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Incontinence-Associated Dermatitis:

A Comprehensive Review and Update

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In 2009, a multinational group of clinicians was charged with reviewing and evaluating the research base pertaining to incontinence-associated dermatitis (IAD) and synthesizing this knowledge into best practice recommendations based on existing evidence. This is the first of 2 articles focusing on IAD; it updates current research and identifies persistent gaps in our knowledge. Our literature review revealed a small but growing body of evidence that provides additional insight into the epidemiology, etiology, and pathophysiology of IAD when compared to the review generated by the first IAD consensus group convened 5 years earlier. We identified research supporting the use of a defined skin care regimen based on principles of gentle perineal cleansing, moisturization, and application of a skin protectant. Clinical experience also supports application of an antifungal powder, ointment, or cream in patients with evidence of cutaneous candidiasis, aggressive containment of urinary or fecal incontinence, and highly selective use of a mild topical anti-inflammatory product in selected cases. The panel concluded that research remains limited and additional studies are urgently needed to enhance our understanding of IAD and to establish evidence-based protocols for its prevention and treatment.

Introduction

In 2005, a panel of clinical experts and researchers based in the United States convened a consensus conference that focused on skin damage associated with exposure to stool or urine; the results of the conference were published in 2007.[1] They advocated labeling the condition incontinence-associated dermatitis (IAD), which was defined as erythema and edema of the surface of the skin, sometimes accompanied by bullae with serous exudate, erosion, or secondary cutaneous infection. Since that conference, recognition of IAD as a distinct form of skin damage has grown among clinicians, and a small but growing body of research is now available to fill gaps in knowledge about its epidemiology, etiology, pathophysiology, prevention, and treatment.

In 2010, a second international consensus conference was convened, consisting of clinicians and researchers from the United States, the United Kingdom, and Western Europe. This consensus group was charged with reviewing and synthesizing the research base pertaining to IAD and with synthesizing this knowledge into best practice recommendations based on existing evidence. Two articles will summarize the results of this consensus conference: The first will focus on updating knowledge of IAD and identifying gaps in our knowledge, and the second will focus on clinical aspects of IAD and its management including challenges in assessment and differential diagnosis, prevention and treatment, cost issues, and educational challenges.

Nomenclature: Acceptance of the Term IAD

The 2005 consensus group recognized absence of a consistent term for identifying skin damage caused by exposure to stool or urine.[1] Instead, a review of the literature

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TABLE 1.

Prevalence of Incontinence-Associated Dermatitis: 2005-2011

Reference	N	Health Care Setting	Incontinence Type	Method of Measurement	Prevalence, %
Junkin and associates[6]	976	Acute care	Urinary and fecal incontinence	Direct observation	27
Bliss and associates[4]	10,215	Long-term care	Urinary and fecal incontinence	Review of electronic database	5.7
Defloor and associates[5]	19,964	Long-term care	Urinary and fecal incontinence	Direct observation	5.7
Arnold-Long and Reed[10]	171	Long-term acute care	Urinary and fecal incontinence	Direct observation	22.8
Beeckman and associates[11]	141	Long-term care	Urinary and fecal incontinence	Direct observation	22.5
Junkin and Selekof[7]	608	Acute care	Urinary and fecal incontinence	Direct observation	20

identified a variety of terms including perineal dermatitis, moisture lesions, and diaper dermatitis. The panel selected IAD because it fits within the taxonomy used by dermatologists to describe this type of skin damage, it recognizes that IAD extends well beyond the perineal skin, and it avoids confusing and potentially pejorative labels such as diaper dermatitis. Consensus panel members attending the 2010 meeting concurred that our experience as clinicians, researchers, and educators supported increasingly widespread acceptance of the term IAD. A review of the electronic database MEDLINE using the key terms incontinence dermatitis or incontinence-associated dermatitis between the years 2007 and 2011 revealed 38 references (incorporating original research reports, case studies, and review articles) in both nursing and medical literature, reflecting a small but growing professional literature focusing on this topic. In addition, the combined National Pressure Ulcer Advisory Panel/European Pressure Ulcer Advisory Panel International Clinical Practice Guideline for Pressure Ulcer Prevention recognized the term IAD as a distinct condition that should be differentiated from pressure ulcers or skin tears.[2] The term IAD was also recognized by a clinical expert panel convened to review current knowledge about moisture-associated skin damage, a term used to describe a variety of clinical disorders occurring with excessive exposure to effluents including urine, liquid stool, perspiration, wound exudate, and output from an intestinal stoma or urostomy.[1,3] Collectively, these observations strongly suggest that IAD is an acceptable term to both clinicians and researchers and this term will be used in both publications authored by this international panel.

Epidemiology

Members of the first consensus panel noted a paucity of studies reporting the prevalence of IAD. Reported prevalence rates varied widely, with estimates ranging from 5.6% to 50%.[1] One of the largest of these studies used data from the Minimum Data Set to calculate the prevalence of IAD in 10,217 nursing home residents from 31 states; they reported a prevalence rate of 5.7%.[4] Defloor and associates[5] found a similar IAD prevalence figure of 5.7% when using direct observation to assess the perineal and perigenital skin of 19,964 hospitalized patients in Belgium. Junkin and associates[6] used visual inspection on a single day to measure prevalence in 976 patients in 3 acute care facilities in the United States; they reported a prevalence rate of 27%.

Multiple studies have been published since the 2005 consensus panel met that enhance our understanding of the prevalence and incidence of IAD (Tables 1 and Tables 2). Junkin and Selekof[7] measured the prevalence of IAD in a community-based and teaching hospital located in the eastern and Midwestern United States, respectively. Subjects were aged 4 years and older and were cared for in 48 inpatient units, including critical care units. The diagnosis of IAD was based on visual inspection of the perineal and perigenital skin during a single day by data collectors who had been trained in assessment of IAD and differential diagnosis of IAD versus other common causes of skin damage in this area including partial-thickness pressure ulcers. Fecal or urinary incontinence was observed in 120 of 608 subjects (19.7%) and the reported prevalence of IAD was 20%.

Driver[8] evaluated 131 patients with fecal incontinence in a critical care unit and reported a 50% prevalence of IAD, using a 2-step skin care regimen with some flexibility in product selection and a 19% prevalence in a group of 177 patients managed with a premoistened cloth combining a cleanser, moisturizing agents, and skin protectant.

