similarity, which also explains the recently described possibility of cross-reactivity between the 2 substances.¹¹ It is also possible that the lower absorption of pimecrolimus is responsible for the lower incidence of this secondary effect, as seen with low-concentration tacrolimus.⁴

References

- Izikson L, English JC 3rd, Zirwas MJ.
 The flushing patient: differential diagnosis, workup, and treatment. J Am Acad Dermatol. 2006;55:193-208.
- Lubbe J, Milingou M. Images in clinical medicine. Tacrolimus ointment, alcohol, and facial flushing. N Engl J Med. 2004;351:2740.

- Sticherling M, Brasch J. Alcohol: intolerance syndromes, urticarial and anaphylactoid reactions. Clin Dermatol. 1999;17:417-22.
- Soter NA, Fleischer AB Jr., Webster GF, Monroe E, Lawrence I. Tacrolimus ointment for the treatment of atopic dermatitis in adult patients: part II, safety. J Am Acad Dermatol. 2001;44: S39-46.
- Alaiti S, Kang S, Fiedler VC, Ellis CN, Spurlin DV, Fader D, et al. Tacrolimus (FK506) ointment for atopic dermatitis: a phase I study in adults and children. J Am Acad Dermatol. 1998;38:69-76.
- 6. Reitamo S, Wollenberg A, Schöpf E, Perrot JL, Marks R, Ruzicka T, et al. Safety and efficacy of 1 year of tacrolimus ointment monotherapy in adults with atopic dermatitis. The European Tacrolimus Ointment Study Group. Arch Dermatol. 2000;136:999-1006.

- Milingou M, Antille C, Sorg O, Saurat JH, Lubbe J. Alcohol intolerance and facial flushing in patients treated with topical tacrolimus. Arch Dermatol. 2004;140:1542-4.
- Knight AK, Boxer M, Chandler MJ. Alcohol-induced rash caused by topical tacrolimus. Ann Allergy Asthma Immunol. 2005;95:291-2.
- Calza AM, Lubbe J. Tacrolimus ointmentassociated alcohol intolerance in infants receiving ethanol-containing medication. Br J Dermatol. 2005;152: 569.
- Ramsay HM, Harden PN. Cyclosporin-induced flushing in a renal transplant recipient resolving after substitution with tacrolimus. Br J Dermatol. 2000;142:832-3.
- Shaw DW, Maibach HI, Eichenfield LF. Allergic contact dermatitis from pimecrolimus in a patient with tacrolimus allergy. J Am Acad Dermatol. 2007;56:342-5.

Congenital Self-Limiting Tufted Angioma

D. Barco, a E. Baselga, a A. Ribé, b and A. Alomara

aServicio de Dermatología and aServicio de Anatomía Patológica, Hospital de la Santa Creu i Sant Pau, Barcelona, Spain

To the Editor:

We report the case of a 1-month-old boy who had had a 5-cm violaceous plaque on the right arm since birth. The plaque was not hot, pulsatile, or painful and had a peau d'orange surface covered with downy hair (Figure 1). The biopsy showed a normal epidermis with dermal vascular proliferation in the form of lobules. The lobules were arranged in a birdshot pattern and were composed of endothelial cells with no signs of atypia or mitosis and with occasional half-moon-shaped peripheral vascular spaces (Figure 2). Immunostaining was negative for glucose transporter-1 (GLUT1). The histologic and immunohistochemical characteristics of the lesion suggested a diagnosis of congenital tufted angioma. The tumor became gradually flatter and had partially disappeared by the time the baby was 1 year old (Figure 3).

Tufted angioma (TA) is a rare benign vascular tumor. Most TAs are acquired

Figure 1. Clinical appearance of the lesion at birth.

and appear during the first year of life or in young people as violaceous macules, plaques, or nodules high on the torso, on the neck, or on the arms. ^{1,2} They may present hyperhidrosis, be painful to the touch, or covered with

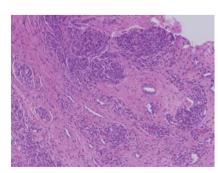


Figure 2. Histologic appearance of the lesion (hematoxylin–eosin, ×100).



Figure 3. Clinical appearance of the lesion at 1 year of age.

lanugo.¹⁻³ Although the tumor appears to be sporadic, familial cases have been reported with autosomal dominant inheritance.⁴ Congenital cases^{2,3} and other cases with a very late onset⁴ have also been reported.

