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Clinical Evidence. Practical Advice.

Editor-in-Chief: Dr Stuart Maddin

Dr. Stuart Maddin, MD, FRCPC

EDITOR-IN-CHIEF

Dr. Stuart Maddin, Chairman of SkinCareGuide, is one of North America's leading dermatologists, and is the author of numerous dermatologic journal articles, monographs and textbooks. In addition to providing consultative input to a number of pharmaceutical and biotech companies, he is the director of the clinical trials unit at the Division of Dermatology, University of British Columbia. Dr. Maddin has also acted in an advisory capacity to a number of drug regulatory agencies, such as the Health Protection Branch (Ottawa), the AAD-FDA Liaison Committee, and WHO (Geneva). He is the founder of the Dermatology Update symposia, now in its 21st year. As well, he is Past President of the Canadian Dermatology Association and served as Secretary-General of the International Committee of Dermatology — International League of Dermatological Societies.

**Jane Hailey, MA, MB, BChir, FRCPC**

PEDIATRIC ADVISOR

Jane Hailey is currently in private practice and a Clinical Associate Professor at the University of British Columbia. For the last 14 years, she has worked in a group practice, working with children of all ages – from newborns to adolescents. She is a staff pediatrician at Children's and Women's Hospital in Vancouver, and is an examiner in Pediatrics for the Royal College of Physicians and Surgeons of Canada. She writes regularly for *Pediatric Notes*, a North American weekly newsletter for the pediatric community. She also writes and narrates a regular medical column on CBC RADIO ONE, discussing a wide variety of topics in general medicine - from newborns to geriatrics!



The Bottom Line – Managing Diaper Dermatitis*

**S. Humphrey, MD; J. N. Bergman, MD, FRCPC;
S. Au, MD, FRCPC**

*Department of Dermatology and Skin Science, University of British Columbia,
Vancouver, Canada*

Irritant Diaper Dermatitis (IDD)

Irritant diaper dermatitis (IDD) is a common inflammatory eruption of the skin in the diaper area, seen in 25% of children wearing diapers. [Ward DB, et al. *Arch Pediatr Adolesc Med* 154:943-6 (2000).]

IDD is caused by the presence of moisture, warmth, urine, feces, and friction. It is difficult to completely eradicate these predisposing factors in a diapered child. Thus, IDD presents an ongoing therapeutic challenge for parents, family physicians, pediatricians, and dermatologists.

Pathogenesis

Four key factors contribute to the development of IDD:

Wetness

- Wet diapers result in excess hydration and maceration of the stratum corneum, leading to impaired barrier function, enhanced epidermal penetration by irritants and microbes, and susceptibility to frictional trauma.

Friction

- IDD is most commonly distributed in areas with greatest skin-to-diaper contact.
- Friction disrupts the macerated stratum corneum, exacerbating barrier dysfunction.

Urine and Feces

- The interaction of urine and feces is key to the pathogenesis of IDD. Bacterial ureases in the stool degrade the urea that is found in urine causing ammonia to be released and local pH to increase.
- Fecal lipases and proteases are activated by the increased pH, causing skin irritation and disruption of the epidermal barrier.
- Ammonia does not directly irritate intact skin; it is thought to mediate irritation by contributing to the high local pH.

Microorganisms

- *Candida albicans* and *Staphylococcus aureus* (less common) infections are associated with IDD; the increased humidity and pH in the diaper provide the ideal milieu for microbial proliferation.

Clinical Features

- Localized asymptomatic erythema can progress to widespread painful erythema with maceration, erosions, and frank ulceration.
- IDD commonly spares the skin folds and affects convex skin surfaces in close contact with the diaper, including the buttocks, genitalia, lower abdomen, and upper thighs.
- Severe variants can occur, which include:
 - Jacquet erosive diaper dermatitis: associated with diarrhea, poor hygiene, infrequent diaper changes, and plastic diapers
 - Granuloma gluteale infantum: associated with topical steroid use, candida infection, and plastic diaper covers.

Always consider other conditions that may occur in the diaper area when assessing a patient with possible IDD. See Table 1.

