16 may 2012

RISK OF INFECTION EAST AFRICA

RECENT OUTBREAKS OF INFECTIOUS DISEASES

<table>
<thead>
<tr>
<th>Country</th>
<th>Disease</th>
<th>Date of Outbreak</th>
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<tbody>
<tr>
<td>Tanzania</td>
<td>Rift valley fever</td>
<td>2007</td>
</tr>
<tr>
<td>Kenya</td>
<td>Rift valley fever</td>
<td>2007, 2006</td>
</tr>
<tr>
<td></td>
<td>Poliomyelitis</td>
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<tr>
<td></td>
<td>Meningococcal disease</td>
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<tr>
<td></td>
<td>Leptospirosis</td>
<td>2004</td>
</tr>
<tr>
<td></td>
<td>Meningococcal disease</td>
<td>2007, 2006</td>
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<tr>
<td></td>
<td>Cholera</td>
<td>2003</td>
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<tr>
<td>Rwanda</td>
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<td>Cholera</td>
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<td>Djibouti</td>
<td>Avian influenza</td>
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<tr>
<td>Eritrea</td>
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<td>Ethiopia</td>
<td>Acute diarrheal syndrome</td>
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<td></td>
<td>Poliomyelitis</td>
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<tr>
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<td>Anthrax</td>
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<td>Somalia</td>
<td>Rift valley fever</td>
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<tr>
<td></td>
<td>Meningococcal disease</td>
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### MODE OF TRANSMISSION, SIGNS AND SYMPTOMS OF SELECTED DISEASES

**AVIAN INFLUENZA**

**Transmission and etiology**

Avian influenza is flu infection in birds. The virus that causes the bird infection can change (mutate) to infect humans. Such mutation could start a deadly worldwide epidemic. Human cases of avian influenza A (H5N1) have been reported in Asia, Africa, Europe, Indonesia, Vietnam, the Pacific, and the near East. Hundreds of people have become sick with this virus. Slightly more than 60% of those who became ill have died.

**Clinical signs and symptoms**

Symptoms of avian flu infection in humans depend on the strain of virus. Infection with the H5N1 virus in humans causes typical flu-like symptoms, which might include: **cough (dry or productive), diarrhea, difficulty breathing, fever greater than 100.4°F (38°C), headache, malaise, muscle aches, runny nose and sore throat.**

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<tr>
<th>Country</th>
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<td>Acute respiratory syndrome</td>
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<td>Malawi</td>
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<td></td>
<td>New virus (Arenoviridae family)</td>
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<td></td>
<td>Plague</td>
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<td>Comoros</td>
<td>Cholera</td>
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<td>Mauritius</td>
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<td>Chikungunya</td>
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<td>Reunion</td>
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<td>Mayotte</td>
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</table>
Prevention

The Food and Drug Administration has approved one vaccine to prevent infection with one strain of H5N1 bird flu virus. This vaccine isn't available to the public, but the U.S. government is stockpiling it and will distribute it in the event of an outbreak. It's intended to help protect adults ages 18 to 64 and could be used early in such an outbreak to provide limited protection until another vaccine, designed to protect against the specific form of the virus causing the outbreak, is developed and produced.

Recommendations for travelers

If you're traveling to Southeast Asia or to any region with bird flu outbreaks, consider these public health recommendations:

Avoid domesticated birds. If possible, avoid rural areas, small farms and open-air markets.

Wash your hands. This is one of the simplest and best ways to prevent infections of all kinds. Use an alcohol-based hand sanitizer containing at least 60 percent alcohol when you travel.

Ask about a flu shot. Before traveling, ask your doctor about a flu shot. It won't protect you specifically from bird flu, but it may help reduce the risk of simultaneous infection with bird and human flu viruses.

Avoid cross-contamination. Use hot, soapy water to wash cutting boards, utensils and all surfaces that have come into contact with raw poultry.

Cook thoroughly. Cook chicken until the juices run clear, and it reaches a minimum internal temperature of 165 F (74 C).

Steer clear of raw eggs. Because eggshells are often contaminated with bird droppings, avoid foods containing raw or undercooked eggs.

