Diaper Dermatitis: Appropriate Evaluation & Optimal Management Strategies
Irritant diaper dermatitis (IDD) is a localized form of contact dermatitis caused by several factors, including warmth, chafing, prolonged contact with urine and feces, and overhydration of the upper portion of the skin (stratum corneum). Many experts believe that the presence of Candida yeast infection also plays a primary, as well as a secondary, role in the development of this frequently troublesome and sometimes painful eruption. IDD is so common that infants in the United States have a 1 in 4 chance of being diagnosed with this condition. In a recent UK study that included the parents of 532 hospitalized children in diapers, 52% of families reported a history of diaper dermatitis (DD), and multivariate analysis demonstrated the risk of DD to be associated with oral thrush, past history of disease, frequency of diaper changes, and diarrhea. There were 4.8 million outpatient visits for DD from 1990 to 1997; pediatrician visits accounted for 75% of these, and the remaining 25% of visits involved family physicians, internists, dermatologists, and other specialists. IDD is a therapeutic challenge for both parents and physicians because of its frequency and the difficulty in eliminating all of the causative factors in diapered infants.

IDD is generally episodic and relatively self-limiting. It peaks in frequency at 9 to 12 months of age. However, moderate-to-severe IDD can be more problematic, and is frequently associated with Candida albicans. Although estimates of candidiasis-associated DD vary, early studies reported recovery of the organism in 41% to 80% of infants with IDD, and a positive correlation...
has been demonstrated between the clinical severity of IDD and the presence and level of C albicans in the diaper, mouth, and anus of infants. 

This panel agrees that IDD often can be managed effectively in its early stages by keeping the diaper area dry and the stratum corneum intact. This can be accomplished with frequent diaper changes, gentle cleansing, and use of barrier protection. In some cases, mild topical corticosteroids, such as hydrocortisone 1%, may be useful. If IDD worsens or persists for more than 3 days, candidiasis-associated DD should be strongly considered as a diagnosis. Candidiasis-associated DD can be severe, and it is important that the clinician recognize its associated signs and symptoms so that the rash can be treated appropriately with an antifungal agent.

Practitioners should be aware that other less common, but potentially more serious, conditions can also “masquerade” as DD, including metabolic and nutritional disorders, malignancies, and a broad set of primary cutaneous disorders, such as psoriasis. These conditions should be considered as diagnoses, particularly if a patient does not respond to appropriate therapy.

Prescribing patterns for moderate-to-severe DD demonstrate that topical antifungal agents, mid- to high-potency steroids, and combinations of both are often prescribed despite the fact that higher potency steroids may not be the best choice for use on the delicate, generally occluded diaper area of infants. 

Because of high rates of candidal colonization and infection associated with DD, patients may benefit from the use of a topical antifungal agent in addition to the traditional approaches for the treatment of persistent DD (appropriate cleansing and the use of barrier agents). A topical antifungal agent such as miconazole can be used for this purpose in conjunction with barrier products to protect the skin. A combination barrier antifungal ointment containing miconazole nitrate 0.25%, zinc oxide 15%, and white petrolatum 81.35% received FDA approval in February 2006 for the treatment of candidiasis-associated DD for pediatric patients aged ≥4 weeks. It is an alternative agent that provides a consolidated and simplified therapy for patients diagnosed with DD where secondary Candida involvement is likely. In severe cases of candidiasis-associated DD that is unresponsive to topical agents, oral agents such as nystatin may be useful in minimizing Candida species in the gastrointestinal (GI) tract, the primary reservoir of Candida spp.

**FACTORS INVOLVED IN THE DEVELOPMENT OF DIAPER DERMATITIS**

Diaper dermatitis arises from the interaction of several factors, the most important of which is prolonged contact of the skin with a mixture of urine and feces. The combination of diaper occlusion, fecal enzyme activity, urine, and diaper chafing leads to overhydration of the stratum corneum and chemical and mechanical abrasion, which compromises barrier function and makes the stratum corneum more susceptible to frictional trauma and the penetration of irritants and microbes. Hydrated skin is more likely to suffer frictional damage than dry skin, and frictional alterations in skin barrier function induced by overhydration can then subsequently change the balance of resident flora and promote growth and likely the invasiveness of C albicans, which is found in as many as 70% of infants with DD.

