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E - NEWS LETTER

Jay Jalaram Ayu Pariwar

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Patron Message-

In year 1991, International Diabetes Foundation and World Health Organizations declared World Diabetes Day on 14th November. This is day delicated to the inventours of Insulin, Frederick Banting and Charles Best. We organized different awareness program for Diabetic care and life style modification as per Ayurveda.

About 422 million people worldwide have diabetes, the majority living in low and middle income countries, and 1.5 million deaths are directly attributed to diabetes each year . Both the numbers of cases and the prevalence of diabetes have been steadily increasing over the past few decades. It creates great burdens on health budget.

Prevention is better than cure is the utmost principle of Ayurveda. We have to follow Dincharya(daily regiment) and Rutucharya(seasonal regiment) to overcome this type of non-comunicable dieases. Ayurveda have great supremacy for controlling and treatment of NCDs

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From Editors Desk:

Abstract

Dengue is an acute viral illness caused by RNA virus of the family Flaviviridae and spread by Aedes mosquitoes. Presenting features may range from asymptomatic fever to dreaded complications such as hemorrhagic fever and shock. A cute-onset high fever, muscle and joint pain, myalgia, cutaneous rash, hemorrhagic episodes, and circulatory shock are the commonly seen symptoms. Oral manifestations are rare in dengue infection; however, some cases may have oral features as the only presenting manifestation. Early and accurate diagnosis is critical to reduce mortality. Although dengue virus infections are usually self-limiting, dengue infection has come up as a public health challenge in the tropical and subtropical nations. This article provide a detailed overview on dengue virus infections, varied clinical manifestations, diagnosis, differential diagnosis, and prevention and treatment.

Introduction

The dengue virus, a member of the genus *Flavivirus* of the family Flaviviridae, is an arthropode-borne virus that includes four different serotypes (DEN-1, DEN-2, DEN-3, and DEN-4).

The World Health Organization (WHO) consider dengue as a major global public health challenge in the tropic and subtropic nations. Dengue has seen a 30-fold upsurge worldwide between 1960 and 2010, due to increased population growth rate, global warming, unplanned urbanization, inefficient mosquito control, frequent air travel, and lack of health care facilities. Two and a half billion people reside in dengue-endemic regions and roughly 400 million infections occuring per year, with a mortality rate surpassing 5–20% in some areas. Dengue infection affects more than 100 countries, including Europe and the United States (USA). The first reported case of dengue like illness in india was in Madras in 1780, the first virologically proved epidemic of DF in India occurred in Calcutta and Eastern Coast of India in 1963-1964. Dengue virus infection presents with a diverse clinical picture that ranges from asymptomatic illness to DF to the severe illness of dengue hemorrhagic fever/dengue shock syndrome (DHF/DSS). Oral mucosal involvement is seen in approximately 30% of patients, although oral features are more frequently associated with DHF than with DF.[9Dengue virus infection exhibit varied clinical presentation, hence, accurate diagnosis is difficult and relies on laboratory confirmation. The condition is usually self-limiting and antiviral therapy is not currently available. Supportive care with analgesics, hydration with fluid replacement, and sufficient bed rest forms the preferred management strategy.

Etiopathogenesis:-

DF is a severe flu-like infection that involves individuals of all age groups (infants, children, adolescents, and adults).Transmission among human beings occurs by the mosquito *Aedes aegypti* and chiefly occurs during the rainy season.

The proposed etiologies for dengue virus infection are:Viral replication, primarily in macrophages

Direct skin infection by the virus

Immunological and chemical-mediated mechanism induced by host–viral interaction.Dengue virus gains entry into the host organism through the skin following an infected mosquito bite. Humoral, cellular, and innate host immune responses are implicated in the progression of the illness and the more severe clinical signs occur following the rapid clearance of the virus from the host organism. Hence, the most severe clinical presentation during the infection course does not correlate with a high viral load. Alterations in endothelial microvascular permeability and thromboregulatory mechanisms lead to an increased loss of protein and plasma. Proposed theories suggest that endothelial cell activation caused by monocytes, T-cells, the complement system, and various inflammatory molecules mediate plasma leakage. Thrombocytopenia may be related to alterations in megakaryocytopoiesis, manifested by infection of human hematopoietic cells and compromised progenitor cell growth. This may cause platelet dysfunction, damage, or depletion, leading to significant hemorrhage.

Classification:-

The WHO classifies DF into two groups: Uncomplicated and severe.Severe cases are linked to excessive hemorrhage, organ impairement, or severe plasma escape, and the remaining cases are considered uncomplicated.

According to the 1997 classification, dengue can be divided into undifferentiated fever, DF, and DHF. DHF was further subdivided into grades I–IV.

Grade I: Only mild bruising or a positive tourniquet test

Grade II: Spontaneous bleeding into the skin and elsewhere

Grade III: Clinical sign of shock

Grade IV: Severe shock - feeble pulse, and blood pressure cannot be recorded.

Here, grades III and IV comprise DSS.

