

Mosquito borne zoonotic diseases: A review

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Abstract

Mosquito borne zoonotic diseases cause huge economic loss in the human and animal population due to expenses in cost of treatment and vaccination. Major problem in these diseases resides on the control of mosquito population. This insect is becoming resistant day by day to the insecticide. This review covers geographical distribution, transmission cycle, symptoms in human, diagnosis and control of the major zoonotic diseases caused by mosquito such as Japanese encephalitis, dengue fever, chikungunya, yellow fever, west Nile fever, and Rift valley fever. These diseases are caused by different species of *Culex* and *Aedes* mosquito. Frequent change in weather condition, adapting new farm practices and irregular use of several insecticide caused the mosquito population to flare up and become resistant from all adverse condition and spreading these disease to new non endemic areas.

Keywords: Mosquito, Japanese encephalitis, Immunity, Dengue virus

Introduction

Mosquitoes are responsible for great vector borne diseases burden such as Japanese encephalitis, Dengue, Chikungunya, Yellow fever, West Nile fever and Rift Valley fever. Over 1 million people die from mosquito-borne diseases every year, and hundreds of millions more experience pain and suffering from illnesses transmitted by mosquitoes. Rift Valley fever, yellow fever causes negative impact on commerce, travel and economies. Yellow fever also causes explosive debilitating outbreaks. Human impact on environment can increase the incidence of Japanese encephalitis. Dengue Fever is the fastest-growing mosquito-borne disease. It is a serious and sometimes fatal condition which affects over 50 million people every year. Chikungunya is a rapidly emerging viral disease that is also spread by the *Aedes* mosquitoes. According to the Centre for Disease Control and Prevention (CDC) symptoms of chikungunya infection include fever, headache, fatigue, nausea, vomiting, muscle pain, rash and joint pain. In the present review we summarized the mosquito borne diseases of zoonotic importance.

Dengue fever

Dengue is common in more than 110 countries (Ranjit and Kissoon, 2011). It infects 50 to 528 million people worldwide a year, leading to half a million hospitalizations (Whitehorn and Farrar, 2010; Bhatt and Gething, 2013) and approximately 25,000 deaths (Varatharaj, 2010). For the decade of the 2000s, 12 countries in Southeast Asia were estimated to have about 3 million infections and 6,000 deaths annually (Shepard et al., 2013). It is reported in at least 22 countries in Africa; but is likely present in all of them with 20% of the population at risk (Amarsinghe et al., 2011). This makes it one of the most common vector borne diseases worldwide (Yacoub and Willes, 2014). Dengue fever, which was once confined to Southeast Asia, has now spread to Southern China, countries in the Pacific Ocean and America (Gubler, 2010) and might pose a threat to Europe (Reiter, 2010). Rates of dengue increased 30 fold between 1960 and 2010 (WHO, 2009). This increase is believed to be due to a combination of urbanization, population growth, increased international travel, and global warming (Whitehorn and Farrar, 2010). Dengue virus is primarily transmitted by *Aedes* mosquitoes, particularly *A. aegypti*. Other *Aedes* species that transmit the disease include *A. albopictus*, *A. polynesiensis* and *A. scutellaris*. Humans are the primary host of the virus (WHO, 2009; Gould and Solomon, 2008) but it also circulates in nonhuman primates (WHO, 2011). An infection can be acquired via a single bite (CDC, 2010). The characteristic symptoms of dengue are sudden-onset fever, headache (typically located behind the eyes), muscle and joint pains, and a rash. The alternative name for dengue, breakbone fever, comes from the associated muscle and joint pains (Whitehorn and Farrar, 2010; Chen and Wilson, 2010). The febrile phase involves high fever, potentially over 40 °C (104 °F), and is associated with generalized pain and a headache; this usually lasts two to seven days. Nausea and vomiting may also occur (Simmons et al., 2012). A rash occurs in 50–80% of those with symptoms (Chen and Wilson, 2010; Wolff and Johnson, 2009). Shock (dengue shock syndrome) and haemorrhage (dengue hemorrhagic fever) occur in less than 5% of all cases of dengue, however those who have previously been infected with other serotypes of dengue virus ("secondary infection") are at an increased risk (Ranjit and Kissoon, 2011; Rodenhuis-Zybert et al., 2010). Severe disease is marked by the problems of capillary permeability and disordered blood clotting (Varatharaj, 2010; Simmons et al., 2012). These changes appear associated with a disordered state of the endothelial glycocalyx, which acts as a molecular filter of blood components (Simmons et al., 2012). A probable diagnosis is based on the findings of fever plus two of the following: nausea and vomiting, rash, generalized pains, low white blood cell count, positive tourniquet test, or any warning sign in someone who lives in an endemic area. The earliest change detectable on laboratory investigations is a low white blood cell count, which may then be followed by low platelets and metabolic acidosis (Ranjit and Kissoon, 2011). Dengue shock syndrome is present if pulse pressure drops to ≤ 20 mm Hg along with peripheral vascular collapse (Simmons et al., 2012). Peripheral vascular collapse is determined in children via delayed capillary refill, rapid heart rate, or cold extremities. While warning signs are an important aspect for early detection of potential serious disease, the evidence for any specific clinical or laboratory marker is weak (Yacoub and Willes, 2014). There are no approved vaccines for the dengue virus (Whitehorn and Farrar, 2010). Prevention thus depends on control of and protection from the bites of the mosquito that transmits it. Generalized spraying with organophosphate or pyrethroid insecticides, while sometimes done, is not thought to be effective (Reiter, 2010). Reducing open collections of water through environmental modification is the preferred method of control, given the concerns of negative health effects from insecticides and greater logistical difficulties with control agents (WHO, 2009). People can prevent mosquito bites by wearing clothing that fully covers the skin, using mosquito netting while resting, and/or the application of insect repellent (DEET being the most effective (CDC, 2010).

