INTRODUCTION — Chikungunya is an arthropod-borne virus (arbovirus) endemic to West Africa that causes acute febrile polyarthralgia and arthritis. The name chikungunya is derived from a local language of Tanzania meaning "that which bends up" or "stooped walk" because of the incapacitating arthralgia caused by the disease.

Multiple outbreaks beyond West Africa have been described. Since 2004, chikungunya has spread broadly, causing massive outbreaks with explosive onset in the Indian Ocean region, India, and other parts of Asia [1-3]. Chikungunya had traditionally been perceived as a tropical disease until an outbreak in Italy in 2007. In addition, thousands of cases have been identified in travelers returning from outbreak areas [4]. The distribution of mosquito vectors capable of transmitting chikungunya virus is wide; since late 2013, chikungunya virus infections have spread widely in the Americas [5].

EPIDEMIOLOGY — The United States Centers for Disease Control (CDC) maintains a page with reported current or previous local transmission of chikungunya virus.

Endemic areas — Chikungunya virus is endemic in parts of West Africa, where it appears to be maintained in a cycle involving humans, Aedes mosquitoes, primates, and perhaps other animals [6,7]. Serosurveys of humans in parts of West Africa have identified antibodies to chikungunya virus in 35 to 50 percent of the population in the absence of recognized outbreaks.

Spread and resurgence — Chikungunya virus spreads by means of travel of infected individuals between regions where competent mosquitoes exist for perpetuation of local transmission [8]. Imported cases have been described in many Asian and European countries as well as in the Americas and Australia [9-16]. Rapid spread in the last few years may also be related to a viral mutation that enhances replication efficiency in the Aedes albopictus mosquito. (See 'Transmission' below and 'Mutation of virus and vector replication' below.)

After chikungunya virus was identified during an outbreak in East Africa in Tanzania in the early 1950s, the virus was noted to be the cause of multiple outbreaks in many countries of central, southern, and western Africa. Outside Africa, the first documented chikungunya fever outbreak was in Thailand in 1958. This was followed by outbreaks in multiple other Asian countries, including India, Sri Lanka, Malaysia, Indonesia, Cambodia, Vietnam, Myanmar, the Philippines, and Pakistan.

After a period of relatively little chikungunya activity, large outbreaks were observed in Africa in the late 1990s. In the Democratic Republic of the Congo, for example, an urban outbreak involved an estimated 50,000 people in 1999 to 2000. Epidemics also occurred in Indonesia in 2001 to 2003 after decades without obvious chikungunya fever cases.

Indian Ocean and Asia — Rapid spread of chikungunya virus has been observed in outbreaks involving the Indian Ocean islands, where attack rates have been extremely high in previously unexposed populations. A massive outbreak in Lamu (an island off the coast of Kenya) affected thousands in 2004; a
seroprevalence study suggested that the widespread epidemic may have affected 75 percent of the island’s 18,000 inhabitants [17]. The epidemic on Reunion Island in 2005 to 2006 involved approximately 266,000 individuals (34 percent of the island’s population); a higher attack rate was observed among older adult residents [18]. In the Comoro Islands, a seroprevalence study found 63 percent positive for chikungunya virus antibodies [19].

In India, nearly 1.4 million cases of chikungunya fever were reported in 2006, and outbreaks have continued to occur. The appearance and spread of chikungunya virus in India was observed after a hiatus of almost 32 years [20,21]. Chikungunya virus also reappeared in Malaysia in 2006 [22], in Thailand in 2008, in Singapore in 2008, and caused an outbreak in Guangdong Province, China, in 2010 [23].

Europe — Although chikungunya fever has traditionally been considered a disease of tropical and subtropical regions, in the summer of 2007, chikungunya virus caused an outbreak in northeastern Italy [24]. The index case was a traveler from India who became ill while visiting Italy, and phylogenetic analysis of the virus demonstrated similarity with chikungunya strains found in the Indian Ocean outbreaks.