ANTHRAX

Transmission and etiology

Anthrax is an infectious disease due to a type of bacteria called Bacillus anthracis. There are three main routes of anthrax infection: infection in humans most often involves the skin, gastrointestinal tract, or lungs. Cutaneous anthrax occurs when anthrax spores touch a cut or scrape on the skin. It is the most common type of anthrax infection. The main risk is contact with animal hides or hair, bone products, and wool, or with infected animals. People most at risk for cutaneous anthrax include farm workers, veterinarians, and tannery and wool workers.

Inhalation anthrax develops when anthrax spores enter the lungs through the respiratory tract. It is most commonly contracted when workers breathe in airborne anthrax spores during processes such as tanning.
hides and processing wool. Breathing in spores means a person has been exposed to anthrax, but it does not mean the person will have symptoms. The bacteria spores must "germinate" or sprout (the same way a seed might sprout before a plant grows) before the actual disease occurs. The process usually takes 1 to 6 days. Forty-three days is the longest known incubation period. Once the spores germinate, they release several toxic substances. These substances cause internal bleeding, swelling, and tissue death. **Gastrointestinal anthrax** occurs when someone eats anthrax-tainted meat. Symptoms of anthrax differ depending on the type of anthrax.

**Clinical signs and symptoms**

Symptoms of **cutaneous** anthrax start 1 to 7 days after exposure: an **itchy sore** develops that is similar to an insect bite. This sore may blister and form a black ulcer (sore). The sore is usually painless, but it is often surrounded by swelling. A scab often forms, and then dries and falls off within 2 weeks. Complete healing can take longer.

Symptoms of **inhalation anthrax**: begins with **fever, malaise, headache, cough, shortness of breath, and chest pain**. Fever and **shock** may occur later.

Symptoms of **gastrointestinal anthrax** usually occur within 1 week and may include: **abdominal pain, bloody diarrhea, fever, mouth sores, nausea and vomiting** (the vomit may contain blood). When treated with antibiotics, cutaneous anthrax is likely to get better. However, up to 20% of people who do not get treatment may die if anthrax spreads to the blood.

People with second-stage inhalation anthrax have a poor outlook, even with antibiotic therapy. Up to 90% of cases in the second stage are fatal. Gastrointestinal anthrax infection can spread to the bloodstream, and may result in death.

**Prevention**

Antibiotics are recommended to prevent infection in anyone exposed to the spores. Ciprofloxacin, doxycycline and levofloxacin (Levaquin) are approved by the Food and Drug Administration for post-exposure prevention of anthrax in adults and children.

An anthrax vaccine for humans is available, but it's not 100 percent effective. The vaccine doesn't contain live bacteria and can't lead to infection, but it can cause side effects, ranging from soreness at the injection site to more-serious allergic reactions. The vaccine isn't recommended for children, pregnant women or older adults.

The vaccine isn't intended for the general public. Instead, it's reserved for military personnel, scientists working with anthrax and people in other high-risk professions.
If you live or travel in a country where anthrax is common and herd animals aren't routinely vaccinated, avoid contact with livestock and animal skins as much as possible. Also avoid eating meat that hasn't been properly cooked.

Even in developed countries, it's important to handle any dead animal with care and to take precautions when working with or processing imported hides, fur or wool.

**CHIKUNGUNYA (CHIKV)**

**Transmission and etiology**

Chikungunya (in the Makonde language "that which bends up") virus (CHIKV) is an insect-borne virus, that is transmitted to humans by virus-carryings Aedes mosquitoes. There have been recent breakouts of CHIKV associated with severe illness.

**Clinical signs and symptoms**

CHIKV infection causes an illness with symptoms similar to dengue fever, with an acute disease that affects the joints of the extremities febrile phase of the illness lasting only two to five days, followed by a prolonged arthralgia (neuralgic pain in a joint or joints). The pain associated with CHIKV infection of the joints persists for weeks or months, or in some cases years. The incubation period of chikungunya disease is from two to five days. Its symptoms include a **fever up to 40 °C (104 °F)**, a petechial or maculopapular **rash** (skin is covered with small confluent bumps) of the trunk and occasionally the limbs, and **arthralgia or arthritis affecting multiple joints**. Other nonspecific symptoms can include headache, conjunctivitis, and slight photophobia. Typically, the fever lasts for two days and then ends abruptly. However, other symptoms: namely joint pain, intense headache, insomnia and an extreme degree of prostration, last for a variable period; usually for about five to seven days. Patients have complained of joint pains for much longer time periods; some as long as two years, depending on their age.