Bliss and associates[9] reported the prevalence of IAD in a group of 185 community-dwelling adults with fecal incontinence (72%) or dual fecal and urinary incontinence (28%). Slightly more than half (52%) reported experiencing IAD; 23% stated that they experienced occasional problems, 10% reported frequent problems, and 5% reported chronic skin damage.

Arnold-Long and Reed[10] evaluated the prevalence and incidence of IAD in 171 adult patients managed in a long-term acute care facility located in the Midwestern United States. Diagnosis was based on direct observation of the skin; assessment was completed at the time of admission to the facility. Incontinence-associated dermatitis was found in 39 patients on admission, yielding a 22.8% prevalence rate. In a nursing home study in Belgium, Beeckman and coworkers[11] identified a group of 141 out of 464 nursing home patients (32.9%) deemed at risk for IAD; visual assessment revealed a 22.5% IAD prevalence.

TABLE 2.

Incidence of Incontinence-Associated Dermatitis: 2005-2011

Reference	N	Health Care Setting	Incontinence Type	Method of Measurement	Period of Observation	Incidence, %
Bliss and associates[19]	981	Long-term care	Urinary and fecal incontinence	Direct observation	6 weeks	3.4
Bliss and associates[12]	45	Critical care	Fecal incontinence	Direct observation	Duration of stay in the critical care unit: median time to onset of 4 d	36
Driver[8]	Phase 1: n = 131	Critical care	Fecal incontinence	Direct observation	Phase 1: Duration of stay in critical care unit: <14 d	Phase 1: 50
	Phase 2: n = 177				Phase 2: Duration of stay in critical care unit: >14 d	Phase 2: 19[a]
Arnold-Long and Reed[10]	132	Long-term care	Urinary and fecal incontinence	Direct observation	Duration of stay: Median time to onset 13.5 d	7.6

[a]Researchers implemented defined skin care regimen, using 3-in-1 washcloth with skin cleanser, moisturizers, and dimethicone-based skin protectant during phase 2 of the study.

Though evidence remains limited, several studies have now been completed to improve our knowledge of the incidence of IAD. Bliss and associates[4] evaluated IAD incidence in a group of 981 residents in 16 nursing homes in 15 states. Residents were on a skin damage prevention regimen and observed over a 6-week period and IAD was diagnosed by visual inspection. Incontinence-associated dermatitis developed in 33, yielding an incidence rate of 3.4%. In a separate study, Bliss and associates[12] evaluated IAD in 45 adult patients admitted to surgical/trauma critical care units in 3 hospitals located in the Midwestern United States. Incontinence-associated dermatitis was diagnosed based on visual inspection of the patient's perineal, groin, and thigh skin. Assessments were completed by the study investigators who were trained by an experienced WOC nurse. Sixteen patients developed IAD during the observation period, indicating an incidence rate of 36%. Arnold-Long and Reed[10] evaluated new cases in 132 patients in a long-term acute care facility found to be free of the condition at the time of admission. Ten developed IAD during the data collection, yielding an incidence rate of 7.6%.

The second consensus panel identified only one study that reported the incidence of cutaneous candidiasis among patients with IAD. Junkin and Selekof[7] reported that 18% of a group of 198 hospitalized patients with urinary, fecal, or double urinary and fecal incontinence had evidence of IAD and secondary cutaneous candidiasis based on visual inspection. Bliss and associates[9] administered the Fecal Incontinence Quality of Life Tool to 185 community-dwelling adults with fecal incontinence or dual fecal and urinary incontinence; 18% reported experiencing a fungal rash.

Participants in the first consensus conference concurred that our knowledge of the natural history of IAD is especially limited, and experts participating in the second consensus conference also found little research in this area. However, findings from 3 studies provide some initial insights into time of onset of IAD. In their study of 981 nursing home residents, Bliss and associates[13] reported that the median time to onset of IAD was 13 days (range, 6-42 days). Arnold-Long and Reed[10] reported a similar time to onset of 13.5 days (range, 3-days) in a long-term acute care facility. In contrast to these findings, Bliss and associates[12] reported a median time to onset of IAD of 4 days (range, 1-6 days) in a group of 45 critically ill patients.

Visscher and coinvestigators[14,15] examined the natural history of diaper dermatitis in 31 healthy, full-term neonates in 2 studies that provide some insight into the response of human skin after its first exposure to urine and stool. In the first, 2 research nurses observed and assessed the skin 25 minutes after diaper removal to allow evaporation from the surface of the skin; visual inspections occurred on days 1, 4, 7, 14, 21, and 28 after delivery. When the skin was observed at day 1, none of the neonates had evidence of erosion or rashes and 6 (19%) had mild erythema. Subsequent observations revealed that 70% developed erosion or rashes by day 7, and 90% had evidence of diaper dermatitis by day 14. The second study compared skin hydration on the perineal and perigenital skin to the skin of the volar surface of the arm, using the same observation schedule. Differences in the hydration of the skin of the arm and perineal area emerged at day 7. Specifically, the perineal and perigenital skin demonstrated greater hydration and a higher pH (more alkaline) when compared to the skin of the arm. Bliss and colleagues[12] evaluated healing of IAD in 45 critically ill patients; the median time from diagnosis to healing was 11 days (range, 1-19 days).

While these studies provide additional knowledge of the epidemiology of IAD, variability in reported incidence and prevalence rates persists. Based on our review of the literature and clinical experience, the consensus panel hypothesizes that observed variability is probably attributable

to differences in care setting as well as the prevalence underlying type of incontinence (urinary incontinence, fecal incontinence, or double urinary and fecal incontinence). Variability in study findings may also be attributable to the lack of a fully developed assessment tool to measure IAD, which remains a research priority.

Etiology and Pathogenesis

A small number of studies were identified that evaluated etiologic factors and the pathogenesis of IAD. Histopathologic analysis of IAD reveals inflammation of the upper dermis resulting in erythema.[16,17] Areas of erosion are associated with dermal and epidermal proliferation and vascular occlusion resulting in necrobiosis and partial tissue destruction. These findings differ from histopathologic analysis of pressure ulcers that revealed ischemia as the predominant factor[17] and ultrasonic imaging that revealed subdermal pockets of edema in deeper tissues nearer underlying bone.[18] These findings support clinical observations that IAD is the result of top-down damage to the skin when exposed to stool or urine whereas pressure ulcers primarily result from bottom-up damage when pressure or shear leads to destruction of deeper tissues such as muscle.

