

Onychomadesis Following Hand-foot-and-mouth Disease

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PRACTICE POINTS

- Onychomadesis is a late complication of hand-foot-and-mouth disease (HFMD) with a latency period of 1 to 2 months.
- Although the mechanism between onychomadesis and HFMD is still unclear, we propose that it is caused by the viral infection itself rather than severe systemic disease.

To the Editor:

Onychomadesis is characterized by separation of the nail plate from the matrix due to a temporary arrest in nail matrix activity. Hand-foot-and-mouth disease (HFMD) is a relatively common viral infection, especially in children. Although the relationship between onychomadesis and HFMD has been noted, there are few reports in the literature.¹⁻⁹ We present 2 cases of onychomadesis following HFMD in Taiwanese siblings.

A 3-year-old girl presented with proximal nail plate detachment from the proximal nail fold on the bilateral great toenails (Figure 1) and a transverse whole-thickness sulcus on the bilateral thumbnails (Figure 2) of several weeks' duration. Her 6-year-old sister had similar nail changes. Hand-foot-and-mouth disease was diagnosed about 4 weeks prior to nail changes. The mother reported that only the younger



Figure 1. Onychomadesis on the bilateral great toenails.



Figure 2. Onychomadesis on the thumbnail.

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sister experienced fever. There was no history of notable medication intake, nail trauma, periungual erythema, vesicular lesion, or dermatitis. In both patients, the nail changes were temporary with spontaneous normal nail plate regrowth several months later. A diagnosis of onychomadesis was made.

The etiology of onychomadesis includes drug ingestion, especially chemotherapy; severe systemic diseases; high fever; infection, including viral illnesses such as influenza, measles, and HFMD; and idiopathic onychomadesis.^{1,2,5,10} In 2000, Clementz and Mancini¹ reported 5 children with nail matrix arrest following HFMD and suggested an epidemic caused by the same virus strain. Bernier et al² reported another 4 cases and suggested more than one viral strain may have been implicated in the nail matrix arrest. Although these authors list HFMD as one of the causes of onychomadesis,^{1,2} the number of cases reported was small; however, studies with a larger number of cases and even outbreak have been reported more recently.³⁻⁸ Salazar et al³ reported an onychomadesis outbreak associated with HFMD in Valencia, Spain, in 2008 (N=298). This outbreak primarily was caused by coxsackievirus (CV) A10 (49% of cases).⁵ Another onychomadesis outbreak occurred in Saragossa, Spain, in 2008, and CV B1, B2, and unidentified nonpoliovirus enterovirus were isolated.⁶ Outbreaks also occurred in Finland in 2008, and the causative agents were identified as CV A6 and A10.^{7,8} The latency period for onychomadesis following HFMD was 1 to 2 months (mean, 40 days), and the majority of cases occurred in patients younger than 6 years.¹⁻⁵ Not all of the nails were involved; in one report, each patient shed only 4 nails on average.⁶

Although there is a definite relationship between HFMD and onychomadesis, the mechanism is still unclear. Some authors claim that nail matrix arrest is caused by high fever¹⁰; however, we found that 40% (2/5)¹ to 63% (10/16)⁴ of reported cases did not have a fever. Additionally, only 1 of our patients had fever. Therefore high fever–induced nail matrix arrest is not a reasonable explanation. Davia et al⁵ observed no relationship between onychomadesis and the severity of HFMD. In addition, no serious complications of HFMD were mentioned in prior reports.

We propose that HFMD-related onychomadesis is caused by the viral infection itself, rather than by severe systemic disease.^{1-5,7} Certain viral strains associated with HFMD can induce arrest of nail matrix activity. Osterback et al⁷ detected CV A6 in shed nail fragments and suggested that virus replication damaged the nail matrix and resulted in temporary nail dystrophy. This hypothesis can explain that only some nails, not all, were involved. In our cases,

we noted an incomplete and slanted cleft on the thumbnail (Figure 2). We also found that incomplete onychomadesis appeared in the clinical photograph from a prior report.⁵ The slanted cleft in our case may be caused by secondary external force after original incomplete onychomadesis or a different rate of nail regrowth because of different intensity of nail matrix damage. The phenomenon of incomplete onychomadesis in the same nail further suggests the mechanism of onychomadesis following HFMD is localized nail matrix damage.

In conclusion, we report 2 cases of onychomadesis associated with HFMD. Our report highlights that there is no racial difference in post-HFMD onychomadesis. These cases highlight that HFMD is an important cause of onychomadesis, especially in children. We suggest that certain viral strains associated with HFMD may specifically arrest nail matrix growth activity, regardless of fever or disease severity.

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