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Encephalomyocarditis

The virus that causes Encephalomyocarditis (EMC) belongs to the genus Cardiovirus (family Picornoviridae). The virus replicates in myocardial cells, causing cell necrosis, frequently resulting in a fatal myocarditis. EMC is a natural infection in rodents, that are considered to be the reservoir host, without producing clinical disease. Identical EMCV strains were found in a captive African elephant and 3 randomly caught free living rats in the same French zoo (Romey, 2021).
Sporadic outbreaks occur in a variety of domestic and wild animals, including elephants living 2006; Canelli, 2010). Both African and Asian elephants have developed EMC in South Africa, Australia, the United States, and Europe (Seaman, 1987; Braack, 1995; Lamglait, 2015) .
Infection most likely occurs via the oral-fecal route from feed or water contaminated with rodent urine or feces. An outbreak of EMC in free-ranging African elephants in Kruger National Park in South Africa was correlated with a population explosion of a particular mouse species (*Mastomys natalensis*), which had a high prevalence of antibodies to EMC virus. The feeding behavior of elephants—i.e., pulling up and ingesting tufts of grass containing rodent nests—may have contributed to infection of the elephants (Grobler, 1995). A total of 64 elephants died during this outbreak. 53 of 64 (83%) bulls died of cardiac failure. Postmortem lesions included hydrothorax, hydropericardium, severe ascites (up to 50 liters), and petechial and ecchymotic hemorrhages on the epicardium. Two genetically distinct lineages of ECMV were found in wild animals in Africa south of the Sahara (van Sandwyk, 2013).
There is no evidence that the infection spreads between elephants.

Clinical signs in elephants

Sudden death is the predominant sign or, in less acute cases, anorexia, lethargy, and moderate-to-severe dyspnea. These are signs associated with congestive heart failure. Myocarditis is the principle effect in elephants, resulting in pulmonary edema, hydropericardium, and ascites (Gaskin, 1980; Seaman, 1987; Simpson, 1972). However, not all infections in elephants result in clinical disease. In a study in Kruger National Park, 53% of the animals had antibody titers to EMC virus (Grobler, 1995). There appears to be a sex bias toward clinical infection in males. It is known that testosterone enhances susceptibility to EMC infection in mice (Friedman, 1972). Recovery from infection may leave myocardial scars, which may cause problems later if an elephant is immobilized. The stress of the immobilization may place an additional burden on a weakened myocardium, making it susceptible to ventricular dysrhythmia caused by catecholamine release following administration of an immobilizing agent (Hattingh, 1994).

Diagnosis

The infection progresses so rapidly that there may be little time to obtain samples for antemortem screening. Serologic tests may be used to survey other members of a herd. Sudden death or signs associated with heart failure plus myocarditis at necropsy provide a presumptive diagnosis. A definitive diagnosis is made in acute cases by isolation of the EMC virus in tissue culture cells. Confirmation is by inhibition of infectivity or hemagglutination by antisera specific for EMC virus (Thomson, 2001). Conventional PCR, reverse-transcription

real-time PCR (RT-rtPCR) and genome sequencing are used for genetical identification (Yuan, 2014; Romey, 2021). Gross lesions observed at necropsy include pale streaks in the myocardium, hydrothorax, and hydropericardium with fibrin in the fluids. Pulmonary edema with froth in the tracheobronchial tree may be seen. This frothy trunk syndrome is seen associated with immobilization following recovery from EMC. Microscopically, myocardial degeneration and necrosis predominate with lymphocytic infiltrates. Virus particles may be visible on electron microscopy. Differential diagnosis. Any disease that causes myocardial necrosis must be considered, including endotheliotropic herpesvirus infection, hypovitaminosis E, and cardioactive glycoside plant poisoning (oleander Nerium oleander).

Treatment and prevention

There is no specific treatment for EMC. Prevention by rodent control is crucial. Rodent population monitoring would reveal a population buildup, enabling initiation of more rigorous control methods. During an outbreak in Kruger National Park, elephants were vaccinated with an in-house–developed aziridine-inactivated vaccine in an oil adjuvant (Hunter, 1998). In clinical trials, vaccinated elephants withstood a potentially lethal challenge with EMC virus (Raath, 1995). Inactivated vaccines were used in outbreaks in the United States with no conclusions reached as to effectiveness in elephants (Backues, 1997; Thomson, 2001).

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