Between July and September 2007, 205 cases of chikungunya fever were identified in Italy by epidemiologic and clinical criteria; 175 cases were confirmed by laboratory testing. The clinical attack rate in the two affected Italian villages was 5.4 and 2.5 percent but increased with age (1.6 percent for residents <40 years old versus 8.8 percent for those 80 years of age and older) [24]. Cases of chikungunya fever related to local transmission ceased in Italy when temperatures dropped, and human cases were not reported in 2008. Local transmission occurred in France in 2010 [25] and was subsequently reported again in 2014 [26].

Because the Indian Ocean islands, India, and Malaysia are popular tourist destinations among European travelers, many imported cases of chikungunya fever appeared in Europe at the time of the outbreaks in India and the Indian Ocean islands [27].

Americas — In December 2013, chikungunya fever was reported in the Caribbean Island of Saint Martin [3,28]. Since then, local transmission has been confirmed in many countries and territories in the Caribbean, North America, Central America, and South America [28-32]. The majority of cases reported in the continental United States have been imported cases [29,30,33]. The first two cases of local transmission in the continental United States were reported in Florida in mid-July 2014 [31,32,34]; local transmission has been reported more widely in Puerto Rico [33,35]. This is the first time that local transmission of chikungunya virus has been reported in the Americas.

The United States Centers for Disease Control maintains a page summarizing the status of chikungunya transmission in the Caribbean and the Americas.

Pacific region — In 2013 and 2014, outbreaks of chikungunya virus infection spread in the Pacific region on multiple islands, including Tonga, American Samoa, Yap, Papua New Guinea, and others. Concurrent outbreaks of dengue and Zika virus infection have been confirmed on some of the islands [36].

TRANSMISSION — In endemic areas of Africa, chikungunya virus transmission occurs in a cycle involving humans, several species of *Aedes* mosquitoes that inhabit forests and villages, and animals (nonhuman primates and perhaps other animals). In Asia and elsewhere, however, major outbreaks are sustained by mosquito transmission among susceptible humans. The virus can spread via travel of
Infected individuals between regions where competent mosquitoes exist for perpetuation of local transmission. (See 'Epidemiology' above.)

**Mosquito vectors** — The major chikungunya virus mosquito vectors are *Aedes aegypti* and *Aedes albopictus*. These vectors are also capable of transmitting dengue virus, which, like chikungunya virus, is increasing in geographic range and intensity of transmission. (See "Mosquito vectors of infectious diseases" and "Epidemiology of dengue virus infections".)

*Aedes aegypti* — *Ae. aegypti* is well adapted to urban settings and is widely distributed in urban areas of the tropics and sub-tropics. It prefers the human host as a source of blood meals and breeds readily in flowerpots and in trash, such as discarded cups. A single *Ae. aegypti* mosquito may be able to infect more than one human, since this species feeds on another host if its blood meal is interrupted. *Ae. aegypti* is found in the southeastern United States and in limited parts of the Southwest [31].

*Aedes albopictus* — *Ae. albopictus* (the so-called Asian tiger mosquito) is competent to transmit a number of arboviruses in the laboratory (including yellow fever, West Nile, Japanese encephalitis, and eastern equine encephalitis viruses). It bites a range of animal species so has been considered a relatively inefficient vector, since blood meals taken from nonsusceptible hosts do not contribute to virus transmission [37]. However, some populations of *Ae. albopictus* may be more anthropophilic (eg, preferring human blood) than others; in some settings, humans may be the most abundant host [37]. *Ae. albopictus* has become widely dispersed beyond Asia; it is now established in many parts of the Americas, Europe, Africa, and the Pacific Islands [38]. In the United States, *Ae. albopictus* is found in southeastern and mid-Atlantic states as well as in parts of the Southwest, Northeast, and lower Midwest [31]. *Ae. albopictus* has been found as far north as New Jersey, southern New York, and Pennsylvania, but, with climate change, its range is predicted to spread farther north [39].

