Oral presentation in dengue hemorrhagic fever: A rare entity

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Abstract

One of the major health hazards which is prevalent and dangerous is the dengue fever which causes the death of many people. This may be associated with a variety of mucocutaneous manifestations which may be of help in early diagnosis. Many biochemical assays and hematological investigations may aid in the further diagnosis and treatment of the fatal disease. Oral lesions are rare to occur and if present, are often mistaken for platelet abnormality. This case report highlights the importance of oral lesions and it is the first of its kind to be reported as dengue hemorrhagic fever.

Key words: Dengue fever, Dengue hemorrhagic fever, Dengue virus
INTRODUCTION

Dengue fever is a severe flu-like illness that affects the infants, children, adolescents, and adults. The disease is transmitted among humans by the mosquito Aedes aegypti and is seen mostly in the rainy season.

The etiologies by which dengue is caused have been hypothesized and are as follows:

- Viral replication, which occurs primarily in macrophages.
- Direct infection of the skin by the virus.
- Immunologic and chemically mediated mechanism induced by interaction of the virus with the host.

There are four serotypes of dengue viruses (DEN 1-4). Infection with dengue virus can cause three clinical syndromes with undifferentiated febrile illness or viral syndrome, classic dengue fever (DF), dengue hemorrhagic fever (DHF) which may occur with shock or as dengue shock syndrome (DSS).

CASE REPORT

A middle-aged female patient had sought our consultation for blisters in her mouth, bleeding gums, and difficulty in swallowing for the past 1 week. She also gave a history of fever since 1 week and the temperature ranged between 102°F and 104°F. Blisters had initially started in the left buccal mucosa and then involved the right buccal mucosa, tongue, and posterior part of the palate. She also complained of red spots in her lower limbs since 3 days. History revealed that she had joint pain from the time of onset of the fever.

On clinical examination, petechiae were present in the upper and lower limbs, face, and neck. She had an axillary temperature of about 102°F. Bilateral submandibular lymphadenopathy was present.

Intraoral examination revealed the presence of raised hemorrhagic plaques both on the right and left buccal mucosa [Figures 1 and 2] as well as on the dorsum of the tongue near the tip. The hemorrhagic plaques were surrounded by the greenish blue mucosa, and the surface of the hemorrhagic plaques was irregular. At the junction of the hard and soft palate, a diffuse area of erosion of 3 × 4 cm was present [Figure 3]. The tonsils on the both right and left sides was enlarged and inflamed. Patient had xerostomia and the tongue appeared to be coated. A tourniquet test was performed and around 20-22 petechiae/2.5 cm² were observed. The patient was then subjected to a series of hematological and biochemical investigations.

The results of the investigation were as follows:

- Thrombocytopenia 4000 cells/mm³, total leukocyte count 3000 cells/mm³, lymphocyte count 12%, serum albumin 3 g/dl, hemoglobin 14.2 g/dl, prothrombin time (PT) was normal, and activated partial thromboplastin time (aPTT) was elevated, and hematocrit value was about 44%.

With the above results, a provisional diagnosis of DHF was made. To confirm further, IgM was detected by an antibody capture enzyme-linked immunosorbent assay (ELISA; 8 days after the onset of symptoms).

Patient was managed with IV fluids and platelet transfusion in the intensive care unit. She recovered within 1 week.

DISCUSSION

DHF is caused by one of four closely related, but antigenically distinct, virus serotypes (DEN 1-4) of the genus Flavivirus. Dengue is caused by Flavivirus, which is small and appears spherical with lipid envelope. Mucosal involvement is seen in about 15-20% of patients with DHF. Most commonly affected sites are the conjunctival and sclera margins, soft palate, and lips and the tongue. More than 50% of cases have been reported in the soft palate by Stanford.

In accordance with the current WHO and Pan American Health Organization, a case of DHF should meet the following clinical criteria: Acute onset fever, hemorrhagic manifestations, thrombocytopenia, and hemoconcentration demonstrated by a rise in hematocrit value by 20% or more.

The incubation period ranges about 4-7 days, after which the patient may experience acute onset of fever followed by non-specific signs and symptoms. The patient in our case also had fever for the past 1 week and the temperature range was around 102°F-104°F, which is in accordance with the previous reported cases.

The febrile period may also be accompanied with rash which appears as maculopapular or macular that becomes diffusely erythematous later. In our case, there was absence of rash in the initial stages of the disease.

Tourniquet test was performed by inflating a blood pressure cuff of the sphygmomanometer on the upper aspect of the arm to a point midway between systolic and diastolic pressures for about 5 min. If there are more than 20 petechiae/2.5 cm², the test is considered to be positive, as in our case.
Hemorrhagic manifestations were seen in most of the patients as petechiae and purpura. The oral manifestations include that of gum bleed and petechiae in the soft palate.[7] Our presentation was typical in that it showed severe hemorrhagic bulla extending in the right and left buccal mucosa and also involving the junction of hard and soft palate. The major pathophysiologic hallmarks which determine the disease severity and distinguish from other viral hemorrhagic fevers are plasma leakage and abnormal hemostasis.[8] Abnormal hemostasis includes capillary fragility, thrombocytopenia, impaired platelet function, and disseminated intravascular coagulation, of which the first three parameters were positive in our case.

The IgM ELISA test is used as a serologic tool and has a sensitivity of 83.9-98.4% and a specificity of 100%,[9] and hence has been used in our patient with positive results.

Differential diagnosis for dengue fever should be considered in case of appearance of rash along with fever and joint pain. It includes viral exanthematous fevers like Chikungunya fever, measles, German measles, roseola infantum, acute retroviral syndrome, and others such as scarlet fever, Kawasaki disease, toxic shock syndrome, secondary syphilis, typhoid fever, leptospirosis, and drug exanthema.[1] In the oral cavity, hemorrhagic plaques are seen in most of the cases of idiopathic thrombocytopenic purpura.

Chikungunya fever presents with polyarthralgia along with myalgia and has fewer signs of easy bleeding. Due to the severe joint pain, patients adapt themselves to the characteristic postures.

A prodromal phase of intraoral koplik spots prior to the occurrence of cutaneous rash is characteristic of measles. There was also presence of erythematous macules and papules that usually heals with desquamation.

German measles shows involvement of soft palate and tonsils during the prodromal phase and there is mild upper respiratory tract infection along with fever and cutaneous rash.

Roseola infantum, an HHV-6 infection commonly seen in infants, exhibits macules and papules that are almond shaped, and there is presence of an enanthem of red papules in the soft palate and uvula, termed as Nagayoma spot. The most characteristic feature is the presence of febrile seizures.

Acute retroviral syndrome presents with generalized morbilliform exanthema that spares the palms and soles, lasts for 4-5 days, and is associated with fever, myalgia, and lymphadenopathy.[1]
To conclude, two clinical observations plus one laboratory finding or at least rising hematocrit value are sufficient to establish a diagnosis of DHF. In our case, petechiae, rise in temperature, positive tourniquet test, thrombocytopenia, and positive IgM ELISA test were evident to establish a diagnosis of DHF.

Clinical diagnosis is sufficient to start the treatment. Ranjith et al. had suggested that the disease be identified at an early stage, and treatment should be started in order to manage shock with fluid resuscitation. Shock due to fluid loss should be managed with fluid resuscitation.

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