

Moisture-Associated Skin Damage

2000 words 24.02.2020

Authors Aby Mitchell and Barry Hill

Introduction

Moisture associated skin damage (MASD) is the term used to cover a range of skin problems following prolonged exposure to moisture (Dorsett & Allen, 2013). MASD is defined as "*inflammation and erosion of the skin caused by prolonged exposure to moisture and its contents, including urine, stool, perspiration, wound exudate, mucus, or saliva*" (Young, 2017; Gray, 2011). MASD encompasses the four forms of moisture-associated skin damage, which include 1) incontinence-associated dermatitis or IAD; 2) intertriginous dermatitis, also called intertrigo or ITD; 3) peristomal moisture-associated dermatitis; and 4) peri-wound moisture-associated dermatitis. Although previously considered as a specific problem associated with urinary and faecal incontinence MASD can also be caused by wound exudate, perspiration, mucus or saliva (Dorsett & Allen; Gray et al., 2011). MASD is a common problem encountered by many patient groups in the community in particular older adults. It is important for community staff to assess, correctly diagnosed and treat MASD as well as promote appropriate skin care and continence regimes.

Function of the skin

The skin performs a variety of roles including an effective barrier from environmental exposure, thermoregulation, internal homeostasis, immune function, preventing desiccation of the body from moisture loss and vitamin D metabolism (Dorsett & Allen, 2013). The barrier function of the skin is predominantly achieved by the stratum

corneum (the outer layer of the epidermis). When the skin is damaged this barrier, function is lost. **Editor please include image of the layers of the skin from the MAG clinical library**

Causes of moisture associated skin damage

When exposed to moisture over a long period of time the skin becomes over-hydrated. This can precipitate inflammation because irritants are able to pass through the skin and cause dermatitis (Woodward, 2019). Over-hydrated skin and is more prone to mechanical damage caused by friction and shearing forces from skin rubbing against clothes, bedsheets or incontinence pads. The skin is more likely to breakdown and there is an increased the risk of infection. Inflammation can also occur because normal skin flora is able to penetrate the skin barrier and activate the skin's immune defenses (Voegeli, 2019). MASD is widely accepted to consist of four distinct conditions (Dorsett & Allen, 2013). Each condition has a slightly different aetiology.

Incontinence-associated dermatitis

Incontinence- associated dermatitis (IAD)is a the most widely studied form of MASD (Beeckham et al., 2011; Gray et al., 2012) with prevalence rates thought to vary from between 5.6 % to 50% (Gray et al., 2007). The highest of those being patients with faecal incontinence in long-term care. It is likely this figure is under estimated without validated assessment and recording tools and under reporting (Borchert et al., 2010).

Aetiology ad predisposing factors

IAD is predominantly a chemical irritation caused by skin contact with urine or faeces. Urinary incontinence may lead to IAD but it is most commonly a result of faecal

incontinence or mixed urinary and faecal incontinence (Voegeli, 2012). Enzymes from stool and ammonia from urine can disrupt the acid mantle of the skin and cause it to break down (Rees and Pagnamenta, 2009). Inflammation is characterized by redness and in severe cases blisters and swelling (Dorsett & Allen, 2013). The affected area will present with maceration and erythema and may progress to painful partial-thickness erosions with a weepy serous exudate (Dorsett & Allen, 2013). IAD poses the greatest risk to the sacrum, buttocks and groin, it can also extend over the lower abdomen, anterior, posterior and medial thighs (Beeckman et al, 2015). Once IAD is present there is an increased the risk of pressure ulcer (PU) development, infection and morbidity (Junkin et al., 2008). IAD and Pus often exist alongside each other creating a combined presentation (Lumbers, 2018).

Treatment & Interventions

The following precautions can help minimize the risk of developing incontinence-associated dermatitis in at-risk patients and to minimize complications in patients already exhibiting symptoms:

- Minimize skin exposure to urine and stool
- Manage incontinence
- Frequent change of continence products
- Develop a consistent regimen of skin care to protect the integrity of the skin barrier, including cleansing, moisturizing and use of a skin protectant
- Avoid frequent cleansing with soap and water this is detrimental to the skin barrier function and damages corneocytes (found in the stratum corneum), increases dryness and creates friction (Beeckham et al., 2015). A skin cleanser

with a PH range similar to normal skin is preferred over traditional soap which is alkaline. Aggressive cleansing can increase frictional forces and abrade skin (Beeckman et al., 2015).