Modes of mosquito dissemination include transport of mosquito larvae and eggs in used tires by container ships and air traffic with subsequent establishment in new areas with suitable environmental and climatic conditions [40,41]. Outbreaks of chikungunya fever in Italy and Reunion have resulted from virus transmission by *Ae. albopictus* [24,37]. Subsequently simultaneous outbreaks of chikungunya fever and dengue infections have been documented in Central Africa (Gabon) in association with the introduction of the *Aedes albopictus* vector. Dengue-chikungunya coinfections have been documented in humans and in a wild-caught mosquito [42].

**Mosquito competence** — Mosquito susceptibility to infection by chikungunya virus and mosquito competence for virus transmission can vary [43]. Among *Ae. aegypti* and *Ae. albopictus* strains in Florida evaluated for susceptibility to infection by and competence for transmission of a chikungunya virus from the Reunion outbreak, all mosquitoes were susceptible to infection and capable of transmitting the virus [44]. Thus, local mosquitoes possess the capacity to serve as vectors for transmission of chikungunya virus in Florida.

Similarly, *Ae. albopictus* collected in southern France demonstrated high susceptibility to infection with chikungunya virus (77.1 percent), which was comparable to the susceptibility of mosquitoes collected from Reunion [45]. Given the wide distribution of mosquito vectors capable of transmitting chikungunya virus, in the future, transmission could occur in regions with no prior case reports [5].

**Seasonal synchrony** — In order for a viremic traveler to initiate a local outbreak in a nonendemic area, there must be seasonal synchrony between the geographic source of ongoing viral transmission and the
geographic destination vulnerable to introduction of infection (due to infestation with competent mosquito vectors) [46]. For example, transmission of chikungunya virus in Italy during the summer of 2007 was successful because of presence of virus in an individual traveling within the northern hemisphere between India and Italy; the period of active transmission in India coincided with Italy's hottest months. Spread from the southern to northern hemisphere is less likely because the warm seasons and vector activity for these regions may not be synchronous.

Extrinsic incubation period — The extrinsic incubation period is the period between a mosquito blood meal from a viremic host and the transmission of virus to a new host. During this period, the virus must replicate and reach the mosquito salivary glands so that it will be transmitted to a new host when the mosquito takes the next blood meal. The extrinsic incubation period varies depending on the virus, the mosquito vector, and environmental conditions including temperature and humidity. In general, the warmer the temperature, the shorter the extrinsic incubation period and the sooner the mosquito can transmit virus to a new host. In cool temperatures and in many temperate areas, a mosquito may die before the extrinsic incubation period is complete.

The mutation in the chikungunya virus strain that caused the massive outbreak on Reunion (described below) may also be capable of shortening the extrinsic incubation period, allowing more mosquitoes to survive long enough to transmit virus [47]. (See ‘Mutation of virus and vector replication’ below.)

Nosocomial and vertical transmission — Additional modes of transmission include nosocomial and vertical transmission. Nosocomial transmission has been described in France, where a nurse was infected by exposure to blood while caring for a patient infected in Reunion [9,11]. Transmission via transfusion of blood products and/or organ transplantation could also occur, since chikungunya viremia (may exceed 10^9 RNA copies/mL plasma) is likely prior to onset of symptoms [11,48]. Chikungunya virus infects the human cornea and could be transmitted via corneal grafts. Infected corneas have been documented in individuals in the absence of systemic symptoms of chikungunya infection [49].

Vertical transmission of chikungunya has been described in Reunion; among 39 women in the outbreak with viremia at the time of delivery, the rate of vertical transmission was 48.7 percent [50]. Symptoms among neonates were observed within three to seven days and included fever, poor feeding, and rash; 89 percent had thrombocytopenia. Among 19 infected neonates, 10 had severe disease, primarily encephalopathy. Caesarean delivery was not protective against vertical transmission.

There is no evidence of congenital infection in infants who were exposed in utero [51].

VIROLOGY — Chikungunya is a single-stranded RNA virus of the genus *Alphavirus* (Togaviridae family). It was first isolated from mosquitoes and humans during an outbreak in Tanganyika (Tanzania) in 1952 to 1953 [52]. Thus far, three lineages distinguishable by genotypic and antigenic characteristics have been identified: the clusters of Central and East Africa, West Africa, and Asia.