Chikungunya

Three genotypes of this virus have been described: West African, East/Central/South African, and Asian genotypes (Powers et al., 2000). Explosive epidemics in Indian Ocean in 2005 and Pacific Islands in 2011, as well as now in the Americas, continue to change the distribution of genotypes. The disease was first described by Marion Robinson (Robinson, 1955) and W.H.R. Lumsden (Lumsden, 1955) in 1955, following an outbreak in 1952 on the Makonde Plateau, along the

border between Mozambique and Tanganyika. The first recorded outbreak of this disease may have been in 1779 (Carey, 1971). This is in agreement with the molecular genetics evidence that suggests it evolved around the year 1700 (Cherian et al., 2009). The virus is passed to humans by two species of mosquito of the genus *Aedes*: *A. albopictus* and *A. aegypti* (Lahariya and Pradhan, 2006; Staples and Fisher, 2014). Animal reservoirs of the virus include monkeys, birds, cattle, and rodents (Schwarz et al., 2012). This is in contrast to dengue, for which only primates are hosts (WHO, 2014). It features the sudden onset of fever usually lasting two to seven days, and joint pains typically lasting weeks or months but sometimes years (Powers and Logue, 2007; Sourisseau et al., 2007; Schilte et al., 2013). Common predictors of prolonged symptoms are increased age and prior rheumatological disease (Schilte et al., 2013; Gerardin et al., 2013; Moro et al., 2012; Sissoko et al., 2009). The cause of these chronic symptoms is currently not fully known. Markers of autoimmune or rheumatoid disease have not been found in people reporting chronic symptoms (Schilte et al., 2013; Manimunda et al., 2010). However, some evidence from humans and animal models suggests chikungunya may be able to establish chronic infections within the host. Viral antigen was detected in a muscle biopsy of a person suffering a recurrent episode of disease three months after initial onset (Ozden et al., 2007). Chikungunya virus is an alphavirus with a positive-sense single-stranded RNA genome of about 11.6kb. It is a member of the Semliki Forest virus complex and is closely related to Ross River virus, O'nyong'nyong virus, and Semliki Forest virus (Powers et al., 2001). Virus isolation provides the most definitive diagnosis, but takes one to two weeks for completion and must be carried out in biosafety level III laboratories (WHO, 2013). Chronic recurrent poly arthralgia occurs in at least 20% of chikungunya patients one year after infection, whereas such symptoms are uncommon in dengue (Morens and Fauci, 2014). The most effective means of prevention are protection against contact with the disease carrying mosquitoes and mosquito control (Caglioti et al., 2013). Currently, no approved vaccines are available. A phase-II vaccine trial used a live, attenuated virus, to develop viral resistance in 98% of those tested after 28 days and 85% still showed resistance after one year (Edelman et al., 2000). However, 8% of people reported transient joint pain, and attenuation was found to be due to only two mutations in the E2 glycoprotein (Gorchakov et al., 2012). A phase-II vaccine trial used a live, attenuated virus, to develop viral resistance in 98% of those tested after 28 days and 85% still showed resistance after one year (Edelman et al., 2000). However, 8% of people reported transient joint pain, and attenuation was found to be due to only two mutations in the E2 glycoprotein (Gorchakov et al., 2012).

