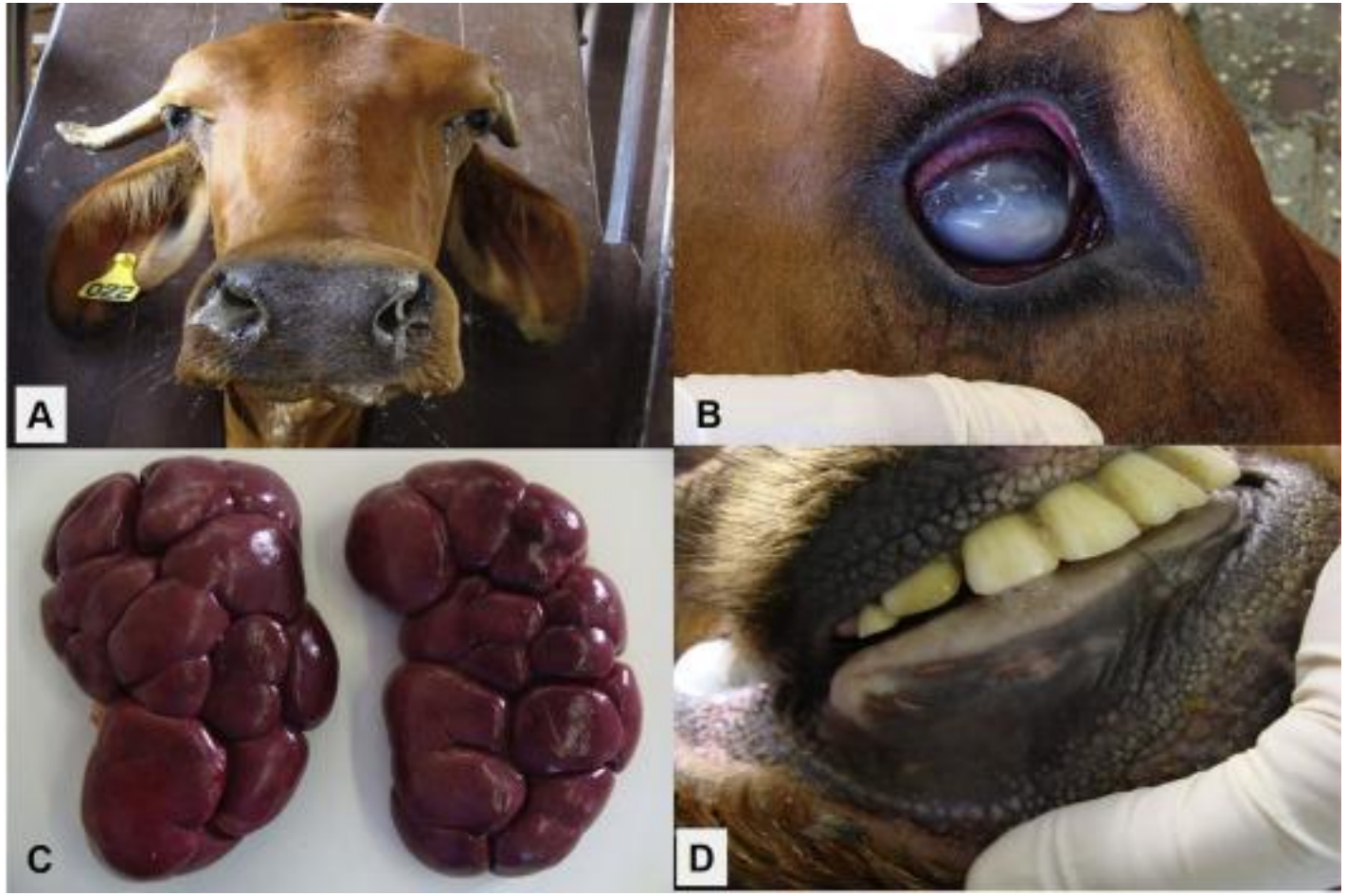
Bovine, oral mucosa. There is a gingival hyperemia and focal erosion

Corneal opacity causes blindness

the surface of the muzzle has been sloughed in this animal

Affected cattle are profoundly depressed with a high fever

http://www.nadis.org.uk/bulletins/malignant-catarrhal-fever-(mcf).aspx



Case 1 had mucopurulent nasal secretion (A) corneal oedema of the right ocular globe (B), fibrinopurulent bronchopneumonia, petechial haemorrhages within the mesenteric, thoracic and pleural surfaces, ulcerative stomatitis and rhinitis, pulmonary oedema, lymphadenomegaly and bilateral multifocal haemorrhagic nephritis (C). The fetus carried by this cow had normal external appearance and measured 30 cm in crown–rump length. Case 2 had bilateral corneal oedema, ulcerative stomatitis (D) and abomasitis.

Headley, S. A., Pimentel, L. A., Oliveira, V. H. S., Toma, H. S., Alfieri, A. F., Carvalho, A. M., ... & Alfieri, A. A. (2015). Transplacental transmission of ovine herpesvirus 2 in cattle with sheep-associated malignant catarrhal fever. Journal of comparative pathology, 153(4), 206-211.

