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# HAND FOOT MOUTH DISEASE AND ISOLATED BENIGN INTRACRANIAL HYPERTENSION, A RARE PRESENTATION

**Paediatrics**

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## ABSTRACT

Hand foot mouth disease (HFMD) is a highly contagious acute viral infection caused by *Enterovirus* and *Coxsackie* virus occurring mostly in infants and children. The illness is usually self-limiting; however, HFMD associated cardiorespiratory and neurological complications may become fatal if left untreated in early stages. We report a rare case of HFMD in a 7-month-old infant who presented with signs and symptoms suggestive of severe neurological complication, but eventually was found to have isolated intracranial hypertension without any evidence of meningo-encephalitis which rapidly responded to supportive therapy.

## KEYWORDS

Hand foot mouth disease, Benign Intracranial hypertension, *Enterovirus*

### INTRODUCTION:

Hand foot mouth disease (HFMD) is a clinical syndrome characterized by oral enanthem associated with macular/maculopapular, vesicular rash of hands and feet caused by *Enteroviruses* of *Picornaviridae* family. Serotypes of *Enterovirus* most frequently implicated are *Coxsackie virus A16*(CAV16) and *Human Enterovirus 71*(HAV71). Other serotypes causing outbreaks of HFMD include *Coxsackie A viruses* 5,6,7,9,10; *Coxsackie B viruses* 2,5.

The disease is usually self-limiting with a mortality rate of about 0.03% (1). However, sometimes fatal complications like myocarditis, hepatitis, pancreatitis, aseptic meningitis, acute flaccid paralysis, encephalitis, Guillain Barre Syndrome, neurogenic pulmonary edema have been reported (2), especially in children less than four years of age. Occurrence of isolated benign intracranial hypertension in cases of HFMD is not widely reported (3). We report a case of 7-month-old infant with HFMD presenting with signs and symptoms of severe encephalopathy but eventually diagnosed as benign intracranial hypertension.

### Case Report:

A 7-month-old male infant was admitted with complaints of high-grade fever for 5 days and excess cry with irritability, poor feeding for 2 days progressing to encephalopathy since the morning of presentation. On examination, he was noted to have erythematous maculopapular skin rash over perioral area, nasal creases, palms soles and back along with intraoral lesions (Figure 1). He was drowsy with tense bulged anterior fontanelle. Pupils were equal in size and pupillary reflex was bilaterally symmetrical. He was febrile, tachycardiac, tachypneic at admission with normal blood pressure and saturation. Other physical findings were normal. Enquiry into the further history revealed that the child developed skin rash after receiving over-the-counter medications and a review by the family physician suggested drug reaction as the likely cause of rash.

### Blood investigations showed:

white blood cell count -10,200/mm$^3$ with 58% neutrophils, CRP- 91 mg/dl. An urgent CT brain study showed no abnormalities. MRI brain was done subsequently which was normal. Fundus examination was normal. Lumbar puncture was done and cerebrospinal fluid (CSF) analysis showed cell count- 2 cells/mm$^3$, lymphocyte predominant; glucose-55mg/dl; protein-76.6mg/dl with gram stain showing no organisms. CSF opening pressure was 28 cm H2O suggestive of raised intracranial pressure.

CSF viral PCR was negative for commonly tested viruses including enterovirus. Based on above findings, a diagnosis of severe HFMD with benign intracranial hypertension was made.

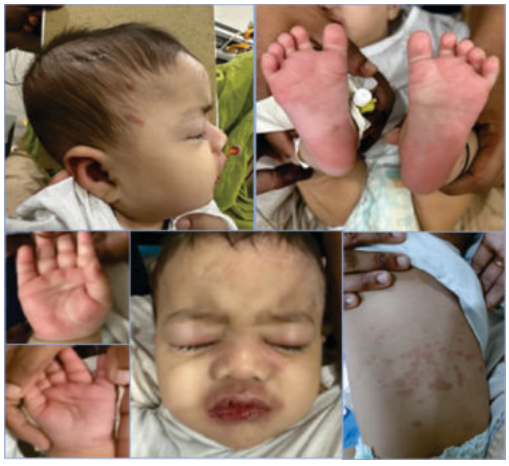

**Figure 1:** Images showing tense bulged anterior fontanelle with erythematous maculopapular skin rash over perioral area, nasal creases, palms soles and back

