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MASD Part 2: Incontinence-Associated Dermatitis and Intertriginous Dermatitis

A Consensus

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A consensus panel was convened to review current knowledge of moisture-associated skin damage (MASD) and to provide recommendations for prevention and management. This article provides a summary of the discussion and the recommendations in regards to 2 types of MASD: incontinence-associated dermatitis (IAD) and intertriginous dermatitis (ITD). A focused history and physical assessment are essential for diagnosing IAD or ITD and distinguishing these forms of skin damage from other types of skin damage. Panel members recommend cleansing, moisturizing, and applying a skin protectant to skin affected by IAD and to the perineal skin of persons with urinary or fecal incontinence deemed at risk for IAD. Prevention and treatment of ITD includes measures to ensure that skin folds are dry and free from friction; however, panel members do not recommend use of bed linens, paper towels, or dressings for separating skin folds. Individuals with ITD are at risk for fungal and bacterial infections and these infections should be treated appropriately; for example, candidal infections should be treated with antifungal therapies.

Introduction

Moisture-associated skin damage (MASD) encompasses a range of conditions, including incontinence-associated dermatitis (IAD) and intertriginous dermatitis (ITD). MASD presents as inflammation of the skin, occurring with or without erosion or secondary cutaneous infection. It is often undetected until significant inflammation, maceration, or skin erosion occurs. Despite the fact that both IAD and ITD are common presentations, we reviewed medical and nursing textbooks and found little information about these conditions. An earlier article focusing on the historical perspective of IAD revealed both a lack of consensus and the scarcity of research on this topic.[1] Even fewer studies have focused on ITD.

To bring deserved attention to these clinically relevant conditions, a panel of 9 clinical experts met in Minneapolis in 2010 to discuss 4 common forms of MASD. The first article from this consensus group discussed shared etiology, pathophysiology, prevention, and treatment principles applicable to 4 forms of MASD.[2] The second in a 3-article series, this discussion details essential clinical knowledge related to IAD and ITD. A third article will offer a detailed examination of peristomal and periwound forms of MASD.

Incontinence-Associated Dermatitis

IAD is a form of irritant dermatitis that develops from chronic exposure to urine or liquid stool.[1] Several terms have been used to describe the change in condition of the skin from exposure to urine and/or stool including diaper dermatitis, maceration, perineal dermatitis, and moisture lesions; these lesions have also been mistakenly labeled

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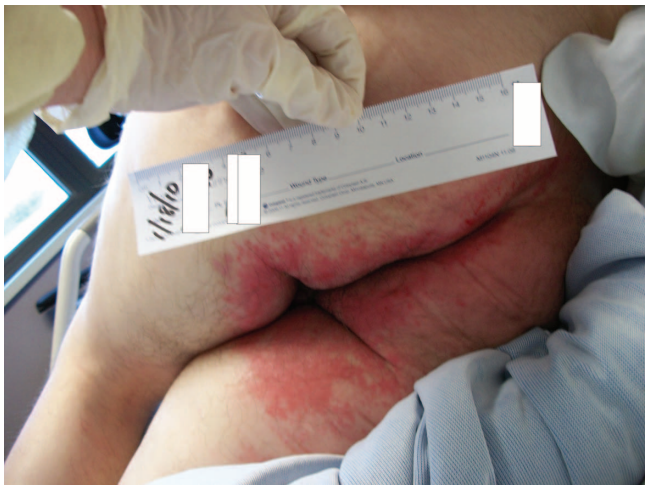

FIGURE 1. Incontinence-associated dermatitis extending to inner aspects of both thighs (photo credit: Courtesy Gretchen Steinmetz, Kindred Healthcare).

as stage I or II pressure ulcers. The benefit of using the term IAD to describe this condition is explained by Gray and colleagues.[1]

IAD occurs not only in the perineum (the skin from the vulva to the anus in women; the skin from the scrotum to the anus in men) but also in the labial folds, groin, buttocks, or scrotum from exposure to urine and in the perianal and gluteal cleft from exposure to stool.[1] Depending upon the skin areas exposed to urine or stool and/or the use of containment devices, IAD can also extend to the inner and posterior thighs and may include other elements such as candidiasis (Figures 1 and 2).

The prevalence of IAD varies widely depending upon the setting. In the long-term care (LTC) setting, Bliss and colleagues[3] used logistic regression analysis to examine

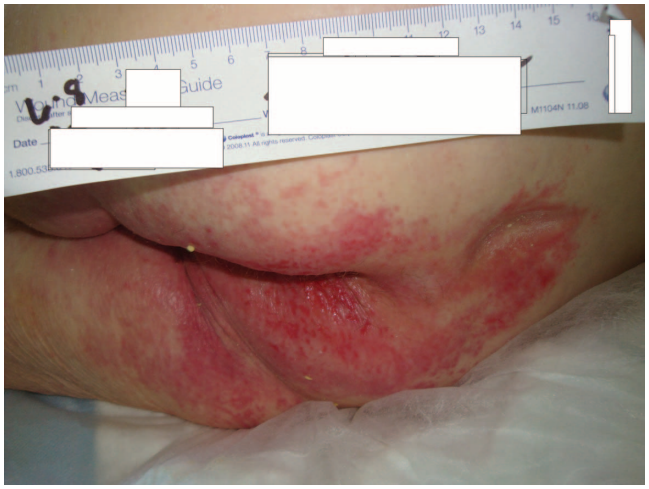

FIGURE 2. Incontinence-associated dermatitis with serous exudate causing the skin to glisten and secondary cutaneous candidiasis with characteristic satellite lesions (courtesy Gretchen Steinmetz, Kindred Healthcare).