- Salivation and oral hyperemia may be an early sign, progressing to erosions of the tongue, hard palate, gums and, characteristically, the tips of the buccal papillae
- Superficial lymph nodes may be enlarged, and limb joints may be swollen
- Nervous signs such as hyperesthesia, incoordination, nystagmus and head pressing may occur alone or with signs described above
- A few infected animals may recover following mild or even quite severe clinical reactions

Forms

- Historically, MCF has been described as having several “forms”—mild, peracute, head and eye, intestinal.

1. Peracute: either no clinical signs are detected, or depression followed by diarrhea and dysentery may develop 12–24 hours prior to death.
2. Intestinal: Fever, Diarrhea, hemorrhagic gastroenteritis or hematuria may also be seen, although these signs are less common than in bison and deer
3. Cutaneal (Skin) form: Skin lesions (erythema, exudation, cracking, crust formation) are common in animals that do not succumb quickly. Skin ulceration and necrosis may be extensive or restricted to the udder and teats
4. Neurologic form: Nervous signs such as hyperaesthesia, incoordination, nystagmus and head pressing may occur alone or with signs described above

5. Head and eye:

- high fever, increased serous lachrymation and nasal exudate progressing to profuse mucopurulent discharge, inappetance, and decreased milk yields.
- In cattle, swollen lymph nodes and severe eye lesions (panophthalmitis, hypopyon, corneal opacity) are more frequent, and hemorrhagic enteritis and cystitis less frequent, than in deer and bison.
- Salivation and oral hyperaemia may be an early sign, progressing to erosions of the tongue, hard palate, gums and, characteristically, the tips of the buccal papillae
- Superficial lymph nodes may be enlarged, and limb joints may be swollen
- Progressive bilateral corneal opacity, starting at the periphery, is characteristic.

Cow with MCF

Malignant catarrhal fever

erosions on the tongue

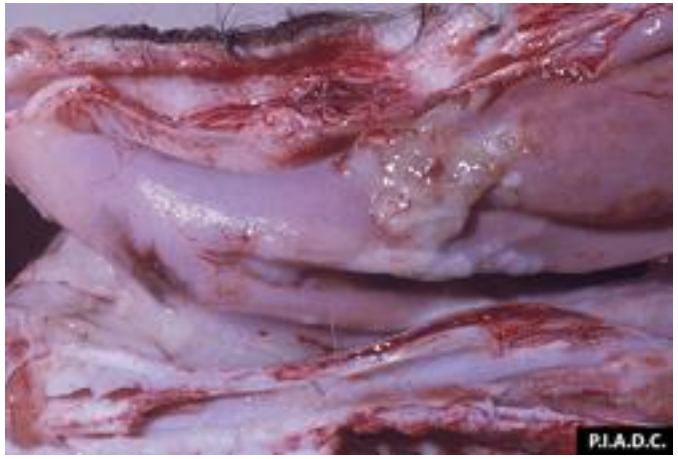
Post Mortem

- The principal lesions are
  - inflammation and necrosis of respiratory, alimentary, or urinary mucosal epithelium;
  - subepithelial lymphoid infiltration;
  - generalized lymphoid proliferation and necrosis;
  - widespread vasculitis.
- Mucosal ulcerations and hemorrhage are common.
- Hemorrhages may be present in many parenchymatous organs, particularly lymph nodes.
- A classic but not pathognomonic histologic lesion is fibrinoid necrosis of small muscular arteries, but vessels of all types may be inflamed, including those in the brain.
- Prominent white nodules representing intramural and perivascular proliferation may be apparent, particularly in the kidneys.

Bovine, hard palate. There are multiple coalescing mucosal erosions.

Bovine, skin. There are numerous raised plaques (multifocal dermatitis).

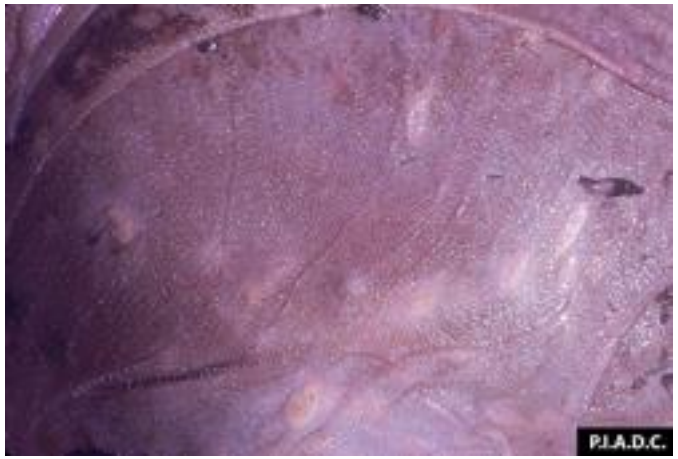
Bovine, head, sagittal section. Mucoid exudate multifocally covers the nasal and pharyngeal



