Journal Homepage: - www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI: 10.21474/IJAR01/11933
DOI URL: http://dx.doi.org/10.21474/IJAR01/11933

REVIEW ARTICLE

VECTOR-BORNE DISEASES IN INDIA

Pragati Dhopte and Irrusappan Hari

Manuscript Info

Manuscript History
Received: 27 August 2020
Final Accepted: 30 September 2020
Published: October 2020

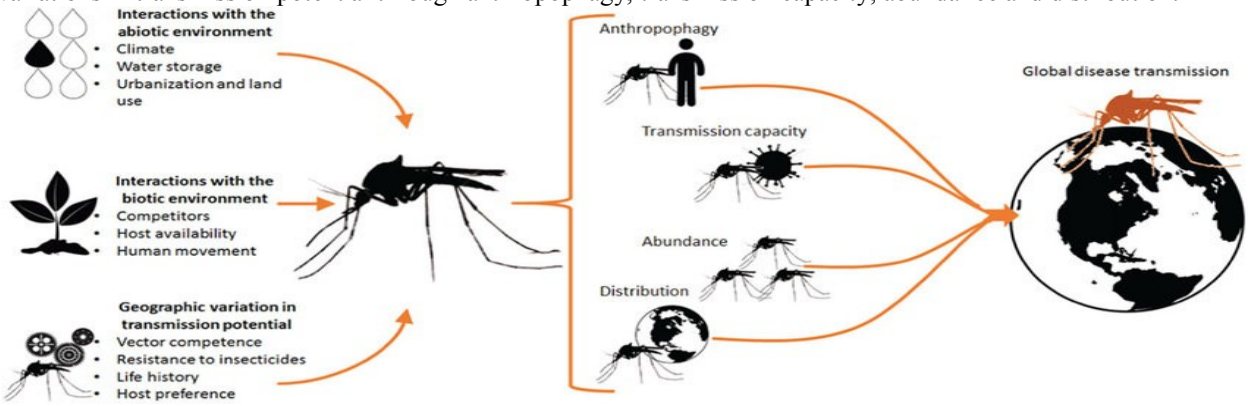
Key words:-
VBD, Malaria, Dengue, JE, Kala Azar, Chikungunya, Transmission, MDA, Deaths

Abstract

Vectors are transmitted diseases from person to person that diseases are known as vactor borne diseases. There are mainly six vector borne diseases present in India, tropical and subtropical rigion also. As per current medical importance, geographic distribution, epidemiology and potential spreading of vector borne diseases, Malaria total cases were 29340 and deaths 2 and Japanese encephalitis total cases were 111. Chikungunya and Kala azar total cases were 700 and no deaths were found in 2020 respectively. 87.25% of MDA were supplied to total population and the dengue cases were 136422 and deaths 132 were observed in 2019. The vector borne diseases in India are reviewed in this article.

Introduction:-

The function of the arthropods is an intermediate or definitive host or reservoir to maintain and /or amplify the host like vertedrates. After pathogen dissimination, they may be refracted to infection and infectious (Reisen et. al., 2010). According to vector definition they may be any arthropod (insect or arachnid) or animal which carries and transmits infectious pathogens and parasites directly or indirectly from an infected animal to a human or from an infected one infected person to another and causes diseases to the human beings (Kalluri et. al., 2007). Vector-Borne diseases are described as many parasites and pathogens responsible for some of the most important diseases in humans, agriculture and in nature (Wilson et al., 2017). According to National vector borne disease control program, Ministry of government of India, there are various types of vector borne diseases like Malaria, Dengue, Japanese encephalitis, Kala-Azar, Lymphatic filariasis and Chikunguniya, etc (WHO, 2014). The below figure shows the human are transmitted as per global diseases transmission, carried out by abiotic, biotic and geographic variations in transmission potential through anthropophagy, transmission capacity, abundance and distribution.



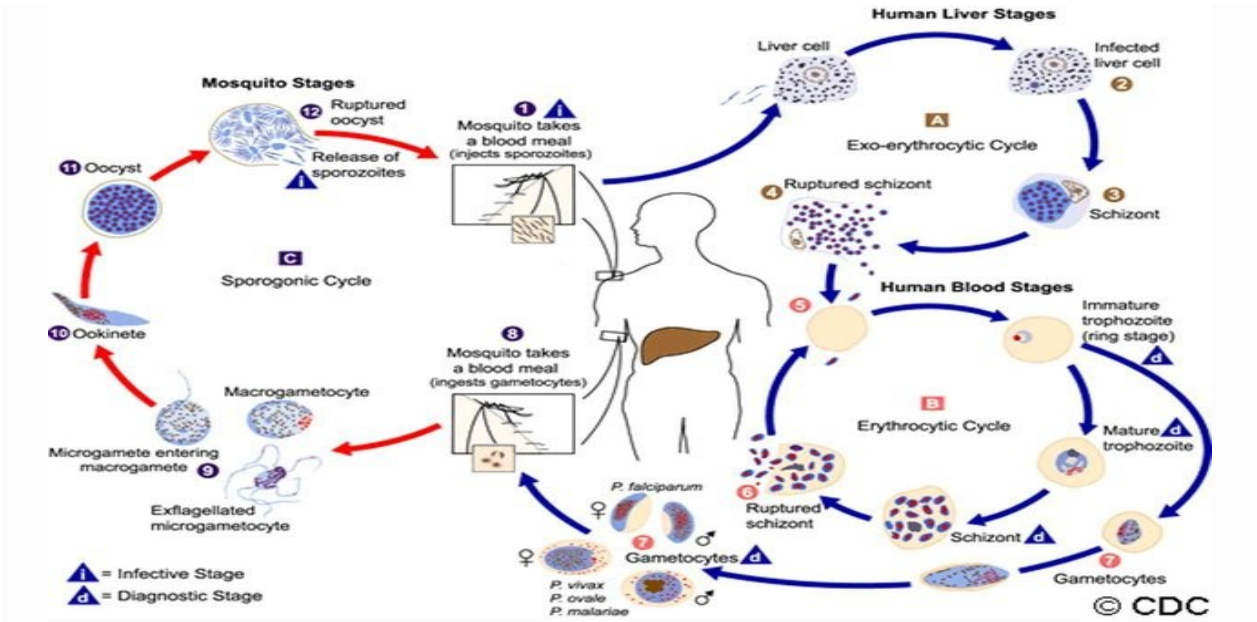
Corresponding Author:- Pragati Dhopte

The complexity of interactions among mosquito vectors, arboviral (Sharma et. al. 2017)
A serious public health problem has been emerged in recent years in the countries of the South- East Asia Region, including India due to vector-borne diseases (VBD) (Patel et. al., 2011). Mosquitoes and other blood-feeding arthropods are vectors of serious parasitic, viral or bacterial diseases. Malaria and dengue, together causes over **300 million cases and kill over one million people every year**. Vector-borne diseases (VBDs) have destroyed in the past but have been either eliminated or reduced to only sporadic cases in the second half of the last century. However, in the last few years, we have witnessed a significant increase in the frequency of VBD cases and outbreaks, and the risk of resurgence now looks ever more probable. **The lack of vaccines and other effective prevention for most VBDs and the complexity of the disease life cycles require highly integrated approaches that target the disease transmission system rather than only the pathogens** (Christophides, G. K., &Crisanti, A., 2013). In tropical and subtropical regions, dengue is known as a **disease of poverty** as it is most closely associated with **poor populations** (Rigau-Perez, 1998; WHO, 2012). The first major epidemic of Japanese encephalitis (JE) was described in 1924 in Japan, in which about 6000 cases were reported. Currently in India, JE is the principal cause of vaccine-preventable encephalitis. JE is now endemic in several states in India, including Bihar, Uttar Pradesh (UP), Assam, Manipur, Andhra Pradesh, Karnataka, Madhya Pradesh, Tamil Nadu, Haryana, Kerala, West Bengal, Orissa, Union territories of Goa and Pondicherry, with epidemic activity in northern and central parts of India (Tiwari et. al., 2012).