records from 59,558 nursing home residents. The prevalence of incontinence was 59.8%; 39.7% had double incontinence, 12.4% had fecal incontinence only, and 7.7% had urinary incontinence only. IAD was present in 5.7% (n = 3405) of residents. The reported prevalence may be low since IAD was not specifically assessed by the nursing home staff as part of routine reporting.

A prospective study by Bliss and associates[4] revealed that of the 1918 nursing home residents screened, 98% (n = 1879) were incontinent, and 51% of those (n = 981) qualified for prospective surveillance of IAD because they were incontinent, free of perineal skin damage, and had no other exclusion criteria. Of those 981 residents, 78.6% had double incontinence, 19.6% had urinary incontinence only, and 1.8% had fecal incontinence only. IAD developed in 3.4% of enrolled patients over a 2-week period.[4] In this study, each facility had a defined skin care regimen in place, which was hypothesized to account for the comparatively low incidence of IAD.

Lyder and colleagues[5] reported a 23% to 25% incidence of IAD in a geropsychiatric unit, and Keller and colleagues[6] reported a 41% prevalence of IAD in an LTC facility. Roe and colleagues[7] conducted a meta-analysis of 10 studies on the prevalence of urinary incontinence in LTC and reported the prevalence of UI to be as high as 65%. However, this meta-analysis provided no data regarding the incidence or prevalence of IAD.

Junkin and Selekof[8] reported a 13.5% prevalence of IAD among patients with urinary incontinence in 2 acute care facilities. Junkin and Selekof hypothesized that this observation was influenced by the fact that 33% of patients were managed by indwelling catheter and deemed continent of urine. When compared to the young adult group, patients over 80 years were statistically significantly more incontinent.[8]

Limited data are available on IAD secondary to fecal incontinence. Driver conducted a quality improvement project and reported that 12% of 131 patients in critical care had fecal incontinence and 50% developed skin breakdown. Of these, 31% developed IAD within 14 days and 19% developed skin damage after 14 days. IAD did not develop in any patients who were continent of stool.[9]

Junkin and Selekof[8] reported a 21.5% prevalence of IAD in patients who were incontinent of stool or both urine and stool. The prevalence of skin damage, including both IAD and pressure ulcers, in these patients was 42.5%; persons with skin injury were more likely to have fecal incontinence than urinary incontinence (45.8% vs 29.7%, respectively, $P = .0875$). Bliss and colleagues[10] reported that one-third of 152 patients in acute care or critical care had fecal incontinence. Antimicrobial use and *Clostridium difficile* diarrhea were associated with fecal incontinence in both groups.

Ehman and colleagues[11] prospectively observed 45 patients in an intensive care unit, all of whom were fecally incontinent and free of skin damage at admission. Over a period of 7 ± 4 days, 35.5% of patients developed IAD; the median time to onset of IAD was 4 days.

In the community setting, Bliss and coworkers[12] studied 188 subjects with fecal incontinence; 28% of their subjects also had urinary incontinence. According to self-reported surveys, 52% (n = 96) had IAD-related skin damage. Skin damage was reported as always present for 5%, often present for 10%, and sometimes present for 23%.

Etiology and Pathophysiology

The pathophysiology of IAD is based on an understanding of the skin's moisture barrier function.[2] Prolonged exposure to moisture of any kind (water, saline, or urine) can act as an irritant and result in contact dermatitis.[13] Occasional exposure to urine alone may not be deleterious, but repeated exposure, especially in the presence of occlusion or fecal material, puts the skin at higher risk.[8] Chronic moisture exposure compromises the skin's barrier function, quantified as transepidermal water loss (TEWL). The use of an absorptive or containment device may exacerbate irritation when it creates prolonged occlusion and hyperhydration of the skin.

Unique aspects to urine-induced IAD include its chemical composition and osmolarity. Ammonia in the urine increases the skin's pH,[14,15] and for many years it was believed that the ammonia in urine damaged the skin. More recent study has shown that the injury is actually due to an alkaline pH that can result from the conversion of ammonia to ammonium by fecal bacteria.[16,17]

Fecal incontinence is also deleterious to the skin. Intestinal and bacterial enzymes can weaken epidermal integrity and cause skin damage.[18,19] The alkaline pH of urine may promote the enzymatic activity of lipases and proteases in patients with dual (urinary and fecal) incontinence. Lipases and proteases are also produced by a variety of fecal bacteria. These enzymes are thought to break down proteins in the keratinocytes, contributing to erosion.[20] Liquid stools are also associated with malabsorption and compromised nutrition, which has been associated with an increased likelihood of IAD in hospitalized patients.[8]

MASD is further associated with alterations in the skin's flora reflected in an increased number of coagulase-negative staphylococci.[21] Secondary infection of denuded skin can develop from coliform bacteria or *Candida albicans*.[22] In addition, pathogenic toxins resulting from *C difficile* increase the risk for secondary infections and skin damage.

