Incontinence-Associated Dermatitis (IAD) and Pressure Ulcers: An Overview

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Introduction

Urinary incontinence is a global health problem affecting 8.2% of the 2008 world population (4.3 billion). By 2018, numbers will increase up to an estimated 423 million individuals being affected with urinary incontinence (21.6% of the population) [1]. Fecal incontinence is also common in men and women aged 65 and older, with a 17% incidence rate over 4 years. Fecal incontinence and urinary incontinence may share common pathophysiologic mechanisms and need regular assessment in older adults [2]. Incontinence is a debilitating disorder (physically, emotionally, socially and psychologically) and has an important impact on quality of life [3]. Not only patients are affected, but also their caring family, relatives and friends, professional caregivers and the society [1, 3].

Together with incontinence, the appearance and function of the skin are altered with aging, resulting in higher rates of skin complaints. One of the most common skin complaints associated with incontinence and ageing is incontinence-associated dermatitis (IAD) [4]. IAD is a type of irritant contact dermatitis that is associated with the prolonged exposure of the skin to urine or faeces [5]. IAD is often associated with skin redness, rash, or vesiculation [6].

There is a small but growing body of evidence that provides insight into the definition, epidemiology, etiology, and pathophysiology of IAD. The past decade has seen a growth in publications focusing on IAD and the differentiation between IAD and pressure ulcers. Today, IAD as a skin disorder seems to be more and more accepted in clinical practice and research worldwide, as indicated by an increasing number of PubMed entries since 2006 [6].

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It is a daily challenge for healthcare professionals in hospitals, nursing homes and the community to maintain a healthy skin in patients with incontinence [7, 8]. Clinicians need to be vigilant both in maintaining optimal skin conditions and in diagnosing and treating minor cases of IAD prior to progression and skin breakdown [9, 10]. Patients with IAD can experience discomfort, pain, burning, itching or tingling in the affected areas. In addition, the development of IAD can result in an undue burden of care, loss of independence, disruption in activities and/or sleep, and reduced quality of life, worsening with frequency and quantity of soiling [11, 12].

Definition and Terminology for Incontinence-Associated Dermatitis

In 2007, IAD was defined by an international expert panel as skin inflammation manifested as redness with or without blistering, erosion, or loss of the skin barrier function that occurs as a consequence of chronic or repeated exposure of the skin to urine or fecal matter [13]. The terminology to describe skin problems associated with incontinence is diverse, and currently more than 18 terms are used in the literature. In the International Statistical Classification of Diseases and Related Health Problems (10th Revision Version for 2007) (ICD-10), the World Health Organization (WHO) classifies incontinence-related skin problems as 'Diseases of the skin and subcutaneous tissue' (Chapter XII, L00-L99) in subcategory 'Dermatitis and eczema' (L20-L30). The current version of the ICD-10 contains coding for diaper dermatitis but does not contain separate coding for IAD [11, 14].

The term used to classify incontinence-related skin problems used in the Medical Subject Heading Terms database of the US National Library of Medicine (MeSH database) is diaper rash. Diaper rash is defined as a type of irritant dermatitis localized to the area in contact with a diaper and occurring most often as a reaction to prolonged contact with urine, faeces, or retained soap or detergent [15]. As IAD occurs frequently in geriatric care settings, the use of the terms 'diaper rash' might not be appropriate for adult persons. In the North American Nursing Diagnosis Association (NANDA), IAD is not mentioned [16].

In international literature, no common terminology is used to indicate the presence of incontinence associated skin problems. The terminology focuses on a description of the skin (e.g. skin maceration), the cause of the irritation (e.g. moisture lesion, incontinence lesion and incontinence dermatitis), the location of the skin problem (e.g. perineal dermatitis) or the material causing the skin problem (e.g. diaper dermatitis) [8, 15].