Dermatosis	Clinical Features
IDD	<ul style="list-style-type: none"> • erythema, maceration, erosions, ulcerations • localized to convex skin surfaces in contact with the diaper while sparing the folds
Candidiasis	<ul style="list-style-type: none"> • beefy red plaques with satellite papules and pustules • can affect entire diapered skin and does not spare the folds
Bacterial infection	<ul style="list-style-type: none"> • impetigo: flaccid bullae, superficial erosions, honey-colored crust • folliculitis: erythematous follicular papules and pustules
Granuloma gluteale infantum	<ul style="list-style-type: none"> • asymptomatic erythematous-violaceous papules and nodules
Jacquet erosive diaper dermatitis	<ul style="list-style-type: none"> • punched-out ulcers or erosions with elevated, heaped-up margins
Psoriasis	<ul style="list-style-type: none"> • well-circumscribed, pink-red plaques in diaper area and inguinal folds • silvery scale usually absent
Allergic contact dermatitis	<ul style="list-style-type: none"> • eczematous eruption localized to area of contact with allergen • pruritic
Langerhans cell histiocytosis	<ul style="list-style-type: none"> • erythematous infiltrated papules, pustules, and nonhealing erosions or ulcerations, with foci of hemorrhage, in diaper area • seborrheic dermatitis-like eruption on scalp and postauricular area • systemic involvement including anemia, diarrhea, organomegaly, lymphadenopathy, and bony involvement
Acrodermatitis enteropathica	<ul style="list-style-type: none"> • eczematous eruption may evolve into crusted and eroded vesiculobullous and pustular lesions • acral, periorificial, and anogenital distribution • triad of dermatitis, alopecia and diarrhea presents upon weaning from breast milk

Table 1: Clinical features of diaper dermatoses.

Risk Factors

- Fecal incontinence and diarrhea
 - e.g., Hirschsprung's disease, fecal impaction and overflow, and anogenital malformations.
- Increased bile acids in stool
 - e.g., short-gut syndrome and conjugated hyperbilirubinemia
- Atopic dermatitis
 - increased sensitivity to irritants and susceptibility to secondary infection.

Treatment

Barriers

- Pastes are the most hardy and desirable barriers, followed by ointments.
- Choose pastes with >10% of a fine powder such as zinc oxide or titanium dioxide.
- Creams and lotions are not appropriate barriers as they are poorly adherent, minimally occlusive, and contain preservatives.
- Barrier should be applied thickly to diaper area like "icing on a cake" after each diaper change.
- Cover barrier with petroleum jelly to prevent it from sticking to diaper.
- Avoid barriers with potentially harmful ingredients such as camphor, phenol, boric acid, and salicylates.

Cleansing

- Bathe daily in a lukewarm bath using irritant and fragrance-free soap.
- Gently pat dry with a towel to avoid any undue friction.

Treatment (continued)

- Do not attempt to remove all the adherent barrier paste when wiping off urine/ feces.
- Mineral oil can help remove residual barrier when necessary.
- Unscented and alcohol-free diaper wipes are appropriate for patients with IDD. Sensitive wipes shown to neutralize pH can be helpful.

Diapers

- Maximizing “diaper-free” time is a widely recommended preventative strategy, but not very practical.
- Change diapers as soon as they are wet or soiled (at least every 3–4 hours, more often in neonates).
- Do not use tight-fitting diapers; looser diapers can minimize contact between skin and urine/ feces.
- Cloth diapers are not recommended for patients with IDD; they increase skin wetness, promote mixing of urine and feces, and are associated with Jacquet erosive diaper dermatitis.
- Common IDD should resolve when children become toilet trained.

Disposable Diapers

“Breathable” superabsorbent disposable diapers (e.g., Pampers®, Procter & Gamble; Huggies®, Kimberly-Clark) are the diapers of choice in IDD:

- They contain a polyacrylate polymer core that forms a gel when hydrated and traps moisture away from the skin surface; the gel controls pH by its buffering capacity and by separating urine from feces.
- “Breathable” backsheet (outer cover) prevents excess humidity but still protects against leaks; it feels like cloth rather than plastic, and is readily identifiable in the office.
- Polyacrylate gel core diapers are associated with reduced skin wetness, superior pH control, and less IDD compared with cellulose core disposable and cloth diapers [Campbell RL, et al. *J Am Acad Dermatol* 17:978-87 (1987)].
- They have been shown to reduce the prevalence of severe IDD by up to 50%. [Akin F, et al. *Pediatr Dermatol* 18:282-90 (2000)].
- Most conventional diapers now use the breathable polyacrylate gel core technology.

