Review Article

Emerging Viral Diseases

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Abstract:

Human life is intricately related to it's surrounding environment which also harbors other animals and some deadly infectious pathogens. Any threat to the environment can thus increase the threat of new and so-called 'emerging infectious diseases' (EIDs) especially novel viral infections called 'emerging viral diseases'. This occurs partly due to changing climate as well as human interference with nature and animal life. An important event in new disease emergence is genetic changes in the pathogen that make it possible to become established in a new host species, productively infect new individuals in the new hosts (typically humans) and create local, regional or worldwide health threats. The world has witnessed some emerging and deadly viral threats in recent past with huge mortality and morbidity. Among them were severe acute respiratory syndrome (SARS), bird flu, swine flu, Middle East respiratory syndrome (MERS), ebola virus disease. Moreover some disease has caused great concern in certain regions including Bangladesh in terms of morbidity, like Nipah virus, Zika virus, Dengue and Chikungunya fever. Here in this article an attempt was made to briefly describe some of these emerging viral infections.

Key Words: Emerging virus

(BIRDEM Med J 2017; 7(3): 224-232)

Introduction

The 20th century has seen significant reductions in ecosystems and biodiversity and equally dramatic increases in the numbers of people and domestic animals inhabiting the Earth.[1] In fact, continued inadvertent human activities like land use changes, population growth, increased contacts with wild animal reservoirs and the degradation of health care resources has increased the opportunity for various pathogenic agents including some deadly viruses to pass from the wild and domestic animals to human beings causing emergence of new diseases.[2] Emerging viral diseases are nothing new. Smallpox probably reached Europe from Asia in the 5th century, and yellow fever emerged in the Americas during the 16th century as a consequence of the African slave trade. Dengue fever arose simultaneously in South-East Asia, Africa, and North America during the 18th century. In 1918-1919 the so-called Spanish flu spread like wildfire through all five continents, killing between 25 and 40 million people. The second half of the 20th century saw the emergence of HIV/AIDS (1981), among other viral diseases.[3] Even more worrying is the fact that emerging and re-emerging viral diseases have had a tendency to spread more quickly and more widely during the last decade, invading whole countries and continents. It is estimated that the majority, some estimates place it as high as 75%, of these emerging diseases are derived from animals.[4] These zoonoses spill over from their natural reservoirs either through direct contact or indirectly through close contact with domestic animals and subsequently into human populations. We focus on few viral pathogens and their diseases that have received increased attention during the recent years, namely severe acute respiratory syndrome (SARS), bird flu, swine flu, Ebola virus disease, Zika and Chikungunya fever. We briefly outline some of their basic biology, epidemiology, clinical presentation and management including prevention.

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Received: June 6, 2017 **Accepted:** July 31, 2017

Table. Recent Deadly Viral Outbreaks in the world[5]

Disease	Virus	Year	Country/ region of disease outbreak	Mortality
Chikungunya Fever	Chikungunya Virus (CHIKV)	2017	Bangladesh	0.1%
Zika Virus Infection	Zika Virus (ZKV)	2014	Brazil	Nil
Ebola Virus Disease (EVD)	Ebola Virus	2013	Guinea, West Africa	40%
Avian Influenza/ Bird Flu	H7N9 Influenza A	2013	China	23%
Middle East respiratory syndrome	MERS-CoV	2012	Saudi Arabia	35%
Swine Flu	H1N1 Influenza A	2009	Mexico	1%
Avian Influenza/ Bird Flu	H5N1 Influenza A	2003	China	60%
Sever Acute Respiratory Syndrome (SARS)	SARS CoV	2002	China	9.6%
Dengue Fever	Dengue virus DEN I-IV	2000	Bangladesh	1.6%

Chikungunya Fever

Epidemiology

Chikungunya fever is a mosquito-borne viral disease.[6] It is an RNA virus that belongs to the alphavirus genus of the family Togaviridae.

Chikungunya virus (CHIKV) was first described by Marion Robinson and Lumsden in 1955, following an outbreak in 1952 on the Makonde Plateau, along the border between Mozambique and Tanzania. The name "chikungunya" derives from a word in the Kimakonde language, meaning "to become contorted", and describes the stooped appearance of sufferers with joint pain (arthralgia). In our local language it is called "Langra jor"

Until recently it has been primarily restricted to countries surrounding or near the Indian Ocean, Beginning in 2004 in East Africa, and continuing across many islands in the Indian Ocean and the Indian subcontinent in 2005–2006 an epidemic thought to affect more than 1.5 million people has occurred.

