Network for wildlife health surveillance in Europe

EWDA Diagnosis Card

Encephalomyocarditis

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Etiology

Encephalomyocarditis virus (EMCV) is a member of the genus *Cardiovirus* of the family Picornaviridae. Two serotypes are known: EMCV-1 and EMCV-2, which can be distinguished by serological and molecular techniques. EMCV has a worldwide distribution. It is stable at pH 3-9, and relatively stable up to pH 13.

Affected species (wildlife, domestic animals, humans)

EMCV has a very broad host range, with naturally acquired infection reported in humans, non-human primates, rodents, marsupials, mongoose species, artiodactyls (including cattle and especially domestic pigs), horses, elephants, tapirs, tigers, dogs and cats.
EMCV is recognized as cause of disease mainly in pigs and wild boars, but outbreaks of EMC are reported also in non-human primates and in a variety of domestic, captive, non-domestic and wild animals. Human cases have been luckily very rare and although the infection is possible the risk appears to be almost negligible.

Epidemiological characteristics and disease course

Rodents, especially rats, are considered to be the natural host and reservoir of EMCV in which the virus usually persists without causing disease. In recent studies it was demonstrated that EMCV could spread easily within rat and mice population by horizontal transmission, the virus being isolated from the faeces of both inoculated and contact-infected rodents at 2 to 29 dpi. Faecal contamination of feed or water, and ingestion of infected rodents' carcasses are the major routes of spread and transmission to susceptible animals in pig farms, zoo, and wild species. Wild boar also represents a potential reservoir of the infection. Horizontal pig-to-pig transmission through virus excretion by acutely infected pigs for 1–4 days was recognised as another infection route. The virus can also be transiently excreted from persistently infected pigs, after reactivation of the infection. Transplacental infection has also been described as a potential route of virus spread. Other factors, such as infectious dose, route of infection, and age of pigs, have been found to be important in the spread of the virus under experimental conditions.

Clinical signs

The clinical signs in animals depends upon both viral and host factors. The strains of EMCV vary in virulence and/or tissue tropism. In the natural hosts, wild rodents, the virus usually persists without causing disease. Certain virus strains cause predominantly fatal encephalitis, widespread myocardial damage, or specific destruction of pancreatic β-cells. In pigs and likely in wild boars, the infection has been recognized either as a cause of mortality in youngs due to acute myocarditis, or reproductive failure in sows. In non-human primates a sudden mortality with mild, not specific symptoms was observed in most cases.

Gross lesions

Not all animals dying from the acute phase of cardiac failure show gross lesion. The lesions vary in severity and consist of multiple white-grey linear or circular areas, which are visible in the wall of the ventricles, especially those of the right ventricle. The results of cardiac dysfunction, as hydropericardium, hydrothorax and pulmonary oedema, are frequently observed.

Histological lesions

Heart: Myocarditis with focal or diffuse accumulation of mononuclear cells, vascular congestion, edema, degeneration of the myocardial fibers with necrosis and occasional mineralization of necrotic heart muscle.

Central Nervous System: Brain tissue can be congested with evidence of meningitis, perivascular infiltration (mononuclear cells) and neuronal degeneration.
Non-suppurative encephalitis and myocarditis has also been seen in naturally infected swine foetuses.

Differential diagnosis

Any infectious or non-infectious diseases that produce sudden death and/or cardiac lesions, as anthrax (waterfowl and elephants), capture myopathy, vitamin E - selenium deficiency.
EMCV-induced reproductive problems in swine, should be differentiated from porcine reproductive and respiratory syndrome, Aujeszky's disease, and porcine parvovirus.

Criteria for diagnosis

The confirmative diagnosis is based on the isolation/identification of the EMCV.

