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"A systemic review: Yellow Fever"

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ABSTRACT:
Yellow fever is a viral hemorrhagic fever with a high mortality which is caused be yellow fever virus (YFV) and spread by the bite of an infected Aedes aegypti mosquito. It affects people and non-human primates in tropical areas of Africa and South America and is a severe concern and also sometimes in temperate zone. Fever, chills, headache, backache, nausea, and vomiting are all symptoms of yellow fever. The skin and eyes may appear yellow, which is known as jaundice and is the symptom that gives the disease its common name. The virus responsible for yellow fever belongs to family of Flavivirus. There are three transmission cycles of yellow fever including sylvatic(jungle), intermediate and urban.The clinical manifestations of yellow fever comprises of subclinical infection, abortive illness without jaundice and life threatening disease. Blood tests that reveal the presence of virus antibodies, as well as the patient's history of visiting an endemic location, are commonly used to make the diagnosis of yellow fever. There are no specific treatments are available for treating tyellow fever hence, only taking preventive measures like vaccination helps in controlling the symptoms. Massive immunisation and vector control can help to prevent yellow fever. In locations where the disease is endemic, the entire community should be vaccinated.

KEYWORDS: Yellow fever, Hemorrhagic, Aedes aegypti, mortality, Flavivirus, sylavatic, vaccination.

I. INTRODUCTION

INTRODUCTION ABOUT YELLOW FEVER

ETIOLOGY

Yellow fever is caused due to yellow fever virus (YFV) and spread by bite of infected Aedes aegypti mosquito.It is a viral hemorrhagic fever resulting in high mortality rate. Although huge outbreaks have occurred in Europe and North America in the past, the illness now only affects Africa, Central and South America, and the Caribbean. Mosquitoes capable of spreading yellow fever exist in areas where the disease does not currently exist and in areas where the disease has never been, such as Asia[1]. The YFV is the prototype of the Flavivirus genus, which belongs to the Flaviviridae family and has genetic material made up of single-stranded positive-sense RNA[2]. The genome of the yellow fever virus predominantly encodes capsomere and attachment proteins. Because it is an RNA virus, the genome contains a replication protein that aids in virus replication[3].Three structural, seven nonstructural, and NS5) proteins are encoded by its genome. Although just one YFV serotype has been identified, there are seven genotypes with minor genetic differences, two from the Americas (South and five from Africa), but no differences in clinical presentation have been reported[2,3].

After three days, the virus begins to replicate in the mosquito's body, first in the cells of the digestive system and fat cells, then in the neurological system, salivary glands, and reproductive system. The life cycle of Aedes mosquitos is short[3].

Within a mosquito, a complex biological process takes place that successfully transmits a pathogen, such as a virus, from one host to another. To begin, the mosquito must be drawn to a specific host. Mosquitoes have a wide range of host preferences. Some insects, such as Aedes aegypti, prefer human blood and will overlook other blood sources if they can locate one [4]. Forest monkeys hold the virus in natural reservoirs, and mosquitoes disseminate it to humans. The disease, known as acute hemorrhagic fever, affects humans, all monkey species, and a few other small mammals, and is spread by several mosquito species [5].

There are three transmission cycles of yellow fever virus including Jungle (sylvatic), Intermediate (Savannah), and Urban.Human-to-human transmission of yellow fever occurs through the biting of the Aedes aegypti mosquito, which thrives in unpolluted urban water and bites primarily during the day[6]. The transmission from

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mammalian host (usually monkey) to human occurs in jungle yellow fever, carried by a variety of mosquito species: Haemagogus and Sabethes spp. in South America; Aedes africanus in East Africa; and a variety of Aedes species in West Africa[5,6].Intermediate yellow fever, also known as savannah yellow fever, is spread by a number of "semidomestic" mosquitoes from animal to person and person to person (e.g., A. furcifer, A. taylori) [3,5,6].