National Vector Borne Disease Control Programme (NVBDCP) is one of the most comprehensive and multifaceted public health activities including prevention and control of mosquito borne diseases (Patel et. al., 2011). Malaria has been known in India from time immemorial and it has always been a **major public health hazard**. India is one of the affected countries in which millions of people die every year due to Malaria. Most of the arid and semiarid in Western India fall in an unstable Malaria zone in the country (Srivastava and Yadav 2000). Malaria unquestionably remains the most important vector borne disease in India as far as the studies of the Central Bureau of Health Education (CBHE) and the Directorate of National Malaria Eradication Programme (NMEP) are concerned. According to CBHE, after the independence, an estimate of malaria problem made in 1953, indicated an annual incidence of 75 million cases with 0.8 million deaths. According to the Directorate of National Malaria Eradication programme (NMEP) the Malaria incidence lead dropped down to 2 million cases in 1958. In the report of Government of India stated an upsurge of Malaria occurred in 1965 and the cases rose from 1.48 lakhs in1966 to 6.4 million in 1976 (Tiwari et. al., 2012).

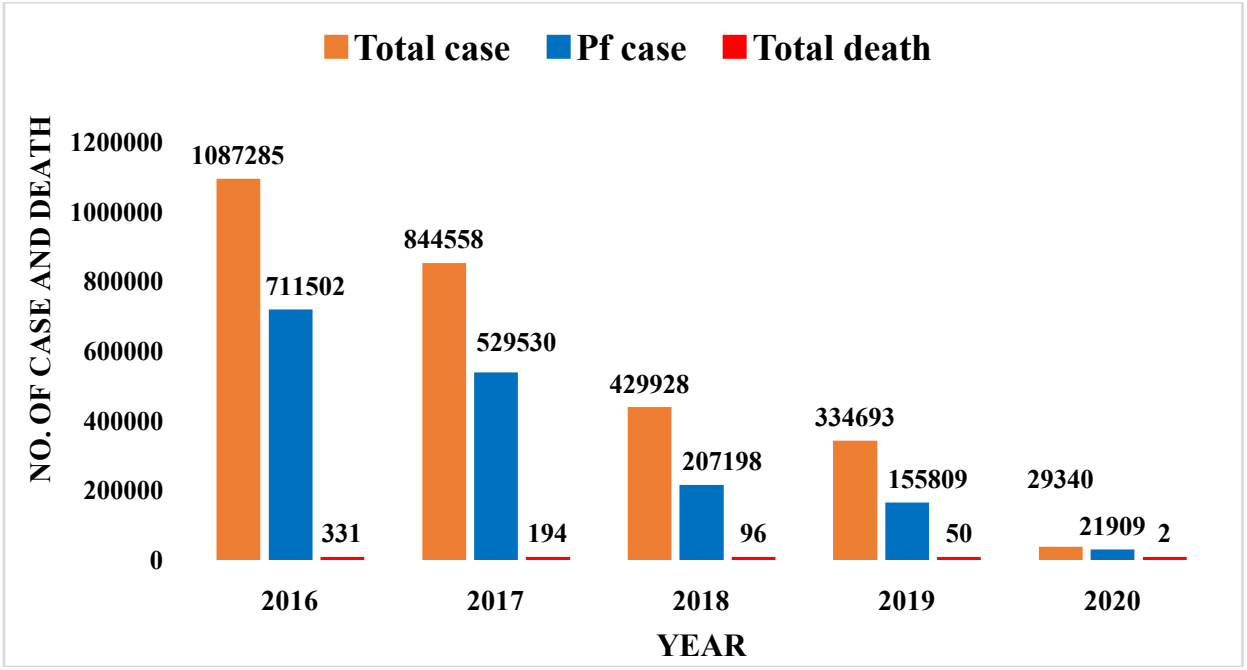
Malaria:
Malaria is caused by obligate intraerythrocytic protozoa of the genus *Plasmodium*. Humans can be infected with one or more of the following four species: *P. falciparum*, *P. vivax*, *P. ovale*, and *P. malariae*. Plasmodia are primarily transmitted by the bite of an infected female *Anopheles* mosquito, but infections can also occur through exposure to infected blood products (transfusion malaria) and by congenital transmission. In industrialized countries, most cases of malaria occur from areas endemic for malaria (imported malaria). Exceptionally, local transmission through mosquitoes occurs (indigenous malaria) (Trampuz et. al., 2003).

When the infected anopheline mosquito takes a blood meal, sporozoites are inoculated into the bloodstream. Within an hour sporozoites enter hepatocytes and begin to divide into exoerythrocyticmerozoites (tissue schizogony). For *P. vivax* and *P. ovale*, dormant forms called hypnozoites typically remain quiescent in the liver until a later time; *P. falciparum* does not produce hypnozoites. Once merozoites leave the liver, they invade erythrocytes and develop into early trophozoites, which are ring shaped, vacuolated and uninucleated. Once the parasite begins to divide, the trophozoites are called schizonts, consisting of many daughter merozoites (blood schizogony). Eventually, the infected erythrocytes are lysed by the merozoites, which subsequently invade other erythrocytes, starting a new cycle of schizogony. The duration of each cycle in *P. falciparum* is about 48 hours. In non-immune humans, the infection is amplified about 20-fold each cycle. After several cycles, some of the merozoites develop into gametocytes, the sexual stage of malaria, which cause no symptoms, but are infective for mosquitoes as shown in figure (Trampuz et. al., 2003).



Life cycle of Malaria parasits (Ali, M.A. 2013)

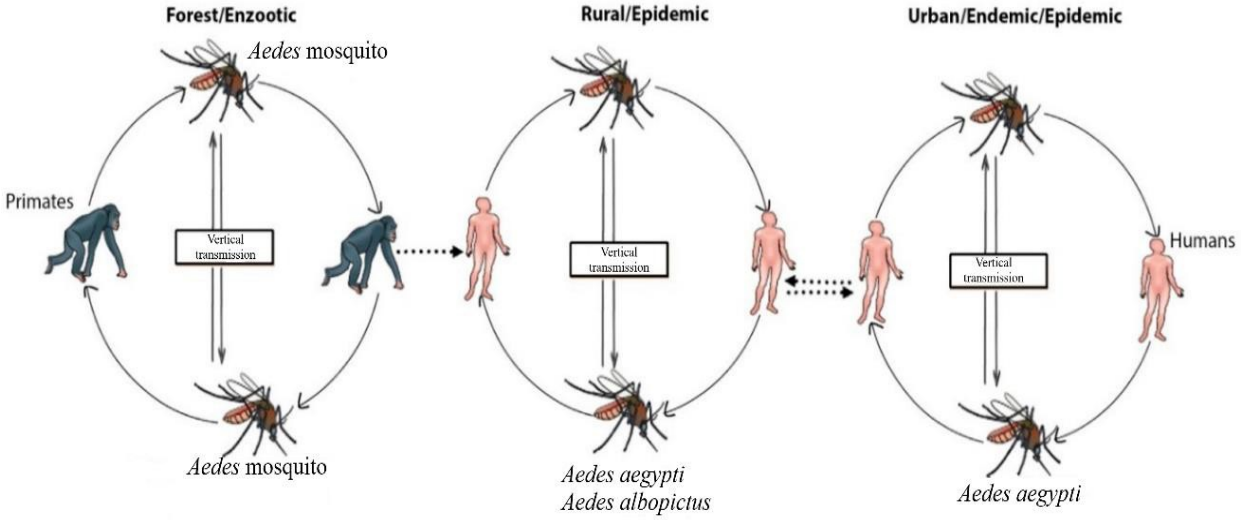
In Gujarat, about 28% of cases of malaria appear during the dry season, most probably caused by P. vivax relapses. In Kheda district of Gujarat showed higher recurrence rates in the 5–10 years age group than in other age groups. A total of 82% of recurrences occurred within 1 year of the primary malaria attack. Recurrences occurred up to 3 years after the primary malaria attack, but were less frequent in the 3rd and 4th year. Data from a semiarid region of Kutch in Gujarat showed significant autocorrelation peaks between the months of August to November and those from January to June, providing an estimated relapse latency period between 5 and 8 months and a mean value of 7 months. This relatively long interval is compatible with infection with temperate P. vivax (Sharma et. al, 1990; Shrivastava et. al. 1996; Roy et. al. 2013). Total cases of malaria are decreases with increase in year i. e.from 2016 to 2020 as shown in graph.



