The pathogenicity of encephalomyocarditis virus (EMC) for domestic animals was first recognized in 1938 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 Picornaviridae, genus Cardiovirus, species cardiovirus. The geographical area of Greatest confined virus activity in Florida where the virus has been isolated from animals of eight species, viz., baboon, chimpanzee, raccoon, cotton tail, quail, macaque, and African elephants. Swine are most often affected.

In swine, infection with EMC virus causes an acutely fatal disease. Some 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 excitement then collapse and die. There are no consistent or identifying clinical signs. Mortality rates of 10 to 50 percent 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. Sometimes described as being caused by the epidemic 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 mesentery are slightly congested or otherwise normal. Histopathologically, the most significant finding is myocarditis with round cell infiltration, vascular congestion, edema, edema, and degeneration of the myocardial fibers with necrosis and calcification. Minimal congestion with mild meningitis and petechial areas of neuronal degeneration are reported in the brain.

The occurrence of an acutely fatal disease of swine with accompanying lesion 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 through 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 often observed. 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 anorexia, fever, and coma. The heart invariably has a higher titer of virus than any other tissue. Virus passenger history, dose and individual animal sensitivity undoubtedly influence the course of infection. An interaction between EMC virus and Trichinella spiralis has been demonstrated in rats and may also be a factor in disease in swine.

The primary source of EMC infection in swine is uncertain but presumably animals acquire the infection by consuming feed or water contaminated by rodents with EMC virus or by consuming diseased rodents. The virus in animals is not high then the concentration in feed and animals feeding on vlees of other animals dying of EMC infection are perhaps more likely to become infected. Inapparent swine may perpetuate the infection within a herd through excretion of the virus and contamination of their own premises although some experimental attempts to transmit disease to contact pigs under hygienic conditions has not succeeded. Nonevidence that swine remain an inapparent reservoir 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 United States. The EMC virus of domestic infection with attempts to transmit EMC infection via mosquitoes have been unsuccessful. Epizootic levels of viremia in animals after experimental infection for infection suggests other transmission in domestic animals. However, epidemiological studies of the natural disease in swine and captive wild animals do not indicate that these sources are an important vector for domestic disease. 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 commercial condition that rat control was effective. EMC virus has been recovered from other wild rodents, viz., raccoon, shrew, mongoose, and cotton rat and this suggest other possible sources of EMC infection in the transmission chain. Rodent control should be practiced in all feed storage and preparation areas. The basic rules for containment of the domestic and exported animals in the United States should be observed to prevent spread of EMC virus to domestic swine. 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 commercial condition that rat control was effective. EMC virus has been recovered from other wild rodents, viz., raccoon, shrew, mongoose, and cotton rat and this suggest other possible sources of EMC infection in the transmission chain. Rodent control should be practiced in all feed storage and preparation areas. The basic rules for containment of the domestic and exported animals in the United States should be observed to prevent spread of EMC virus to domestic swine. 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 commercial condition that rat control was effective. EMC virus has been recovered from other wild rodents, viz., raccoon, shrew, mongoose, and cotton rat and this suggest other possible sources of EMC infection in the transmission chain.