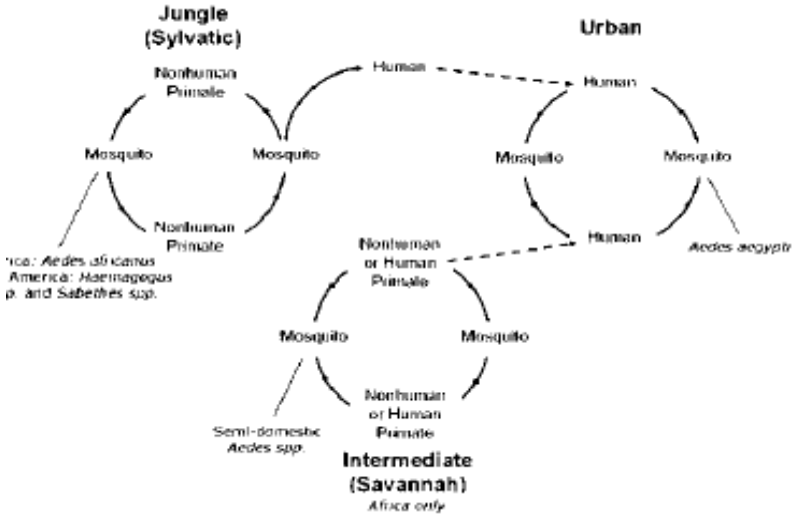

Fig.1 Transmission cycles of Yellow Fever Virus[3]

Yellow fever has a short incubation period. There is a several-day incubation period after the infecting mosquito bites, during which the virus grows within the body. During the incubation period, most people do not notice any signs or symptoms of the disease. Infection or invasion occurs after the incubation period. After the incubation phase ends, this, lasts for two to five days. Viruses are rapidly replicating and spreading throughout the body[3,6,7].

EPIDEMIOLOGY

The growing prevalence of yellow fever in Africa was mostly caused by a succession of epidemics and lesser outbreaks of the disease in West African countries, but the first epidemic reported in Kenya showed that the disease's distribution was changing as well [8]. Although the incidence of endemic disease is unknown, yellow fever is thought to be responsible for about 1% of severe hepatitis cases in endemic parts of Africa [1,8].

In Africa, transmission is sustained by a dense population of vector mosquitos living in close proximity to mainly uninfected human populations. Although the yellow fever vaccine has been included in certain nations' paediatric immunisation regimens, vaccine coverage is not perfect[4]. Yellow fever is a disease that arises in epidemics and has a wide range of occurrence throughout Africa. Between December 2015 and July 2016, a massive Aedes aegypti–borne epidemic swept Angola and the adjacent Democratic Republic of the Congo in south/central Africa, resulting in over 2930 confirmed or suspected cases and 253 deaths, as well as the emergency distribution of 30 million vaccine doses[4,8].

Yellow fever transmission is lower in South America than it is in Africa, in part because high vaccination coverage occurs predominantly as part of mass immunisation efforts in reaction to illness outbreaks.

Recent human cases in South America demonstrate sylvatic transmission, which involves virus circulation among nonhuman primates and overflow into the human population, transmitted by mosquitos common in forested areas (such as Haemagogus and Sabethes spp)[4].In 1995, Peru had the greatest outbreak of yellow fever in South America since the 1950s, with cases reported in Bolivia, Brazil, Colombia, Ecuador, and Peru between 1985 and 1994 [1].

Due to underreporting of the disease (particularly in remote locations), limits of passive monitoring, a lack of diagnostic competence in many areas where yellow fever is common, and the frequency of asymptomatic infection, accurate data on the disease's burden are difficult to come by.As a result of these difficulties, support for

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immunisation programmes as the cornerstone of prevention is growing [9,10].

Unvaccinated travellers have contracted yellow fever. In unimmunized visitors from the United States and Europe, 9 cases were documented between 1970 and 2002; illness was acquired in Brazil (3 cases), Senegal (2 cases), Venezuela, Ivory Coast, Gambia, and West Africa. The mortality rate was 89%. Another case occurred in 1987, when a Spanish immunise traveller visited four West African countries[11,12].

The last yellow fever pandemic in Brazil was reported in Rio de Janeiro state in 1929, and the final yellow fever urban case was identified in Acre state in 1942[13].

Many outbreaks and epidemics have occurred, including in Nigeria (1984 onward), Cameroon (1990), Kenya (1992–1993), Ghana (1993–1994), Gabon (1994–1995), Liberia (1995, 2000), Senegal (1995–1996, 2002), Benin (1996), Guinea (2000), Côte d'Ivoire (2001), and Sudan (2003)[11].

