INCONTINENCE-ASSOCIATED DERMATITIS
Prof. Dimitri Beeckman
Ghent University, Belgium

Learning objectives

At the end of this lecture, students will be able to:

- Describe the pathogenesis of incontinence-associated dermatitis (IAD)
- Articulate key risk factors for the development of IAD
- Describe and use the Ghent Global IAD categorization Tool (GLOBIAD)
- Describe key IAD prevention and treatment principles
INTRODUCTION

– Clinical pictures of IAD
INTRODUCTION

– Clinical pictures of IAD

WHAT ABOUT SLOUGH?

• Moist devitalized host tissue
• The colour varies from cream, yellow and tan depending on hydration level
• It can firmly attached or loose
• May be slimy, gelatinous, stringy, clumpy or fibrinous consistency
• Maybe liquefying necrosis
• May be related to the end of the inflammatory stage in the healing process
• Maybe biofilm related
• May contain:
  • Proteinaceous tissue
  • Fibrin
  • Neutrophils
  • Bacteria

INTRODUCTION

– DEFINITION AND CODING

• IAD = part of a broader group of skin conditions, referred to as Moisture-Associated Skin Damage (MASD).
• IAD = 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 faeces.
• IAD = different levels of severity (associated with selection of interventions and outcomes)
An important function of the skin is to protect the body against pathogens.

The stratum corneum (outermost layer of the epidermis) provides this critical barrier by prohibiting the invasion of micro-organisms.

- Stratum corneum = 70% protein, 15% lipids, 15% water
- Lipids and water are important components in the skin’s barrier function
- In older patients, the volume of water decreases to less than 10%.
Skin surface ‘wetness’, increased skin surface pH, digestive intestinal enzymes (protease, lipase), repeated skin cleansing activities, and a possible occlusive environment (e.g. due to incontinence pads) contribute to irritation and inflammation.

**PATHOGENESIS**

- **INFLAMMATION:**
  - May come in many forms, from occasional rashes accompanied by skin itching and redness, to chronic conditions such as eczema, seborrhea, and psoriasis.
  - Can be characterized as acute or chronic:

  **ACUTE**
  - Exposure to e.g. UV radiation, allergens, or to contact with chemical irritants (urine, faeces, soaps, etc.)
  - Typically resolved within 1 to 2 weeks with little accompanying tissue destruction (if well managed)
INFLAMMATION:

- May come in many forms, from occasional rashes accompanied by skin itching and redness, to chronic conditions such as eczema, seborrhea, and psoriasis.
- Can be characterized as acute or chronic:

CHRONIC

- Sustained immune cell mediated inflammatory response within the skin itself
- Long lasting and can cause significant and serious tissue destruction

PATHOGENESIS

- Incontinence: water is pulled into and held in the corneocytes.
- Overhydration: swelling and disruption of the structure of the stratum corneum, and leads to an increase of the stratum corneum thickness and visible skin changes
- Excessive hydration: irritants may more easily penetrate the stratum corneum to exacerbate inflammation.
- Overhydrated skin: epidermis more prone to injury from friction.
PATHOGENESIS

- Urease transforms urea into ammonium thus increasing the skin surface pH
- Increased skin surface pH: decreased stratum corneum cohesion and decreased recovery capacity of skin barrier function, micro-organisms to thrive and increase the risk of skin infection
- Impaired skin barrier and occlusive skin conditions: facilitate the infiltration of the stratum corneum by the Candida Albicans, from the gastrointestinal tract, and Staphylococcus, from the perineal skin
- Lipases and proteases attack the the stratum corneum proteins and lipids.

PATHOGENESIS

- ABOUT WHAT DO EXPERTS AGREE WHEN THEY OBSERVE IAD?
  - Erythema and edema of the skin
  - Sometimes accompanied by bullae with serous exudate, erosion, and infection
  - No common language
INDEPENDENT RISK FACTORS

• Knowledge of risk factors is helpful to tailor IAD prevention and management.

