Back to basics: understanding moisture-associated skin damage

KEY WORDS
- Moisture-associated skin damage (MASD)
- Incontinence-associated dermatitis
- Intertriginous dermatitis
- Periwound MASD
- Peristomal MASD

This article discusses the aetiology and clinical presentations of the four conditions collectively known as moisture-associated skin damage (MASD). It explores the normal status of the skin and how this is altered in the presence of excess and prolonged moisture. In all manifestations the barrier property of the skin is impaired, allowing irritants to penetrate the epidermis. Moisture damage disrupts the lipid matrix surrounding the cells in the stratum corneum and effectively dissolves the physical barrier of the skin. The pH of the skin also increases, resulting in an alkaline environment conducive to bacterial proliferation and infection. Urine and faeces are the offending contaminants in incontinence-associated dermatitis, whereas perspiration is the primary cause of intertriginous dermatitis. Effluent from the ostomy causes peristomal damage and wound exudate instigates moisture damage to the periwound skin. The clinical picture of MASD ranges from mild erythema to extensive and painful skin breakdown often complicated by bacterial and fungal infection. A structured skin-care regimen that involves cleansing, protecting and restoring the barrier properties of the skin will assist in the prevention and management of all MASD.

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Moisture-associated skin damage (MASD) is the umbrella term for four clinical manifestations, namely incontinence-associated dermatitis (IAD), intertriginous dermatitis (ITD), periwound moisture-associated dermatitis and peristomal moisture-associated dermatitis. Excess moisture and the associated chemical irritants cause MASD. The difference between the four conditions is the type of moisture that induces the skin damage. Urine and faeces cause IAD, ITD is caused by perspiration, peristomal damage is due to effluent from the stoma coming into contact with the skin, and periwound moisture-associated dermatitis is a result of wound exudate being present on the skin surrounding the wound (Gray et al, 2011; Beeckman et al, 2017).

The global prevalence of MASD is not accurately known, with figures varying greatly between clinical settings and geographical locations (Gray et al 2011; 2013; Kottner et al, 2014; Beeckman et al, 2017). A recent national audit in Wales in which the skin of 8,365 hospital inpatients was inspected identified IAD in 4.3% of the cohort (Clark et al, 2017).

The lack of global prevalence data is not helped by the lack of consensus on the terminology to describe the injured tissue and lack of validated and accepted data collection method (Beeckman et al, 2015). The heterogeneity and variation of reported outcomes in research studies on IAD was highlighted in a recent Cochrane review on the subject (Beeckman et al, 2016). Misdiagnosis, especially in relation to IAD and category II pressure ulceration, hinders the accuracy of data collected. Clinicians are faced with confusion and uncertainty when trying to diagnose superficial damage on the sacrum, which could be due to pressure damage, IAD or a combination of both (National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance, 2014).

The recent publication of the Ghent Global IAD (GLOBIAD) categorisation tool has created an