The course of TA consists of appearance during the first year of life⁵ and growth over months or years, 1,2 with subsequent stabilization,1,4 and little tendency to spontaneously remit.4 Acquired or late-onset TAs with spontaneous remission are rare^{5,6} and lesions are thought to usually persist.1 Unlike late-onset TA, congenital and early-onset cases show a greater tendency toward spontaneous remission,6,7 and only 1 case of congenital TA that did not improve over time has been reported.2 Remission of TA occurs over months or years and is partial or total.^{6,7}

The appearance of disseminated intravascular coagulation with severe thrombopenia (Kasabach-Merritt syndrome [KMS]) is a rare complication of congenital TA.^{1,3} KMS is associated with vascular lesions including kaposiform hemangioendotheliomas (KHE) and TA.⁸

The microscopic appearance of TA is characteristic²⁻⁴ and shows multiple cell lobules in the middle and lower dermis and in the subcutaneous tissue. The lobules are composed of endothelial cell aggregates arranged in a spiral pattern around the vascular plexuses. When these lobules protrude into the surrounding vascular walls, they take on a half-moon shape,

characteristic of TA. Immunostaining is negative for GLUT1, unlike in infantile hemangiomas.

The histology of KHE is similar to that of TA as the cells are also organized in capillary lobules (though they are larger, deeper, less circumscribed, and separated by connective tissue). KHE is associated with a greater number of cells, with a higher proportion of fusiform cells. Both diseases show half-moon-shaped capillary spaces around the vascular lobules^{4,9}; the spaces appear to correspond to lymph channels.¹⁰

Enjolras et al⁸ performed a histologic analysis of vascular lesions compatible with TA or KHE complicated by KMS and found a lymphatic component in most of them. The use of the D2-40 monoclonal antibody, which is a specific marker for lymphatic endothelium, ¹⁰ has made it possible to show the presence of lymphatic capillaries in the periphery of the vascular lobules in TA and KHE, though with a different distribution. ¹⁰

TA and KHE share a similar clinical appearance, 9 sometimes share a common complication (KMS), have similar histologic characteristics,9 and have lymph vessels around the capillaries.¹⁰ In another study by Enjolras et al,¹¹ the histologic analysis of vascular tumors complicated by KMS showed findings compatible with KHE during the active phase and more typical of TA thereafter. This set of characteristics common to both tumors supports the idea of a single spectrum of vascular lesions in which the aggressive extreme would be occupied by KHE and the more benign extreme by TA.

A wait-and-see approach in earlyonset or congenital cases of TA seems to be the most acceptable course of treatment,^{5,6} as no cases of malignant transformation have been reported.^{1,2} Treatment is considered in cases where vital organs are compromised or symptoms are marked, or to improve the appearance of the patient.⁷ The recommended treatment is surgical removal.⁷ Systemic corticosteroids, vincristine, or other alternatives may be used, though with highly variable results. ^{1,2} A wait-and-see approach may be valid for adult forms. A review by Ishikawa et al⁶ found that treatment of adult TA tended to be applied at an early stage, thus making it difficult to obtain information on the course of the primary lesion and on whether there is a tendency to spontaneously remit.

We present the case of a congenital TA—a rare form that appears to have a greater tendency toward spontaneous remission than later-onset forms. For this reason, we recommend a wait-and-see approach in cases of early-onset TA or TA present at birth.

References

- Kamath GH, Bhat RM, Kumar S. Tufted angioma. Int J Dermatol. 2005; 44:1045-7.
- Satter EK, Graham BS, Gibbs NF. Congenital tufted angioma. Pediatr Dermatol. 2002;19:445-7.
- Enjolras O, Picar A, Soupre V. Hemangiomes congenitaux et autre tumeurs vasculaires infantiles rares. Ann Chir Plast Esthet. 2006;51:339-46.
- Requena L, Sangueza OP. Cutaneous vascular proliferation. Part II. Hyperplasias and benign neoplasms. J Am Acad Dermatol. 1997;37:887-919; quiz 920-2.
- 5. McKenna KE, McCusker G. Spontaneous regression of a tufted angioma. Clin Exp Dermatol. 2000;25:656-8.
- Ishikawa K, Hatano Y, Ichikawa H, Hashimoto H, Fujiwara S. The spontaneous regression of tufted angioma. A case of regression after two recurrences and a review of 27 cases reported in the literature. Dermatology. 2005;210: 346-8.
- Browning J, Frieden I, Baselga E, Wagner A, Metry D. Congenital, selfregressing tufted angioma. Arch Dermatol. 2006;142:749-51.
- Enjolras O, Wassef M, Mazoyer E, Frieden IJ, Rieu PN, Drouet L, et al. Infants with Kasabach-Merritt syndrome do not have «true» hemangiomas. J Pediatr. 1997;130:631-40.
- 9. Bienaime A, Rojat-Habib MC, Hesse S, Pelissier JF, Bonerandi JJ. Giant