• IAD prevalence studies identified following key risk factors for IAD:
  - Liquid stool
  - Critical illness
  - Fever
  - Diminished perfusion and oxygenation
  - Poor skin condition (e.g. steroid use/diabetes)
  - Restricted mobility and activity
  - Higher score on care dependency
  - Poor nutritional status
  - Risk of friction and shear
  - Restricted cognitive awareness

WHAT DO WE KNOW FROM ICU PATIENTS?

• Matched case-control study in critically ill patients
• 30 hospitals (46 ICU/CCU wards) in Belgium
• 206 patients
• Fecal incontinent

Clinical trial registration: https://clinicaltrials.gov/ct2/show/NCT02996357
INDEPENDENT RISK FACTORS

WHAT DO WE KNOW FROM ICU PATIENTS?

<table>
<thead>
<tr>
<th></th>
<th>N = 206</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>65.1 (SD 14.9)</td>
</tr>
<tr>
<td><strong>Incontinence</strong></td>
<td></td>
</tr>
<tr>
<td>• Urinary incontinence</td>
<td>N/A</td>
</tr>
<tr>
<td>• Fecal incontinence</td>
<td>96.6%</td>
</tr>
<tr>
<td>• Double incontinence</td>
<td>3.4%</td>
</tr>
<tr>
<td><strong>IAD Cat. 2A (prevalence)</strong></td>
<td>46.1%</td>
</tr>
</tbody>
</table>

Van Damme et al. 2017 (in progress)

---

INDEPENDENT RISK FACTORS

WHAT DO WE KNOW FROM ICU PATIENTS?

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>OR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liquid stool</td>
<td>4.686</td>
<td>2.283 - 9.619</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Diabetes</td>
<td>2.893</td>
<td>1.336 - 6.266</td>
<td>0.007</td>
</tr>
<tr>
<td>Age</td>
<td>1.049</td>
<td>1.021 - 1.078</td>
<td>0.001</td>
</tr>
<tr>
<td>Smoking</td>
<td>2.670</td>
<td>1.206 - 5.914</td>
<td>0.015</td>
</tr>
<tr>
<td>Use of diapers</td>
<td>0.337</td>
<td>0.158 - 0.720</td>
<td>0.005</td>
</tr>
<tr>
<td>Fever</td>
<td>2.603</td>
<td>1.226 - 5.528</td>
<td>0.013</td>
</tr>
<tr>
<td>Inotropics</td>
<td>4.109</td>
<td>0.845 - 19.970</td>
<td>0.080</td>
</tr>
<tr>
<td>SpO₂ &lt; 95%</td>
<td>2.154</td>
<td>1.034 - 4.484</td>
<td>0.040</td>
</tr>
<tr>
<td>Parenteral nutrition</td>
<td>2.839</td>
<td>1.000 - 8.055</td>
<td>0.050</td>
</tr>
<tr>
<td>Vasopressors</td>
<td>1.964</td>
<td>0.905 - 4.265</td>
<td>0.088</td>
</tr>
</tbody>
</table>

Nagelkerke R² = 0.377; Hosmer-Lemeshow P = 0.301

Van Damme et al. 2017 (in progress)
OBSERVATION OF IAD WITH THE GLOBIAD

Step 1: Separate and inspect skin folds

- Opposing skin surfaces trap and harbour moisture.
- Inflammation typically most pronounced at deepest crease of skin fold.
- Allows secondary evaluation of hygiene/access to skin fold.
OBSERVATION

Step 1: Separate and inspect skin folds

- Opposing skin surfaces trap and harbour moisture.
- Inflammation typically most pronounced at deepest crease of skin fold.
- Allows secondary evaluation of hygiene/access to skin fold.

OBSERVATION

Step 2: Assess for skin erosion

- May present initially as islands of partial thickness erosion.
- Often see multiple areas of erosion closely spaced.
- Entire dermis may be eroded in severe cases.
- Natural history not well defined.
OBSERVATION

Step 3: Inspect for secondary cutaneous infection, especially candidiasis

- Opportunistic infection with Candida Albicans.
- Thrives in warm, moist environment and damages stratum corneum.
- Seen in 18% of one group of 608 acute care inpatients (Junkin & Selekof, 2007).