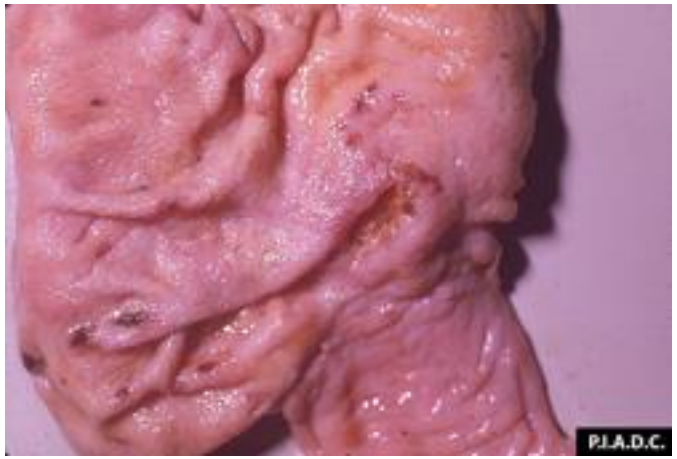
Bovine, nasal turbinate. There is a small amount of mucoid exudate.

Bovine, prescapular lymph nodes: Moderately (left) to markedly enlarged (right) due to MCF.

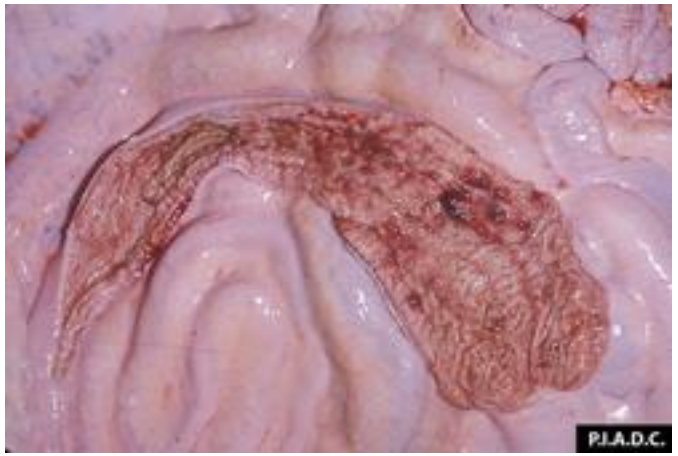
Bovine, prescapular lymph node. There are foci of hemorrhage (and necrosis) in the cortex, and the medulla is edematous.



Bovine, omasum. Omasal leaves contain multiple pale foci of necrosis; on the right there are several ulcers.



Bovine, cecum and ileum. There are scattered small foci of mucosal hemorrhage and erosion.



Bovine, spiral colon. There are multiple mucosal hemorrhages.

Bovine, kidney. Multiple pale foci in the cortex are foci of institial nephritis.

Bovine, colon. There is severe longitudinal linear congestion of the mucosa.

Diagnosis

- Both AlHV-1, and OvHV-2 have been transmitted experimentally to cattle, rabbits and hamsters, which develop lesions characteristic of MCF.
- Viral DNA has been detected in clinical material from MCF cases caused by both AIHV-1 and OvHV-2 using the PCR. PCR is becoming the method of choice for diagnosing both forms of the disease.
- Serological tests
  - Virus neutralisation
  - Immunoblotting
  - Enzyme-linked immunosorbent assay (ELISA)
  - Immunofluorescence
  - Paired serum samples (5 ml) taken 3–4 weeks apart
- Virus isolation from peripheral blood leukocytes (AlHV-1) or lymphoid cells

Differential diagnosis

- Rinderpest
- Bovine viral diarrhoea mucosal disease
- Infectious bovine rhinotracheitis
- Bluetongue
- Epizootic haemorrhagic disease
- Foot and mouth disease
- Vesicular stomatitis
- Ingestion of caustic materials or some toxic plants

Control and Prevention

- The prognosis is severe.
- No treatment has been found to provide any consistent benefit. Stress reduction of subclinical or mildly affected animals is indicated.
- No vaccine is currently available.
- Virus-free sheep can be produced through early weaning and isolation.
- The only other effective control strategy is separation of carriers from susceptible species.
- When large numbers of potent shedders are present, such as in lamb feedlots, distances **more than 1 km** may be necessary to protect highly susceptible species.

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- http://www.cfsph.iastate.edu/Factsheets/pdfs/malignant_catarrhal_fever.pdf
- http://www.msdvetmanual.com/generalized-conditions/malignant-catarrhal-fever/overview-of-malignant-catarrhal-fever
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Feline Herpesvirus 1

- Feline viral rhinotracheitis (FVR) is an upper respiratory tract infection of the nose and throat in cats.
- It is caused by feline herpesvirus 1 (FHV-1).
- FHV-1 is a common cause of ocular and upper respiratory disease in cats and kittens, and a potential cause of eosinophilic dermatitis.
- Cats of all ages are susceptible, but kittens are at a higher risk and may be infected at about five weeks of age. Pregnant cats or cats suffering from a lowered immunity due to a pre-existing disease are also at higher risk.

