**New insights on Incontinence-Associated Dermatitis.**

**David Voegeli. PhD, BSc, RN.**

**Associate Professor of Nursing,**

**Skin Health Research Group,**

**Faculty of Health Sciences,**

**University of Southampton,**

**A Level South Academic Block,**

**University Hospital Southampton**

**Southampton.**

**SO16 6YD.**

**Tel: +44 (0)23 8077 7222 Extn: 3162.**

**Email:** [**D.Voegeli@soton.ac.uk**](mailto:D.Voegeli@soton.ac.uk)

**Incontinence-associated Dermatitis.**

**Introduction.**

Prolonged contact of the skin with urine or faeces leads to a specific form of moisture associated skin damage, known as incontinence associated dermatitis (IAD). While this is a common condition encountered in all areas of nursing practice, gaps remain in our understanding of the many contributing factors. A lack of standardised definitions of IAD, differences in terminology, and a bewildering increase in products available to prevent and manage IAD, makes it difficult for nurses to deliver evidence-based care. However it is an area where nursing research has made a considerable contribution over the past few years, culminating in the development and publication of a set of international best practice principles based on expert consensus (Beeckman et al, 2015). This paper explores the main principles developed and the implications for nursing practice.

**What is IAD?**

IAD may be regarded as a type of contact dermatitis that occurs in patients who have urinary of faecal incontinence, often causing significant discomfort, and reduced quality of life (Doughty et al, 2012). Typically IAD presents as inflammation of the skin surface characterised by redness, and in extreme cases, swelling and blister formation (Figure 1). In urinary incontinence this generally affects the labia in women, and the scrotum in men, as well as the inner thigh and buttocks in both sexes. If untreated this usually rapidly leads to excoriation and skin breakdown, which may subsequently become infected by the skin flora, leading to a viscous circle of increased inflammation and skin breakdown. It is generally agreed that urinary incontinence on its own does not necessarily lead to IAD, but combined with faecal incontinence or the passage of liquid stool significantly increases the risk. There is also the possibility that certain medications the patient might be taking also contributes, either by a direct action of drug metabolites excreted in the urine or faeces on the skin, or by drugs increasing the passage of liquid stools as a side effect, such as some antibiotics (Shiu et al, 2013). The exact mechanisms that lead to IAD are not fully understood, although some significant insights have been achieved over the past few years, mainly driven by a continued interest in this area by nurse researchers. The main factors precipitating the skin inflammation are thought to be overhydration of the epidermis and an increase in the skin pH away from the protective acidic range, disrupting the normal skin barrier (Voegeli, 2012).

**How many patients are affected?**

It is suggested that as many as half of all of nursing home residents and 10–35% of community-dwelling adults may be affected by urinary incontinence (Newman et al. 2007). Similarly faecal incontinence has been reported in 23–66% of nursing home residents (Newman et al. 2007). Not surprisingly IAD is reported as being a significant problem across the world. However it is likely that the prevalence of IAD is underestimated (Borchert et al, 2010) due to a variety of reasons. In many countries data is not collected, and where it is there are often problems recognising it or confusing it with pressure damage (Beeckman et al, 2014). Where data does exist there is often confusion in the way it is presented, with the terms prevalence and incidence being used interchangeably. The prevalence of IAD (i.e. proportion of patients at a defined time point) varies from 5.6% to 50% and is highest in those with faecal incontinence (Gray et al, 2007). Whilst the incidence of IAD (proportion of patients developing IAD over time) is 3.4% to 25% (Long et al, 2010). Given the large number of patients affected, the prevention and management of IAD presents a significant financial burden for healthcare systems. In England alone 903,500 prescriptions for barrier products were issued in 2014, at a cost of £3.27 million (HSCIC, 2014). However the true cost is likely to be much higher, with many patients self-funding their own preferred product.