Associated Factors

Brown[23] proposed 3 factors leading to IAD: tissue tolerance, perineal environment, and toileting ability. Factors affecting tissue tolerance include age, health status, nutritional status, oxygenation, perfusion, exposure to shear or friction, and core body temperature. The perineal environment is altered by the frequency and type (urinary, fecal, or dual) of incontinence, condition of the skin (inflamed, edematous), and factors that impair the perineal skin (hydration, pH of urine or stool, pathogens in the stool or on the skin). The ability of the patient to reach the toilet, as well as cognitive inability to recognize the need to toilet, comprises the final factors in this model. The framework was validated in 166 hospitalized patients and the findings showed a statistically significant correlation between IAD and fecal incontinence, frequency of incontinence, poor skin condition, pain, poor skin oxygenation, fever, and compromised mobility. Additional contributing factors included the severity of illness and comorbid conditions as well as low serum albumin, which is associated with a marked rise in the likelihood of skin damage.[8]

In the LTC patient population, Bliss and colleagues[24] reported on a prospective surveillance study of residents who were incontinent of urine or feces. The majority of patients who developed IAD were female, elderly, and white. All patients with fecal incontinence developed IAD; rates of urinary and double incontinence were similar in those who did and did not develop IAD. There was no difference in cognitive impairment between patients with and without IAD. The time to the development of IAD was 6 to 42 days (median, 13 days) in a study of 981 residents in 16 nursing homes.

IAD, Pressure, Shear, and Friction

Friction can physically damage the stratum corneum.[25] Wet, soft skin does not glide against bed linens or clothing and is therefore more vulnerable to friction injury and erosive skin loss.[26] IAD is associated with partial thickness wounds; existing evidence does not reveal an association with full-thickness wounds. Consensus panel members noted that in contrast to the more discrete lesion characteristic of a pressure ulcer, IAD tends to present as a larger area of complete epidermal erosion. Therefore, it is possible that IAD renders the skin more vulnerable to damage from pressure, shear, friction, and mechanical stripping. It is not known whether IAD-damaged skin can advance to complete erosion without the complicating factors of pressure, shear, and friction.

IAD and pressure ulcers often coexist.[27,28] The precise nature of the relationship between these conditions is not entirely understood, but existing research suggests that moisture-injured skin has an impaired tolerance to friction or pressure, resulting in a higher risk for pressure ulceration. The risk of skin ulceration from pressure is reduced by offloading. Repositioning, while cleansing the skin and changing incontinence products, is seldom adequate "offloading of pressure" because of the short periods of time spent moving the patient during the cleansing regimen. Fader and colleagues[29,30] reported that in a study of incontinence pad changing regimens, subjects were seldom turned or repositioned at the time of pad changing.

Assessment

A focused history identifying the presence of urinary or fecal incontinence, comorbid factors associated with a higher likelihood of IAD, and functional factors associated with increased risk such as inability to toilet should be obtained.

Visual inspection and patient history are key elements in accurate diagnosis of IAD. These techniques are used whether the clinician is examining outpatients, whose IAD tends to be mild to moderate, or acutely ill inpatients, who often present with more severe cases. Etiology, while always a consideration, is especially important when evaluating more severe cases of denudation or erosion, in part to differentiate IAD from pressure ulcers. For instance, the finding of moist or macerated skin strongly suggests that the skin damage is due at least in part to moisture, and this finding can aid in proper differentiation and diagnosis. A thorough examination of the perineal and perigenital areas, including skin folds, helps the clinician to differentiate skin damage associated with moisture and occlusion from that associated with pressure and shear. When inspecting the patient's skin, the clinician should look for evidence of maceration, changes in pigmentation, areas of skin loss, and evidence of cutaneous infection such as candidiasis. Based on patients' self-reporting, inflammation in dark-skinned individuals may present as either hyperpigmentation or hypopigmentation. Indicators of candidiasis include a central maculopapular rash with characteristic satellite lesions.

Several instruments have been developed for assessing the risk of IAD and the severity of IAD. The Perineal Assessment Tool[31] assesses the risk for IAD based on the type and intensity of the irritant, the duration of exposure, the condition of the perineal skin, and other contributing factors including the use of antibiotics, *C difficile* diarrhea, tube feeding, and low serum albumin levels. Three tools have been developed for systematic assessment and grading of IAD. The Perineal Dermatitis Grading Scale is a descriptive scale that includes skin color, skin integrity, lesion size, and symptoms of pain.[32] The IAD Skin Condition Assessment Tool generates a score based on the area of skin breakdown, the degree of redness, and the amount of erosion.[33] More recently, the IAD severity instrument has been developed and has undergone initial validation. This instrument assesses 13 body locations for IAD including the perianal skin, genitalia, buttocks, inner thighs, lower abdomen, and suprapubic area. The instrument requires the clinician to determine the degree of redness, magnitude of skin loss, and presence of rash. Face and content validity were established via feedback from 247 WOC nurses, 100 staff nurses, and 16 nursing assistants.[34]

Because of the complexity associated with diagnosis based on visual inspection alone, researchers are seeking to develop physiological measures to aid clinicians in differentiating IAD and partial thickness damage due to other factors. Two examples under investigation include high-resolution ultrasound[35,36] and subepidermal moisture.[37] None have yet proven practical for use in the clinical setting.