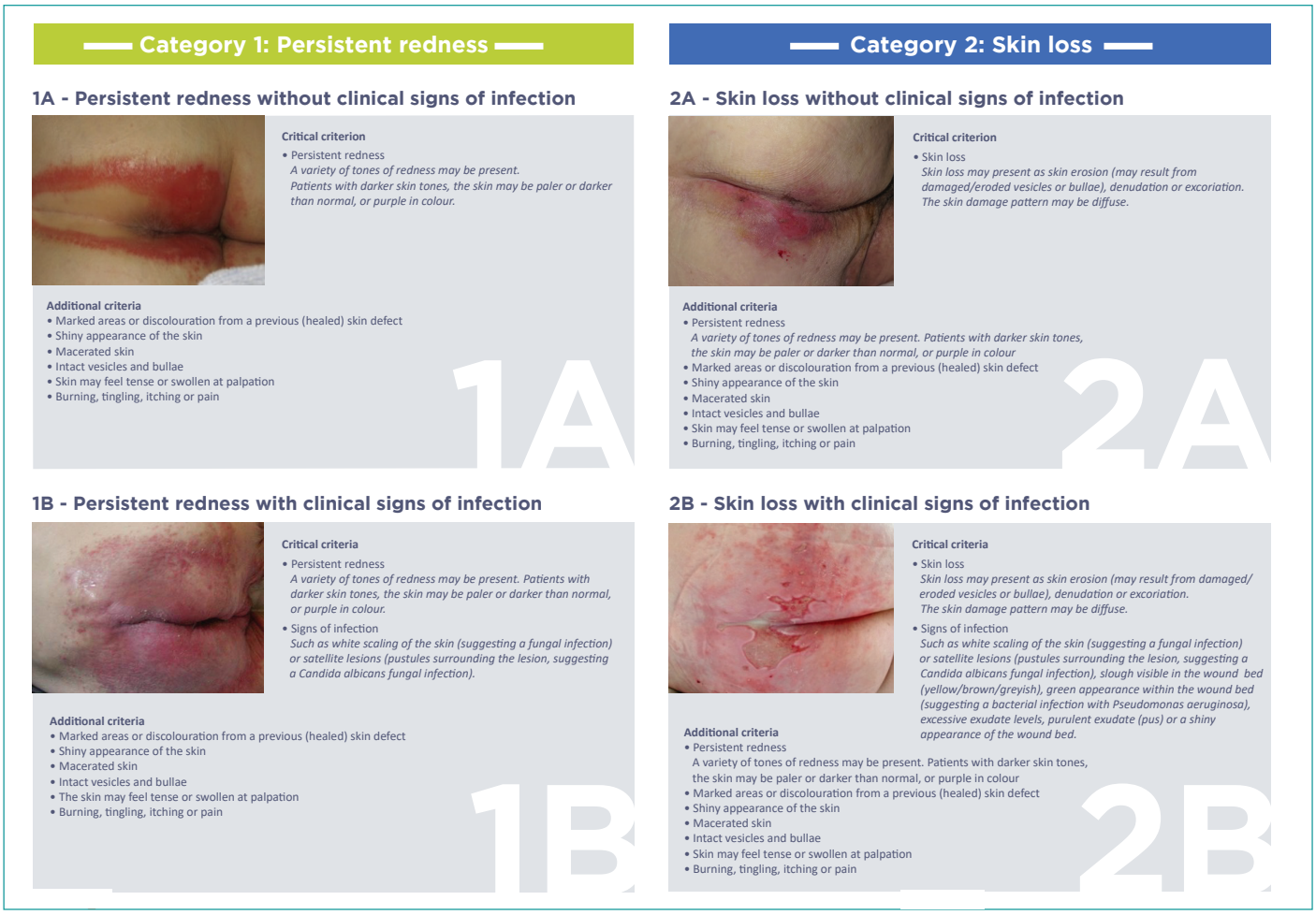

Figure 1. The Ghent Global IAD (GLOBIAD) categorisation tool (with kind permission from Dimitri Beeckman from Ghent University)

internationally-agreed description of the clinical presentation of IAD (*Figure 1).* The GLOBIAD tool was developed as a 2-year research project involving 22 international experts and 832 clinicians from 30 countries. The tool categorises IAD from its initial presentation to the more severe forms that are exacerbated by infection. Use of the tool in clinical practice and research will standardise the description of IAD by facilitating accurate recording of the severity of damage based on a visual inspection of the affected areas (Beeckman et al, 2017).

BARRIER PROPERTIES OF THE SKIN

It is important to understand how the barrier properties of the skin are formed and how they function in order to comprehend the damage caused by MASD. The skin is composed of five layers, with the outer layer the stratum corneum providing the main barrier function. There are 15–20 layers of corneocytes in the stratum corneum that have evolved from keratinocytes in the basal layer of the epidermis. The cells are held together by proteins called desmosomes and a lipid matrix (Voegeli, 2016). The lipids of the stratum corneum are composed of ceramides, cholesterol and free fatty acids and are collectively termed the lipid lamellae (Moncrief et al, 2013). The compact assembly of the cells in the stratum corneum has been likened to a brick wall that prevents substances from passing through into the deeper layers of the epidermis (Penzer et al, 2012).