**Normal skin barrier and IAD.**

One of the major functions of healthy skin is the maintenance of a physical barrier against the external environment. This barrier function prevents the entry of harmful substances and pathogens, provides a well-developed immunological defence mechanism, as well as acting as an important moisture barrier, preventing excessive fluid gain and loss from the body. This is principally achieved by the uppermost layer of the skin, the epidermis and in particular the outermost part of the epidermis, the stratum corneum. The structure of the stratum corneum is classically viewed as a ‘bricks and mortar’ arrangement (Figure 2). Protein-rich corneocytes act as the bricks, held together by a lipid-rich matrix mortar and protein ‘rivets’ called desmosomes. Enzymes within the epidermis act on phospholipids to produce a mixture of ceramides, free fatty acids and cholesterol (Harding, 2004) which help to regulate stratum corneum function. As well as a rigid protein structure, the corneocytes contain substances that actively attract and hold water in the stratum corneum. Collectively they are known as natural moisturizing factor (NMF) and the increase in intracellular water they promote helps the corneocytes to retain their turgidity and shape, thus maintaining a flexible, coherent barrier (Voegeli 2012). The skin barrier is further enhanced by the maintenance of an acidic surface pH (normally between 4 to 6) termed the acid mantle. Not only does this help maintain a healthy balance of resident skin bacteria, but it is now recognised that skin pH plays an important role in regulating skin health and stratum corneum cohesion (Ali and Yosipovitch, 2013).

If urine is allowed to remain in contact with the skin for prolonged periods of time, the excessive moisture overwhelms normal moisture regulation leading to overhydration and disruption of the stratum corneum structure, which may present as maceration (Ichikawa-Shiegeta et al, 2014). The urea in urine can be broken down by the skin bacteria to form the highly alkaline ammonia, which shifts the pH of the skin further disrupting the barrier. If faeces are present, the change to a more alkaline pH activates enzymes present in the faeces, which then further contribute to the damage caused to the epidermis. Liquid stool tends to be richer in digestive enzymes (lipases and proteases), which, when combined with its elevated water content, is particularly damaging to the skin (Gray et al 2007, Beeckman et al 2009). It is thought that penetration of skin bacteria through the damaged barrier also plays a role in the development of the inflammatory component of IAD. Evidence for this was provided by Houwing et al (2007) who studied tissue samples from patients with IAD and grade 1 pressure ulcers. They found that ischaemic changes were evident in the pressure ulcers, whilst the moisture lesions demonstrated inflammatory changes similar to those seen in contact dermatitis. More recently Mugita et al, 2015 have explored the mechanisms of IAD in an animal model. They have shown that the histology of IAD is distinct from contact dermatitis and demonstrate the ability of gut flora to penetrate the skin, along with proteolytic enzymes to produce inner tissue damage. The end result of these processes is the initiation of an inflammatory response, and IAD, which if not managed correctly sets up a vicious cycle that further drives the inflammation and skin breakdown. This may be further complicated by secondary infection of the damaged skin by pathogens, with fungal infection being very common.

**Patient Assessment**

Numerous risk factors for the development of IAD have been identified (Table 1), the main ones being incontinence of urine, faeces (or both), frequency of incontinence episodes, use of occlusive containment products, pre-existing skin condition poor mobility / dexterity, and inability to maintain personal hygiene (Kottner et al, 2014). Interestingly, although many of these risk factors are more common with ageing, age itself does not appear to be an independent risk factor for the development of IAD (Kottner et al, 2014).

Patients at risk of developing IAD should have their skin assessed at least daily, or more frequently if they are considered to be at very high risk. This should form part of a general skin assessment and can easily be incorporated into routine skin inspection for pressure ulcer risk (Beeckman et al, 2015). Although tools exist for assessing IAD risk (Storer-Brown 1993, Nix 2002) they have not gained popularity in clinical practice and there is the danger that they can become confused with Pressure Ulcer risk assessment tools, and vice versa. Similarly several tools have been developed to guide skin assessment and IAD severity (Table 2), but again their use in practice is limited. They suffer many of the same problems as many other assessment tools, in that overall there is limited evidence that they actually improve clinical decision making or patient care. Current recommendations advise using a simple 3 stage IAD severity tool (Table 3; Beeckman et al, 2015) to assess, guide interventions and importantly, evaluate whether these are working.