Currently, IAD is considered a part of a broader group of skin conditions that are referred to as moisture-associated skin damage (MASD) [17]. MASD is used as an umbrella to cover damage of the skin caused by different types of moisture sources, including urine or faeces, perspiration, wound exudate, mucus, and saliva. The most common forms of MASD are IAD, intertriginous dermatitis, periwound moisture-associated dermatitis, and peristomal moisture-associated dermatitis [17]. The term IAD is preferred over the more general term MASD as it distinguishes the skin problem directly with the urine and/or fecal incontinence and not with other conditions (such as perspiration or wound exudate).

Etiology and Pathophysiology of Incontinence-Associated Dermatitis

The etiology of IAD is complex and related to both recurrently chemical and physical irritation of the skin barrier, triggering inflammation and subsequent skin damage [6]. Some studies provide insights into time of IAD onset. Bliss et al. [18] reported that the median time to onset of IAD was 13 days (range 6 to 42 days, n = 981 nursing home residents). In 2011, Arnold-Long et al. [19] reported a time to onset of 13.5 days with a range of 3–25 days. In an intensive care study (2011), Bliss et al. [20] found a median time to onset of IAD of 4 days (range 1–6 days, n = 45 critically ill patients). The development of IAD is attributable to multiple factors having a negative impact on the skin barrier function, including [6, 15, 21]:

- Chemical irritants in incontinence (such as the digestive intestinal enzymes protease and lipase);
- Changes in the skin surface pH;
- Associated microorganisms (such as the Candida Albicans causing fungal infections);
- Repeated skin cleansing activities;
- An occlusive perineal environment (due to the use of incontinence pads);
- Mechanical factors such as friction.

The IAD pathophysiology is summarized in Fig. 7.1.

In particular older patients are affected by IAD as the ageing process is associated with a decline of cell replacement in the skin, compromised barrier function and mechanical protection, delayed wound healing, decreased sweat and sebum production, and reduced content of natural moisturizing factors and lipids [4].

The Stratum Corneum (SC) and the Barrier Function of the Skin

The outermost layer of the epidermis, the stratum corneum (SC), is responsible for the biomechanical barrier function of the skin. The SC is continuously renewed and comprises between 15 and 20 layers of flattened skin cells (corneocytes). The number of layers (and so the thickness of the stratum corneum) depends on the skin area [22]. Corneocytes comprise keratinocytes in the epidermis and contain a variety of components, such as proteins, sugars and other substances that together are known as natural moisturising factor (NMF). NMF comprises filaggrin proteolysis and includes water-soluble, hygroscopic molecules that are mainly located in the corneocytes. The NMF supports skin hydration and leads to an effective and flexible barrier [22]. The SC is adversely transformed with age [23, 24]. The main reasons are:

- the keratin filaments within the corneocytes are prone to crosslinking;
- the amount of intercellular lipids decreases resulting in fewer lipid bilayers;
- the rate of corneocytes turnover decreases.

