PORCINE ENCEPHALOMYOCARDITIS

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The pathogenicity of encephalomyocarditis virus (EMC) for domestic animals was first recognized in 1958 with the recovery of EMC virus from swine during an outbreak of an acutely fatal disease on a farm in Panama. Large outbreaks have repeatedly occurred in swine again in Panama and in Florida, New South Wales, Australia, New Zealand and recently in Cuba.

EMC viruses are strains of a single RNA of the family Picornaviradae, genus Enterovirus, species cardiovirus. The geographical area of greatest confirmed virus activity is Florida where the virus has been isolated from animals of eight species, viz., baboon, chimpanzees, raccoon, cotton rats, squirrel swine, calves and African elephants. Swine are most often affected.

In swine, infection with EMC virus causes an acutely fatal disease. Swine of varying age groups in large and small herds on commercial farms have been affected. The disease has not been associated with any specific husbandry or vaccination practices. Animals often die suddenly without apparent signs of disease. Occasionally, an animal may be observed to exhibit a brief period of exitation then collapse and die. There are no consistent or identifying clinical signs. Mortality rates of 10 to 100 per cent have been reported in specific outbreaks. It is generally highest in young pigs. Inapparent infections also occur. The disease in swine herds may extend over a period of two or three weeks. The outstanding lesion in swine dying of the disease is severe myocarditis with myocardial degeneration.

Death is apparently attributable to myocardial failure arising from a heart block or acute arrhythmia.

The dead swine are typically in good flesh and size consistent with farm conditions and feeding practices. At necropsy, hydrothorax, hydropericardium and ascites are frequently observed. Gross myocardial lesions are observed in most cases. The heart is soft and pale. Minute yellowish or white areas suggestive of necrosis are seen. These lesions are most prevalent on the epicardium of the right ventricle and may extend to varying depths within the myocardium. The lungs are usually congested and edematous. The liver may be normal, or slightly congested. Abdominal congestion may be pronounced. The meninges are slightly congested or otherwise normal. Histopathologically, the most significant finding is myocarditis with round cell infiltration, vascular congestion, edema and degeneration of the myocardial fibers with necrosis and calcification. Minimal congestion with mild meningitis and spotty areas of neuronal degeneration are reported in the brain.

The occurrence of an acutely fatal disease of swine with accompanying lesions of myocarditis may provide a preliminary diagnosis of EMC infection. Virus isolation and identification are essential for a conclusive diagnosis. Inapparent infection may be disclosed though examinations for EMC virus neutralizing and/or hemagglutination inhibition antibodies.

EMC infection has been reproduced in swine by oral feeding of the virus and parenteral inoculation. Infection, as evidenced by viremia and myocardial lesions may occur without signs of illness. Early transitory fever, inappetence, listlessness and paralysis are reported occasionally. As in the natural disease, death may occur suddenly without prodromal signs of illness or following a very brief agonal episode. Virus has been recovered from all organs, feces and urine. The heart invariably has a higher titer of virus than any other tissue. Virus passage history, dose and individual animal sensivity undoubtedly influence the course of infection. An interaction between EMC virus and Trichinella spiralis has been demostrated in rats and may also be a factor in disease in swine.

The primary source of EMC infection in swine is uncertain but presumedly animals acquire the infection by consuming feed or water contaminated by rodents with EMC virus or by consuming diseased rodent carcasses. The virus titer in animals is much higher than the concentration in feces and animals feeding on viscera of other animals dying of EMC infection are perhaps more likely to become infected. Infected swine may perpetuate the infection within a herd through excretion of the virus and contamination of their own premises although one experimental attempt to transmit disease to contact pigs under unhygienic conditions was unsuccessful. There is no evidence that swine remain an inapparent shedder of EMC virus.

Aside from early reports of the recovery of EMC virus from wild caught mosquitoes in Africa, EMC virus has also been isolated from mosquitoes in Brazil and the Unites States and from ticks in India. Experimental attempts to transmit EMC infection via mosquitoes have been unsuccessful. Persistent high levels of viremia in animals offer an opportunity for insect vectors to transmit disease. However, epidemiological studies of the natural disease in swine and captive wild animals do not indicate that those outbreaks of disease were vector borne.

Considerable attention has been focused on rats as the primary source of EMC virus infections without conclusively implicating rats in outbreaks of disease in animals or cases of human infection. In at least two outbreaks, the owners were confident that rat control was effective. EMC virus has been recovered from other wild rodents, viz., raccoon, squirrel, mongoose and cotton rat and this suggest other possible sources or links in the transmission chain.

Rodent control should be practiced in all feed storage and preparation areas. The basic rules for confinement at the diseased and exposed animals to the focus of infection should be observed to prevent spread of EMC virus to adjoining or neighboring premises. All feed and water equipment in an affected area should be cleansed. Animals dying of the disease should be disposed of promptly and sanitarily. There is no biological or chemical prophylaxis for prevention or treatment of EMC infection.

Although man is susceptible to EMC virus there have been few clinical cases of EMC infection. One human epidemic is known to have occurred. Workers, in intimate contact with the animal epidemics in Panama, Florida and Australia have not experienced any associated illness nor have limited serological surveys disclosed evidence of infection among these workmen. Infected swine pose a potential source of infection for slaughterhouse workers but there has been no evidence of infection among this occupational group. There is a potential danger to human households from handling and consuming infected or contaminated pork products.

Selected reference: Acland and Littlejohns. EMC virus infection of pigs, in Australia. Austr. Vet.J., 51,409-415,1975; Craighead, et al., Oral infection of swine with EMC virus. J. Infect, Dis., 112,205-212,1963; Gainer. EMC virus infections in Florida, 1960-1966 J. Am. Vet. Med. Assoc. 151,421-425,1969; Luya, et al., Características del Virus EMC aislado de cerdos en Cuba. Cienc. Tec. AGric. Vet. 1, 1-2, 73-90. 1979; Murnane, et al., Fatal disease of swine due to EMC virus, Science 131, 498-499, 1960; Sutherland, et al., An outbreak of EMC in pigs. New Zealand Vet. J., 25,225, 1977; - Tesh and Wallace. Observations on the natural history of EMC virus. Am J. Trop. Med. and Hyg., 27,133-143, 1977; Portions of the chapter, EMC Virus, by Thomas G. Murnane from the Handbook Series in Zoonoses, James H. Steele, Volume II, Section B, Viral Zoonoses are reproduced with permission of the CRC Press, Inc.