Findings from clinical studies evaluating urine versus stool as an etiologic factor for IAD are mixed. Bliss and colleagues[4,19] reported that fecal incontinence and double urinary and fecal incontinence were associated with an increased likelihood of IAD in nursing home patients and urinary incontinence was not. In contrast, Junkin and associates[6] and Junkin and Selekof[7] reported that hospitalized patients with fecal incontinence were no more likely to experience IAD than were patients with urinary incontinence alone. Urine promotes irritation by overhydrating exposed skin, increasing its pH, and creating more friction as the skin moves against absorptive incontinence devices, clothing, or bedding. Urine is also postulated to diminish tissue tolerance to friction, shear, or pressure. Mayrovitz and Sims[20] studied healthy adult volunteers and found that wetting the skin with synthetic urine impaired its elasticity, temperature, and blood flow when challenged by an increased pressure load when compared to dry skin challenged with a similar load.

Early studies of babies in diapers identify water as a primary agent in the etiology of IAD. Berg's model of "diaper dermatitis" postulates that water penetration of the skin leads to increased permeability to irritants (including fecal enzymes) and chemicals and increased friction coefficient (leading to raised susceptibility to mechanical abrasion).[21] Studies of patch testing on hairless mice demonstrate that urine alone acts as a mild skin irritant, stool alone acts as a moderate irritant, and urine and stool together are highly irritating to skin.[22]

Clinical research strongly suggests that stool acts as an etiologic agent.[7,12,13,23] Patch testing reveals that stool irritates skin in the perianal area and volar surface of the forearm.[24] Research in humans and animal models demonstrates that this irritation is at least partially attributed to the presence of digestive enzymes, mainly lipases and proteases.[21,22] Although the mechanisms that lead to irritation are not completely understood, evidence suggests that proteases and lipases have a synergistic effect such that the keratin in the stratum corneum is rendered more vulnerable to lysis by the proteases when lipases are also present.[25] Enzymatic activity and irritation are also exacerbated in the presence of an alkaline pH and common in patients with urinary and fecal incontinence.

Though research is lacking, clinical experience overwhelmingly suggests that liquid fecal matter is more irritating than solid stool. The irritating qualities of liquid fecal materials stool may occur because liquid stool usually comes into contact with a larger area of skin and because it contains more bile salts and pancreatic lipases that are especially irritating to the skin.[21,22] These enzymes also increase the skin's vulnerability to irritation by other low-molecular-weight irritants, including ammonia.

Associated Factors

The role of colonization or infection with bacterial or fungal species in the pathogenesis of IAD is not well understood. Two microorganisms, *Candida* sp. and *Staphylococcus aureus,* were identified in a study comparing the microbiology of the skin in a group of 28 infants with diaper dermatitis to 48 infants with healthy skin.[26] *Candida* sp. was significantly more common in the perianal skin, inguinal fold, and mouth of infants with diaper dermatitis as compared to infants without IAD ($P < .0003$). In contrast, differences in the prevalence of colonization with *Staphylococcus aureus* were not statistically significant ($P > .34$). While colonization with either organism is not pathognomonic for IAD, high colony counts of both microorganisms were associated with more severe cases, suggesting that a process of critical colonization may occur, similar to the phenomenon observed in some patients with indolent chronic wounds. From a clinical perspective, cutaneous infections with bacterial species are not associated with IAD, but infections with *Candida albicans*, based on visual inspection of a candidal rash, were observed in 18% of the perineal and perigenital skin.[7]

The role of absorptive products in the pathogenesis of IAD is also not entirely understood. Research shows that covering the skin with an absorptive product in the absence of exposure to urine or stool increases perspiration, raises transepidermal water loss (TEWL), and results in a more alkaline pH at the surface of the skin.[27] Absorptive products also may contribute to irritation by provoking a hypersensitivity response, but documented cases are rare.[28-32] Clinical experience suggests that the risk of IAD appears to be related to occlusion, combined with prolonged contact with urine or stool if the device is allowed

to become saturated with urine or soiled with stool. Fader and coinvestigators[33] compared the effect of frequent *versus* infrequent pad changes (every 4 hours vs every 8 hours) on the perineal and perigenital skin of a group of 81 nursing home patients and found that both groups had similar evidence of erythema, implying that simply changing a pad more frequently may not reduce the incidence of IAD. However, measurements of skin hydration via TEWL showed that the skin of those on the less-frequent pad changing regimens was significantly wetter than when their pads were changed more frequently. In addition, residents who were managed with 4-hour pad changes were less likely to have partial-thickness skin damage (rated as stage II pressure ulcers) than were residents managed by brief changes every 8 hours, although this finding did not reach statistical significance. More work into the effect of the frequency of pad changing is therefore needed to establish the optimum regimen for patients.

Although definitive evidence is lacking, the design of wrap around briefs may influence the likelihood that an individual will develop IAD.[34] Disposable infant diapers containing absorbent polymers and cellulose fluff have been found to reduce pH, increase the volume of urine absorbed prior to saturation, and diminish the incidence of diaper dermatitis when compared to reusable cloth diapers. Incorporation of a microporous film that increased the air permeability of a disposable infant diaper exerted a similar effect on the incidence of IAD.[35] Beguin and associates[36] described developing and testing an incontinence brief for adults that incorporated a cellulose fiber that reduced pH at the surface of the skin to a mean value of 4.6 as compared to 7.1 for standard briefs. The brief was also modified to include side panels made of a nonwoven material that significantly increased the air permeability from 0 to 1200 L/m^2/s and water vapor transmission from 0 to 3500 g/m^2/d. A single-group before-after study in 12 patients with IAD resulted in resolution of 8 (67%).[36]

Differentiation of IAD From Pressure Ulcers

The assessment of IAD, including risk assessment and differentiation from other forms of skin damage such as pressure ulcer or skin tear, remains a challenge for both expert and nonspecialty nurses. Some authors have proposed circumventing this challenge by classifying and managing all such lesions as decubitus (lying down) ulcers, and basing prevention or treatment on this broader diagnosis.[17,37] Several members of the consensus group participated in debates held at continuing education seminars focusing on whether clinicians should even attempt to differentiate IAD from pressure ulcers. However, the 2010 consensus panel identified several persuasive rationales for differentiating skin damage based on its underlying etiology as IAD, pressure ulcer, or skin tear.

