

can lead to confusion with other vasculitides, particularly with the ANCA-positive vasculitides (polyarteritis nodosa, microscopic polyangiitis, eosinophilic granulomatosis with polyangiitis, granulomatosis with polyangiitis). As no pathognomonic laboratory or histopathologic criteria exist, the diagnosis is purely clinic and is made by exclusion. A detailed clinical history and a high level of clinical suspicion are paramount.^{1,3-9}

The diagnostic approach to cases of suspected cocaine-induced cutaneous vasculitis should include complete blood count, biochemistry including liver and kidney function tests, erythrocyte sedimentation rate, urinalysis, chest X-ray, fecal occult blood, full thickness skin biopsy, antiphospholipid antibodies, coagulation studies including homocysteine and proteins C and S, cryoglobulins, serum ANCA and ANA antibodies, double-stranded DNA antibodies, rheumatoid factor and complement levels, and serology for HIV, HBV, and HCV. Other tests such as blood, urine, or skin microbiology should be performed as required.^{1,3,4}

The skin lesions usually resolve within 2–3 weeks after cessation of cocaine use. Normalization of laboratory tests can take 2–14 months, though the neutropenia recovers fully in less than 10 days.^{1,3,5,7,8}

There is no consensus regarding treatment of this condition. Obviously, removal of the cause is the most important measure, together with symptom relief. Good clinical outcomes have been reported with the use of nonsteroidal anti-inflammatory drugs for arthralgia and colchicine, dapsone, oral antihistamines, and pentoxifylline for the skin lesions.^{1,3,4} Systemic corticosteroids have not been shown to be effective.

Conflict of interest

The authors declare no conflict of interest.

References

1. Magliocca KR, Coker NA, Parker SR. The head, neck, and systemic manifestations of levamisole-adulterated cocaine use. *J Oral Maxillofac Surg.* 2013;71:487–92.

2. Larocque A, Hoffman RS. Levamisole in cocaine: unexpected news from an old acquaintance. *Clin Toxicol (Phila).* 2012;50:231–41.
3. Yachoui R, Kolasinski SL, Eid H. Limited cutaneous vasculitis associated with levamisole-adulterated cocaine. *J Clin Med Res.* 2012;4:358–9.
4. Salas-Espindola Y, Peniche-Castellanos A, López-Gehrke I, Mercadillo-Pérez P. Leukocytoclastic vasculitis related to cocaine use. *Actas Dermosifiliogr.* 2011;102:825–7.
5. Lawrence LA, Jiron JL, Lin HS, Folbe AJ. Levamisole-adulterated cocaine induced skin necrosis of nose, ears, and extremities. Case report. *Allergy Rhinol.* 2014;5:132–6.
6. Pillow MT, Hughes A. Levamisole-adulterated cocaine induced vasculitis with skin ulcerations. *West J Emerg Med.* 2013;14:149–50.
7. Souied O, Baydoun H, Ghandour Z, Mobarakai N. Levamisole-contaminated cocaine: an emergent cause of vasculitis and skin necrosis. *Case Rep Med.* 2014;2014:434717.
8. de la Hera I, Sanz V, Cullen D, Chico R, Petiti G, Villar M, et al. Necrosis of ears after use of cocaine probably adulterated with levamisole. *Dermatology.* 2011;223:25–8.
9. Chung C, Tumei PC, Birnbaum R, Tan BH, Sharp L, McCoy E, et al. Characteristic purpura of the ears, vasculitis, and neutropenia – a potential public health epidemic associated with levamisole-adulterated cocaine. *J Am Acad Dermatol.* 2011;65:722–5.
10. Neynaber S, Mistry-Burchardi N, Rust C, Samtleben W, Burgdorf WH, Seitz MA, et al. PR3-ANCA-positive necrotizing multi-organ vasculitis following cocaine abuse. *Acta Derm Venereol.* 2008;88:594–6.

A. Imbernón-Moya,* R. Chico, I. de la Hera, M.Á. Gallego-Valdés

Servicios de Dermatología, del Hospital Universitario Severo Ochoa, Avda. de Orellana s/n. 28911 – Leganés (Madrid), Spain

Corresponding author.

E-mail address: adrian.imber88@hotmail.com (A. Imbernón-Moya).