Case of yellow fever in unvaccinated travelers have been reported in **Table 1.** From the United States and Europe since 1996. The majority of people become sick on short-term visits, with some staying in endemic areas for less than a week. They estimate the risk of yellow fever illness to be 1:267 and the chance of death to be 1:1333 for a 2-week visit to a location in Africa where there is an outbreak of yellow fever. For a 2-week visit in Africa during an interepidemic period, the chance of sickness is 1:2000 and death is 1:10,000[11,12].

Table 1. Reported cases of Yellow Fever in unvaccinated travellers

Year	Country of Residence	Age	Sex	Place of Exposure	Outcome	Reference
1979	France	42	M	Senegal	Died	12
1985	The Netherlands	27	F	Guinea-Bissau, The Gambia, Senegal	Survived	15
1996	Switzerland	53	M	Brazil	Died	14
1996	United States	45	M	Brazil, Rio Negro, Amazon River	Died	11
1999	Germany	40	M	Ivory Coast	Died	15
2001	Belgium	47	F	The Gambia	Died	16
2002	United States	47	M	Brazil, Rio Negro	Died	17

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Fig. 2: Areas in Africa where yellow fever is endemic, 2005[1].

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Fig.3: Areas in Central and South America where yellow fever is endemic, 2005 [1].

II. CLINICAL MANIFESTATIONS

The clinical spectrum of yellow fever include sbubclinical infection, abortive (nonspecific fabrile) illness without jaundice and life threatening diseases [18].The wide range of clinical symptoms is likely due to differences in viral strains as well as poorly understood host immunological mechanisms. Viremia peaks 2–3 days after infection, and viremia lasts longer in fatal cases than it does in survivors[1].

Yellow fever affects people of all ages, but the intensity and fatality of the disease are highest among the elderly. The sickness begins three to six days (median 4.3 days) after being bitten by an infected mosquito[19].

There are three phases of clinical yellow fever including:

PERIOD OF INFECTION:

The infection is characterised by viremia, in which virises are present in blood and which lasts three to four days. The patient has a fever and is suffering from widespread malaise, headaches, photophobia, lumbosacral pain, lower extremity pain, myalgia, anorexia, nausea, vomiting, restlessness, irritability, and dizziness. Yellow fever has a wide range of symptoms and indications, making it difficult to identify it from other acute diseases at this stage [1,8].

The patient has flushed skin, reddened conjunctivae and gums, and epigastric discomfort on physical examination. There may be soreness and enlargement of the liver. The tongue has a white coating in the centre and is crimson at the tip and sides. Faget's sign is a modest pulse rate compared to the intensity of the fever. The average temperature is 39°C, however it can reach 41°C in the summer.

Leukopenia, which is present at the start of the disease, and an increase in serum transaminase levels on days 2–3 of the sickness, before the onset of jaundice, are two laboratory abnormalities [1].

PERIOD OF REMISSION

Following the period of infection, a period of remission lasting up to 48 hours may occur, defined by the absence of fever and symptoms. At this point, patients who have had abortive infections recover. Approximately 15% of people infected with the yellow fever virus progress to the third stage of the illness[8].

PERIOD OF INTOXICATION

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The third phase of yellow fever consists of return of fever, nausea, vomiting, jaundice, and bleeding diathesis. Although continuous viremia has been observed, the viremia fades at this stage and antibodies emerge in the blood[20]. The participation of many organ systems is common. Serum transaminase levels are proportionate to the severity of the sickness; in patients who recover, they peak early in the second week of illness. High levels of proinflammatory cytokines are linked to multiorgan failure in yellow fever[20,21].

Hepatic dysfunction: Yellow fever hepatic dysfunction is distinct from other viral hepatitides in that serum aspartate aminotransferase (AST) levels are higher than alanine aminotransferase (ALT) levels (ALT). This could be related to myocardial and skeletal muscle damage caused by the virus. The severity of the condition is reflected in the levels[8].

