

Rosacea in the Pediatric Population

Nicole L. Lacz, MD; Robert A. Schwartz, MD, MPH

GOAL

To gain a thorough understanding of rosacea in the pediatric population

OBJECTIVES

Upon completion of this activity, dermatologists and general practitioners should be able to:

1. Discuss the etiology of rosacea.
2. Explain the possible clinical presentation of rosacea in children.
3. Outline the treatment options for rosacea in the pediatric population.

CME Test on page 112.

This article has been peer reviewed and approved by Michael Fisher, MD, Professor of Medicine, Albert Einstein College of Medicine. Review date: July 2004.

This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of Albert Einstein College of Medicine and Quadrant HealthCom, Inc. Albert Einstein College of Medicine

is accredited by the ACCME to provide continuing medical education for physicians.

Albert Einstein College of Medicine designates this educational activity for a maximum of 1 category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only that hour of credit that he/she actually spent in the activity.

This activity has been planned and produced in accordance with ACCME Essentials.

Drs. Lacz and Schwartz report no conflict of interest. The authors report discussion of off-label use for numerous acne vulgaris medications, including oral tetracycline and doxycycline, as well as topical erythromycin, clindamycin, azelaic acid, and isotretinoin. Dr. Fisher reports no conflict of interest.

Rosacea is a condition of vasomotor instability characterized by facial erythema most notable in the central convex areas of the face, including the forehead, cheek, nose, and perioral and periocular skin. Rosacea tends to begin in childhood as common facial flushing, often in response to stress. A diagnosis beyond this initial stage of rosacea is unusual in the pediatric population. If a child is identified with the intermediate stage of rosacea, consisting of papules and pustules,

an eye examination should be performed to rule out ocular manifestations. It may be beneficial to recognize children in the early stage of rosacea; however, it is uncertain if prophylactic treatment is necessary.

Cutis. 2004;74:99-103.

Accepted for publication March 29, 2004.

Dr. Lacz is a staff physician at Memorial Sloan-Kettering Cancer Center, New York, New York. Dr. Schwartz is Professor and Head of Dermatology, University of Medicine and Dentistry of New Jersey-New Jersey Medical School, Newark.

Reprints: Nicole L. Lacz, MD, Dermatology, UMDNJ-New Jersey Medical School, 185 S Orange Ave, Newark, NJ 07103-2714 (e-mail: nicole.lacz@verizon.net).

Epidemiology

Rosacea in childhood is most likely underreported because of its clinical similarity to other erythematous facial disorders.¹ Rosacea is generally thought of as a disease of fair-skinned, young to middle-aged adults, though it has been noted to affect people of other complexions and ages.² Most full-blown cases in the pediatric population have been in light-skinned children ranging from infants to adolescents.

Etiology

The etiology of rosacea is unknown, though certain exacerbating factors undoubtedly have a role in predisposed individuals.^{3,4} Emotions such as anger, anxiety, and embarrassment can lead to flushing. Environmental conditions such as wind, cold, humidity, or heat from any source (eg, sun, sauna, whirlpool, vigorous exercise) can do the same. Vasodilators such as alcohol or vasodilatory medications can lead to flushing, though these are not likely causes in children. Spicy foods such as chili, curry, and peppers, as well as hot foods and beverages including coffee, tea, and hot chocolate, may contribute to symptoms. Irritants such as alcohol-based cleansers, astringents, perfume, shaving lotion, certain soaps, sunscreen, and facecloths may aggravate rosacea.^{3,4} Saprophytic mites (*Demodex folliculorum* and *Demodex brevis*) may cause an inflammatory or allergic reaction by either blocking hair follicles or acting as vectors for microorganisms that some believe may be responsible for or may trigger rosacea.⁵ Immuno-deficiency, as in patients with human immunodeficiency virus, also may contribute to the development of rosacea.⁶

Genetics plays an uncertain role in the development of blushing and ultimately rosacea. If vasodilator substances or mediators are implicated in the development of rosacea as postulated, the disease may have a genetic basis because such mediators are often under the control of single genes.⁷ In one study, 20% of children with rosacea were found to have a history of rosacea in their immediate families, though this number may be an underestimate because only one parent of each patient was examined and half of the parents clinically diagnosed with rosacea reported no familial involvement.⁸ A family history of perioral dermatitis also may be important, as this condition may be related to rosacea.⁹

Pathophysiology

Chronic transient vasodilation, as occurs with blushing, is the earliest representation of rosacea. The warmth and redness associated with flushing is caused by vasodilation, allowing excess blood flow, and by engorgement of the subpapillary venous plexus.² Flushing without sweating is typically seen in children and is likely due to circulating vasodilator substances or mediators such as bradykinin, catecholamines, cytokines, endorphins, gastrin, histamine, neuropeptides, serotonin, substance P, and vascular endothelial growth factor.¹⁰ A flaw in the autonomic innervation of the cutaneous vasculature also is a likely mechanism.¹⁰

Clinical Description

The first stage of rosacea consists of blushing, in which the face becomes bright red in response to certain stimuli (Figure). Episodes of erythema are recurrent and last longer than normal physiologic flushing, which typically subsides within minutes.¹¹ Telangiectasias can become apparent over time. Children in this early stage of the condition may complain of burning or irritation.