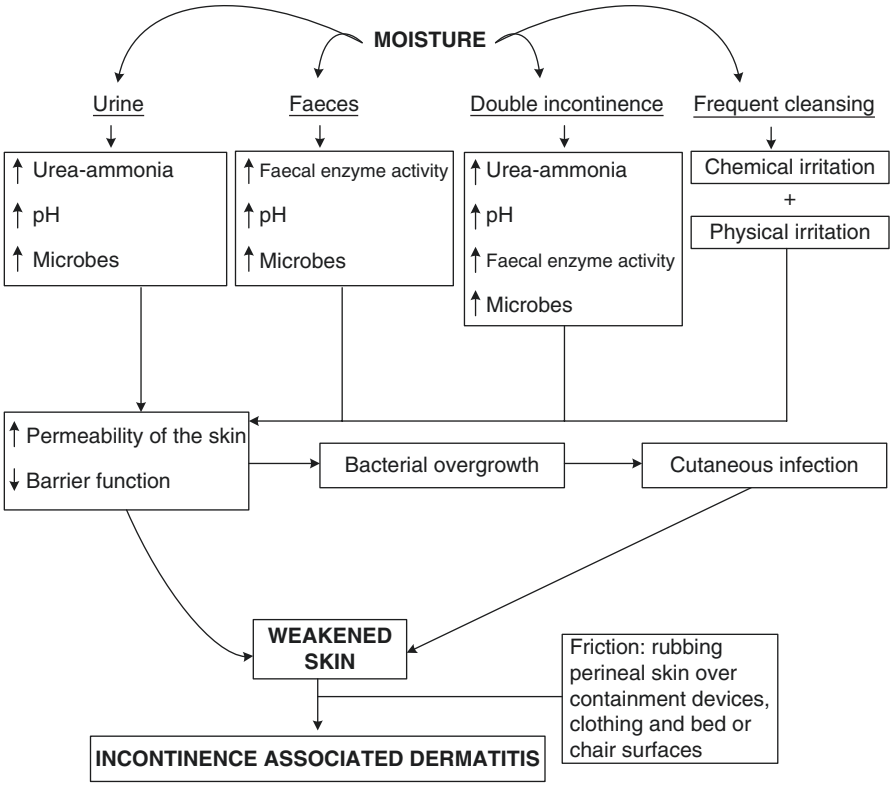

Fig. 7.1 Etiology of Incontinence-Associated Dermatitis (based on the systematic review by Beeckman and associates [15])

Prolonged exposure to moisture from incontinence leads to SC damage. Hyperhydration of the keratinocytes and disruptions of the intercellular lipid bilayers are caused by excessive skin surface moisture [25, 26]. As a result, the corneocytes swell and the thickness of the SC increases. Furthermore, lipases and proteases from the gastro-intestinal tract (in case of faecal incontinence) attack the SC proteins and lipids.

The Impact of pH on the Barrier Function of the Skin

The pH plays a fundamental role in the barrier function of the skin, the SC cohesion and in regulating the resident bacteria on the skin [22]. The healthy skin surface is acidic with a pH of 4–6 [27]. An increase of the skin pH will increase swelling of the SC, will cause the skin to be more permeable, will increase the risk of bacterial colonization (and thus cutaneous infections), will alterate the lipid rigidity and will reduce the skin barrier function. Furthermore, it increases the activity of lipid processing enzymes, resulting in abnormal lipid processing [27]. Lipolytic (lipid digesting) and protolytic (protein digesting) enzymes in faeces significantly increase the risk of damaging the SC. Liquid faeces have an even more damaging effect

compared to formed stool as they tend to have significant more digesting enzymes [13]. In the latest expert opinion document by Beeckman et al. [11] the experts concluded that enzymes are even more active in a higher pH environment. This may explain why patients with mixed incontinence (urinary and fecal) may experience more skin problems linked with their incontinence [11].

Excessive skin surface moisture itself (or hyperhydration) does not alter the skin surface pH [28]. However, the chemical process of urease transforms the urea in the urine into ammonium; thus eventually increasing the skin surface pH [29].

External Factors Increasing the Risk of Incontinence-Associated Dermatitis Development

A reduced skin barrier function and additional occlusive skin conditions (caused by diapers and/or incontinence pads) may further facilitate the degeneration of the SC infiltration by and other irritants and microorganisms (such as the Candida Albicans) [15, 21]. In addition, frequent incontinence episodes (requiring frequent skin cleansing) will lead to the chemical irritation because of the repeated use of water and skin cleansing agents. Furthermore, the use of washcloths for skin washing and towels for drying will to physical irritation [15, 22]. Reduced mobility and limited ability to move independently in bed and chairs causes friction and shear loads in the SC and the epidermis diminishing the strength of the epidermal barrier further [22].