Despite the evolution of instruments for assessment and classification of IAD, the most common scale used to report IAD is the National Pressure Ulcer Advisory Panel pressure ulcer staging system. Stage II pressure ulcers were redefined in 2005 to exclude skin lesions from etiologies other than pressure, yet today most open skin wounds, even those due to etiologies other than pressure, are still incorrectly identified using the pressure ulcer staging system.[38] Thus increased awareness of MASD and IAD is critical, as is increased skill in differential assessment. Table 1 compares the findings of skin ulceration from IAD to that of stage I and II pressure ulcers.

TABLE 1.

Differentiation of IAD Versus Stage I and II Pressure Ulcers

Factors	IAD	Stage I Pressure Ulcers	Stage II Pressure Ulcers
History of condition	Exposure to urine or stool	Exposure to pressure, shear, and/or microclimate from immobility or inactivity	Exposure to pressure, shear, and/or microclimate from immobility or inactivity
Location of affected skin	Skin folds in areas where urine or stool can accumulate	Skin usually over bony prominences or exposed to other external pressure (eg, medical device)	Skin usually over bony prominences or exposed to other external pressure (eg, medical device)
Color of wound bed	Shiny, red, glistening, no slough in wound bed	Nonblanchable erythema of intact skin	Shiny, pink, or red open wound, no slough in wound bed
Color of periwound tissue	Red, irritated, edematous	Normal for race/ethnicity, edema may be palpable	Normal for race/ethnicity, edema may be palpable
Characteristics of involved area	Blotchy, not uniform in appearance	Tend to be single areas of erythema	Tend to be single ulcers with distinct ulcer wound margin
Pain	Burning, itching, and tingling	Sharp pain, usually no itching; pain may intensify when patient is initially moved off of injured areas	Sharp pain, usually no itching; pain may intensify when patient is initially moved off of injured areas
Odor	Urine, fecal odor	None	None unless infected and then may have odor of infecting organism
Other	Candidiasis common (seen as satellite lesions)	Redness tends to resolve with offloading or repositioning of device	Ulcer bed is shallow and heals through epithelialization

Abbreviation: IAD, incontinence-associated dermatitis.

Prevention and Treatment

Prevention of IAD is centered on prompt removal of irritants from the skin, combined with application of a skin protectant. Adherence to a defined, consistent skin care regimen is the best way to remove irritants and restore as much of the inherent skin barrier as possible. Studies examining defined skin care programs for prevention of IAD have not yet provided a solid evidence base for specific recommendations. Studies have compared soap and water to no-rinse products[39,40]; products that clean, moisturize, and repel moisture from the perineum to prior skin care practices[5]; and the comparative effectiveness of acrylate polymer barrier film, 43% petrolatum, zinc oxide with dimethicone, and 98% petrolatum.[4] Bliss and colleagues studied use of these 4 products in 1918 residents of LTC facilities. The incidence of IAD did not differ between programs, but the overall incidence was 3.4%, which is quite low in the high-risk population enrolled in their study.[4] While none of these studies showed a clear preference for any given product or method, the best evidence supports the use of a defined skin care program and quality products (Table 2). The consensus panel agreed on 2 principles for any defined skin care regimen: (1) cleansing and moisturization are advised for any patient, and (2) cleansing, moisturization, and application of a skin protectant are advised for any patient experiencing urinary or fecal incontinence because of the increased risk for IAD.

Cleanse

Gentle mechanical actions should be used when cleansing the skin, and scrubbing should be avoided. The cleanser should be free of perfumes or potential irritants, and its pH should be similar to that of normal skin. Since towel-drying has been shown to compromise the skin's moisture barrier,[41] a no-rinse formulation should be used for frequent bathing. Some clinicians have expressed concern about the efficacy of no-rinse formulations when compared to soap and water, but a recent comparative study reported equivalent efficacy in removal of gram-negative (coliform) bacteria and potentially pathogenic gram-positive organisms in human subjects.[42] No-rinse options include incontinence or perineal cleansers, disposable wipes, or 3-in-1 sprays. Some single-step formulations provide both cleansing and moisturizing, and some provide cleansing, moisturizing, and application of a skin protectant.

Moisturize

Most cleansers contain either humectants or emollients, or both, to preserve the lipid barrier. Humectants (eg, glycerin, urea, α-hydroxy acids, and sugars) aim to attract

TABLE 2.

