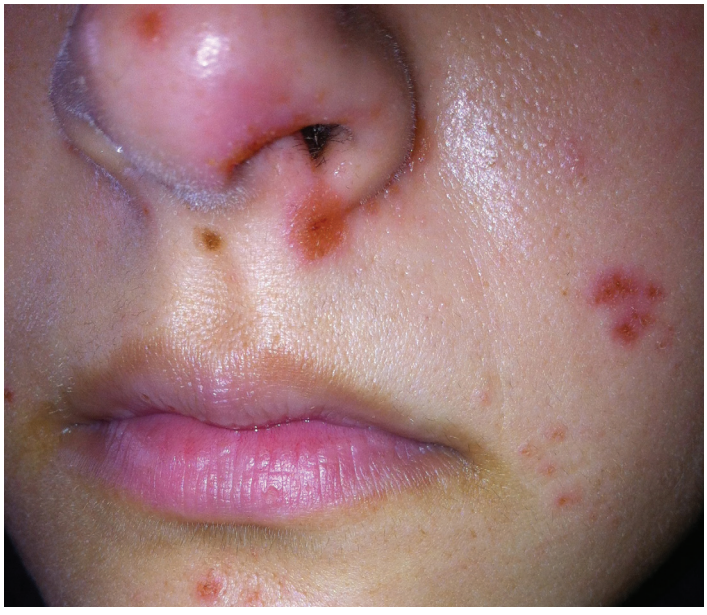
Woman, 32, With Crusty Red Blisters

Purvi Amin, MPA, PA-C, Lisa Daitch, MPAS, PA-C

FIGURE 1



The patient's facial lesions were papulovesicular, with weeping, honey-colored crusting.

A 32-year-old Korean woman presented with a rash on her scalp, face, palms, soles, and genital region and with sores in the oral cavity. The blisters were red and flat with some crusting, particularly on the scalp and face. The patient described the blisters as very painful, adding that it hurt to walk, grasp objects, and drink fluids. Associated symptoms included painful urination, sore throat, malaise, and fever of up to 103°F. She was taking acetaminophen and ibuprofen to alleviate the fever and pain.

Medical history was unremarkable. Social history was negative for recent changes in sexual partner or travel to foreign countries.

Physical examination revealed numerous flat, erythematous lesions. Lesions on the face and scalp had developed a weeping, honey-colored crust (see Figure 1, left, and Figure 2, page 40). The lesions were tender to the touch, particularly on the palms and soles.

Further questioning revealed that the patient's 18-month-old son had exhibited similar symptoms two to three days prior to her illness.

DIFFERENTIAL DIAGNOSIS

Because multiple bacterial and viral diseases manifest in this fashion, the differential diagnosis included the following disorders:

Erythema multiforme. This skin condition may result from an allergic or hypersensitivity reaction to certain drugs or from infections. Infections that can cause erythema multiforme include herpes simplex virus and mycoplasma. Pa-

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Purvi Amin is in private practice near Orlando. **Lisa Daitch** is an Associate Professor at Georgia Regents University in Augusta.

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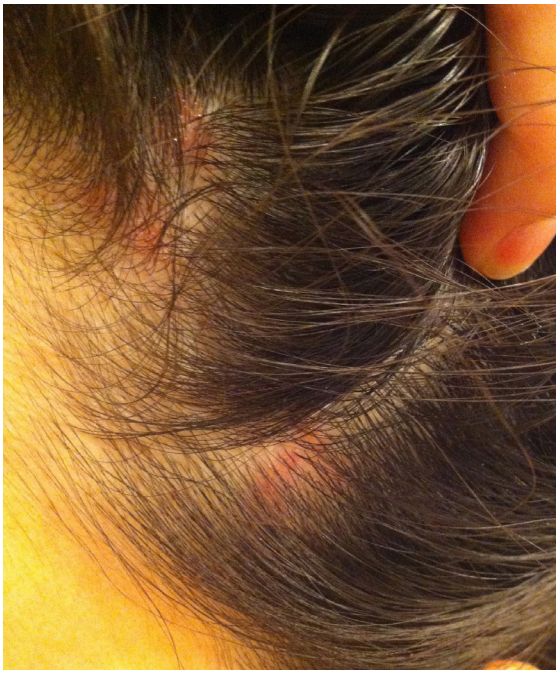
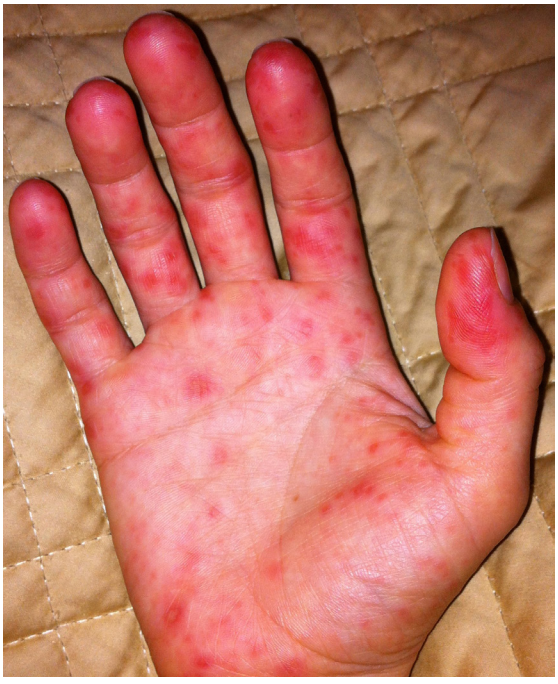
FIGURE 2