The barrier function of the epidermis helps to regulate passive water loss, which is known as trans-epidermal water loss (TEWL). A high TEWL exists when the skin barrier function is weakened. Ideally the barrier function of the skin should prevent excessive fluid gain or

loss (Voegeli, 2016). A normal water content is necessary for the correct functioning and maturation of the epidermis (Ousey et al, 2016). The cells of the stratum corneum also contain a natural moisturising factor that helps to maintain hydration of the epidermis by attracting and holding water in the cells (Voegeli, 2016).

EFFECT OF MOISTURE ON THE SKIN

Overhydration of the skin causes disruption of the barrier properties of the stratum corneum and allows irritants to penetrate the epidermis. The moisture damage disrupts the lipid matrix surrounding the corneocytes and effectively dissolves the physical barrier (Voegeli, 2016), leaving it more vulnerable to trauma from pressure and friction (Beeckman et al, 2015).

The pH of healthy skin is between 4 and 6, providing an acidic environment that supports the resident, commensal bacteria on the surface of the skin. In overhydration, the pH of the skin increases, resulting in an alkaline environment that is conducive to bacterial proliferation and infection. If urine is the offending cause of the moisture, the urea it contains is converted to the alkaline substance ammonia (Beeckman et al, 2015).

Mature skin is more susceptible to breakdown because of the ageing process, e.g. slow epidermal turnover, fewer sweat glands and less sebum production (Minematsu et al, 2011; Penzer et al, 2012; Moncrief et al, 2013; Beeckman et al, 2017).

INCONTINENCE-ASSOCIATED DERMATITIS

IAD is the most commonly recognised form of MASD. This is mainly due to the work of the global IAD Expert Panel and its publication on best practice principles (Beeckman et al, 2015). Prevention of IAD is based upon the assessment and management of incontinence.

IAD is a form of contact dermatitis. The substances responsible for causing IAD are urine and/or faeces (Beeckman et al, 2017). Faeces contain enzymes that damage the stratum corneum (Mugita et al, 2015). Liquid faeces causes more damage than solid faeces as the enzymes are more destructive in the liquid form (Campbell et al, 2016). The enzymes in faeces also exacerbate the effects of urine on the skin, hence incontinence of urine and faeces is more damaging to the skin than either type of incontinence on its own. Skin damage is normally found in the perianal area, although it can extend further depending on the degree of the incontinence and speed with which the contaminants are removed from the skin (Beeckman et al, 2015).

Initially IAD presents as erythema of the skin, which may be patchy or completely cover the affected area. The erythema is a result of inflammation of the skin and therefore will feel warm to the touch. The erythema may develop into superficial wounds (loss of epidermis) and may present with vesicles, bullae, papules or pustules (Beeckman et al, 2015). The skin may also present with a glossy surface (Nakagami et al, 2006). IAD can cause considerable pain (often burning in nature) and suffering for the individual, especially following each episode of incontinence.

The factors that place an individual at risk of developing IAD in addition to incontinence are fragile skin, compromised mobility, diminished cognitive awareness, inability to perform personal hygiene, pain, pyrexia medication, poor nutritional status and critical illness (Beeckman et al, 2015). Furthermore, Kottner et al (2014) specify that an increased body mass index (BMI) and diabetes mellitus are risk factors for IAD. They identified these factors following a secondary data analysis of a multicentre prevalence study (the Netherlands and Austria) to identify the personal and health-related variables most strongly related to the development of IAD. Over 3,500 individuals were included in the data set. A high BMI and diabetes were found to be statistically significant characteristics of individuals with IAD. The purpose of this work was not to prove cause and effect; however the authors suggest that a high BMI may increase the occlusive effects on the skin's surface and the chronically elevated serum glucose levels associated with diabetes can result in pathological changes in all tissues including the skin, triggering an increased susceptibility to injury and infection.

