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Moisture-Associated Skin Damage

Expanding and Updating Practice Based on the Newest ICD-10-CM *Codes*

Mikel Gray ◆ Donna Z. Bliss ◆ Laurie McNichol

ABSTRACT

Moisture-associated skin damage (MASD) occurs when skin is repeatedly exposed to various sources of bodily secretions or effluents, often leading to irritant contact dermatitis with inflammation, with or without denudation of affected skin. In 2020, the Wound, Ostomy and Continence Nurses Society took an initiative that led to the addition of multiple *International Classification for Diseases* codes for irritant contract dermatitis caused by various forms of MASD for use in the United States (*ICD-10-CM*). In the last issue of the *Journal of Wound, Ostomy and Continence Nursing*, a clinical practice alert identifying the various new codes was published that summarized each of the new codes and provided highlights of the descriptions of each of the these codes. This is the first in a series of 2 articles providing a more detailed description of the newest irritant contact dermatitis codes linked to MASD. Specifically, this article reviews the clinical manifestations and assessment, pathophysiology, epidemiology, prevention, and management of irritant contact dermatitis due to saliva, respiratory secretions, and fecal or urinary incontinence.

KEY WORDS: Incontinence-associated dermatitis, Intertriginous dermatitis, Intertrigo, Irritant contact dermatitis, Moisture-associated skin damage, Respiratory secretions, Saliva, Tracheostomy.

INTRODUCTION

Moisture-associated skin damage (MASD) occurs with exposure to various sources of moisture (bodily secretions or effluents) such as urine or fecal matter, perspiration, wound exudate, mucus, digestive secretions, respiratory secretions, or saliva.[1] Historically, the concept of MASD arose from recognition that many skin lesions identified by nurses in patients at risk for pressure injury, stage 2 pressure injuries in particular, were not attributable to pressure or shearing forces. In 2005, the European Pressure Ulcer Advisory Panel released a document that differentiated moisture lesions, defined as erythema with or without partial-thickness skin erosion, from lesions caused by the pressure or shearing forces (pressure injuries or ulcers).[2] In 2007, Gray and colleagues[3] held a round table discussion that identified the concept of MASD. Gray's group observed that that MASD has multiple clinical manifestations and that each of these manifestations varied based on the type and chemical constitution of the moisture source (bodily secretion or effluent) and its effects on the skin.[3] This recognition ultimately led to the publication of a series of articles in 2011 that reviewed clinical manifestations and the pathophysiology of MASD, along with 4 prevalent forms: incontinence-associated dermatitis (IAD), intertriginous dermatitis (ITD), peristomal MASD, and periwound MASD.[1,4,5] While the authors of this series of articles recognized these forms of MASD as prevalent, they acknowledged that multiple other forms of MASD exist that deserve additional research and development of evidence-based interventions for their assessment, prevention, and management.

Since these foundational publications, multiple authors have investigated various forms of MASD, increasing our knowledge of its pathophysiology, classifications, assessment, prevention, and management.[6-8] Based on this growing recognition of MASD as a significant cause of skin damage, the latest version of the World Health Organization's *International Classification of Diseases* (*ICD*) codes (11th edition) for the first time includes codes for various forms of MASD.[9] However, unlike most other countries in the world, the United States ties *ICD* codes into reimbursement for health care and will continue to use a Clinical Modification of *ICD* version 10 (*ICD-10-CM*). Therefore, the Wound, Ostomy and Continence Nurses (WOCN) Society led a group that worked with the *ICD-10-CM* Coordination and Maintenance Committee to develop codes for use in the United States until we moved to use of the 11th edition of the *ICD* codes.[10] The original intent of this group was to achieve a code for a single form of MASD (IAD). Ultimately, the group elected to request multiple codes recognizing multiple forms of MASD. Consistent with the original intent of the group that defined IAD and distinguished it from pressure injuries or other forms of skin damage,[11] each of these codes is defined as a form of irritant contact dermatitis and each also identified the moisture source that leads to a particular form of MASD.[12]

In the previous issue of the *Journal of Wound, Ostomy and Continence Nursing*, Bliss and colleagues[13] authored a Clinical Practice Alert that outlined each of the codes and provided a

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photographic image of the various forms of MASD. The purpose of this article is to provide a more detailed description of the *ICD-10-CM* codes for irritant contact dermatitis due to saliva, respiratory, and exposure to fecal or urinary incontinence, followed by a second article describing irritant contact dermatitis related to digestive secretions or effluent from a urinary or fecal ostomy or fistula.

IRRITANT CONTACT DERMATITIS DUE TO EXPOSURE TO UNSPECIFIED MOISTURE SOURCE (*ICD-10-CM* CODE L24.A0)

The *ICD* coding taxonomy is divided into categories based on organ or organ system, as well as type of disease or disorder. The various MASD codes are classified as irritant contact dermatitis.[12] Many substances have the potential for causing irritant contact dermatitis including chemicals, drugs, cosmetics, detergents, dyes, foods, and contact with animals or plants, among other substances. In the *ICD-10-CM* system, irritant codes begin with L24 or L25. They must be distinguished from other forms of dermatitis such as allergic or radiation dermatitis. Code L24.A0 is described as irritant contact dermatitis due to friction or contact with body fluids, unspecified. This unspecified code is intended for use when the moisture source leading to irritant contact dermatitis is unknown.

Clinical Manifestations

The irritant dermatitis of all forms of MASD is characterized by inflammation of the skin, sometimes accompanied by erosion or denudation of the skin, and a serous exudate. The location reflects areas where skin is exposed to the underlying moisture source, and the borders tend to be indistinct when compared to partial- or full-thickness pressure injuries. Symptoms may include burning and itching of inflamed skin.[1,4,5] While this code may be used for initially characterizing MASD, the WOC or wound care nurse's assessment should focus on determining the underlying factors leading to irritant contact dermatitis and moisture source in order to provide a more specific diagnosis and guide treatment (Table).

Accurate documentation of these characteristics enables colleagues or a coder reviewing notes in the electronic medical record to easily identify and accurately code these clinically relevant forms of MASD. Accurate labeling of the various forms of MASD is not only important for advanced practice providers such as nurse practitioners and clinical nurse specialists, it is just as important for every WOC nurse caring for patients with MASD. The WOCN Society is currently working with a consultant, Donna Cartwright, who is a professional coder to work with professional coding societies to recognize and accurately code these forms of MASD based on documentation in nursing or progress notes in a manner similar to that used to document pressure injury occurrences in acute-, post-acute, and long-term care facilities.

IRRITANT CONTACT DERMATITIS DUE TO SALIVA (*ICD-10-CM* CODE L24.A1)

Irritant contact dermatitis due to saliva occurs when the skin is exposed to excessive saliva leading to erythema and local irritation.[13] Saliva is produced and secreted from the acini of specialized (salivary) glands, located in the mouth.[14] Comprising more than 95% water, saliva also contains mucus, electrolytes including sodium, potassium, and bicarbonate, along with α-amylase (a digestive enzyme that degrades various starches initiating their digestion), lysozyme, lactoferrin, peroxidase, and secretory IgA (these substances exert antibacterial properties). The pH of saliva varies from 6.0 to 7.0. Saliva serves a variety of physiologic functions including binding and lubricating food to ease swallowing, solubilizing food to enable tasting, and antimicrobial activity via a combination of mechanical flushing and the actions of various components of saliva described earlier.