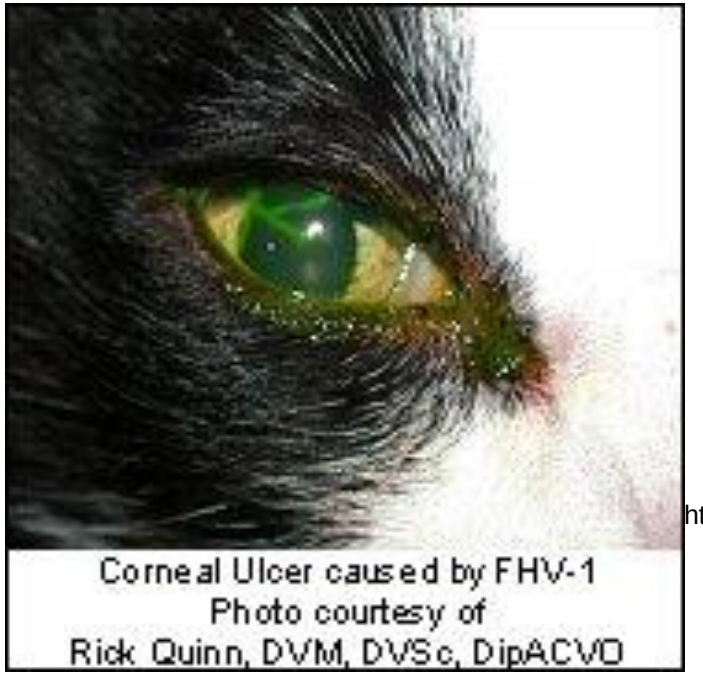
Transmission

- The most common way for the herpesvirus to spread is through contact with discharge from an infected cat's eyes, mouth or nose.
- Cats can contract this virus by sharing litter boxes, food and water bowls and mutual grooming with an infected cat.
- An infected pregnant cat might also pass the virus on to kittens who are still in the uterus. Because the virus is highly contagious, it is common in catteries, shelters and multi-cat households.
- Some cats who become infected with feline herpes are latent carriers.
  - Even though they will never display symptoms, they can still pass the virus on to other cats. Stress can cause these carriers to "shed" the virus, exhibiting mild symptoms, which clear up on their own after a few days.