Japanese encephalitis

Countries with the proven epidemics of Japanese encephalitis (JE) are India, Pakistan, Nepal, Sri Lanka, Burma, Laos, Vietnam, Malaysia, Singapore, Philippines, Indonesia, China, maritime Siberia, Korea, and Japan (Vaughn and Hoke, 1992). Since 1990s the virus has continued to spread in Pakistan (Igarashi et al., 1994) Nepal (Zimmerman et al., 1997) and Australia (Hanna et al., 1996). First clinical case of JE in India was observed in 1955 at Vellore (former North Arcot district, Tamil Nadu) (Namachivayam and Umayal, 1982). A total of about 65 cases were reported between 1955 and 1966 in South-India (Carey et al., 1968). In 1973, the first major outbreak occurred in Burdwan and Bankura, the two districts of West Bengal with about 700 cases and 300 deaths (Chakravarty et al., 1975). Subsequently, another outbreak in the same state occurred in 1976 with 307 cases and 126 deaths (Vaughn and Hoke, 1992). In these natural amplifying hosts, however; virus does not produce encephalitis, although abortion occurs in pregnant sows (Guerin and Pozzi, 2005). Virus is transmitted to humans by the bite of infected mosquito, which serves as a dead end host due to its short duration and low viremia in man (Scherer et al., 1959). Most important mosquito vector in Asia is *Culex tritaeniorhynchus* which breeds in stagnant water like paddy fields or drainage ditches (Innis, 1955). Other species are *Culex vishnui* (India), *C. gelidus*, *C. fuscocephala* (India, Malaysia, Thailand), *C. pipiens*. About 50-60% of the survivors suffer from serious long-term neurologic sequelae manifested as convulsions, tremors, paralysis, ataxia, memory loss, impaired cognition, behavioural disturbance and other such symptoms (Halstead and Jacobson, 2003). There is an incubation period of 4-14 days in humans during JEV infection and patients are presented with few days of fever including coryza, diarrhea and rigors (Soloman, 1997). Convulsions occur more frequently in children in upto 85% cases than in adult patients i.e. 10% (Kumar et al., 1990). Vector control alone cannot be relied upon to prevent JE since it is practically almost impossible to control mosquito density in the rural areas which are the worst affected areas due to poor socio-economic conditions (Tiroumourogane et al., 2002). Three types of JE vaccine are currently in use: mouse-brain derived inactivated, cell-culture derived inactivated and cell-culture derived live attenuated JE vaccine. Formalin inactivated vaccines are the safe and effective against JEV for at least 30 years (Tsai et al., 1999).

Yellow fever

Yellow fever is common in tropical and subtropical areas of South America and Africa. Although yellow fever is most prevalent in tropical-like climates, the northern United States was not exempted from the fever. The first outbreak in English-speaking North America occurred in New York in 1668, and a serious one afflicted Philadelphia in 1793 (Miller and Jacquelyn, 2005). The first definitive outbreak of yellow fever in the New World was in 1647 on the island of Barbados (McNeill, 2004). Transovarial and transstadial transmission of the yellow fever virus within *A. aegypti*, that is, the transmission from a female mosquito to her eggs and then larvae, are indicated. This infection of vectors without a previous blood meal seems to play a role in single, sudden breakouts of the disease (Fontenille et al., 1997). Three epidemiologically

different infectious cycles occur (Barrett and Higgs, 2007) in which the virus is transmitted from mosquitoes to humans or other primates. In the "urban cycle", only the yellow fever mosquito *A. aegypti* is involved. It is well adapted to urban areas and can also transmit other diseases, including dengue fever and chikungunya. Besides the urban cycle, both in Africa and South America, a sylvatic cycle (forest cycle or jungle cycle) is present, where *Aedes africanus* (in Africa) or mosquitoes of the genus *Haemagogus* and *Sabethes* (in South America) serve as vectors. Yellow fever begins after an incubation period of three to six days (CDC, 2010). Most cases only cause a mild infection with fever, headache, chills, back pain, fatigue, loss of appetite, muscle pain, nausea, and vomiting (WHO, 2009). In severe epidemics, the mortality may exceed 50%. (Tomori, 2004). Surviving the infection provides lifelong immunity (Modrow, 2002), and normally there is no permanent organ damage (Rogers et al., 2006). Yellow fever is caused by the yellow fever virus, a 40- to 50-nm-wide enveloped RNA virus, the type species and namesake of the family Flaviviridae (Lindenbach, 2007). If yellow fever is suspected, the virus cannot be confirmed until six to 10 days after the illness. A direct confirmation can be obtained by reverse transcription polymerase chain reaction where the genome of the virus is amplified (Tolle, 2009). Vaccination is recommended for those traveling to affected areas, because non-native people tend to suffer more severe illness when infected. Protection begins by the 10th day after vaccine administration in 95% of people (Barrett and Teuwen, 2009), and lasts for at least 10 years. About 81% of people are still immune after 30 years. The attenuated live vaccine stem 17D was developed in 1937 by Max Theiler (Barrett and Teuwen, 2009). In 2009, the largest mass vaccination against yellow fever began in West Africa, specifically Benin, Liberia, and Sierra Leone. When it is completed in 2015, more than 12 million people will have been vaccinated against the disease (BBC News, 2009). Control of the yellow fever mosquito *A. aegypti* is of major importance, especially because the same mosquito can also transmit dengue fever and chikungunya disease. A list of the countries that require yellow fever vaccination is published by the WHO (WHO, 2013). If the vaccination cannot be conducted for some reasons, dispensation may be possible. In this case, an exemption certificate issued by a WHO-approved vaccination center is required. Although 32 of 44 countries where yellow fever occurs endemically do have vaccination programmes, in many of these countries, less than 50% of their population is vaccinated. Insecticide-treated mosquito nets are effective, just as they are against the *Anopheles* mosquito that carries malaria (Barrett and Teuwen, 2009).