**Prevention**

The best way to prevent chikungunya virus infection is to avoid mosquito bites. There is no vaccine or preventive drug currently available. Prevention tips are similar to those for other viral diseases transmitted by mosquitoes, such as dengue or West Nile.
- Use insect repellent containing DEET, Picaridin, oil of lemon eucalyptus, or IR3535 on exposed skin. Always follow the directions on the package.
- Wear long sleeves and pants (ideally treat clothes with permethrin or another repellent).
- Have secure screens on windows and doors to keep mosquitoes out.
- Get rid of mosquito sources in your yard by emptying standing water from flower pots, buckets and barrels. Change the water in pet dishes and replace the water in bird baths weekly. Drill holes in tire swings so water drains out. Keep children's wading pools empty and on their sides when they aren't being used.

Additionally, a person with chikungunya fever should limit their exposure to mosquito bites to avoid further spreading the infection. The person should use repellents when outdoors exposed to mosquito bites or stay indoors in areas with screens or under a mosquito net.

**CHOLERA**

**Transmission and etiology**

Cholera is an infection in the small intestine caused by the bacterium *Vibrio cholerae*. Cholera is typically transmitted by either contaminated food or water. In the developed world, seafood is the usual cause, while in the developing world it is more often water. Cholera has been found in only two other animal populations: shellfish and plankton. People infected with cholera often have diarrhea, and if this highly liquid stool, colloquially referred to as "rice-water" or "faucet butt", contaminates water used by others, disease transmission may occur. The source of the contamination is typically other cholera sufferers when their untreated diarrheal discharge is allowed to get into waterways, groundwater or drinking water supplies. Drinking any infected water and eating any foods washed in the water, as well as shellfish living in the affected waterway, can cause a person to contract an infection. Cholera is rarely spread directly from person to person. Both toxic and nontoxic strains exist. Nontoxic strains can acquire toxicity through a temperate bacteriophage.

Coastal cholera outbreaks typically follow zooplankton blooms, thus making cholera a zoonotic disease.

**Clinical signs and symptoms**

For every symptomatic person, 3 to 100 people get the infection but remain asymptomatic. The primary symptoms of cholera are profuse, painless diarrhea and vomiting of clear fluid. These symptoms usually start suddenly, one to five days after ingestion of the bacteria. The diarrhea is frequently described as "rice water" may have a fishy odor. An untreated person with cholera may produce 10–20 liters of diarrhea a day with fatal results. Cholera has been nicknamed the "blue death" due to a patient's skin turning a bluish-gray hue from extreme loss of fluids If the severe diarrhea is not treated with intravenous rehydration, it can result in life-threatening dehydration and electrolyte imbalances. The typical symptoms of dehydration include low blood pressure, poor skin turgor (wrinkled hands), sunken eyes, and a rapid pulse.

**Prevention**

Although cholera may be life-threatening, prevention of the disease is normally straightforward if proper sanitation practices are followed.
EBOLA HEMORRHAGIC FEVER (EHF)

Transmission and etiology

Fruit bat

The Ebola virus is transmitted by direct contact with the blood, body fluids and tissues of infected persons. Transmission of the Ebola virus has also occurred by handling sick or dead infected wild animals (chimpanzees, gorillas, monkeys, forest antelope, fruit bats). The absence of clinical signs in bats is characteristic of a reservoir species.