Other alphaviruses that cause illnesses associated with fever and arthralgia or arthritis include O’nyong-nyong (East and Central Africa), Barmah and Ross River viruses (Australia and the Pacific), Semliki virus (Africa), Mayaro virus (South America), and Sindbis group viruses (widespread excluding the Americas) [27,53]. (See "Specific viruses that cause arthritis".)

The pathogenesis of the severe and persistent joint symptoms that characterize chikungunya virus infections is uncertain. Some data suggest that macrophage-derived products such as tumor necrosis
factor-alpha, interferon-gamma, and macrophage chemoattractant protein-1 may play important roles in the joint tissue damage [54].

**Mutation of virus and vector replication** — The replication efficiency of chikungunya virus within the *Ae. albopictus* mosquito may be enhanced in the presence of a mutation in the viral gene encoding the envelope protein of the virus (mutation A226V). This mutation was observed in >90 percent of viral sequences in the latter period of the Reunion outbreak, although it was not observed in the initial outbreak strains, suggesting an enhanced survival benefit for this mutant (A226V) [55]. The mutation was also found in chikungunya virus isolates from the outbreak in Italy [56].

The presence of this mutation has been associated with enhanced *Ae. albopictus* susceptibility to infection and more rapid viral dissemination into the mosquito salivary glands [47]. As a result, a lower level of human viremia is required for transmission of this mutant virus to a biting mosquito, facilitating the cycle of infection.

**CLINICAL MANIFESTATIONS**

**Acute infection** — Clinical signs and symptoms begin abruptly with fever and malaise following an incubation period of two to four days (range 1 to 14) [57].

Fever may be high grade (40°C); the usual duration of fever is three to five days (range 1 to 10 days). Polyarthralgia begins two to five days after onset of fever and commonly involves multiple joints (often 10 or more joint groups) [9,21,58,59]. Joints affected include hands (50 to 76 percent), wrists (29 to 81 percent), and ankles (41 to 68 percent). Arthralgia is symmetrical in 64 to 73 percent and involves distal joints more than proximal joints. Involvement of the axial skeleton was noted in 34 to 52 percent of cases. Pain may be intense and disabling, leading to immobilization.

Skin manifestations have been reported in 40 to 75 percent of patients [21,59]. The most common skin manifestation is macular or maculopapular rash (usually appearing three days or later after onset of illness and lasting three to seven days). The rash often starts on the limbs and trunk, can involve the face, and may be patchy or diffuse. Islands of normal skin may be seen along with the diffuse rash. Pruritus has been reported in 25 to 50 percent of patients in some series. Bullous skin lesions have also been described, most often in children. Hemorrhagic manifestations are uncommon.

Additional manifestations may include headache, myalgia, and gastrointestinal symptoms.

On physical examination, periarticular edema or swelling has been observed in 32 to 95 percent of cases. In one series, large joint effusions were noted in 15 percent of cases. Peripheral lymphadenopathy (most often cervical) may be present (9 to 41 percent of cases) [10,60]. Conjunctivitis may be observed [61]. The most common laboratory abnormalities are lymphopenia and thrombocytopenia. Liver enzymes may be elevated.

**Persistent symptoms** — Following acute illness (usually lasting 7 to 10 days), some patients may experience persistent signs and symptoms including arthritis/arthralgia, edematous polyarthritis of fingers and toes, morning pain and stiffness, and severe tenosynovitis (especially of wrists, hands, and ankles) [62]. Carpal tunnel syndromes may result from hypertrophic tenosynovitis. In addition, patients may report joint or bone pain at sites of previous injury. Occasionally, unusual joints (such as sternoclavicular or temporomandibular joints) are involved. New onset Raynaud phenomena in the second or third month following infection have been described in up to 20 percent of cases [58]. Cryoglobulinemia has also been
found in patients with persistent symptoms attributed to chikungunya infection, >90 percent in one series [63].