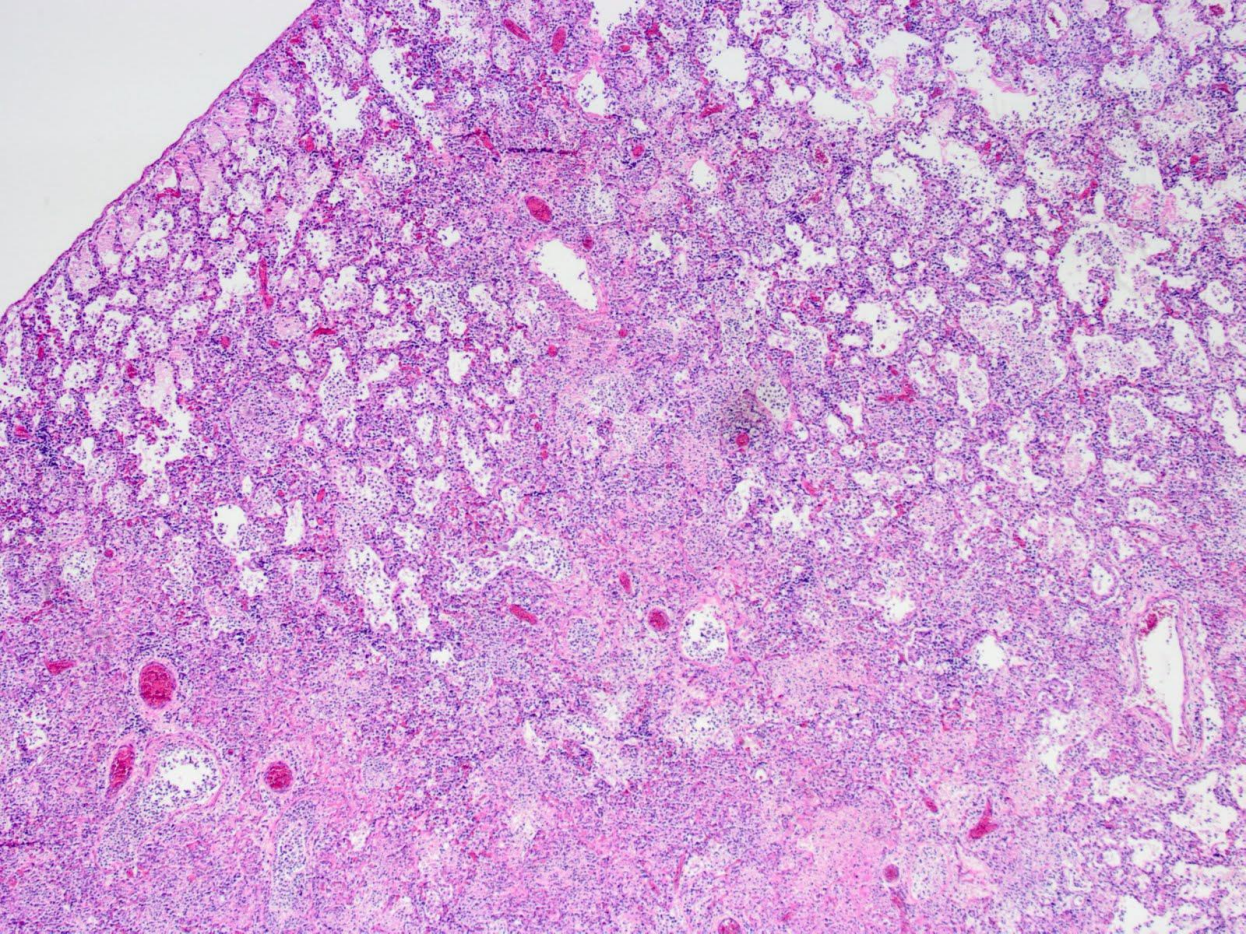
Clinical Signs

- Some infected cats can remain without symptoms, yet act as carriers and spread the infection to other non-infected cats. The following symptoms may also be sporadic in a FHV-1 carrier:
- Sudden, uncontrollable attacks of sneezing
- Watery or mucopurulent nasal discharge
- Loss of sense of smell
- Spasm of the eyelid muscle resulting in closure of the eye (blepharospasm)
- Eye discharge
- Inflammation of the conjunctiva of the eye (conjunctivitis)
- Keratitis (inflammation of the cornea causing watery painful eyes and blurred vision)
- Lack of appetite
- Fever
- Loss of pregnancy
- Cats weakened by the virus may also develop secondary infections.

http://homepage.usask.ca/~vim458/virology/studpages2010/felineuri/rhino_transmission.html

