Case Report

Hand Foot and Mouth Disease: A case report

Sah VK*1

Janaki Medical College Teaching Hospital
Ramdaiya, Dhanusha, Nepal

1 Assistant Professor, Department of Pediatrics, Janaki Medical College

ABSTRACT

Hand, foot & mouth disease (HFMD) is a common emerging infectious disease caused by intestinal viruses of the picornaviridae family. A strain of Coxsackie virus (A16, A5, A6) is chiefly instrumental for producing this condition however more severe form is caused by Enterovirus-71. Though HFMD is a mild disease and can often be effectively and competently managed in an outpatient setting, recent outbreaks in the western pacific region with fatal form of disease have attracted the concern about the clinical assessment and appropriate management of HFMD. Early detection and good clinical judgments not only can prevent the fatal progression but also can reduce overall morbidity and mortality regarding HFMD. I am presenting a case report of the aforementioned disease and the subsequent early clinical manifestation of the fatal form of HFMD to enlighten on the nature of the disease so that early diagnosis and management of the condition can be carried out to halt the disease progression and prevention for the betterment of children especially under five years.

Key Words: Picornaviridae, Hand foot and mouth disease, Children

INTRODUCTION

Enteroviruses are non-enveloped, single-stranded, positive-sense viruses in the Picornaviridae ("small RNA virus") family, which also includes the genera Rhinovirus, Hepatovirus (hepatitis A virus), and Parechovirus and genera containing related animal viruses resulting in a wide variety of diseases in human. Hand foot and mouth disease (HFMD) is one of the emerging benign and common infectious diseases predominantly involving the children under 5 years caused by serotype of enterovirus most frequently Coxsackie virus A16 (CAV16) and human enterovirus 71 (HEV71)[1]. Other serotypes causing outbreaks of HFMD are coxsackie A viruses 5, 6, 7, 9, and 10; coxsackie B viruses 2 and 5 [2].

Over the last decade, many outbreaks of HFMD have been reported in countries of the Western Pacific Region, including Japan, Malaysia, Singapore and across China. After a number of these outbreak noted since 1970, a number of young children had been the victim of serious manifestation of HFMD though majority of the children remain either
asymptomatic or present with mild clinical symptoms [3]. The common manifestation of the diseases includes fever, skin eruption on hand and feet and vesicle in the mouth. The fatal presentation involves central nervous system and/or pulmonary edema predominantly those caused by human enterovirus 71 [4]. Coxsackie virus A16 also can occasionally be associated with complications such as encephalitis, acute flaccid paralysis, myocarditis, pericarditis, and shock [5].

**Case report**

A 5 years boy presented to the outpatient department with appearance of small, round and painless fluid filled skin lesion surrounded by red margin and few red rashes over both palm and foot for the last 3 days. The lesion developed simultaneously over hand and foot and was non-pruritic. The child was a febrile with a prodromal symptom of runny nose since last 15 days. The child was attending his regular school without any discomfort. His sibling also demonstrated similar type of lesion with a same day of appearance of lesion. He was the known case of seizure disorder with a history of 3 episode of seizure in last 3 month and managed with sodium valproate from last 8 month. There was no known drug allergy history in past. The child was active playful and cooperative with stable vitals. There were few nodular (2-3mm) and other pustular lesion over palmar surface of hand and great toe of foot of 4-5mm. Few number of vesicular eruption were present over palm and sole of size 2-4 mm. Buccal cavity revealed a vesicular lesion (4-6mm) on left sided buccal mucosa and a 2-3mm sized ulcer over the base of tongue and these lesions were painless. Beside above mentioned lesion, few yellow like ulcer lesion were also noted surrounded by an erythematosus halo of 5-6mm. The remaining skin part; particularly buttock, perianal and other region were free of any lesion.

![Figure 1: Right and left hand showing the nodular, vesicular and pustular lesions distributed over palm and fingers](image1)

![Figure 2: Oral cavity revealing the ulcerating lesion over base of the tongue and a vesicular lesion on the left buccal mucosa](image2)

![Figure 3: Left and right feet showing nodular and vesicular lesion](image3)

**DISCUSSION**

Hand, foot, and mouth disease is a viral infection caused by a strain of Coxsackie virus and enterovirus 71. Nishimura, Yamayoshi and Yang with their colleagues, recently independently demonstrated that human P-selectin glycoprotein ligand-1 (PSGL-1), human scavenger receptor class B, member 2 (SCARB2), and sialic-acid-linked glycanact as functional receptors for EV71 by using different human cell lines and different cloning strategies [6, 7, 8].
Epidemiology

Enterovirus infections are common and ubiquitous in distribution. In temperate climates there is an annual epidemic peak in summer/fall, although some transmission occurs year-round. Factors associated with increased incidence and/or severity includes young age, male sex, exposure to children, poor hygiene, overcrowding and low socioeconomic status [9]. In an age wise distribution; more than 25% of symptomatic infections occur in children younger than 1 year of age [10]. Factor that reduce the risk is breastfeeding; likely via enterovirus-specific antibodies. Till now humans are the only known reservoir for human enteroviruses, although some nonhuman primates can be infected. The replication of EV71 occurs in predominantly intestinal tract and its shedding typically occur between 2-4 weeks and may prolong for 12 week’s post-infection. Positive evidence from throat swab for up to 2 weeks of post-infection support its replication in upper respiratory tract. Fecal-oral decontamination and the respiratory secretions through direct person-to-person contact, droplets or fomites become possible mode of infection [11]. The incubation period is roughly around 3-7 days.

