Denouement and Discussion

Hand-Foot-and-Mouth Disease

Results of enteroviral polymerase chain reaction obtained from an unroofed vesicle were positive, confirming a diagnosis of hand-foot-and-mouth disease (HFMD). Supportive care with use of topical emollients and mupirocin ointment on any crusted or eroded areas was recommended, and the rash resolved without sequelae during the subsequent 2 weeks.

Generally, HFMD is a clinical diagnosis. It most frequently affects children younger than 5 years. It is caused by one of several serotypes of enterovirus, typically coxsackievirus A16, although coxsackievirus A5, A7, A9, A10, B2, and B5 have also been implicated in HFMD in the United States. In Southeast Asia and China, enterovirus 71 has been associated with outbreaks of HFMD with associated neurological complications, including encephalomyelitis. Epidemics of HFMD often occur during the summer and fall. Typically, HFMD begins with a low-grade fever, malaise, and pharyngitis, with mucocutaneous manifestations appearing 1 to 2 days later. The typical lesions are often limited to macules, papules, and/or vesicles involving the palms and soles as well as painful erosions (herpangina) involving the oral mucosa. As with all enteroviral infections, HFMD is transmitted via the fecal-oral route and can be transmitted via contact with saliva, vesicular fluid, respiratory secretions, and feces.

The serotype of enterovirus implicated recently in more severe presentations of HFMD is coxsackievirus A6, which historically has been an uncommon cause of HFMD worldwide and has not been previously reported in the United States. However, since 2008, coxsackievirus A6 has been implicated in several epidemics of HFMD in Europe and Asia[1-5] as well as an atypical outbreak of HFMD in the United States that started in November 2011 with ongoing cases continuing to be reported to the Centers for Disease Control and Prevention.[6,7] The clinical and epidemiologic features of the outbreak reported in the United States were unusual and notable for reports of clinical disease in adults, presentation during the winter and spring months, and more severe clinical presentation. The clinical manifestations associated with coxsackievirus A6 infection include hemorrhagic vesicles and bullae that often progress to crusted erosions and a more widespread and/or generalized distribution that favors the extremities but may also involve the face, buttocks, and trunk. Oral mucosal involvement may be seen but may be minimal or absent. More severe cutaneous manifestations may occur in children with underlying atopic dermatitis or other skin disease.

Complications of HFMD are rare and include bacterial superinfection of cutaneous lesions, dehydration secondary to painful oral erosions and decreased oral intake, and viral meningitis or meningoencephalitis. Onychomadesis, or shedding of the nail plate, is not uncommonly seen after HFMD, including that caused by coxsackievirus A6.

Treatment of HFMD is largely supportive, focusing on supportive care and treatment of associated pain and pruritus, if present. Oral analgesics as mouthwash, lozenge, or spray can be helpful for painful oral erosions. Spontaneous resolution of skin lesions occurs in 1 to 2 weeks.

The differential diagnosis of a widespread papulovesicular exanthem in a young child includes disseminated herpes simplex virus infection, eczema herpeticum, varicella-zoster virus infection, erythema multiforme, Stevens-Johnson syndrome, papular urticarial/arthropod bite reaction, and Gianotti-Crosti syndrome. When HFMD is suspected but the clinical presentation is atypical, diagnostic evaluation may include enterovirus polymerase chain reaction from a skin lesion to confirm the diagnosis. Serotyping, if indicated, may be performed at some reference laboratories but is not typically required for cases of HFMD.

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