

Oral manifestations of dengue fever: A rarity and literature review

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ABSTRACT

Dengue is a viral infection with fatal potential complications. It is also called break-bone fever. Worldwide, dengue infection is the most common mosquito-borne viral disease. It is caused by vector *Aedes aegypti* and represents a major public health issue in more than 100 tropical countries. This may be associated with a variety of mucocutaneous manifestations, which may be of help in early diagnosis. Dengue viral infections are characterized by abrupt febrile illness, but can also lead to significant morbidity and mortality. Hence, it requires an early and correct diagnosis. Gingival bleeding is the most common oral manifestation of dengue infections. Many biochemical assays and hematological investigations may aid in the further diagnosis and treatment of the fatal disease. Although oral lesions are uncommon in dengue infections and if occur, may be mistaken for platelet abnormality or hemorrhagic disorders. This review emphasizes the significance of oral lesions as it may be the early indicators of dengue hemorrhagic fever.

Key words

Dengue fever, oral manifestations, platelet count

INTRODUCTION

Oral cavity is the mirror of the whole body. Many systemic and infectious diseases show their significant presence in the oral cavity. Dengue fever (DF) is a severe flu-like condition that affects almost all age groups.^[1] It has now become a common epidemic and affecting people in all parts of our country.^[2] Among humans, it is being caused by the mosquito *Aedes aegypti* and is prevalent mostly in the rainy season. The etiology of dengue has been hypothesized as follows:

- Viral replication, which occurs primarily in macrophages^[3]
- Direct infection of the skin by the virus
- Immunologic and chemically mediated mechanism induced by the interaction of the virus with the host.^[4]

Serotype of dengue virus: Four serotypes of dengue viruses occur (DEN 1–4). Infection with dengue virus can cause three clinical syndromes with undifferentiated viral syndrome, classic DF, and dengue hemorrhagic fever (DHF), which may occur with shock or as dengue shock syndrome (DSS). Infections with the dengue virus can cause a spectrum of three clinical syndromes with classic DF, DHF, and DSS. The World Health Organization (WHO) criteria exist for the classification of dengue into these three clinical categories.^[5] However, there is a significant overlap between the categories.^[6] Oral findings are secondary to the general manifestations. Limited literature exists which presents the oral manifestation of DF.^[7] Approximately 2.5 billion people live in dengue-risk regions with about 100 million new cases each year worldwide. The cumulative dengue

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diseases' burden has attained an unprecedented proportion in recent times with sharp increase in the size of human population at risk. Dengue disease presents highly complex pathophysiological, economic, and ecologic problems. In India, the first epidemic of clinical dengue-like illness was recorded in Madras (now Chennai) in 1780 and the first virologically proved epidemic of DF occurred in Calcutta (now Kolkata) and Eastern Coast of India in 1963–1964. During the last 50 years, a large number of physicians have treated and described dengue disease in India.^[8] This case report is an attempt to add the scientific literature rarity of oral lesions in a patient with DF.

CASE REPORT

A 19-year-old male patient was referred from the Department of Medicine, Indira Gandhi Medical College and Hospital to the department of Periodontology, Government Dental College, Shimla for the treatment of blisters in his mouth, soreness on the gums, alveolar mucosa on soft palate, and difficulty in swallowing for the past 10 days. He was reported with a history of high-grade fever since more than 1 week. Blisters initially present on the left side of maxillary alveolar mucosa followed by similar lesions on the upper right mucosa. Later on it has involved the posterior part of the palate. He also complained of bluish red vesicles on lower limbs since 4–5 days. There was persistent joint pain from the time of onset of the fever.

On clinical examination, petechiae were present all over the body involving the upper and lower limbs, face, and neck. Axillary temperature of 103°F was noted. On palpation, bilateral submandibular lymphadenopathy was evident.

Intraoral examination revealed the presence of typical ulcerative and bleeding lesions on both on the left and right gingiva in relation to teeth no. 24, 25 and teeth no. 14, 15 regions. Diffuse area of erosion lesions measuring from 1.5 cm × 1.3 cm on the right side and 0.8 cm × 0.7 cm on the left side [Figures 1 and 2] were present. At the junction of the hard and soft palate, predominantly on right side small blood-filled vesicles were present [Figure 3]. Petechiae were also present on lower limbs [Figure 4]. A tourniquet test was performed and around 18–24 petechiae/2.5 cm² were observed. There was no conjunctival involvement. The patient was then subjected to relevant hematological and biochemical investigations to establish the diagnosis.