Graph: Malaria Situation in India from 2016-2020
(Source: NVBDCP, 2020)

Dengue:

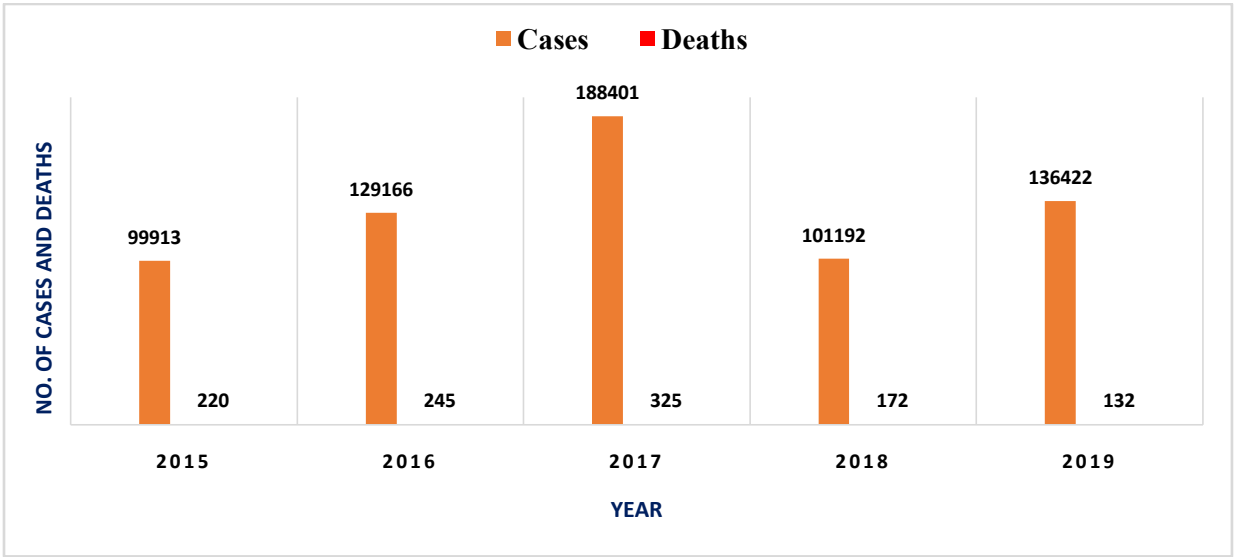
Dengue is an acute febrile disease caused by the mosquito-borne dengue viruses (DENVs), consisting of four serotypes (DENV 1 to 4), that are members of the flaviviridae family, genus flavivirus (Westaway et. al., 1985). All four DENV serotypes have emerged from sylvatic strains in the forests of South-East Asia (Wang et. al., 2000). Dengue (pronounced Den' gee) is a disease caused by any one of closely related dengue viruses (DEN1, DEN 2, DEN 3 & DEN 4). The viruses are transmitted to human by the bite of an infected mosquito, AedesAegypti but 2001 outbreak in Hawaii was transmitted by AedesAlbopictus. The Asian genotypes of DEN-2 and DEN-3 are frequently associated with severe disease (Vaddadi Srinivas, 2015). Dengue virus is a RNA virus of the family flaviviridae; they are otherwise called arboviruses. The dengue virus genome contains 11,000 nucleotide bones. They have 3 different protein molecules that form virus partied (C, prM and E) and 7 other types of protein molecules (NSI, NS2a, NS2b, NS3, NS4a, NS4b, NS5) that are found in infected host cells and are required for replication of virus. There are 4 strains of virus, ex; DEN1, DEN2, DEN3, DEN4. ALL 4 serotypes can cause full blown disease. Infection with 1 serotype is believed to produce lifelong immunity to that serotype, but he can be infected with other serotypes in future (Shrinivas, 2011). The humans are the primary host for dengue viruses & transmitted by Aedes mosquitoes. A mosquito that takes a blood meal from an infected person becomes infected with a virus. In 8 to 10 days the virus spreads to tissues like salivary gland from the gut of the mosquito. The virus seems to have no detrimental effect on the mosquito. Aedes mosquitoes live in close proximity to humans. Dengue may also get transmitted via infected blood products and through organ donation. Vertical transmission from mother to child can also occur during pregnancy (A TuiskunenBäck, Å Lundkvist, 2013). When a mosquito carrying DENV bites a person, the virus enters the skin together with the mosquito's saliva. It binds to and enters the white blood cells, and reproduces inside the cells while they move throughout the body. The white blood cells respond by producing a number of signalling proteins (Such as interferon) that are responsible for many of the symptoms, such as the fever, the flu-like symptoms and the severe pains. In severe infection, the virus production inside the body is much increased, and many more organs (Such as the liver and the bone marrow) can be affected, and fluid from the bloodstream leaks through the wall of small blood vessels into body cavities. As a result, less blood circulates in the blood vessels, and the blood pressure becomes so low that it cannot supply sufficient blood to vital organs. Furthermore, dysfunction of the bone marrow leads to reduced numbers of platelets, which are necessary for effective blood clotting; this increases the risk of bleeding, the other major complication of dengue (Shrinivas, 2011).



Transmission of Dengue (Ahammad F. et al., 2019)

Severe disease it is not entirely clear why secondary infection with a different strain of DENV places people at risk of dengue hemorrhagic fever and dengue shock syndrome. The most widely accepted hypothesis is that of antibody-dependent enhancement (ADE) (Tuiskuhen et. al., 2013). Host immunity, vector capacity, circulating DENV, weather or climate, dengue control capacity and population movement is the risk factors involved in the transmission of Dengue. The climatic factors influence the dengue epidemiology because of its indirect impact on lifecycle of mosquitoes and on incubation periods of DENV within mosquitoes (Morin *et al.,* 2013). Primarily the dengue infection can be controlled by the control of dengue vectors which can be aimed against the immature

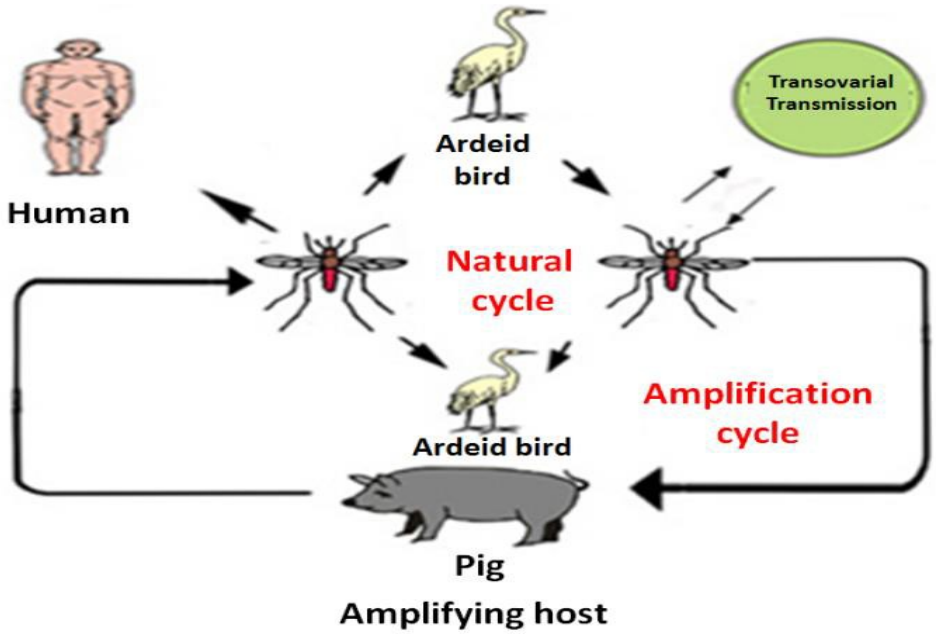
aquatic stages (larvae and pupae) or the adult mosquitoes. Direct vector control measures include, use of insecticides to kill the mosquitoes or prevent them from biting by employing repellents. Environmental modification or sanitation improvements that reduce potential larval development sites or house improvements that prevent mosquito entry can be used as indirect vector control methods (WHO, 2009 and Kuehn, 2014). The Indian encounter with dengue is interesting and intriguing. The epidemiology of dengue in India is very complex and ever changing. Though the first reported occurrence of dengue fever in India was in 1946, there were no major outbreaks in the country for almost 20 years, until a major epidemic occurred in 1963–1964 in Kolkata.16–18 It gradually spread to involve North India in 1967–1968 and also South India.19,20 All four serotypes of the virus were reported from South India.21 Again, after almost three decades of very low incidence, the first major outbreak of DF/DHF occurred in Delhi in 1996 where 10,252 cases and 423 deaths were reported.10 This outbreak was caused by DENV-2, genotype IV strain of the virus.22 Similar strains of the DENV-2 were reported from central India (Gwalior) and southern India, indicating that the predominant circulating strain in India that time was DENV-2.23 In the post-epidemic period in Delhi in 1997, DENV-1 was also seen in circulation (Gupta and Bullani, 2014). Dengue cases are increased in 2019 as compare to 2018.