IAD can be exacerbated by inappropriate care delivery, such as a delay in cleaning an individual after an episode of incontinence, incorrectly fitted and infrequent changing of continence garments/pads, or blocking of the absorptive capacity of

continence garments with skin barrier products. The staff time required to clean an individual is a major factor in the health-related costs associated with IAD (Morris, 2011).

IAD is a recognised risk factor for pressure ulceration and the two conditions can coexist in an individual (Lachenbruch et al, 2016). PuClas 4 is an excellent on-line collection of e-learning modules on pressure ulceration and IAD. The scientific content of the four modules is based on the joint 2014 National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance position statement on pressure ulcer classification and IAD differentiation, and provides an overview of causative factors and of typical wound-related characteristics. The modules can be accessed at http://puclas4.ucvvgent.be/

INTERTRIGINOUS DERMATITIS

ITD is inflammation of the skin folds related to perspiration, friction and bacterial/fungal bioburden. ITD occurs when moisture becomes trapped in the naturally large skin folds of the body, e.g. axillary and inguinal areas, although it can occur wherever two skin surfaces come into close contact with each other (Black et al, 2011). It can occur under the breasts and in children it can be found in the folds of the neck (Voegeli, 2016). In obese individuals, the skin folds are more pronounced and ITD is often found in the abdominal and pubic panniculi (Voegeli, 2016).

Factors predisposing to ITD are hyperhidrosis and incontinence (Black et al, 2011). The clinical presentation is one of shiny, moist erythema that can progress to skin erosion and breakdown. It can be difficult to distinguish IAD from ITD in skin folds exposed to urine and faeces (Black et al, 2011).

PERIWOUND MOISTURE-ASSOCIATED SKIN DAMAGE

Wound exudate contains proteolytic enzymes that damage the stratum corneum. There is a difference in the composition of wound exudate, with chronic wounds producing fluid with higher levels of proteolytic enzymes (Voegeli, 2016). There is an increase in the volume of exudate when infection is present (Vowden et al, 2015). Wound exudate can vary in viscosity depending on its constituents, e.g. biofilm material. In addition, its composition is influenced by periwound oedema and the position of the patient (hydrostatic pressure) (Vowden et al, 2015). Exudate with a high protein content will have a thick viscosity; it will be thin when low in protein.

The skin surrounding the wound becomes macerated and excoriated when it encounters wound exudate. Maceration is defined as a reversible pallor of the skin that is caused by overhydration and saturation of the skin (Gray and Weir, 2007). Excoriation is an injury to the skin caused by trauma, chemical or thermal burn (Dowsett et al, 2015).

PERISTOMAL MOISTURE-ASSOCIATED DAMAGE

Peristomal moisture-associated damage is a known complication of an ostomy, and is more commonly seen with colostomies and ileostomies (Gray et al, 2013; Voegeli, 2016). The damage occurs when the surrounding skin encounters effluent from the stoma. Consequently, the skin becomes inflamed and erosion can occur. The damage can occur soon after the initial surgery and reduces as the individual becomes more competent at caring for the stoma. Alternatively, it may develop later as the body shape changes (Voegeli, 2016). Management depends upon the correct choice and application of the containment device, along with a structured skin care routine (Gray et al, 2013).

SECONDARY SKIN INFECTION

MASD places the individual at risk of developing a secondary skin infection, commonly *Candida albicans* (Beeckman et al, 2015). This can be difficult to diagnose. Microbiological cultures can aid the identification of the fungal species (Beeckman et al, 2015). *Candida albicans* is treated with topical antifungal creams or powders. These treatments are not recommended, however, as part of preventive care in MASD (Beeckman et al, 2015).