The most clinically relevant argument for differentiating IAD versus pressure ulcer is the impact of accurate prevention and treatment. Patients deemed at risk for a pressure ulcer are often placed on a pressure redistribution surface, and protective garments or pads may be used for patients deemed at risk for skin tears. While a defined skin care regimen is advocated for all patients, only those at risk for IAD require addition of a skin protectant to their regimen, often combined with aggressive means of preventing or transiently containing urinary or fecal incontinence. In contrast, the emphasis of a defined skin care regimen for the person deemed at risk for skin tears focuses on application of a moisturizer, along with protective devices placed on the elbows or knees.[38] Many patients are at risk for all of these conditions, and individualization of care based on risk is strongly recommended based on both clinical and economic considerations. Regulatory, reimbursement, and legal policies and guidelines also favor differentiation of skin damage based on underlying etiology. For example, differentiating IAD from a pressure ulcer influences quality indicators of care, the likelihood of reimbursement, and risk for subsequent litigation. Finally, correct identification of skin damage influences classification when measuring national or international benchmark data for quality-sensitive indicators such as facility-acquired pressure ulcers.[39]

The 2005 consensus group identified several characteristics designed to aid clinicians in differentiating IAD from skin tears or various categories of pressure ulcers (Table 3).[1] Black and colleagues[4] included a table in their second consensus statement that provides a useful guide for differentiating IAD from stage I and II pressure ulcers. In addition, the European Pressure Ulcer Advisory Panel,[40] Defloor and associates,[41] Gray and associates,[42] and Beeckman's group[43] have published resources to aid clinicians in the differentiation of IAD and pressure ulcers. Black and colleagues[4] emphasized the significance of the patient's medical history when determining the etiology of perineal skin damage. This includes regular skin assessment, including a pressure ulcer risk assessment, and identification of fecal or urinary incontinence, as well as other sources of moisture that may impact the risk for moisture-associated skin damage such as excessive perspiration or a wound with high-volume exudate. Despite these models, the consensus panel acknowledges that patient history and skin observation alone cannot clearly distinguish one skin condition from another (specifically IAD from pressure ulceration) where the condition is present on an area of skin on which pressure ulcers may form and the patient has urinary and/or fecal incontinence.

Research suggests that nursing students and nonspecialty professional nurses experience difficulty differentiating IAD from pressure ulcers.[37,43-45] Kottner and Halfens[37] evaluated interrater reliability based on outcomes of 339 nursing assessments of patients in a home health care setting. While nurses agreed on presence or absence of

TABLE 3.

Visual Inspection for Differentiation of IAD From Pressure Ulcers or Skin Tears

Characteristic	IAD	Pressure Ulcer
Color	Bright red in persons with lighter skin tones and subtle red in persons with darker skin tones	Deep red (maroon) to reddish or bluish purple in suspected deep tissue injury
Location	Perineal or perigenital skin, especially near anus, in skin folds or underneath absorptive incontinence product such as underpad or body-worn brief	Typically found over bony prominence
Lesions	One or more islands or erosion to extensive denudation of epidermis and dermis	Varies from partial-thickness to full-thickness wounds
Borders	Diffuse	Demarcated
Necrotic tissue	None	Black eschar or yellow slough may be present
Exudate	None or clear, serous exudate	Volume varies; high-volume purulent exudate seen in some cases
Symptoms	Burning pain, itching	Pain and itching, may be exacerbated by dressing change

Abbreviation: IAD, incontinence-associated dermatitis.

moisture-associated skin damage in 95% of cases, the interrater reliability, when queried about the primary cause of the damage (ie, exposure to urine, stool, perspiration, or wound exudate), was 0.67 (95% confidence interval, 0.61-0.73).

Research also suggests that education of nurses improves their ability to differentiate IAD from pressure ulcers. Beeckman and coworkers[43] evaluated 212 professional nurses and 214 nursing students in the final year of their education and found both groups experienced difficulty differentiating stage II pressure from IAD with erosion and from skin damage attributable to both moisture and pressure. However, differential diagnosis skills improved significantly in both groups following education using either the Pressure Ulcer Classification (PUCLAS) e-learning program or a traditional lecture/PowerPoint presentation format; student nurses gained greater benefit from the PUCLAS e-learning technique. The PUCLAS education tool was further tested on 1217 professional nurses practicing in Belgium, Holland, Portugal, and the United Kingdom. Baseline evaluation found that 55.5% of photographs were identified correctly; following administration of the PUCLAS, 70.7% of IAD photographs were classified correctly as compared to a 35.5% rate of correct classification in a control group.

Mahoney and coworkers[39] evaluated differential diagnosis of IAD versus pressure ulcers and skin tears in a group of 100 nurses with clinical experience and formal education in wound care. Respondents viewed 9 photographs of skin damage involving either the intergluteal cleft or the fleshy aspects of the buttocks. The level of agreement was calculated with 100% representing perfect agreement and 0% reflecting no agreement. The level of agreement was greatest for skin damage with either incontinence or pressure as the sole etiologic factor (62% and 70%, respectively). In contrast, Fleiss kappa for interrater agreement for photographs of skin damage to the intergluteal cleft was 0.1558 (99% confidence interval, 0.1467-0.1670) and the Fleiss kappa for skin damage involving the fleshy surface of the buttocks was 0.1227 (99% confidence interval, 0.1081-0.1372). When responses were pooled for all photographs, analysis revealed that even experienced wound care nurses achieved only "slight agreement" based on visual inspection of photographs alone.

Instruments for Assessing IAD Severity and Risk

Three instruments for evaluation of IAD were identified by the 2005 consensus group: the Perineal Assessment Tool,[46] Perirectal Skin Assessment Tool,[47,48] and the IAD Skin Condition Assessment Tool.[49] The Perineal Assessment Tool is designed to assess risk for IAD based on type of irritant (solid stool, liquid stool, urine, etc), the duration of contact, condition of the perineal skin, and presence of contributing factors such as *Clostridium difficile* infection, poor nutrition, or tube feeding. Nix[46] established content validity for this instrument, using a group of WOC nurses, and its interrater reliability was found to be 87%. The Perineal Dermatitis Grading Scale is a descriptive instrument; items evaluate erythema, skin integrity, area of affected skin measured in centimeters, and associated symptoms including itching and pain. A review of CINAHL and MEDLINE electronic databases from 2002 to 2011 did not identify any published study establishing this instrument's validity or reliability. The IAD Skin Condition Assessment Tool is designed to describe IAD and measure its severity; items include the area of affected skin, erythema, and presence of erosion. A review of CINAHL and MEDLINE databases up to 2011 did not identify any study evaluating this tool's validity or reliability.

