THE TRAGEDY OF ENCEPHALOMYOCARDITIS VIRUS INFECTION IN ZOOLOGICAL PARKS OF FLORIDA

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Authors' Note: This report reflects the cooperative efforts of many people. Dr. Schobert first encountered the problem and submitted tissue specimens to Dr. Lewis. Dr. Lewis made all the virus recoveries from BG and LC animals. Dr. Jorge recovered the virus from the JZ elephant. Dr. Simpson, Dr. Lewis and Dr. Gaskin collaborated in identifying the virus as encephalomyocarditis virus. Dr. Schobert, Dr. Woolenman, Ms. Marlowe, and Dr. Curtis dealt directly with the diseased animals and freely contributed case histories and necropsy findings.

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Encephalomyocarditis virus (EMCV), an enterovirus in the family *Picornaviridae*, was first isolated from cotton rats (*Sigmodon hispidus*) in 1940.(9,23) Since then it has been recovered from a variety of avian and mammalian sources, from 3 genera of mosquitoes (*Aedes, culex,* and *Mansonia*, spp.) and 2 genera of ticks (*Ixodes* and *Haemophysalis* ssp.).(18)

The virus occurs worldwide.(17) Many reports of isolations in the USA originated in Florida (Table 1) and the first documentations of EMCV infections in chimpanzees,(7) baboons,(10) raccoons,(5) and African elephants(16) were from Florida. The first reported North American outbreak of fatal porcine EMCV-induced myocarditis also occurred in Florida.(4) Although Florida may have an unusually high incidence of EMCV infection, the frequency of documented infection may merely reflect increased surveillance.

This report describes the devastating occurrence of EMCV infection at 3 separate zoological parks in Florida. It is presented to alert zoological personnel that EMCV may cause mortality in elephants, non-human primates, and other exotic species. EMCV infection was not confirmed by virus recovery in all instances, but the timing of deaths as well as the clinical, necropsy, and histological findings give circumstantial evidence of its role.

I. Busch Gardens (Tampa, Florida)

In the morning of August 25, 1974, a fifteen-year-old female African elephant, which had lived at Busch Gardens for 10 years, was restrained to collect blood by aural venipuncture. In the afternoon she became recumbent and died abruptly. At necropsy a ruptured blood vessel was found in the midthoracic area near the aorta. There was hemorrhage in the mediastinum and the pericardial sac. An 18-19 month male calf (220) lbs) was found in the uterus, and all other organs appeared normal except for the presence of nematodes in the bile duct.

On September 3, 1974, a 10-year-old African elephant female, which had been imported as a replacement animal, died peracutely. She was non-gravid. There was a severe pneumonia, but aside from nematodes in the bile duct, no other abnormalities were observed.

On September 20, 1974, a 15-year-old male African elephant had difficulty walking from the display area to the elephant barn. He finally collapsed and died after a 10-minute struggle. Upon necropsy, the liver was noted to be 3 times normal size and ascites was present. The kidneys were pale in the glomerular area and hemorrhagic in the tubular region. The mesenteric and mediastinal lymph nodes were edematous, the lungs were edematous, and the heart, although normal in size, had a mottled myocardium. The gastrointestinal tract was normal but nematodes were present in the bile duct.

On February 2, 1975, a 16-year-old female African elephant (BG-1) died peracutely while under a 24-hour surveillance for calving. Upon necropsy, a near-term male fetus was found and there was a muddy discoloration of the amniotic fluid. The dam's kidneys were pale and the liver was enlarged, with rounded edges and a bronze coloration. The spleen was normal but there was catarrhal enteritis. The most striking lesions were in the heart and lungs. The heart was flaccid, especially the right ventricle. Paleness of the myocardium and petechiation of the ventricular epicardium were present. The lungs were edematous and essentially no functional lung tissue was left. Frozen tissue specimens were submitted for virologic studies.

On August 30, 1975, a 10-year-old Asian elephant died peracutely. Although there was no pneumonia present, the lungs were hemorrhagic. The heart was slightly flaccid and there was evidence of myocarditis. A hemorrhagic ileotyphlitis was observed, and there was hemorrhage in the anterior large intestine. The kidneys were hemorrhagic in the tubular region. A presumptive diagnosis of toxemia or enterotoxemia was made.