STRUCTURED SKIN CARE ROUTINE

The prevention and treatment of MASD should

Table 1. Interventions for prevention and management based on the severity of incontinence-associated dermatitis (adapted from Beeckman et al, 2015)

Patient with urinary ± faecal incontinence	Actions			
	MANAGE INCONTINENCE EDUCATE PATIENTS AND CAREGIVERS	Cleanse*, protect† and restore‡		REFER FOR SPECIALIST ADVICE if there is no improvement within 3–5 days OR if a skin infection is suspected
No redness and skin intact (at risk)		Prevention: select option 1 or 2		
		1	Continence care wipe (3-in-1: cleanser + skin protectant + moisturiser) **ADD** skin protectant if extra skin protection is required	
		2	Skin cleanser OR bathing/cleansing wipe **PLUS** skin protectant	
GLOBIAD Category 1A: persistent redness		Management: select option 1 or 2		
		1	Continence care wipe (3-in-1: cleanser + skin protectant + moisturiser) **ADD** skin protectant (e.g. acrylate terpolymer barrier film) if worsening erythema/skin condition	
		2	Skin cleanser **OR** bathing/cleansing wipe **PLUS** skin protectant (e.g. acrylate terpolymer barrier film or dimethicone-containing product)	
GLOBIAD Category 2A: skin loss (moderate–severe)		1	Continence care wipe (3-in-1: cleanser + skin protectant + moisturiser) **ADD** skin protectant if worsening arythema/skin condition	
		2	Skin cleanser **OR** bathing/cleansing wipe **PLUS** skin protectant	
			AND consider containment devices (e.g. faecal management system/faecal pouch)	
GLOBIAD Categories 1A and 2B: With infection			As for category 2 **PLUS** take a microbiological sample when possible and use result to decide on appropriate therapy (e.g. antifungal cream, topical antibiotic, anti-inflammatory product)	

*Cleansing should take place daily and after each episode of faecal incontinence

†Skin protectants should be applied according to the manufacturer's instructions

‡For skin that is overhydrated or where maceration is present, do not use skin care products that trap moisture or are formulated to attract moisture

start with a thorough skin assessment that includes all skin folds. In obese individuals, the skin inspection may require the input of a team of staff.

Skin should be cleansed as soon as possible following every episode of incontinence. The skin should be cleaned with a pH-balanced cleanser. One containing surfactants will help to remove debris including faeces (Beeckman et al, 2014). Non-rinse cleansers prevent additional friction during the cleansing process.

Following cleansing of the skin, the use of barrier products can repel/protect the skin from subsequent excess moisture. Emollients are available in the form of ointments, creams, lotions and gels. Emollients can help to repair broken skin and keep intact skin supple and moisturised, and thus better able to protect itself from moisture damage. Emollients increase the amount of water held in the stratum corneum by rehydrating the corneocytes (Penzer et al, 2012). They work by providing a protective layer on the surface of the skin preventing TEWL, or alternatively they may contain humectants that actively hydrate the skin by drawing water from the dermis into the epidermis. Emollients containing humectants are not recommended for overhydrated skin, however, as they attract additional water to an already wet area (Penzer et al, 2012; Beeckman et al, 2015). The clinician needs to take skin overhydration into account when choosing an emollient to manage MASD.

The best practice principles for the prevention and management of IAD suggest that the structured skin care regimen be based upon the acronym CPR: cleanse, protect and restore (Beeckman et al, 2015), *Table 1*. A reduction in IAD was found following the introduction of a structured skin care routine for critically ill patients with faecal incontinence (Park and Kim, 2014). It could be postulated that the principles and positive impact of using a structured skin care

regimen in IAD could be extended to all aspects of MASD; however, further research is required to establish its effectiveness in other types of MASD.

CONCLUSION

MASD is the collective term for four types of moisture damage to the skin (IAD, ITD, periwound and peristomal damage). The damage is not caused by moisture alone; chemical irritants, proteolytic and lipolytic enzymes and an alteration in the skin pH all contribute to the destruction of the barrier function of the skin. In all cases, the damage can range from superficial erythema to extensive skin breakdown and can be complicated by bacterial and fungal infection. A structured skin care routine that involves cleansing, protecting and restoring damaged skin will help to prevent and manage MASD. WUK

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