The predominant treatment is general supportive therapy. There are five distinct species of the Ebola virus: Bundibugyo, Côte d’Ivoire, Reston, Sudan and Zaïre. Bundibugyo, Sudan and Zaïre species have been associated with large outbreaks of Ebola hemorrhagic fever in Africa causing death in 25-90% of all clinically ill cases, while Côte d’Ivoire and Reston have not. EHF is clinically indistinguishable from Marburg virus disease (MVD), and it can also easily be confused with many other diseases prevalent in Equatorial Africa, such as other viral hemorrhagic fevers, falciparum malaria, typhoid fever, shigellosis, rickettsial diseases, cholera, gram-negative septicemia or enteritis. The most detailed studies on the frequency, onset, and duration of EVD clinical signs and symptoms were performed during the 1995 outbreak in Kikwit, Zaire (EBOV) and the 2007-2008 outbreak in Bundibugyo, Uganda (BDBV).
Clinical signs and symptoms

EVD begins with a sudden onset of an influenza-like stage characterized by general malaise, fever with chills, arthralgia and myalgia, and chest pain. Nausea is accompanied by abdominal pain, diarrhea, and vomiting. Respiratory tract involvement is characterized by pharyngitis with sore throat, noexia, cough, dyspnea, and hiccups. The central nervous system is affected as judged by the development of severe headaches, agitation, confusion, fatigue, depression, seizures, and sometimes coma. The circulatory system is also frequently involved, with the most prominent signs being edema and conjunctivitis. Hemorrhagic symptoms are infrequent (fewer than 10% of cases for most serotypes), (the reason why Ebola hemorrhagic fever (EHF) is a misnomer) and include hematemesis, hemoptysis, melena, and bleeding from mucous membranes (gastrointestinal tract, nose, vagina and gingiva). Cutaneous presentation may include: maculopapular rash, petechiae, purpura, ecchymoses, and hematomas (especially around needle injection sites). Development of hemorrhagic symptoms is generally indicative of a negative prognosis. However, contrary to popular belief, hemorrhage does not lead to hypovolemia and is not the cause of death (total blood loss is low except during labor). Instead, death occurs due to multiple organ dysfunction syndrome (MODS) due to fluid redistribution, hypotension, disseminated intravascular coagulation, and focal tissue necrosis. Prognosis is generally poor (average case-fatality rate of all EVD outbreaks to date = 68%). If a patient survives, recovery may be prompt and complete, or protracted with squeals, such as orchitis, arthralgia, myalgia, desquamation or alopecia. Ocular manifestations, such as photophobia, hyperlacrimation, iritis, iridocyclitis, choroiditis and blindness have also been described. Importantly, EBOV and SUDV are known to be able to persist in the sperm of some survivors, which could give rise to secondary infections and disease via sexual intercourse.

Prevention

There are currently no Food and Drug Administration-approved vaccines for the prevention of EVD. As an outbreak of Ebola progresses, bodily fluids from diarrhea, vomiting, and bleeding represent a hazard. Due to lack of proper equipment and hygienic practices, large-scale epidemics occur mostly in poor, isolated areas without modern hospitals or well-educated medical staff. Many areas where the infectious reservoir exists have just these characteristics. In such environments, all that can be done is to immediately cease all needle-sharing or use without adequate sterilization procedures, isolate patients, and observe strict barrier nursing procedures with the use of a medical-rated disposable face mask, gloves, goggles, and a gown at all times, strictly enforced for all medical personnel and visitors.
LEPTOSPIROSIS

Transmission and etiology

Leptospirosis is caused by infection with bacteria of the genus Leptospira, and affects humans as well as other mammals, birds, amphibians, and reptiles. Leptospirosis is a relatively rare bacterial infection in humans. Leptospirosis is also transmitted by the semen of infected animals. Slaughterhouse workers may contract the disease through contact with infected blood or body fluids.

Humans become infected through contact with water, food, or soil containing urine from these infected animals. This may happen by swallowing contaminated food or water, or through skin contact. The disease is not known to be spread from person to person and cases of bacterial dissemination in convalescence are extremely rare in humans. Leptospirosis is common among water-sport enthusiasts in specific areas as prolonged immersion in water is known to promote the entry of the bacteria. Surfers and whitewater paddlers are at especially high risk in areas that have been shown to contain the bacteria, and can contract the disease by swallowing contaminated water, splashing contaminated water into their eyes or nose, or exposing open wounds to infected water. Occupations at risk include veterinarians, slaughterhouse workers, farmers, sewer workers, and people working on derelict buildings. Rowers are also sometimes known to contract the disease.