**Role of Urine And Feces**

Although urinary ammonia was traditionally thought to be an important etiologic factor in the development of IDD, a small study found no difference in the mean level of ammonia in the morning diaper obtained from 26 infants with IDD compared with the mean level obtained from 82 control patients. Furthermore, the experimental application of highly ammoniacal urine on intact infant and adult skin did not
elicit a dermatologic reaction. Erythema could be induced when ammoniacal urine was applied to skin that had been scratched sufficiently enough to compromise the skin barrier. In addition to the capacity of urine to irritate damaged skin, its most important role in the development of IDD is its proclivity for skin hydration. Thus, urinary ammonia on intact skin without secondary contamination does not appear to be a primary etiologic factor in IDD. However, the interaction of urine and feces is fundamental in the development of IDD. Berg and colleagues demonstrated that bacterial ureases in the stool degrade urea in urine, which releases ammonia; however, ammonia does not actually irritate the skin, but rather mediates irritation by increasing local pH. Increased pH reactivates fecal enzymes such as lipase and protease, which irritate the skin and disrupt the epidermal barrier. It has also been demonstrated that the incidence and severity of IDD is significantly higher in infants with a 48-hour history of diarrhea. While it has been suggested that this higher rate of IDD could be due to increased exposure of the skin to irritation from fecal enzymes, it may be possible that additional moisture present in diarrhea versus normal stool could contribute to the increased incidence and severity of IDD in patients with loose stools. Supporting this theory is the demonstration that skin wetness strongly correlates with both the risk and severity of IDD. It is feasible that the excess moisture that accompanies diarrhea hydrates the skin, and when occluded, this increased hydration causes the skin barrier to further deteriorate. Thus, irritation may be enhanced by excess moisture in diarrheal stool, as well as the increased amount of fecal proteases and lipases in contact with the diaper area during bouts of diarrhea.

CANDIDIASIS-ASSOCIATED DIAPER DERMATITIS

The cutaneous bacterial flora of the diaper area are abundant, regardless of the underlying condition of the skin. Various organisms, including Staphylococcus aureus, Escherichia coli, and Proteus, have been recovered from the skin of infants with IDD, although these organisms are not considered causative. Calbicans, in contrast, has been widely implicated in moderate-to-severe DD.

Candida is a genus of yeast-like fungi that is often part of the flora of the skin, mouth, and GI tract of infants. Excessive growth of Candida appears to increase the likelihood of invasive disease. Colonization by Candida spp appears to be significantly more frequent in children with IDD than in healthy controls. In a study by Ferrazzini et al, researchers compared the rates of colonization at 3 body sites in children with IDD and healthy controls. The results demonstrated greater rates of colonization at all sites among the affected children versus the healthy controls: perianal, 75% vs 19%; inguinal, 50% vs 10%; and oral, 68% vs 25%. The same study also demonstrated a highly significant, positive correlation between the extent of Candida spp colonization.
Primary and Secondary Candida Infection

The observation of relatively low levels of *C. albicans* at some rash sites underscores the challenge in determining whether *Candida* is a causative factor in candidiasis-associated DD or if it is a secondary contaminant that flourishes in the moist, warm environment of the diaper area and then capitalizes on the disrupted skin barrier of the diaper environment.

In support of the latter statement that *Candida* is a secondary contaminant, one study suggests that if sufficient moisture is present, adequate nutrients for the growth of *C. albicans* exist on human skin. In another study, *Candida* spp were identified in the diaper area of children who had healthy skin, which demonstrated that this area can be populated by *Candida* spp to a small degree without causing symptoms. In a study that used an experimental human infection model of erosio interdigitalis blastomycetica, it was demonstrated that recovery of *C. albicans* decreased once the organism established itself on the skin and intense inflammation developed. C. albicans appears to be the initial contaminant, but infection was attributed to synergism of the organism with gram-negative rods, a synergy that might reproduce itself similarly with resident flora in candidiasis-associated DD. Once inflammation was present, the recovery of the organism decreased. Other observations support a primary role for *Candida* in the development of DD. It has been demonstrated that *Candida* does not require clinically inflamed skin to proliferate and cause infection. *C. albicans* infection requires only moist occlusion on normal skin to grow. However, without moisture and occlusion, *C. albicans* cannot compete against resident microflora.