Epidemiology and Pathophysiology

Evidence concerning the epidemiology of saliva-associated irritant contact dermatitis (SAICD) is too sparse to enable definitive measurements of its prevalence or incidence. The most prevalent form of SAICD is probably related to excessive licking of the lips and surrounding skin, leading to several forms

TABLE.
Clinical Characteristics and Distributions of Irritant Contact Dermatitis Due to Saliva, Respiratory Secretions, Fecal and/or Urinary Incontinence, and Perspiration Trapped in a Skinfold

MASD Type/ *ICD-10-CM* Code	Moisture Source	Clinical Manifestations	Location and Distribution of Skin Damage
Irritant contact dermatitis due to saliva/L24.A1	Saliva	Erythema of affected skin, appearance ranges from shades of light pink to red to purple on light, medium, and dark skin tones, partial-thickness skin erosion may occur, the borders of skin damage are irregular and will reflect the areas exposed to the moisture source, signs of secondary cutaneous infections will be present in some cases, serous exudate may occur causing symptoms that include burning or itching	*Intact oral cavity*: Vermillion and skin adjacent to the vermillion including the corners of the mouth and skin and the skin that can be reached by the tongue *Loss of control of salivary secretions* (usually seen with head and neck cancers): Any skin exposed to chronic salivary secretions, often incudes the chin and neck
Irritant contact dermatitis due to respiratory secretions/L24.B2	Respiratory secretions from an intentional tracheostomy or tracheocutaneous fistula		The skin immediately adjacent to the tracheal stoma, including the skin underneath the dressing or solid skin barrier placed underneath a tracheostomy tube if present.
Intertriginous dermatitis/ intertrigo/L30.4	Perspiration trapped in a skinfold that cannot evaporate		Any fold where 2 skin surfaces are in chronic contact such as the axilla, underneath the breasts, and between the toes. Additional skinfolds beneath the abdomen (pannus), neck, or shoulders may be present in persons with severe to morbid obesity
Incontinence-associated dermatitis/L24.A2	Fecal matter or urine		Fourteen areas most likely to result in IAD have been identified; they include the perineal and perigenital skin, posterior thigh, lower abdomen, crease between the genitals, and thigh.

Abbreviations: IAD, incontinence-associated dermatitis; MASD, moisture-associated skin damage.

of cheilitis.[15,16] Cheilitis is also observed in critically ill patients with an endotracheal tube that keeps the mouth partially open. Cheilitis simplex (often referred to as chapped lips) is characterized by damage to the vermillion of the lips due to excessive lip licking in an attempt to compensate for dryness.[15,16] Angular cheilitis occurs when the dermatitis extends to adjacent skin at the corners of the mouth. In addition, some patients with neurologic disorders, dementia, or obsessive compulsive behaviors may engage in compulsive or chronic lip licking and develop irritant contact dermatitis of the vermillion and adjacent skin.[17,18] We searched the literature and found a single article that evaluated the epidemiology of 202 patients diagnosed with cheilitis; 5.4% of cases were attributed to irritant contact dermatitis.[19] We searched the literature but found no studies exploring the pathophysiologic mechanisms when skin is exposed to saliva.

Clinical Manifestations

Clinical manifestations of SAICD in persons with an intact oral cavity include erythema, scaling of affected skin, along splitting of the vermillion of the lips, sometimes accompanied by erosion of skin exposed to saliva, itching, and burning.[13] The pattern of skin damage will reflect the skin that can be reached by the tongue. The resulting irritant contact dermatitis is usually mild to moderate in severity.[15,16]

Excessive production of saliva (sialorrhea) affecting skin other than that surrounding the vermillion is attributable to loss of control of the ability to retain saliva in the mouth (drooling) with or without alterations in the rate or character of saliva production (Figure 1).[13] Clinical experience strongly suggests that SAICD is particularly common among adults with head and neck cancers. The most prevalent forms of head and neck cancers are squamous cell carcinomas arising from the mouth, throat, and larynx.[20] While the majority of these patients will experience xerostomia (abnormally low-volume saliva production), some will suffer from sialorrhea. Excessive salivary production is associated with the tumor itself or surgical resection of malignant tissue, especially when surgery involves the middle third of the mandible. In addition to raising the risk for irritant contact dermatitis when in contact with the skin, some patients will also experience a salivary (spit) fistula or salivary mucocele (sialocele). In these cases, saliva comes into contact with skin beyond the border of the vermillion, sometimes leading to larger areas of irritant contact dermatitis. Diagnosis is based on the patient's history and physical assessment, focusing on salivary production and inability to control salivary flow, combined with erythema, flaking, and dried, broken skin characteristics of this form of MASD.[13,21]

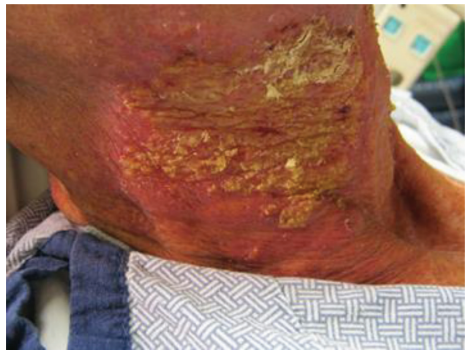

Figure 1. Saliva-associated irritant contact dermatitis affecting the skin and neck.

Prevention and Management

Prevention and management of SAICD in persons with an intact oral cavity focus on modifying excessive licking behaviors and enhancing the epithelial/moisture of the vermillion and adjacent skin, combined with application of topical skin protectants such as petrolatum-based products. In selected cases, treatment also may include use of a corticosteroid cream if concurrent allergic contact dermatitis is suspected.[15,16]

Because of the complexity of the underlying disease and treatments, management of SAICD in patients with head and neck cancers focuses on removing the irritant source by controlling the production and/or flow of saliva.[21] Surgical interventions include creation of a planned salivary (spit) fistula to control the flow of saliva, salivary gland excision, and salivary duct ligation and transposition. Targeted radiation therapy may be used to reduce salivary production. Several drugs have been used to reduce saliva production including anticholinergics and botulinum toxin; none are approved by the US Food and Drug Administration for this indication. Topical therapies include regular cleansing and application of a skin protectant to prevent excessive exposure to saliva. Given the limited research in this area and lack of guidelines for practice statements, significant additional research is needed to further our understanding of the epidemiology, etiology, pathophysiology, assessment, prevention, and treatment of SAICD.

IRRITANT CONTACT DERMATITIS DUE TO RESPIRATORY SECRETIONS (*ICD-10-CM* CODE L24.B2)

Respiratory secretions comprise water and salts, along with multiple glycoproteins including multiple mucins (MUC 1, 2, 4, 5AC, 5B, 7, 8, and 13) and a number of proteoglycans, lipids, and numerous proteins (chemokines and cytokines) with antimicrobial activity; its pH in healthy individuals is around 6.[22,23] The respiratory tract plays host to microbiota that aid immune function while interacting with the respiratory mucus and cilia to remove potential pathogens from the respiratory system. The primary physiologic function of respiratory mucus is to form a self-cleaning barrier to inhaled allergens, pathogens, and other potential irritants.

