VIRGINIA
EPIDEMIOLOGY BULLETIN

C.M.G. Buttery, M.D., M.P.H., Commissioner
Grayson B. Miller, Jr., M.D., Epidemiologist
Editor: Carl W. Armstrong, M.D.

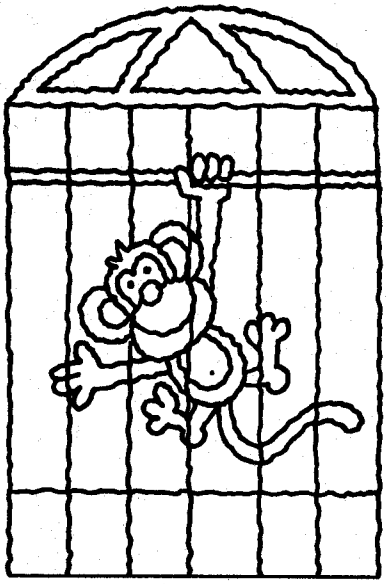
December, 1989
Volume 89, Number 12

Ebola Virus Infection in Imported Primates—Virginia*

In late November 1989, Ebola virus was isolated from cynomolgus monkeys *(Macaca fascicularis)* imported into the United States from the Philippines via Amsterdam and New York. During quarantine in a primate facility in Virginia, numerous macaques died, some with findings consistent with simian hemorrhagic fever (SHF). The U.S. Army Medical Research Insitutue of Infectious Diseases tested 10 animals and, from there, isolated SHF from tissues and serum; however, five other animals of the 10 tested were positive for Ebola virus. Monkeys from a later shipment quarantined in a second room also had unusually high mortality and were tested by a rapid antigen detection enzyme—linked immunosorbent assay. Ebola viral antigen was detected in serum and/or tissues from seven of these monkeys. Primary liver material from animals in both rooms exhibited particles with typical filovirus morphology by electron microscopy and Ebola virus antigen by immunohistochemistry.

All persons who might have come in contact with the monkeys or with tissue or blood specimens from them have been identified and will be under surveillance by the Virginia State Department of Health for three weeks after the last possible exposure for each contact. As of December 6, no evidence of infection has appeared in any of the exposed persons. Any person with symptoms compatible with Ebola infection will be admitted to a local hospital and cared for under CDC guidelines for suspected cases of viral hemorrhagic fevers (1). Appropriate guidelines for management of newly imported primates have been sent to all U.S. primate importation and quarantine facilities, and surveillance for hemorrhagic disease in staff members and in recently imported primates is being instituted. An investigation is under way by CDC, in cooperation with foreign health officials, to identify the source of infection in the monkeys.

Reported by: D. Dalgard, DVM, Hazelton Research Products, Inc. Reston; J. Y. Baumgardner, MAS, C. W. Armstrong, MD, S. R. Jenkins, VMD, C. D. Woolard, MPH, G. B. Miller, Jr, MD, State Epidemiologist, Virginia State Dept. of Health. P. B. Jahrling, PhD, T. G. Ksiazekdum, PhD, E. O. Johnson, PhD, C. J. Peters, MD, U.S. Army Medical Research Institute of Infectious Diseases, Frederick, Maryland. Div. of Quarantine, Center for Prevention Svcs; Div. of Viral and Rickettsial Diseases, Center for Infectious Diseases, CDC.



Editorial Note: Unlike SHF virus, which does not cause clinical illness in humans, Ebola virus can cause severe disease in humans. This report describes the first isolation of Ebola virus in the United States.

Ebola hemorrhagic fever was first recognized in 1976, when two epidemics occurred in southern Sudan and in Zaire (2). A subsequent outbreak occurred in 1979 in Sudan (3). All three outbreaks were associated with high case-fatality rates in humans. In these epidemics, nosocomial transmission (often by contaminated needles) was followed by person-to-person transmission to household members in close contact with blood or secretions from seriously ill patients.