In Bangladesh it was first identified in 2008 in ChapaiNawabganj district, cases probably acquired disease in India. Later in 2011 some cases were identified in Dohar, Dhaka as an atypical outbreak of dengue. Then the recent outbreak started in April-May of this year.[7]

Chikungunya epidemics display cyclical and seasonal trends. There is an inter-epidemic period of 4-8 years. Outbreaks are most likely to occur in the post-monsoon period (starting in May, peak in July-August). In susceptible populations, chikungunya fever can have attack rates as high as 40 to 85%.

Transmission

Transmission primarily by bites of infected female mosquitoes - *Aedes aegypti* and *Aedes albopictus*, which bite throughout daylight hours, though there may be peaks of activity in the early morning and late afternoon. Maternal-fetal transmission is possible. Risk is highest during intrapartum period (two days before delivery to two days after delivery).Vertical transmission occurs in approximately 50% of cases. Cesarean delivery was not protective against vertical transmission. CHIKV was not found in breast milk.[8] Transmission via blood products has been described in France. Transmission via organ transplantation could also occur. Might be transmitted via corneal grafts even in the absence of systemic manifestations of chikungunya infection.[9]

Pathogenesis

After initial viremia and release of inflammatory cytokines. Organs targeted for chikungunya virus replication are - lymphoid tissues, liver, central nervous system, joints including synovial fluid, and muscle.

Clinical Features

Incubation Period is 3-7 days (range 2-12 days). 72% to 97% of those infected will develop symptoms. Asymptomatic seroconversion occurs in less than 15% of patients.[10] It is usually an acute febrile illness that may have 3 phases: a) Acute phase : upto 3 weeks of fever, b) Sub-acute phase : > 3 weeks to 3 months and

c) Chronic phase : > 3 months. 10-15% of the patient those who present with severe Chikungunya progress to Sub-acute or chronic phase.

Acute phase is characterized by the triad of Fever (92%), Arthralgia/arthritis (87%) and Rash. Fever is sudden onset, high grade with chills and lasts for 3 to 5 days (range 1 to 10 days). Fever can be biphasic.[11] Joint pain occurs initially or begins 2-5 days after onset of fever. Arthralgia is bilateral, symmetrical, distal joints more than proximal joints. Most affected joints are hands (50 to 76%), wrists (29 to 81%), and ankles (41 to 68%). Involvement of the axial skeleton was noted in 34 to 52% of cases. Periarticular edema or swelling has been observed in 32 to 95% of cases. Large joint effusions were noted in 15% of cases.[12] Skin rash has been reported in 40 to 75% of patients which may be macular or maculopapular, usually occurs within first 4 days of fever, starts on the limbs and trunk, pruritic in 25 to 50% of patients. Atypical dermatologic manifestations include bullous skin lesions (most often in children) and hyperpigmentation.[13] Cervical lymphadenopathy may occur in10-40% of patients, mostly in children or young adults.[14] Few patients may present or develop complications like severe sepsis or septic shock, meningoencephalitis, Guillian-Barre syndrome (GBS), myocarditis, decomposition of cardiovascular diseases, fulminant hepatitis in patients with chronic liver disease (CLD), pancreatitis, extensive epidermolysis, kidney failure, respiratory failure. Complications occur in patients of extreme ages - > 65, children, pregnancy, co-morbidity – diabetes mellitus, hypertension, ischemic heart disease and other co-infection.[15,16] In the subacute phase the predominant features is arthritis/arthralgia and fatigue.[17] In 10-15% patients the joint symptoms progress beyond 3 months among whom 50 % patients evolve into rheumatoid arthritis or seronegative spondyloarthitides or non-specific viral arthritis.[18]

Investigations

Investigations include testing blood or serum for direct or indirect evidences of CHIKV infection. Complete blood count (CBC) will reveal leukopenia with marked lymphopenia, or sometimes pancytopenia and mild thrombocytopenia. In all patients dengue should be excluded by doing NS-1. For confirmation of the case at least one of the followings in the acute phase of illness should be present: virus isolation by cell culture (only research purpose), detection of viral RNA by real-time reverse-transcription polymerase chain reaction (rRT-PCR) (within 5 days of onset of Illness), detection of antibody by enzyme linked immunosorbent assay (ELISA) - Viral specific IgM antibody in single serum sample collected within 5 to 28 days of onset fever or four-fold rise of IgG antibody in samples collected at least three weeks apart (1st sample after 7 days).[19] In CNS affected patients cerebro-spinal fluid (CSF) may be studied.