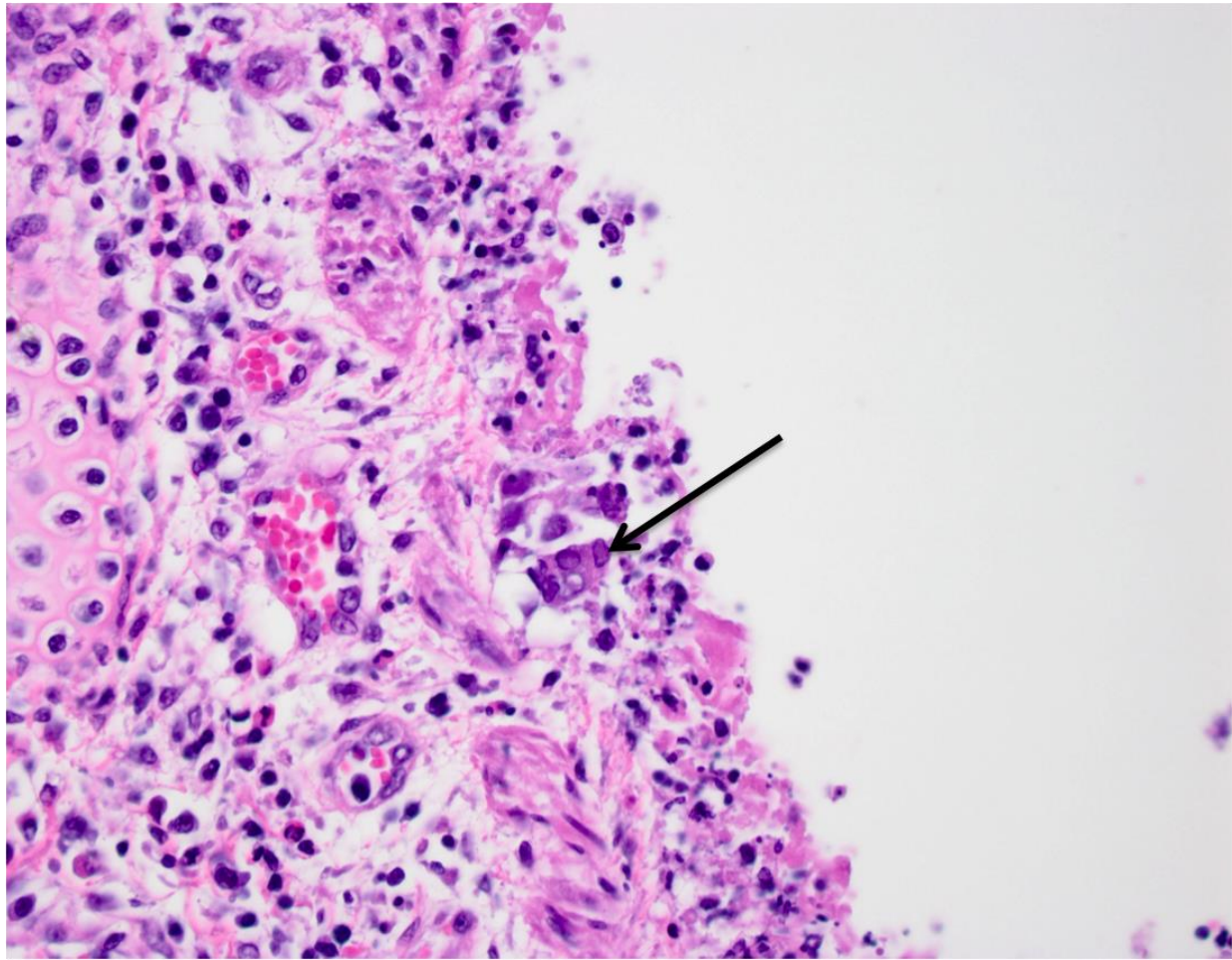


http://www.critterology.com/feline_herpesvirus_1_feline_viral_rhinotracheitis-112.h



The usually clear air spaces in the lung are filled with inflammatory cells & debris

The arrow points to a syncitial cell containing multiple nuclei. These nuclei contain characteristic viral inclusion bodies



http://www.parrymedicalwriting.com/herpesvirus-infection-in-cats/

Diagnosis

- Virus isolation
- PCR
- Virus neutralization
- IF
- Samples taken from the conjunctiva of the eye are stained to detect the intranuclear *inclusion* bodies

Differantial Diagnosis

Table 1: Summary of clinical signs produced in cats during primary infections by three common pathogens

Clinical Signs	FHV-1	FCV	Chlamydia
Malaise/Anorexia	+++	++	+/-
Sneezing	+++	+	++
Nasal Discharge	+++	++	++
Oral Ulceration	-	+++	-
Ptyalism	+	+++	+/-
Ocular Discharge	+++	+	++
Conjunctivitis	+++ (Hyperemic)	-	+++ (Chemotic)
Keratitis	+++	-	-

Prevention

- Vaccination!
- The standard 'core' vaccines that are given to cats include a vaccine against feline viral rhinotracheitis.
- The FVR vaccine will not completely prevent an infection from occurring if your cat is exposed to the virus, but it will significantly reduce the severity of the infection and will shorten the length of the illness.
- Vaccine guidelines recommend the initial kitten series of two or more vaccinations when kittens are 6 to 16 weeks of age, a single booster vaccine one year later, then revaccination every three years thereafter.

- Preventing direct contact between your cat and other cats will greatly minimize the of your cat becoming infected, while following good sanitation and hygiene practices, such as
  - washing your hands thoroughly before and after petting another cat will further reduce the likelihood of disease spread between cats.
- If your cat has had an FVR infection, you should keep the cat indoors to prevent spread of this infection to other cats in your neighborhood.

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AKABANE DISEASE

Congenital Arthrogryposis – Hydranencephaly Syndrome, A–H Syndrome, Akabane Disease, Congenital Bovine Epizootic A-H Syndrome, Acorn Calves, Silly Calves, Curly Lamb Disease, Curly Calf Disease, Dummy Calf Disease, Bovine Epizootic Encephalomyelitis

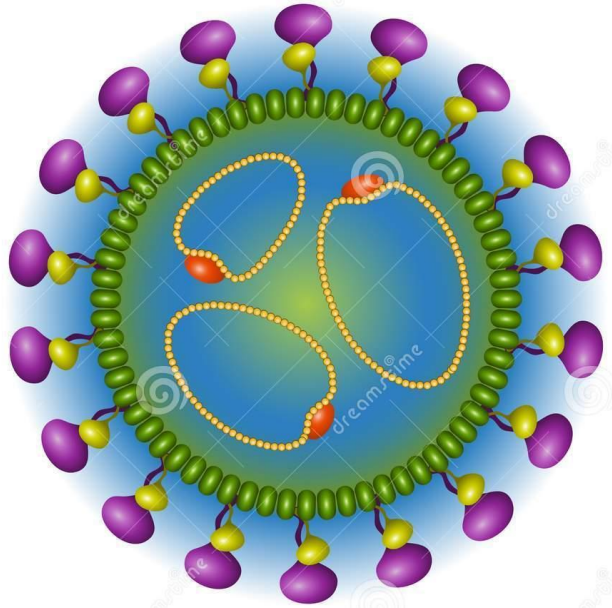
- Akabane is an arthropod-borne viral disease that can affect cattle, sheep and goats. In the predominant syndrome, inapparent infections in adults can lead months later to abortions, stillbirths and congenital defects in newborns.

Etiology

- Bunyaviridae → genus Orthobunyavirus → Akabane virus (arbovirus)

single-stranded RNA
enveloped

causes intrauterine infection of the fetus in pregnant cattle, sheep and goats by invading the endothelial cells of the placenta, replicating in the trophoblastic cells and finally in the fetus itself.
The result is abortions, stillbirths, premature births and deformities of the fetus or newborn with no clinical signs in the dam during pregnancy.



BunyaVirus

- Akabane is named after the Japanese village where the virus was first isolated from mosquitoes (*Aedes vexans, Culex tritaeniorhychus*) in 1959.