On April 19, 1978, a 5-year-old female African elephant (BG-2) was found dead with cyanotic mucous membranes. Upon necropsy, the stomach and intestines were normal and full of food. There was bronchiectasis of all airways in the lungs. The heart was slightly enlarged and very flaccid (especially on the right side) and the myocardium was mottled. The spleen was slightly enlarged and the liver, rounded and edematous. The kidneys were hemorrhagic through the cortices. The thymus and all lymph nodes were enlarged and the mandibular and anterior thoracic nodes were hemorrhagic. Frozen tissue specimens were submitted for virologic studies.

On April 30, 1978, a second 5-year-old female African elephant (BG-3) was found dead. Upon necropsy the lungs were completely edematous and consolidated. The heart was enlarged and flaccid with a mottled myocardium and petechiation of the epicardium. The spleen was enlarged. The liver was also enlarged and had a "parboiled" appearance. There was a quarter-inch-thick layer of subcapsular edema. The hepatic lymph node was enlarged and hemorrhagic, and the mesenteric lymph nodes were edematous. The kidneys were slightly hemorrhagic. Tissue specimens were submitted for virologic studies.

A young Asian elephant, which had been chained between BG-2 and BG-3, remained healthy and still lives at Busch Gardens.

II. Lion Country Safari (West Palm Beach, Florida)

A number of animals died at Lion Country Safari between December 1976 and February 1978. Although various tissue specimens were submitted for bacteriological, histopathological, and toxicological analysis at several different laboratories, specimens were not submitted for virological studies until March of 1977. At that time EMCV was recovered from tissues of an adult female African elephant that had died February 26, 1977. Although a detailed description of the deaths preceding this definitive diagnosis may not be warranted, the species involved and the timing of the deaths are very interesting.

In late August, 1976, a group of 5 adult African elephants were shipped from a zoological park in Georgia to Lion Country Safari. They were separated from the other Lion Country elephants and were chained on an isolated island. In December of 1976 one female was noted to stay recumbent for a prolonged time but quickly arose when approached. Blood was collected but no abnormalities were found. The elephant seemed normal in the succeeding 3 weeks but was found recumbent on the morning of December 21, 1976. She was unable to rise when prodded and had severe dyspnea. Fluid therapy, steroides, antihistamines, calcium, antibiotics and stimulants failed to have any effect and the elephant died approximately 2 hours later. Her body was removed from the island.

The following morning (December 22) the female next to the one which had died was found to be recumbent and dyspneic. Repetition of the same treatment failed to produce any positive result and this elephant died within 2 hours. Necropsies on both elephants revealed only enlargement of the cardiac ventricles and a gelatinous edema of the coronary fat.

A juvenile white African rhinoceros died peracutely with no premonitory signs on December 26, 1976. The lungs were congested and edematous but no other gross lesions were noted.

For economic reasons the management decided to feed the carcasses of the 2 elephants and the rhinoceros to the lions. On December 31, 1976, several lions appeared anorectic and fatigued easily when taken to the shelter of their huts. On January 2, 1977, 3 lions died after arrival at the huts. Subsequently more lions died and the only consistent gross findings were heart and lung lesions. The deaths of lions occurred as listed in Table 2.

Histological studies revealed an acute necrotizing myocarditis with or without pulmonary edema and pneumonia. These lesions were felt to account for the difficult breathing, fatigue, and subsequent death occurring within 12 hours of onset of clinical signs.

On January 4, 1977, an adult male giraffe (2000 lbs.) collapsed only 10 minutes after being observed in a healthy state by the section warden. In spite of extensive supportive therapy, this animal died within 2 hours. No gross lesions were noted and toxicologic, bacteriologic, histopathologic, and virologic studies (National Animal Disease Center, Ames, Iowa) were not revealing.

On January 19, 1977, a third African elephant (from the original group of 5 that had been shipped from Georgia) became recumbent. This animal was mechanically hoisted and given over 100 liters of fluids and electrolytes as well as cortisone, antibiotics, antihistamines and stimulants. Death occurred in the early hours of January 21, 1977. Upon necropsy, petechial hemorrhages were noted on the surface of the heart and there was pulmonary congestion.

On January 28, 1977, a male chimpanzee was found dead on the island on which he was confined. This 12-year-old ape had been at Lion Country for 7 years and had no history of health problems. Upon necropsy, extensive congestion of all organs was observed. Histologically there was evidence of mild myocardial hypertrophy.