Prevalence and Incidence of Incontinence-Associated Dermatitis

Prevalence and incidence figures for IAD that are internationally comparable are missing. The prevalence varies between 5.6 and 50%, and incidence between 3.4 and 25% [14]. In many countries, the precise number of patients affected by IAD is even unknown. The lack of an ICD-10 coding for IAD an internationally validated and standardized method for IAD data collection contribute to the wide variation in prevalence and incidence figures [11]. Besides, variation is caused by the complexity of recognising the condition and distinguishing it from other skin lesions (such as superficial pressure ulcers) [30–32]. This finding clearly indicates that a standardisation in outcome definition and methods in epidemiological and clinical IAD research is urgently needed.

Risk Assessment and Risk Factors for Incontinence-Associated Dermatitis

Comparable to the domain of pressure ulcers, researchers attempted to develop tools to assess the risk of patients to develop IAD [33, 34]. Different from pressure ulcer risk assessment tools (such as the Braden, Waterlow, Norton tool), IAD assessment tools are not widely used by clinicians. In the expert opinion document (2015) [11], the panel of experts does not recommend the use of a separate risk

assessment tool for IAD; although awarness of key risk factors for IAD is needed. Besides, the panel pointed out that the previously mentioned pressure ulcer risk assessment tools are not designed for IAD, nor do they adequately predict risk of IAD development [11].

Essentially, all patients with urinary and/or fecal incontinence are at risk of developing IAD. Besides, following additional factors will increase the risk of IAD development in incontinent patients: frequent episodes of incontinence (especially faecal), the use of occlusive containment products, poor skin condition (e.g. due to aging/steroid use/diabetes), reduced mobility, diminished cognitive awareness, inability to perform personal hygiene, pain, increased body temperature (pyrexia), certain medications (antibiotics, immunosuppressant), poor nutritional status, and critical illness. Although increased age is associated with higher prevalence of incontinence, age does not appear to be an independent risk factor for IAD [8, 22]. More research is needed in this domain.

Observation of Incontinence-Associated Dermatitis

The diagnosis of IAD is based on visual inspection of the perineal and perigenital skin. Typical locations for IAD to occur are the perianal and sacrococcygeal areas, the thighs and the buttocks [14]. IAD only occurs in skin areas being exposed to urine and/or faeces [11, 14]. If similar skin damage occurs in areas that not have been exposed to urine or faeces, another type of MASD should be considered (intertriginous dermatitis, periwound moisture-associated dermatitis, and peristomal moisture-associated dermatitis).

In patients with light skin tones, early signs of IAD are erythema (ranging from pink to red) and a whitened appearance and slight swelling of the surrounding skin (indicating maceration). Evidence points out that the appearance of erythema is strongly associated with skin maceration in older incontinent patients [22, 35]. In patients with darker skin tones, the skin looks paler, darker, purple, dark red or yellow.

In both patients groups (light skin tones and darker skin tones), the affected area has poorly demarcated edges and may be patchy or continuous over large areas. Because of the underlying inflammation, areas of IAD where skin is intact may feel warmer and firmer than surrounding unaffected skin. Lesions including vesicles or bullae, papules or pustules may be observed. The epidermis may be damaged to varying depths; in some cases the entire epidermis may be eroded exposing moist, weeping dermis [8, 11, 14] (See Fig. 7.2).

An inflamed and eroded skin has a high risk for secondary infection [6]. Candida Albicans is the most frequent fungal infection in geriatric IAD patients [6]. In 2007, Junkin and Selekof [21] found that 18% of 198 hospitalized patients with urinary, fecal or double urinary and fecal incontinence had evidence of IAD with secondary cutaneous candidiasis based on visual inspection. Clinical signs are punctuate pustules and satellite lesions spreading around the IAD area. Bacterial infections may also occur in the course of IAD. In 2014, Campbell and Coyer [36] found that 32% of patients with IAD had a rash indicative of a fungal infection.