These studies illustrate that in a moist environment, *Candida* can colonize multiple areas on its own—even in a highly functional stratum corneum—and suggest that failure to recover the organism does not preclude the possibility of prior *C. albicans* involvement. Although it is likely that an impaired stratum corneum is vulnerable to secondary colonization, a compromised skin barrier does not appear to be a prerequisite for colonization. It would seem that failure to consistently recover *C. albicans* in clinically infected skin has been emphasized in the literature too heavily, and the colonization characteristics of *C. albicans* too seldom. The data provide supportive evidence for both the primary and secondary contamination of *C. albicans* in the diaper area.

Candida and the Gastrointestinal Tract

The consistently higher rate of recovery of *C. albicans* from the GI tract relative to the rash site supports the GI tract as a reservoir for *C. albicans*, with infection resulting from the spread of the organism onto the diaper area. There is a strong correlation between the level of *C. albicans* in the feces and disease severity. In a study assessing 30 infants with candidiasis-associated DD, the authors recovered *C. albicans* from satellite pustules in all 30 infants, from the central area of erythema in 66% of patients, and from the rectum in 93% of infants.

Candida and Antibiotic Use

Clinical situations promoting *C. albicans* growth in the GI tract presumably increase the risk of candidiasis-associated DD. In a study quantifying *C. albicans* colonization before and after antibiotic therapy, a 10-day course of systemic antibiotics administered to infants with otitis media who were otherwise healthy was associated with a 2-fold increase in *C. albicans* recovery from the rectum and skin. Furthermore, the infants who developed IDD had significantly greater numbers of *C. albicans* organisms recovered from these sites. The presence of thrush also is an important diagnostic clue in the evaluation of an infant with DD. Thrush is highly correlated with GI colonization, and it has been demonstrated that 52% of infants with oral candidiasis develop candidiasis-associated DD.

These studies demonstrate that candidiasis-associated DD is linked to predisposing factors including diarrhea, antibiotic exposure, and/or the presence of thrush, but can also present as primary disease. Candidiasis should be suspected in any case of IDD that lasts more than 3 days. Potassium hydroxide (KOH) preparation and fungal culture should be collected from satellite lesions/satellite papules and pustules rather than the central area of rash, and though recovery of *C. albicans* from the skin can be challenging, the GI tract usually manifests heavy growth. In the case of persistent dermatitis with a low yield from KOH and culture, the best chance of recovering *C. albicans* is with a rectal swab. It is important that candidiasis-associated DD is treated immediately; clinicians should use their judgment in the case of a negative KOH and con-
sider treating with an antifungal agent if candidiasis is suspected clinically.

**DIFFERENTIAL DIAGNOSIS OF DIAPER DERMATOSIS**

The diagnosis of diaper dermatoses is usually straightforward, but occasionally rarer, more serious disorders can “masquerade” as DD. Therefore, it is important to always consider other diagnoses, particularly if a patient does not respond to appropriate therapy for DD (Figure 1). Table 1 provides a comprehensive list of differential diagnoses, which are discussed in more detail below.

**IDD, the most common form of DD, presents with confluent erythema and papules (Figure 1).** Severe cases may also exhibit areas of erosion. This dermatitis is usually localized to convex areas in contact with the diaper, including the buttocks, medial thighs, mons pubis, scrotum, and labia majora. IDD is often accompanied by perianal erythema, but the most distinctive feature of IDD is sparing of the skin folds (Figure 2).

Tidewater-mark, Lucky Luke, and diaper-dye dermatoses are variants of contact dermatitis, and each has recognizable patterns to differentiate them from uncomplicated IDD.

Diaper-dye dermatitis can be distinguished by its well-demarcated erythematous papules at the colored edges of the diaper. There may be crossover between this and tidewater-mark dermatitis, which presents as band-like erythema on the thighs and abdomen at the diaper edges, sparing the skin folds. Lucky Luke DD also has an easily recognizable pattern that resembles a cowboy’s gunbelt holster and is located on the outer buttocks and hips (Figure 3a); it develops in response to rubber components in the diaper. Because of the potential for contact dermatitis, parents should be cautioned to avoid products with additives that are known to cause a reaction in their child.