Treatment

Treatment of CHIKV in acute phase is mostly supportive with rest, adequate electrolyte containing fluid (at least 2-3 L/ day) and paracetamol upto maximum 4 gm/day for fever and joint pain. Additional tramadol or tapentadol may be used if joint pain is not relieved by paracetamol. There is no specific anti-viral therapy.[20] There is no indication of steroid in acute phase. Non-steroidal anti-inflammatory drugs (NSAIDs) should best be avoided in the acute phase, but if required may be used once dengue is excluded, but avoid aspirin. Cold compression of joints is helpful. Hospitalization is required for only those with hypotension, altered consciousness, bleeding manifestations, co-morbidity, extreme age or decreased intake. In the subacute phase the mainstay of treatment is adequate doe of NSAIDs and physiotherapy. In some patients short course systemic steroid may be necessary. In the chronic phase patient must be evaluated by a rheumatologist for the initiation of disease modifying anti-rheumatic drugs (DMARDs).[21]

Prognosis

Prognosis is good compared to dengue. Younger patients recover within 5 to 15 days. Recovery is delayed in the elderly. In few patients joint pain can persist up to 2 years. The risk of death is around 1 in 1,000 which is relatively low compared to dengue hemorrhagic fever (DHF).[22] After a single infection it is believed most people become immune.

Prevention

Preventing mosquito bite and vector control by control of mosquito breeding places are the best way to prevent CHIKV infection.

Zika Virus Infection

Epidemiology

Zika virus (ZKV) came into the light during the time of last World cup football in Brazil. The outbreak of ZKV will subsequent spread to the world through the travelers also spread fear. Although it is not a killer virus like others but the major issue is the possibility of neurological and fetal malformation if the mother is affected.[23] Zika virus is a mosquito-borne single-stranded RNA virus. It is related to dengue, yellow fever, West Nile and Japanese encephalitis, viruses that are also members of the virus family Flaviviridae.[24] Its name comes from the Zika Forest of Uganda, where the virus was first isolated in 1947 from rhesus monkey. The first well documented human case of Zika virus was in 1964; ZIKV first isolated from human in 1968 in Nigeria. Since then it was endemic in Africa and Asia. In 2007 there was an outbreak in Yap Island in Pacific Micronesia. In 2015, ZIKV first appeared outside of Africa and Asia when it was isolated in Brazil where is has caused a minor outbreak following the 2014 World Cup Football. Between January 2014 and February 2016, a total of 33 countries have reported Zika virus.[25,26]

Transmission

It is primarily transmitted by bite of female Aedes aegypti mosquito, but Aedes albopictus and africanus can also transmit the virus; same mosquitoes that spread dengue and chikungunya viruses.[27] Zika virus can be transmitted from a pregnant mother to her fetus during pregnancy or around the time of birth. There is no evidence of transmission through breastfeeding. Spread of the virus through blood transfusion and sexual contact (a biologist in 2008) have been reported. Zika was discovered in saliva and urine of infected persons in Brazil.[28]

Clinical Features

About 1 in 5 people infected with Zika virus become ill (develop Zika fever). Incubation period is likely to be a few days to a week. Features are like mild Dengue fever or Chikungunya fever. It is an acute febrile illness of 1-7 days with generalized maculopapular rash, arthralgia, myalgia, headache and conjunctivitis.[29] Although complications are rare, but there have been cases of meningo-encephalitis and Guillain-Barré Syndrome (GBS) reported in patients following suspected Zika virus infection.[30]