Clinical Manifestations

The clinical manifestations of irritant contact dermatitis usually include inflammation of the peristomal skin adjacent to the tracheal stoma (Figure 2).[13] Erosion with partial skin loss may occur with irregular borders reflecting the area exposed to respiratory secretions. However, these areas must be differentiated from medical-related pressure from a tracheostomy tube or ties used to secure the tracheostomy tube or ventilator tubes.

Epidemiology and Pathophysiology

Tracheostomy is a surgically created ostomy (stoma) connecting the trachea directly to the skin.[24] Evidence concerning the frequency of tracheostomy creation is not known, but the available research strongly suggests that intentional tracheostomy is the most frequent scenario underlying irritant contact dermatitis due to respiratory secretions.[25-27] For example, Fischler and colleagues[25] evaluated 90,412 patients receiving care for a combined 243,921 days in intensive care units (ICUs). They found that 10% of patients who required ventilation for more than 24 hours underwent tracheostomy.[25]

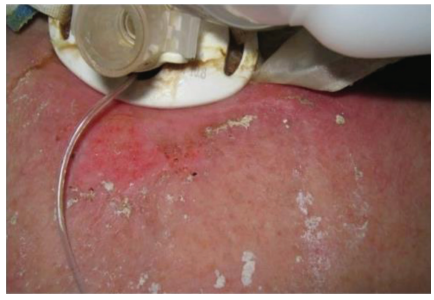

Figure 2. Irritant contact dermatitis of the peristomal skin of a tracheostomy.

Similarly, Blot and colleagues[26] evaluated 35,332 critically ill patients cared for in 708 ICUs and reported that a median of 7.2% had undergone tracheostomy within 7 days of ventilation. Clearly, the number of patients requiring ICU admission and ventilator support has surged precipitously during the years 2020-2021 due to the global COVID-19 pandemic. Guarnieri and colleagues[27] investigated 151 patients with SARS CoV-2 and found that 48% underwent tracheostomy; they further observed that patients who required tracheostomy had longer ICU stays and more complications related to their tracheostomy. The proportion of these complications associated with peristomal MASD was not recorded.

In addition to critically ill patients, a number of chronic diseases may require tracheostomy for long-term ventilation.[28-30] For example, Pereira and colleagues[28] examined residents of 33 skilled nursing facilities and reported that 3.36% had long-term tracheostomies. Guarnieri and colleagues[27] evaluated 209 patients with amyotrophic lateral sclerosis and reported that 16% had a tracheostomy. Similarly, Branco's group[30] reviewed medical records of 5265 patient with cervical spinal cord injuries and reported that 20.6% required long-term tracheostomy for ventilatory support.[29] In addition to these individuals requiring short- or long-term management with tracheostomy, as many as 70% of adults and children with a tracheostomy for more than 16 weeks will experience a persistent tracheocutaneous fistula that typically requires 1 or more surgical procedures to repair.[29-31]

The pathophysiology of irritant contact dermatitis created when skin is exposed to respiratory secretions is not known. Research concerning the prevalence of peristomal irritant contact dermatitis is also sparse; a single report in Chinese indicated a 38.5% prevalence of respiratory secretion irritant contact dermatitis in persons with tracheostomies.[32,33]

Prevention and Management

A review of the literature reveals no international guidelines for care of the peristomal skin surrounding a tracheostomy. Available guidelines for nursing care tend to focus on suctioning and securement methods for tracheostomy tubes.[34-36] Generalized recommendations for peristomal skin care include cleansing with saline and avoidance of hydrogen peroxide. Securement of tracheostomy ties or tube sufficiently tight to avoid life-threatening removal of the tube, along with prevention of medical device–related pressure injury when the ties are excessively tight, was described. Best practice standards also recommend selection of an absorbent prepackaged tracheostomy dressing rather than cotton gauze in order to avoid inadvertent introduction of cotton fibers into the respiratory system.[34]

Limited evidence provides a basis for additional interventions for prevention and management specifically designed at reducing exposure of the peristomal skin to respiratory secretions. Considered collectively, available evidence suggests that a solid skin barrier is more effective than an absorbent dressing or gauze placed over the peristomal skin of a tracheostomy. Specifically, Chuang and colleagues[32] extended this line of research via a randomized crossover trial comparing gauze to a cut-to-fit hydrocolloid (pectin) skin barrier in a group of 34 participants cared for in ICUs in 2 facilities. They found that use of the hydrocolloid skin barrier reduced erosion of the peristomal skin and increased nurse satisfaction when caring for these critically ill patients. Ahmadinegad and colleagues[37] reported findings from a randomized controlled trial comparing an absorbent foam dressing to sterile gauze placed over the peristomal skin in 80 critically ill adults from a single-layer application immediately following tracheostomy. They found no difference between bacterial colonization or infections of the tracheostomy site versus respiratory secretions based on selection of a gauze versus foam dressing. Karaca and Korkmaz[38] evaluated the use of a barrier cream applied to the peristomal skin beneath a gauze dressing in a quasi-experimental study and reported differences in several characteristics of the peristomal skin (cutaneous pH, mean peristomal skin moisture, and temperature), prompting them to recommend use of the barrier cream in patients with tracheostomies. Similarly, Schuren and colleagues[39] reported findings of a systematic review of a liquid polymer and cyanoacrylate moisture barrier for a variety of MASD forms and concluded that its use also reduced erosion and inflammation of the peristomal skin. Additional research is needed concerning the use of both skin barriers versus absorbent dressings and topical moisture barriers for prevention and management of irritant contact dermatitis due to respiratory secretions.

INTERTRIGINOUS DERMATITIS/INTERTRIGO (*ICD-10-CM* CODE L30.4)

An *ICD-10-CM* code for intertriginous erythema (identified as erythema intertrigo) is classified under "other and unspecified" forms of dermatitis. It is found on skin surfaces in contact with each other, such as the axillae, neck creases, intergluteal fold, and between the toes. Despite its characterization under the other and unspecified category of L30, it is attributed to moisture and friction.[12] This code was approved for use in the *ICD-10-CM* taxonomy prior to inclusion of MASD codes for irritant contact dermatitis related to salivary, respiratory, digestive, urinary and fecal incontinence, ostomy effluent, and effluent from a fistula; the rationale for this categorization is not immediately apparent.

The moisture source for ITD is perspiration. Perspiration is almost entirely composed of water, with trace amounts of sodium, potassium, calcium, magnesium, and zinc.[40] Sweat is produced by eccrine, apocrine, and apoeccrine sweat glands located throughout the skin.[41] The pH of sweat in otherwise healthy persons is around 6.3.[42] The anticipated range of sweat production of humans in a moderate climate is 600 to 700 mL.[1] Humans sweat in response to rises in core body temperature in response to the surrounding environment, physical exertion, and systemic infection. The main purpose of sweating is thermoregulation that occurs as perspiration evaporates from the skin. In the presence of excessive environmental heat or occlusion of the skin and its sweat glands, the core body temperature can rise to dangerous levels, resulting in hypothermia and heat stroke. In addition, an increased risk of

ITD occurs when perspiration is trapped within a skinfold or underneath occlusive clothing or footwear that does not allow prompt evaporation of sweat.