Clinical Feature

Undifferentiated fever

This stage is seen mostly in the primary infection but may also occur following the initial secondary infection. Clinically, it is difficult to differentiate from numerous other viral diseases and often remains undiagnosed.

Dengue fever

DF follows both primary and secondary infections, and is most frequently encountered in adults and older children. Onset of symptoms is characterized by a biphasic, high-grade fever lasting for 3 days to 1 week. Severe headache (mainly retrobulbar), lassitude, myalgia and painful joint, metallic taste, apetite loss, diarrhea, vomiting, and stomachache are the other reported manifestations. Dengue is also known as breakbone fever because of the associated myalgia and pain in joints. Of patients with DF, 50–82% report with a peculiar cutaneous rash. The initial rash is the result of capillary dilatation, and presents as a transient facial flushing erythema, typically occuring before or during the first 1–2 days of fever. The second rash is seen at 3 days to 1 week following the fever, and presents as a asymptomatic maculopapular or morbilliform eruption. Sometimes, individual lesions may merge and present as widespread confluent erythematous areas with pinpoint bleeding spots and rounded islands of sparing, giving a typical appearance of "white islands in a sea of red." The cutaneous rash is usually asymptomatic, and pruritis is reported only in 16-27% cases. Bleeding episodes are infrequently seen in DF, although epistaxis and gingival bleeding, substantial menstruation, petechiae/purpura, and gastrointestinal tract (GIT) hemorrhage can occur.

Dengue hemorrhagic fever

DHF is frequently seen during a secondary dengue infection. However, in infants it may also occur durring a primary infection due to maternally attained dengue antibodies. The proposed diagnostic criteria for DHF includes:

Clinical parameters: Acute-onset febrile phase – high-grade fever lasting from 2 days to 1 week. Hemorrhagic episodes (at least one of the following forms): Petechiae, purpura, ecchymosis, epistaxis, gingival and mucosal bleeding, GIT or injection site, hematemesis and/or malena
Positive tourniquet and hepatomegaly.

- b.

Laboratory parameters: Thrombocytopenia (platelet count <100,000/cu mm)The hemorrhagic episodes in DHF are associated with multifactorial pathogenesis. Vasculopathy, deficiency and dysfunction of platelets and defects in the blood coagulation pathways are the attributed factors. Decreased production of platelets and increased destruction of platelets may result in thrombocytopenia in DHF. The impaired platelet function causes the blood vessels to become fragile and this results in hemorrhage.

The clinical course of DHF is characterized by three phases: Febrile, leakage, and convalescent phase. High-grade fever of acute onset along with constitutional signs and facial erythema characterizes the commencement of the febrile illness. The initial febrile illness is marked by a morbilliform rash and hemorrhagic tendencies. The fever persists for 2 days to 1 week and then drops to normal or subnormal levels when the patient either convalesces or advances to the plasma leakage phase. High plasma escape cases are marked by frank shock with low pulse pressure, cyanosis, hepatomegaly, pleural and pericardial effusions, and ascites. Severe ecchymosis and gastrointestinal bleeding followed by epistaxis may also be noted in a few cases. Bradycardia, confluent petechial rashes, erythema, and pallor are seen during this phase.

Dengue shock syndromeDSS is defined as DHF accompanied by a unstable pulse, narrow pulse pressure (<20 mmHg), restlessness, cold, clammy skin, and circumoral cyanosis. Progressively worsening shock, multiorgan damage, and disseminated intravascular coagulation account for a high mortality rate associated with DSS. The shock persists for a short span of time and the patient promptly recovers with supportive therapy.

Orafacial Features

Oral features are infrequently seen in dengue virus infection and are more commonly associated with DHF. Erythema, crusting of lips, and ntongue and soft palatal vesicles constitute the prominent oral features in dengue virus infection. Chadwick *et al*. reported higher cases involving the mucosa with scleral injection (90%), whereas Sanford noticed vesicular eruptions of the soft palate (>50%). Byatnal *et al*., reported numerous hemorrhagic bullae on the sublingual mucous membrane, lateral surface of the tongue, and floor of the mouth. Brown-colored plaquelike lesions with a rough surface were seen on the buccal mucosa that showed bleeding on touch along with spontaneous bleeding from the gingiva and the tongue. Petechiae, purpura, ecchymoses, and nasal bleeding have also been reported. Mitra *et al*. reported bleeding gums, hemorrhagic plaques, and inflamed tonsils in a dengue-infected patient. Isolated hypoglossal nerve palsy following dengue infection is a rare occurence. Taste alteration, conjunctival redness, and lymphadenopathy may also be reported in DFdepicts the reported orofacial features of dengue.