Initial validation of a new tool, the incontinence-associated dermatitis and its severity (IADS) instrument, was reported in 2010.[50] The IADS is a descriptive tool that ranks the severity of IAD. The location of IAD is identified based on 13 locations including the perianal skin, buttocks, genitalia, upper thigh, and skin folds between

genitalia and thigh. The magnitude of erythema is based on a 3-point ordinal scale that varies from none to pink to red; the tool includes 2 pallets designed to aid the clinician in evaluating persons with lighter and darker skin tones. Erosion and rash are scored as present or absent. Borchert and colleagues[50] evaluated content validity based on responses of 247 WOC nurses, and criterion validity was tested by comparing IADS scores of testers with 2 WOC nurse experts and the principal investigator. Interrater reliability was tested in 3 groups: nurses' assistants and staff nurses at a single facility in the Midwestern United States and WOC nurses. Preliminary findings indicated that the IADS had good interrater reliability but further studies are needed to establish reliability in various clinical settings. A literature review did not reveal any studies that have yet used the IADS, but the developers report that the instrument is being used in several studies now underway (D. Bliss, oral personal communication, 2011).

Technological Assessment

The challenges associated with distinguishing stage I and II pressure ulcers from IAD have led several researchers to explore the potential of several technologies to aid clinicians when assessing visible or suspected skin damage in the perineal and perigenital area. For example, intermediate frequency ultrasound, varying from 10 to 20 MHz, has been used to create images of erythematous skin and subcutaneous tissues.[18,51,52] Several findings have been associated with an increased likelihood of development of a pressure ulcer including loss of visible layering underneath the dermis, discontinuous fascia, and hypoechoic areas.[51] Quintavalle and associates[18] reported pockets of edema in the subdermal tissues of patients at risk for pressure ulcers based on Braden Scale scores of 18 or less. These findings support the hypothesis that pressure ulcers are caused by pressure and resulting in ischemic damage in deeper tissues near the bone whereas tissue damage caused by friction creates superficial damage and a partial-thickness wound.

Bates-Jensen and coinvestigators[53] used a handheld device that measures subepidermal moisture on the sacrum and buttocks to identify progression to stages I and II pressure ulcers. The device calculates electrical capacitance at the surface of the skin, which reflects localized edema in epidermal and subepidermal tissues. Results are quantified as dermal phase units from 0 to 999. Higher readings indicate localized edema and were found to predict 26% to 32% of stage I pressure ulcers within a 7-day period.[53,54] Elevated readings were also found to differentiate between erythema and early ischemic insult categorized as a stage I pressure ulcer.

Findings from these studies suggest that ultrasonic imaging may be used to aid clinicians to more accurately differentiate deep tissue injury at high risk for progression to a pressure ulcer from the erythema associated with IAD. However, consensus group members also commented on limitations associated with clinical application of this technique including equipment cost and the technical expertise required for generating and interpreting high-resolution ultrasonic images. Measurement of subdermal moisture has the potential to avoid these limitations, but further study is needed to determine whether either of these technologies will prove clinically useful for differentiation of IAD versus partial-thickness pressure ulcers.

A number of other techniques have also been evaluated for their potential role in aiding clinicians when evaluating perineal skin damage including TEWL, color, temperature, and elasticity.[52,55] Transepidermal water loss is a measurement of the volume of water that diffuses from the body's internal environment, through the moisture barrier, and into the atmosphere via passive diffusion. Two principal techniques have been used to measure TEWL: open and closed chamber.[55] Open-chamber devices measure water vapor pressure via 2 thermistors and hygrosensors (capable of measuring moisture), mounted at 2 heights within a hollow sensor. Measurements using this device are typically based on standardization of temperature and humidity, a level of control that is not feasible in the clinical setting. Condenser chamber TEWL systems use a cooled closed chamber to determine water vapor flux. Alternatives to these traditional approaches include an unventilated chamber system that uses a battery-operated device with a humidity sensor that is placed on the skin for 20 seconds for measurement,[55] or optothermal radiometry that measures TEWL via a neodymium or erbium YAG lasers mounted in a hand-held probe.[56,57] While these technologies continue to evolve, there is insufficient evidence to determine whether measurement of TEWL is useful in identifying IAD or differentiating this condition from suspected deep tissue injury or stage I or II pressure ulcers.

Prevention and Treatment

The 2007 consensus panel found few studies evaluating the effectiveness of different perineal skin care protocols for the prevention and treatment of IAD.[1] Key prevention recommendations comprised following a defined skin care regimen that includes gentle cleansing, moisturization, and application of a skin protectant or moisture barrier. Treatment goals include protecting the skin from further exposure to irritants, establishing a healing environment, and eradicating any cutaneous infection. Further recommendations for treatment were mainly based on the opinions and experiences of the expert panel. For mild to moderate IAD, a structured skin care regimen with the addition of a daily application of a skin protectant was recommended. A structured skin care program combined with a more regular application of a skin protectant was advocated for more severe IAD. The 2007 consensus panel concluded that additional and larger trials were needed to

fully determine the validity of these recommendations regarding prevention and treatment.

To date, few rigorously performed research studies have addressed the effectiveness of different skin care regimens to prevent or treat IAD (Table 4). A number of studies have compared the use and effect of different types of skin care, but design weaknesses are common, with small sample size a frequent limitation. The panel recognizes that IAD prevention and treatment remain a fertile area of investigation, which may have been lagging behind other evidence-based topics because of the lack of agreed objective and measurable outcomes. Robust methods for measuring hyperhydration of the skin, wetness, barrier function damage, and bacterial load are needed to pave the way for more reliable research about mechanisms of IAD healing. The panel suspects that, in time, some of these outcomes may become more easily measurable and provide a greater rationale for the effectiveness of prevention and treatment interventions.