Clinical manifestation

It is common in children particularly between 2 years and 5 year but can also occur in adolescent and young adults. It is characterized by a mild prodromal period of mild grade of fever, sore throat, runny nose and a rash with vesicular or pustular lesions. The illness begins with either an asymptomatic child with a typical rash, blister and vesicular or pustular lesion frequently followed by ulceration or with a mild fever, poor appetite, malaise ("feeling sick") and frequently a sore throat followed by painful lesion as described above. Generally fever is followed by sore throat by 2-3 days involving buccal mucosa, tonsillar pillar and occasionally posterior pharynx. They begin as small red spots that blister and then often become vesicular or pustular lesion followed by ulceration of the lesion. The skin rash which are not itchy in children develops over 1 to 2 days with flat or raised red spots, some with blisters and ulceration surrounded by erythematous halo. It is usually located on the palms of the hands and soles of the feet and occasionally appear on the buttocks, perianal reason and groins [3]. A person is contagious when the first symptoms appear and may continue until the blister-like skin lesions disappear [12]. A person is most contagious during the first week of illness. A person may have only the rash or the mouth ulcers. Less commonly the lateral and dorsal surface of hands and feet, and perioral skin can be affected.

Approximately 10%-30% of hospitalized cases during EV71 associated HFMD epidemics in Asia have developed a spectrum of CNS complications, including aseptic meningitis, encephalitis and acute flaccid paralysis [13, 14, 15]. Brainstem encephalitis, a distinctive form of encephalitis with stereotypic neuro-pathological characteristics has become the hallmark of severe EV71-associated HFMD in the recent recurrent EV71 epidemics in Asia, which began in the late 1990s [16, 17]. The most severely affected children can develop fulminant cardio-respiratory failure, which is often fatal and causes a high incidence of severe neurological and possible psycho-behavioral sequelae among survivors, despite intensive care support [18].
Diagnosis

Despite of the presence various advance technique in the diagnosis HFMD, the diseases can be diagnosed clinically. Throat and vesicle (if available) swab samples in virus transportation medium can be used for varied of molecular technique, cell culture for virus isolation using PCR and several other technique [19, 20]. This become important in patients presenting with other manifestations of EV71, such as CNS disease or cardiovascular collapse, rapid virological diagnosis is even more helpful because of the broad differential for those conditions and the specific treatments available. Identification of the agents responsible for outbreaks of HFMD is also important with regards to predicting the severity of the outbreak and initiating appropriate public health interventions.

The differential diagnoses for HFMD include herpetic gingivo stomatitis, aphthous stomatitis, scabies infestation, chickenpox (varicella), measles and rubella.

Management

The management of the HFMD remains a clinical judgement depending upon the assessment of the disease. A clinical challenge emerges when the disease is assessed for the involvement of the CNS, cardiopulmonary and ANS system which might require intensive care and management. Most of the symptomatic child having muco-cutaneous lesion are managed symptomatically with acetaminophen with the benefit of self-limited diseases course. Daily follow-up by the clinic may be advisable for at least seven days after the onset of illness. There is no specific antiviral treatment available till now. The remaining small proportion of children with HFMD progressing to potentially fatal cardiopulmonary failure requires intensive care including ventilation and newer therapy including milrinone [21]. The early onset of myoclonic jerk (during sleep in early stage and later also in awake state), truncal ataxia and wandering eyes (rotatory eye movement without fixation) are the early manifestation of fatal disease course [22].

The severe disease throughout its course is characterized by three distinct stages:

(I) those with CNS involvement (II) those with ANS dysregulation (profuse sweating and respiratory abnormalities, persistent tachycardia and hypertension) and, (III) later, those with frank cardiopulmonary failure, including pulmonary edema or hemorrhage [23]. CSF study support the assessment with the finding of leukocytosis, thrombocytosis and hyperglycemia.

One of the newer therapy is the use of intravenous immunoglobulin (IVIG). IVIG has shown promises result in halting the severe progression of diseases especially ANS manifestation as a long term sequel. However, the use of IVIG has yet to be supported by evidence from randomized clinical trials [24].

Seizure is the uncommon manifestation of the disease. Especially the myoclonic jerk, if the occurs may require the anticonvulsant (phenytoin) therapy and with frequent myoclonic jerk may require the continuous sedation with midazolam and/or phenytoin.

CONCLUSION

The recent outbreak of the HFMD in the Asia and its uncommon manifestation crediting life's of the under 5 children especially between 1 and 2 years warns the pediatrician
for careful clinical assessment and management of disease. Since no antiviral and vaccines are available for the management, and also children with high risk of fatal clinical course often present with a subtle clinical feature during early phase; the early assessment and symptomatic management remain the key modalities of therapy for the success and prevention of the fatal outcome of the disease. The range of the management of a milder form of disease with acetaminophen to the requirement of intensive care demand the good clinical skill to reduce the morbidity and mortality from HFMD.

AUTHOR'S CONTRIBUTION

VKS- Studied the case in detail, critical reviews, Inscription and drafting and approval of final manuscript.

SOURCE OF SUPPORT: Clinical support and Diagonosis, Chitwan Medical College, Bharatpur, Nepal

CONFLICT OF INTEREST: Author declared that there is no conflict of interest.

REFERENCES


Correspondence to:
Dr. Vikash Kumar Sah
Assist. Professor
Department of Pediatrics
Janaki Medical College teaching Hospital
Janakpur, Nepal
Email: sendvikash@icloud.com