Clinical signs and symptoms

Leptospiral infection in humans causes a range of symptoms, and some infected persons may have no symptoms at all. Leptospirosis is a biphasic disease that begins with flu-like symptoms (fever, chills, myalgia, and intense headache). The first phase resolves, and the patient is briefly asymptomatic until the second phase begins. This is characterized by meningitis, liver damage (causing jaundice), and renal failure. The infection is often wrongly diagnosed due to the wide range of symptoms. This leads to a lower registered number of cases than exists. Symptoms of leptospirosis include high fever, severe headache, chills, muscle aches, and vomiting, and may include jaundice, red eyes, abdominal, diarrhea, and rash. Initial presentation may resemble pneumonia. The symptoms in humans appear after a 4–14 day incubation period. Cardiovascular problems are also possible.

Prevention

The risk of acquiring leptospirosis can be greatly reduced by not swimming or wading in water that might be contaminated with animal urine, or eliminating contact with potentially infected animals.

Protective clothing or footwear should be worn by those exposed to contaminated water or soil because of their job or recreational activities.

MARBURG HEMORRHAGIC FEVER

Transmission and etiology

The Marburg virus is transmitted by direct contact with the blood, body fluids and tissues of infected persons. Transmission of the Marburg virus also occurred by handling ill or dead infected wild animals (monkeys, fruit bats)
Clinical signs and symptoms

Illness caused by Marburg virus begins abruptly, with severe headache and severe malaise. Many patients develop severe hemorrhagic manifestations between days 5 and 7, and fatal cases usually have some form of bleeding, often from multiple sites. The disease has no vaccine and no specific treatment. Fatality rates have varied greatly, from 25% in the initial laboratory-associated outbreak in 1967, to more than 80% in the Democratic Republic of Congo from 1998-2000, to even higher in the outbreak that began in Angola in late 2004.

Prevention

No vaccine is available. Avoid contact with monkeys and fruit bats.

POLIOMYELITIS (POLIO)

Transmission and etiology

Poliomyelitis often called polio or infantile paralysis is an acute, viral, infectious disease spread from person to person, primarily via the fecal-oral route. Although approximately 90% of polio infections cause no symptoms at all, affected individuals can exhibit a range of symptoms if the virus enters the blood stream. In about 1% of cases, the virus enters the central nervous system, preferentially infecting and destroying motor neurons, leading to muscle weakness and acute flaccid paralysis. Different types of paralysis may occur, depending on the nerves involved. Spinal polio is the most common form, characterized by asymmetric paralysis that most often involves the legs. Bulbar polio leads to weakness of muscles innervated by cranial nerves. Bulbospinal polio is a combination of bulbar and spinal paralysis. Two basic patterns of polio infection are described: a minor illness which does not involve the central nervous system (CNS), sometimes called abortive poliomyelitis, and a major illness involving the CNS, which may be paralytic or nonparalytic. In most people with a normal immune system, a poliovirus infection is asymptomatic. Rarely, the infection produces minor symptoms; these may include upper respiratory tract infection (sore throat and fever), gastrointestinal disturbances (nausea, vomiting, abdominal pain, constipation or, rarely, diarrhea), and influenza-like illness. The virus enters the central nervous system in about 3% of infections. Most patients with CNS involvement develop nonparalytic aseptic meningitis, with symptoms of headache, neck, back, abdominal and extremity pain, fever, vomiting, lethargy and irritability. About one to five in 1000 cases progress to paralytic disease, in which the muscles become weak, floppy and poorly controlled, and finally completely paralyzed; this condition is known as acute flaccid paralysis. Depending on the site of paralysis, paralytic poliomyelitis is classified as spinal, bulbar, or bulbospinal. Encephalitis, an infection of the brain tissue itself, can occur in rare cases, and is usually restricted to infants. It is characterized by confusion, changes in mental status, headaches, fever, and less commonly, seizures and spastic paralysis.

Because Poliomyelitis is highly contagious via the oral-oral (oropharyngeal source) and fecal-oral (intestinal source) routes, in endemic areas, wild polioviruses can infect virtually the entire human population. Poliomyelitis is seasonal in temperate climates, with peak transmission occurring in summer and autumn. These seasonal differences are far less pronounced in tropical areas.