WHY ANOTHER NEW TOOL?

- A few tools have been developed for assessment of IAD.
- While some of these have been investigated for validity, reliability and their use in day-to-day practice remains limited.
- This is due in part to the lack of evidence that these tools improve clinical decision making and care.
• In 2015, an IAD expert panel recommended the adoption of a simplified approach to categorizing IAD based on the level and severity of the lesion

• In 2016 - 2017 an international expert panel representing > 30 countries developed the Ghent Global IAD Categorisation Tool (GLOBIAD) to create an internationally agreed description of IAD, and to standardize the documentation within clinical practice and for research purposes.

• Tested by 823 clinicians worldwide

WHAT IS THE GLOBIAD?

TRULY INTERNATIONAL …

34 Experts in Delphi

823 clinicians in psychometric testing

823 clinicians worldwide
1A - Persistent redness without clinical signs of infection

Critical criterion
- Persistent redness
  A variety of tones of redness may be present. Patients with darker skin tones, the skin may be paler or darker than normal, or purple in colour.

Additional criteria
- Marked areas or discoloration from a previous (healed) skin defect
- Shiny appearance of the skin
- Macerated skin
- Intact vesicles and bullae
- Skin may feel tense or swollen at palpation
- Burning, itching, itching or pain may be present
**CATEGORY 1B**

--- Category 1: Persistent redness ---

1B - Persistent redness with clinical signs of infection

**Critical criteria**
- Persistent redness
  A variety of tones of redness may be present. Patients with darker skin tones, the skin may be paler or darker than normal, or purple in colour
- Signs of infection
  Such as satellite lesions (e.g. pustules or maculopapular rash) or white scaling of the skin (indicating a fungal infection e.g. Candida albicans)

**Additional criteria**
- Marked areas or discoloration from a previous (healed) skin defect
- Shiny appearance of the skin
- Macerated skin
- Intact vesicles and bullae
- The skin may feel tense or swollen at palpation
- Burning, tingling, itching or pain may be present

---

**CATEGORY 2A**

--- Category 2: Skin loss ---

2A - Skin loss without clinical signs of infection

**Critical criterion**
- Skin loss
  Skin loss may present as skin erosion, denudation, excoriations, open vesicles, or open bullae.
  The skin damage pattern may be diffuse.

**Additional criteria**
- Persistent redness
  A variety of tones of redness may be present. Patients with darker skin tones, the skin may be paler or darker than normal, or purple in colour
- Marked areas or discoloration from a previous (healed) skin defect
- Shiny appearance of the skin
- Macerated skin
- Intact vesicles and bullae
- The skin may feel tense or swollen at palpation
- Burning, tingling, itching or pain may be present
**TIME FOR ACTION …**
**Category 1: Persistent redness**

1B - Persistent redness with clinical signs of infection

- **Critical criteria**
  - Persistent redness
  - A variety of tones of redness may be present. Patients with darker skin tones, the skin may be paler or darker than normal, or purple in colour
  - Signs of infection
    - Such as satellite lesions (eg. pustules or maculopapular rash) or white scaling of the skin (indicating o fungal infection eg Candida albicans)

- **Additional criteria**
  - Marked areas or discoloration from a previous (healed) skin defect
  - Shiny appearance of the skin
  - Macerated skin
  - Intact vesicles and bullae
  - The skin may feel tense or swollen at palpation
  - Burning, tingling, itching or pain may be present

---

**Category 2: Skin loss**

2A - Skin loss without clinical signs of infection

- **Critical criteria**
  - Skin loss
    - Skin loss may present as skin erosion, abrasion, excoriations, open vesicles, or open bullae.
    - The skin damage pattern may be diffuse

- **Additional criteria**
  - Persistent redness
    - A variety of tones of redness may be present. Patients with darker skin tones, the skin may be paler or darker than normal, or purple in colour
  - Marked areas or discoloration from a previous (healed) skin defect
  - Shiny appearance of the skin
  - Macerated skin
  - Intact vesicles and bullae
  - Skin may feel tense or swollen at palpation
  - Burning, tingling, itching or pain may be present
**DIAGNOSIS?**