The ecology, natural history, and mode of transmission in nature of Ebola virus and the related Marburg virus are unknown. Before this indicent, no monkey had ever been found to be naturally infected with Ebola virus. The incubation period for Ebola virus is 5-9 days (range: 2-15 days) but can be shorter with parenteral transmission.

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Happy Holidays

Ebola Virus Infection in Imported Primates—Virginia*

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Disease onset is abrupt and characterized by severe malaise, headache, high fever, myalgia, joint pains, and sore throat. The disease course is rapid and includes pharyngitis, conjunctivitis, diarrhea, abdominal pain, and occasionally facial edema and jaundice.. Severe thrombocytopenia can occur, and hemorrhagic manifestations include petechiae and frank bleeding. Death occurs primarily as a result of hypovolemic shock and its consequences. There is no specific therapy (1), and patient management is usually directed at supportive measures.

The only previous documentation of transmission of this family of virus from primates to humans occurred in 1967, when African monkeys infected with Marburg virus were imported into Europe (4). In that outbreak, human infection occurred in 25 workers handling blood and tissues from infected monkeys, and six secondary (person-to-person transmission) cases occurred; seven persons died. Animal caretakers did not become infected.

As a result of the 1967 Marburg virus outbreak, the United States and several other countries instituted a 31-day quarantine for imported monkeys. The facility in the Virginia outbreak routinely has used a 45-day quarantine. In addition to quarantine measures, the use of universal precautions in handling animals or their specimens minimizes the risk for human infections.

Suspected cases of illness in potentially exposed persons should be promptly reported to the Virginia Department of Health, which is working closely with the Special Pa;thogens Branch, Division of Viral and Rickettsial Diseases, Center for Infectious Diseases, CDC.

**Adapted from MMWR 1989;38(47).*

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Interim Information and Guidance for Physicians on Eosinophilia—Myalgia Syndrome

Center for Environmental Health & Injury Control Centers for Disease Control Atlanta, Georgia 30333

Rationale and Limitations

The Centers for Disease Control (CDC) has recently received a large volume of calls from physicians caring for patients with either clear-cut or suspected cases of eosinophilia-myalgia syndrome (EMS), an apparently new clinical entity recently recognized as having occurred in epidemic form over at least the last several months.[1]

The recommendations we set forth here are based largely on the collective but unsystematically derived impressions of a number of us who have discussed over the phone a total of several dozen specific cases, either with other physicians and public health officials or with the patients themselves. To a lesser extent, we have been guided by data developed regarding the toxic oil syndrome (TOS), a disease that was epidemic in Spain in 1981 and whose intermediate and chronic phases resemble certain features of EMS.[2-5]

The physician who chooses to use this document must accept the possibility that our preliminary impressions may later be shown to have been wrong or misleading. Similarly, there can be no guarantee at this time that our recommendations will ultimately turn out to have been those that were most appropriate.

Given the current dearth of information, an individual practitioner's own clinical observations and judgement are particularly important in caring for known or suspected EMS cases and should be the ultimate determining factor in choosing any particular course of treatment. It is nonetheless understandable that physicians should wish to base their judgements, in part, on whatever information may exist regarding observations and judgements that have already been made by their colleagues in similar cases.

Description

EMS appears to have a subacute onset, with symptoms developing over several weeks. Patients typically complain of myalgia and fatigue. The myalgia is very intense and often incapacitating. There may be associated arthralgia. Many patients have respiratory complaints—dyspnea and cough are particularly common—but these are not universal. There may be a skin rash (maculopapular, vesicular or urticarial) that may vanish while other symptoms persist. Some patients have complained of swelling of the extremities, and there may be frank muscle weakness distinct from any loss of extremity function caused by the severe myalgia.

The physical examination may reveal the skin rash, occasionally hepatomegaly (generally without splenomegaly), and extremity edema, but there are no universal or pathognomonic findings. Some patients show signs of heart failure, and there have been reports of arrhythmias (e.g., atrial fibrillation, second degree heart block).