Doubt often exists as to whether the skin damage is IAD, pressure damage or a reaction to something that has come into contact with the skin. Often the obvious is missed, in that if the patient is not incontinent then it’s not IAD. However as those with incontinence are often at increased risk of pressure ulcers, it can be difficult to distinguish between the two (Mahoney et al, 2013). The main differences centre on the cause, location, and appearance of the skin damage (Table 4). Pressure ulcers usually form over a bony prominence as a result of unrelieved pressure or friction, whilst moisture lesions are often more widespread and the skin usually looks shiny due to presence of moisture. The edges of a pressure ulcer are generally distinct, producing a wound with a regular or circular shape, as opposed to the widespread and diffuse appearance of a moisture lesion. However it is important to remember that although distinctions can be made between the two, they can co-exist and are closely related. A recent systematic review and meta-analysis by Beeckman et al (2014) concluded that there was a probable association between IAD and pressure ulcer development, and a meta-analysis of pressure ulcer risk assessment scales showed that their predictive capability was increased if incontinence / moisture was included (Garcia-Fernandez et al, 2014). Demonstrating the importance of taking a holistic view to skin care.

**Prevention & Management**

Identification of urinary or faecal incontinence during the nursing assessment should lead to the implementation of appropriate protocols aimed at preventing IAD or promoting healing if skin damage is already present, and link to those for preventing pressure ulcers. Protocols for the prevention or management of IAD should address two key aspects together:

1. Assessing and managing the incontinence,

2. Using a structured skin care regime.

The simple purpose of assessing and managing incontinence is to reduce exposure of the patient’s skin to the main causative agents of IAD. It also provides an ideal opportunity to identify reversible causes of incontinence, such as medication (e.g. diuretic therapy in someone with impaired mobility), urinary tract infection, and constipation (Gray 2010). Simple incontinence management interventions should be tried first, such as optimising nutritional and fluid intake, urinary sheaths for men, and ‘toileting’ techniques. Combined with the use of appropriate absorbent products, that are designed to wick moisture away from the skin, avoid overhydration and occlusion of the skin, these initial steps are often effective (Langemo et al, 2011). In patients with severe IAD and excoriated skin, more complex interventions may be needed to ensure the skin does not come into contact with irritants and promote healing, such as urinary catheters and faecal management systems (Black et al, 2011, Morris 2011). However in the case of urinary catheters, these should always be regarded as the last resort due to the substantial risk of infection. It is recommended that if there is no improvement in the condition of the patient’s skin after 3 to 5 days of initiating a continence management plan, then a reassessment is made and specialist advice sought (Beeckman et al, 2015).

Ensuring optimal skin care is provided following each major incontinence episode, particularly if faeces are present, is one of the most important actions that can be taken to prevent IAD. The skin care provided should be based on a structured regime and involve the use of a skin cleanser and a protectant. The use of ordinary soap and water should be avoided as in most cases the pH of the soap is too alkaline, and may contribute to the skin irritation (Voegeli 2008; Voegeli 2012). Following cleansing the skin needs to be protected against subsequent contact with urine or faeces by using a skin protectant or barrier product. These are designed to repel moisture and protect the skin from the harmful effects of the incontinence. Basic barrier preparations generally consist of a lipid / water emulsion base with the addition of metal oxides (e.g. zinc or titanium) which form a thin layer on the surface of the skin to repel potential irritants. The more sophisticated ones often contain a water repellent silicone-based ingredient such as demeticone, as well as mild antiseptic agents such as cetrimide or benzalkonium. Unfortunately there is the potential for some of these ingredients to cause irritation in sensitive individuals, with even seemingly innocuous preparations such as zinc and castor oil cream containing arachis (peanut) oil. This should always be kept in mind, particularly if skin irritation appears to worsen when using any preparation. Advances in polymer science have led to the development of a new generation of product, which allow a thin semi-permeable protective silicone-based polymer coating to be applied to the skin. It would appear that in some situations these polymers have an advantage over more conventional products, by offering greater protection against repeated moisture exposure. Many of the newer products available combine a no-rinse cleanser with a protectant and are pH balanced to help maintain the skin’s acid mantle. If assessment of the skin breakdown suggests that fungal infection is present, then the incorporation of an antifungal cream into the skin care regime will be needed as well.

Concern has been expressed that the use of barrier products, particularly greasy creams and ointments might ‘clog’ incontinence pads, leading to pad failure and leakage, a problem that can certainly occur with the overuse of talcum powder. Early work by Bolton et al (2004) suggested that, if applied sparingly, according to manufacturers’ instructions, most barrier products are safe to use in combination with incontinence pads and do not significantly affect pad performance. However the range of products developed for IAD has increased significantly since then, coupled with advances in material science and pads, therefore further work is needed to build up a picture of the performance of all products currently available to help clinicians choose the right product for their patient.