- Clinical cases have been seen only in cattle, sheep and goats.
- Antibodies to Akabane virus have been found in horses, donkeys, buffalo, deer, camels, pigs and wild boar, but there has been no indication that the virus is pathogenic in these species.
- Serological evidence of infection has also been reported in wild ruminants and other ungulates.
- Our livestock population is highly susceptible to the virus.
- Based on the severe mortality that can occur with Akabane disease in naïve populations, the economic impact of the disease can be great.
- Additionally, the potential vectors for the disease are found in the Türkiye.

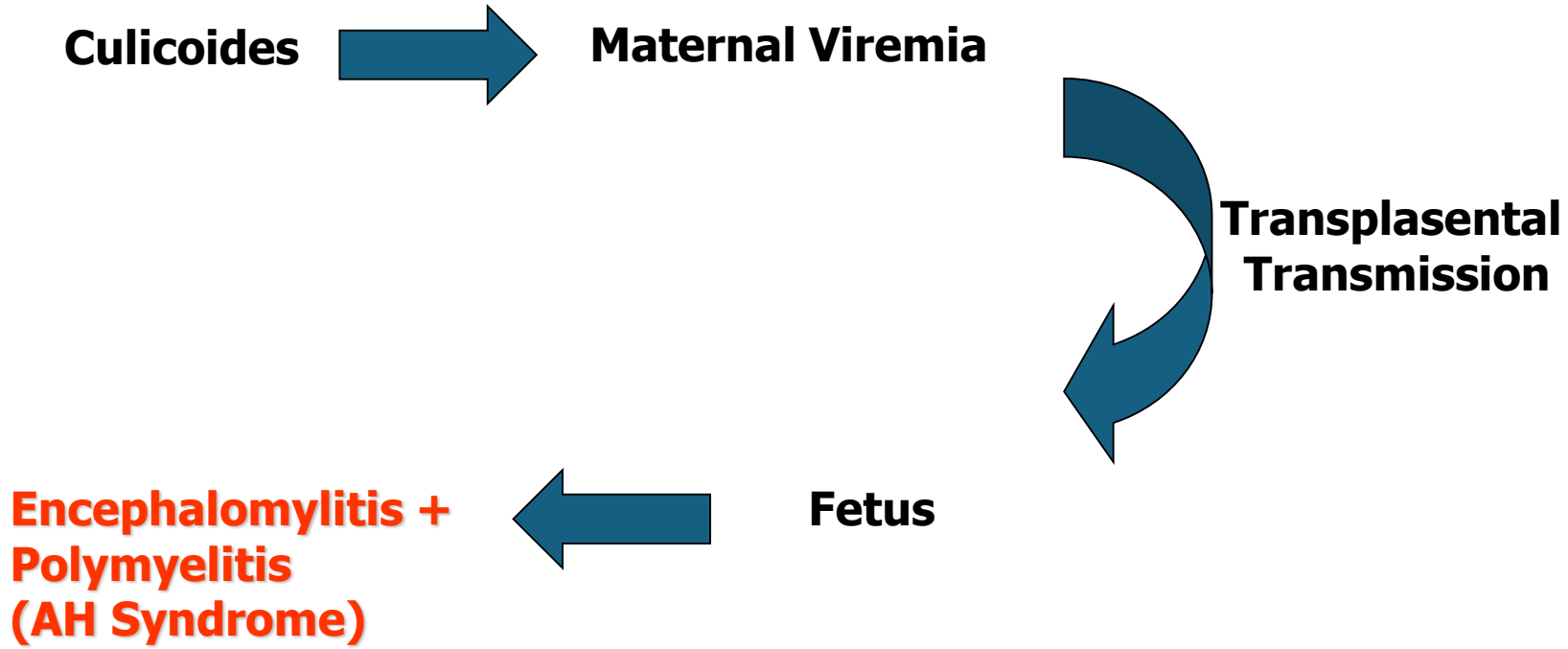
Geographic Distribution

- Akabane virus is common in many tropical and subtropical areas in the Eastern Hemisphere, including parts of Asia, Africa, the Middle East and Australia. However, outbreaks tend to occur mainly in the far northern and far southern ranges of its distribution.
- Some countries that have reported Akabane disease include Japan, Korea, Taiwan, Australia, Israel and Türkiye.

Transmission

- Akabane virus is primarily transmitted by biting midges in the genus Culicoides.
- Vertical transmission is important in the epidemiology of this disease.
- In animals that are not immune to Akabane virus, it is transmitted across the placenta to the fetus, and causes congenital defects.
- This virus does not appear to be contagious by casual contact; horizontal transmission has only been reported via insect vectors.
- It has not been found in the semen of bulls.
- Ruminants do not become long-term carriers of Akabane virus.

Pathogenesis and Pathology



Clinical Signs

- Akabane virus infections are asymptomatic in most adult animals, but viremia usually occurs 1 to 6 days after infection.
- Akabane virus is transmitted across the placenta to the fetus.
- Fetal infections do not become evident until the animal is either born or aborted due to severe defects.