Both Jacquet’s dermatitis and granuloma gluteale
infantum are severe variants of IDD. Jacquet’s dermatitis presents with easily recognizable, punched-out ulcered papules and is usually associated with infrequent diaper changes. However, it has become increasingly rare with the advent of super-absorbent diapers. Granuloma gluteale infantum can be distinguished from uncomplicated IDD by its asymptomatic violaceous papules and nodules on prominent areas of the groin, abdomen, genitalia, and thighs. Potential risk factors include a history of topical corticosteroid use, Candida infection, or use of occlusive plastic diaper covers.

Candidiasis-associated DD consists of bright red, confluent areas of involvement with irregular, scaly border and papulopustular satellite lesions. The intertriginous areas are involved, which distinguishes it from uncomplicated IDD. Figures 3b and 3c illustrate presentations of candidiasis-associated DD. Candidiasis-associated DD is also differentiated from IDD by a rash duration >3 days (Figure 1). Many patients may have a history of recent diarrhea, concomitant oral thrush, or antibiotic exposure.

Seborrheic dermatitis presents as salmon-colored, well-demarcated scaling plaques, especially at the inguinal folds. It can also be associated with candidiasis-associated DD, but it is often distinguished by the presence of greasy, scaling lesions elsewhere on the body, especially on the scalp, face, postauricular folds, neck, and axilla. Seborrheic dermatitis usually appears by 3 to 4 weeks of age and often resolves spontaneously by 3 to 4 months of age.

Intertrigo presents as moist and sharply demarcated erythema at the skin folds with minimal scale and can be distinguished from candidal infection by a lack of satellite lesions. Intertrigo is a superficial inflammatory process on skin surfaces in close opposition with one another arising from a combination of moisture, heat, sweat retention, and friction.
Secondary streptococcal infection can develop in the intertriginous folds of the diaper area (Figure 4), as well as the neck and axillae. This infection is associated with a bright red and moist appearance with sharply demarcated borders. Bullous impetigo (Figure 5) can also develop in the diaper area, and may occasionally be mistaken for candidiasis-associated DD. *S. aureus* folliculitis superimposed on IDD (Figure 6) is another bacterial infection that can involve the diaper area, and must be recognized so that appropriate antibiotic therapy can be instituted. This disorder can sometimes be confused with the more common candidiasis-associated dermatitis, but KOH and culture evaluation will distinguish between the two. Severe deep-seated bacterial infections, including furunculosis, and severe life-threatening infections such as Fournier’s gangrene can develop if the bacterial etiology of the skin findings is not recognized and treated appropriately.²⁰

Atopic dermatitis presents as mild erythema and scaling, and can occasionally involve weeping crusted lesions.⁸ Atopic dermatitis is usually associated with concurrent or previous flares of rash on the face and extensor limbs, and though it usually spares the diaper area, it can sometimes resemble IDD or seborrheic dermatitis.⁸ Diagnosis is often based on a positive personal or family history of allergic rhinitis, hay fever, or asthma. In the differential diagnosis, concurrent or previous rash elsewhere on the body is its primary feature.⁸

Unfortunately, other more serious disorders can sometimes initially be mistaken for DD. Langerhans’ cell histiocytosis (LCH) is a rare and potentially life-threatening condition caused by bone marrow-derived cells that are dendritic in nature and can ac-
genital zinc defects. Acrodermatitis enteropathica (Figure 9) is a congenital disorder in which there is a defect in zinc absorption from the gut. Breast milk zinc deficiency can occur when maternal breast milk does not have sufficient zinc. Affected children quickly recover from the latter disease when supplements are provided. In contrast, children with classic acrodermatitis enteropathica may require significant zinc supplementation for life. In these disorders, extensive refractory disease usually develops and involves the perioral, genital, and acral areas. Erythema and peeling of the skin on the hands and feet, as well as paronychial changes of the nail may occur.

