

2. Nanda A, Alsaleh QA, Al-Sabah H et al. Geroderma osteodysplastica/wrinkly skin syndrome: report of three patients and brief review of the literature. *Pediatr Dermatol* 2008;25:66–71.
3. Cohen PR, Grossman ME, Almeida L et al. Tripe palms and malignancy. *J Clin Oncol* 1989;7:669–678.
4. Skiljevic DS, Nikolic MM, Jakovljevic A et al. Generalized acanthosis nigricans in early childhood. *Pediatr Dermatol* 2001;18:213–216.

ARTI NANDA, M.D., D.N.B.E.
Kuwait
e-mail: artinanda@hotmail.com.

POPSICLE PANNICULITIS IN A 5-MONTH-OLD CHILD ON SYSTEMIC PREDNISOLONE THERAPY

To the Editor:

Popsicle panniculitis is a type of cold panniculitis that affects the cheeks of young children, is self-limited, and may be mistaken for cellulitis. The diagnosis may be missed unless a detailed history is elicited. We present a 5-month-old child who developed bilateral popsicle panniculitis on his cheeks despite being on systemic prednisolone for a large facial hemangioma.

A 5-month-old African-American child being treated for a large facial hemangioma presented to clinic with new painful, indurated, erythematous plaques on both cheeks. He had begun oral prednisolone 10 weeks earlier (3 mg/kg/day initially, tapered to 2 mg/kg/day over the first month, and 1.8 mg/kg/day over the next 6 weeks). Three days prior to presentation, the patient had been given a popsicle by his mother because he was teething. Later that day, he developed tender nodules on his cheeks. On physical examination, the infant was healthy-appearing and afebrile. He had a large midline facial hemangioma on the nose and forehead and erythematous, edematous plaques on both cheeks (Fig. 1). A



Figure 1. The patient demonstrates erythematous plaques on both cheeks 3 days after being given a popsicle.

diagnosis of popsicle panniculitis was made. Within 2 weeks, the lesions fully resolved without therapy, while his prednisolone was decreased to every other day dosing.

Popsicle panniculitis is rarely documented, but likely much more common than is reported (1–3). Popsicle panniculitis is a type of cold panniculitis, a well-described condition that usually occurs in neonates and infants following cold injury. Although popsicle panniculitis may sometimes be misdiagnosed as bacterial cellulitis, the clinical appearance and history are generally sufficient to make the diagnosis. Biopsy specimens of cold panniculitis show a lobular mixed inflammatory cell infiltrate, most prominent at the dermo-subcutaneous junction (4,5). Cold panniculitis is self-limited and patients generally fully recover several weeks after exposure. The cold sensitivity declines as children age. Of interest, our patient was on high-dose oral prednisolone therapy for the treatment of his facial hemangioma. Systemic corticosteroid therapy might be expected to suppress inflammation and decrease the likelihood of developing inflammation after cold injury. We doubt that the steroid taper contributed to his panniculitis because the amount of steroid he was receiving had not been decreased over the preceding 6 weeks. To our knowledge, this is the first report of popsicle panniculitis in the setting of systemic corticosteroid therapy.

Although the exact mechanism is unknown, the pathogenesis of cold panniculitis may be similar to that of subcutaneous fat necrosis of the newborn (SFN) (6). In SFN, the greater concentration of saturated fatty acids in the subcutaneous fat of neonates compared with adult subcutaneous fat has been theorized to explain the increased susceptibility to crystallization and necrosis under cold stress (7,8).

Our patient is interesting because he developed cold panniculitis despite being on systemic corticosteroid therapy. As popsicle panniculitis resolves spontaneously, we suggest that unnecessary biopsies or treatment may be avoided by eliciting a careful history in consideration of the physical presentation.

REFERENCES

1. Epstein EH Jr, Oren ME. Popsicle panniculitis. *N Engl J Med* 1970;282:966–967.
2. Rajkumar SV, Laude TA, Russo RM et al. Popsicle panniculitis of the cheeks. A diagnostic entity caused by sucking on cold objects. *Clin Pediatr (Phila)* 1976;15:619–621.
3. Day S, Klein BL. Popsicle panniculitis. *Pediatr Emerg Care* 1992;8:91–93.
4. Duncan WC, Freeman RG, Heaton CL. Cold panniculitis. *Arch Dermatol* 1966;94:722–724.