IAD observation is complex and different stages of severity are described [14, 22]. IAD severity can range from intact skin (with different levels of erythema),

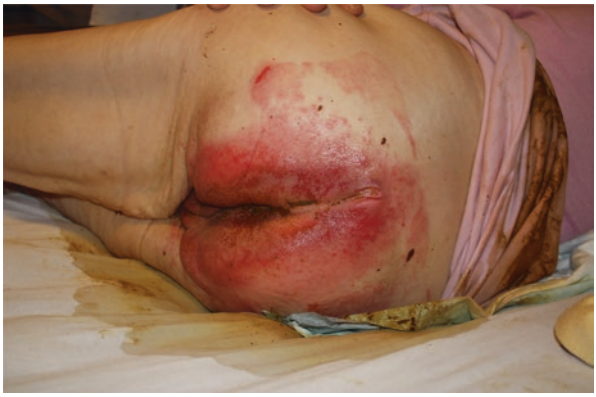

Fig. 7.2 IAD Category 2 with poorly demarcated edges over a large area with clear signs of inflammation. The epidermis is damaged to varying depths; in some areas the entire epidermis is eroded exposing moist, weeping dermis. (Photo courtesy of D. Beeckman)

maceration and swelling, disappearance of the skin structure and erosions, eventually leading to cutaneous wounds.

A study in 2015 pointed out that pain is an essential characteristic of IAD, especially occurring during skin cleansing activities [12]. Pain can range from tingling, over itching and burning [12]. During the last years, a series of IAD classification tools and scores have been published and tested regarding psychometric properties. In 2015, Clarke-O'Neill et al. [37] concluded that the existing IAD instruments are too time-consuming and linguistically complex for use in routine clinical practice in nursing homes. The research also concluded that observation with an instrument could be improved by adding reference photographs of skin illustrating the categories. In 2015 a simplified classification system consisting of three categories supported by photographs (including being "at risk") was proposed:

- **Category 0 =** No redness and skin intact (at risk): Skin is normal as compared to rest of body (no signs of IAD)
- **Category 1 =** Red but skin intact (mild)—edema can be present
- **Category 2 =** Red with skin breakdown (moderate-severe)—edema, vesicles/bullae/skin erosion, denudation of skin, skin infection can be present

Such a classification may be useful for documentation, clinical decision making and research purposes [6, 11]. However, further validation studies are needed before this tool can be introduced in practice.

Incontinence-Associated Dermatitis and Pressure Ulcers

Association Between IAD and Pressure Ulcers

Clinicians often experience difficulties to correctly identify IAD and to distinguish it from pressure ulcers (mainly erythema or up to the level of partial thickness skin loss) and other (moisture related) skin conditions [8, 38]. In healthcare systems where pressure ulcer occurrence is used to assess the quality of care and is linked with reimbursement and litigation, misdiagnosis of IAD as a pressure ulcer has potentially serious

implications [11, 39]. From a clinical point of view, we know that IAD frequently occurs in the absence of any causal factor for pressure ulcer development (pressure/shearing forces on the skin and the underlying soft tissue) and that babies are frequently affected with irritant diaper dermatitis (similar pathophysiological process for IAD) when their skin is exposed to prolonged wetness. The international guidelines for pressure ulcer prevention and treatment in 2014 [39] and the IAD best practice document in 2015 [11] underline the need to correctly diagnose pressure ulcers and to differentiate them from other skin lesions which occur in the same areas on the skin. Even though the clinical presentation of partial thickness pressure ulcers and IAD is similar, the underlying etiologic factors differ.