Graph:- Dengue Situation in India from 2014 to 2019.
(Source: NVBDCP 2020)

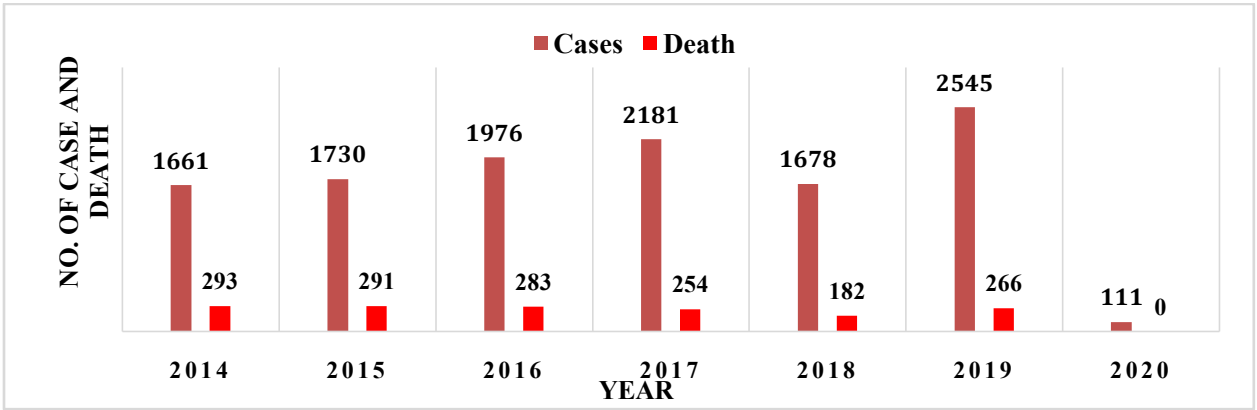
Japanese encephalitis:

Japanese encephalitis virus (JEV) is a single-stranded RNA virus belonging to the family Flaviviridae, genus Flavivirus. It is one of the leading forms of viral encephalitis worldwide, mostly prevalent in eastern and southern Asia, covering a region with a population of over three billion. Most infections of JE are asymptomatic, but if clinical illness develops, it causes significant morbidity and mortality. Though underreported, JE causes an estimated 50,000 cases and 15,000 deaths annually. JE is a disease of public health importance because of its epidemic potential and high fatality rate. In endemic areas, the highest age-specific attack rates occur in children of 3 to 6 years of age. Approximately one third of patients die and half of the survivors suffer severe neuropsychiatric sequelae from the disease. Japanese encephalitis virus (JEV) belongs to the family flaviviridae and genus Flavivirus. It is a single stranded, positive-sense polarity RNA genome of approximately 11 kb in length. The virion of JEV contains three structural proteins – nucleocapsid or core protein (C), non-glycosylated membrane protein (M), and glycosylated envelope protein (E), as well as seven non-structural (NS) proteins – NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS. JEV exists in a zoonotic cycle between mosquitoes and pigs and/or water birds. This study reviewed JEV literature from 2000 to 2010, outlining the Indian scenario, clinical depictions, diagnosis, and the prevention of this deadly disease (Tiwari et. al., 2012).



Transmission of JE (Bhattacharya. S, 2014)

The JEV is transmitted to vertebrates by mosquitoes. Mosquito transmission was suspected during the early 1930s; in 1938, Mitamura et al. reported isolation from Culextritaeniorynchus. The ecology of JEV has come from various studies carried out in Japan by Scherer et al., and JEV ecology has been the subject of several reviews. Many species of Culex mosquitoes can transmit JE. For Southern Asia, Eastern Asia, and Southeastern Asia, the main vector of JE is C. tritaeniorhynchus. For Northern Australia, the main vector is C. annulirostris. However, various other secondary vectors may be important. Indian studies in particular have revealed a number of secondary vectors, including Mansoniaindiana, C. pseudovishnui, C. whitmorei, C. gelidus, C. epidesmus, Anopheles subpictus, A. peditaeniatus, and M. uniform. The natural cycle of JE virus in Asia involves water birds and Culex mosquitoes. However, unlike many other mosquito-borne diseases, an amplifying host is important in the epidemiology of human JE. In Asia, pigs are considered to be the most important amplifying host, providing a link to humans through their proximity to housing. The life cycle of the virus is illustrated in Figure. There are two epidemiological patterns of transmission: an endemic pattern in tropical areas with viral circulation in most months of the year, but with a broad seasonal peak, probably resulting from irrigation practices; and an epidemic pattern in more temperate areas with clear summer seasonality (Tiwari et. al., 2012).

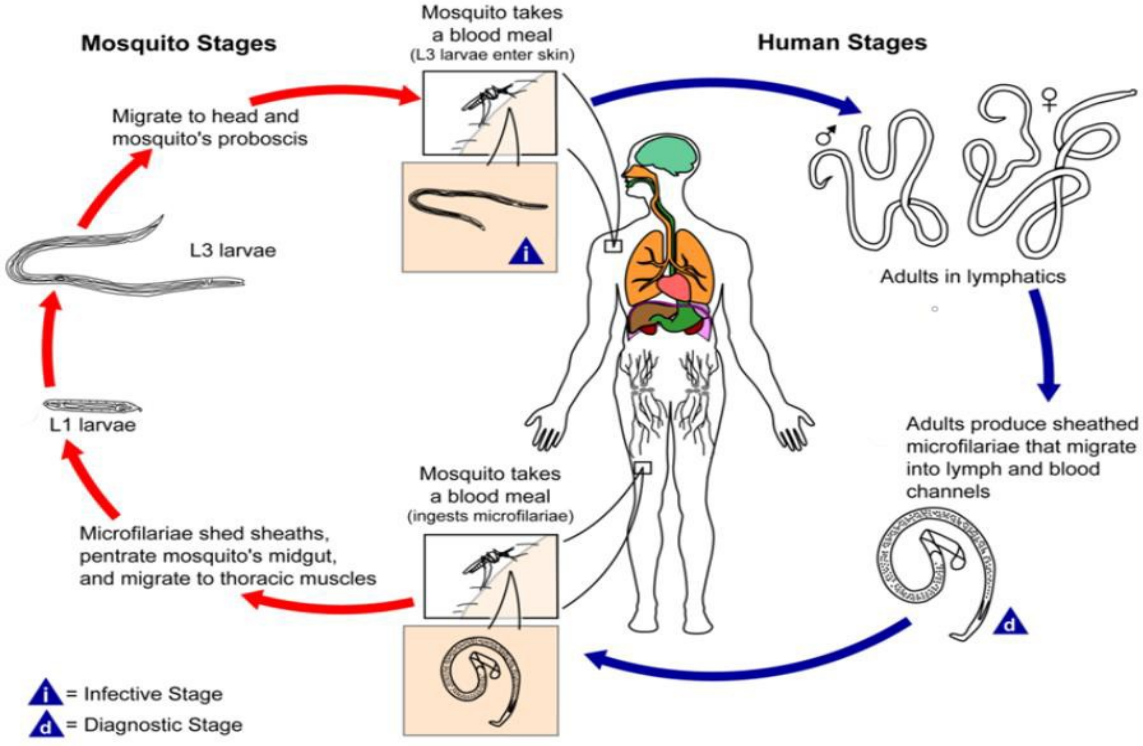


Graph:- Japanese Encephalitis Situation in India from 2014-2020.
(Source: NVBDCP, 2020)