One distinct subset of DD may occasionally be a harbinger of future psoriatic disease. Napkin psoriasis presents as sharply demarcated erythematous plaques, especially at the inguinal folds and on the scrotum or convex areas. There is scaling, but this can be difficult to discern due to the moisture of the area. The initial lesion usually develops in the intertriginous groin area, followed by a secondary dermatitis that resembles psoriasis. Involvement of the umbilical area is a clue to this diagnosis. Napkin psoriasis is due to Koebnerization, and may resolve once the infant is toilet trained; however, a strong correlation between the appearance of psoriasis in infancy and later in life has been demonstrated.

Distinguishing one form of DD from another can be challenging, but an accurate diagnosis can be made if the clinician concentrates on the location and morphology of the rash, particularly regarding involvement or lack of involvement of the skin folds or other sites on the body such as the scalp and umbilicus. The presence of papules, purpura, or petechiae is an important clue, as is the response to traditional barrier therapy with antifungal therapy added as appropriate. If a patient does not improve with such measures, less common but potentially more serious disorders should be considered.

MANAGEMENT OF IDD
The treatment of uncomplicated IDD is outlined in Figure 1. Educating caregivers about an optimal diapering routine is crucial to successful treatment. Extensive clinical practice has long supported a stepwise approach to the treatment of DD that includes a combination of frequent diaper changes, gentle cleansing, barrier protection, and antifungal/anti-inflammatory treatment (if the eruption lasts more than a few days) (Figure 1). First, it is important to emphasize prevention, and the most effective prevention is to ensure that the infant’s skin stays clean and dry.

FIGURE 8. Langerhans’ Cell Histiocytosis. Photograph courtesy of Victoria Barrio, MD

FIGURE 9. Zinc Deficiency. (Note severe confluent erosions and scale.) This child did not respond to routine diaper dermatitis therapy, and developed periorificial and acral (fingers and toes) lesions. Photograph courtesy of Lawrence F. Eichenfield, MD

Improved Diaper Technology
It has been estimated that disposable diapers account for more than 90% of diapers used in developed nations. Disposable diaper technology has improved significantly over the last few decades and continues to evolve. Disposable diapers that contain superabsorbent gelling materials are associated with a reduced incidence and decreased severity of IDD. Although studies have reached various conclusions regarding the benefits of disposable diapers over cloth diapers, it is intuitive that diapers with the capacity for maintaining normal skin pH and hydration are important in the prevention and treatment of DD. One study demonstrated that infants diapered exclusively in disposable diapers developed less rash than those diapered exclusively or occa-
sionally in cloth diapers. Further innovations include a disposable diaper that provides continuous topical administration of a zinc oxide/petrolatum formulation. Use of this diaper is associated with improvements in erythema and diaper rash in patients with mild-to-moderate IDD. A helpful component in the treatment of moderate-to-severe candidiasis-associated DD is ensuring the use of a breathable diaper. Highly breathable diapers have been associated with a 38% to 50% reduction in severe IDD cases among infant patients, including those with confirmed candidiasis. In a study involving adult volunteers, it was demonstrated that when C albicans cells were occluded with a patch from either a highly breathable diaper or a standard diaper on the volar forearm, Candida colonization was observed nearly two-thirds less in the highly breathable diaper-covered sites compared to the control sites.

**Cleansing**

Excessive washing of the diaper area should be avoided as a preventive and treatment approach for DD. Once the diaper is soiled, the feces should be removed from the skin as soon as possible with water and a soft cloth. If a barrier preparation has been used, the caregiver should focus on removing the stool from the barrier product rather than completely removing the barrier. Then an additional layer of cream or ointment should be applied to the remaining barrier preparation. Parents who work to remove all the ointment often induce more trauma and irritation at the site. The use of baby wipes minimizes the risk of frictional injury. Baby wipes that contain alcohol should be avoided because some types of alcohol can be drying; however, most baby wipes are alcohol-free and contain 98% water. pH-buffered baby wipes and products formulated for sensitive skin have minimized the likelihood of irritancy from these products.

If the caregiver needs to wash the diaper area more thoroughly, a nonirritating cleanser such as Cetaphil (Galderma Laboratories) or mineral oil can be used. Many practitioners use cotton swabs or pads to apply the cleanser. Cloth or pressed cotton cosmetic pads are less likely to adhere to the skin surface and break apart, and are more effective for removing feces.