Clinical Manifestations

The inflammation and erythema of ITD may be found in any fold where 2 skin surfaced rub together such as the axilla, inframammary folds, and groin. In obese individuals, skinfolds also may occur between the lower abdomen and genital area due to an abdominal pannus or other areas created as body mass index (BMI) increases.[4] The severity of inflammation and the highest likelihood of denuded skin are found at the deepest portion of skinfold because it is where the friction and chronic moisture are greatest and the severity of inflammation diminishes at the edge of skinfold where the skin has greater exposure to air, enabling prompt evaporation of perspiration. Secondary cutaneous infections may occur due to the warm and humid microclimate created by the skinfold.

Pathophysiology and Epidemiology

Little is known about the pathophysiology of ITD. Obesity is associated with a variety of physiologic stressors including an increased risk for ITD.[43] The relationship between BMI and transepidermal water loss (TEWL, a measure of the skin's epithelial/moisture barrier) in severe to morbid obesity is associated with a body-wide increase in TEWL.[44] Persons with severe to morbid obesity also have greater areas of skin contained within skinfolds formed by the abdominal pannus, underneath the breasts in females and males, and the groin.[45] Nevertheless, it is important to note that not all cases of ITD are found in obese persons. For example, ITD has been described in the inframammary skinfolds, axillae, and between the toes of persons with normal and higher BMIs.[45-47] Friction is also postulated to play a significant role in the pathophysiology of ITD, particularly when moist skinfolds rub together.[1] The warm and humid local microclimate is also associated with changes in commensurate bacterial species found on the skin; coliform bacterial species, cyanobacteria, actinobacteria, and streptococcal infections found in moist skinfolds that may result in secondary cutaneous infections in skinfold as have various viral species, though the frequency and contribution of these potential pathogens to ITD are not well understood.[48,49] Multiple *Candida* species (*C albicans*, *C glabrata*, *C tropicalis*, *C krusei*, *C parapsilosis*, *C dubliniensis*, and *C famata*) are associated with ITD; in this case, treatment must incorporate both interventions to promote evaporation of the moisture created by perspiration with topical application of antifungal agents.[50] Yosipovitch and coworkers[51] measured cutaneous pH in a group of 50 participants with type 2 diabetes and a group of 40 controls and reported a significantly higher pH in diabetic individuals, suggesting diabetes mellitus may act as a risk factor for candidiasis in persons with ITD.

Research into the epidemiology of ITD is limited, but a number of recent studies have significantly increased our knowledge of its prevalence and incidence in various care settings. Kottner and colleagues[52] reported findings from a secondary analysis of data from 4 prevalence studies conducted in the Netherlands between 2013 and 2016. The combined sample size used for this secondary analysis was 40,340 patients. The prevalence of ITD was 9.6% (95% CI, 8.6-10.6%) in patients receiving home care, 6.7% (95% CI, 6.4-7.0) in patients residing in long-term care facilities, and 2% (95% CI, 1.8-2.3) in hospitalized patients. Risk factors associated with an increased likelihood of ITD were a higher BMI, having diabetes mellitus, and being care dependent; these factors were associated with the presence of intertrigo in all 3 settings. Gabriel and colleagues[53] reported an ITD prevalence of 16.1% (95% CI, 11.6-21.2) in a group of 223 patients residing in long-term care facilities in Berlin, Germany. The most common locations were the inframammary folds, found in 9.9% of participants, followed by the inguinal region (9.4%), axilla (0.5%), and abdominal region (0.5%). Risk factors for ITD identified in this study were increased age and care dependency.

Arnold-Long and Johnson[54] measured prevalence and incidence of ITD in 417 adult patients admitted to a single acute care facility in the United States between 2014 and 2016. The mean prevalence over this 3-year data collection period was 40% (range, 36%-42%) and the mean incidence was 30% (range, 32%-39%). Obesity was linked to an increased prevalence and incidence of ITD.

Prevention and Management

Mistiaen and colleagues[55] systematically reviewed the literature related to prevention and treatment of ITD in larger skinfolds. Sixty studies, including 4 randomized controlled trials, characterized as having a significant risk for bias, were retrieved and evaluated. No studies were found that focused on prevention. In addition, the studies evaluating various interventions focused on patients with secondary infections. These interventions were various topical antiseptics, antimycotic (antifungal) agents, antibiotics, and corticosteroids for alleviation of inflammation. Nine studies with a median sample size of 17.2 participants were located that evaluated the effects of tea tree oil (*Melaleuca alternifolia*), tacrolimus, betulin (a topical anti-inflammatory agent made from triterpene from the bark of birch trees), Mericleri salt (an antifungal agent), witch hazel (derived from the flowering shrub *Hamamelis virginiana* and used for its anti-inflammatory actions), and polynoxyin (a formalin-releasing antiseptic agent) in patients with ITD and secondary cutaneous infections. These studies also examined the effects of soap and water, alone or combined with application of talcum powder, and barrier creams or hydrocolloid dressings. The authors also identified 15 studies that focused on surgical management of ITD; all focused on the use of mammoplasty for management of inframammary ITD. Based on the paucity of studies and the higher risk of bias in these studies, the authors concluded that there are no effective treatments of ITD that can be considered evidence-based and no evidence concerning the efficacy of interventions for its prevention. Current best practices for prevention and treatment of ITD in larger skinfolds include routine skin cleansing, possibly followed by application of a skin protectant.[4] This may be followed by drying of the skin with a hair dryer set on a low (cool) setting once daily in an attempt to enhance the skin's natural epithelial border and encourage evaporation of moister within the skinfold. Cleansing may be followed by application of a skin protectant, but the characteristics of the optimal skin protectant in a skinfold remain controversial. Ointments are generally avoided due the risk of occluding the surface of the skin, paradoxically preventing the evaporation of local perspiration. Liquid polymer acrylates or creams are possible alternatives, but evidence concerning their efficacy for prevention and treatment of ITD is lacking.[4,55] Placement of fabrics or absorbent products capable of reducing moisture between large skinfolds has been advocated.[4] These products are placed so that an edge protrudes from the skinfold in order to wick moisture away from the

skinfold while preventing friction as the skin rubs together rather than just absorption of perspiration. A wicking fabric that is impregnated with silver may be used that is designed to wick moisture, reduce friction, and exert an antiseptic effect against a variety of bacterial and fungal species.

INCONTINENCE-ASSOCIATED DERMATITIS (*ICD-10-CM* CODE L24.A2)

Incontinent-associated dermatitis is a type of irritant contact dermatitis that is due to prolonged exposure to urinary, fecal, or dual incontinence.[4,56] Although research remains limited, the evidence base underpinning our understanding of the epidemiology, pathophysiology, diagnosis, prevention, and treatment of IAD is more robust than the body of research for other forms of MASD discussed in this article.