The chest X-ray may show interstitial infiltrates but is often clear, even in the presence of respiratory symptomatology. Arterial blood gases may demonstrate hypoxia. Liver function tests are normal in most patients, although there may be mild to moderate elevations in transaminases and other liver enzymes. Uncommonly, measurements of CPK and aldolase may be elevated. There is striking eosinophilia. The vast majority of patients have absolute eosinophil counts greater than 2,000 cells/cu mm, but eosinophil counts as high as 10,000-30,000 cells/cu mm are not unusual, and higher counts have been reported. Bone marrow examination has generally shown hyperplasia of eosinophil precursors. Muscle biopsy usually demonstrates perivascular inflammatory infiltrates that are typically composed of eosinophils and round cells with relative sparing of the muscle interstitium. Clear-cut vasculitis has occasionally been reported. A few patients have been described to us whose presentations suggest concurrent thromboembolic phenomena (e.g., stroke, pulmonary embolism, or systemic embolization downstream from an area of atherosclerosis).

In a very few patients, the syndrome appears to clear rapidly after discontinuation of L-tryptophan-containing products (LTCPs; see below), with a noticeable improvement in the degree of eosinophilia and other clinical manifestations within a few days. However, improvement is generally slower, and disease in some patients appears to progress even after LTCPs have been stopped. A few patients who have been ill for some months have apparently developed scleroderma-like skin thickening.

An undetermined percentage of patients develop progressive and potentially fatal ascending polyneuropathy. Electrical studies in such patients have been read as most consistent with axonal loss, and pathologically, the pattern is said to be that of mononeuritis multiplex.

Association With L-Tryptophan-Containing Products

The coincidence of EMS with the ingestion of LTCPs is striking. Two case-control studies show a strong and statistically significant association of LTCP use with EMS.[6] It is our current best judgement that this association represents cause-and-effect, although the pathogenic mechanism underlying the development of EMS has yet to be elucidated. Although EMS may well be due to the ingestion of a contaminant or impurity in LTCPs, no such contaminant has yet been identified.

Diagnosis

The current CDC surveillance definition of EMS requires fulfillment of three criteria: (1) Eosinophil count > 1000 cells/cu mm, (2) Myalgias of severity sufficient to interfere with a patient's ability to pursue his or her usual activities, and (3) exclusion of other infectious or neoplastic ill-

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*Eosinophilia-Myalgia Syndrome (EMS)**

On October 30, 1989, the New Mexico Department of Health and Environment (NMDHE) was notified of three patients with eosinophilia and severe myalgia who had been taking oral preprations of the amino acid L-tryptophan (LT). Even though the patients had undergone extensive clinical evaluation and testing, their illnesses were not consistent with any known diagnostic entity. Public announcement of the cluster led rapidly to reports of similar cases in New Mexico and, subsequently, in other states as well.

As of December 6, 707 cases of eosinophilia-myalgia syndrome (EMS) had been reported by state health departments to CDC, including seven cases from Virginia. Studies examining an association of L-tryptophan-containing products (LTCPs) with the EMS epidemic have been completed in New Mexico, Minnesota, and Oregon. In addition, a fatal case in New York has been reported.

New Mexico. In a New Mexico case-control study, EMS cases (N=12) were compared with controls (two per case) who had been matched with case-patients by age (plus or minus 5 years), sex, and neighborhood of residence. Comparisons were made for factors such as the use of different vitamins, other health foods or raw food products, medications, and different water sources. All case-patients and two (8%) controls used LTCPs (p=6.9 × 10-6). There were no statistically significant differences between cases and controls on 32 other potential risk factors studied.

Minnesota. In Minnesota, investigators had no prior knowledge of patients' use of LTCPs. Twelve cases were identified and compared with controls (one per case) matched by age, sex, and telephone exchange. All case-patients and no controls used LTCPs (p=8 × 10-4) during the month before onset of illness for case-patients and during a similar time period for matched controls. Illness was not associated with consumption of vitamins and health-food products, wild game, undercooked meat or fish products, or medications. A follow-up study compared 30 EMS cases with 36 asymptomatic users of LTCPs who responded to a public request and contacted the Minnesota Department of Health. Twenty (67%) case-patients reported using brands of LTCPs from one particular tablet manufacturer, compared with eight (22%) asymptomatic users (OR=7.0; 95% confidence interval (CI)=1.5-24.6 (p less than 0.0002)). Asymptomatic LTCP users were similar to case-patients for age, sex, and geographic areas of residence.