Chronic symptoms usually involve joints affected during the acute illness and can be relapsing or unremitting and incapacitating. Study with detailed analysis found arthralgias were typically polyarthralgia [64]; 63 percent had associated local swelling.

The duration of persistent symptoms is variable. Among 47 patients with acute chikungunya fever followed in Marseilles, France, 82 percent had persistent joint symptoms. At one, three, and six months following acute illness, symptoms persisted in 88, 86, and 48 percent of patients, respectively; at 15 months, 4 percent remained symptomatic [58]. In contrast, among 88 patients in Reunion evaluated a mean of 18 months after confirmation of acute chikungunya infection, 63 percent reported persistent polyarthralgia [60]. Morning stiffness was reported by 75 percent of individuals, and almost half reported that the pain had a negative impact on daily activities. Another study of 180 patients from Reunion with viremic chikungunya infection found that at 36 months 60 percent still had arthralgias [64]. One study in South Africa reported arthralgia three years after the acute illness in 12 percent of patients [65].

**Severe complications** — In older reports, chikungunya fever has been described as a self-limited illness, although severe complications and death have been reported in the more recent outbreaks. It is unclear whether these differences reflect a modulation in virus virulence, improved epidemiologic observation, or both. Severe complications and death occur more often among patients older than 65 years and in those with underlying chronic medical problems.

Severe complications include respiratory failure, cardiovascular decompensation, myocarditis, acute hepatitis, renal failure, and neurologic involvement. Meningoencephalitis is the most common neurologic complication; other manifestations include acute flaccid paralysis and Guillain Barré syndrome [66-68]. Ocular manifestations (iritis, retinitis, episcleritis, macular choroiditis) and sensorineural hearing loss have also been described [61,69,70]. In Reunion, the estimated incidence of severe disease (eg, hospitalized patients with complications, such as respiratory failure, meningoencephalitis, acute hepatitis, or kidney failure) was 17 per 100,000 population [18,71].

Deaths associated with chikungunya virus infection were reported during outbreaks in Mauritius, Reunion, and India [71-74]. In Reunion there were 228 deaths; the mean age was 78 years [71]. During the chikungunya epidemic in Ahmedabad, India, in 2006, about 60,000 cases were described; the number of deaths during the four months of peak epidemic activity exceeded the average death rate during those months in the previous four years by almost 3000 [74].

**Subclinical infection** — Some individuals have serologic evidence for exposure to chikungunya virus infection in the absence of clinical symptoms. As noted above, in West Africa seropositivity has been noted in 35 to 50 percent of the individuals in the absence of recognized outbreaks. During the Reunion outbreak, a survey found that about 3.2 percent of military policemen were seropositive in the absence of clinical symptoms [75]. A Thai study noted a ratio of clinical-to-subclinical chikungunya infection of about 1.8:1 [76].

**DIFFERENTIAL DIAGNOSIS** — Prominent arthralgia, high fever, diffuse rash, and absence of respiratory symptoms can help to distinguish chikungunya from other illnesses. The differential diagnosis includes:

- Dengue fever – Dengue virus and chikungunya infections share some clinical symptoms and areas of geographic distribution; distinguishing them may be difficult in the setting of acute febrile illness
with rash. However, polyarthralgia occurs in virtually all cases of chikungunya fever but is not typical of dengue fever [77]. Cytopenia, particularly thrombocytopenia, may distinguish dengue from chikungunya infection. The diagnosis is established via serology. (See "Clinical manifestations and diagnosis of dengue virus infection").

- Seronegative rheumatoid arthritis – Clinical manifestations of seronegative rheumatoid arthritis include inflammatory arthritis involving three or more joints for >6 weeks, with negative rheumatoid factor and anti–cyclic citrullinated peptide (CCP) antibody tests. It is a diagnosis of exclusion. (See "Diagnosis and differential diagnosis of rheumatoid arthritis", section on 'Patients not meeting above criteria'.)