The 2 remaining adult female African elephants from the original 5 shipped from Georgia were mixed with 5 other African elephants which had been at Lion Country for some time. The 2 new elephants were introduced on February 1, 1977, and there was little resultant social harassment. One of the 2 had been unthrifty in appearance since December 25, 1976, and had been put on an especially nutritious diet. On February 26, 1977, this female (LC-1) became anorectic and remained separated from the other elephants. She became uncoordinated, then recumbent, and died within 2 hours. Some salivation was observed prior to death, and frothy nasal discharge was present at the end of the trunk. At necropsy, the lungs were edematous with occasional areas discolored by congestion. Large hemorrhages were noted on the epicardial surface and the myocardium was pale in color. The liver was yellow, swollen, and friable. The surface of the spleen had small areas of hemorrhage and the parenchyma was jam-like in consistency. Tissue samples from this elephant were submitted for virologic studies.

A male oryx-addax antelope cross (LC-2), born October 16, 1977, was rejected by his mother and was moved to the Lion Country hospital. The calf had a good appetite and thrived. After 2 weeks he was placed on display in the Pets' Corner. On December 13, 1977, he became anorectic and had a temperature of 105.6°F. After treatment with antibiotics and steroids, the animal resumed eating and had a normal temperature. However, on December 15, 1977, he was found dead. Upon necropsy, pulmonary edema was evident with purple discoloration of lobes and bloody froth in the trachea. The atria of the heart were mottled with white and blue. Parallel linear hemorrhages were observed in the rumen. The kidneys were swollen and white spots were visible on the liver. Histologically, a diffuse subacute myocarditis, and pulmonary edema and congestion were reported. Tissue specimens were submitted for virologic studies.

Two Debrazza monkeys, a male and a female, were housed together and seemed in excellent health. On December 27, 1977, the male was found dead and some 12 hours later the female died. No gross lesions were observed at necropsy. The only histologic changes noted were hepatic congestion and minimal pulmonary edema and congestion. Tissue specimens from the female (LC-3) were submitted for virologic studies.

A 19-year-old female chimpanzee (LC-4) had been acquired by Lion Country in 1968 and was confined on an island. She was found dead on February 15, 1978. Only a reluctancy to rise had been observed earlier that day. With considerable difficulty 4 employees removed her body from the island in spite of interference from other chimpanzees in the well-organized social unit. Paleness of the myocardium, pulmonary edema, and hepatic swelling were noted upon necropsy. Histologically, myocarditis and subcortical renal necrosis were observed. Tissue specimens were submitted for virologic studies.

On February 21, 1978, it was observed that the 3-year-old daughter of LC-4 was very lethargic. She was moved from the island to the hospital. Her body temperature was subnormal (92°F) and she was given supportive medical treatment. She exhibited incoordination, dyspnea, and disorientation, and died the following afternoon. Necropsy findings included petechiation of the respiratory system, paleness of the myocardium, hepatomegaly, and hemorrhage around some of the mesenteric lymph nodes. Tissue specimens (LC-5) were submitted for virologic studies.

A 6-year-old male chimpanzee (LC-6) had been born on an island at Lion Country and was found dead there on February 22, 1978. No clinical signs were noted and he had been in excellent health. The only gross lesions observed at necropsy were pulmonary edema and paleness of the myocardium. Tissue specimens were submitted for virologic studies.

III. Jacksonville Zoo (Jacksonville, Florida)

A 9-year-old Asian elephant (JZ), which had lived at the zoo since February of 1976, was treated for a jaw abscess during 1976 and a severe case of diarrhea in July of 1978. In the morning of September 29, 1978, he began vomiting, and had to be chained to prevent self-mutilation due to head-pressing against the wall of the elephant house. His rectal temperature was 101.5°F. His condition worsened as he showed signs of severe colic with cramping and tenesmus. There was slight diarrhea but continued retching. Fluid, antibiotic, analgesic, and antispasmodic therapy failed to alter the course of the illness and he became recumbent in the early morning of September 30 and died 4 hours later. Necropsy examination revealed that the lungs were slightly congested and there were petechial and ecchymotic hemorrhages on the epicardium. The mucosa in the area of the duodenum and proximal jejunum was greatly inflamed and the colon was distended with gas. The mesenteric lymph nodes were slightly congested, the spleen was congested, and the liver appeared normal. Tissue specimens were submitted for virologic studies.