Corticosteroids

- A short course of a mild topical corticosteroid is frequently necessary in moderate-to-severe IDD.
- Hydrocortisone 1% ointment can be applied to affected areas twice daily for a limited duration.
- Mid-to-high potency corticosteroids should never be used in the diaper area (risk of atrophy, systemic absorption, candidiasis, granuloma gluteale infantum).
- Avoid combination antifungal-corticosteroid products that often contain mid-to-high potency steroids.

Infection

- *Candida* infection is often associated with moderate-to-severe cases of IDD and presents with beefy red erythema and satellite papules and pustules; the skin folds may be involved.
- The azoles, nystatin, and ciclopirox are all appropriate topical anticandidal agents; twice-daily application is recommended until resolution.
- Mupirocin is also effective in the treatment of IDD superinfected with *Candida*.

Remember the ABCDEs of Diaper Dermatitis

Antimicrobial/Anti-inflammatory
Barriers
Cleansing
Diaper
Education

Conclusion

IDD is a common dermatosis afflicting diapered children. It is caused by wetness, friction, urine, stool, and microorganisms. A proactive approach targeting predisposing factors is the best defense against IDD.

*Adapted from Humphrey S, Bergman JN, Au S. Practical management strategies for diaper dermatitis. *Skin Therapy Lett* 11(7):1-4 (2006 Sep). [Can be reviewed online at www.SkinTherapyLetter.com.]

Treatment of Head Lice*

C. H. Hong, MD, FRCPC

Department of Dermatology and Skin Science, University of British Columbia, and
Skinfit MD Clinic, Vancouver, Canada

Head Lice

An estimated 1 in 10 children in Canada will be affected by a lice infestation this year and it is more common worldwide than the common cold. Head lice infestations, i.e., *Pediculus humanus capitis*, are the most common type, which can occur worldwide and affect people of all ages and socioeconomic groups. Lice are wingless, bloodsucking insects. They are difficult to see because they are about the size of a sesame seed and they adapt to take on the color of their surroundings.

- Occurs most commonly in school-aged children, especially girls between 3 and 11 years of age.
- Is often epidemic. Most common places for outbreaks include schools, day cares, and play groups.
- Personal hygiene and socioeconomic status are not related to the likelihood of developing lice infestation.
- Not known to transmit bloodborne diseases.
- Spread through close head-to-head contact or through fomites (inanimate objects) that come into contact with the infested scalp and then are shared (e.g., combs, brushes, and hats).

Clinical Manifestations and Diagnosis

Head lice are typically confined to the scalp, and while itching is the main symptom of lice infestation, the lice themselves are not itchy and the bites are generally painless. Itching is caused by the body's immune reaction to the saliva injected into the skin at the time of the blood meal. Redness and scaling may also be seen in the scalp as can small, itchy bumps on the posterior neck. Enlargement of lymph glands in the neck can be occasionally seen.

Diagnosis can be made ONLY by identification of a living louse. Nits or eggs are often empty shells and not an indication of an active infestation. Nits are also often mistaken for dandruff, sand or dirt, or dried hair gel.

Myths and Facts

Myths about head lice are abundant and belief in these myths is often why treatments are not used properly and why people believe their lice treatment has failed. Some common myths include:

- **Myth:** All children with lice scratch or itch.
Fact: Initial infestation may produce no signs or symptoms for 4–6 weeks; only 1 in 3 children will complain of itching.
- **Myth:** Lice jump or fly from head to head.
Fact: Lice can be dislodged from hair by air movements giving the appearance of flying.
- **Myth:** Lice live in carpets, beds, clothes, and sofas.
Fact: Lice can only live for 24 hours away from a human host.
- **Myth:** Lice die immediately after treatment.
Fact: Lice may take several hours to die following treatment.
- **Myth:** One treatment is enough.
Fact: Due to loss of residual activity of pediculicides, two treatments, 7 days apart, are recommended to kill newly hatched nymphs.
- **Myth:** Permethrin based products are 100% ovicidal.
Fact: Permethrin kills 70% of eggs with one treatment.
- **Myth:** Everyone in the family should be treated.
Fact: Only those with a proven infestation should be treated, although everyone should be checked daily to weekly.
- **Myth:** Head lice prefer long or dirty hair.
Fact: Lice do not care about hair length or cleanliness.