**Treatment Options for Irritant Diaper Dermatitis**

The management of all diaper dermatoses should include reducing moisture in the diaper area, minimizing contact with urine and feces, and eradicating infectious organisms (Figure 1). With the exception of candidiasis-associated DD, IDD is episodic and tends to be relatively self-limiting in nature if the offending irritants are removed. Occasionally, patients may have a true allergic contact dermatitis, in which case it is important to eliminate the causative agent, which may be present as a coloring additive or elastic/rubber agent in the diaper.

Numerous over-the-counter (OTC) barrier formulations are available. Table 2 lists some of the more commonly used products and their active ingredients. Petrolatum is a safe and effective OTC barrier product that protects the skin, reduces transepidermal water loss, and repels water. Petrolatum provides a thick, protective barrier and is useful in the prevention of IDD. It is also a commonly used ingredient in other OTC barrier products such as Vitamin A & D® Ointment (Schering-Plough Healthcare) and in pre-
scription products such as Vusion® Ointment (Barrier Therapeutics). Lanolin is another effective skin protectant that also is used in Vitamin A & D Ointment and other skin lotions and creams. Many infants will benefit from traditional lanolin-containing therapies; however, the healthcare provider must be aware that in children who are allergic to lanolin, such products may aggravate the condition.

Zinc oxide preparations such as Desitin® (Johnson & Johnson) are available in 10% and 40% ointment formulations (Table 2). For treatment of uncomplicated IDD, a petrolatum product, Vitamin A & D Ointment, or a product containing 10% zinc oxide may be sufficient. For moderate-to-severe IDD, a barrier ointment with a greater concentration of zinc oxide can also be used. Zinc oxide pastes are highly effective barriers, but they are difficult to wash off, and aggressive cleansing of the skin when removing a paste is very irritating. Mineral oil is more effective than soap and water to remove a paste. A thin layer of petrolatum can be applied to the paste to prevent opposing skin surfaces from sticking together and to ensure that the paste does not adhere to the diaper.

The use of cornstarch is recommended over talcum powder to reduce frictional injury, especially if the caregiver uses cloth diapers. Although it has been suggested that powders and cornstarch enhance microbial growth, in a study by Leyden it was demonstrated that neither product is associated with proliferation of C albicans. The use of talcum powder has been associated with severe respiratory distress caused by accidental inhalation.

**Zinc oxide pastes are highly effective barriers, but they are difficult to wash off, and aggressive cleansing of the skin when removing a paste is very irritating.**

**Table 2. Commonly Used Barrier Preparations and Their Active Ingredients**

<table>
<thead>
<tr>
<th>Barrier</th>
<th>Active ingredient(s)</th>
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<tbody>
<tr>
<td>Vitamin A &amp; D ointment®</td>
<td>Vitamin A, vitamin D, lanolin, white petrolatum, paraffin</td>
</tr>
<tr>
<td>A&amp;D diaper rash cream with zinc oxide and aloe®</td>
<td>Zinc oxide (10%), dimethicone (1%)</td>
</tr>
<tr>
<td>Aquaphor®</td>
<td>Petrolatum, lanolin</td>
</tr>
<tr>
<td>Aveeno diaper cream®</td>
<td>Zinc oxide (13%), dimethicone (5%)</td>
</tr>
<tr>
<td>Balmex diaper rash ointment®</td>
<td>Zinc oxide (11.3%)</td>
</tr>
<tr>
<td>Balmex daily protective clear ointment®</td>
<td>White petrolatum (51.1%)</td>
</tr>
<tr>
<td>Balmex creamy lotion barrier®</td>
<td>Zinc oxide (13%)</td>
</tr>
<tr>
<td>Boudreaux’s butt paste®</td>
<td>Zinc oxide (16%)</td>
</tr>
<tr>
<td>Burt’s bees baby bee, diaper ointment with vitamin A and vitamin E®</td>
<td>Zinc oxide</td>
</tr>
<tr>
<td>California baby diaper rash cream®</td>
<td>Zinc oxide (12%), lanolin</td>
</tr>
<tr>
<td>Desitin diaper rash ointment, creamy®</td>
<td>Zinc oxide (10%)</td>
</tr>
<tr>
<td>Desitin diaper rash ointment, original®</td>
<td>Zinc oxide (40%)</td>
</tr>
<tr>
<td>Gerber diaper rash ointment with oatmeal soothers®</td>
<td>Zinc oxide (40%), petrolatum (33%)</td>
</tr>
<tr>
<td>Johnson’s baby diaper rash cream with zinc oxide®</td>
<td>Zinc oxide (13%)</td>
</tr>
<tr>
<td>Mustela bebe vitamin barrier cream®</td>
<td>Zinc oxide (10%)</td>
</tr>
<tr>
<td>Palmer’s diaper rash cream, cocoa butter formula®</td>
<td>Petrolatum (30%), dimethicone (1%)</td>
</tr>
<tr>
<td>Triple paste®</td>
<td>Petrolatum</td>
</tr>
<tr>
<td>Vaseline®</td>
<td>Petrolatum</td>
</tr>
<tr>
<td>Vaseline baby, baby fresh scent®</td>
<td>Petrolatum</td>
</tr>
<tr>
<td>Weleda diaper care®</td>
<td>Zinc oxide (12%)</td>
</tr>
<tr>
<td>Zinc oxide ointment</td>
<td>Zinc oxide (20%)</td>
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</tbody>
</table>