The moisture sources for IAD are urine or fecal materials (stool). Urine is mainly composed of water (>95%), but it also contains a rich and variable mixture of urea, sodium, potassium, chloride, creatinine, and traces of multiple other metabolites.[57] The pH of urine varies significantly from as low as 4.0 to as high as 8; numerous factors include dietary intake, presence of metabolites, concentration of citrate, and other substances in the urine, along with various bacterial species with the potential to raise pH via ammonia production.[58,59] Urinary pH tends to be higher in women, historically attributed to be related to differences in dietary intake; more recent research indicates that this difference is caused by a gender-specific higher absorption of food anions in females as compared to males.[58]

In the otherwise healthy individual, the composition of normal (soft, formed) stool is about 75% water along with organic solids.[60] Approximately 25% to 54% of these organic solids are bacterial biomass, up to 25% is protein or nitrogenous matter, 25% comprises undigested plant matter or carbohydrates, and as much as 15% is fatty matter. Liquid stool contains a much higher proportion of water, along with digestive enzymes including lipases and proteases. The pH range of soft, formed stool is around 6.3.[61] In contrast, the pH of semi-formed to liquid stool tends to be higher. A study of 428 stool samples obtained from 138 critically ill individuals with systemic inflammatory response syndrome found a mean pH of 7.1; the measured pH in individual patients was as high as 9.2.[62]

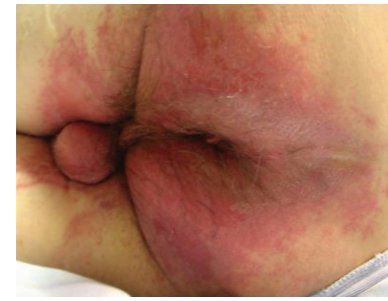

Figure 3. Incontinence-associated dermatitis due to dual urinary and fecal incontinence affecting the perineal skin, gluteal cleft, both buttocks, and inner thighs.

Clinical Manifestations

The main clinical manifestation of IAD is redness (erythema) of the skin (Figure 3).[63-65] Redness of the skin can range from shades of light pink to red to purple on light, medium, and dark skin tones.[66] Incontinent-associated dermatitis on dark-toned skin may also manifest as lighter or darker shades of underlying skin tones. Areas of skin erosion from IAD often appear shiny or glistening due to serous fluid accumulation or have different shades of redness of the various layers exposed. Localized edema can be present.[4] The edges of IAD are typically irregular, and a fungal or bacterial cutaneous rash may develop.[64,67] The Incontinence-Associated Skin Damage scoring tool (v. IASD.D.2) identified 14 body areas that can be affected by IAD based on consensus from WOC nurse experts (Figure 4).[68] Some of these areas are anterior, others are posterior, and some are bilateral. Symptoms of IAD include the sensations of itching, stinging, burning, and discomfort of the affected areas.[63-65]

Pathophysiology and Epidemiology

The pathophysiology of IAD is only partially understood. Research in healthy volunteers has shown that exposure of healthy skin to synthetic urine results in inflammation within a relatively brief period of time (4 hours).[65] A study that exposed the skin of healthy volunteers to synthetic urine and stool found that both prompted the release of inflammatory cytokines (IL-1α, IL-1RA, IL-1β, TNF-α, and IL-8) and that exposure to synthetic urine was associated with a higher rise in IL-1α whereas exposure to synthetic stool was associated with

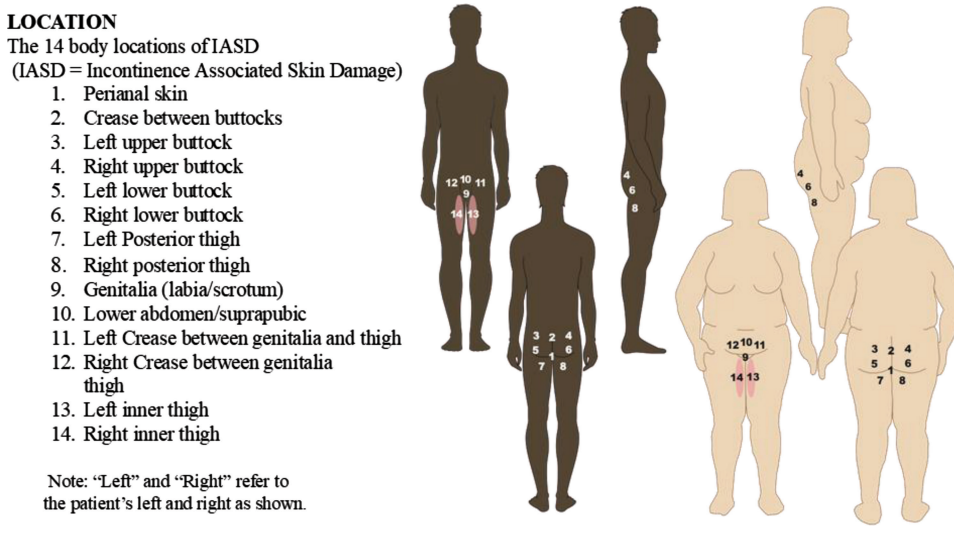

Figure 4. Fourteen body locations of incontinence-associated dermatitis.

a greater rise in TNF-α.[69] Exposure of the skin to synthetic urine and a controlled period of occlusion have also been found to increase inflammation of the skin and permeability to environmental water and other irritants.[70]

Clinical experience and research demonstrate that IAD can occur relatively quickly after exposure to urine and especially with exposure to liquid or semisolid stool common among critically ill adults. Bliss and colleagues[71] evaluated the incidence of IAD in a group of 45 critically ill adults and reported a median of 4-day (range, 1-6 day) time to the onset of IAD. In addition, IAD may reoccur after healing if fecal and/or urinary leakage persists and the skin is not adequately protected.[63]

Reported prevalence of IAD in hospitalized patients varies from 1.5% to 20%.[72-79] In critically ill patients, IAD prevalence is 46%[80] and the prevalence among residents in long-term care facilities varies from 3.1% to 6.5%.[64,81-83] The prevalence of IAD among admissions to acute long-term care facilities is 23%.[83] Among those living independently in the community, 52% of individuals with fecal or dual incontinence reported having IAD.[63] The incidence of IAD in critically ill patients ranges from 15% to 36% and in hospitalized/acute rehabilitation patients the incidence is 7.6%.[54,71,77,84] Among residents in long-term care facilities, IAD incidence rates vary from 3.4% to 30%.[85-87]

Multiple factors are associated with an increased likelihood of IAD, and various frameworks organizing these factors have been developed at different times.[88-90] A recent conceptual framework for IAD organized the risk and associated factors for IAD into 4 main categories: etiological factors (such as type and severity of incontinence), mechanisms for skin damage (such as skin pH and overhydration), factors that reduce skin tolerance (such as inadequate skin care, obesity, impaired tissue oxygenation, and inappropriate use of absorbent products), and general (person-related) factors (such as comorbidities, toileting ability, and cognitive status).[91]

Incontinence-associated dermatitis is linked to an increased risk for pressure injury development, and full-thickness pressure injuries in particular.[73,92,93] In addition, a study of nursing home residents who were incontinent and had a perineal pressure injury, they were twice as likely to develop IAD than those without a pressure injury.[86] These findings highlight that IAD and pressure injury can both be present in an area of skin damage, an important consideration when applying *ICD-10* codes.

Prevention and Management

One of the key interventions for successful treatment of IAD is to institute and follow a structured skin care regimen or algorithm using quality products.[85,94] The fundamental principles for prevention and treatment of IAD are similar.[95] Treatment of IAD includes prompt cleansing of urine and feces from the skin using a product that does not raise skin pH and gentle, thorough drying of the skin.[96] There are a variety of approaches aimed at protecting the skin from direct contact with leaked urine or feces to promote healing of IAD. The topical application of a skin protectant (also called a skin barrier) is suitable for most patients. Skin protectants come in a variety of formulations and applications including creams, pastes, films, sprays, skin cleansers and wipes, etc.[96-98] Healing of damaged skin may involve replenishing moisture or lipids using topical products such as a moisturizer, humectant, or emollient.[96,99]

CONCLUSIONS

Moisture-associated skin damage may result in multiple forms of irritant contact dermatitis. This article described the *ICD-10-CM* codes, moisture sources, clinical manifestations, epidemiology, pathophysiology, treatment, and prevention of MASD due to excessive exposure to saliva, respiratory secretions, fecal and urinary incontinence, and perspiration trapped in skinfolds. A second article will review irritant contract dermatitis associated with digestive secretions and fecal or urinary effluent from an abdominal stoma or enterocutaneous fistula.