**Conclusion**

IAD is a common problem affecting as many as half of the patients with urinary or faecal incontinence that are managed with absorptive products. The high incidence of incontinence in the older population, particularly those in long-term care settings means they are at increased risk. Confusion still exists in separating IAD from pressure damage, but careful assessment can help distinguish between the two and enable appropriate prevention and treatment. Key factors in the successful prevention and management are careful patient assessment, good continence care, and clear evidence-based skin care protocols. All of which can improve the patient experience, and improve clinical outcomes, thus demonstrating once again the importance of good ‘fundamental’ care.

**Key Points:**

* IAD is a very common inflammatory skin disorder seen in patients with urinary or faecal incontinence.
* Confusion exists in differentiating sin breakdown due to IAD from pressure ulcers.
* If the patient isn’t incontinent, they don’t have IAD.
* Prevention and management relies on careful patient assessment, good continence care, and clear evidence-based skin care protocols.

**References.**

Ali S.M., Yosipovitch, M. (2013) Skin pH: from basic science to basic skin care. *Acta. Derm. Venereol.* 93, pp261-267.

Beeckman D, Schoonhoven L, Verhaeghe S, Heyneman A, Defloor T. (2009) Prevention and treatment of incontinence-associated dermatitis: literature review. *Journal of Advanced Nursing*. 65(6), p1141–1154.

Beeckman D, Woodward S, Gray M (2011) Incontinence-associtated dermatitis: step-by-step prevention and treatment. *Br J Community Nurs* 16(8), pp382–389.

Beeckman D, Van Lancker A, Van Hecke A, Verhaeghe S.(2014) A systematic review and meta-analysis of incontinence-associated dermatitis, incontinence and moisture as risk factors for pressure ulcer development. *Research in Nursing and Health.* 37, pp204-218.

Beeckman D, et al. (2015) Proceedings of the Global IAD Expert Panel. Incontinence-associated dermatitis: moving prevention forward. *Wounds International* available at: <http://www.woundsinternational.com/media/other-resources/_/1154/files/iad_web.pdf>

Black JM, Gray M, Bliss DZ, Kennedy-Evans KL, Logan S, Baharestani MM, Colwell JC, Goldberg M, Ratliff CR (2011) MASD part 2: incontinence-associated dermatitis and intertriginous dermatitis: a consensus. *Journal of Wound Ostomy and Continence Nursing*. 38(4), p 359-370.

Bolton C. Flynn R. Harvey E. Morris J. (2004) Assessment of pad clogging. *Journal of Community Nursing*. 18(6), pp18-20.

Borchert K, Bliss D, Savik K, Radosevich DM. (2010) The incontinence-associated

dermatitis and its severity instrument: development and validation. *J Wound Ostomy Continence Nurs*. 37(5), pp527-535.

Defloor T, Schoonhoven L, Fletcher J. et al (2005) Statement of the European Pressure Ulcer Advisory Panel —Pressure Ulcer Classification Differentiation Between Pressure Ulcers and Moisture Lesions. *J Wound Ostomy Continence Nurs*. 32(5), pp302-306.

Doughty D, Junkin J, Kurz P et al. (2012) Incontinence-associated dermatitis.

Consensus statements, evidence-based guidelines for prevention and treatment, current challenges. *J Wound Ostomy Continence Nurs*. 39(3), pp303-315.

Garcia-Fernandez F.P, Pancorbo-Hidalgo P.L, Agreda J.J.S. (2014) Predictive capacity of risk assessment scales and clinical judgement for pressure ulcers: a meta-analysis. *J Wound Ostomy Continence Nurs.* 41, pp24-34.

Gray M. (2010) Optimal management of incontinence-associated dermatitis in the elderly. *American Journal of Clinical Dermatology.* 11(3), pp201-210.

Gray M. Bliss DZ. Doughty DB. Ermer-Seltun J. Kennedy-Evans KL. Palmer MH. (2007) Incontinence-associated dermatitis: a consensus. *J Wound Ostomy Continence Nurs*. 34(1), pp45-54.