Pressure ulcers are localized injuries to the skin and/or underlying tissue, usually over a bony prominence due to the impact of mechanical forces (pressure and shear) [39]. Pressure ulcers can be divided in four categories: non-blanchable erythema (cat. I), partial thickness skin loss (cat. II), full thickness skin loss (cat. III) and full thickness tissue loss (cat. IV). IAD and pressure ulcers have different etiologies but may co-exist: IAD is a 'top down' injury, i.e. damage is initiated on the surface of the skin, while pressure ulcers are believed to be 'bottom up' injuries, where damage is initiated by changes within soft tissues below and within the skin. The pathophysiological and histopathological differences between IAD and pressure ulcers are largely understudied. Only in 2007, Houwing and colleagues [40] performed 14 skin biopsies and concluded that an ischemic and irritation pattern emerged, in both pressure ulcers and IAD. However, the pattern of irritation appeared to be more associated with lesions that clinically fitted the description of IAD. Based on this small-scale study, the researchers concluded that there was no justification for the introduction of an (at that time) new diagnostic entity such as IAD. In a recent systematic review and meta-analysis by Beeckman et al. [4] incontinence and IAD were found to be risk factors for pressure ulcer development.

Luboz et al. [41] relate this association between prolonged exposure to skin surface moisture and irritants to changes of the mechanical skin properties of the skin and underlying tissue. They link the associated risk for pressure ulcer development with the increase of the coefficient of friction and tissue stiffness changes. Additionally, local inflammation will increase the temperature of the skin leading to further diminishing of the cutaneous resistance against tissue deformation. On the other hand, we know that category I and partial thickness skin lesions will increase the susceptibility for IAD development [6]. Research points out that determining if the inflammation of the skin in the buttock and sacral areas is primarily due to pressure or irritation is difficult and confusing [8].

Differentiation Between IAD and Pressure Ulcers

Multiple studies showed that pressure ulcer classification is difficult [8, 14] and that misclassification between pressure ulcers and incontinence associated dermatitis (IAD) frequently occurs [11, 42]. As previously mentioned, the differential diagnosis between pressure ulcers and IAD is mainly based on visual examination. Misclassification has significant implications for prevention, treatment, and for

Table 7.1 Synthesis of the EPUAP position statement on pressure ulcer classification and IAD differentiation

	Pressure ulcer	Incontinence-associated dermatitis (IAD)
Cause	Pressure and/or shear must be present	Moisture must be present (e.g. shining, wet skin caused by urinary incontinence or diarrhoea)
Location	A wound over a bony prominence is likely to be a pressure ulcer	IAD may occur over a bony prominence. However, pressure and shear should be excluded as causes, and moisture should be present
Shape	If the lesion is limited to one spot, it is likely to be a pressure ulcer	Diffuse, different superficial spots are more likely to be IAD
Depth	Partial thickness skin loss and full thickness skin loss	Superficial (partial thickness skin loss)
Necrosis	A black necrotic scab on a bony prominence is a pressure ulcer grade 3 or 4. If there is no or limited muscular mass underlying the necrosis, the lesion is a pressure ulcer grade 4	No necrosis
Edges	Distinct edges	Diffuse or irregular edges
Colour	If redness is non-blanchable, this is most likely a pressure ulcer grade 1	Blanchable or non blanchable erythema Pink or white surrounding skin due to maceration

reporting and benchmarking on quality of care. Classification skills are likely to benefit from education [38]. In 2005, Defloor and colleagues [42] published the EPUAP statement on the differentiation between pressure ulcers and IAD. In this statement, wound-related characteristics (causes, location, shape, depth, edges, and colour) and patient-related characteristics were defined to clarify the difference between a pressure ulcer and IAD. Based on this statement, an international working group of experts developed and tested the e-learning PuClas education tool (http://www.puclas.ugent.be/puclas/), a world-wide used tool to learn and teach about pressure ulcer classification, translated in many languages. Currently a revised version of the tool (PuClas3, http://puclas3.ucvvgent.be/) is published and online available [43]. A summary of the PuClas guideline is provided in Table 7.1.

Management of Incontinence-Associated Dermatitis

Management (prevention and treatment) of IAD is a significant challenge for healthcare professionals. Delivering care that is based on state-of-the-art research is hampered by the absence of an internationally and inter-professionally accepted terminology, a standardized definition, high quality studies and national and international guidelines [6].