In India, epidemics of JE are reported from many parts of the country, and it is considered a major pediatric problem. The first recognition of JE based on serological surveys was in 1955, in Tamil Nadu, India.16 A total of approximately 65 cases were reported between 1955 and 1966 in Southern India.17 Subsequent surveys carried out by the National Institute of Virology of Pune indicated that approximately half of the population in Southern India has neutralizing antibodies to the virus. Since 1955, many major outbreaks in different parts of the country have been reported. A major outbreak resulting in a 42.6% fatality rate was reported in the Bankura District of West Bengal in 1973. Subsequently, the disease spread to other states and caused a series of outbreaks in different parts of the country. In 1978, cases were reported from 21 states and union territories.15 In Uttar Pradesh, the first major JE epidemic occurred in Gorakhpur in 1978, with 1,002 cases and 297 deaths reported. Many outbreaks were reported in Gorakhpur after the 1978 JE outbreak, with varying intensity and magnitude. Since 1978 to 2005, this encephalitis has taken more than 10,000 lives in the state.18 The 2005 epidemic surpassed all previous reported outbreaks in the country. In that year, Uttar Pradesh faced a devastating outbreak of JE, mostly confined to Gorakhpur, with 6,061 cases and 1,500 deaths; another outbreak occurred in 2006, with 2,320 cases and 528 deaths. Similarly, JE cases in Uttar Pradesh were confined predominantly to Gorakhpur during 2007, with 3,024 cases and 645 deaths,18 and then onwards till 2007 there have been 103,389 reported cases in India, and 33,729 deaths.19 Approximately 597,542,000 people in India live in JE-endemic regions, and 1,500 to 4,000 cases are reported every year.20 These figures are based on total reported cases; it is possible that many cases are unreported and hence the actual magnitude of the threat of JE may be considerably higher, both in the Indian and in the global context. JE incidence during the past few years is given in graph. The trend of JE suggests that the problem in Northern India is escalating, and larger epidemics may occur in the future (Tiwari et. al. 2012).

Lymphatic filariasis:

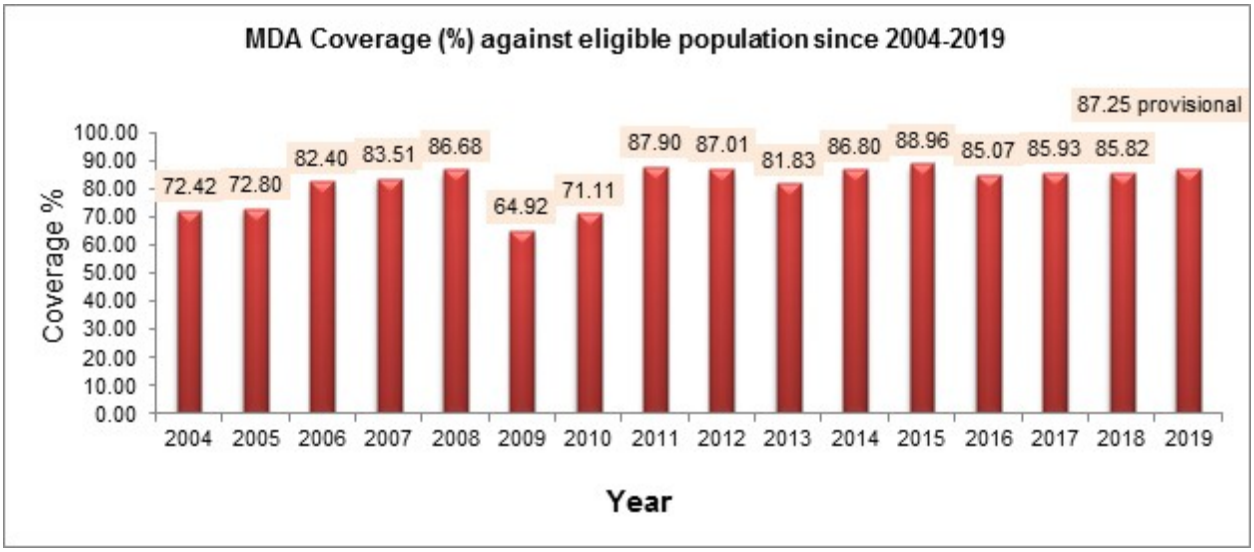
Filariae are microscopic roundworms that dwell in the blood and tissues of humans. The most important filarial diseases for humans are lymphatic filariases, in which the adult worms are found in the lymphatic system. The lymphatic form of filariasis will be the focus of the site. Lymphatic is also referred to sometimes as “elephantiasis.” Elephantiasis is actually Infection with lymphatic filariasis, commonly known as elephantiasis, occurs when thread-like, filarial parasites are transmitted to humans through mosquitoes.



Transmission Cycle of Lymphatic Filariasis (CDC 2018)

Lymphatic filariasis is transmitted by different types of mosquitoes, for example by the *Culex*mosquito, widespread across urban and semi-urban areas; *Anopheles*, mainly in rural areas; and *Aedes*, mainly in the Pacific Islands and

parts of the Philippines. It is also transmitted by 3 types of parasite (*Wuchereriabancrofti*, responsible for 90% of cases, *Brugiamalayi*and *B. timori*). Microscopic parasitic worms lodge in the lymphatic system and disrupt the immune system. They live for 6-8 years and, during their lifetime, produce millions of microfilariae (tiny larvae) that circulate in the blood.



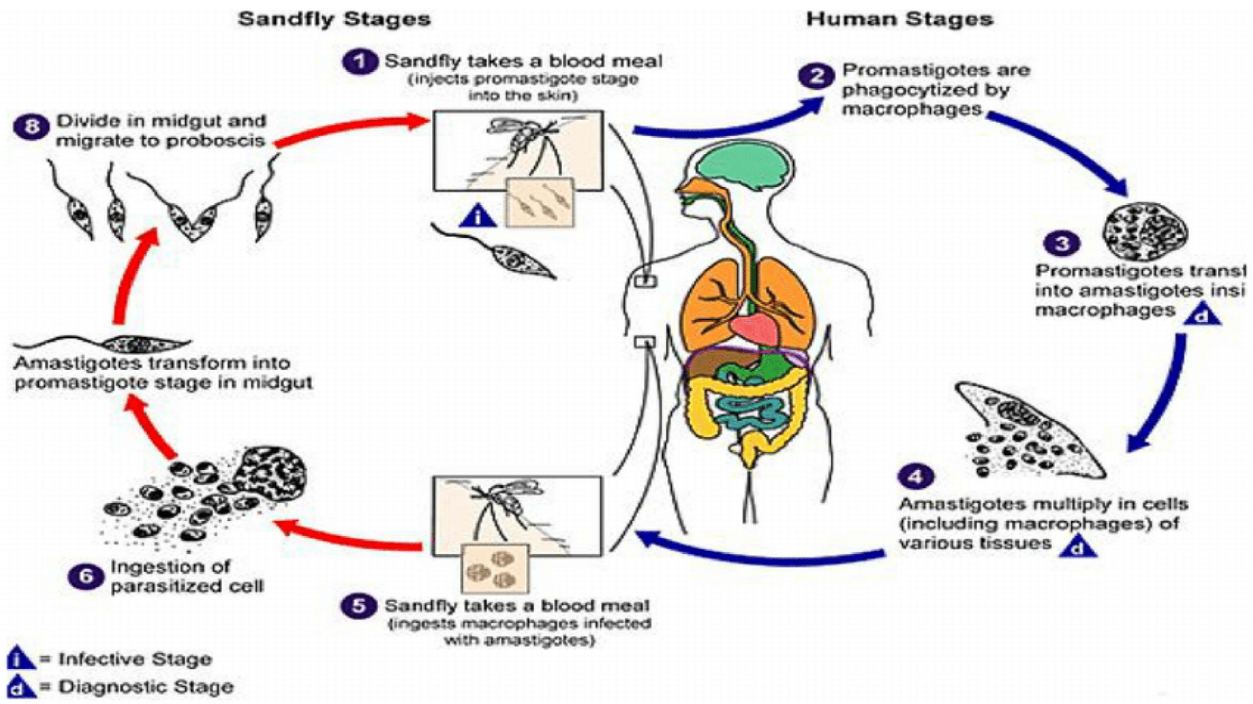
MDA Coverage (%) against eligible population since 2004-2019 (Source: NVBDCP, 2020)