Zika and Pregnancy

Course of Zika virus disease is similar to that in the general population. There is no evidence to suggest that pregnant women are more susceptible or experience more severe disease during pregnancy. The panic is with the effect of ZKV on the fetus. There have been reports of 35 congenital microcephaly in babies of mothers who were infected with Zika virus while pregnant in Brazil in 2015. Zika virus infections have been confirmed in several infants with microcephaly.[31,32] Microcephaly can lead to seizures, vision or hearing problems, developmental/ intellectual disabilities which may often be lifelong and life threatening. Several birth defects have been reported in infants with suspected Zika virus infection including brain abnormalities like - intracranial calcifications, ventriculomegaly, neuronal migration disorders (lissencephaly and pachygyria), congenital contractures and clubfoot.[33]

Diagnosis

In suspected patients blood, urine or saliva can be tested for evidence of ZKV. In mother placenta or amniotic fluid and in infant cord blood or CSF can be tested. CBC may show mild leucopenia and thrombocytopenia. Confirmation can be done by any of the following – RT-PCR to detect viral RNA in blood and body fluids within 10 days of illness, virus specific IgM antibody by ELISA after first week of illness or plaque-reduction neutralization testing (PRNT) to measure virus-specific neutralizing antibodies titre (e 4 fold greater than Dengue).

Treatment

No specific antiviral medications are available to treat Zika infections. Treatment is symptomatic with rest, fluids, paracetamol to control fever. It is a self-limiting disease.

Prevention

The Center for Disease Control and Prevention (CDC) recommends that pregnant women in any trimester should consider postponing travel to an area where Zika virus transmission is ongoing. If she travels, she should strictly follow steps to avoid mosquito bites during the trip.[34] Some governments are urging women not to get pregnant at the moment. In some country, considering to give permission for termination of pregnancy.

Ebola Virus Disease (EVD)

Epidemiology

EVD is a zoonotic disease. Ebola virus (EBV) was first identified in 1976 in Zaire (Democratic republic of Congo). Name comes from the river Ebola around where it was identified. Since then it had caused several epidemics in Africa with variable mortality. The recent outbreak seems to have started in a village near Guéckédou, Guinea where bat hunting is common. Suspected index case was a boy who contacted the virus by eating bush meat (Bat). The Zaire species of Ebolavirus was the causative agent of the 2014-2016 epidemic in West Africa, where there were nearly 29,000 total cases (suspected, probable, or confirmed), more than 15,000 laboratory-confirmed cases, and 11,000 deaths.[35] Based on the total estimated case count, the overall case fatality rate was approximately 40 percent. In earlier outbreaks caused by the same virus species in Central Africa, case fatality rates reached 80 to 90 percent.

Ebola virus is a nonsegmented, negative-sense, single-stranded RNA virus. It is a member of the family Filoviridae, taken from the Latin "filum," meaning thread-like, based upon their filamentous structure. It has 5 subspecies. Among them Zaire type is most virulent.

Transmission

Ebola is spread through direct contact (through broken skin or mucous membranes) with blood or body fluids (urine, saliva, feces, vomit, sweat, breast milk, semen) of a person who is sick with Ebola, objects (like needles and syringes) that have been contaminated with the virus and infected animals.[36] There is no evidence to date that filoviruses are carried by mosquitoes or other biting arthropods.

Pathogenesis

It is the host response to infection, rather than any toxic effect of the virus, that is responsible for the effect of EVD. Immune mediated tissue damage, systemic inflammatory response, coagulation defect and impaired adaptive immunity lead to the morbidity and mortality related to EVD.

Diagnosis

Patients present with high fever, respiratory and gastro-intestinal symptoms, rash and bleeding manifestations. By 6^{th} day patient either starts improving or deteriorates.[37] Confirmation can be done by RT-PCR or antigen/ antibody capturing ELISA in blood or body fluids.

Treatment

The mainstay of treatment for Ebola virus disease involves supportive care to maintain adequate organ function (eg, cardiovascular, respiratory, renal) while the immune system mobilizes an adaptive response to eliminate the infection.[38] Several experimental antiviral therapies were used to treat patients during the 2014-2016 outbreak in West Africa, but their efficacy is unclear, and the availability of these drugs is limited.[39]

Prevention

Although no specific vaccine is available, some experimental vaccines have been developed and tested with good result in the recent outbreak.