Source: Ref 33

**Treatment of Candidiasis-Associated Diaper Dermatitis**

In 2006, Vusion, the first antifungal agent indicated for the treatment of candidiasis-associated DD became available. This combination barrier-antifungal
product is an efficient means of treating candidiasis-associated DD. Until 2006, management consisted of off-label use of an antifungal agent unapproved for candidiasis-associated DD or a combination antifungal/corticosteroid topical product. Some practitioners still prescribe these and other medications off-label to treat candidiasis-associated DD.

**The Risks of Mid- to High-Potency Steroids**

The use of a mid- to high-potency corticosteroid is a relatively common, and sometimes hazardous, practice for the treatment of pediatric dermatoses. Use of such agents in infants and children, particularly in occluded areas, can lead to a number of complications, including atrophy, striae, and adrenal axis suppression. Unfortunately, caregivers are not always aware of these risks, and clinicians may need further education.

Until recently, clinicians had a limited choice of antifungal and antifungal/corticosteroid products in their armamentarium, none of which were formulated specifically to treat candidiasis-associated DD.

Mid- to high-potency corticosteroids are generally contraindicated for use in intertriginous and occluded areas of the skin, especially in infants, as their use has the potential to cause a variety of adverse events including systemic absorption, skin atrophy, striae, tachyphylaxis, and growth delay. Furthermore, the abraded skin of the diaper area of an infant with IDD leads to an enhanced rate of percutaneous absorption of a topical agent, which is further increased by the occlusiveness of the diaper. The risks of corticosteroid use in infants need to be communicated to clinicians, and safer therapeutic alternatives need to be developed.

Commonly Used Antifungal Agents for Candidiasis-Associated Diaper Dermatitis

Few well-designed comparator trials have assessed the efficacy of antifungal agents for the treatment of candidiasis-associated DD. Imidazole antifungal agents (eg, miconazole, econazole, ketoconazole), have varying fungicidal activity against cutaneous Candida spp. Topical azoles are also well-tolerated. Side effects sometimes seen with systemicazole therapy are not associated with topical use of these types of drugs.

If an infant has a rash secondary to antibiotic use and the infant must continue antibiotic therapy, a thick petrolatum ointment barrier may provide optimal protection. The practitioner also must consider the possibility of another diagnosis in patients who do not respond to appropriate treatment of candidiasis-associated DD.

Until recently, clinicians had a limited choice of antifungal and antifungal/corticosteroid products in their armamentarium, none of which were formulated specifically to treat candidiasis-associated DD. Management considerations have been simplified for the clinician with the FDA approval of Vusion Ointment (miconazole nitrate 0.25%/zinc oxide 15%/petrolatum 81.35%), which is a safe, effective, and convenient treatment option for candidiasis-associated DD.