Both the prevention and treatment of IAD include the removal of occlusive conditions, gentle skin cleansing, skin protection (e.g. “barrier products”), and the

application of therapeutic ointments like dexpanthenol, zinc, or antimycotics [6]. Although the number of studies about prevention and treatment of IAD is increasing, the current evidence is still limited [44]. One reason is the use of many different and sometimes ill-defined outcome parameters in clinical studies [11]. As previously mentioned, reviews identified different operational definitions of IAD, the use of various clinical severity scales with varying numbers of categories, different biophysical skin barrier and appearance parameters (e.g. erythema, transepidermal-water loss, skin surface pH), bacterial loads to name but a few [15, 45, 46]. Skin surface interleukin levels, stratum corneum hydration, or skin surface roughness are other parameters recently used to characterize diapered skin and to measure intervention effects in infants and adults. Furthermore there are more than six published clinical scores to measure the risk and/or the severity of IAD [8] but their usefulness as outcomes in clinical research is unexplored so far.

Prevention of IAD includes three strategies:

- skin cleansing to remove dirt, debris and microorganisms;
- skin moisturization to repair or augment the skin's barrier, retain and/or increase its water content, reduce transepidermal water loss and restore or improve the intercellular lipid structure; and
- the application of a skin barrier product to prevent skin breakdown by providing an impermeable or semi-permeable barrier on the skin.

Recent studies and expert opinions recommend the application of a skin barrier product as an essential element of skin care to prevent or treat IAD. A wide range of creams, ointments, pastes, lotions and films is available as well as different skin barrier formulations such as petrolatum-based, dimethicone-based, zinc oxide–based, or liquid film-forming acrylate. The terminology used to describe the properties of products is not standardized. Despite their widespread use, little is known about the efficacy and effectiveness of products from well-designed randomized controlled clinical trials. A number of studies compared the use and effect of different types of skin regimens, but study design weaknesses are common.

Barrier products are necessary to protect the skin of patients who suffer urinary and fecal incontinence [6]. The presence of high moisture and corrosive enzymes from intestinal fluids can lead to devastating breakdown of the skin leading to denuding and erosion of the skin [14]. Current products which are used to protect the skin from these challenges include occlusive barrier ointments, creams, pastes, polymeric film formers and cyanoacrylates. Determining the relative performance of these barrier materials is difficult for clinicians and is generally anecdotal [11]. A wide variety of products and formulas with both moisturizing and barrier capacity exists. Skin protectants probably vary in the magnitude of protection from exposure to irritants, but we have inadequate evidence to rank these products based on their barrier function while preventing maceration of underlying skin [8]. In addition, we have inadequate evidence to determine the effect of the concentration of active ingredients [8, 14]. For example, dimethicone is classified as a non-occlusive emollient and skin protectant, but some products contain 1% dimethicone, while others

contain 3% or 5% dimethicone. Because of these deficits in knowledge and clinical evidence, it is not surprising that product selection remains a challenge for clinicians when preventing and managing IAD.

Evidence on the effectiveness alone of skin care regimens to prevent or treat IAD are yet insufficient for policy making. Health care budgets are limited, hence policy makers are facing the problem how to set priorities in the allocation of health care resources to different treatment options. Knowledge on this can be obtained by performing economic evaluations of health care interventions providing payers and regulatory bodies with better insights how to establish priorities within cost-constrained health care budgets. To date, there are limited cost-effectiveness studies with regard to IAD.

Conclusion

To conclude, managing IAD can correctly be described as an important challenge for clinical practice and research. Too many patients suffer from it, and still too little effort is done to improve outcomes in these patients. Problems are mainly related to accurate observation, differentiation, and appropriate management. Significant efforts, mainly in terms of education, are made to improve the differentiation between IAD and pressure ulcers. However, these efforts are mainly locally organised and they vary in term of intensity between organisations. A more general awareness about the association between IAD and pressure ulcers is needed. Tissue viability experts and incontinence specialists must play a leading role in developing this area.

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