Network for wildlife health surveillance in Europe
Diagnosis Card

Marine Morbilliviruses

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Etiology

Cetacean morbillivirus (CeMV) and *Phocine distemper virus* (PDV) are the two known marine species of the genus *Morbillivirus*. The terrestrial Canine Distemper Virus (CDV) may also infect seals. Morbilliviruses are single stranded negative sense RNA viruses of the family of *paramyxoviridae*.

Affected species (wildlife, domestic animals, humans)

PDV affects mainly seals. North Atlantic harbor seals (*Phoca vitulina*) are the most susceptible species. Two large scale epidemics decimated the North-East Atlantic population of harbor seals in 1988 and 2002. Gray seals (*Halichoerus grypus*), harp seals (*Pagophilus groenlandicus*) and hooded seals (*Cystophora cristata*) are less susceptible. There is no evidence that walruses (*Odobenus rosmarus*), polar bears (*Ursus maritimus*) or eared seals (Otariidae) are susceptible to disease despite serological evidence of exposure.

CeMV has been documented in many odontocete species, two mysticete species and one seal species. Multiple strains of CeMV have been identified. Mass mortalities have been reported in populations of bottlenose dolphins (*Tursiops truncatus* and *Tursiops aduncus*), common dolphins (*Delphinus delphis*), long finned pilot whales (*Globicephala melas*) and striped dolphins (*Stenella coeruleoalba*). Sporadic mortalities due to morbillivirus infection have occurred in harbor porpoises (*Phocoena phocoena*), and pygmy sperm whales (*Kogia breviceps*). CeMV is considered to be endemic in bottlenose dolphins, pilot whales, dusky dolphins (*Lagenorhynchus obscurus*), long beaked common dolphins *(Delphinus capensis*), Fraser's dolphins (*Lagenorhynchus hosei*) and melon headed whales (*Pepenocephala electra).* CeMV infection has been documented in the mysticetes: minke whales (*Baleanoptera acutorostrata*) and fin whales (*B. physalus*). Monk seals (*Monachus monachus*) have suffered a mass mortality due to a *Cetacean morbillivirus* outbreak in Western Africa. Caspian seals (*Pusa caspica*) have suffered mass mortalities due to the terrestrial *Canine distemper virus*. The latter two incidents underscore the potential of morbillivirus to cross the species barrier.

Domestic animals, humans and terrestrial wildlife have not been infected with marine morbilliviruses.

Epidemiological characteristics and disease course

Marine morbilliviruses are highly immunogenic viruses, which are mainly transmitted horizontally by inhalation of virus particles present in expired aerosols from infected animals. The infectious period is short as animals either quickly succumb to an infection or mount an immune response which clears the virus from their body. A large population is therefore necessary for marine morbilliviruses to persist endemically. These viruses are also able to persist in small (possibly multi-host species) metapopulations in a manner which has not been clarified yet. In immunologically naive populations mass mortalities may occur upon introduction of morbillivirus. Morbilliviruses initially target cells of the immune system and subsequently infect endothelial and epithelial cells of various organ systems and the central nervous system. Infection is either acutely fatal, in case that the host is unable to mount an adequate immune response, uneventful if the host does mount an adequate immune response, or sub-acutely fatal if the host succumbs to secondary infections due to morbillivirus-induced immune suppression. CeMV sporadically causes fatal chronic central nervous system infections.

Clinical signs

Susceptibility and thus clinical signs depend on host species, morbillivirus strain and immune status of the population. In harbour seals infection may result in systemic clinical signs (depression, lethargy, fever), ocular signs (abnormal discharge, redness of conjunctiva and sclera, corneal opacities), respiratory signs (mucopurulent nasal discharge, coughing, tachypnoea, dyspnoea), digestive signs (diarrhoea), reproductive failure (abortion) and skin lesions (dermatitis). Little information is available on

clinical signs in cetaceans. Most cetaceans are observed moribund or stranded dead. Striped dolphins can show systemic clinical signs (poor body condition, subcutaneous oedema), nervous signs (disorientation, muscle tremors, passive behaviour), respiratory signs (abnormal respiratory rate) and digestive signs (ulcers in the oral mucosa). Common dolphins can exhibit seizures, uncontrolled trembling and dyspnoea.