The child was administered antiedema measures to reduce intracranial pressure along with other symptomatic treatment. His encephalopathy, lethargy, AF bulge and oral intake started to improve within 72 hours of supportive management. Rash and fever also subsided by day 7 of admission. He was seizure free throughout. He started to accept feeds well and was discharged from the hospital on day 10 of admission.

### DISCUSSION:

HFMD is a common, usually mild childhood illness caused by enteroviruses. The term HFMD derives from typical maculopapular or vesicular lesions involving the skin of the hands, feet and oral mucosa.

The prodromal phase includes low-grade fever, malaise and sore throat followed by the sudden appearance of erythematous papulo-vesicular eruptions which appear in crops and persist in groups over some specific areas like hand, feet, perioral area, knees, buttocks and also intraorally. Lesions over thick skin such as palms and soles may not develop classical vesicle; they may instead persist as erythematous papules. The illness usually improves spontaneously and is considered a benign disease of self-limiting nature.

However, complications can occur in a minority involving cardiorespiratory and neurological systems. Cases of HFMD associated with onychomadesis have been documented which could be due to viral replication damaging the nail matrix resulting in transient nail dystrophy (4). Neurological involvement in HFMD cases is usually seen 1–5 days after the initial infection. Early symptoms include headache, lethargy, poor feeding, irritability, but most cases resolve without consequence, although a minority of them progress to develop severe disease (5). Common neurological complications include acute cerebellar ataxia, polio-like syndrome, acute transverse myelitis, encephalitis, benign intracranial hypertension, Guillain-Barre syndrome and aseptic meningitis. *Enterovirus-71* is associated with a higher rate of neurological involvement compared to *Coxsackievirus*. Cardiovascular complications include myocarditis, pulmonary edema, dyspnoea, pulmonary haemorrhage. Risk factors for the development of complications including death have been identified in various studies and these are lethargy, altered sensorium, tachycardia, tachypnoea, leucocytosis with neutrophilia, positive EV-A71, pneumo-edema/pneumorrhagia and seizures (6).

Idiopathic intracranial hypertension (IIH) is a rare neurological disorder characterized by raised intracranial pressure (ICP) in the absence of brain parenchymal lesion, vascular malformations, hydrocephalus, or central nervous system (CNS) infection. The diagnosis is usually confirmed by high opening pressure of CSF with exclusion of secondary causes of intracranial hypertension. Without treatment, it can have deleterious effects on vision including optic atrophy. Although associations of *EV71* and *Coxsackie virus B4* infection with benign intracranial hypertension is mentioned, occurrence of isolated intracranial hypertension without features of meningitis or encephalitis in HFMD is not widely reported in the literature till date. In this report, we document a rare case of 7-month-old infant with HFMD presenting with benign intracranial hypertension without any evidence of neuro-infection which quickly and completely responded to supportive therapy.

HFMD is essentially a clinical diagnosis, although the other differentials such as varicella zoster, papular urticaria, impetigo, Steven-Johnson syndrome, Toxic epidermal necrolysis, erythema multiformae, Kawasaki, etc need to be excluded. Our case was misdiagnosed as drug reaction until later when the rash was closely inspected along with detailed clinical history.

HFMD patient is contagious until the vesiculobullous skin lesions disappear. Therefore, patients should be isolated until the fever, skin and mucosal lesions have disappeared. Patient/parental education is paramount in reducing the transmission of illness. Handwashing has been proven to be an effective strategy in the prevention of HFMD transmission (7).

## CONCLUSION:

The constellation of features in HFMD are unique enough to aid instant clinical diagnosis with certainty in almost all cases. HFMD is often a benign self-limiting illness, but those who develop signs of severe disease or lethargy should be admitted for close monitoring and prompt therapy which can prevent adverse outcomes.

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