**Category 2: Skin loss**

**2B - Skin loss with clinical signs of infection**

**Critical criteria**
- Skin loss
  - Skin loss may present as skin erosion, denudation, exfoliation, open vesicles, or open bullae. The skin damage pattern may be diffuse.
  - Signs of infection: Such as satellite lesions (e.g., papules or maculopapular rash), white scaling of the surrounding skin, or in the wound bed (indicating a fungal infection, e.g., Candida albicans). Slough visible in the wound bed (yellow/brown/greyish), green appearance within the wound bed (indicating a bacterial infection, e.g., Pseudomonas aeruginosa), excessive exudate levels, purulent exudate (pus), or a shiny appearance of the wound bed.

**Additional criteria**
- Persistent redness
- A variety of tones of redness may be present. Patients with darker skin tones, the skin may be paler or darker than normal, or purple in color.
- Marked areas or discoloration from a previous (healed) skin defect
- Shiny appearance of the skin
- Macerated skin
- Intact vesicles and bullae
- Skin may feel tense or swollen at palpation
- Burning, tingling, itching, or pain may be present

---

**Category 1: Persistent redness**

**1A - Persistent redness without clinical signs of infection**

**Critical criterion**
- Persistent redness
  - A variety of tones of redness may be present. Patients with darker skin tones, the skin may be paler or darker than normal, or purple in color.

**Additional criteria**
- Marked areas or discoloration from a previous (healed) skin defect
- Shiny appearance of the skin
- Macerated skin
- Intact vesicles and bullae
- Skin may feel tense or swollen at palpation
- Burning, tingling, itching, or pain may be present
**DIAGNOSIS?**

Grade 4: Extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures with or without full thickness skin loss.

---

**Category 2: Skin loss**

**2B - Skin loss with clinical signs of infection**

**Critical criteria**
- Skin loss
- Skin loss may present as skin erosion, demarcation, excoriation, open vesicles, or open bullae. The skin damage pattern may be diffuse.
- Signs of infection
  - Such as satellite lesions (eg. pustules or maculopapular rash), white scaling of the surrounding skin or in the wound bed (indicating a fungal infection eg Candida albicans), slough visible in the wound bed (yellow/brown/greenish), green appearance within the wound bed (indicating a bacterial infection eg Pseudomonas aeruginosa), excessive exudate levels, purulent exudate (pus) or a shiny appearance of the wound bed.

**Additional criteria**
- Persistent redness
- A variety of tones of redness may be present. Patients with darker skin tones, the skin may be paler or darker than normal, or purple in color.
- Marked areas or discoloration from a previous (healed) skin defect
- Shiny appearance of the skin
- Macerated skin
- Intact vesicles and bullae
- Skin may feel tense or swollen at palpation
- Burning, tingling, itching or pain may be present

---

**BEST PRACTICE FOR MANAGEMENT**
MANAGEMENT OF IAD

Incontinence care

- If incontinence is not treatable, use suitable incontinence material and replace as frequent as needed
- Consider a bladder and/or bowl program and implement in close collaboration with continence specialists
- Revise the incontinence approach regularly as changes will occur over time
- Provide a holistic approach to incontinence with attention to financial impact and psychosocial well-being

MANAGEMENT OF IAD

Gentle cleansing of the genital, peri-anal and groin region

- Use gentle cleansing techniques (cfr. neonates)
- Reduce frequency of skin cleansing
- Avoid traditional soap (alkaline pH)
- Avoid influence on bacterial flora, leading to a selection of pathogens
- Consider using bath oil or shower oil without perfume, a pH neutral cleansing foam, soft disposable washcloths
- Do not rub the skin after washing, simple patting of the skin is sufficient
MANAGEMENT OF IAD

Skin hydration

- Hydrate dry skin with a hydrating topical product
- Consider the application of a cream (emulsions of water and oil), ointments are greasy and tend to be more occlusive
  - Dry skin: oil in water cream
  - Very dry skin: rich cream (water in oil emulsion)
- Limit the amount of cream, especially in the folds, to avoid softening of the skin and maceration
- Apply the cream in a gentle way, avoid rubbing