In the second, or intermediate, stage of rosacea, the rash consists of papules and pustules on a background of erythema with telangiectasias confined to the child's face. Although peripheral involvement of the back, upper chest, and scalp may be seen in adults, these areas seem to be spared in the pediatric population.¹²

The third, or late, stage of rosacea involves coarse skin, inflammatory nodules, or gross enlargement of facial features.¹¹ Such chronic changes do not occur in children as they do in adults, presumably because the disease process takes more time to evolve.¹³

Eye involvement can occur in children.¹⁴ It may include manifestations such as blepharoconjunctivitis, episcleritis, keratitis, meibomianitis, chalazia, hordeola, and hyperemic conjunctivae.^{15,16} Although any of these eye conditions can potentially occur in children, meibomian gland inflammation and keratitis are the common findings noted.¹⁷ Peripheral vascularization followed by subepithelial infiltrates can lead to scarring or perforation in the lower two thirds of the cornea. The disease may be unilateral, but most commonly it affects both eyes.¹⁷

Steroid-induced rosacea also has been termed *iatrosacea* because of its mode of acquisition.¹⁸ Topical fluorinated and low-dose corticosteroids can cause a rosacealike dermatitis of the face consisting of persistent erythema with papules, pustules, telangiectasias, and sometimes atrophy.¹⁹⁻²² Corticosteroids may be an exacerbating factor leading to classic rosacea rather than the cause of an independent disease.² The distribution of steroid rosacea to the eyelids and lateral face may help distinguish it from the centrally located typical rosacea.²³ A case of pediatric rosacea associated with the use of topical fluorinated glucocorticosteroids was identified in a 9-month-old boy and 16-year-old girl, both with erythematous patches and papules on the cheeks, paranasal areas, and chin.^{18,23} Forty-six boys and 60 girls, ranging in age of onset from 6 months to 13 years (average, 7 years), were diagnosed with steroid rosacea. Nearly all of the children had perinasal and perioral involvement of erythematous skin interspersed



A rosy tint on the cheeks of a boy at age 2 years (A) and 4 years (B). The child's mother has rosacea.

with papules and/or pustules. The lower eyelids were affected in roughly half of the patients.⁸

Diagnosis

Consistent flushing in children may be a sign of vasomotor instability and early rosacea. These children may blush more frequently and with greater intensity for longer periods than their peers exposed to the same stimuli.²⁴ Thus, blushing in the early stage of rosacea may be an accentuation of the body's normal physiologic response system. A diagnosis of pediatric rosacea beyond the initial stage should be considered when a healthy child has acuminate papules and small pustules of the face, especially if there also exists flushing, telangiectasias, or a family history of rosacea.¹⁴

Differential Diagnosis

The earliest form of rosacea, facial blushing, may be difficult to distinguish from flushing due to other causes. Blushing due to emotions such as embarrassment or anger and to exercise-induced flushing are both appropriate reactions to such stimuli, whereas blushing in the first stage of rosacea may be an exaggeration of this phenomenon.² The main pathway for thermoregulatory and emotional flushing is the cervical sympathetic outflow tract.²⁵ Gustatory blushing, as occurs with consumption of spicy foods, is mediated by autonomic neurons via a branch of the trigeminal nerve.² Sweating often occurs in conjunction with the aforementioned causes of flushing, but it is rarely associated with rosacea flushing.

Thus, sweating may be helpful in reaching a diagnosis; however, exceptions exist.² Frey syndrome (auriculotemporal syndrome), which is characterized by warmth and sweating in the malar region caused by aberrant autonomic fiber connections after damage in the parotid region, may mimic the early stage of rosacea.