Thrombocytopenia 5000 cells/mm³, total leukocyte count 3500 cells/mm³, lymphocyte count 9%, serum albumin 2.8 g/dl, hemoglobin 12.8 g/dl, and prothrombin time were normal, activated partial thromboplastin time was elevated and hematocrit value was about 45%.

Based on the above hematological tests a provisional diagnosis of DHF was made. An antibody capture enzyme linked immunosorbant assay (ELISA) has confirmed the diagnosis by detecting IgM (Dengue-2 serotype) 8 days after the onset of symptoms. In addition to the medical line of treatment, i.e., intravenous fluids and platelet-rich plasma transfusion in the Intensive Care Unit by the physician in medical college, for the intraoral lesions, mucopain ointment was prescribed as topical application, and hexidine mouthwash for the maintenance of oral hygiene and to reduce the load of pathogenic microbes. After the recovery of the patient from general symptoms, intraoral lesions healed uneventfully within 2 weeks.

DISCUSSION

DHF is caused by one of the four closely related, but antigenically distinct, virus serotypes (DEN 1–4) of the genus *Flavivirus*.^[2] Dengue is caused by *Flavivirus*, which is small and appears spherical with lipid envelope.^[1] 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.^[3]

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.^[5]

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

The febrile period may also be accompanied with rash which appears as maculopapular or macular that becomes diffusely erythematous later.^[2] 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.^[6] If there are more than 20 petechiae/2.5 cm², the test is considered to be positive, as in the present 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.^[8] Our presentation was typical in that it showed severe hemorrhagic bulla

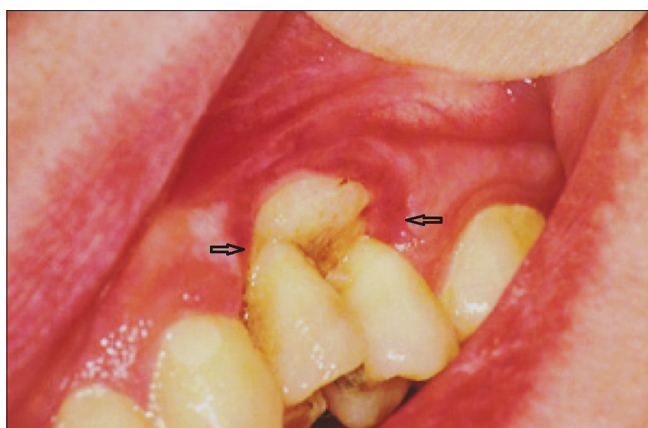


Figure 1: Presence of hemorrhagic ulcerative erosion in relation to gingivoalveolar mucosa with respect to 24 and 25

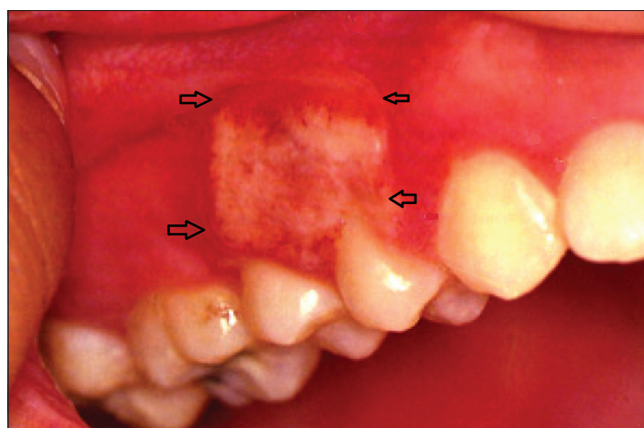


Figure 2: Mucosal lesion on the gingival area with respect to 14 and 15

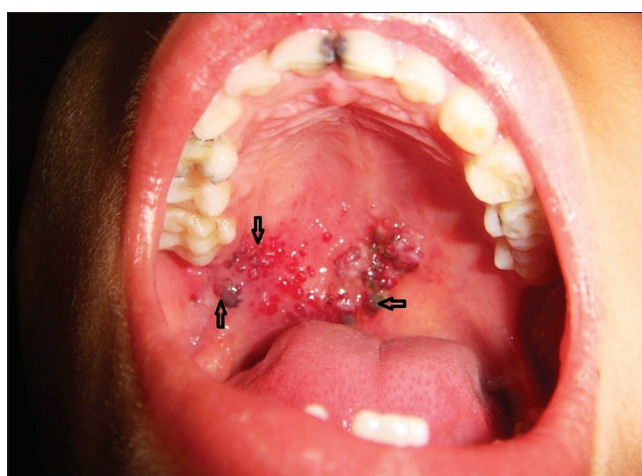


Figure 3: Small blood-filled vesicles at the junction of the hard and soft palate