Treatment Options

There are many proposed “natural” and alternative cures for treating head lice including various oils (e.g., tea tree oil), petroleum jelly, peanut butter, and mayonnaise; however, the effectiveness of most are dubious. There are no clinical studies to demonstrate effectiveness of these agents and many are messy and difficult to use.

Most patients will need to be treated with a commercial product designed to kill lice. Most of these are available over-the-counter and work by attacking the central nervous system of the louse. No pediculicide is 100% ovicidal. In order to achieve maximum effectiveness, the pediculicide needs to be applied to dry or nearly dry hair. It should be saturated to allow sufficient pediculicide to penetrate lice and nits and left on for the entire recommended time.

- Permethrin 1% cream rinse (Nix®/Kwellada-P®)
 - Wash hair with conditioner-free shampoo and towel dry until hair is almost dry; then apply at least 25ml, and up to 50ml for thick or long hair, to hair and scalp especially behind the ears and the nape of the neck.
 - Leave product on for 10 minutes then rinse with cool water over a sink (not in a shower or bath).
 - Comb wet hair with nit comb to remove any dying lice and nits.
 - A second treatment in 7 days should be carried out for maximum efficacy.
 - There is minimal systemic absorption and low risk for toxicity.
 - It is contraindicated in patients with chrysanthemum allergy.
- Synergized pyrethrins (R & C® shampoo)
 - Apply to dry hair and scalp, especially behind the ears and the nape of the neck. Hair must be completely dry.
 - Leave on 10 minutes, then add water to form lather. Rinse with cool water over a sink. Do not use conditioner.
 - A second treatment in 7 days should be carried out for maximum efficacy.
 - There is low risk of toxicity.
 - Do not use if known allergy/sensitivity to chrysanthemum or ragweed.
- Lindane shampoo 1%
 - It is inexpensive.
 - Apply to dry hair, leave on for 5 minutes and rinse with cool water over a sink (not in a shower or bath).
 - Comb wet hair with nit comb to remove any dying lice and nits.
 - Concern re: neurotoxicity with high dose or repeated exposure
 - Contraindicated in neonates, young children (<2 years of age), pregnant women, and nursing mothers or those with a history of seizures.
 - Resistance has been seen for more than 2 decades.

Regardless of the type of treatment recommended, patients should be encouraged to use a nit comb (i.e., a fine toothed metal comb such as the LiceMeister® comb), on wet hair to manually remove the nits, which can become cemented to the hair shafts. Treated patients should also be monitored for reinfestation.

Fomite Control

Following treatment, all clothing should be machine laundered and dried in the dryer (hot cycle). In addition, bed linens, towels, stuffed animals, and any headgear should be cleaned. Clothing that cannot be washed can be dry cleaned or sealed in a bag for 2 weeks. Combs and brushes can be covered in the pediculicide and then washed in hot water for 20 minutes; alternatively they can be soaked in a disinfectant solution (e.g., 2% Lysol® for 1 hour). All interior areas should also be cleaned and vacuumed to remove any shed hairs.

Reasons for Failure of Treatment

- Wrong diagnosis
- Poor adherence / improper use of chemical lice treatment (e.g., applied to wet hair, insufficient product applied)
- Inadequate time to evaluate treatment (lice do not die on contact with product)
- Poor manual removal of nits
- New exposure to lice (re-infestation)
- Not repeating treatment 1 week later

Conclusion

Head lice are a common and embarrassing problem with many good treatments. Patients should be appropriately counseled to minimize spread of infestation and then advised on appropriate topical treatments. For further information about lice and lice eradication as well as counseling tips and patient material go to www.SkinPharmacies.ca/CE.