Results

Table 3 summarizes the temporal relationship of suspected and confirmed cases of EMC at the 3 zoological parks. Table 4 summarizes the recovery of EMC virus from the tissues submitted for virologic study. The identification of the agent as EMC virus has been previously described.(16)

Discussion

Although EMCV is infective for a wide variety of animals and arthropods, only certain species are consistently affected adversely. Swine are quite susceptible with significant mortality having been reported in porcine outbreaks in Panama,(14) Florida, (4) and New South Wales, Australia.(1) Although the virus has been associated with some cases of human neurologic disease (aseptic meningitis, encephalitis, and Guillain-Barre (1) syndrome), the infection is usually not serious in man. (18,23) Since serologic evidence indicates that human EMCV infection is quite common (especially in countries where the level of hygiene and standard of living are low), most human infections are probably asymptomatic and/or remain unrecognized.(17) In contrast non-human primates are rather severely affected.(6) Elephants, especially the African type, may also be affected severely.(16)

A variety of factors influence the pathogenic potential of EMCV. Younger animals are generally more susceptible than older ones. Suckling rats, for example, may die after infection by several routes, but adults may survive even intracerebral challenge.(12) Pregnant mice are more likely to be adversely affected than are males and non-pregnant females.(3) Infected pregnant mice have more extensive heart lesions and produce higher titers of virus in cardiac muscle.(3) Certain strains of EMCV may have different tissue affinities. An M variant causes mainly acute myocarditis in mice, while an E variant is highly neurotropic.(22)

Seasonality definitely seemed to play a role in the EMCV infections described in this report (Table 3). The Busch Gardens' cases occurred between August 25, 1974, and April 30, 1975, with confirmed cases in February and April. The Lion Country cases occurred December 21, 1976, through February 26, 1977, and December 15, 1977, through February 2, 1978, with confirmed cases in December

and February. The Jacksonville case occurred September 30, 1978. Thus, most of the cases occurred in the cooler months of the year. Similarly the porcine cases described by Gainer in 1961 began in October.(4)

Cool weather (which was especially evident during the Lion Country cases) might have limited the availability of food supplies in the wild and prompted wild animal reservoir hosts to seek feed from sources in the zoological parks. In Florida, EMCV has been recovered from cotton rats,(4,5) raccoons, (5) and the blood of an opossum;(13) the virus has also been recovered from squirrels.(20,21) Such animals would have relatively easy access to food supplies within the 3 parks and rodent control in zoos is always a difficult problem. The occurrence in cool weather would tend to discount the possibility of infection via haemophagous arthropods. Arthropod transmission of EMC virus has never been demonstrated.(17)

There is controversy concerning the role of rodents in the dissemination of EMCV. Tesh and Wallace(17) reported that *Rattus rattus* (common large black rats), *Rattus norvegicus* (Norway rat), and *Mus musculus* (common house mouse) are susceptible to oral infection with EMCV but do not excrete significant amounts of virus in their feces nor develop an intestinal carrier state which permits contact transmission. Similar results were obtained with *Rattus rattus* and *Rattus norvegicus* by Kilham *et al.*, (11,12) Other workers have presumed that rodents are the natural reservoir of EMCV.(19,23) EMCV infections in such species as the squirrel, cotton rat, oppossum, and raccoon should be investigated to determine the role that these feral animals play as reservoir hosts.

Whatever the wildlife reservoir and however EMCV was transmitted to the animals described in this report, the end result was tragic. It is unlikely that this problem is unique to Florida, and zoo personnel in other parts of the world are cautioned to keep aware of it.

Summary

1. EMCV infection at 3 zoological parks in Florida is described. Confirmed EMCV-induced deaths are reported in 4 African elephants, 1 Asian elephant, 1 Debrazza monkey, 1 African antelope cross, and 3 chimpanzees. A number of presumptive cases of EMCV infection are presented, including 20 deaths in lions.
2. Deaths were usually acute or peracute and occurred in young as well as older animals.
3. Pulmonary edema or congestion and cardiomyopathy were the most consistent necropsy findings.
4. Most deaths were associated with the cooler months and the possibility that wild animal reserovir hosts were attracted to feeding areas and contaminated these areas with EMCV is raised.
5. EMCV occurs throughout the world and should not be a problem unique to Florida. Therefore, zoo personnel are cautioned to be alert to the possibility of EMCV infection at their institutions.

Table 1. Reported recoveries of EMCV in the continental United States (1960-1977).