- Reduce frictional forces (which are also likely to contribute to pressure damage) (Mitchell, 2019)
- Apply skin barrier to protect the skin from further damage
- Treat any secondary infection topically

All patients with urinary or faecal incontinence should have their skin assessed at least once daily dependant on the number of incontinent episodes. Nurses should consider the use of the IAD Assessment and Intervention Tool (IADIT) (Junkin, 2014), Incontinence-associated dermatitis and its severity (IADS) (Borchert et al. 2010) and Skin Assessment Tool (Beeckman et al., 2011)

Peri-wound moisture associated dermatitis

The production of exudate is a normal result of the inflammatory stage of wound healing. However, the advent of moist wound healing has brought with it an understanding that moisture balance is the key to optimal outcomes. Excessive amounts of wound exudate can cause the peri-wound (within 4 cm of wound edge) skin to become macerated and even break down (Dorsett & Allen, 2013).

Aetiology and predisposing factors

There is no evidence to suggest that normal moist wound healing causes maceration Voegeli (2012) but the chemical composition of the wound exudate can greatly affect the skin. The presence of bacteria, specific proteins, or proteolytic enzymes, as well

as the volume of wound exudate greatly reduces the barrier function of the skin and can lead to maceration. Specifically, exudate from chronic wounds has been found to contain a higher concentration of proteolytic enzymes compared to exudate from acute wounds (Voegeli, 2012). Another factor affecting the occurrence of peri-wound maceration is damage to skin by aggressive removal of adhesive wound dressings, which affect the integrity of the skin barrier by stripping away parts of the epidermis.

Intertriginous dermatitis

Intertrigo (intertriginous dermatitis), also referred to as Intertriginous dermatitis (ITD), is an inflammatory condition of skin folds, induced or aggravated by heat, moisture, maceration, friction, and lack of air circulation (Vakharia, 2019). Intertrigo frequently is worsened by infection, which most commonly is with *Candida*. Bacterial, viral or, other fungal infection may also occur. Intertrigo commonly affects the axilla, perineum, inframammary creases, and abdominal folds, neck creases and interdigital areas. Those that use continence pads show a significant overlap with intertrigo and Intertrigo is a common complication of obesity and diabetes.

Aetiology and Predisposing Factors

Intertrigo most often occurs in patients with obesity (body mass index more than 30 kg per m²), diabetes mellitus, or human immunodeficiency virus infection, and in those who are bedridden. It also occurs in patients with large skin folds and those who wear diapers or other items that trap moisture against the skin. There is a linear increase in the severity of obesity and the presence of intertrigo. Patients who are obese sweat more profusely because of their thick layers of subcutaneous brown fat, generating

more heat than persons with normal body mass. This increases thermal, frictional, and moisture components of the skin.

As the stratum corneum becomes macerated because of hyperhydration, the friction intensifies and further weakens and damages the epidermal tissue. The condition can progress to severe inflammation and skin breakdown. This erosion of the epidermal barrier may create an entry point for micro-organisms that cause secondary infections.

Peristomal moisture-associated dermatitis

Colwell et al., (2011) define peristomal moisture-associated dermatitis as *'inflammation and erosion of skin related to moisture that begins at the stoma/skin junction and can extend outward in a 10cm radius'*. As part of the application of the stoma pouch, solid skin barriers are placed around the stoma to protect the underlying skin from detrimental components of the stoma output (urine or stool). These barriers work to keep the skin dry by absorbing effluent from the stoma and moisture from the underlying skin. If too much moisture is absorbed from the stoma, the barrier will cease to be effective, letting the effluent encounter the peristomal skin. Too much moisture beneath the barrier (sweat or exudate from an existing peristomal wound) can occlude the underlying skin and lead to maceration.

Treatment & Interventions

The following precautions can help minimize the risk of developing peristomal moisture-associated dermatitis in at-risk patients and to minimize complications in patients already exhibiting symptoms:

- Manage peristomal moisture sources such as perspiration, wound exudate, and external sources to ensure proper pouch adhesion.
- Make sure the pouch is not left in place for too long or too short of a period. Longer wear times may lead to compromised pouch adhesion and occlusion of the underlying skin, and shorter wear times can result in mechanical stripping of the skin
- When cutting or molding the skin barrier to fit the stoma, it is recommended that frequent measurements of the stoma be conducted over the first 6 weeks to adjust to the changing shape of the stoma.

Treatment of peristomal moisture-associated dermatitis should be focused on preventing further irritation and healing the irritated skin. The pouching system should be re-evaluated to ensure proper fitting and drainage, with the skin barrier suited to the type of output. Topical therapies such as skin barrier powders, pastes or rings can be used to absorb moisture under the skin barrier, provide an additional physical barrier, reduce existing irritation, and allow for proper adhesion of the solid skin barrier. If exudate from an underlying wound is the source of moisture, the aetiology of the wound should be addressed, and exudate managed with an appropriate absorptive dressing.