5. Hultcrantz E. Haxthausen's disease. Cold panniculitis in children. *J Laryngol Otol* 1986;100:1329–1332.
6. Hicks MJ, Levy ML, Alexander J et al. Subcutaneous fat necrosis of the newborn and hypercalcemia: case report and review of the literature. *Pediatr Dermatol* 1993;10:271–276.
7. Borgia F, De Pasquale L, Cacace C et al. Subcutaneous fat necrosis of the newborn: be aware of hypercalcaemia. *J Paediatr Child Health* 2006;42:316–318.
8. Katz DA, Huerter C, Bogard P et al. Subcutaneous fat necrosis of the newborn. *Arch Dermatol* 1984;120:1517–1518.

FRANKLIN W. HUANG, Ph.D.
 DAVID R. BERK, M.D.
 SUSAN J. BAYLISS, M.D.
 St. Louis, Missouri
 e-mail: DBerk@im.wustl.edu

CHROMOSOME 6 ABNORMALITY WITH ASSOCIATED DYSMORPHOLOGICAL FEATURES AND ERYTHROKERATODERMA

To the Editor:

In Sobey et al (1) "Mosaic Chromosome 6 Trisomy in an Epidermal Nevus," the authors document a case which showed skin biopsy findings of a mosaic trisomy 6 from a linear epidermal nevus. Previously, chromosome 6 mosaicism has only been reported in an infant, with multiple other genetic abnormalities. It has also been reported in biopsy samples of some neoplastic conditions of the skin, such as Merkel cell carcinoma. This article prompted us to share an interesting case of a 3-year-old boy who has a chromosome 6 abnormality, along with skin findings.

A 3-year-old boy patient presented to our clinic with a diagnosis of a chromosome 6 duplication with a karyotype of 46, XY, dup(6)(q13.2-q16.2) in 2004. At the time, he had marked developmental delay, febrile seizures, dysmorphological features, and erythrokeratoderma. Birth history is only significant for exposure to second-hand smoke, and amoxicillin which was used to treat pharyngitis early in pregnancy. Otherwise, the mother had no history of medication use, or of toxic or chemical exposures during pregnancy. Our patient had a normal spontaneous vaginal delivery and was borderline small for gestational age. Family history did not reveal any dermatologic diseases. The father was noted to have hepatitis C. Dysmorphological features present in this patient include posteriorly rotated, downward tipped ears, reverse epicanthus folds, broad nasal root, and a small normally formed head (tenth percentile). At presentation, he had transient, migrating well-defined erythematous to pink, thin plaques with a fine white scale appearing in a blaschkonian arrangement on the bilateral arms and lower extremities. The patient's mother



Figure 1. Erythematous thin plaque with fine white scale located on upper extremity.

stated the plaques first appeared when the patient was approximately 1 year old. She also noticed that the plaques seem to come and go and would migrate on the skin (Fig. 1). The differential diagnosis at clinical presentation included erythrokeratoderma variables, ichthyosis linearis circumflexa, and atopic dermatitis. However, no double-edged scale was present, as seen in ichthyosis linearis circumflexa. The family refused the biopsy we requested to further characterize this migratory eruption and the patient was presumptively diagnosed with an unclassified migratory ichthyosis. Treatment included keratolytics: ammonium lactate cream was used in one area and tretinoin 0.025% cream in another area without much therapeutic success.

Karyotyping was repeated and our patient was found to have an insertion of maternal chromosomal material of unknown origin in 6q15 in all cells. Karyotype was 46, XY, ins(6;?)(q15;?). In reviewing the previous gene mapping of disease to this locus, no mention has been made of a syndrome which incorporates dermatologic findings with dysmorphological features (2). Symptoms associated with chromosome trisomy 6q are similar to the presentation of our patient, and include growth delays, severe mental retardation, psychomotor retardation, downward slanting palpebral fissures, malformed ears, and neck abnormalities (3). In the Klein et al (4) article on 6q deletions, they report patients who have all had developmental delay, craniofacial dysmorphism, and functional eye disorders. This suggests that the genes affecting craniofacial development are located in 6q16.2-q21. Another article reported that duplication of 6q21q22.1 resulted in moderate mental retardation and facial dysmorphism (5). While it is unclear whether the origin of material is a duplication, or an insertion at locus 6q.15, it nevertheless represents another chromosome 6 abnormality with interesting dermatologic findings.

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.