Figure 4: Petechiae present on lower limbs

extending in the right and left buccal mucosa and also involving the junction of the 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.^[9] Abnormal hemostasis includes capillary fragility, thrombocytopenia, impaired platelet function, and disseminated intravascular coagulation, of which the first three parameters were positive in our case.^[10]

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 DF should be considered in case of appearance of rash along with fever and joint pain. DF should be considered in the differential diagnosis of fever and rash in a patient residing or returning from an endemic area, and dermatologists should be aware of the distinctive exanthem of DF. Recognition of DF rash permits a rapid and early

diagnosis, which is critical, as DF can progress to life-threatening DHF or DSS.^[11]

It includes various viral exanthematous fevers such as measles, German measles, roseola infantum, acute retroviral syndrome, and others such as Kawasaki disease, scarlet fever, toxic shock syndrome, syphilis, typhoid fever, leptospirosis, and drug exanthema.^[12] In the oral cavity, hemorrhagic plaques and erosions are seen in most of the cases of idiopathic thrombocytopenic purpura.^[13]

CONCLUSION

Two clinical observations plus one laboratory finding or at the least rising hematocrit value are sufficient to establish a diagnosis of DHF.^[6] In our case, petechiae, rise in temperature, positive tourniquet test, thrombocytopenia, and positive IgM ELISA test were sufficient to establish a diagnosis of DHF.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Ligon BL. Dengue fever and dengue hemorrhagic fever: A review of the history, transmission, treatment, and prevention. *Semin Pediatr Infect Dis* 2005;16:60-5.
2. Arshad I, Malik FA, Hussain A, Shah SA. Dengue fever; clinico-pathologic correlations and their association with poor outcome. *Prof Med J* 2011;18:57-63.
3. Wu SJ, Grouard-Vogel G, Sun W, Mascola JR, Brachtel E, Putvatana R, *et al.* Human skin Langerhans cells are targets of dengue virus infection. *Nat Med* 2000;6:816-20.
4. Chadwick D, Arch B, Wilder-Smith A, Paton N. Distinguishing dengue fever from other infections on the basis of simple clinical and laboratory features: Application of logistic regression analysis. *J Clin Virol* 2006;35:147-53.
5. World Health Organization. *Dengue Hemorrhagic Fever: Diagnosis, Treatment, Prevention and Control*. 2nd ed. Geneva: World Health Organization; 1997.
6. Bandyopadhyay S, Lum LC, Kroeger A. Classifying dengue: A review of the difficulties in using the WHO case classification for dengue haemorrhagic fever. *Trop Med Int Health* 2006;11:1238-55.
7. Pontes FS, Frances LT, Carvalho Mde V, Fonseca FP, Neto NC, do Nascimento LS, *et al.* Severe oral manifestation of dengue viral infection: A rare clinical description. *Quintessence Int* 2014;45:151-6.
8. Gupta N, Srivastava S, Jain A, Chaturvedi UC. Dengue in India. *Indian J Med Res* 2012;136:373-90.
9. Thomas EA, John M, Bhatia A. Cutaneous manifestations of dengue viral infection in Punjab (North India). *Int J Dermatol* 2007;46:715-9.
10. Pincus LB, Grossman ME, Fox LP. The exanthem of dengue fever: Clinical features of two US tourists traveling abroad. *J Am Acad Dermatol* 2008;58:308-16.
11. Sabin AB. Research on dengue during World War II. *Am J Trop Med Hyg* 1952;1:30-50.
12. Boonpucknavig S, Boonpucknavig V, Bhamarapavati N, Nimmannitya S. Immunofluorescence study of skin rash in patients with dengue hemorrhagic fever. *Arch Pathol Lab Med* 1979;103:463-6.
13. Itoda I, Masuda G, Suganuma A, Imamura A, Ajisawa A, Yamada K, *et al.* Clinical features of 62 imported cases of dengue fever in Japan. *Am J Trop Med Hyg* 2006;75:470-4.