- vascular tumour in an adult: tufted angioma or kaposiform hemangioendothelioma. Ann Dermatol Venereol. 2006;133:553-6.
- 10. Arai E, Kuramochi A, Tsuchida T, Tsuneyoshi M, Kage M, Fukunaga M,
- et al. Usefulness of D2-40 immunohistochemistry for differentiation between kaposiform hemangioendothelioma and tufted angioma. J Cutan Pathol. 2006;33: 492-7.
- Enjolras O, Mulliken JB, Wassef M, Frieden IJ, Rieu PN, Burrows PE, et al. Residual lesions after Kasabach-Merritt phenomenon in 41 patients. J Am Acad Dermatol. 2000;42:225-35.

Staphylococcus aureus Sepsis as a Complication of Scabies

S. Mallo-García, E. Taborga-Díaz, S. Menéndez-Cuervo, and J. Santos-Juanes-Jiménez

^aServicio de Dermatología II and ^aUnidad Pediátrica de Cuidados Intensivos, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain

To the Editor:

Scabies is a parasitosis considered to be a public health problem especially in developing regions of the world. Local and systemic secondary infections and other complications like acute poststreptococcal glomerulonephritis are major causes of morbility in this type of patient.¹

A 2-month-old infant was admitted to hospital with a diagnosis of scabies and fever, and treatment with permethrin cream, 5%, was prescribed. The day before admission the infant developed a hot, erythematous, edematous plaque below the left knee (Figure 1). Two days later he was transferred to the pediatric intensive care unit with a high fever and respiratory distress. Physical examination revealed pale skin and mucous membranes, tachypnea, and subcostal and intercostal retractions. A hard, erythematous, and hot plaque had formed below the left knee with residual scabies lesions on the overlying skin. Vital signs included a heart rate of 108 beats per minute, respiratory rate of 55 breaths per minute, blood pressure of 51/21 mm Hg, oxygen saturation of 93% by pulse oximetry (oxygen mask with reservoir at 15 L/min), and a body temperature of 38°C.

Laboratory analysis revealed a white cell count of 13 200 cells per mm³ (16% band neutrophils and 20% segmented

neutrophils) and an increase in acute phase reactants (C-reactive protein, 187 mg/L; procalcitonin, 50.5 ng/mL). Empiric treatment was started with intravenous cefotaxime (25 mg/kg/d) and teicoplanin (10 mg/kg/d).

The general condition of the patient deteriorated in the 24 hours following admission to the pediatric intensive care unit with increased signs of respiratory distress. A further chest x-ray revealed empyema and hemothorax in the left lung that required drainage of 35 cm³ of yellowish fluid with a pH of 6.86. The abscess on the knee was also drained (Figure 2). Blood cultures and cultures from the skin lesion and the empyema were positive for Staphylococcus aureus. The prescribed antibiotics were replaced with intravenous cloxacillin (25 mg/kg/d) for 14 days, and a favorable response was seen in the patient.



Figure 1. Hot erythematous and edematous plaque on the leg.

Scabies is an infestation caused by the *hominis* variant of the *Sarcoptes scabiei* mite, a human parasite that tunnels under the epidermis. It affects both sexes and all age groups equally. There are more than 300 million new cases each year all over the world.² Scabies is a highly contagious disease that is generally transmitted by direct human contact, although cases have been described in which transmission occurred through contact with fomites and contaminated animals.

Secondary bacterial infections can sometimes occur in skin lesions and cause local and, less commonly, systemic complications.^{3,4}

A study by Itzhak Brook³ analyzed the bacterial flora found in lesions with secondary infections and found the most common aerobe was *S aureus*, while the most common anaerobes were *Peptostreptococcus* species.



Figure 2. Drainage of purulent material from the abscess on the knee.