Harding, CR (2004) The stratum corneum: structure and function in health and disease. *Dermatologic Therapy*, 17, pp6-15.

Houwing RH, Arends JW, Canninga-van Dijk MR, Koopman E, Haalboom JR (2007) Is the distinction between superficial pressure ulcers and moisture lesions justifiable? A clinical-pathologic study. *SKINmed* 6(3): p113–1177.

HSCIC (2014) Prescription Cost Analysis: England 2014 available at: [www.hscic.gov.uk](http://www.hscic.gov.uk)

Ichikawa-Shiegeta Y, Sugama J, Sanada H, Nakatani T, Konya C, Nakagami G, Minematsu T, Yusuf S, Supriadi, Mugita Y. (2014) Physiological and appearance characteristics of skin maceration in elderly women with incontinence. *J Wound Care*. 23(1), pp18-30.

Junkin J. (2014) An incontinence assessment and intervention bedside tool (IAD IT)

assists in standardising the identification and management of incontinence

associated dermatitis. Poster presented Wounds UK, Harrogate.

Kottner J, Blume-Peytavi U, Lohrmann C, Halfens R. (2014) Associations between

individual characteristics and incontinence-associated dermatitis: A secondary data analysis of a multi-centre prevalence study. *Int J Nurs Studies*. 51: pp1372-1380.

Langemo D, Hanson D, Hunter S, Thompson P, Oh I.E. (2011) Incontinence and incontinence-associated dermatitis. *Adv Skin Wound Care*. 24(3), pp126-140.

Long M, Reed L, Dunning K, Ying J. (2012) Incontience-associated dermatitis in a

long-term acute care facility. *J Wound Ostomy Continence Nurs.* 39(3), pp318-27.

Mahoney M, Rozenboom B, Doughty D. (2013) Challenges in classification of gluteal

cleft and buttock wounds. *J Wound Ostomy Continence Nurs.* 40(3), pp239-245.

Morris L. (2011) Flexi-Seal® faecal management system for preventing and

managing moisture lesions. *Wounds UK.* 7(2), pp88-93.

Mugita Y, Minematsu T, Huang L, Nakagami G, Kishi C, Ichikawa Y, et al. (2015) Histopathology of Incontinence-Associated Skin Lesions: Inner Tissue Damage Due to Invasion of Proteolytic Enzymes and Bacteria in Macerated Rat Skin. *PLoS ONE* 10(9): e0138117. doi:10.1371/journal.pone.0138117.

National Association for Tissue Viability Nurses (Scotland) (2009) Skin Excoriation Tool For Incontinent Patients. Available online: <http://www.healthcareimprovementscotland.org/programmes/patient_safety/tissue_viability_resources/excoriation_tool.aspx>

Newman D, Preston A, Salazar S. (2007) Moisture control, urinary and faecal incontinence, and perineal skin management. In *Chronic Wound Care: A Clinical Source Book for Healthcare Professionals*, 4th edn (Krasner D., Rodeheaver G, Sibbald R, eds), HMP Communications, Malvern, pp. 609–627.

Nix DH. (2002) Validity and reliability of the perineal assessment tool. *Ostomy Wound Manage.* 48(2), pp43-49.

Shiu SR , Hsu MY, Chang SC , et al. (2013) Prevalence and predicting factors of

incontinence-associated dermatitis among intensive care patients. *J Nurs*

*Healthcare Res.* 9(3), pp210.

Storer-Brown D. (1993) Perineal dermatitis: can we measure it? *Ostomy Wound*

*Manage.* 39(7), pp8-32.

Voegeli D. (2008) The effect of washing and drying practices on skin barrier function. *J Wound Ostomy Continence Nurs*. 35(1), pp84-90.

Voegeli D. (2012) Moisture-associated skin damage: aetiology, prevention and

treatment. *Br J Nurs.* 21(9), pp517-521.

**Figures and tables:**

**Figure 1 – Severe incontinence-associated dermatitis:**



Taken from McDonagh D. (2008) Moisture lesion or pressure ulcer? A review of the literature. Journal of wound Care. 17(11), p461-466 and previously used in: Voegeli D. (2012) Moisture-associated skin damage: aetiology, prevention and treatment. *Brit. Journ. Nurs.* 21(9), pp517-521.