Rift valley fever

The disease was first reported among livestock in Rift Valley of Kenya in the early 1900s (Palmer, 2011), and the virus was first isolated in 1931. Outbreaks usually occur during periods of increased rain which increase the number of mosquitoes (WHO, 2010). The virus is transmitted through mosquito vectors, as well as through contact with the tissue of infected animals. Two species—*Culex tritaeniorhynchus* and *Aedes vexans*—are known to transmit the virus (Jup et al., 2002). The mild symptoms may include: fever, muscle pains, and headaches which often last for up to a week. The severe symptoms may include: loss of the ability to see beginning three weeks after the infection, infections of the brain causes severe headaches and confusion, and bleeding together with liver problems which may occur within the first few days. Those who have bleeding have a chance of death as high as 50%. Diagnosis relies on viral isolation from tissues, or serological testing with an ELISA (Dessau and Modis, 2013). Once infected there is no specific treatment.

West Nile fever

WNV was first isolated from a feverish 37-year-old woman at Omogo in the West Nile District of Uganda in 1937 during research on yellow fever virus (Smithburn et al., 1940). The first appearance of WNV in the Western Hemisphere was in 1999 (Nash et al., 2001). West Nile virus is now endemic in Africa, Europe, the Middle East, west and central Asia, Oceania (subtype Kunjin), and most recently, North America and is spreading into Central and South America. The mosquito species that are most frequently infected with WNV feed primarily on birds (Kilpatrick, 2011). The important mosquito vectors vary according to geographical area; in the United States, *Culex pipiens* (Eastern United States, and urban and residential areas of the United States north of 36-39°N), *Culex tarsalis* (Midwest and West), and *Culex quinquefasciatus* (Southeast) are the main vector species (Hayes et al., 2005). Approximately 80% of West Nile virus infections in humans are subclinical, which cause no symptoms. In the cases where symptoms do occur—termed West Nile fever in cases without neurological disease—the time from infection to the appearance of symptoms (incubation period) is typically between 2 and 15 days. Symptoms may include fever, headaches, fatigue, muscle pain or aches (myalgias), malaise, nausea, anorexia, vomiting, and rash. Less than 1% of the cases are severe and result in neurological disease when the central nervous system is affected. Risk factors independently associated with developing a clinical infection with WNV include a suppressed immune system and a patient history of organ transplantation. Definitive diagnosis of WNV is obtained through detection of virus-specific antibody IgM and neutralizing antibodies. Cases of West Nile virus meningitis and encephalitis that have been serologically confirmed produce similar degrees of CSF pleocytosis and are often associated with substantial CSF neutrophilia. Specimens collected within eight days following onset of illness may not test positive for West Nile IgM, and testing should be repeated. A positive test for West Nile IgG in the absence of a positive West Nile IgM is indicative of a previous flavivirus infection and is not by itself evidence of an acute West Nile virus infection.

There should be continuous monitoring of the mosquito borne diseases. Dengue and chikungunya are transmitted by the *Aedes* mosquito which used to be measured an urban mosquito but Chikungunya is common today in both rural and urban areas today. *Aedes* can breed in clean as well as fairly polluted water, has an average flight range of 100 metres, bites in the daytime, and chiefly human beings. Thus it breeds in and around human habitations, in water bodies such as storage tanks, desert coolers, even septic tanks. *Aedes* eggs can survive long periods of drying and dormant eggs inside old tires are known to have been transported over great distances. In short, human activities add greatly to the survival and spread of *Aedes* mosquitoes. The only way to put a stop to dengue and Chikungunya is for people to know where the mosquito is breeding in each house and locality, and take individual and community action to eradicate it. Mosquito repellents are useful but beyond the reach of the poor. Space spraying to kill adult mosquitoes should be used carefully, in exceptional cases.

Authors' Contributions

N Singh and P Singh collected information and prepared the initial version of the manuscript. S Shukla, V Gupta and N Tandia drafted and revised the manuscript. All the authors read and approved the final manuscript.

Competing Interests

The authors declare that they have no competing interests.

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