The time between first exposure and first symptoms, known as the incubation period, is usually six to 20 days, with a maximum range of three to 35 days. Virus particles are excreted in the feces for several
weeks following initial infection. Factors that increase the risk of polio infection or affect the severity of the disease include immune deficiency, malnutrition, tonsillectomy, physical activity immediately following the onset of paralysis, skeletal muscle injury due to injection of vaccines or therapeutic agents, and pregnancy. Although the virus can cross the placenta during pregnancy, the fetus does not appear to be affected by either maternal infection or polio vaccination. Maternal antibodies also cross the placenta, providing passive immunity that protects the infant from polio infection during the first few months of life. As a precaution against infection, public swimming pools were often closed in affected areas during poliomyelitis epidemics.

Clinical signs and symptoms

Early symptoms of paralytic polio include high fever, headache, stiffness in the back and neck, asymmetrical weakness of various muscles, sensitivity to touch, difficulty swallowing, muscle pain, loss of superficial and deep reflexes, paresthesia (pins and needles), irritability, constipation, or difficulty urinating. Paralysis generally develops one to ten days after early symptoms begin, progresses for two to three days, and is usually complete by the time the fever breaks. The likelihood of developing paralytic polio increases with age, as does the extent of paralysis. In children, nonparalytic meningitis is the most likely consequence of CNS involvement, and paralysis occurs in only one in 1000 cases. In adults, paralysis occurs in one in 75 cases. In children under five years of age, paralysis of one leg is most common; in adults, extensive paralysis of the chest and abdomen also affecting all four limbs (quadriplegia) is more likely. Paralysis rates also vary depending on the serotype of the infecting poliovirus; the highest rates of paralysis (one in 200) are associated with poliovirus type 1, the lowest rates (one in 2).

Spinal polio, the most common form of paralytic poliomyelitis, results from viral invasion of the motor neurons of the anterior horn cells, or the ventral (front) gray matter section in the spinal column, which are responsible for movement of the muscles, including those of the trunk, limbs and the intercostal muscles. Virus invasion causes inflammation of the nerve cells, leading to damage or destruction of motor neuron ganglia. When spinal neurons die, Wallerian degeneration takes place, leading to weakness of those muscles formerly innervated by the now-dead neurons. With the destruction of nerve cells, the muscles no longer receive signals from the brain or spinal cord; without nerve stimulation, the muscles atrophy, becoming weak, floppy and poorly controlled, and finally completely paralyzed. Progression to maximum paralysis is rapid (two to four days), and is usually associated with fever and muscle pain. Deep tendon reflexes are also affected, and are usually absent or diminished; sensation (the ability to feel) in the paralyzed limbs, however, is not affected.

The extent of spinal paralysis depends on the region of the cord affected, which may be cervical, thoracic, or lumbar. The virus may affect muscles on both sides of the body, but more often the paralysis is asymmetrical. Any limb or combination of limbs may be affected—one leg, one arm, or both legs and both arms. Paralysis is often more severe proximally (where the limb joins the body) than distally (the fingertips and toes).

Making up about 2% of cases of paralytic polio, bulbar polio occurs when poliovirus invades and destroys nerves within the bulbar region of the brain stem. The destruction of these nerves weakens the muscles supplied by the cranial nerves, producing symptoms of encephalitis, and causes difficulty breathing, speaking and swallowing. Critical nerves affected are the glossopharyngeal nerve, which partially controls swallowing and functions in the throat, tongue movement and taste; the vagus nerve, which sends signals to the heart, intestines, and lungs; and the accessory nerve, which controls upper neck movement. Due to the effect on swallowing, secretions of mucus may build up in the airway, causing suffocation. Other signs and symptoms include facial weakness, caused by destruction of the trigeminal
nerve and facial nerve, which innervate the cheeks, tear ducts, gums, and muscles of the face, among other structures; **double vision; difficulty in chewing; and abnormal respiratory rate, depth, and rhythm**, which may lead to **respiratory arrest. Pulmonary edema and shock** are also possible, and may be fatal.