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

Identification of the agent

Virus culture & identification: in acute cases, the identification of the virus in tissues, usually myocardium or spleen, of the affected animals is commonly used. BHK-21 cell culture is the most sensitive method for virus isolation (rapid and complete cytopathic effect). The diagnosis is confirmed by inhibition of infectivity by antisera specific for EMCV or hemagglutination test.
Immunohistochemistry: in more chronic cases, isolation of the virus is not always possible. In these cases, immunohistochemistry (also using monoclonal antibodies) may be helpful to demonstrate viral antigen in the affected tissues.
Molecular Biology: RT-PCR methods may be applied for direct identification of EMCV in target organs (heart, spleen, liver, and lung).
Serology*:* In live animals, the commonly used serological test is virus-neutralization test. Enzyme-linked immunosorbent assay (ELISA) may also be used, if available. VN-antibody production starts as soon as 3 days post infection and may persist for an estimated period of 6 months. Haemagglutination inhibition test may be used but it shows differences among EMCV strains.

EWDA proposed protocol (for harmonization at large scale)

Direct diagnosis: PCR is the universal method able to ascertain viral positivity in short time with high sensitivity.
Indirect diagnosis: The virus-neutralization test is the commonly used serological test but for investigation at population level ELISA is likely the most suitable method. However, serology should not be used alone, as EMCV antibodies have been detected in areas with no clinical disease, presumably due to non-pathogenic EMCV strains.

Laboratories that can be contacted for diagnostic support

- Sciensano, Dr. Kris De Clercq, kris.declercq@sciensano.be
- Institute for Animal Health (IAH), Dr. David Paton, david.paton@iah.ac.uk
- Wageningen Bioveterinary Research, Dr. Aldo Dekker aldo.dekker@wur.nl
- Istituto Zooprofilattico Sperimentale della Lombardia e dell´Emilia-Romagna (IZSLER), Dr. Santina Grazioli, santina.grazioli@izsler.it
- Friedrich Loeffler Institut – Bundesforschungsinstitut fuer Tiergesundheit (FLI), Dr. Dr. Martin Beer, martin.beer@fli.bund.de
- Laboratory of Microbiology & Parasitology, Faculty of Veterinary Medicine, University of Thessaly, Karditsa, Greece, Dr. Charalambos Billinis, billinis@vet.uth.gr

Recommended literature

1. KOENEN, F, PAUL, E., GIBBS, J., RUIZ-FONS, F., & BARLOW A.M. (2012). Picornavirus Infections. 168–180. “Infectious Diseases of Wild Mammals and Birds in Europe.” Wiley-Blackwell (Eds.).
2. CANELLI, E., LUPPI, A., LAVAZZA, A., LELLI, D., SOZZI, E., MARTIN, A. M., GELMETTI, D., PASCOTTO, E., SANDRI, C., MAGNONE, W., & CORDIOLI, P. (2010). Encephalomyocarditis virus infection in an Italian zoo. *Virol J*, *7*(6), 4.
3. BILLINIS C. (2009). Encephalomyocarditis Virus Infection in Wildlife Species in Greece. Journal of Wildlife Diseases, 45(2), 2009, pp. 522–526
4. MAURICE, H., NIELEN, M., BROCCHI, E., NOWOTNY, N., BAKKALI KASSIMI, L., BILLINIS, C., LOUKAIDES, P., O'HARA, R. S. & KOENEN, F. (2005). The occurrence of encephalomyocarditis virus (EMCV) in European pigs from 1990 to 2001. Epidemiology and infection, 133(3), 547-558.
5. GELMETTI, D., MERONI, A., BROCCHI, E., KOENEN, F., & CAMMARATA, G. (2005). Pathogenesis of encephalomyocarditis experimental infection in young piglets: a potential animal model to study viral myocarditis. Veterinary research, 37(1), 15-23.
6. SPYROU V, MAURICE H, BILLINIS C, PAPANASTASSOPOULOU M, PSALLA D, NIELEN M, KOENEN F, PAPADOPOULOS O. (2004). Transmission and pathogenicity of encephalomyocarditis virus (EMCV) among rats. Vet Res. 35(1):113-22.