More than 120 million people are currently infected with lymphatic filariasis; about 40 million of them are disfigured and incapacitated by the disease. Lymphatic filariasis afflicts more than 25 million men with genital disease and more than 15 million people with lymphoedema. The majority of infections has no symptoms, but silently cause damage to the lymphatic system and the kidneys as well as alters the body's immune system. Acute episodes of local inflammation involving skin, lymph nodes and lymphatic vessels often accompany chronic lymphoedema (Fig. 4d). Approximately 65% of those infected live in the WHO South-East Asia Region, 30% in the African Region, and the rest in other tropical areas. Treatment is rarely recommended to clear the adult parasites from the bloodstream by using a single dose of albendazole together with either diethylcarbamazine or ivermectin. Interruption of transmission of infection by mosquitoes bite is mostly recommended (Khan, 2015).

Nocturnal periodic form of W. bancrofti is widely distributed in tropical and subtropical regions like Africa, Asia and Latin America while non-periodic or diurnal sub-periodic form is prevalent in the Islands of the South Pacific regions where maximum densities of microfilaria (mf) count is observed around 1630 hrs; and the distribution of sub-periodic form of W. bancrofti is limited to western Thailand where peak mf density in the peripheral human blood is observed at 2030 hrs13-15. In India also, sub-periodic form of W. bancrofti was recorded in Andaman and Nicobar Islands. Bancroftianfilariasis, in India is transmitted mainly by Culexquinquefasciatus which is a night biting mosquito and the mf periodicity is nocturnal except for those reported from Andaman and Nicobar Islands16-19. There is no report on microfilarial periodicity of W. bancrofti from Assam; therefore, the present study was undertaken in Dibrugarh district of Assam to observe the pattern of microfilarial periodicity (Khan et. al., 2015).

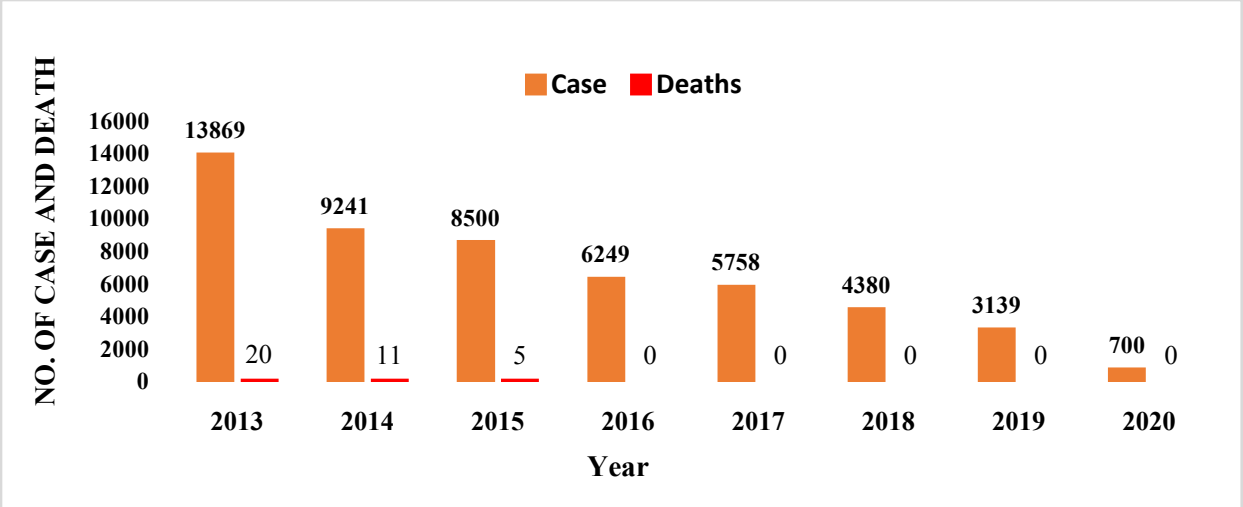
Kala-Azar:

The genus Leishmania belongs to a family Trypanosomatidae (order Kinetoplastida). The parasite is categorized in two main groups; the old world species occurring in Europe, Africa and Asia, and the new world species occurring in America. About 53 species of the parasite have been described from different regions of the world; of these, 31 species are known to be parasites of mammals and 20 species are pathogenic for human beings. Many of the leishmania species infecting human are zoonotic, having a complex variation in domestic and wild mammal reservoir hosts; while, other species of the parasite are anthroponotic, having human-to-human transmission in the presence of the vector. Leishmaniadonovani is usually considered to be an anthroponotic parasite though studies reported the presence of parasite or circulating antibodies against the parasite antigens in domestic and wild animals of India and East. The global distribution of each of Leishmania species determines the type of disease that occurs in an area. L. donovani causes visceral leishmaniasis in South Asia and Africa; while L. infantum causes this disease in the Leishmania species have a heteroxenous life cycle.



Transmission Cycle of Leishmaniasis (Hailu A et al., 2005)