Swine Flu (H1N1)

Epidemiology

In late March and early April 2009, an outbreak of H1N1 influenza A virus infection was detected in Mexico, with subsequent cases observed in many other countries, including the United States.[40] The pandemic that began in March 2009 was caused by an H1N1 influenza A virus that represented a quadruple reassortment of two swine strains, one human strain, and one avian strain of influenza; the largest proportion of genes came from swine influenza viruses. More than 214 countries and territories reported laboratory-confirmed cases of pandemic H1N1 influenza A. During this period approximately 61 million cases of pandemic H1N1 influenza occurred in the United States, including approximately 274,000 hospitalizations and 12,470 deaths.[41] The pandemic was declared to be over in August 2010.

Diagnosis

The signs and symptoms of influenza caused by pandemic H1N1 influenza A virus were similar to those of seasonal influenza, although gastrointestinal manifestations appeared to be more common with pandemic H1N1 influenza A. The most common clinical findings of the 2009 H1N1 influenza A pandemic were fever, cough, sore throat, malaise, and headache; vomiting and diarrhea were also common, both of which

are unusual features of seasonal influenza. Other frequent findings included chills, myalgias, and arthralgia. [42,43] Complications like rapidly progressive pneumonia, respiratory failure, acute respiratory distress syndrome (ARDS), and multisystem organ failure were reported in some cases during the 2009 to 2010 H1N1 influenza A pandemic.

rRT-PCR is the most sensitive and specific test for the diagnosis of pandemic H1N1 influenza A virus infection.[44]

Treatment

Specific anti-viral treatment is available for swine flu. During the 2009 to 2010 H1N1 influenza A pandemic, the United States Centers for Disease Control and Prevention (CDC) released guidelines for the use of antivirals for patients with confirmed or suspected influenza virus infection, which include a neuraminidase inhibitor (orally inhaled or intravenous zanamivir, oral oseltamivir or newly developed intravenous peramivir).[45] Prompt or early initiation of antiviral therapy was recommended for children, adolescents, or adults with suspected or confirmed influenza infection and any of the following features-illness requiring hospitalization, progressive, severe, or complicated illness, regardless of previous health status, children <5 years of age, adults e"65 years of age, pregnant women and women up to two weeks postpartum and individuals with co-morbid conditions.[46]

Prevention

Since the trivalent influenza vaccine did not give protection against swine flu, it was necessary for a new vaccine. Starting with the 2010 influenza season in the southern hemisphere and the 2010 to 2011 season in the northern hemisphere, the trivalent influenza vaccine included antigen from the 2009 pandemic H1N1 influenza A virus. Some monovalent pandemic H1N1 influenza A vaccines are also available with variable efficacy.[47] The CDC recommended that H1N1 influenza vaccine be given to all persons six months of age and older.

Avian Influenza/ Bird Flu

Epidemiology

There has been 2 different strains of avian origin Influenza A virus that has caused pandemics in different times. The first one is a highly pathogenic avian influenza (HPAI) virus H5N1 with a high mortality rate. The first outbreak of H5N1 bird flu was in December 2003. Since then, over 700 cases have been reported, in Africa, Asia, and Europe. The highest numbers have been in Indonesia, Vietnam, and Egypt. The most recent case of H5N1 was reported in Malaysia in March 2017.[48] It killed a number of chickens, but no human cases were reported. It is not easy for humans to catch it, but it is fatal in 60 percent of cases. H5N1 affects several types of birds. It has mostly been reported in farmed poultry, such as chickens, geese, turkeys, and ducks. Most people with the virus have had direct contact with infected poultry or objects contaminated with bird feces or secretions.

In late March and April 2013, human cases of novel avian influenza A H7N9 infection in China were reported to the World Health Organization.[49] The number of new cases peaked in April 2013 and then declined due to implementation of control strategies including closure of live bird markets and increased public awareness. Since then, annual epidemics have occurred during influenza season; most cases have occurred in China.[50] The largest wave was the fifth wave in late 2016 and early 2017. Since 2013, annual epidemics of avian influenza A H7N9 have resulted in >1300 reported cases.

Avian influenza A H7N9 virus appears to have derived from multiple reassortment events of at least 4 avian influenza viruses. This occurred because of migration of wild birds and growing chicken and ducks in close proximity, thus giving the chance to genetic reassortment and development of a novel virus.[51]

Diagnosis

The usual incubation period has been estimated to be from 3 to 7 days but has been reported to be as long as 10 days. Patients have presented with respiratory tract infections, many of which have progressed to severe pneumonia. Presenting signs and symptoms may include fever, cough, dyspnea, headache, myalgias, and malaise. Complications like fulminant pneumonia, respiratory failure, ARDS, septic shock, multiorgan failure, rhabdomyolysis, disseminated intravascular coagulation, and encephalopathy can develop in some patients.