Father-to-Newborn Transmission of Herpes Simplex Virus Infection: A Sweet but Bitter Kiss



Herpes neonatal tras contacto con herpes labial paterno: dulce y amargo beso

Dear Editor,

While rare, neonatal herpes simplex virus (HSV) infection is one of the most severe perinatal infections. Only 10% of cases are acquired after birth. The disease is classified according to clinical presentation as follows: skin, eye, and mouth (SEM) disease (prevalence of ≈45%); central nervous system (CNS) disease (≈30%), and disseminated disease

(≈25%), which has the worst prognosis.¹ No deaths have been reported for SEM disease, but 2% of those affected may develop some degree of impairment by the age of 12 years.² Early recognition and treatment with high-dose acyclovir (60 mg/kg/d) reduces mortality and may improve long-term outcomes.³

We present the case of a 10-day-old newborn female admitted to the hospital with generalized skin lesions that had appeared 5 h previously. The lesions consisted of groups of vesicles and pustules overlying erythematous skin, affecting the trunk and tongue (Fig. 1). The infant did not have fever or neurologic symptoms and was systemically well. There had been no complications during the pregnancy, vaginal delivery, or immediate postpartum period. The mother had had systemic lupus erythematosus for the past 6 years, but this was clinically stable. She had had chickenpox during her childhood but had no genital lesions suggestive of herpes



Figure 1 Groups of vesicles and pustules overlying erythematous skin.

infection. The father, however, had active herpes simplex labialis and admitted to having repeatedly kissed his baby while he had active lesions. HSV type 1 was isolated from vesicular lesions in the neonate by real-time polymerase chain reaction (PCR). The rest of the tests were normal or negative, and included biochemical analysis, blood cell count, coagulation studies, blood culture, PCR for varicella-zoster virus from the newborn's vesicles, and culture and PCR for HSV, enterovirus, and varicella-zoster virus in cerebrospinal fluid. The fundus of the eye and ultrasound imaging of the brain were also normal. Due to the high suspicion of HSV infection, high-dose intravenous acyclovir was administered empirically while awaiting the PCR test results. Once the diagnosis had been confirmed, acyclovir was continued to complete 14 days of treatment. Contact isolation was also established. The girl's condition gradually improved over the next few days, and no sequelae were observed in the follow-up period.

HSV is a common human pathogen. Between 30% and 100% of adults have serologic evidence of the virus. It has been shown that oral HSV excretion is episodic and may occur in the presence or absence of herpes labialis. Importantly and contrary to general belief, reactivation of HSV leads more frequently to asymptomatic viral shedding than to recurrent mucocutaneous lesions.¹

HSV infection in neonates typically presents within the first 4 weeks of life. Skin lesions are an important diagnostic clue, but they are absent in 39% of cases of disseminated disease, 32% of cases of CNS disease, and 17% of cases of SEM disease.⁴ The proportion of neonatal infections due to

HSV type 1 has increased in the last decade, possibly as a consequence of changing sexual habits among parents. An early diagnosis is mandatory and the method of choice is detection of HSV DNA by PCR or virus culture.

Our findings led to a diagnosis of neonatal HSV type 1 infection (SEM disease). We believe that the most likely source of transmission was the father's herpes simplex labialis, although we cannot be entirely certain because virus isolation was not performed. We initiated early empirical treatment about 8 h after onset of the lesions, and may, therefore, have prevented progression to CNS involvement.

The aim of this article is to alert dermatologists to the possibility of postnatally acquired HSV infection in newborns. We emphasize the importance of establishing an early diagnosis and initiating empirical treatment upon the first suspicion. Based on our case, we strongly recommend separation of the newborn from any person with active HSV lesions (mothers with herpes labialis should wear a mask). We also recommend offering anticipatory guidance for parents.

Conflict of interests

The authors declare no conflict of interest.

Acknowledgments

We thank the patient's parents for providing the photographs used in this article and for authorizing their publication. We would also like to thank Rabia Sofia Rashid for correcting the manuscript.

References

1. Gantt S, Muller WJ. The immunologic basis for severe neonatal herpes disease and potential strategies for therapeutic intervention. *Clin Dev Immunol.* 2013;2013:369172.
2. Kimberlin DW, Lin CY, Jacobs RF, Powell DA, Frenkel LM, Gruber WC, et al. Natural history of neonatal herpes simplex virus infections in the acyclovir era. *Pediatrics.* 2001;108:223-9.
3. James SH, Whitley RJ. Treatment of herpes simplex virus infections in pediatric patients: current status and future needs. *Clin Pharmacol Ther.* 2010;88:720-4.
4. Allen UD, Robinson JL. Prevention and management of neonatal herpes simplex virus infections. *Paediatr Child Health.* 2014;19:201-6.

O. Guergué Diaz de Cerio,* M. Rubio Lombraña, A. Barrutia Borque, M.R. González Hermosa

Servicio de Dermatología del Hospital Universitario Cruces, Vizcaya, Spain

Corresponding author.

E-mail addresses: olaneguergue06051988@gmail.com, olane.guerguediazdecerio@osakidetza.eus

(O. Guergué Diaz de Cerio).