REFERENCES

1. Gray M, Black JM, Baharestani MM, et al. Moisture-associated skin damage: overview and pathophysiology. *J Wound Ostomy Continence Nurs*. 2011;38(3):233-241.
2. Defloor T, Schoonhoven L, Fletcher J, et al. Statement of the European Pressure Ulcer Advisory Panel—Pressure Ulcer Classification: differentiation between pressure ulcers and moisture lesions. *J Wound Ostomy Continence Nurs*. 2005;32(5):302-306.
3. Gray M, Bohacek L, Weir D, Zdanuk J. Moisture vs pressure: making sense out of perineal wounds. *J Wound Ostomy Continence Nurs*. 2007;34(2):134-142.
4. Black J, Gray M, Bliss DZ, Kennedy-Evans KL, Logan S, Baharestani MM. MASD part 2: incontinence-associated dermatitis and intertriginous dermatitis: a consensus. *J Wound Ostomy Continence Nurs*. 2011;38(4):359-370.
5. Colwell JC, Ratliff CR, Goldberg M, et al. MASD part 3: peristomal moisture-associated dermatitis and periwound moisture-associated dermatitis: a consensus. *J Wound Ostomy Continence Nurs*. 2011;38(5):541-553.
6. Voegeli D, Hillery S. Prevention and management of moisture-associated skin damage. *Br J Nurs*. 2021;30(15):S40-S46.
7. Johansen E, Lind R, Sjobo B, Petosic A. Moisture associated skin damage (MASD) in intensive care patients: a Norwegian point-prevalence study. *Intensive Crit Care Nurse*. 2020;60:102889.
8. Werth SL, Justice R. Prevalence of moisture-associated skin damage in an acute care setting: outcomes from a quality improvement project. *J Wound Ostomy Continence Nurs*. 2019;46(1):51-54.
9. World Health Organization. *International Classification of Diseases*: 11th ed. https://icd.who.int/en. Accessed November 2, 2021.
10. Gray M, Bliss DZ, McNichol L, Cartwright D. View from here: moisture-associated skin damage an historic step forward. *J Wound Ostomy Continence Nurs. 2022*;48(6):581-583.
11. Gray M, Bliss DZ, Doughty DB, Ermer-Seltun J, Kennedy-Evans KL, Palmer MH. Incontinence-associated dermatitis: a review. *J Wound Ostomy Continence Nurs*. 2007;34(1):45-56.
12. Centers for Medicare & Medicaid Services. ICD 10 resources. https://www.cms.gov/Medicare/Coding/ICD10/ICD-10Resources. Accessed November 15, 2021.
13. Bliss DZ, McNichol L, Cartwright D, Gray M. Clinical practice alert: new *ICD-10* codes for MASD. *J Wound Ostomy Continence Nurs*. 2022;49(1):15-19.
14. Tiwari M. Science behind human saliva. *J Nat Sci Biol Med.* 2011;2(1):53-58.
15. Fonesca A, Jacob SE, Sindle A. Art of prevention: practical interventions in lip-licking dermatitis. *Int J Womens Dermatol*. 2020;6(5):377-380.
16. Greenberg SA, Schlosser BJ, Mirowski GW. Diseases of the lips. *Clin Dermatol*. 2017;35:e1-e14.
17. Hisa T, Hamada T, Hirachi Y, Yoshioka M, Takigawa M, Shigenaga Y. Senile lip licking. *Dermatology*. 1995;191(4):339-340.
18. French L, Usatine R. Angular cheilitis. In: Usatine R, Smith M, Mayeaux EJ, et al, eds. *Color Atlas of Family Medicine*. 2nd ed. New York, NY: McGraw-Hill; 2013:203-205.
19. Lim SW, Goh CL. Epidemiology of eczematous cheilitis at a tertiary dermatological referral center in Singapore. *Contact Dermatitis*. 2000;43(6):322-326.
20. Chow LQM. Head and neck cancer. *N Engl J Med*. 2020;382(1):60-72.
21. Bomeli SR, Desai SC, Johnson JT, Walvekar RR. Management of salivary flow in head and neck cancer patients—a systematic review. *Oral Oncol*. 2008;44:1000-1008.
22. Lillehoj EP, Kim KC. Airway mucus: its components and function. *Arch Pharmacal Res*. 2002;25(6):770-780.
23. Hansson GC. Mucins and the microbiome. *Annu Rev Biochem*. 2020;89:769-793.
24. Parker LC. Tracheostomy care. *Nurs Crit Care*. 2014;9(6):38-41.