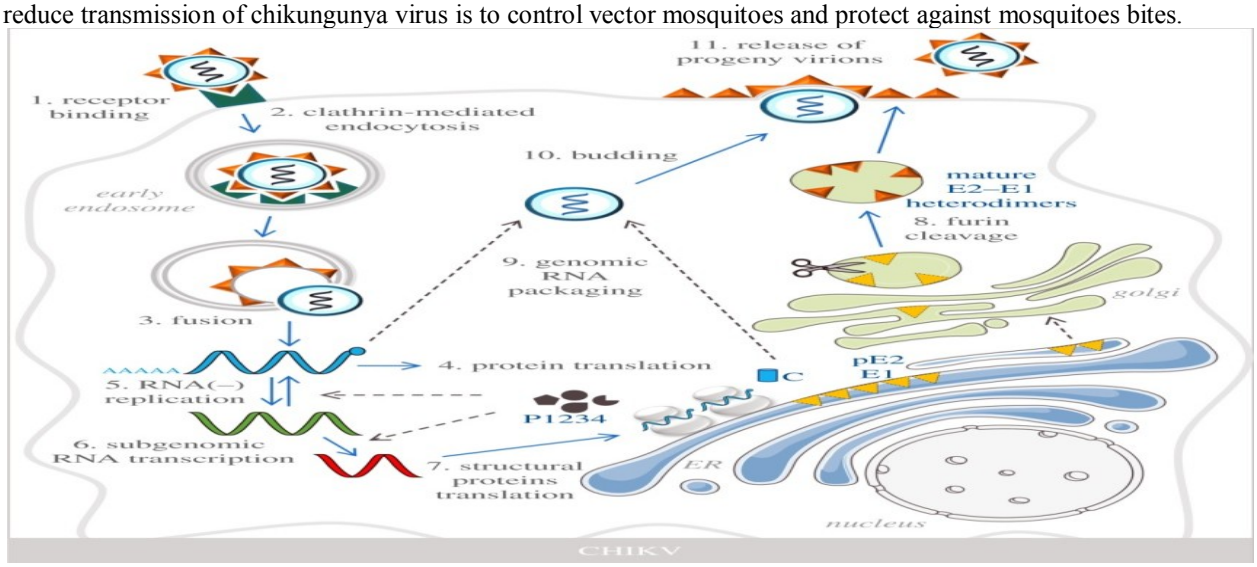
The parasite exhibits two morphological forms in its life cycle, amastigote in macrophages of the mammalian host and promastigote in the gut of the sand fly vectors. Human stage of the life-cycle starts when a parasitized female sand fly injects metacyclicpromastigotes into human body. The promastigotes are then phagocytosed by the host's macrophages, and consequently, the parasite transforms into non-flagellated form, amastigote, which reproduce by binary fission. The multiplication of the parasites occurs inside the macrophages. The macrophage lyses and the multiplication cycle continue when other hosts' phagocytes are infected Leishmaniasis is transmitted by the bite of infected female sand flies. There are over 600 species of sand flies divided into five genera: Phlebotomus and Sergentomyia in the Old World and Lutzomyia, Brumptomyia, and Warileya in the New World. Although human-biting sandflies occur in various genera, the only proven vectors of human leishmaniasis are species and subspecies of the genus Phlebotomus and Lutzomyia. Various species in the genus Phlebotomus are responsible for transmission of leishmaniasis in the Old World and Lutzomyia species in the New World. Each sand fly species typically transmits only one species of parasite and each parasite leads to a particular type of disease [The development of leishmania parasite within the vector sand flies is an inevitable stage for the transmission of leishmaniasis among various hosts. Female sand flies acquire leishmania parasites when they feed on an infected mammalian host in search of a blood-meal. The amastigote forms of the parasites taken up by sand flies are not usually found in the peripheral circulation; rather they are present in the skin itself. Parasites present in organs such as liver and spleen are not accessible to sand flies. Amastigotes are intracellular parasites found in phagolysosomes of macrophages and other phagocytes, and their uptake by the blood-feeding sand fly is assisted by the cutting action of the mouthparts. Thus sand flies are pool feeders, meaning they insert their saw-like mouthparts into the skin and agitate them to produce a small wound into which the blood flows from superficial capillaries. It is this tissue damage associated with the creation of the wound that releases skin macrophages and/or freed amastigotes into the pool of blood, and enables their subsequent uptake into the abdomen of the sand fly. Then the parasite multiplies and further differentiates into other stages, metacyclicpromastigote being the final mammalian-infective stage which movies to the foregut of the vector sand fly. The metacyclicpromastigotes are deposited in the skin of a new mammalian host when the fly takes another blood meal, leading to the transmission of disease (Alemayehuet et. al., 2017).



Graph:- Manifesting no. of Kala Azar Cases and Death from 2013-2020 in India.
(Source: NVBDCP, 2020).

The incidence of kala-azar in India is among the highest in the world. The global estimate for the incidence and prevalence of kala-azar cases per year is 0.5 million and 2.5 million, respectively. Sixty-six countries have reported confirmed kala-azar cases' but 90% of these occur in India, Nepal, Bangladesh and Sudan. In India, the calculated DALYs (disability-adjusted life years) lost due to kala-azar in 1990 were 6.8 million for men and 0.5 million for women. The corresponding global figures are 12 million for men and 8.6 million for women. II following the resurgence of the disease in the 1980s, various reports have stressed the need to control the disease (Bora D., 1999).

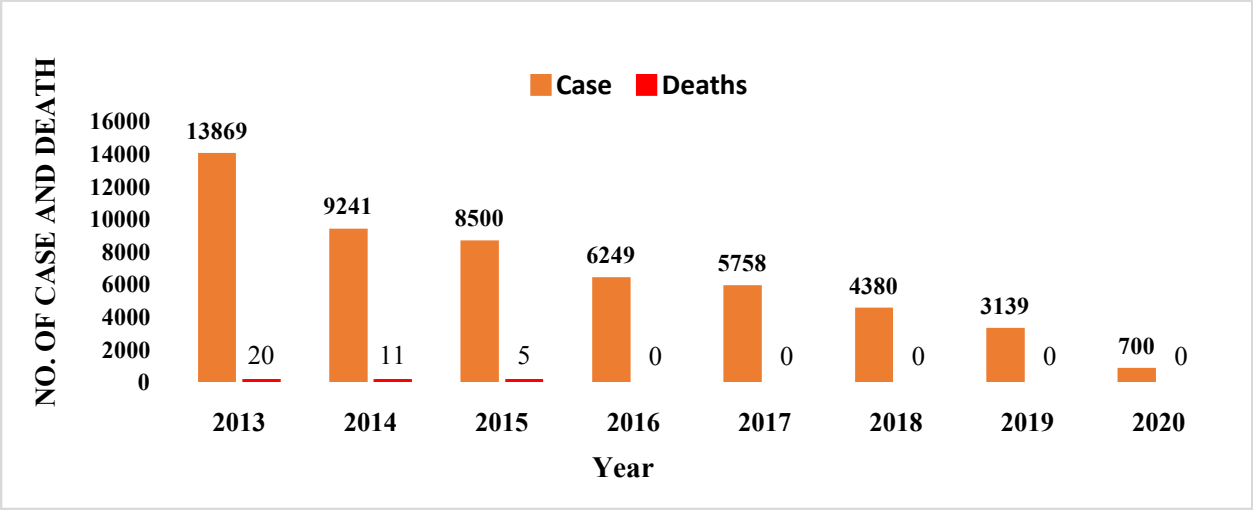
Chikunguniya:
Chikungunya fever is an acute febrile illness caused by an arthropod-borne alphavirus, Chikungunya virus (CHIKV). The virus is primarily transmitted to humans by the bite of an infected *Aedes* species mosquito Chikungunya is a viral tropical disease transmitted also by *Aedes*mosquitoes. It is relatively uncommon and poorly documented. The disease has been found in Africa, Asia, and on islands in the Caribbean, Indian and Pacific Oceans. Typical symptoms are an acute illness with fever, skin rash and incapacitating joint pains that can last for weeks. The latter distinguishes chikungunya virus from dengue, which otherwise shares the same vectors, symptoms and geographical distribution. There is no cure or commercial vaccine for the disease. Most patients recover fully but, in some cases, joint pain may persist for several months or even years. As with dengue, the only method to reduce transmission of chikungunya virus is to control vector mosquitoes and protect against mosquitoes bites.



Viral Replication during Transmission of Chikungunya
(Source- Echavarria-Consuegra, L., Smit, J. M., & Reggiori, F., 2019**).**

Historically, chikungunya virus (CHIKV) has circulated in Africa, Asia, and the Indian and Pacific Ocean Islands. In 2013, the virus spread to the Americas and caused outbreaks in countries that harbor the vectors, Aedesaegypti and Aedes albopictus. In India epidemic of Chikungunya fever was reported during 60s & 70s, 1963 (Kolkata), 1965 (Pondicherry and Chennai in Tamil Nadu, Rajahmundry, Vishakapatnam and Kakinada in Andhra Pradesh; Sagar in Madhya Pradesh and Nagpur in Maharashtra) and 1973 (Barsi in Maharashtra). Subsequently in 2008, 2009, 2010, 2011 and 2012, 95091, 73288, 48176, 20402 and 15977 suspected Chikungunya fever cases with nil death were reported. During 2013, 18840 suspected Chikungunya cases were reported.

Chikungunya is an important public health concern as the virus continues to emerge into previously non- endemic areas. Most of the intervention strategies have focused on mosquito control and mosquito bite prevention as there is currently no treatment or vaccine for CHIKV infection in humans. Actually success of these intervention strategies depends on social factors such as knowledge, attitudes, and perceptions of the disease. The Government of India has initiated several public health measures to control the epidemic of chikunguniya, including IEC/Behavior Change Communication activities through print, electronic media, inter- personal communication, and outdoor publicity as well as an inter- sectoral collaboration with civil society organizations such as non- governmental organizations. It is important to understand how affected populations are educated regarding its transmission cycle and the importance of control measures to determine what prevention strategies are likely to be successful. Knowledge assessment of people will also be helpful in determining how to allocate optimally, the limited resources available for chikungunuya and vector control, and in evaluating the impact of such activities globally. World Health Organization (W.H.O.) focuses on priority areas for research in Chikungunya and Dengue and has recommended an evaluation of social, cultural and community behavioral practices leading to disease transmission, including Knowledge Attitude and Practices studies. As very few studies have been done in central India on role of health education in chikungunya, therefore the present study was carried out to assess impact of health education intervention regarding the awareness of chikunguniya (Nagar et. al., 2018).