Avian influenza A H5N1 and H7N9 can be detected by rRT-PCR from nasopharyngeal and/or lower respiratory tract samples. On chest radiograph and computed tomography (CT) scanning, patients with pneumonia have had multilobar patchy consolidations and diffuse ground-glass opacities.

Treatment

Specific anti-viral therapy is available for bird flu. Avian influenza A H7N9 virus appears to be resistant to the adamantanes (amantadine and rimantadine) but most isolates have been susceptible to the neuraminidase inhibitors (oseltamivir and zanamivir). H5N1 is sensitive to bothe the groups. Analysis suggested that the use of oseltamivir was associated with a significant reduction in mortality, benefit was greatest when treatment was started within the first two days following symptom onset, but some benefit persisted for up to six to eight days following symptom onset.[52] The standard dosing and duration of oseltamivir is 75 mg twice daily for five days, but a higher dose of 150 mg twice daily and a longer duration of 10 days may be considered, especially in patients with pneumonia or clinical progression.[53] The World Health Organization recommends that household contacts of patients with H5N1 avian influenza should receive post-exposure prophylaxis with 75 mg of oseltamivir once daily for seven to ten days.

Severe Acute Respiratory Syndrome (SARS)

Epidemiology

Cases of SARS were first noted in Guangdong Province, China, in November 2002. Initial cases were recognized as atypical pneumonia characterized by high fever, shortness of breath, cough, and pneumonia. Most early cases were associated with people in the wild animal trade and their contacts. The index case for the illness in Hong Kong was a physician from Guangdong Province who traveled to Hong Kong five days after the onset of symptoms, was identified as the source of 16 cases which led to the world-wide outbreak.[54] By the time the epidemic was contained in August 2003, more than 8400 cases and more than 900 fatalities were identified.[55] Cases of SARS occurred in 29 countries in Asia, Europe, and North America. China, including Hong Kong, had 83 percent of all cases.

Transmission

Transmission was believed primarily by droplet spread, and less frequently by direct contact or fomites. Viral shedding in feces also has been reported.

Virology

The etiologic agent (SARS-CoV) was identified as a previously unrecognized Coronavirus (Family Coronaviridae), an enveloped single stranded positive-sense RNA virus.[56]

Diagnosis

Clinically, cases present following a 2–10 day incubation with fever (> 38°C). Frequently there is malaise, nonproductive cough, dyspnea, chills, rigors, and headache. Rhinorrhea and a sore throat are rare.[57] Radiological signs after the onset of fever show consolidation that increases progressively in size, predominantly in the lower lung fields but pleural effusions are absent. Biopsy shows interstitial inflammation and oxygen saturation is decreased in about half of patients. Laboratory tests show leucopenia, lymphocytopenia and thrombocytopenia.[58] Diagnosis can be done by viral isolation and characterization, RT-PCR or serology [ELISA or immunofluorescence assay (IFA)]. However, the duration of detectable viremia or viral shedding is unknown. Currently, paired sera and a seroconversion or a rising titer is considered confirmation of suspected cases but is not used clinically. Diagnosis is based on clinical findings and exclusion of other causes of pneumonia.

Treatment

No specific treatment is currently recommended except for meticulous supportive care. As with other viral infections, antibacterial agents are ineffective. In addition, no antiviral agents have been found to provide benefit for treating SARS.[59]

Prognosis

Mortality was strongly age-dependent, with children and young adults rarely developing fatal disease, while more than half of the clinical cases over the age of 65 years died. Overall mortality was close to 10%.

Conclusion

Emerging viral diseases are a major cause of morbidity and mortality in different parts of the world. Humans are responsible for this to a large extent by causing disruption of environmental homeostasis and intruding and altering wild life habitat as well as domestic animals. In order to survive these deadly viruses, much attention is needed to be paid to prevention of transmission of these zoonotic diseases and preserving a healthy environment for both human being and animals. Among all of these the recent outbreak of Chikungunya in our country has threatened the health sector just like Dengue fever did in year 2000. Hopefully with growing knowledge and clinical experience we will be able to overcome this hurdle and contain the disease.

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