Diagnosis:-Cautious attention should be directed at DF if a patient suffers from high fever within 2 weeks of being in the tropics or subtropics. A decreased number of white blood cells (leukopenia), accompanied by a decreased number of platelet count (thrombocytopenia) and metabolic acidosis are the initial changes on laboratory examinations. Microbiological laboratory testing confirms the diagnosis of DF. Virus segregation in cell cultures, nucleic acid demonstration by polymerase chain reaction (PCR), and serological detection of viral antigens (such as NS1) or particular antibodies are the preferred microbiological assays. Viral segregation and nucleic acid demonstration provide precise diagnosis, although the high cost limits the availability of these tests

Management

Fluid replacement and antipyretic therapy with paracetamol is the preferred therapy following the febrile phase. Care should be taken not to use other nonsteroidal antiinflammatory drugs.Judicious fluid administration forms the mainstay of treatment during the critical phase of the infection. Normal saline, Ringer's Lactate, and 5% glucose diluted 1:2 or 1:1 in normal saline, plasma, plasma substitutes, or 5% albumin are the routinely administered fluids.

WHO guidelines summarize the following principles of fluid therapy: Oral fluid supplementation must be as plentiful as possible. However, intravenous fluid administration is mandatory in cases of shock, severe vomiting, and prostration (cases where the patient is unable to take fluids orally)Crystalloids form the first-line choice of intravenous fluid (0.9% saline)Hypotensive states that are unresponsive to boluses of intravenous crystalloids, colloids (e.g., dextran) form the second-line measuresIf the patient remains in the critical phase with low platelet counts, there should be a serious concern for bleeding. Suspected cases of bleeding are best managed by transfusion of fresh whole blood.

Dental Management Oral lesions are infrequently seen and are often misguided as platelet defects. Significant hemorrhagic manifestations need platelet transfusions. In general, there is no need to give prophylactic platelets even at <20,000/cu mm. Prophylactic platelets may be given at a level of <10,000/cu mm in absence of bleeding manifestations. In case of systemic massive bleeding, platelet transfusion may be needed along with red cell transfusion. Liver functions should be monitor.

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Principal, JJAMC

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STUDENT ARTICLE
Nimba

Prepared by : Mihir Patel(S.Y. BAMS)
Guided by: Dr.Deepa Mehta , Dr.Pradeep Tidake

• **Latin Name : Azadirachta indica** •

Family : Meliaceae

• **Synonyms :**

1. हिङ्गनिर्यास:ु - हिङ्ग इव निर्यासोऽस्र्

The exudate from Nimbresembles Hingu Niryasa

2.अरिष्ट:िरिष्टमशभस्र्मयत्सवाि◌ोगियशक ु इत्र्ाि◌ः|

Nimba cures many diseases, does well always if used.

3. कृममघ्ि कृमीि ◌्िन्ति इनि ।

Nimba is very efficacious in helminthiasis.

• **Morphology :**

Habit→ A large tree

Leaves→ Compound alternate, imparipinnate

Inflorescence → Axillary panicle

Fruit →Drupaceous with one seed

Stem →Woody bran-ched solid, erect and cy-lindrical

Useful part→ Patra ,Twak, Bija,Phala,Puspa, Niryasa

- **Rasapanchak :** Rasa – Tikta,Kasaya
  Guna – Laghu, Rükşa
  Virya – Sita
  Vipaka – katu
- **Dosa karma :** Pitta kapha samaka
- **Karma :** krimighna,Netrya,Pramehahara,sothaha ra,Visghna,Rakta sodhaka,Kusthaghna,.

• **Amayika Prayoga :** - Kustha Surameha,Twagroga,Arumsika

- **Formulations :** 1.Nimbadi curana 2.Nimbadi kwath
  3.Panchanimba curana
- **Reference :** Bhavprakash Nighantu, Dravyaguna Vijnana by Dr.Prakash .L . Hegde

STUDENT ARTICLE
Arka-RAKTA

Prepared by : Kripal (S.Y. BAMS)
Guided by: Dr.Deepa Mehta,Dr.Pradeep Tidake

Latin Name : Calotropis procera
Family : Asclepidaceae
Synonyms :

1. गणरूपः–Plant grows in a gregarious nature.

2. क्षीरपर्णः Arka leaves have latex.

3. सदापुष्प–Arka always bear flowers.

Morphology : Habit→An erect shrub usually grows 2 to 2.5 mtr high.

Leaves→ Subsessile, 6 to 15 cm long. 4.5 to 8 cm wide, broadly ovate, ovate-oblong.
Inflorescence → Umbel inflorescence. Flowers White or greenish white and very small.
Fruit →Etaerio of folicles, 7.5 to 10 cm long. 5-7.5 cm wide, subglobose, ellipsoid or Ovoid.
Seeds 6x4 mm, broadly ovate, acute, flattened, narrowly marginal, light brown

Useful part→ Mula twak and pushpa

Rasapanchak : Rasa – Katu, Tikta
Guna – Laghu, Rükşa, tiksna
Virya – Usna
Vipaka – katu

Dosa karma : kapha vata hara
Karma : Dipana, Rucikara, Sulahara, Hridya, Vrsya, Balya, Krimighna, Chardighna
Rogaghnata : - Klaivya, Aršas, Käsa, Świtra, Kuştha, Krmi, Raktapitta. (Latex) - Kustha, Gulma, Udara, Świtra.
Formulations : Arka lavana.2.Arkeswara rasa.
Reference : Bhavprakash Nighantu, Dravyaguna Vijnana by Dr.Prakash .L . Hegde