**Figure 2 – Structure of Stratum Corneum.**

Please use figure 2 from: Voegeli D. (2012) Moisture-associated skin damage: aetiology, prevention and treatment. *Brit. Journ. Nurs*. 21(9), pp517-521.

**Table 1: IAD risk factors.**

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| **Main risk factors for IAD:** |
| **Incontinence:**  Faecal, Urinary, Double incontinence |
| **Frequent episodes of incontinence** |
| **Use of occlusive containment products** |
| **Poor existing skin condition** |
| **Reduced mobility** |
| **Diminished cognitive awareness** |
| **Inability to maintain personal hygiene** |
| **Pain** |
| **Increased body temperature** |
| **Medications** (steroids, antibiotics) |
| **Poor nutritional status** |
| **Critical illness** |

Adapted from Beeckman D, et al. (2015) Proceedings of the Global IAD Expert Panel. Incontinence-associated dermatitis: moving prevention forward. Wounds International available at: <http://www.woundsinternational.com/media/other-resources/_/1154/files/iad_web.pdf>

**Table 2: Example IAD assessment tools.**

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| --- | --- |
| **IAD Assessment tools** | |
| **IAD Assessment and Intervention Tool (IADIT)** | Junkin J. (2014) |
| **Incontinence-associated dermatitis and its severity (IADS)** | Borchert K, Bliss DZ, Savik K, Radosevich DM (2010) |
| **Skin Assessment Tool** | Beeckman D, Woodward S, Gray M (2011) |
| **Skin Excoriation Tool For Incontinent Patients** | National Association for Tissue Viability Nurses (Scotland) (2009) |

**Table 3: Recommended 3 Stage Severity Scoring for IAD:**

|  |  |
| --- | --- |
| **Severity of IAD** | **Signs** |
| No redness and skin intact (at risk) | Skin is normal compared to rest of body, no signs of IAD |
| Category 1 – Red but intact skin (mild IAD) | Erythema ± oedema |
| Category 2 – Red with skin breakdown (moderate – severe IAD) | As for category 1 plus:  Vesicles / bullae/ Skin erosion;  Denudation of skin;  Infection. |

Recommended 3 stage severity scoring system for IAD adapted from Beeckman D, et al. (2015) Proceedings of the Global IAD Expert Panel. Incontinence-associated dermatitis: moving prevention forward. Wounds International available at: <http://www.woundsinternational.com/media/other-resources/_/1154/files/iad_web.pdf>

**Table 4: Differences between Superficial Pressure Ulcers and Moisture Lesions (Adapted from Defloor et al 2005).**

|  |  |  |
| --- | --- | --- |
|  | **Pressure Ulcer** | **Moisture Lesion** |
| **Causes** | Pressure and/or shear must be present. | Moisture must be present (eg, shining wet skin caused by urinary incontinence or diarrhoea). |
| **Location** | A wound not over a bony prominence is unlikely to be a pressure ulcer. | A moisture lesion may occur over a  bony prominence. However, pressure  and shear should be excluded as  causes and moisture should be  present.  A combination of moisture and friction  may cause moisture lesions in skin  folds.  A lesion that is limited to the anal cleft  only and has a linear shape is not a  pressure ulcer and is likely to be a  moisture lesion.  Perianal redness/skin irritation is most  likely to be a moisture lesion  resulting from faeces. |
| **Shape** | Circular wounds or wounds with a regular shape are most likely pressure ulcers | Diffuse different superficial spots are  more likely to be moisture lesions. |
| **Depth** | Partial-thickness skin loss is present when only the top layer of the skin is damaged (Grade 2).  In full-thickness skin loss, all skin layers are damaged (Grade 3 or 4). | Moisture lesions are superficial (partial thickness skin loss).  In cases where the moisture lesion gets  infected, the depth and extent of the  lesion can be enlarged/deepened  extensively. |
| **Edges** | If the edges are distinct, the lesion is most likely a pressure ulcer. | Moisture lesions often have diffuse or  irregular edges. |
| **Colour** | If redness is nonblanchable, this is most likely a pressure ulcer Grade 1. | If the redness is not uniformly distributed, the lesion is likely to be a moisture lesion (exclude pressure and shear as causes).  *Pink or white surrounding skin:*  Maceration resulting from moisture. |