EDITORIAL

DENGUE FEVER

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The viral etiology of dengue was established by the 1940s, and records of dengue-like illness date back more than 200 years. Major changes in the epidemiology of dengue virus infections began after World War II and have continued to date. Given current estimates of over 100 million infections worldwide each year and over 2.5 billion individuals at risk for infection, the dengue[1].

Both epidemic and endemic transmission of dengue viruses are maintained through a human-mosquito-human cycle involving Aedes. Transmission of dengue viruses between mosquitoes and nonhuman primates has been demonstrated to occur in Asia and Africa, but there is no evidence that such transmission is an important reservoir for transmission to humans. Susceptible humans become infected after being bitten by an infected female Aedes mosquito. Viremia in humans begins towards the end of a four to six-day incubation period and persists until fever abates, which is typically three to seven days. An uninfected Aedes mosquito may acquire the virus after feeding on the subject during this viremic period. The mosquito has an incubation period of 8 to 12 days before it is capable of transmitting the virus to susceptible people. Once infected, mosquitoes carry the virus for their lifespan and remain infective for the rest of life[1].

Aedes aegypti mosquitoes, the principal vector for the transmission of dengue virus, have many characteristics that make them ideal for dissemination of the virus. A. aegypti typically breed in or close to houses, laying eggs in both man-made and natural water containers. The typical flight distance is relatively short. A. aegypti are daytime feeders that prefer to bite humans and are frequently unnoticed. They are easily interrupted in their feeding and move on to another host, frequently taking multiple blood meals in a single breeding cycle. Thus, an infected A. aegypti mosquito may transmit dengue virus to several individuals in a small area. For these reasons, family members who are at home during the day, typically women and young children are thought to be at particularly high risk for infection.

The worldwide incidence of dengue and DHF has been increasing in the past several decades, and the geographic distribution of these diseases has expanded. The emergence of DHF as a public health problem has largely been a result of human behaviors including population growth, poorly planned urbanization, associated with overcrowding, poor water distribution, and poor sanitation, changing lifestyles, such as increased reliance on plastic containers and tires; standing water can easily collect in these modern transportation, with increased movement of viruses, mosquitoes, and susceptible humans, lack of effective mosquito control.

Aegypti are widely distributed in India, Pakistan, and Sri Lanka. Dengue virus transmission, particularly in India and Sri Lanka, increased substantially during the 1980s and 1990s. Pakistan and India experienced major outbreaks of dengue in 2007. In 2010, due to torrential monsoon raining in Pakistan and the subsequent flooding has resulted in mosquito over breeding has resulted in the current epidemic in Punjab and Sindh.

The clinical manifestations of dengue range from self-limited dengue fever to dengue hemorrhagic fever with shock syndrome, which carries a high mortality rate. The risk of severe disease is much higher in sequential rather than primary dengue infection. • Classic dengue fever (DF) is an acute febrile illness accompanied by headache, retroorbital pain, and marked muscle and joint pains, which evoked the term "breakbone fever. Fever typically lasts for five to seven days. Hemorrhagic manifestations can also occur in patients with DF. Physical examination is non-specific, but may include a macular or maculopapular rash in approximately 50 percent of cases• Dengue hemorrhagic fever (DHF) is the most serious manifestation of dengue virus infection and can be associated with shock. The four cardinal features of DHF include increased vascular permeability, fever, hemorrhage, and marked thrombocytopenia (less than 100,000 cells/mm3). Plasma leakage is the most specific and life-threatening feature of DHF and usually occurs rapidly over a period of hours and can lead to shock. Severe plasma leakage can occur in patients with minimal hemorrhagic manifestations. Hemorrhagic manifestations of dengue virus infection can range from spontaneous petechiae to profuse bleeding.

The diagnosis of acute dengue virus infection is based mainly on clinical signs and symptoms in endemic countries. In countries where serologic assays are available, it is recommended to check an acute phase serum plasma sample for use in an IgM immunoassay. If the clinical suspicion of dengue virus infection is high and the assay results are negative, to have the test in 2 weeks time. The most frequently used serologic tests for the diagnosis of acute dengue virus infection are the hemagglutination inhibition (HI) assay and IgG or IgM enzyme immunoassays.

Complement fixation and neutralizing antibody assays are more technically demanding and are used in specialized laboratories only.

Up till now there is no vaccine available for dengue fever. Since there is no specific therapy available for dengue virus infections, it is important to exclude other treatable diagnoses. Patients at risk for dengue can acquire other diseases with similar clinical features, such as malaria, typhoid fever, and leptospirosis. Patients with dengue fever should be cautioned to maintain their intake of oral fluid to avoid dehydration. Fever and myalgias can be managed as needed with acetaminophen. Aspirin or nonsteroidal antiinflammatory agents should generally be avoided because of the risk of bleeding complications and in children because of the potential risk of Reye's syndrome. Gastrointestinal bleeding or menorrhagia in patients with DHF, and occasionally in patients with dengue fever as well, can be severe enough to require blood transfusion. Factors that contribute to bleeding include thrombocytopenia due to decreased platelet survival and, in severe cases, frank disseminated intravascular coagulation. Plasma leakage in DHF is important to manage with aggressive intravascular volume repletion to prevent or reverse hypovolemic shock. In mild cases, particularly when medical attention is received early, oral rehydration may be sufficient. However, in patients with established intravascular fluid loss, intravenous fluid administration is recommended. Blood transfusion is appropriate in patients with significant bleeding; subsequent hematocrit measurements must be interpreted with caution since it is also critical to assess the adequacy of fluid repletion. A protocol for intravenous fluid therapy has been developed by the World Health Organization (WHO) based upon clinical experience mainly in children from Southeast Asia. For patients with hypotensive shock, an initial bolus of five percent dextrose in normal saline or Ringer's lactate (20 mL per kg of body weight) infused over 15 minutes is recommended, followed by continuous infusion (10 to 20 mL/kg per hour depending on the clinical response) until vital signs and urine output normalize. For patients who improve, the infusion rate should then be gradually reduced until it matches plasma fluid losses.

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