An 8-year-old boy presented with flesh-colored lesions of 3 years’ duration in the perioral area. He had a habit of lip licking. The lesions were asymptomatic. The boy was otherwise healthy and was not taking any medication. There was no family history of similar skin lesions.

On physical examination, numerous flesh-colored, flat-topped, papular and plaque-like lesions were visible in the perioral area. No other cutaneous or systemic findings were noted.

What’s your diagnosis?
Answer: Flat warts (verrucae plana)

Flat warts, also known as plane warts or verrucae plana, are caused by human papillomavirus (HPV). The virus is transmitted directly from one person to another. Flat warts might spontaneously resolve over a period of months to years. A variety of treatment options are available, including watchful observation.

**Etiology**

HPV is a double-stranded DNA virus of the Papovaviridae family. Approximately 120 serotypes of HPV have been identified. The virus is species-specific and tissue-specific. Most flat warts are caused by HPV types 3 and 10; less commonly, the culprits are types 26 to 29 and 41, which are trophic to human skin.

**Epidemiology**

Cutaneous warts affect approximately 20% of children at some point. Flat warts account for approximately 4% of cutaneous warts. The condition is seen mainly in children and young adults. Flat warts are more common in white children than in nonwhite children.

The virus is transmitted by close physical contact, including person-to-person and autoinoculation. The virus, however, does not spread to histologically dissimilar sites, such as the oral cavity and genitalia. Moist environments and disruption of the epidermal barrier increase the chance of infection. Children with a family member or many classmates with flat warts have a higher risk of having flat warts themselves. Although the condition primarily is seen in healthy individuals, those with immunodeficiency are at increased risk.

**Histopathology**

Histologic examination of a classic lesion shows hyperkeratosis and acanthosis. In contrast with common warts and planar warts, papillomatosis is either mild or absent. The stratum corneum is characterized by basket-weave orthokeratosis with no parakeratosis, and the rete ridges are less branched than in common warts.

**Clinical Manifestations**

Typically, flat warts present as multiple, flat-topped, smooth-surfaced papules, sometimes grouped or confluent. The lesions usually are yellow-brown or flesh-colored. Individual lesions generally are 2 to 5 mm in diameter. The lesions often are asymptomatic.

Sites of predilection include the face, neck, dorsum of the hands, and along scratch marks and, in adults, areas that are shaved. A linear array of lesions, due to autoinoculation, is characteristic. In individuals with immunodeficiency or immune reconstitution syndrome, the lesions can be extensive and large.

**Diagnosis**

The diagnosis is usually clinical. Dermoscopy aids visualization of the morphologic features. A skin biopsy should be considered if the diagnosis is in doubt.

**Differential Diagnosis**

Flat warts should be differentiated from molluscum contagiosum, acne, skin tags, seborrheic keratoses, angiofibromas, chickenpox, epidermodysplasia verruciformis, and papular acrodermatitis of childhood (Gianotti-Crosti syndrome).

**Complications**

The lesions can be cosmetically unsightly and socially embarrassing, especially if they occur on exposed areas. Affected individuals can spread the disease to other individuals or to themselves (autoinoculation).

**Prognosis**

Approximately 40% of cutaneous warts resolve spontaneously within 2 years. Flat warts have higher rates of spontaneous resolution. Regression may be preceded by an eruption of numerous warts. In the immunocompromised host, lesions tend to persist. Recurrence after treatment is common as a result of the persistence of HPV in grossly normal appearing squamous epithelium beyond the area of treatment.

**Treatment**

To avoid spread of the infection, bathtub isolation is important. Some authors suggest watchful observation of the lesions while awaiting spontaneous resolution. Most authors suggest active treatment of lesions out of concern about transmission and autoinoculation and concern that spontaneous resolution may take months to years.

Active treatments can be mechanical (eg, liquid nitrogen cryotherapy, duct tape, laser therapy, excision), chemical (eg, salicylic acid, cantharidin, bleomycin), and immune-modulating (eg, imiquimod). Each treatment decision must be individualized, taking into consideration the efficacy and cost of the treatment; the experience level of the physician; the location, size and number of lesions; and the preference of the patient.

The two most common treatments are salicylic acid and cryotherapy with liquid nitrogen. Salicylic acid is a kerolytic agent. Topical application of salicylic acid has a cure rate of 75%. Advantages of salicylic acid include convenience (it can be bought over the counter), minimal cost, minimal discomfort, and reasonable effectiveness. Disadvantages
include length of time to achieve response and perilesional irritation.\textsuperscript{11,12} Salicylic acid liquid must be applied daily, and salicylic acid patches every other day.\textsuperscript{11}

Topical vitamin A acid (tretinoin), as well as immunotherapy with imiquimod or 5-fluorouracil, also can be considered in some cases.

Cryotherapy with liquid nitrogen has a similar cure rate as salicylic acid therapy.\textsuperscript{12} Liquid nitrogen, which has a temperature of $-196^\circ$C, is used to freeze the wart. Traditionally, liquid nitrogen is applied with a cotton-tipped applicator or via a spray gun until the wart has a 2 mm white halo around it. Repeating the freeze, with a complete thaw in between, increases the effectiveness of the treatment.\textsuperscript{12}

The cryotherapy procedure is painful and might be unacceptable for very young patients. Application of a eutectic mixture of local or topical anesthetic can be considered before the liquid nitrogen is applied.\textsuperscript{5}

Other treatment modalities usually are reserved for more resistant warts. Consultation with a dermatologist should be considered.

Due to the abundance of warts and his young age, this child was treated with a regimen of imiquimod in the morning and topical tretinoin at night, with complete resolution occurring after 3 months.

Alexander K. C. Leung, MD—Series Editor, is a clinical professor of pediatrics at the University of Calgary and a pediatric consultant at the Alberta Children’s Hospital in Calgary.

Benjamin Barankin, MD, is medical director and founder of the Toronto Dermatology Centre.

REFERENCES