The intermediate stage of pediatric rosacea may be confused with other papulopustular disorders such as acne vulgaris, perioral dermatitis, and lupus erythematosus (Table). Careful attention to symptoms, distribution of facial lesions, and potential biopsy results are warranted to distinguish between the conditions. Steroid rosacea and perioral dermatitis may be variants of rosacea or completely separate conditions.^{9,26}

Perioral dermatitis is a rosacealike dermatitis characterized by erythematous papules and pustules usually confined to the perioral region, though the perinasal and periocular areas may be involved.²⁷ A granulomatous perioral dermatitis with tiny, closely spaced, flesh-colored papules in the perioral, perinasal, and periorbital areas was described in children aged 3 to 11 years.²⁸ All cases had spontaneous resolution of symptoms regardless of treatment. The patients did not exhibit flushing or telangiectasias.²⁸

The classic butterfly rash, consisting of erythema and telangiectasia of the malar region and associated with systemic lupus erythematosus, also can be confused with rosacea. Histopathologic examination and direct immunofluorescence of the lesion may help to differentiate lupus from rosacea.¹⁴

Laboratory Diagnosis

There is no specific histologic change unique to rosacea.¹² The most common findings are telangiectasia, edema, elastosis, a variable amount of superficial and deep perivascular lymphohistiocytic inflammatory infiltrate loosely arranged around the hair follicles, and especially architectural disruption of the upper dermis.^{12,28} Depending on the variant of rosacea, there may be an exaggeration of one or more histopathologic signs.¹² For example, granulomatous rosacea may contain collections of granulomas with multinucleated giant cells.²⁸

Treatment

Treatment is gradual and largely determined by the clinical type of rosacea. Children with early or intermediate stages of rosacea are encouraged to avoid their individual local triggers to prevent flares. Topical corticosteroids, especially fluorinated medications, should be discouraged, because even low-potency steroids, including over-the-counter preparations and hydrocortisone 1%, have been shown to cause worsening of the condition.⁸

Traditional therapy for rosacea includes topical and systemic antibiotics, topical metronidazole, and topical retinoids. Oral tetracycline can be used for adolescents in doses similar to those prescribed for adults. It should not be used in children younger than approximately 9 years¹⁷ because it is known to cause dental staining and to be deposited in the skeletal system where it can cause temporary depression in bone growth.^{29,30} Azithromycin or a low dose of doxycycline can be used with good results.^{31,32} For younger children, oral erythromycin is safe and effective to eliminate the erythema, papules, and pustules of rosacea.³² Topical erythromycin and clindamycin have been used with varying results. Azelaic acid and isotretinoin also may be effective.³³ Topical metronidazole 0.75% gel has proven effective in clinical trials.^{34,35} A combination of systemic antibiotics and topical treatment may lead to a substantial reduction in inflammatory lesions, erythema, and the size and diameter of telangiectatic vessels.³²

Eyelid hygiene and erythromycin or bacitracin ointment, to improve meibomian gland function, are appropriate initial treatments for the ocular manifestations of childhood rosacea.^{17,36} A low dose of steroid drops can manage significant irritation when needed. Systemic therapy with tetracycline or other oral pharmaceuticals used to treat the face also may work for ocular symptoms.¹⁷

The treatment of steroid rosacea is a slow process often involving antiacne agents such as benzoyl peroxide and oral or topical antibiotics.²³ Abrupt

Diseases Often Mistaken for Intermediate Stage Rosacea in Children

Acne vulgaris
Granulosis rubra nasi
Haber syndrome
Jessner lymphocytic infiltrate
Papular granuloma annulare
Perioral dermatitis
Polymorphous light eruption
Sarcoid
Seborrheic eczema
Systemic lupus erythematosus
Tinea faciale

discontinuation of topical steroids followed by administration of antibiotics is a suitable treatment option. Prior recommendation has been to taper all topical steroids to prevent rebound flare; however, one study found clearing of symptoms by week 3 in 22% of patients, by week 4 in 86% of patients, and by week 8 in 100% of patients following abrupt cessation of topical steroids and a regimen of oral erythromycin stearate or topical clindamycin phosphate in children with erythromycin allergy or intolerance.⁸ Thus, a gradual withdrawal of topical nonfluorinated steroids may not be necessary. However, it is more common than not for children to experience an initial flare of their condition upon withdrawal from topical fluorinated steroids. This is followed by a slow and steady fading.²³

REFERENCES

1. Buxton PK. Acne and rosacea. *BMJ*. 1988;296:43-45.
2. Greaves MW. Flushing and flushing syndromes, rosacea and perioral dermatitis. In: Champion RH, Burton JL, Burns DA, et al, eds. *Textbook of Dermatology*. Vol 3. Oxford, England: Blackwell Science; 1998:2099-2111.
3. McDonnell JK, Tomecki KJ. Rosacea: an update. *Cleve Clin J Med*. 2000;67:587-590.
4. Zuber TJ. Rosacea. *Prim Care*. 2000;27:309-318.
5. Roihu T, Kariniemi A-L. *Demodex* mites in acne rosacea. *J Cutan Pathol*. 1998;25:550-552.
6. Vin-Christian K, Maurer TA, Berger TG. Acne rosacea as a cutaneous manifestation of HIV infection. *J Am Acad Dermatol*. 1994;30:139-140.