Panel members agreed that both prevention and treatment of IAD must include a consistently applied and well-defined skin care regimen, including (1) gentle perineal cleansing, (2) moisturization, and (3) the application of a skin protectant or moisture barrier. The addition of antifungal products, steroidal-based topical anti-inflammatory products, and topical antibiotics to treat IAD is recommended only in specific situations. Interventions such as the use of absorptive or containment products and/or indwelling devices are also indicated in specific situations to support both prevention and treatment of IAD. These recommendations will be discussed and supported by recent evidence where appropriate.

Defined Skin Care Regimen

A growing body of evidence supports the importance of a consistently applied, defined skin care regimen to prevent IAD.[11,58-61] Although we found some variation in the components to be included in such a regimen, all of them included (1) perineal cleansing, (2) the application of a moisturizer, and (3) the application of a skin protectant. Beeckman and coinvestigators[11] reported a randomized controlled trial of 464 nursing home patients (including 141 deemed at risk for developing IAD) that compared a 3-in-1 washcloth containing a skin cleanser, moisturizers, and a 3% dimethicone-based skin protectant, was compared to usual care comprising washing with a pH neutral soap and water. The incidence of IAD was significantly lower in those receiving the defined skin care regimen using the washcloth after 120 days of treatment (8.1% in the intervention group vs 27.1% in the group managed with soap and water, $F = 3.1$, $P = .003$). Bale and associates[59] completed a pre-post trial of 164 patients, using a defined skin care regimen that included a perineal skin cleanser, moisturizer, and polymer acrylate skin protectant. Introduction of this defined skin care regimen resulted in a statistically significant reduction in IAD incidence (4.7% vs 25.3%) and reduced severity of skin lesions when compared to the unstructured protocol used at baseline. In a double-blinded, randomized, controlled trial in 190 patients,[58] an exercise and incontinence intervention achieved significant improvement in urinary and fecal incontinence and skin wetness (limited to the back distal perineal area) when compared to usual care.

Perineal Cleansing

Evidence shows that perineal skin cleansing should involve a product whose pH range reflects the acid mantle of healthy skin (pH between 5.4 and 5.9).[62] Increasing the pH of the skin may enhance the risk of skin colonization by potentially pathogenic microorganisms, which may ultimately invade the skin should the barrier function be disturbed.[63] The pH of normal soap is alkaline and ranges from 9.5 to 11.0.[64] The ability of soap to clean the skin is based on releasing free alkali and insoluble acid salts. The alkali pH of soap is produced by the hydrolysis of soap in an aqueous solution, by which a quantity of alkali is

TABLE 4.

Research on the Prevention and Treatment of IAD: 2004-2011

Reference	N	Study Design	Intervention	Outcome
Bale and associates[59]	164	Single-group before-after study	Defined skin care regimen with perineal skin cleanser and moisturizer, application of polymer acrylate barrier film skin protectant	Reduction in occurrence rate of IAD from 25.3% prior to intervention to 4.7% following intervention
Baatenburg de Jong and Admiraal[94]	40	Randomized controlled trial	Application of polymer acrylate skin protectant vs zinc oxide oil–based skin protectant	Less cost when polymer acrylate skin protectant compared to zinc oxide oil–based protectant
Beeckman and associates[11]	464	Randomized controlled trial	Defined skin care regimen with 3-in-1 washcloth containing skin cleanser, moisturizers, and dimethicone-based skin protectant	Significant reduction in IAD prevalence 8.1% vs 27.1% (intervention vs control group, $P = .003$), reduction in IAD severity using IAD Skin Condition and Assessment Tool was not statistically significant ($P = .06$)

Abbreviation: IAD, incontinence-associated dermatitis.

released, raising the pH of the water to about 10.0 to 11.0.[65] Ananthapadmanabhan and coworkers[66] demonstrated that higher pH (pH 10.0) solutions can increase stratum corneum swelling and alter lipid rigidity, thereby suggesting that cleansers with neutral or acidic pH, close to normal pH 5.5, may be potentially less damaging to skin.

Skin cleansers provide an alternative to soap and water when washing the perineal and perigenital skin.[63] They may reduce some of the adverse effects of soap, due to their chemical composition, and help to maintain a pH level that minimizes disruption of the skin's moisture barrier.[67] Many no-rinse skin cleansers are "pH balanced" in order to ensure that their pH is closer to that of healthy skin. Cleansers emulsify dirt and microorganisms on the skin surface so that they can be easily removed. During cleansing, there is a complex interaction between the cleanser, the moisture skin barrier, and skin pH. No-rinse skin cleansers combine detergents and surfactant ingredients to loosen and remove dirt or irritants; many also contain emollients and/or humectants to restore or preserve optimal barrier function. Therefore, the panel members agree that a no-rinse skin cleanser with a pH range similar to normal skin is preferred over soap and water for the prevention of IAD.

Rönner and associates[68] compared the ability of soap and water versus a no-rinse skin cleaner to remove transient microbial flora (*Escherichia coli* and *Staphylococcus aureus*) in healthy volunteers. The amount of residual bacteria was comparable for both washing procedures. Both washing procedures resulted in a low level of residual bacteria on the skin. The authors concluded that no-rinse skin cleansers could be safely used as an alternative to soap and water washing of fragile skin. In addition, 1 randomized controlled trial[69] and 1 crossover study[70] found no-rinse cleansers were more effective than soap and water in preventing incontinence-related skin problems. Three studies,[71-73] of which one was a randomized controlled trial,[72] found reduced skin erythema with the combined use of a no-rinse skin cleanser and a skin protectant compared to controls. Further research is required to evaluate the effectiveness of many nonsoap skin cleanser alternatives available.

If water and soap are needed to remove dirt or irritants, gentle cleansing is preferred over scrubbing techniques and a soft cloth is recommended to minimize friction damage. This recommendation is in agreement with Voegli,[63] who found that repeated washing with soap and water caused significant skin damage in 15 healthy volunteers receiving 6 different washing and drying techniques to the volar aspect of the forearm. Comparisons were made between washing with soap or water alone, and drying using a towel (rubbing and patting) or evaporation. The results showed that washing with soap and water and towel drying had a significant disrupting effect on the skin's barrier function. The authors also concluded that drying the skin by patting with a towel offered no advantage to conventional gentle rubbing as it leaves the skin significantly wetter and at greater risk of frictional damage. Panel members realize that more work is required to fully elucidate the role of drying techniques in the prevention of IAD. Therefore, they prefer no-rinse cleanser over towel drying for preventing IAD.