- African tick bite fever – African tick bite fever occurs as a result of infection with Rickettsia africae and is observed among travelers to Africa [78]. It is associated with headache, fever, myalgia, solitary or multiple eschars with regional lymphadenopathy, and generalized rash. The diagnosis is established via serology. (See "Other spotted fever group rickettsial infections").

- Enteric fever – Clinical manifestations of enteric fever include fever, bradycardia, abdominal pain, and, infrequently, a rash (rose spots). The presentation of enteric fever is typically subacute, whereas the presentation of chikungunya infection is typically abrupt in onset. The diagnosis is established by stool and/or blood culture. (See "Epidemiology, microbiology, clinical manifestations, and diagnosis of typhoid fever").

- Leptospirosis – Leptospirosis is characterized by fever, rigors, myalgia, and headache. Less common symptoms include cough, nausea, vomiting, diarrhea, abdominal pain, and arthralgia. It may be distinguished from chikungunya infection by the presence of conjunctival suffusion and jaundice. The diagnosis is established via serology. (See "Epidemiology, microbiology, clinical manifestations, and diagnosis of leptospirosis").

- Malaria – Malaria is characterized by fever, malaise, nausea, vomiting, abdominal pain, diarrhea, myalgia, and anemia. Fever in the setting of malaria is often intermittent, whereas fever in the setting of chikungunya infection is typically persistent. The diagnosis of malaria is established by visualization of parasites on peripheral smear. (See "Clinical manifestations of malaria").

- Relapsing fever – Clinical manifestations of relapsing fever include fever, headache, neck stiffness, arthralgia, myalgia, and nausea. Fever in the setting of relapsing fever is typically intermittent, whereas fever in the setting of chikungunya infection is typically persistent. Diagnostic tools include direct smear and polymerase chain reaction (PCR). (See "Clinical features, diagnosis, and management of relapsing fever").

- Ross river virus – Clinical manifestations of Ross River virus infection include fever, arthritis, and rash. Epidemiologic history can help to exclude Ross River virus infection as it is transmitted only in Australia. The diagnosis of Ross River virus is typically established by serology. (See "Ross River virus infection").

- Measles – Clinical manifestations of measles include fever, cough, sore throat, coryza, conjunctivitis, and lymphadenitis. Koplik spots may precede generalized rash. The diagnosis is established via serology. (See "Clinical manifestations and diagnosis of measles").

- Rubella – Clinical manifestations of rubella include low-grade fever, coryza, conjunctivitis, and lymphadenopathy. Macular rash begins on the face and spreads to the trunk, and arthritis may be present. The diagnosis is established via serology. (See "Rubella").
Epstein-Barr virus – Clinical manifestations of mononucleosis include fever, malaise, and pharyngitis. Lymphadenopathy and splenomegaly may be present along with atypical lymphocytosis. The diagnosis is established via detection of heterophile antibodies.

Meningococcal infection – Meningococcal infection may be associated with meningitis and hemorrhagic rash. The diagnosis is established based on cerebrospinal fluid examination. (See "Clinical manifestations of meningococcal infection".)

Other infections in the differential diagnosis include primary HIV infection and rickettsial infections. The possibility of dual infection should be considered if the clinical course is atypical or fever persists longer than five to seven days [79]. Chikungunya virus outbreaks have occurred simultaneously with outbreaks of dengue, Zika virus [36], or yellow fever [80], and coinfection with chikungunya virus and other pathogens has been described (eg, chikungunya and dengue [81], chikungunya and yellow fever [79], chikungunya and amebiasis [82]).

**DIAGNOSIS** — Diagnostic techniques for identification of chikungunya virus include serology, viral culture, and molecular techniques [11,21,53]. Serology is the primary tool for diagnosis in the clinical setting. Immunoglobulin M (IgM) anti-chikungunya virus antibodies (detected by direct enzyme-linked immunosorbent assay [ELISA]) are present starting about five days (range 1 to 12 days) following onset of symptoms and persist for several weeks to three months. Immunoglobulin G (IgG) antibodies begin to appear about two weeks following onset of symptoms and persist for years.