Renal Dysfunction: Oliguria, azotemia, and very high protein levels in the urine are all signs of renal impairment. Creatinine levels in the blood are three to eight times higher than normal. Renal failure predominates in certain patients who survive the hepatic phase[22].

Haemorrhage: The third phase of sickness is characterised by haemorrhage. Circulatory collapse may be exacerbated by gastrointestinal bleeding. Thrombocytopenia, a prolonged prothrombin time, and a general reduction in clotting factors generated by the liver are among the laboratory abnormalities[8].

Myocardial injury: Myocardial injury's clinical relevance is poorly understood, and clinical investigations have likely underestimated it. Acute cardiac enlargement has been recorded in some cases during the course of illness. Extrasystoles, sinus bradycardia without conduction problems, ST-T abnormalities, particularly increased T waves, and extrasystoles may all be seen on the ECG[23].

Central nervous system dysfunction: Delirium, agitation, convulsions, stupor, and coma are some of the symptoms of central nervous system (CNS) dysfunction. The cerebrospinal fluid is under heightened pressure in extreme cases, and it may include high protein but no cells. Microscopic perivascular haemorrhages and edoema are pathologic alterations[8].

The patient's prognosis is established during the second week following onset, when he or she either dies or recovers quickly. The condition kills 20 to 50 percent of individuals who enter the period of intoxication. Anuria, shock, hypothermia, agitation, delirium, intractable hiccups, seizures, hypoglycemia, hyperkalemia, metabolic acidosis, Cheyne-Stokes respirations, stupor, and coma are all poor prognostic indicators.

Convalescence is often accompanied by weariness that lasts several weeks. Although these patients may have yellow fever superimposed on other hematologic or hepatic illnesses, jaundice and serum transaminase increases can last months in some circumstances. Yellow fever appears to have the same outcome in people with and without hepatitis B surface antigenemia.

Bacterial superinfections, such as pneumonia, parotitis, and sepsis, are common complications of yellow fever. Myocarditis, arrhythmia, and heart failure have all been linked to late fatalities during convalescence[8].

III. DIAGNOSIS AND TREATMENT:

DIAGNOSIS:

Diagnosis of yellow fever is difficult specially in early stages. According to WHO recommendations, any country at risk of yellow fever outbreaks/endemics should establish at least one national laboratory for illness diagnosis, as well as training programmes to assist medical personnel and public health officials in performing and understanding the tests [24]. Yellow fever can be diagnosed with the help of special laboratory tests such as serology, Polymerase Chain Reaction (PCR) in blood and urine, by viral isolation or histopathology, and immunohistochemistry on postmortem tissues.

Serology: An enzyme-linked immunosorbent test (ELISA) for immunoglobulin (Ig)M and microsphere-based immunoassay (MIA) are the best way to make a serologic diagnosis. Antibodies attached to a substrate are used in both approaches to identify and detect specific types of proteins. The antibodies employed in ELISA and MIA bind to yellow fever attachment proteins and antibodies that are unique to the virus. A rise in titer between matched acute and convalescent samples, or a decline between early and late convalescent samples, offers a tentative diagnosis; confirmation is obtained by a reduction in titer between early and late convalescent samples[3,8].Other kinds of examinations To detect viral proteins or antibodies, serological tests use blood cells and immune system components known as complements. Both tests include methods for identifying whether viral proteins or antibodies adhere to the test chemicals[3].

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Rapid Diagnostic tests:PCR is used to detect viral genomes in blood or tissue, while ELISA is used to determine IgM antibody levels[25]. Even in severe infections, the viral genome is found in extremely low amounts in the body, therefore a polymerase chain reaction (PCR) is used to create numerous copies of the viral RNA. Because PCR was designed to replicate DNA, a unique modification of the PCR reaction called RNA amplification is used. The RNA is then examined using electrophoresis, a technique that allows molecules to be separated by size and electrical charge for identification. This method is effective in discriminating between the many strains of yellow fever viruses[3].A yellow fever diagnostic test based on reverse transcription loop-mediated isothermal amplification (RT-LAMP), which does not require thermocycling equipment and can be read visually, has showed promise as a sensitive and rapid field test [26].