Skin cleansing should occur as soon as possible to limit contact with urine and stool. However, limited evidence suggests that a cumulative effect may exist with skin damage increasing as cleansing frequency with water and soap increases,[63] which might affect patients who experience frequent incontinent episodes. Therefore, the routine washing of patients following each episode of urinary incontinence needs to be reevaluated, and washing frequencies reduced where possible. Since fecal incontinence is implicated as a more powerful factor for development of IAD when compared to urinary incontinence alone, panel members advocate the importance of providing timely cleansing following an episode of fecal incontinence.

Moisturize

The barrier function of the skin depends on intercellular lipids as well as intact keratinocytes.[74-76] In the epidermis, keratinocytes bear an important responsibility for maintaining the skin structure and homeostasis.[76] Loss of water from the skin must be carefully regulated, a function dependent on the complex nature of the outer layer of the skin, the stratum corneum. The retention of water in the stratum corneum is dependent on the intercellular lipids orderly arranged to form a barrier (protective surface layer of lipids) to TEWL.[74] Epidermal keratinocytes provide the rigid stratified structure through a sophisticated differentiation program. Through a complex differentiation process, keratinocytes control skin features such as moisturization.[75]

Moisturization is designed to repair or augment the skin's moisture barrier, retain and increase its water content, reduce TEWL, and restore the lipid barriers' ability to attract, hold, and redistribute water.[77] Moisturizers contain varying combinations of emollients, occlusives, humectants, and water to achieve their beneficial effects.[78] Lipids are major components of emollients and include fats, waxes, or oils.[79] Sources of lipids include animal products such as lanolin (arising from sheep wool fat), petrolatum and dimethicone (arising from mineral oils), and vegetables (including nuts, seeds, and some fruits).[67] They exert their benefits through positive effects on the skin barrier, partially through improved repair and permeability,[80] resulting in better hydration. Some emollients also contain humectants, such as glycerine.[79] Humectants enhance water absorption from the dermis into the epidermis, and in humid conditions they support the stratum corneum to absorb water from the external environment. The expert panel agrees that moisturizers with a high lipid content (rather than a high water or humectant content) should be used in patients with hyperhydrated skin.

Routine use of moisturizers is essential in patients with dry skin because it replaces intercellular lipids and enhances the moisture content of the stratum corneum.[81-83] The panel also agrees that the application of a moisturizer is beneficial for dry skin because it promotes hydration of the skin and reduces the likelihood of other forms of cutaneous damage such as skin tears. However, it is important to keep in mind that moisturizer formulations can lead to allergic contact dermatitis, often as a result of preservatives, fragrances, or perfumes found within many commercial preparations.[84] Typical symptoms include stinging or burning sensations when the product is applied. Proper knowledge of moisturizers and scientifically based recommendations for the use are important.[85]

Apply Skin Protectant

A skin protectant primarily aims to prevent skin breakdown by providing an impermeable or semipermeable barrier on the skin, thus preventing penetration of water and biologic irritants found in stool and urine.[86] Evidence suggests that a defined skin care regimen for prevention of IAD must include the application of a skin protectant/barrier.[4,19] Multiple products and formulas are commercially available with both moisturizing and/or protecting/barrier capability. Many skin protectants are based on occlusive substances, such as petrolatum or dimethicone.[87] Moisturizers are not only used to restore the barrier function of the epidermis, but they also provide a soothing protective film.[77] Draelos[87] identified multiple over-the-counter skin care products, but also found commonality among 80% of the formulations.[87]

Currently used skin protectants include petrolatum-based ointments, dimethicone-based ointments, zinc oxide creams, oils, and liquid film-forming acrylates. Evidence suggests that commercially available skin protectants vary in their ability to protect the skin from irritants, prevent maceration, and maintain skin health. Hoggarth and coinvestigators[86] addressed 6 products' efficacy against insult from a known irritant (sodium lauryl sulphate), skin hydration potential, and maintenance of skin barrier and barrier efficacy against maceration in 18 healthy volunteers. The results indicated that each one of the products tested has different performance properties. Products containing petrolatum demonstrated protection against irritants and maceration and provided some skin hydration. Products containing dimethicone varied in protection against irritants and have good skin hydration potential and low barrier efficacy. Zinc oxide–based products showed protection against irritants but poor skin hydration and barrier properties to prevent maceration. The study concluded that only the water-in-oil petrolatum-based product performed effectively within all the parameters tested. This study suggests that skin barrier protection involves more than the inclusion of an active barrier ingredient.

As described previously, multiple products that act as cleanser, moisturizer, and protectant can be recommended. The expert panel agreed that a defined skin care regimen that reduces steps or staff time should be encouraged.[1] Furthermore, a single intervention has the potential to maximize time efficiency and to encourage adherence to the skin care regimen.[71,73] Combination products may include moisturizing cleansers, moisturizer skin protectant creams, and disposable washcloths that incorporate cleansers, moisturizers, and skin protectants into a single product. In a recent randomized, controlled, clinical trial in nursing home residents, Beeckman and associates[11] found that a 3-in-1 washcloth (cleansers, moisturizers, and skin protectant), impregnated with a 3% dimethicone formula, resulted in a significantly reduced IAD prevalence and a trend toward less-severe lesions. They concluded that this 1-step intervention was effective against the use of water and a pH neutral soap to prevent and/or treat IAD.

Because of a lack of valid tools to compare the clinical effectiveness of specific products to prevent IAD, the expert panel strongly recommends additional research to establish a benchmark for measuring a product's ability to block exposure to a specific irritant, preserve skin health, and prevent maceration. The existing model for ranking the magnitude of protection from various elements of sunlight in the current SPF (sun protection factor) system may serve as a useful model for a skin protectant taxonomy.

Supportive Interventions

Supportive interventions include the use of absorptive or containment products and the use of an indwelling device (urinary catheter or stool diversion system) in order to restore or maintain skin integrity. The panel recognizes that the use of absorptive or containment products is a fertile area of investigation; however, evidence indicates that when used correctly, absorptive or containment products comprise an important intervention for preventing IAD.[33] In a Cochrane review on the use of absorbent products for moderate to heavy urinary and/or fecal incontinence, Fader and coworkers[34] stated that no particular design seemed to be better or worse to maintain skin health. Instead, they found that individuals differed in their preference for absorbent product designs and using a combination of designs for various activities such as day versus night, or going out versus staying in was preferred to use of a single design. Shigeta and coinvestigators[88] reported results of a comparative cross-sectional study that examined perineal skin characteristics in 45 elderly female nursing home residents with fecal incontinence or double urinary and fecal incontinence. Absorbent pad surface pH ($P < .01$) and excessive sweating ($P = .006$) were significantly related to a more alkaline skin pH. Results show that the perineal skin in this particular population was significantly affected by occlusion with pads, increasing the risk of IAD.