Oregon. The Oregon Health Division studied 29 EMS patients, all users of LTCPs, and compared them with users of LTCPs identified by a random telephone survey of Oregon residents (control group A; N=32) and asymptomatic LT users who contacted the Oregon Health Division (control group B; N=24). Fourteen (48%) case-patients were exposed to LTCPs from a single lot of 4500 bottles, compared with two (6%) persons in control group A and two (8%) persons in control group B (ORs=14.0 (95% CI=2.5-103.0) and 10.3 (95% CI=1.8-76.8), respectively) who were so exposed. This association remains statistically significant when controlled for age, sex, or average daily LTCP consumption.

New York. In New York, a 58-year-old woman with EMS died September 17, 1989. The patient, who had become ill in July 1989 with myalgia, fatigue, and marked progressive weakness, had been taking 5-6 g of LT daily. She had leukocytosis (19,800 cells/mm3) with 18% eosinophils. Electromyelographic and nerve conduction studies were most consistent with axonal neuropathy. Studies considered to be within normal limits included: cerebrospinal fluid glucose, protein, and cell counts and celiac and renal arteriograms. Serologic tests for a variety of autoimmune diseases were negative. The patient developed an ascending polyneuropathy with near-total quadriplegia and a bifacial hemiparesis. She failed to improve on corticosteroid and cyclophosphamide treatment and died following cardiorespiratory arrest.

Editorial Note: Although EMS shares some features with previous case reports (1-3), it has not been described in epidemic form. In addition, EMS closely parallels the intermediate and chronic phases of toxic-oil syndrome (TOS), which occurred in epidemic form in Spain in 1981. In that epidemic, patients also had severe myalgia and intense eosinophilia, as well as other manifestations (4-7). LT is an essential amino acid that is normally ingested as a constituent of dietary protein. LT supplements are used by some persons for disorders such as insomnia, depression, and premenstrual syndrome (8).

Because this syndrome represents an apparently new clinical entity, diagnostic criteria have not yet been established. Many of the potential cases reported to CDC had initially been diagnosed as other illnesses, such as eosinophilic myositis, eosinophilic fasciitis, polyarteritis nodosa, and suspected trichinosis.

For surveillance purposes, CDC recommends defining a case of EMS as an illness characterized by all of the following: 1) eosinophil count greater than or equal to 1000 cells per mm3; 2) generalized myalgia (at some point during the course of illness) of severity sufficient to affect a patient's ability to pursue his or her usual daily activities; and 3) absence of any infection or neoplasm that could account for 1 or 2 above.

The findings of the lot and brand-name studies in Minnesota and Oregon, suggest multiple interpretations: some LTCPs could contain a contaminant that is causally associated with EMS; or host factors mediating the response to LT may be unique to patients who use a particular brand or set of brands associated with illness. Studies under way include identifying possible chemical or

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Interim Information and Guidance for Physicians on Eosinophilia—Myalgia Syndrome

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ness(es) that might account for #1 or #2. The aim of this definition is to achieve uniformity in statistical reporting of EMS and these criteria should not be considered necessarily applicable to the diagnosis of individual patients. Ultimately, diagnosis of an individual case should represent the patient's physician's best judgement as to the nature of the disease process affecting his or her patient and should not be unduly swayed by case definitions developed for epidemiologic studies or surveillance. Although the diagnosis of EMS would be difficult to support in the absence of eosinophilia, there is no specific laboratory test for EMS. The illness should be diagnosed by its conforming more or less to the lines of the description of the syndrome outlined above. It is important, however, that due care be taken to thoroughly evaluate the patient for other possible causes of eosinophilia and myalgia. Although the diagnosis of EMS is more likely in a patient with a history of use of LTCPs in the month before onset, one must not assume that any patient with eosinophilia who has been using LTCPs has EMS.