7. Bamford JT. Rosacea: current thoughts on origin. *Semin Cutan Med Surg.* 2001;20:199-206.
8. Weston WL, Morelli JG. Steroid rosacea in prepubertal children. *Arch Pediatr Adolesc Med.* 2000;154:62-64.
9. Weston WL, Morelli JG. Identical twins with perioral dermatitis. *Pediatr Dermatol.* 1998;15:144.
10. Landow K. Unraveling the mystery of rosacea. *Postgrad Med.* 2002;112:51-58.
11. Jansen T, Plewig G. Rosacea. In: DJ Demis, ed. *Clinical Dermatology.* Philadelphia, Pa: Lippincott Williams and Wilkins; 1997:1-11.
12. Marks R, Harcourt-Webster JN. Histopathology of rosacea. *Arch Dermatol.* 1969;100:683-691.
13. Savin JA, Alexander S, Marks R. A rosacea-like eruption of children. *Br J Derm.* 1972;87:425-429.
14. Drolet B, Paller AS. Childhood rosacea. *Pediatr Dermatol.* 1992;9:22-26.
15. Browning DJ, Proia AD. Ocular rosacea. *Surv Ophthalmol.* 1986;31:145-158.
16. Jenkins MS, Brown SI, Lempert SL, et al. Ocular rosacea. *Am J Ophthalmol.* 1979;88:618-622.
17. Erzurum SA, Feder RS, Greenwald MJ. Acne rosacea with keratitis in childhood. *Arch Ophthalmol.* 1993;111:228-230.
18. Litt JZ. Steroid-induced rosacea. *Am Fam Physician.* 1993;48:67-71.
19. Leyden JJ, Thew M, Kligman AM. Steroid rosacea. *Arch Dermatol.* 1974;110:619-622.
20. Hogan DJ, Epstein JD, Lane PR. Perioral dermatitis: an uncommon condition? *Can Med Assoc J.* 1986;134:1025-1028.
21. Hogan DJ, Rooney ME. Facial telangiectasia associated with long-term application of a topical corticosteroid to the scalp. *J Am Acad Dermatol.* 1989;20:1129-1130.
22. Coskey RJ. Perioral dermatitis. *Cutis.* 1984;34:55-56, 58.
23. Franco HL, Weson WL. Steroid rosacea in children. *Pediatrics.* 1979;64:36-38.
24. Wilkin JK. Flushing reactions: consequences and mechanisms. *Ann Intern Med.* 1981;95:468-476.
25. Drummond PD, Lance JW. Facial flushing mediated by the sympathetic nervous system. *Brain.* 1987;110:793-803.
26. Manders SM, Lucky AW. Perioral dermatitis in childhood. *J Am Acad Dermatol.* 1992;27:688-692.
27. Boeck K, Abeck D, Werfel S, et al. Perioral dermatitis in children—clinical presentation, pathogenesis-related factors and response to topical metronidazole. *Dermatology.* 1997;195:235-238.
28. Frieden IJ, Prose NS, Fletcher V, et al. Granulomatous perioral dermatitis in children. *Arch Dermatol.* 1989;125:369-373.
29. Howard R, Tsuchiya A. Adult skin disease in the pediatric patient. *Dermatol Clin.* 1998;16:593-608.
30. Gruber GG, Callen JP. Systemic complications of commonly used dermatologic drugs. *Cutis.* 1978;21:825-829.
31. Caputo R, Barbareschi M, Veraldi S. Azithromycin: a new drug for systemic treatment of inflammatory acneic lesions. *G Ital Dermatol Venereol.* 2003;138:327-331.
32. Bikowski JB. Subantimicrobial dose for acne and rosacea. *SKINmed.* 2003;2:234-245.
33. Szepietowski J. Azelaic acid gel: use in the treatment of rosacea. *Dermatol Klin (Wroclaw).* 2003;3:150.
34. Anonymous. Topical metronidazole for rosacea. *Med Lett Drugs Ther.* 1989;31:75-76.
35. Micali G, Licastro R, Lembo D. Treatment of rosacea with metronidazole gel 0.75%. *G Ital Dermatol Venereol.* 1992;127:247-250.
36. McCully JP, Dougherty JM, Deneau DG. Classification of chronic blepharitis. *Ophthalmology.* 1982;89:1173-1180.

DISCLAIMER

The opinions expressed herein are those of the authors and do not necessarily represent the views of the sponsor or its publisher. Please review complete prescribing information of specific drugs or combination of drugs, including indications, contraindications, warnings, and adverse effects before administering pharmacologic therapy to patients.

FACULTY DISCLOSURE

The Faculty Disclosure Policy of the Albert Einstein College of Medicine requires that faculty participating in a CME activity disclose to the audience any relationship with a pharmaceutical or equipment company that might pose a potential, apparent, or real conflict of interest with regard to their contribution to the activity. Any discussions of unlabeled or investigational use of any commercial product or device not yet approved by the US Food and Drug Administration must be disclosed.