*This article was adapted from Hong CH. Treatment of Head Lice. *Skin Therapy Letter – Pharmacist Edition* 1(2):4-5 (2006 Sep-Oct). [Can be reviewed online at www.SkinTherapyLetter.com.]

Common Bacterial Skin Infections

M. H. Lupin, BSc, MD, FRCPC

Department of Dermatology and Skin Science, University of British Columbia, Vancouver, Canada

The skin has a complex flora with the number of microorganisms far outnumbering the number of human cells, and infections can result when there is a breakdown in the integrity of the skin, or our immune defense is compromised.

Classification

	Discrete Lesions	Diffuse Lesions
Superficial Infections	impetigo, folliculitis	erysipelas
Deep Infections	ecthyma, furuncles, carbuncles, abscess, paronychia	cellulitis, necrotizing fasciitis (rare)

There may be a continuum of these various infections in any one individual. Cultures and sensitivities are recommended for suspected skin infections especially with the increase of community acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA). These infections can affect healthy children and can be potentially life threatening.

Most Common Pathogens	Impetigo/Ecthyma		Folliculitis/ Abscess	Erysipelas/Cellulitis	
	Nonbullous	Bullous		Face	Extremities
<i>Staphylococcus aureus</i> (<i>S. aureus</i>)	+	+	+	+	
<i>Streptococcus pyogenes</i> (<i>S. pyogenes</i>)	+			+	+

Types

Impetigo and Ecthyma

- Typically honey-colored crust with erythematous vesicles, papules, pustules or erosions; common area around nose and face
- More common in atopic dermatitis
- Ecthyma is a deeper version of impetigo, more commonly seen in patients with malnutrition and/or poor hygiene - vesicles and bullae, deep ulcers; legs are the most common and healing leaves scars.
- Consider insect bites, eczema, herpes, and candida and thermal burns in the differential diagnosis.
- Swab for culture and sensitivity.
- TREATMENT
 - Nonbullous: fusidic acid (Fucidin® 2% Cream) t.i.d. or mupirocin (Bactroban® Cream) t.i.d.
 - Bullous or extensive or multiple lesions: fever or constitutional symptoms, + nodes, immunocompromised, valvular heart disease: cloxacillin (Cloxapen®) 40–50mg/kg/d divided (div) q.i.d., or cephalexin (Keflex®) 40mg/kg/d div q.i.d., or erythromycin 40mg/kg/d div q.i.d.

Folliculitis, Furunculosis, and Carbunculosis (Folliculitis Group)

- A spectrum of infections involving the hair follicles.
- Often characterized by asymptomatic or mildly pruritic red follicular based papules and pustules.
- Hot tub folliculitis is less common and due to *Pseudomonas aeruginosa*. It usually clears spontaneously and is commonly distributed over the trunk, buttocks, and thighs.
- Furunculosis is deeper, presenting with tender, erythematous nodules and suppurative drainage
- Carbuncles are a coalescence of furuncles presenting as larger, tender, fluctuant, draining nodules.
- Swab for culture and sensitivity.
- TREATMENT
 - Warm compresses for 10–15 minutes q.i.d. may give relief.
 - Bacterial folliculitis and bacterial paronychia
 - Fusidic acid 2% cream, t.i.d.
 - Cloxacillin 40–50mg/kg/d div q.i.d., or cephalexin 40mg/kg/d div q.i.d., erythromycin 40mg/kg/d div q.i.d. All would be used for 1 week.
 - Recurrent Furunculosis – Consider *Staphylococcus* nasal carriage;
 - Mupirocin 2% cream b.i.d. intranasally for 5 days. MRSA resistance is increasing in Canada and fusidic acid unguent could be considered in those cases.

Types (continued)

- Carbuncles
 - Cloxacillin 40–50mg/kg/d div q.i.d. or cephalexin 40mg/kg/d div q.i.d.
 - MRSA or penicillin allergy: clindamycin 20mg/kg/d po q.i.d. or trimethoprim/sulfa 6–12mg/kg/d po div b.i.d. for 1 week.