Virological diagnosis:

Molecular diagnostics: Molecular approaches such as conventional (end-point) or real-time reverse transcription polymerase chain reaction (RT-PCR) can identify viral RNA in serum samples throughout the first 10 days after the beginning of symptoms (viremic phase) or even longer in severe instances. A positive result from molecular testing validates the diagnosis of YFV infection (when used with the proper controls and interpretation)[27].

Viral isolation: Viral isolation can be accomplished in mice via intracerebral injection or in cell culture. It's also possible to recover the virus from postmortem liver tissue. This methodology is rarely employed as a first-line diagnostic tool due to its intricacy. However. The ability to isolate viruses is critical for characterisation of circulating strains, production of diagnostic reagents, and research studies [8,27].

Immunohistochemistry: The "gold standard" technique for diagnosing yellow fever in fatal cases is histopathological investigation with immunohistochemistry done on liver slices (and other tissues). Molecular detection can also be used to confirm fatal instances in fresh or formalin-fixed (paraffin-embedded) tissue samples [3,8,27].

Pathology: A liver biopsy should never be conducted during a yellow fever infection because it can result in deadly bleeding. The classic signs of yellow fever, such as midzonal necrosis, are frequently seen in postmortem histopathologic examinations of the liver. Immunocytochemical staining for yellow fever antigen in the liver, heart, spleen, or kidney can provide a definitive postmortem diagnosis[27,28].

TREATMENT:

Antiviral drugs are not available to treat yellow fever. Supportive treatment is provided so that the patient feels at ease and the disease's effects are minimised. Giving plenty of water to make up for fluids lost during vomiting is standard supportive treatment. Oxygen is also given to compensate for the loss of gas exchange due to the injured lungs[3]. Blood pressure and heart rate can both be controlled with medications. Severe yellow fever patients may require blood and renal dialysis. Some patients require blood transfusions to replace proteins that aid in blood coagulation and the healing of wounds caused by haemorrhaging.

IV. PREVENTION

VACCINATION:

Vaccination is the most efficient way to prevent yellow fever in epidemic areas, with seroconversion rates of up to 95%[29]. A single vaccination dose will be enough to establish long-term protection using the 17D Live attenuated vaccine strain.A booster dose of yellow fever vaccine is not needed[30].

To prevent outbreaks, several vaccine tactics are utilised, including routine baby immunisation, mass vaccination campaigns to enhance coverage in at-risk countries, and vaccination of travellers visiting yellow fever-endemic areas. The most successful public health technique for reducing yellow fever epidemics is a mass vaccination programme [29].

Rapid detection and control of epidemics through mass immunisation is crucial in high-risk areas where vaccination coverage is poor. In a place where there is a yellow fever outbreak, it is critical to vaccinate the majority of the people at risk (at least 80%) [7].

Vaccine Development:

The 17D strain is the source of all contemporary yellow fever vaccinations.During the early years of yellow fever vaccine manufacture in the United States and Brazil, between 1937 and 1941, two primary lineages of the 17D line (17D–204 and 17DD) were used[1].

Vaccine response:

Yellow fever vaccination provides excellent protection, with seroconversion rates of >95 percent in both children and adults and a ≥10-

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year immunity duration [9]. Neutralizing antibodies develop in 90% of vaccination recipients within 10 days of inoculation, and in 99 percent within 30 days[31].

Mild viremia develops 3–7 days after the first dose of yellow fever vaccination is administered and lasts 1–3 days. Increases in IFN-a, TNF-a, and T-cell activation indicators occur at this time, and are likely mediators of the yellow fever vaccine's usual moderate side effects. As neutralising antibodies form, the viremia disappears. With future vaccine doses, viremia does not occur, and side effects are less severe[1].

There have been rare reports of serious side-effects from the yellow fever vaccine.

Common adverse effects

Yellow fever vaccine side effects are often modest, and include headaches, myalgia, and low-grade fever, which occurred 5–10 days after immunisation in about 25% of people who took part in clinical studies [32,33].People over 60 years old, as well as those who have significant immunodeficiency due to symptomatic HIV/AIDS or other causes, or who have a thymus disease, are at a higher risk of adverse effects. After a comprehensive risk-benefit analysis, anyone over the age of 60 should be administered the vaccine [7].