Nursing Process—Prevention and Treatment of Incontinence-Associated Dermatitis

Diagnosis	Goal and Expected Outcomes	Interventions
At risk for skin impairment (due to urinary and/or fecal incontinence)	Maintain skin integrity by minimizing exposure to irritants (urine, stool, excessive perspiration), evidenced by lack of redness in perineum or skin folds	Use a structured skin care regimen: 1. Cleanse perineal skin after each episode of incontinence, and daily, with a no-rinse cleanser close to 5.5 pH. 2. Avoid using bath basins for perineal care; these basins are often contaminated. 3. Do not scrub the skin; gently soak the skin by applying damp cloths for a few minutes to soiled skin or use the manufactures' suggested products to remove prior applications of skin protectant products. 4. Check closely in skin folds for residual stool and urine, remove as noted earlier. 5. After the skin is cleaned, moisturize using products that contain humectants and emollients. Avoid products with strong concentrations of humectants (ie, urea, glycerin, α-hydroxy acids, lactic acid) since these products can add too much water to the skin. 6. Protect the skin to reduce exposure to urine and stool with products that contain petrolatum, zinc oxide, dimethicone, or a combination of these. Consider a copolymer film to seal the skin if the patient is at very high risk due to frequent fecal incontinence. 7. Educate all care providers on preferred method of skin care. 8. Keep skin care products near bedside (as permissible in facility) to improve compliance.
Impaired skin integrity	Improve skin integrity: minimize and/or eliminate redness, pain, candidiasis	1. Implement the prevention program described earlier. 2. Apply a skin protectant (zinc oxide, petrolatum, dimethicone, or a combination) or a skin sealant (a copolymer film product). 3. Treat areas of cutaneous candidiasis with antifungal powder or cream. 4. Consider diversion of urine or stool.

water to the stratum corneum. Emollients, which aim to replace intercellular lipids in the stratum corneum, are designed to smooth the skin's surface and to prevent "gaps" in the skin barrier. Examples are cholesterols, fatty acids, and squalene. An emollient may be applied as a separate product or it may be incorporated into a no-rinse or perineal cleanser. The panel concurred that emollients may provide a greater advantage to humectants for MASD because this condition is associated with overhydration of the skin.

Protect

Skin protectants, classified by dermatologic specialists as occlusive moisturizers, should serve as a moisture barrier for the skin, protecting the stratum corneum from exposure to irritants and excess moisture. In addition, protectants should maintain both hydration and a normal level of TEWL, while avoiding maceration from prolonged use. Options include (1) ointment-based skin protectants—zinc oxide, dimethicone, and petrolatum, and (2) liquid acrylates, also known as protective skin barriers (Table 3). One retrospective clinical trial showed similar outcomes in IAD with polymer acrylate barriers versus 3 ointment-based skin protectants; however, these results must be viewed with some caution since the study was powered for economic rather than efficacy outcomes.[4] Expert opinion advocates selection of a skin barrier with fewer ingredients because impaired skin absorbs materials placed on it and this can lead to allergy.[43]

Containment or Diversion of Urine and Stool

Body-worn external collection devices such as male external catheters or anal pouches may also be used. However, the skin will require protection by other means if there is risk of leakage around the device.[8] Absorptive pads, including including bed pads and chair pads, maybe used. Wrap-around absorbent briefs are an option and are often advisable for diagnostic tests, treatment procedures, or occasional use for those in the home setting in order to contain incontinence and promote dignity. Anal-rectal dressings are placed between the buttocks for small-volume leaks.[44] Anal plugs (small foam devices that expand when inserted into the anus) act as a physical barrier to stool leakage. They are available only in Europe.[45,46]

IAD treatment strategies vary, which is not surprising, given the dearth of research and education. The following strategies are recommended based on existing evidence: (1) remove irritants and aggressively manage exposure to urine or stool, (2) eradicate cutaneous infections, and (3) transiently divert urine or stool when indicated. The cleanse-moisturize-protect regimen from prevention efforts must carry over to treatment as well, to protect against further inflammation and damage.

Transient diversion of urine or stool may involve insertion of an indwelling urinary catheter or indwelling fecal drainage system. One study revealed that, for patients with fecal incontinence and diarrhea in a surgical intensive care unit, diversion of stool reduced the incidence of skin damage from 43.0% to 12.5%.[47] However, use of any indwelling device must be carefully weighed against the risk of hospital-acquired urinary tract infection or mucosal erosion and bleeding from prolonged use of a fecal drainage system. "Butt pastes," various products that contain zinc oxide, balsam of Peru, and citric acid are sometimes used, but we identified no research evaluating their efficacy for prevention or treatment of IAD.

Treatment for secondary candidiasis includes topical antifungals, which can effectively treat cutaneous infections. Effective agents include the polyene antibiotics, azoles, and the allylamines.[48] An antifungal powder may be applied to affected skin in a thin layer, followed by application of a skin protectant. Alternatively, several ointment or cream-based skin protectants are available that may be applied in a single step.

TABLE 3.