Treatment and Prognosis

The only clear-cut recommendation that can be made at this time is that LTCPs be stopped. If illness is mild or does not appear to be progressive, it may be reasonable to simply observe the patient off LTCPs and not to give any pharmacologic therapy.

Glucocorticoids (e.g., prednisone) have been used in a number of patients. Data are insufficient for us to recommend a particular dose, but risk-benefit considerations would argue for initial use of a high dose in criticaly ill or severely affected patients and an initially low dose in patients with milder illness. The eosinophil count may fall rapidly with steroid treatment, but there can be substantially less improvement in other disease manifestations. The improvement that may occur with steroid therapy has to be weighed against possible complications, particularly if the degree of improvement is slight and as the anticipated period of treatment lengthens.

Whether or not glucocorticoids are given, non-steroidal anti-inflammatory agents and narcotic analgesics may be useful for the relief of severe muscle pain.

One might reasonably contemplate therapy with cytotoxic agents in some patients with severe, progressive illness. However, we have insufficient information to make any clear-cut recommendation on this point.

It is presently unclear to what extent the pathology of EMS is reversible, particularly in severely affected patients.

Case Reporting

So that an accurate assessment of the impact of this syndrome on the nation's health can be made, all cases and suspected cases of EMS should be reported to state health departments. Special note should be taken of the fact that a history of use of LTCPs is not required in the surveillance definition on which case reporting is based.

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Eosinophilia—Myalgia Syndrome (EMS*)

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microbial contaminants in LTCPs, tracing the sources of individual brands and lots, identifying host factors related to clinical manifestations, and determining factors associated with use and purchase of LTCPs.

On November 17, the Food and Drug Administration (FDA) announced its intention to seek a nationwide recall of all LTCPs in which LT is the sole or major component; this reinforced a November 11 alert to the public to refrain from using LTCPs. At least one case has been reported, however, in a patient taking a combination product, i.e., a product not identified as an LT product but as a multicomponent supplement where LT is one of many ingredients. FDA is attempting to trace suspect lots of LTCPs and is evaluating production procedures at the companies in Japan where LT is produced for eventual sale and consumption in the United States.

Adapted from MMWR 1989;38:765-767, and 785-788

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Letter to the Editor

To the Editor:

I have several questions about the article on Lyme disease that appeared in the August issue of the *Bulletin* (Vol. 89 No. 8). I would like to know the recovery rate of Lyme disease spirochetes from ticks in the presumed endemic counties. In addition, I would like to know how one defines erythema chronicum migrans (ECM). It is my opinion that some physicians may be confusing tinea corporis (ringworm) or granuloma annulare, both of which are quite common, with ECM. Your statistics, which show that 95% of reported cases had ECM, are high compared to the average in other endemic areas, supporting my suspicion. Finally, the disparate geographic areas of presumed endemic disease are unusual. Your response will be most welcome.

Robert A. Silverman, MD
Annandale, VA

In reply:

The definitions that the Virginia Department of Health uses in defining Lyme disease cases and Lyme endemic localities are the ones suggested by the Centers for Disease Control and the Council of State and Territorial Epidemiologists. By using national, standardized definitions our data are more comparable with data from the rest of the country. As stated in the article, "a locality is defined as endemic if a definite case has been documented or if *Ixodes dammini* ticks are found in the area." The case definition was also provided in the article. As with all definitions used for disease surveillance, there may be bona fide cases that do not meet the definition and vice versa. Hopefully, the proportion of such cases will not vary greatly over time and across different geographic areas, thus allowing us to analyze trends over time and geographic differences.