Gross lesions

Gross lesions may be due to the direct action of the virus or to secondary infections, and are mostly restricted to the lungs, and occasionally, the brain. Morbilliviral gross lesions are multifocal pulmonary consolidation or atelectasis, and pulmonary emphysema. Lymph nodes may be grossly enlarged and oedematous, and there may be subcutaneous emphysema. Toxoplasmosis and mycosis are frequent secondary infections, especially in cetaceans, causing granulomatous-necrotizing (toxoplasmosis) or haemorrhagic necrotizing (mycosis) inflammatory changes, frequently in lymph nodes, brain and lungs.

Histological lesions

Histologic lesions are mainly observed in lymphoid organs, brain and lungs. Lymphoid organs often have lymphoid depletion and lymphocytic necrosis, with syncytial cells. Multifocal broncho-interstitial pneumonia is the main lesion of the respiratory tract. Syncytia in alveolar and bronchial lumina occur more or less prominent depending upon host species. Intracytoplasmic (ICIB) and intranuclear (INIB) inclusion bodies are observed in syncytia and macrophages, again its prominence depending on host species. In the brain, mainly the cerebrum, non-suppurative encephalitis is observed. Characteristic changes in the grey matter are neuronal degeneration and necrosis, gliosis and perivascular cuffing. Acidophilic or amphophilic viral inclusions ICIB and or INIB can be present in many tissues.

Differential diagnosis

Pneumonia and encephalitis (the two main lesions of morbillivirus infection) can be caused by many other infectious agents, like viruses (herpesviruses), bacteria (Brucella ceti), protozoa (*Toxoplasma goindii*, *Sarcocystis* sp., and *Neospora* sp., fungi (*Aspergillus* sp., *Mucor* sp.) and macroparasites. The infection by some of these organisms might be exacerbated or facilitated by morbillivirus infection due to the immunosuppression caused by morbillivirus infection. The proximate cause of death might well be any of these secondary infections while the ultimate cause of death is the morbillivirus infection.

Criteria for diagnosis

Broncho-interstitial pneumonia, non-suppurative encephalitis, and lymphoid depletion, with nuclear and cytoplasmic viral inclusions (INIB and ICIB) and syncytial cells, are strongly suggestive of morbillivirus infection. Diagnosis should be confirmed by immunohistochemistry (IHC) on target tissues, and by RT-PCR, especially in a new host species or new geographic areas of morbillivirus infection.

Serology is useful for monitoring morbillivirus infection at population level or in live animals. Indirect enzyme linked immunosorbent assays (iELISA) can be used on haemolyzed serum samples or on serum samples of deceased animals. Antigen coating with the homologous strain is preferred. Antigen of heterologous strains is less sensitive, and its use may therefore increase the amount of false negative results.

Recommended diagnostic method(s) and preferred samples (incl. recommended amount and appropriate storage)

Dead animals: small amounts of tissue (1 cm^3) of brain (cerebrum), lung, tracheobronchial lymph node, spleen, liver, kidney and urinary bladder in 10% neutral buffered formalin for histology and IHC. Pea sized samples of same organs in RNAlater® for RT-PCR. Sample to volume formalin or RNAlater® volume 1:10. Storage: formalin at room temperature, RNAlater® samples should be left at room temperature overnight and may then be stored at minus 20 ºC for indefinite storage after discarding the supernatant.

Live animals: nasal and pharyngeal swabs in RNAlater® for RT-PCR. Serum samples (1-2 ml) for iELISA. Whole blood should be centrifuged between 30 minutes to two hours after sampling when stored at room temperature or within 24 hours if stored at 4 to 8 ºC. Serum can be stored up to a week at 4 to 8 ºC. For longer periods serum should be stored at -20 ºC.

EWDA proposed harmonized protocol (for harmonization at large scale)

RT-PCR on tissue samples of dead animals and pharyngeal and swabs of live animals. Primers as described by Barrett et al., 1993, followed by sequencing to determine the strain of morbillivirus involved. For dead animals, histopathology and IHC (only freshly dead animals) of target organs (lung, respiratory lymph nodes, brain). For prevalence monitoring, iELISA on serum samples is recommended.

Laboratories that can be contacted for diagnostic support

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Recommended literature

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