- Most strains of Akabane virus infect non-pregnant animals subclinically, but a few can cause encephalomyelitis in calves and adult cattle.
- Neurological signs that have been reported in these animals include tremors, ataxia, lameness, paralysis, nystagmus, opisthotonos and hypersensitivity.
- More often, Akabane disease is characterized by asymptomatic infections in postnatal animals, and abortions, stillbirths, premature births and congenital defects (arthrogryposis and congenital malformations of the brain) in fetuses and newborns.
- Birth complications may cause injuries to the dam that result in infertility or death, particularly when the fetus has malformed joints.

- The effects of AKAV infection of a fetus depend on when during gestation the infection occurs; this is more evident in cattle, due to their longer gestation periods:

➢Congenital lesions in the brain may be present in calves born to cows infected early in pregnancy.

➢These calves exhibit behavioral abnormalities even though their motor faculties seem normal. Neurological signs may occur, and calves may be blind, deaf, depressed, or unaware of surroundings; they may have difficulty suckling and generally die soon after birth or must be euthanized.

➢Infection in the second trimester of pregnancy is likely to result in a calf born with arthrogryposis, or rigid joints with atrophied muscles.

- Fetuses infected at a later stage tend to be aborted, stillborn, or premature; often signs of disease are not noticeable until examination of the cranial cavity reveals hydranencephaly.
- Newborn calves may be alert but typically cannot stand, and they are likely to be uncoordinated, partially or fully paralyzed, have bulging or tearing eyes, and make unusual vocalizations.

Bovine calf, calvarium. The cerebral hemispheres consist of thin-walled sacs that contained cerebralspinal fluid prior to necropsy

Post Mortem

- Arthrogryposis and hydranencephaly are the most commonly noted lesions.
- Most of the affected joints are ankylosed and cannot be straightened even by force. [Arthrogryposis (persistent flexion of jointsis the most frequently observed lesion.
- Hydranencephaly (absence of the cerebral hemispheres and their normal site being occupied by cerebrospinal fluid) can also be seen with Akabane infection.

Bovine neonate. This live calf cannot stand due to severe arthrogryposis, primarily affecting the hindlimbs.

Bovine neonate (Aino). This stillborn calf exhibits torticollis and arthrogryposis.

Bovine neonate, brain. The entire brain is reduced in size (microencephaly), and surrounded by cerebrospinal fluid.

Bovine calf. The head is hyperextended. The limb joints are fixed and vary from hypercontracted to hyperextended.

Bovine calf. The head is rotated and tilted to the side (torticollis). There is abnormal rotation of the thoracic limbs and the joints are fixed at unusual angles (arthrogryposis). The thoracolumbar spine is curved to the right (kyphosis).

The cerebral hemispheres have failed to develop.

- Central nervous system lesions may include hydrocephalus, agenesis of the brain, microencephaly, proencephaly, cerebellar cavitation.
- Other abnormalities may include fibrinous leptomeningitis or ependymitis, spinal cord agenesis or hypoplasia, torticollis, scoliosis, brachygnathism, cataracts, ophthalmia [severe inflammation of the eye], hypoplastic skeletal muscles and lungs, fibrinous polyarticular synovitis.

Diagnosis

- Clinically
  - Fetal arthrogryposis ve hydranencephaly (AH Syndrome)
- Congenital Akabane disease is often diagnosed by serology, using serum samples or body fluids (pericardial, pleural or peritoneal) from the fetus or presuckle neonate.
- Most affected fetuses and calves have mounted an antibody response to this virus, although fetuses that were infected before they became immunocompetent may be seronegative.
- Virus neutralization and ELISAs are the most commonly used serological tests.
- Other assays that have been employed include agar gel immunodiffusion, hemagglutination inhibition and hemolysis inhibition assays.

- Antigenic tests can also be useful sometimes in fresh fetuses aborted before they have developed an immune response.
- Samples should be taken from the placenta and fetal skeletal muscle, brain and spinal cord.
- RT-PCR, qRT-PCR
- Akabane virus can be isolated in a number of cell lines (e.g., Vero, HmLu-1, BHK-21) cells), and the virus can be identified by immunofluorescent or immunohistochemical straining or virus neutralization.

Differential Diagnosis

- Schmallenberg virus;
- disease caused by Aino or Cache Valley (Chuzan) viruses;
- bovine viral diarrhea virus;
- border disease;
- Wesselsbron disease;
- bluetongue (in sheep); and
- other nutritional, genetic, and toxic diseases.

Prevention and Control

- Akabane virus does not appear to be transmitted between animals except by arthropods.
- If this virus is introduced into an area where it is not endemic, care should be taken to prevent it from infecting potential vectors, especially Culicoides spp.
- Vaccines are available in some countries, and can prevent fetal losses.
- Akabane disease can also be controlled in pregnant animals by moving them into an endemic area in time to develop immunity before they are first bred.
- Seropositive animals have no problem with the pregnancy.

References

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