**Vusion Ointment (miconazole nitrate 0.25%/zinc oxide 15%/petrolatum 81.35%)**

Vusion Ointment is indicated for the treatment of candidiasis-associated DD and approved for use in immunocompetent pediatric patients aged 4 weeks and older with microscopic evidence of pseudohyphae and/or budding yeast. Before prescribing Vusion Ointment, the role of the clinician is to ensure that the correct diagnosis is made, which includes considering the more uncommon causes of IDD and performing ancillary tests (eg, KOH), to confirm diagnosis if doubt exists as to the etiology of the eruption.
Vusion Ointment is formulated with a low concentration (0.25%) of miconazole, which limits exposure to the antifungal component. The level of systemic absorption is lower with 0.25% miconazole than with 2% miconazole cream. Infant skin, as opposed to adult skin, can absorb a greater proportion of topical medication because infants have a greater surface-to-body weight ratio. Thus, a lower concentration formulation of miconazole can provide effective antifungal activity on infant skin while theoretically decreasing the risk of extensive absorption of miconazole. Furthermore, the combination of miconazole and zinc oxide is synergistic; zinc oxide also has anti-Candida effects, which potentiates the antifungal activity of miconazole.

Clinical Studies with Vusion Ointment
The efficacy and tolerability of Vusion Ointment in the treatment of candidiasis-associated DD have been established in a double-blind, randomized, multicenter, vehicle-controlled study of 0.25% miconazole nitrate ointment. The study included 330 patients with an IDD average severity grade ≥3 as assessed by the Diaper Dermatitis Severity Score, including a severity grade of ≥2 for erythema. Patients with a positive KOH preparation and positive culture of Candida (n=236) composed the modified intent-to-treat (MITT) group, on which efficacy analyses were based. Patients were randomized to receive active treatment or vehicle for 7 days at every diaper change and after bathing. The primary end point of overall cure rate (clinical cure plus microbiologic cure) was assessed on post-treatment Day 14.

Results demonstrated that 23% of the patients in the active treatment group achieved the primary end point, compared with 10% of those in the vehicle group (P=.005) (Figure 10). A total of 38% and 11% of patients in the active and vehicle treatment groups, respectively, achieved the secondary end point of clinical cure (P<.001), and 50% and 23% of patients, respectively, achieved microbiologic cure. Vusion Ointment was demonstrated to be well tolerated, and the rate of adverse events was comparable between groups (active treatment group, 22% vs vehicle group, 19%; P=.585). All adverse events reported among patients in the miconazole group were mild or moderate in severity and were considered unlikely to be related to the active study agent.

CONCLUSIONS
IDD is a common problem that accounts for frequent visits to the pediatrician each year and causes concern to families. An appropriate approach to diagnosis and treatment is essential. Clinicians should become familiar with the benefits and drawbacks of the many OTC products available for the treatment of IDD. Adherence to proper cleansing and diapering routines are the key primary measures in preventing the onset of diaper dermatitis. Proper diagnosis of candidiasis-associated DD and the elimination of off-label use of high-potency topical steroids from the treatment paradigm are also important. Early-stage IDD can be managed effectively by keeping the diaper area dry and the stratum corneum intact with frequent diaper changes, gentle cleansing, and the use of barrier protection.

For more severe forms of IDD, proper diagnosis is essential. Use of a topical antifungal agent can be very useful if IDD is complicated by Candida. Clinicians should recognize the signs and symptoms of the more severe forms of IDD, including candidiasis-associated DD, to ensure the proper course of treatment. Clinicians must be aware of other less common but serious conditions that may masquerade as IDD and consider these diagnoses if a patient does not respond to an antifungal treatment regimen.
REFERENCES


VUSION®
125% miconazole nitrate, 15% zinc oxide, and 81.35% white petrolatum

DOSAGE AND ADMINISTRATION

VUSION® Ointment should not be used to prevent the occurrence of diaper derma-
titis, since preventative use may result in the development of drug resistance.

Before applying VUSION® Ointment, gently cleanse the skin with lukewarm water and pat dry with a soft towel. Avoid using any soaps, shampoos, or lotions on the diaper area.

VUSION® Ointment should be applied to the affected area at each diaper change for 7 days. Treatment may be repeated every 7 days if the diaper rash is still present. Thoroughly wash hands after applying VUSION® Ointment.

OVERDOSAGE

VUSION® Ointment is intended for topical use only. Young children are at risk for acciden-
tally ingesting VUSION® Ointment. To prevent giving VUSION® Ointment to children or poison control should be contacted in the event of accidental ingestion.

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