Approximately 19% of all paralytic polio cases have both bulbar and spinal symptoms; this subtype is called respiratory or **bulbospinal polio**. Here, the virus affects the upper part of the cervical spinal cord (cervical vertebrae C3 through C5), and paralysis of the diaphragm occurs. The critical nerves affected are the phrenic nerve, which drives the diaphragm to inflate the lungs, and those that drive the muscles needed for swallowing. By destroying these nerves, this form of polio affects breathing, making it difficult or impossible for the patient to breathe without the support of a ventilator. It can lead to paralysis of the arms and legs and may also affect swallowing and heart functions.

**Prevention**

The, live, oral polio vaccine (OPV) and inactivated poliovirus vaccine (IPV) are available. Because OPV is inexpensive, easy to administer, and produces excellent immunity in the intestine (which helps prevent infection with wild virus in areas where it is endemic), it has been the vaccine of choice for controlling poliomyelitis in many countries. On very rare occasions (about one case per 750,000 vaccine recipients), the attenuated virus in OPV reverts into a form that can paralyze. Most industrialized countries have switched to IPV, which cannot revert, either as the sole vaccine against poliomyelitis or in combination with oral polio vaccine. Salk vaccine, or inactivated poliovirus vaccine (IPV), is based on poliovirus grown in a type of monkey kidney tissue culture (vero cell line), which is chemically inactivated with formalin. After two doses of IPV (given by injection), 90% or more of individuals develop protective antibody to all three serotypes of poliovirus, and at least 99% are immune to poliovirus following three doses.

**RIFT VALLEY FEVER**

**Transmission and etiology**

![Culex Mosquito](image1.jpg)  ![Aedes aegypti Mosquito](image2.jpg)

Culex Mosquito  Aedes aegypti Mosquito
Rift Valley fever (RVF) is a viral zoonosis that was first identified in Kenya in 1931. This mosquito-borne disease primarily affects animals but that also has the capacity to infect humans. The vast majority of human infections result from direct or indirect contact with the blood or organs of infected animals. Such contact may occur during the care or slaughtering of infected animals or possibly from the ingestion of raw milk. Human infection can also result from the bites of infected mosquitoes. In humans, the virus can cause several syndromes.

Clinical signs and symptoms.

Usually, sufferers have either no symptoms or only a mild illness with fever, headache, myalgia and liver abnormalities. In a small percentage of cases (< 2%), the illness can progress to hemorrhagic fever syndrome, meningoencephalitis (inflammation of the brain), or affecting the eye. Patients who become ill usually experience fever, generalized weakness, back pain, dizziness, and weight loss at the onset of the illness. Typically, patients recover within two to seven days after onset. While most human cases are relatively mild, a small percentage of patients develop a much more severe form of the disease that appears as one or more of three distinct syndromes: ocular disease, meningoencephalitis and viral hemorrhagic fever. RVF in Africa are closely associated with periods of heavy rainfall that occurs during the warm seasons.

Prevention

The vast majority of human infections result from direct or indirect contact with the blood or organs of infected animals. The virus can be transmitted to humans through the handling of animal tissue during slaughtering or butchering, assisting with animal births, conducting veterinary procedures, or from the disposal of carcasses or fetuses. Certain occupational groups such as herders, farmers, slaughterhouse workers and veterinarians are therefore at higher risk of infection. The virus infects humans through inoculation, for example via a wound from an infected knife or through contact with broken skin, or through inhalation of aerosols produced during the slaughter of infected animals. The aerosol mode of transmission has also led to infection in laboratory workers.

There is some evidence that humans may also become infected with RVF by ingesting the unpasteurized or uncooked milk of infected animals.

Human infections have also resulted from the bites of infected mosquitoes, most commonly the Aedes mosquito.
Transmission of RVF virus by hematophagous (blood-feeding) flies is also possible.

To date, no human-to-human transmission of RVF has been documented, and no transmission of RVF to health care workers has been reported when standard infection control precautions have been put in place.

There has been no evidence of outbreaks of RVF in urban areas.

**ACKNOWLEDGE**

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Since our knowledge of potential vaccine efficacy, adverse effects and contraindications changes over the time, the Guide will be reviewed and updated quarterly. Please be aware of date of publication when reading the Guide.

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