Ticks of the *I. dammini* species have been identified from Accomack County, Fort A. P. Hill and Fort Pickett in Virginia. *Borrelia burgdorferi* were identified in one *I. dammini* tick collected in Accomack County and several other species of ticks from the Eastern Shore of Virginia. However, the surveillance definition for an endemic locality does not require the identification of *B. burgdorferi* in ticks. To date, *I. dammini* ticks have represented a small percentage of the ticks in these areas. This is consistent with the low case numbers of Lyme disease reported in Virginia compared with areas in the Northeast that have a preponderance of *I. dammini* ticks and many more human cases.

Several different forms are presently being used to report Lyme disease to the Department of Health. They all describe erythema chronicum migrans (ECM) as a red, circular expanding lesion with central clearing. If a physician reports that a lesion fitting this description was present, we accept his/her professional determination. As the article states, we believe that our 95% ECM rate is more related to ECM being a major part of the case definition, than to over-diagnosis.

Lyme disease does occur in Virginia, but the number of cases is very low compared to highly endemic parts of the country like the northeast, upper midwest and California. We intend to continue monitoring, not only through case reports of human disease, but also by tick and animal studies, to identify changes in the status of Lyme disease in Virginia.

We appreciate your comments and questions.

Cases of selected notifiable diseases, Virginia, for the period November 1 through November 30, 1989.

DISEASE	TOTAL CASES REPORTED THIS MONTH						TOTAL CASES REPORTED TO DATE		
	STATE	REGIONS					THIS YEAR	LAST YEAR (STATE TOTALS)	5 YEAR AVERAGE
		N.W.	N.	S.W.	C.	E.			
Acquired Immunodeficiency Syndrome	27	4	6	4	5	8	352	345	—
Campylobacter Infections	38	7	9	8	10	4	632	665	620
Gonorrhea	1363	—	—	—	—	—	14788	12957	16026
Hepatitis A	44	0	6	2	28	8	312	341	191
B	34	1	6	10	3	14	296	306	438
Non A-Non B	3	0	0	0	0	3	66	73	72
Influenza	16	0	0	0	0	16	1954	2486	2003
Kawasaki Syndrome	0	0	0	0	0	0	22	13	21
Legionellosis	3	1	0	1	0	0	12	11	19
Lyme Disease	9	0	0	0	0	9	52	27	12
Measles	0	0	0	0	0	0	22	220	63
Meningitis — Aseptic	50	2	14	12	5	17	385	194	274
Bacterial*	23	8	2	5	2	6	173	162	196
Meningococcal Infections	7	0	0	3	1	3	62	54	60
Mumps	7	3	0	1	1	2	118	136	65
Pertussis	1	0	0	0	1	0	34	23	31
Rabies in Animals	27	5	3	1	15	3	246	338	246
Reye Syndrome	0	0	0	0	0	0	2	0	2
Rocky Mountain Spotted Fever	0	0	0	0	0	0	16	17	32
Rubella	0	0	0	0	0	0	0	11	3
Salmonellosis	107	12	21	15	38	21	1358	1645	1505
Shigellosis	24	1	10	1	2	10	389	433	202
Syphilis (Primary & Secondary)	74	2	10	12	35	15	548	399	338
Tuberculosis	31	5	9	3	14	0	333	372	385

Localities Reporting Animal Rabies: Amelia 1 raccoon; Augusta 1 bat; Chesterfield 2 raccoons; Cumberland 1 raccoon; James City 1 fox; Loudoun 2 raccoons, 1 skunk; Madison 1 skunk; Nottoway 3 raccoons, 4 skunks; Prince George 4 raccoons; Shenandoah 2 cows, 1 raccoon; Smith 1 skunk; York 2 raccoons.

Occupational Illnesses: Asbestosis 21; Carpal Tunnel Syndrome 32; Coal Workers' Pneumoconiosis 35; Loss of Hearing 10; Poisoning—Lead 1; Poisoning—Mercury 1; Repetitive Trauma Disorder 3.

*Other than meningococcal

Published Monthly by the
VIRGINIA HEALTH DEPARTMENT
Office of Epidemiology
109 Governor Street
Richmond, Virginia 23219