ASSESSMENT OF MOISTURE-RELATED SKIN DAMAGE

Patient assessment

A full and detailed patient review should include an assessment of the patient's continence status, mobility, nutrition, allergies and previous skin problems/wounds. Establish the patient's bathing routine and skin care regimen, including his/her ability to self-care and involvement of caregivers. A risk assessment for skin breakdown that

includes pressure ulcer risk assessment should be performed using a recognised tool (NPUAP/EPUAP 2009). The effect of any identified skin problems on the patient's quality of life should be documented and explored.

Skin assessment

Patient assessment should be followed by a detailed skin assessment that includes inspection and palpation of the skin, assessment of skin colour, temperature and moisture levels. Document the cause, location and type of lesion and ensure moisture lesions are differentiated from pressure ulcers (Defloor et al, 2005). The evidence suggests moisture lesions are often mistaken for pressure ulcers (Beeckman et al, 2007) and this can affect the patient's treatment and organisation's ability to achieve targets to reduce the incidence of pressure ulcers as part of the 'Safety Thermometer' and quality agenda (Dorsett & Allen, 2013).

TREATMENTS

BARRIER AGENTS

In uncomplicated intertrigo, numerous agents and mechanisms can be used to keep the skin folds dry, clean, and cool. Applying barrier protectants reduces skin breakdown and alleviates pruritus and pain. Skin protectants include zinc oxide ointment and petrolatum. Separating skin surfaces with absorbent products, such as gauze, cotton, and products with water vapor-permeable sheets, may also help reduce friction.

DRYING AGENTS

Aluminum sulfate, calcium acetate solution, and antiseptic drying agents (e.g., talcum powder) may be used. Powder drying agents should not be applied at the same time as antifungal creams or ointments because this will create a tacky paste. If both are used, they should be applied two to three hours apart. If symptoms do not improve after treatment, potassium hydroxide preparation, and bacterial culture and sensitivity testing should be performed.

SECONDARY FUNGAL INFECTIONS

Intertrigo complicated by fungal infection should be managed with topical antifungals. Nystatin is effective only for candidal intertrigo. Clotrimazole, ketoconazole, oxiconazole (Oxistat), or econazole may be used for both *Candida* and dermatophyte infections. Topical treatments are applied twice daily until the rash resolves. Fluconazole (Diflucan), 100 to 200 mg daily for seven days, is used for resistant fungal infections, although patients who are obese may require an increased dosage. Oral azoles may potentiate the effects of hypoglycemic agents, leading to low blood glucose levels, and patients with diabetes should be instructed to monitor their blood glucose levels with concomitant use of these medications.

SECONDARY BACTERIAL INFECTIONS

The moist, damaged skin associated with intertrigo provides an opportunistic environment for bacterial microorganisms; therefore, secondary cutaneous infections are common. *Staphylococcus aureus* infection may occur independently or with group A beta-hemolytic streptococcal infection. *Pseudomonas aeruginosa*, *Proteus mirabilis*, or *Proteus vulgaris* also may be present independently or simultaneously.

The optimal treatment for patients with intertrigo and group A beta-hemolytic streptococcal infections includes single or multiple regimens of topical therapies (e.g.,

mupirocin [Bactroban], erythromycin); oral antibiotics (e.g., penicillin, first-generation cephalosporins); and low-potency topical steroids (e.g., hydrocortisone 1% cream). Low-potency topical steroids can also be useful to treat intertrigo associated with seborrheic or atopic dermatitis.

Cutaneous erythrasma is best managed with erythromycin (topical, applied twice daily until rash resolves, or oral, 250 mg four times daily for two weeks). Oral erythromycin is more effective than a topical regimen, but it can cause adverse reactions, such as nausea, vomiting, abdominal pain, and diarrhea. Topical clindamycin, Whit-field ointment, and antibiotic soaps may also be beneficial.

Preventing Recurrent Infections

Keeping the area affected by intertrigo dry and exposed to air can help prevent recurrences. Weight loss should be encouraged if obesity is a predisposing factor. Some patients with large, pendulous breasts may benefit from reduction mammoplasty.

Nurses have a responsibility to reduce the impact of MASD by developing public awareness that MASD can affect anyone in their lifetime. Ensuring that healthcare staff recognise risk factors and have the knowledge and expertise to promote prevention and treatment strategies.

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