Erythematous macular lesions, some weepy and crusting, were found on the scalp.

FIGURE 3



Palmar lesions were maculopapular with an erythematous base and were tender to the touch.

tients present with lesions on the palms (see Figure 3), soles, extremities, face, or trunk. The lesions can appear as a nodule, papule, macule, or vesicle. Initially, in the mild form, the lesions may appear as hives or target-shaped rashes, occurring on the face and acral surfaces. A severe form of erythema multiforme, known as *Stevens-Johnson syndrome*, is characterized by rash, mucosal involvement, and systemic symptoms.[1]

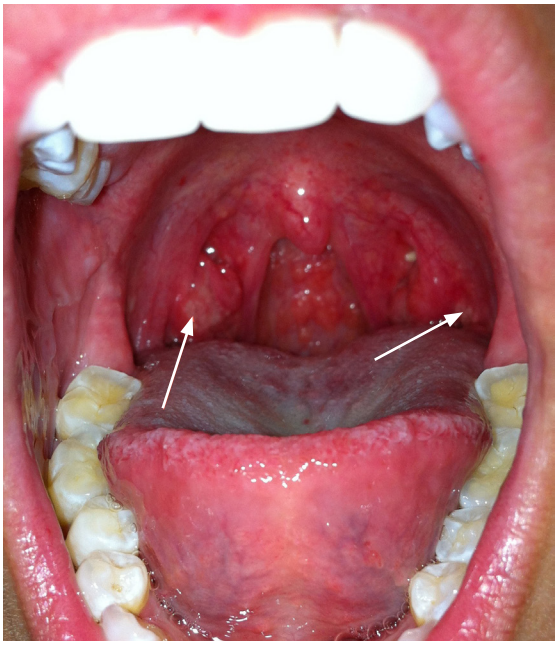
Herpes zoster. This viral infection is caused by the varicella-zoster virus, which also causes chicken pox. The virus lies dormant within a single sensory ganglion and may reappear as shingles along the dermatome of that nerve. Patients may experience burning or shooting pain with tingling or itching before the rash appears; vesicular lesions with erythematous bases appear days later. The rash occurs unilaterally on the body or face and does not cross the midline. A viral culture may be obtained for identification.[2]

Herpetic gingivostomatitis. This infection is most commonly caused by herpes simplex virus type 1, the same virus that causes cold sores. Patients may present with ulcerations along the buccal mucosa and gums. The infection manifests with systemic symptoms, including malaise, fever, irritability, and cervical adenopathy. A viral culture will identify the etiology.[3]

Impetigo. This skin infection is typically caused by bacteria, predominantly *Staphylococcus aureus*, *Streptococcus pyogenes*, or a combination. Infections generally occur after a break in the skin surface. The most common presentation is a rash that spreads to different parts of the body after scratching. Skin lesions can occur on the face, lips, or extremities. Initially vesicular, the lesions generally form a honey-colored crust after fluid discharge. The clinician should take a skin or fluid sample from the lesion to culture, which may identify the pathogen.[4]

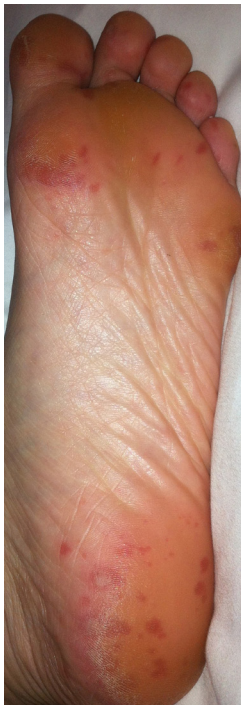
Syphilis. This sexually transmitted infection is caused by the spirochete *Treponema pallidum*. In primary syphilis, patients can develop a painless sore, or *chancre*, on the genitals, rectal area, or mouth. If left untreated, the disease can progress to secondary syphilis, manifesting as a pale pink or reddish maculopapular rash on the palms and soles. The rash can be associated with fever, sore throat, myalgia, and fatigue. It is important to rule out syphi-

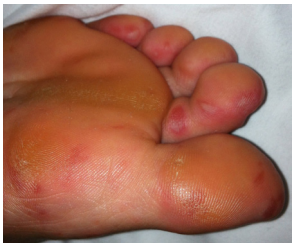
FIGURE 4



Lesions appeared in the oral cavity along the anterior pharynx.