Abscess

- Fluctuant cystic nodule, may have a pointing pustule, usually caused by *S. aureus*.
- Incision and Drainage (I&D) is very important, as well as taking cultures and sensitivities.
- If there is a cellulitic component greater than 5cm, if abscess cannot easily be drained, if location is on face, or if there are systemic symptoms (fever, chills), add a systemic antibiotic.
- Suspect community acquired MRSA.
- TREATMENT
 - I&D; warm compresses for 10–15 minutes q.i.d. may give relief.
 - Fusidic acid 2% cream, t.i.d., is indicated and is useful especially in smaller lesions alone or most commonly in combination.
 - Cloxacillin 40–50mg/kg/d div q.i.d., or cephalexin 40mg/kg/d div q.i.d., or erythromycin 40mg/kg/d div q.i.d. for 1 week.
 - MRSA or penicillin allergy: clindamycin 20mg/kg/d po q.i.d. or trimethoprim/sulfa 6–12mg/kg/d po div b.i.d. for 1 week.

Erysipelas and Cellulitis

- Erysipelas is a superficial infection with a predilection for young children and the elderly.
 - Abrupt onset of tender, well-defined erythematous, indurated plaques most commonly on the face or legs. Involvement of the dermis and often the lymph nodes, usually *S. pyogenes*.
- Cellulitis is a deeper process extending to the subcutis. In children the head and neck is usually involved; caused by *S. aureus*, and less commonly, *H. influenzae*
- Strep perianal disease is usually seen in those under 4 years old. Well-defined redness extends up to 2–3cm away from the anus. Pharyngitis may precede it. Local discomfort/itching can be present.
- Pain inhibiting defecation may lead to fecal retention and overflow soiling or blood stains on the underwear. Psoriasis or candida should be considered as alternatives.
- Orbital, umbilical, or neonatal cellulitis will not be considered.
- TREATMENT
 - Face: Cephalexin 40mg/kg/d po div q.i.d. for 10–14 days will usually be adequate for *S. aureus*, *H. influenzae*. Those under the age of 5 years could be treated with cefuroxime 100–150mg/kg/d q8h for 10–14 days. For those over 5, cefazolin 75mg/kg/d IV div q8h for 10–14 days. Clindamycin for those with β -lactam allergy.
 - Perianal cellulitis: this can be polymicrobial. Amoxicillin-clavulanate 40mg/kg/d po for 10–14 days for mild cases. Clindamycin plus gentamicin or cefazolin plus metronidazole can be used depending on the culture results.

[Blondel-Hill E, Fryters S, Editors. *Bugs & Drugs*. Edmonton: Capital Health (2000).]

Bacterial Resistance

- Due to increased bacterial resistance to drugs in general, always obtain culture and sensitivity.
- In 2005, a five-fold increase in MRSA was noted in Canada and the US.
- A recent presentation at the Canadian Association for Clinical Microbiology and Infectious Disease conference reported high (96%) MRSA sensitivity to topical fusidic acid.
- The Centre for Disease Control recommended that MRSA be a reportable disease.
- CA-MRSA is usually resistant *in vitro* to β -lactams (penicillin and cephalosporins) as well as macrolides/azalides (erythromycin, clarithromycin, azithromycin).
- CA-MRSA can be sensitive to TMP/SMX (trimethoprim/sulfamethoxazole), tetracyclines (e.g., doxycycline (Doryx®), minocycline (Minocin®)) and clindamycin (Cleocin®) though resistance can occur.
- Hospital acquired MRSA (HA-MRSA) is usually resistant *in vitro* to multiple classes of antibiotics.
- Canadian Committee on Antibiotic Resistance, www.ccar-ccra.com, is a good reference for up-to-date information on resistance patterns.

PEARLS

Nasal carriage of *Staphylococcus aureus* is approximately 20%–30%, so it is important to treat the nose if there is frequent recurrence of infection or there is an outbreak in close family members. Mupirocin cream q.h.s. for 6 weeks is helpful. Fusidic acid 2% cream is also effective. Rifampin can be added if topicals are insufficient. Routine treatment of the nose is not recommended.

Conclusion

Selecting the right therapy from the beginning should help minimize complications, reduce the number of hospitalizations, and may also help reduce the climbing incidence of bacterial resistance.

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