Skin Protectants[a,b]

Type	Description	Strengths	Limitations
Dimethicone (ointment)	Silicone-based oil	Good skin hydration	Variable protection against irritants (especially at lower concentrations), modest protection against maceration
Petrolatum (ointment)	Blend of castor seed oil and hydrogenated castor oil	Good protection against irritants, avoids maceration	Modest skin hydration
Zinc oxide (ointment)	White powder, mixed with cream or ointment	Good protection against irritants	Does not avoid maceration, offers poor skin hydration, is difficult to remove
Liquid acrylate	Protective skin barrier	Less drying; nonalcohol formulation results in less pain	
		Resists washing off; potentially fewer applications	

[a] Adapted from Bliss and colleagues[4] and Hoggarth and colleagues.[84]
[b] Ointments should be applied in a thin layer, and to the skin only (not to a brief), because they can interfere with the absorptive ability of briefs or underlying pads.[85]

Intertriginous Dermatitis

ITD is an inflammatory dermatosis of opposing skin surfaces caused by moisture. It is commonly found in the inframammary, axillary, and inguinal skin folds. ITD can develop in any skin fold; people with more skin folds, especially the obese, often have ITD under the abdominal or pubic panniculi. Following weight loss surgery, surplus skin remains and is also a site for the development of ITD.[49] ITD can coexist with IAD. ITD is a relatively common condition, yet there is a significant lack of information about its epidemiology, etiology, pathophysiology, assessment, and treatment.

Epidemiology

ITD occurs in many patient groups, and the data on epidemiology of ITD are generally grouped by patient type. McMahon[50] examined 1116 female patients in a hospital and reported that 11.2% had new cases or resolving cases of inframammary ITD. Brown and colleagues[51] surveyed 100 obese patients for the prevalence of skin problems, including rash, dryness, and "broken skin." Of the respondents, 63% indicated that they had more than 1 skin problem, the most common being broken skin and itching. The self-reported cause of the skin problem was perspiration and friction. ITD is likely underreported due to patient self-management. In addition, a lack of uniform nomenclature for ITD has hampered systematic study.

Etiology and Pathophysiology

ITD is hypothesized to arise from skin-on-skin friction that initially leads to mild erythema and may progress to more intense inflammation with erosion, oozing, exudation, maceration, and crusting.[52-54] Intertrigo is facilitated by trapped moisture in the skin folds where air circulation is limited. Predisposing factors include hyperhidrosis and urinary and fecal incontinence.[52]

Because many cases of ITD occur in obese patients, the changes in the skin associated with obesity play a major role in the development of ITD. In the average adult, the skin covers 2 square meters and weighs 4.5 to 5 kg. Obese patients, however, have comparatively less skin surface for the increased body mass requiring thermoregulation. This imbalance may account for the linear trend in the severity of obesity and ITD.[55] Specifically, bariatric patients may sweat more profusely after becoming overheated. The combination of increased perspiration and larger skin folds increases the risk for maceration and for friction damage.[56]

The skin's barrier function is also impacted by obesity.[57] Obese patients demonstrate significantly higher TEWL and greater erythema than leaner patients. These obesity-associated changes in skin physiology may be related to increased sweat gland activity.[58] Adipose tissue microcirculation, which aids in metabolite transport and contributes to immunity and inflammation response, is impaired by adipose tissue expansion.[59] Skin surface pH may vary as well; one study revealed women with a body mass index >25 had higher skin surface pH in inguinal skin folds.[57] Diabetes, steroids, and broad-spectrum antibiotics have also been suggested as contributory factors in the development of ITD.[60,61]

Self-neglect and poor hygiene are sometimes considered to be the primary etiology of ITD.[62-65] Although some morbidly obese patients may be unable to reach all body areas to provide self-care, panel members believe that the perception that ITD is typically caused by poor hygiene potentially serves to bias healthcare providers regarding obesity and skin fold disease. Rand's findings therefore may not be surprising: a survey of obese individuals revealed that nearly 80% reported disrespectful treatment from the medical community.[66]

When moisture is not evaporated or absorbed, it accumulates in the skin fold. The overly hydrated stratum corneum does not glide on opposing skin surfaces and friction damage is common.[26] In addition, macerated skin can become inflamed and denuded, which produces a fertile breeding ground for many microorganisms.[52] Even normal skin flora have the potential to become pathogenic since, according to Todar,[67] the normal flora of human skin includes *Staphylococcus epidermidis*, *Staphylococcus aureus*, *Streptococcus pyogenes*, corynebacteria, and mycobacteria.

ITD cultures have grown *S aureus*, group A β-hemolytic streptococcus,[68] *Pseudomonas*,[52,69] *Proteus mirabilis* and *Proteus vulgaris*, enterococci, and vancomycin-resistant enterococci.[70] Group A β-hemolytic streptococci have been reported in pediatric patients in the neck, axillae, or inguinal spaces.[68] The skin pH in the axillae, genitoanal area, and interdigital areas are more alkaline and therefore support the growth of colonic bacterial flora.[71]

The most common fungal organisms in ITD are *Candida*.[72,73] However, a recent study of the bacteriology of inflamed skin folds revealed that *Candida* was not the most prevalent organism in skin folds.[70] Cultures at the time of hospital admission revealed *Pseudomonas*, colonic bacteria, methicillin-resistant *S aureus*, and vancomycin-resistant enterococci in skin folds. In addition, dermatophytes commonly complicate interdigital ITD.[52] If ITD is not well managed, the plethora of bacteria present on damaged skin increases the risk for serious soft tissue infections such as cellulitis and/or panniculitis.