Year	Species	State	Reference
1940	Cotton rat	New York	9, 23
1943	Man	New York	8, 23
1945	Chimpanzee	Florida	7
1952	Baboon	Florida	10
1953	Monkey	Florida	10
1954	Squirrel	Florida	10
1960	Swine, rat	Florida	4
1961	Squirrel, raccoon, calf	Florida	5
1962	Swine, baboon, Chimpanzees	Florida	5
1965	Swine	Florida	5
1966	Swine, raccoon, cotton rats, calf	Florida	5
1976	Mosquitos (*Culex* sp.)	Colorado	2
1977	African elephant	Florida	16

Table 2. Summary of Deaths of Lions at Lion Country Safari, West Palm Beach, Florida

1/2/77	1. 17 mo male
	2. 17 mo male
	3. 17 mo male
1/3/77	4. 6 yr female
	5. 4 yr male
	6. 14 mo female
1/4/77	7. 4 yr male
	8. 20 mo female
1/5/77	9. 3 yr female
1/6/77	10. 11 yr male
1/7/77	11. 2 yr male
	12. 2 yr male
1/8/77	13. 4 yr male
	14. 17 mo male
	15. 14 mo male
1/10/77	16. 4 yr male
	17. 17 mo female
1/28/77*	18. 10 yr male
	19. 10 yr male
	20. 10 yr male

* Euthanatized due to unspecified chronic problems.

Table 3. Summary of Suspected and Confirmed EMCV-Associated Deaths of Zoo Animals at Busch Gardens, Lion Country Safari, and Jacksonville Zoo.

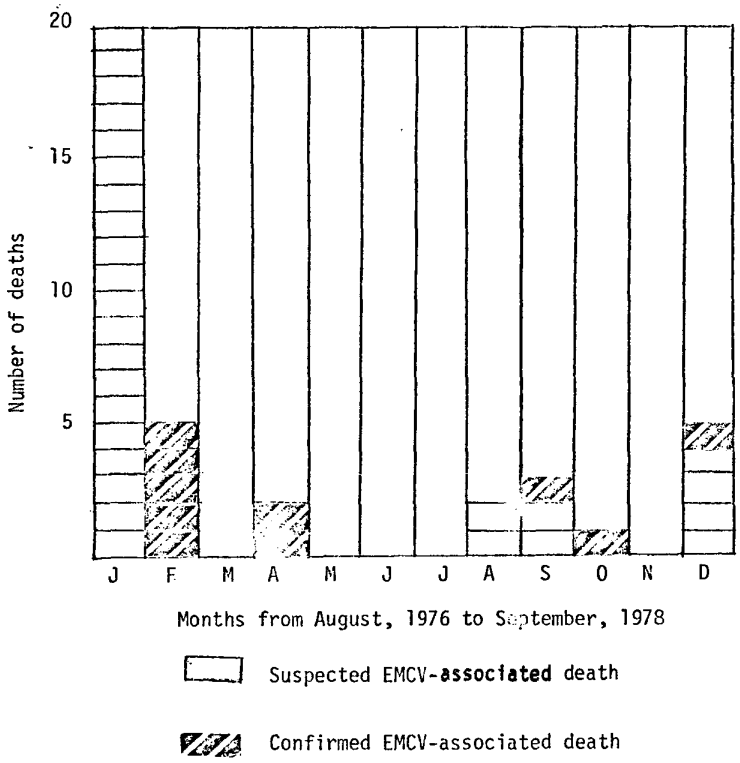

Table 4. Recovery of EMCV from Tissues of Zoo Animals from Busch Gardens (BG), Lion Country Safari (LC), and Jacksonville Zoo (JZ).

Designation	Animal	Date of Death	Tissues from which virus was isolated	Tissues from which virus was not recovered
BG-1	African elephant	2/2/75	Heart, spleen, kidney, skeletal muscle	N.S.
BG-2	African elephant	4/19/78	Heart, spleen	Lung, kidney
BG-3	African elephant	4/30/78	Heart	Liver, spleen, skeletal muscle
LC-1	African elephant	2/26/77	Adrenal, colon, skeletal muscle, heart, mesenteric, lymph node, kidney, liver	N.S.
LC-2	Oryx-addax antelope	12/15/77	Heart, skeletal muscle, liver, spleen, intestine	Kidney, brain
LC-3	DeBrazza monkey	12/27/77	Heart, spleen, liver, lung	N.S.
LC-4	Chimpanzee	2/15/78	Lung, kidney, liver, spleen, heart, thymus	N.S.
LC-5	Chimpanzee	2/21/78	Heart, skeletal muscle, liver, spleen	N.S.
LC-6	Chimpanzee	3/36/78	Skeletal muscle	Heart, liver, spleen
JZ	Asian elephant	9/39/78	Heart, lung, kidney, intestine	Liver

N.S. = No other tissues submitted.

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