Severe adverse effects

Beginning in 1996, cases of severe multiorgan failure following yellow fever vaccination administration were recorded, raising medical community awareness of the vaccine's side effects. Immediate hypersensitivity reactions, neurotropic disease, and viscerotropic disease are the three types of serious side events linked to yellow fever vaccine[1]

Indications, Precautions and contraindications for yellow fever immunization

Unless there are special contraindications, travellers to countries or regions where there is an elevated risk of yellow fever infection should obtain a single dose of vaccination at least 10 days before departure[34].Yellow fever vaccine must be administered at official yellow fever vaccine sites and must be accompanied by an International Certificate of Vaccination (valid from 10 days through 10 years after the date of immunization)[35]. Vaccines are prohibited in newborns and infants under the age of six months, should be administered to infants aged six to nine months only if there is a considerable risk of disease and other measures of protection are unavailable, and should be used with caution in infants aged nine to twelve months[36].

Yellow fever vaccination is not recommended if you have thymus illness [35]. Immunocompromised people should avoid vaccinations as well. Immunization should be offered to symptomatic HIV-infected patients with a CD4 cell count of 1200 cells/mm3 who are at risk of yellow fever infection and cannot avoid exposure [35,37].Individuals over the age of 60 who are inoculated for the first time appear to be at an elevated risk of yellow fever vaccine-related side effects. Individuals who have a family member who has had a serious adverse reaction to the yellow fever vaccine may also be at risk. The use of yellow fever vaccine in such people necessitates a thorough examination of risk during travel and clarification of information.

The safety of yellow fever vaccination during pregnancy has yet to be determined, and little is known regarding the possibility of vaccine-associated viral strains infecting the foetus. There have been no instances of yellow fever vaccination viral transmission from nursing women to their infants, and it is unknown whether the virus is secreted in breast milk. Immunization is recommended for lactating women who travel to areas where yellow fever is endemic[35].

Table 2. Contraindications and Precautions of Yellow fever vaccination[1,7,35,36]

Contraindications
Age, <6 months Thymus disease or history of thymus disease Immunosuppression
Precautions
Age, 6–12 months Age, ≥60 years for first-time vaccinees Pregnancy Lactation Asymptomatic HIV infection with laboratory verification of adequate immune system function Hypersensitivity to eggs

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Hypersensitivity to gelatin Family history of adverse events associated with yellow fever vaccine

VECTOR CONTROL:

Eliminating possible mosquito breeding sites, such as water storage containers and other places where standing water collects, can minimise the risk of yellow fever transmission in urban settings.

Vector surveillance and control are essential components of vector-borne disease prevention and management, particularly in epidemics. Vector surveillance targeting Aedes aegypti and other Aedes species for yellow fever will assist determine where an urban outbreak is likely to occur[29].Spraying pyrethroid insecticides, larval control using larvicides, insect growth regulators, and bacterial toxins, and biological agents such as predatory copepods, fish, and Toxorhynchites larvae are among the current vector control techniques. Many other strategies are being investigated[38].

PROGNOSIS:

Individuals with simple yellow fever usually have a good prognosis. However, depending on the underlying health of the patient and the availability of supportive services, case fatality rates range from 20% to 50% for those who develop the toxic phase of yellow fever [3,39]. Death usually occurs 10-14 days after the hazardous phase begins. Infants and people over the age of 50 are more likely to get sick and die. In addition, the susceptibility of the infecting strain and its virulence can affect fatality rates. There is usually no long-term organ damage in people who survive yellow fever [39].

V. CONCLUSION

Yellow fever is a virus-borne hemorrhagic disease spread by mosquitos. In most African and South American nations, the disease is endemic. The yellow fever transmission involves three phases including urban, intermediate and jungle. The clinical menifestations of yellow fever consists of period of infection, period of remission and period of intoxication. Yellow fever can be diagnosed with the help of diagnostic tests like serology, Polymerase chain reaction, immunohistochemistry, pathology. There are no specific antiviral drugs available for yellow fever treatment. Vaccination is the best way to prevent yellow fever. Yellow fever vaccination and disease vector control should be available in countries with a high risk of the disease. Yellow fever outbreaks have continued and expanded into new locations in recent years, despite the availability of a very effective vaccine.

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