Assessment

No formal risk assessment tool exists for ITD. High-risk patients for ITD include any patient with a skin fold, such as women with pendulous breasts, children with skin folds and stooped posture, athletes with stocky necks, and obese persons.[52]

For optimal assessment, the patient should be lying as flat as possible, and all skin folds should be visualized.[74]

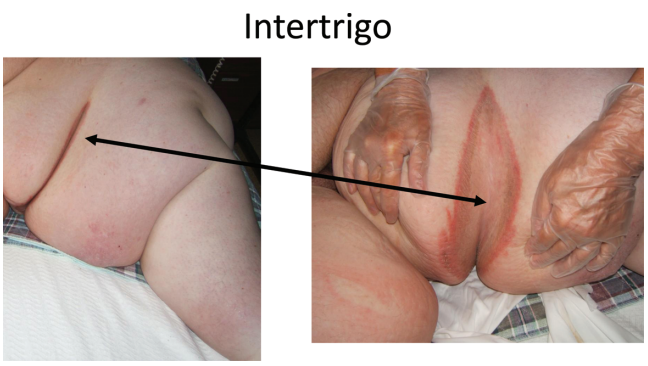

FIGURE 3. Intertriginous dermatitis is apparent when separating the fold on the upper inner thigh, illustrating the importance of fully examining all skin folds (courtesy Karen L. Kennedy-Evans).

Clinicians should examine the entire area of the skin folds, including the base. It is frequently necessary to enlist assistance in order to gently lift the fold without creating or exacerbating traction and fissure formation. Depending on the individual, neck creases, axilla; inframammary; antecubital; subscapular area; waist; inguinal fold; abdominal and flank area; abdominal panniculus, mons pubis; behind the knees; ankle; and interdigital areas should be inspected (Figure 3).

ITD initially presents as mild erythema, typically in a mirror-image manner on each side of the skin fold.[74] It can progress to a more intense inflammation with maceration, erosions, oozing, exudation, and crusting. Symptoms of ITD also include pain, itching, burning, and odor.[52,63,64] Skin biopsies are rarely needed, but culture and sensitivities may be obtained if a secondary infection is suspected.[52]

In pediatric patients, ITD can be difficult to recognize.[68] When *Pseudomonas* presents in the groin of pediatric patients, blue discoloration is evident in the underclothing, sometimes called the "blue underpants sign."[75] Group A β-hemolytic streptococcus has been reported in pediatric patients' skin folds of the neck, axillae, or inguinal spaces.[68] These infections present as fiery red erythema and maceration with a foul odor.

Differential diagnosis of ITD includes allergic contact dermatitis and IAD. Generally, allergic skin diseases are associated with intense pruritus, history of eczema, and patches of eczema in other areas, such as the knees or elbows. Drug reactions may sometimes mimic ITD. Wolf and colleagues[76] reviewed 11 cases of allergic dermatitis from drugs limited to the skin folds. The most common and difficult differentiation is between ITD and IAD in skin folds exposed to urine and feces; it may not always be possible to distinguish ITD from IAD in these situations as the conditions have similar manifestations.

Prevention

Skin folds should be kept as clean and dry as possible. When cleansing, clinicians should use a product with a pH similar to that of healthy skin (pH 5.5). One option is pH-balanced disposable cleansing cloths or soft baby washcloths, since regular washcloths can be abrasive. Cleansing should be gentle, and scrubbing should be avoided. Hand-held showerheads are advisable for patients bathing in the home setting. No-rinse bathing products, cloths, or solutions may be preferred since they offer a one-step process, and the residual product is beneficial to the skin. One patient described wiping ITD-affected skin folds as similar to friction from a rope burn; therefore, skin folds should be "patted" instead of wiped dry.[74] Skin folds can also be fanned to dry or dried with a hairdryer on cool setting.

There is general agreement that prevention of ITD is accomplished by reducing skin-on-skin friction, reducing heat and moisture in the skin fold, and providing skin hygiene.[63] Patients should be encouraged to wear loose-fitting, lightweight clothing made from natural fibers to absorb moisture from skin folds. Other options include athletic clothing specifically designed to draw moisture away from the skin. Textiles that use a polyurethane coating are effective in wicking and translocating moisture, thus reducing moisture in from the skin fold.

Moving the skin folds carefully and maintaining drier skin in the folds will help to reduce frictional forces. Although evidence is lacking, many strategies traditionally used to manage ITD have questionable benefit and some may paradoxically exacerbate the condition. Table 4 summarizes the panel's clinical experience and expert opinion concerning these management strategies.

Treatment

A defined skin care regimen should be established or continued. Clinicians should avoid products containing chlorhexidine gluconate, alcohol, or perfumes because they can be absorbed by damaged skin. Measures to ensure continued drying of the skin fold is a primary treatment strategy. Perhaps, due to the fact that ITD is common and occurs in patients of all ages, there are a wide variety of treatment options noted in the literature. Recommended treatments include wet tea bags,[65] Domeboro soaks,[77] hydrocolloid dressings,[78] diluted vinegar,[79] and Burow's solution.[80] These remedies have not been tested in controlled studies and may potentially prove harmful. The lack of empirical evidence could explain why McMahon and colleagues,[62] who interviewed nurses about ITD treatment, found that 16.5% recommended talcum powder while 15.7% recommended avoiding it.