Panel members concurred that more research is required to fully elucidate the role of absorptive or

containment products in maintaining skin health. They support Fader's group[34] in saying that research priorities should include development of methodologies for measuring characteristics of leaked urine and stool such as volume, flow, and consistency, and that techniques for measuring incontinence-related skin health are needed to provide data on which to base design specifications.

In selected cases, urine or stool may be transiently diverted from the skin via an indwelling device such as a urinary catheter or stool diversion system in order to restore or maintain skin integrity. Diversion must be limited to 28 days in some countries. Because of a lack of research focusing on this topic, the panel acknowledges that this recommendation is based on clinical experience and expert opinion alone.

Treatment

Literature review revealed scant evidence concerning treatment of IAD and the need for further studies that involve comparing protocol and product efficacy to determine best practice for IAD treatment. The panel agrees that referral to a continence specialist should be considered for assessment and treatment of the underlying incontinence. As for IAD prevention, the panel recommends a consistent, defined skin care regimen, including gentle perineal cleansing, moisturization, and the application of a skin protectant or moisture barrier. Specific interventions, such as the application of antifungal products, steroidal-based topical anti-inflammatory products, or topical antibiotics should not be used for routine treatment of IAD. Antifungal products should only be used if a fungal rash is present. Patients who do not respond to treatment within 2 weeks should be referred for additional evaluation. Atypical presentations of IAD have been described (eg, Jacquet diaper dermatitis and hyperkeratotic IAD) and the panel agreed that these require additional assessment and treatment.[89,90]

One small study reported on the effectiveness of a moisturizer to treat IAD.[91] This moisturizer was defined as a hydrogel-based barrier/repair cream and acted as a humectant with a barrier function. The researchers used a split body approach, in a double-blinded randomized design. One body site of 80 patients with a variety of dermatologic conditions (only 5 patients were affected with IAD) was treated with a hydrogel-based barrier/repair cream, while the other side was treated with a traditional petrolatum-based cream. A significant overall reduction in erythema, roughness, and desquamation of the skin with use of a hydrogel/barrier repair cream versus a petrolatum-based moisturizing cream was found. However, no segregated results were reported on the effect on IAD.

Four trials were identified that reported the effectiveness of a skin protectant to treat IAD. Anthony and associates[92] reported a double-blinded, randomized, controlled trial involving 57 subjects and found that a topical zinc oxide preparation with added antiseptic properties (Sudocrem) was superior to traditional zinc cream to treat IAD. Campbell and coinvestigators[93] reported a descriptive study on 33 patients in a rehabilitation unit and found a reduction in erythema, skin maceration, and skin stripping when using a liquid film-forming acrylates to treat IAD. One randomized controlled trial involving 39 subjects found reduced erythema and denudation with liquid film-forming acrylates compared to zinc oxide oil.[94] Beeckman and coinvestigators[11] reported a randomized controlled trial comparing a 3-in-1 washcloth to washing with pH neutral soap and water in a group of 464 nursing home patients; 22.3% of patients in the control group and 22.8% in the intervention were found to have IAD at baseline measurement. After 120 days of treatment, subjects treated with the washcloth had a reduction in IAD severity of 3.8 of 10 versus a reduction of 6.9 of 10 in the control group; this difference was not statistically significant ($P = .06$).

Discussion

Although limited, a small number of studies are now available that provide reasonable estimates of the prevalence of IAD in the acute and long-term care settings. Additional research is needed to provide more robust estimates of IAD prevalence in other settings including critical care units, long-term acute care facilities, the home health care setting, and among community-dwelling adults. A standard protocol, procedure, and data collection instrument are recommended to perform these IAD prevalence/incidence studies and to allow comparison between data and figures. Furthermore, appropriate training should be provided to allow observers to make a correct distinction between IAD and other types of skin lesions. Additional research is needed to more accurately determine the incidence of IAD. Considered collectively, the panel also observed that acuity of illness might influence the risk for IAD. Additional research is needed to determine the influence of acuity on IAD and its severity.

Research focusing on the etiology and pathogenesis of IAD is particularly sparse. The majority of the research the panel identified tends to be several decades old and tends to focus on diaper dermatitis in infants rather than IAD in adults. The consensus panel recommends that additional research focus on urine as an etiologic or synergistic factor, and on factors influencing the irritating effects of stool. The panel also recommends additional research addressing the microbiology of IAD in the adult, including the role of critical colonization and infection with *Candida* sp.

The panel recognizes the ongoing challenges associated with differentiating IAD from pressure ulcers faced by specialty practice and generalist nurses alike. However, rather than abandoning attempts to accurately describe perineal skin damage based on etiology, the panel recommends ongoing research into the development of effective educational interventions for IAD assessment such as the PUCLAS

and incorporation of knowledge about differential assessment and treatment into the curriculum of nursing schools. The panel also recognizes the urgent need for further development and validation of instruments designed to aid nurses to accurately identify IAD and rank its severity such as the IADS tool. The panel also recognizes the potential of noninvasive technologies such as subepidermal moisture measurement as an aid for early diagnosis and differentiation between IAD and pressure ulcers, so preventive interventions can be initiated before skin damage progresses.

The panel concurs that a small but growing body of evidence shows that effective prevention and treatment can reduce IAD incidence and increase healing rate. An effective strategy will consist of gentle perineal cleansing, moisturization, and the application of a skin protectant or moisture barrier. For treatment, this strategy might be combined with a customized addition of antifungal products, steroidal-based topical anti-inflammatory products, or topical antibiotics.

Conclusion

A review of the literature over the past 5 years since the first IAD consensus met reveals progress in our understanding of the epidemiology, pathophysiology, diagnosis, and treatment of this clinically relevant condition. Nevertheless, research remains limited and ongoing work is needed to enhance our understanding of IAD and to establish evidence-based protocols for its prevention and treatment.

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