Graph:- Chikungunya Situation in India from 2014-2020.
(Source: NVBDCP, 2020)

Discussion & Conclusion:-

Vector borne diseases are a public health problem, not only for Asia (India) but also for entire world. Due to outbreak of vector borne diseases like Malaria, Dengue, Chikungunya, JE, Kala-Azar and Filariasis, many diseases control programs are carried out by NVBDCP and WHO. There are specific treatments for diseases but no vaccine is developed for some of them. Hence it is necessary to control the factors like environmental and ecological. By implementing high quality immunization and vaccination program, modification of agricultural practices, vector control and improving standard of living, we can decrease the rate of number of patients of vector borne diseases.

References:-

1. Alemayehu, B., &Alemayehu, M. (2017). Leishmaniasis: a review on parasite, vector and reservoir host. *Health Science Journal*, *11*(4), 1.

2. Ali, M.A.M.A. (2013). Identification and characterization of microRNAs and their putative target genes in Anopheles funestus ss.
3. Ahammad, F., Tengku Abd Rashid, T.R., Mohamed, M., Tanbin, S. and Ahmad Fuad, F.A. (2019). Contemporary Strategies and Current Trends in Designing Antiviral Drugs against Dengue Fever via Targeting Host-Based Approaches. Microorganisms, 7(9), p.296.
4. Bhattacharya, S. and Basu, P. (2014). Japanese Encephalitis Virus (JEV) infection in different vertebrates and its epidemiological significance: a Review. International Journal of Fauna and Biological Studies, 1 (6), pp. 32-37.
5. Bora, D. (1999). Epidemiology of visceral leishmaniasis in India. *National Medical Journal of India*, *12*, 62-68.
6. Center for Disease Control and Prevention. 2018. National Center for Emerging and Zoonotic Infectious Diseases, (NCEZID), Division of Vector-Borne Diseases (DVBD). www.cdc.gov.in.
7. Christophides, G. K., & Crisanti, A. (2013). Vector and vector-borne disease research: need for coherence, vision and strategic planning. *Pathogens and global health*, *107*(8), 385.
8. Echavarria-Consuegra, L., Smit, J. M., & Reggiori, F. (2019). Role of autophagy during the replication and pathogenesis of common mosquito-borne flavi-and alphaviruses. *Open biology*, *9*(3), 190009.
9. Gupta, E., & Ballani, N. (2014). Current perspectives on the spread of dengue in India. *Infection and drug resistance*, *7*, 337.
10. Hailu, A., Musa, A.M., Royce, C. and Wasunna, M. 2005. Visceral leishmaniasis: new health tools are needed. PLoS medicine, 2(7).
11. Kalluri, S., Gilruth, P., Rogers, D., & Szczur, M. (2007). Surveillance of arthropod vector-borne infectious diseases using remote sensing techniques: a review. *PLoS Pathog*, *3*(10), e116.
12. Khan, A. M., Dutta, P., Das, S., Pathak, A. K., Sarmah, P., Hussain, M. E., &Mahanta, J. (2015). Microfilarial periodicity of Wuchereriabancrofti in Assam, northeast India. *Journal of vector borne diseases*, *52*(3), 208.
13. Khan, M. A. H. N. A. (2015). Important vector-borne diseases with their zoonotic potential: present situation and future perspective. *Bangladesh Journal of Veterinary Medicine*, *13*(2), 1-14.
14. Kuehn, B. M. (2014). Chikungunya virus transmission found in the United States: US health authorities brace for wider spread. *Jama*, *312*(8), 776-777.
15. Morin, C. W., Comrie, A. C., & Ernst, K. (2013). Climate and dengue transmission: evidence and implications. *Environmental health perspectives*, *121*(11-12), 1264-1272.
16. Nagar, V., Gupta, S. K., Yadav, K., Kale, S., & Prasad, P., 2018. Impact of health education intervention regarding the awareness of chikunguniya. GJMEDPH 2018; Vol. 7, issue 2.
17. Patel, A. B., Rathod, H., Shah, P., Patel, V., Garsondiya, J., & Sharma, R. (2011). Perceptions regarding mosquito borne diseases in an urban area of Rajkot city. *Natl J Med Res*, *1*(2), 45-47.
18. Reisen, W. K. (2010). Landscape epidemiology of vector-borne diseases. *Annual review of entomology*, *55*, 461-483.
19. Rigau-Pérez, J. G., Clark, G. G., Gubler, D. J., Reiter, P., Sanders, E. J., &Vorndam, A. V. (1998). Dengue and dengue haemorrhagic fever. The Lancet, 352(9132), 971-977.
20. Roy, M., Bouma, M. J., Ionides, E. L., Dhiman, R. C., &Pascual, M. (2013). The potential elimination of Plasmodium vivax malaria by relapse treatment: insights from a transmission model and surveillance data from NW India. *PLoSNegl Trop Dis*, *7*(1), e1979.
21. Sharma, R. C., &Gautam, A. S. (1990). Studies on outbreak of malaria in Muliad village of Kheda district, Gujarat. *Indian journal of malariology*, *27*(3), 157-162.
22. Shragai, T., Tesla, B., Murdock, C. and Harrington, L.C. (2017). Zika and chikungunya: mosquito-borne viruses in a changing world: Global change and vectors of chikungunya and Zika. Annals of the New York Academy of Sciences.
23. Srivastava, H. C., Sharma, S. K., Bhatt, R. M., & Sharma, V. P. (1996). Studies on Plasmodium vivaxrelapse pattern in Kheda district, Gujarat. *Indian journal of malariology*, *33*(4), 173-179.
24. Srinivas, V., & Srinivas, V. R. (2011). Dengue fever: A review article. *Indian J Med Res*.
25. Tiwari, S., Singh, R. K., Tiwari, R., & Dhole, T. N. (2012). Japanese encephalitis: a review of the Indian perspective. *Brazilian Journal of Infectious Diseases*, *16*(6), 564-573.
26. Trampuz, A., Jereb, M., Muzlovic, I., & Prabhu, R. M. (2003). Clinical review: Severe malaria. *Critical care*, *7*(4), 315.
27. Tuiskunen Bäck, A., & Lundkvist, Å. (2013). Dengue viruses–an overview. *Infection ecology & epidemiology*, *3*(1), 19839.
28. Vaddadi, S., &Vaddadi, R. S. (2015). Dengue fever: A review article. *J. Evol. Med. Dent. Sci*, *4*, 5048-5058.

29. Wang, E., Ni, H., Xu, R., Barrett, A. D., Watowich, S. J., Gubler, D. J., & Weaver, S. C. (2000). Evolutionary relationships of endemic/epidemic and sylvatic dengue viruses. *Journal of virology*, *74*(7), 3227-3234.
30. Westaway, E. G. (1985). Brinton. *MA, GAIDAMOVICH, SYA, HORZINEK, MC, IGARASHI, A., K&~ RI&NEN. L., Lvov, DK, PORTERFIELD, JS, RUSSEL, PK, and TRENT, DW*, 24-125.
31. Wilson, A. J., Morgan, E. R., Booth, M., Norman, R., Perkins, S. E., Hauffe, H. C., ...& Fenton, A. (2017). What is a vector?. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *372*(1719), 20160085.
32. World Health Organization, 2009. Dengue Guidelines For Diagnosis, Treatment, Prevention And Control. WHO.
33. World Health Organization, 2012. Neglected tropical diseases. WHO.
34. World Health Organization, 2014. A global brief on vector-borne diseases. WHO.