Mistiaen and coworkers[63] completed a systematic literature review of ITD in large skin folds, noting that most studies included only small subsamples of ITD patients, mostly under the umbrella label of dermatomycoses. Only 6 studies were randomized controlled trials and most of them were conducted in Europe. While no recent research has been conducted, antifungal powders, lotions, or creams are recommended when candidiasis is present.

TABLE 4.

Expert Opinion: Ineffective or Potentially Harmful Strategies for Preventing or Managing ITD

Intervention	Expert Opinion Concerning Feasibility
Use absorptive talc or cornstarch-based powder to reduce moisture in skin fold.	These topical treatments have no proven benefit and may facilitate growth of fungal species in the skin fold.[86]
Use antiperspirants in skin folds	Untested remedy, these products were not developed or tested in large skin folds.
Place sterile gauze (4 × 4, 5 × 9, 8 × 10) in skin fold to absorb moisture.	They are ineffective because they do not effectively translocate the moisture, which is necessary to keep the skin dry.
Place bed or bathing linens (eg, towels, pillowcases, draw sheets) in skin folds to absorb moisture.	These linens absorb moisture but do not allow it to evaporate, which paradoxically exacerbates skin hydration and subsequent damage.
Place paper towels in the skin fold to absorb moisture.	Paper towels initially absorb moisture but do not allow it to evaporate, which paradoxically exacerbates skin hydration and subsequent damage.
Various home remedies have been recommended for preventing or treating ITD including placing wet tea bags in skin folds,[65] Domeboro soaks,[77] hydrocolloid dressings,[78] diluted vinegar,[79] and Burow's solution.[80]	None of these remedies are associated with clinical research demonstrating benefit.
Ask patients to disrobe for 30 minutes twice daily and expose skin folds to a fan, hairdryer, or light bulb.[64]	The efficacy of this intervention is unknown; the consensus group considers it impractical for most morbidly obese patients in the home setting and for patients in an acute or LTC facility.

Abbreviations: ITD, intertriginous dermatitis; LTC, long-term care.

Specific antifungals include the imidazoles, terbinafine, and ciclopirox.[52] A "dusting" of topical antifungal powders should be prescribed; when applied in heavy amounts, the skin moisture causes the powder to cake in the skin fold. Topical steroids should be used cautiously since they can promote candidal growth.[81]

Surgical intervention is uncommon, but removal of redundant tissues may be undertaken in selected cases to reduce skin fold friction and promote dryness. Chadbourne and colleagues[82] conducted a meta-analysis of reduction mammaplasty outcomes in 4173 patients. They reported that the frequency of intertrigo decreased from a mean of 50.3% to 4.4% following reduction mammaplasty.[82] Surgical removal of redundant skin may be performed in patients who have undergone significant weight loss or bariatric surgery. Although ITD is not typically a primary indication for these procedures, we have noted that removal of redundant skin can confer an unintended, and beneficial, side effect. Orthodontics or orthodontic surgery to reposition the jaw may be an indication when severe skeletal retrognathia leads to a pronounced labiomental fold, resulting in chronic and symptomatic labiomental ITD.[83]

Summary

Though the etiology of IAD and ITD differs, both are forms of MASD that typically begin with maceration and inflammation and may progress to denudation and cutaneous infection if left untreated. ITD and IAD can often be prevented with routine application of a defined skin care regimen combined with interventions designed to prevent friction and moisture within skin folds and to minimize contact with urine or stool. Aggressive preventive interventions should be focused on both patients deemed at risk for ITD, including morbidly obese persons, and those deemed at risk for IAD, including persons with urinary or fecal incontinence.

KEY POINTS

✔ MASD is defined as inflammation and erosion of the skin caused by prolonged exposure to various sources of moisture, including urine, stool, perspiration, wound exudate, mucus, or saliva.

✔ The forms of MASD outlined here are IAD and ITD, which involve similar mechanisms and pose the risk of skin breakdown.

✔ Due to a lack of clinical data on the efficacy of assessment, prevention, and treatment protocols related to IAD and ITD, the authors have prepared this consensus document.

✔ To prevent MASD, clinicians should be vigilant both in maintaining optimal skin conditions and in diagnosing and treating minor cases of IAD or ITD before progression and skin breakdown occur.

Glossary Terms

Cleansing: a specialized form of washing the skin to remove debris from (1) at-risk areas of skin and (2) the skin of at-risk patients.

Friction: force generated when 2 surfaces rub together; may be produced by rubbing of (1) skin surfaces or (2) skin and incontinence containment device.

Incontinence-associated dermatitis (IAD): inflammation of the skin associated with exposure to leaked urine or stool.

Intertriginous dermatitis (ITD): inflammation of skin folds from skin-on-skin friction caused by moisture trapped in the folds.

Maceration: softening of tissue by soaking until connective fibers can be teased apart. Macerated tissue often exhibits a lighter-colored appearance.

Moisture-associated skin damage (MASD): inflammation and erosion of the skin caused by prolonged exposure to various sources of moisture and its contents, including urine, stool, perspiration, wound exudate, mucus, or saliva.

Occlusion: reduction or prevention of evaporation that can occur with a device (eg, containment briefs, absorptive pads), thus precluding the drying of the skin.

Washing: cleaning of intact skin; with skin at risk for IAD or ITD, typically involves cleaning with a bathing product.

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