FIGURE 5





Tender erythematous maculopapular lesions on the soles spread to interdigital areas.

lis because, left untreated, it can lead to cardiac and neurologic complications. Screening tests include VDRL and the rapid plasma reagin (RPR) test, both of which assess for antibodies to the organism, and dark field microscopy of ulcerations to identify the organism.[5]

Also included in the differential diagnosis for the patient's symptoms was hand-foot-mouth disease (HFMD), discussed below.

DISCUSSION

HFMD is an acute viral illness most often affecting children younger than 5 and occurring in summer to early fall months. HFMD manifests with fever and papulovesicular eruptions; lesions often appear in the oral cavity first, spreading to the palms, soles, and buttocks. Route of transmission is usually fecal-oral or through respiratory droplets, oral secretions, or direct contact with fluid-filled vesicles.[6-8] The highly contagious nature of the virus causes it to spread to close contacts and family members and leads to outbreaks in schools and daycare centers.[9,10]

In the United States, the most common etiology of HFMD is coxsackievirus A16.[7] Another causative agent, enterovirus 71 (EV71), has been found responsible for HFMD epidemics in southeast Asia and Australia.[11,12] Recently, the coxsackievirus A6 (CV A6) strain has been linked to outbreaks of HFMD.[7,9] This strain may produce an atypical manifestation of skin lesions on the face, trunk, and extremities. The lesions may also appear larger than usual and have a vesiculobullous rather than the more typical papulovesicular appearance. The course of the illness differs in severity depending on the strain of the virus causing HFMD.[9]

CLINICAL PRESENTATION

The acute phase of HFMD typically begins with prodromal symptoms such as fever, malaise, and sore throat. Erythematous ulcerations usually appear in the oral cavity first (see Figure 4) and often cause symptoms such as sore throat, dysphagia, or dryness. As the disease progresses, cutaneous lesions spread to the face, extremities, interdigital areas (see Figure 5), trunk, and perianal area (which may cause dysuria). The lesions may initially appear as erythematous macules or papules, transforming to vesicles as the disease progresses. The mucocutaneous lesions are usually asymptomatic but can be tender to touch or pressure and may leak fluid.[7,10]

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In only a few cases—caused by CV A6—have lesions in the scalp been reported; the mechanism of action is unknown.[10] Instances of lesions invading the nails have been reported, causing desquamation and shedding. This condition is known as onychomadesis.[9,11]

DIAGNOSIS

Serologic testing and viral cultures can identify the exact strain of virus causing HFMD and are particularly useful in unusual presentations. Polymerase chain reaction (PCR) testing yields a high sensitivity and specificity for the causative agent. Histologic examination of skin biopsies may show lymphocytic infiltrates and areas of degeneration along the epidermis. However, most cases are diagnosed based on clinical presentation alone.[6,12]

TREATMENT AND MANAGEMENT

Management of HFMD is primarily symptomatic, consisting of supportive care that includes use of antipyretics, NSAIDs, and adequate fluid intake to prevent dehydration. The disease is usually self-limited, resolving within seven to 10 days without sequelae.[6,9] Aseptic meningitis and other severe complications (especially pulmonary and neurologic), most often associated with EV71 infection, can occur in vulnerable populations, including elderly, pregnant, and immunocompromised patients.[9,11] Because the virus is excreted directly from palmar lesions onto the hands, proper hygiene and handwashing techniques offer an exceptionally strong protective effect, preventing transmission and reducing morbidity.[8]

PATIENT OUTCOME

Based on clinical findings and patient history, the patient was diagnosed with HFMD, which she contracted from her son. Laboratory testing and viral cultures were deemed unnecessary in this case. Treatment was symptomatic, and her skin lesions resolved in one to two weeks.

At follow-up, the patient stated her skin lesions resolved completely without leaving any scars. She also indicated that her nails peeled and shed approximately four weeks after diagnosis, but began to regrow normally four months after diagnosis.

CONCLUSION

Clinicians need to recognize that, although it is uncommon outside the pediatric population, HFMD may occur in adults with intact immune systems. The presentation of HFMD in adults may be atypical, including cutaneous lesions in the scalp and shedding of the nails several weeks after diagnosis. Depending on the viral strain involved, adult patients may have more severe illness and may take longer to recover. Therefore, early diagnosis is important to help prevent the spread of infection and reduce the severity of complications. **CR**

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