25. Fischler L, Erhart S, Kleger GR, Frutiger A. Prevalence of tracheostomy in ICU patients. A nation-wide survey in Switzerland. *Intensive Care Med*. 2000;26(10):1428-1433.
26. Blot F, Melot C; Commission d'Epidémiologie et de Recherche Clinique. Indications, timing, and techniques of tracheostomy in 152 French ICUs. *Chest*. 2005;127(4):1347-1352.
27. Guarnieri M, Andreoni P, Gay H, et al. Tracheostomy in mechanically ventilated patients with SARS-CoV-2-ARDS: focus on tracheomalacia. *Respir Care*. 2021;66(12):1797-1804.
28. Pereira F, Silva AM, Vaz IM, Viamonte S, Winck JC. Tracheostomy prevalence at skilled nursing facilities. *Pulmonology*. 2020;26(2):90-94.
29. Ceriana P, Surbone S, Segagni D, Schreiber A, Carlucci A. Decision-making for tracheostomy in amyotrophic lateral sclerosis (ALS): a retrospective study. *Amyotroph Lateral Scler Frontotemporal Degener*. 2017;18(7/8):492-497.
30. Branco BC, Plurad D, Green DJ, et al. Incidence and clinical predictors for tracheostomy after cervical spinal cord injury: a National Trauma Databank review. *J Trauma*. 2011;70(1):111-115.
31. Chorney SR, Stow J, Javia LR, Zur KB, Jacobs IN, Sobol SE. Tracheocutaneous fistula after pediatric open airway reconstruction. *Ann Otol Rhinol Laryngol*. 2021;130(8):948-953.
32. Chuang WL, Huang WP, Chen MH, Liu IP, Yu WL, Chin CC. Gauze versus solid skin barrier for tracheostomy care: a crossover randomized clinical trial. *J Wound Ostomy Continence Nurs*. 2013;40(6):573-579.
33. Lu HM, Wu SC, Chu CL, Yang JM, Chen SC. Improvement approach of wound care with tracheostomy. *Chang Gung Nurs*. 2007;18:573-584.
34. Lawrence PR, Chambers R, Faulkner MS, Spratling R. Evidence-based care of children with tracheostomies: hospitalization to home care. *Rehabil Nurs*. 2021;46(2):83-86.
35. Bolsega TJ, Sole MA. Tracheostomy care practices in a simulated setting: an exploratory study. *Clin Nurse Spec*. 2018;32(4):182-188.
36. Schreiber ML. Tracheostomy: site care, suctioning, and readiness. *Medsurg Nurs*. 2015;24(2):121-124.
37. Ahmadinegad M, Lashkarizadeh MR, Ghahreman M, Shabani M, Mokhtare M, Ahmadipour M. Efficacy of dressing with absorbent foam versus dressing with gauze in prevention of tracheostomy site infection. *Tanaffos*. 2014;13(2):13-19.
38. Karaca T, Korkmaz F. A quasi-experimental study to explore the effect of barrier cream on the peristomal skin of patients with a tracheostomy. *Ostomy Wound Manage*. 2018;64(3):32-39.
39. Schuren J, Becker A, Sibbald RG. A liquid film-forming acrylate for peri-wound protection: a systematic review and meta-analysis (3M™ Cavilon™ no-sting barrier film). *Int Wound J*. 2005;2(3):230-238.
40. Montain SJ, Cheuvront SN, Lukaski HC. Sweat mineral-element responses during 7 h of exercise-heat stress. *Int J Sport Nutr Exerc Metab*. 2007;17(6):574-582.
41. Baker LB. Physiology of sweat gland function: the roles of sweating and sweat composition in human health. *Temperature*. 2019;6(3):211-259.
42. Jadoon S, Karim S, Akram MR, et al. Recent developments in sweat analysis and its applications. *Int J Anal Chem*. 2015;2015:164974. doi:10.1155/2015/164974.
43. Yosipovitch G, DeVore A, Dawn A. Obesity and the skin: skin physiology and skin manifestations of obesity. *J Am Acad Dermatol*. 2007;56(6):901-916.
44. Monteiro Rodrigues LM, Palma L, Santos O, Almeida MA, Bujan J, Tavares L. Excessive weight favours skin physiology—up to a point: another expression of the obesity paradox. *Skin Pharmacol Physiol*. 2017;30(2):94-101.
45. McMahon R. The prevalence of skin problems beneath the breasts of in-patients. *Nurs Times*. 1991;87(39):48-51.
46. Karaca S, Kulac M, Cetinkaya Z, Demirel R. Etiology of foot intertrigo in the district of Afyonkarahisar, Turkey: a bacteriologic and mycologic study. *J Am Podiatr Med Assoc*. 2008;98(1):42-44.
47. Metin A, Dilek N, Demirseven DD. Fungal infections of the folds (intertriginous areas). *Clin Dermatol*. 2015;33(4):437-447.
48. Tuzun Y, Wolf R, Engin B, Kecici AS, Kutlubay Z. Bacterial infections of the folds (intertriginous areas). *Clin Dermatol*. 2015;33(4):420-428.
49. Adisen E, Onder M. Viral infections of the folds (intertriginous areas). *Clin Dermatol*. 2015;33(4):429-436.
50. Metin A, Dilek N, Bilgili SG. Recurrent candidal intertrigo: challenges and solutions. *Clin Cosmet Investig Dermatol*. 2018;11:175-185.
51. Yosipovitch G, Tur E, Cohen O, Rusecki Y. Skin surface pH in intertriginous areas in NIDDM patients. Possible correlation to candidal intertrigo. *Diabetes Care*. 1993;16(4):560-563.
52. Kottner J, Everink I, van Haastregt J, Blume-Peytavi U, Schols J. Prevalence of intertrigo and associated factors: a secondary data analysis of four annual multicenter prevalence. *Int J Nurs Stud*. 2020;104:103437.
53. Gabriel S, Hahnel E, Blume-Peytavi U, Kottner J. Prevalence and associated factors of intertrigo in aged nursing home residents: a multi-center cross-sectional prevalence study. *BMC Geriatr*. 2019;19(1):105. doi:10.1186/s12877-019-1100-8.
54. Arnold-Long M, Johnson E. Epidemiology of incontinence-associated dermatitis and intertriginous dermatitis (intertrigo) in an acute care facility. *J Wound Ostomy Continence Nurs*. 2019;46(3):201-206.
55. Mistiaen P, Poot E, Hickox S, Jochems C, Wagner C. Preventing and treating intertrigo in the large skin folds of adults: a literature overview. *Dermatol Nurs*. 2004;16(1):43-46, 49-57.
56. Gray M, Beeckman D, Bliss DZ, et al. Incontinence-associated dermatitis: a comprehensive review and update. *J Wound Ostomy Continence Nurs*. 2012;39(1):61-74.
57. LibreTexts Chemistry. Urine composition and function. https://chem.libretexts.org/Bookshelves/Introductory_Chemistry/Map%3A_Fundamentals_of_General_Organic_and_Biological_Chemistry_(McMurry_et_al.)/29%3A_Body_Fluids/29.08%3A_Urine_Composition_and_Function. Accessed November 25, 2021.
58. Worcester EM, Bergsland KJ, Gillen DL, Coe FL. Mechanism for higher urine pH in normal women compared with men. *Am J Physiol Renal Physiol*. 2018;314(4):F623-F629.
59. Koudounas S, Bader DL, Voegeli D. Knowledge gaps in the etiology and pathophysiology of incontinence-associated dermatitis. *J Wound Ostomy Continence Nurs*. 2020;47(4):388-395.
60. Rose C, Parker A, Jefferson B, Cartmell E. The characterization of feces and urine: a review of the literature to inform advanced treatment technology. *Crit Rev Environ Sci Technol*. 2015;45(17):1827-1879.
61. Fallingborg J, Christensen LA, Ingeman-Nielsen M, Jacobsen BA, Abildgaard K, Rasmussen HH. pH-profile and regional transit times of the normal gut measured by a radiotelemetry device. *Aliment Pharmacol Ther*. 1989;3(6):605-613.
62. Osuka A, Shimizu K, Ogura H, et al. Prognostic impact of fecal pH in critically ill patients. *Crit Care*. 2012;16(4):R119.
63. Rohwer K, Bliss DZ, Savik K. Incontinence-associated dermatitis in community-dwelling individuals with fecal incontinence. *J Wound Ostomy Continence Nurs*. 2013;40(2):181-184.
64. Arnold-Long M, Reed LA, Dunning K, et al. Incontinence-associated dermatitis in a long-term acute care facility. *J Wound Ostomy Continence Nurs*. 2012;39(3):318-327.
65. Phipps L, Gray M, Call E. Time of onset to changes in skin condition during exposure to synthetic urine: a prospective study. *J Wound Ostomy Continence Nurs*. 2019;46:315-320.
66. Bliss DZ, Gurvich OV, Hurlow J, et al. Evaluation of validity and reliability of a revised incontinence-associated skin damage severity instrument (IASD. D. 2) by 3 groups of nursing staff. *J Wound Ostomy Continence Nurs*. 2018;45(5):449-455.
67. Bliss DZ, Funk T, Jacobson M, et al. Incidence and characteristics of incontinence associated dermatitis in community-living individuals with fecal incontinence. *J Wound Ostomy Continence Nurs*. 2015;42(5):525-530.
68. Bliss DZ, Hurlow J, Cefalu J, Mahlum L, Borchert K, Savik K. Refinement of an instrument for assessing incontinent-associated dermatitis and its severity for use with darker-toned skin. *J Wound Ostomy Continence Nurs*. 2014;41(4):365-370.
69. Koudounas S, Bader DL, Voegeli D. Investigating the release of inflammatory cytokines in a human model of incontinence-associated dermatitis. *J Tissue Viability*. 2021;30(3):427-433.
70. Koudounas S, Bader DL, Voegeli D. Elevated skin pH is associated with an increased permeability to synthetic urine. *J Wound Ostomy Continence Nurs*. 2021;48(1):61-67.
71. Bliss DZ, Savik K, Thorson MA, Ehman SJ, Lebak K, Beilman G. Incontinence-associated dermatitis in critically ill adults: time to development, severity, and risk factors. *J Wound Ostomy Continence Nurs*. 2011;38(4):433-445.
72. Clark M, Semple MJ, Ivins N, Mahoney K, Harding K. National audit of pressure ulcers and incontinence-associated dermatitis in hospitals across Wales: a cross-sectional study. *BMJ Open*. 2017;7(8):e015616.
73. Gray M, Giuliano KK. Incontinence-associated dermatitis, characteristics and relationship to pressure injury: a multisite epidemiologic analysis. *J Wound Ostomy Continence Nurs*. 2018;45(1):63-67.
74. Barakat-Johnson M, Barnett C, Lai M, Wand T, White K. Incontinence, incontinence-associated dermatitis, and pressure injuries in a health district in Australia: a mixed-methods study. *J Wound Ostomy Continence Nurs*. 2018;45(4):349-355.
75. Johansen E, Bakken LN, Duvaland E, et al. Incontinence-associated dermatitis (IAD): prevalence and associated factors in 4 hospitals in southeast Norway. *J Wound Ostomy Continence Nurs*. 2018;45(6):527-531.