In endemic areas, chikungunya infection can be suspected based on characteristic clinical findings in outbreaks; in endemic areas where good laboratory facilities are unavailable, many infections may remain undiagnosed.

Viral culture and molecular techniques are important research tools. Chikungunya virus can be isolated from blood during the first week of illness using mosquito cells, mammalian cells (Vero), or in mice. Sensitivity of viral culture is high in early infection but drops five days after onset of illness. Chikungunya virus RNA can also be detected by reverse transcription polymerase chain reaction (RT-PCR) during the first five days following onset of symptoms with excellent sensitivity and specificity [11]. Virus isolation allows identification of the viral strain and can be important for epidemiologic and research purposes, but RT-PCR is faster than culture with greater sensitivity and specificity.

**TREATMENT AND PREVENTION** — Treatment of chikungunya infection consists of supportive care including antiinflammatory agents that relieve symptoms in many patients and analgesic agents [58]. No antiviral agents have been shown to be effective in human infection, although ribavirin and interferon-alpha appear to have in vitro activity against virus replication [83]. Chloroquine sulfate has been suggested as a possible treatment because of its antiinflammatory properties but has not been demonstrated to be effective [84]. For seriously ill patients, there are insufficient data for use of corticosteroids or antiviral therapy. The documentation of long-term viral persistence in nonhuman primates raises questions about the roles immune dysregulation and persistent virus in chronic symptoms [85].

Thus far, there is no licensed vaccine for prevention of chikungunya infection; active research is under way to develop a vaccine using variety of approaches [86-90]. Active work is also underway to develop monoclonal antibodies for treatment [91].

Prevention consists of minimizing mosquito exposure [92]. Patients receiving care in an area inhabited by mosquitoes competent to transmit chikungunya virus should be treated in screened, mosquito-free areas.
or under a bednet to avoid spread. (See "Prevention of arthropod and insect bites: Repellents and other measures").

SUMMARY AND RECOMMENDATIONS

● Chikungunya is an arthropod-borne virus (arbovirus) endemic to West Africa that causes acute febrile polyarthralgia and arthritis. (See 'Introduction' above.)

● Chikungunya virus appears to spread across wide geographic areas via travel of infected individuals between regions where competent mosquitoes exist for perpetuation of local transmission. Multiple outbreaks beyond West Africa have been described; since 2004, chikungunya virus has spread broadly, causing massive outbreaks with explosive onset in the Indian Ocean region, India, other parts of Asia, Europe, and, most recently, the Americas. (See 'Epidemiology' above.)

● Clinical manifestations of acute infection include high fever and bilateral polyarthralgia with intense pain. The most common skin manifestation is macular or maculopapular rash (usually appearing three days or later after onset of illness and lasting three to seven days). Additional manifestations may include headache, myalgia, and gastrointestinal symptoms. (See 'Acute infection' above.)

● Many patients experience persistent rheumatologic symptoms following acute illness. These may include polyarthralgia, morning stiffness, tenosynovitis, and Raynaud phenomena. Severe complications (including meningoencephalitis, cardiopulmonary decompensation, acute renal failure, and death) have been described with greater frequency among patients older than 65 years and those with underlying chronic medical problems. (See 'Persistent symptoms' above and 'Severe complications' above.)

● Serology is the primary tool for diagnosis in the clinical setting. Immunoglobulin M (IgM) anti-chikungunya virus antibodies are present starting about five days (range 1 to 12 days) following onset of symptoms and persist for several weeks to three months. Immunoglobulin G (IgG) antibodies start to appear about two weeks following onset of symptoms and persist for years. (See 'Diagnosis' above.)

● Treatment of chikungunya infection consists of supportive care including antiinflammatory and analgesic agents. No antiviral agents have been shown to be effective in human infection. Prevention consists of minimizing mosquito exposure. Patients receiving care in an area inhabited by mosquitoes competent to transmit chikungunya should be treated in screened, mosquito-free areas or under a bednet to avoid spread. (See 'Treatment and prevention' above.)