76. Kayser SA, Phipps L, VanGilder CA, Lachenbruch C. Examining prevalence and risk factors of incontinence-associated dermatitis using the international pressure ulcer prevalence survey. *J Wound Ostomy Continence Nurs*. 2019;46(4):285-290.
77. Wang X, Zhang Y, Zhang X, Xian H. Incidence and risk factors of incontinence-associated dermatitis among patients in the intensive care unit. *J Clin Nurs*. 2018;27(21/22):4150-4157.
78. Junkin J, Selekof JL. Prevalence of incontinence and associated skin injury in the acute care inpatient. *J Wound Ostomy Continence Nurs*. 2007;34(3):260-269.
79. Kayser SA, Koloms K, Murray A, Khawar W, Gray M. Incontinence and incontinence-associated dermatitis in acute care. *J Wound Ostomy Continence Nurs*. 2021;48(6):545-552.
80. Van Damme N, Clays E, Verhaeghe S, Van Hecke A, Beeckman D. Independent risk factors for the development of incontinence-associated dermatitis (category 2) in critically ill patients with fecal incontinence: a cross-sectional observational study in 48 ICU units. *Int J Nurs Stud*. 2018;81:30-39.
81. Bliss DZ, Zehrer C, Savik K, Thayer D, Smith G. Incontinence-associated skin damage in nursing home residents: a secondary analysis of a prospective, multicenter study. *Ostomy Wound Manage*. 2006;52(12):46-55.
82. Bliss DZ, Savik K, Harms S, Fan Q, Wyman JF. Prevalence and correlates of perineal dermatitis in nursing home residents. *Nurs Res*. 2006;55(4):243-251.
83. Boronat-Garrido X, Kottner J, Schmitz G, Lahmann N. Incontinence-associated dermatitis in nursing homes: prevalence, severity, and risk factors in residents with urinary and/or fecal incontinence. *J Wound Ostomy Continence Nurs*. 2016;43(6):630-635.
84. Coyer F, Campbell J. Incontinence-associated dermatitis in the critically ill patient: an intensive care perspective. *Nurs Crit Care*. 2018;23(4):198-206.
85. Bliss DZ, Zehrer C, Savik K, Smith G, Hedblom E. An economic evaluation of four skin damage prevention regimens among nursing home residents with incontinence. *J Wound Ostomy Continence Nurs*. 2007;34(2):143-152.
86. Bliss DZ, Gurvich O, Savik K, et al. Incidence and predictors of incontinence associated skin damage in nursing home residents. *J Wound Ostomy Continence Nurs*. 2017;44(2):165-171.
87. Van Damme N, Van den Bussche K, De Meyer D, Van Hecke A, Verhaeghe S, Beeckman D. Independent risk factors for the development of skin erosion due to incontinence (incontinence-associated dermatitis category 2) in nursing home residents: results from a multivariate binary regression analysis. *Int Wound J*. 2017;14(5):801-810.
88. Faria DT, Shwayder T, Krull EA. Perineal skin injury: extrinsic environmental risk factors. *Ostomy Wound Manage*. 1996;42(7):28-30, 32-24, 36-27.
89. Brown DS, Sears M. Perineal dermatitis: a conceptual framework. *Ostomy Wound Manage*. 1993;39(7):20-22, 24-25.
90. Berg RW. Etiologic factors in diaper dermatitis: a model for development of improved diapers. *Pediatrician*. 1987;14(suppl 1):27-33.
91. Rodriguez-Palma M, Verdu-Soriano J, Soldevilla-Agreda JJ, Pancorbo-Hidalgo PL, García-Fernández FP. Conceptual framework for incontinence-associated dermatitis based on scoping review and expert consensus process. *J Wound Ostomy Continence Nurs*. 2021;48(3):239-250.
92. Lachenbruch C, Ribble D, Emmons K, VanGilder C. Pressure ulcer risk in the incontinent patient: analysis of incontinence and hospital-acquired pressure ulcers from the International Pressure Ulcer Prevalence™ Survey. *J Wound Ostomy Continence Nurs*. 2016;43(3):235-241.
93. Demarre L, Verhaeghe S, Van Hecke A, Clays E, Grypdonck M, Beeckman D. Factors predicting the development of pressure ulcers in an at-risk population who receive standardized preventive care: secondary analyses of a multicenter randomized controlled trial. *J Adv Nurs*. 2015;71(2):391-403.
94. Gates BP, Vess J, Long MA, Johnson E. Decreasing incontinence-associated dermatitis in the surgical intensive care unit: a quality improvement project. *J Wound Ostomy Continence Nurs*. 2019;46(4):327-331.
95. Beeckman D, Van Damme N, Schoonhoven L, et al. Interventions for preventing and treating incontinence-associated dermatitis in adults. *Cochrane Database Syst Rev*. 2016;11(11):CD011627.
96. Doughty D, Junkin J, Kurz P, et al. Incontinence-associated dermatitis: consensus statements, evidence-based guidelines for prevention and treatment, and current challenges. *J Wound Ostomy Continence Nurs*. 2012;39(3):303-315.
97. Lian Y. Barrier products in the treatment of incontinence-associated dermatitis. *Nurs Stand*. 2016;30(47):59-69.
98. Kon Y, Ichikawa-Shigeta Y, Iuchi T, et al. Effects of a skin barrier cream on management of incontinence-associated dermatitis in older women. *J Wound Ostomy Continence Nurs*. 2017;44(5):481-486.
99. Sugama J, Sanada H, Shigeta Y, et al. Efficacy of an improved absorbent pad on incontinence-associated dermatitis in older women: cluster randomized controlled trial. *BMC Geriatr*. 2012;12:22.

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