MANAGEMENT OF IAD

Application of a barrier leave-on product

- Aim is to protect the skin against further adverse effects of moisture and incontinence
- Formulation often include Dimethicone or zinc oxide
- Overall formula vary widely, the product selection should depend on the skin status.
- Most of the barrier products are creams combining barrier with hydrating properties and are to be used on intact skin
- Some of the formula are liquid and able to desiccate open lesions
MANAGEMENT OF IAD

• Pressure ulcer prevention remains important and is even more important in patients with IAD. There is growing evidence that incontinence and IAD are risk factors for the development of pressure ulcers.
• Control the nutritional status of the patient, more particular the protein and vitamin intake, especially in patients with erosions or ulcerations.

COMBINING GLOBIAD AND MANAGEMENT
COMBINING GLOBIAD AND MANAGEMENT

PREVENTION

• Cleans the perineal region
  o Use X® foam and soft (single-use) washcloths
  o Do not use water or soap
• Always dry the skin thoroughly after cleansing, if still moist
  o Use soft towels
  o Do not rub, apply a gentle patting technique
  o Be sure that the skin is completely dry before applying other products and diaper/underpads
• Hydration
  o Apply a thin layer of Y® hydrating barrier cream 2X/day
  o Do not use any other topicals
  o In patients with faecal incontinence and frequent stool: increase frequency of Y® hydrating barrier cream to 3x/day
• Evaluate the skin at least daily
  o Whenever persistent redness is observed: apply the category 1 protocol

COMBINING GLOBIAD AND MANAGEMENT

TREATMENT OF CAT. 1A IAD

• Cleans the perineal region
  o Use X® foam and soft (single-use) washcloths
  o Do not use water or soap
• Always dry the skin thoroughly after cleansing, if still moist
  o Use soft towels
  o Do not rub, apply a gentle patting technique
  o Be sure that the skin is completely dry before applying other products and diaper/underpads
• Hydration
  o Apply a thin layer of Y® hydrating barrier cream 2X/day
  o Do not use any other topicals
  o In patients with faecal incontinence and frequent stool: increase frequency of Y® hydrating barrier cream to 3x/day
• Evaluate the skin at least daily
  o Whenever open lesions (skin loss) are observed: apply the category 2A protocol
  o Whenever clinical signs of major colonization or infection are observed: apply the category 1B protocol
  o Whenever open lesions (skin loss) and major colonization of clinical signs of infection are observed: apply the 2B protocol
COMBINING GLOBIAD AND MANAGEMENT

TREATMENT OF CAT. 2A IAD

- Cleans the perineal region
  - Use X® foam and soft (single-use) washcloths
  - Do not use water or soap
- Always dry the skin thoroughly after cleansing, if still moist
  - Use soft towels
  - Do not rub, apply a gentle patting technique
  - Be sure that the skin is completely dry before applying other products and diaper/underpads
- Treatment of exuding lesions
  - Apply Y® barrier lotion 2x/ day on the exudation zones
  - Shake the lotion and apply a small amount on a sterile gauze and apply it to the lesion
- Hydration
  - Apply a thin layer of Y® hydrating barrier cream 2x/ day
  - Faecal incontinence and frequent stool: increase frequency of Y® barrier lotion or Y® hydrating barrier cream to 3x/day
- After desiccation of the exudation lesions
  - Replace the Y® barrier lotion by Y® hydrating barrier cream
- Evaluate the skin on at least daily base
  - Whenever the erosion becomes ulceration: clean the wound with saline and apply the wound care protocol
  - Whenever clinical signs of major colonization or infection are observed: apply the category 2B protocol


COMBINING GLOBIAD AND MANAGEMENT

TREATMENT OF CAT. 1B IAD

- Apply the 1A IAD protocol
- Evaluate the infection according to table 1
  - In case of local signs of fungal infection:
    - Apply a paste containing Miconazol, 1 to 2 times a day. The paste should not be removed before applying a new layer
    - Replace the paste containing Miconazol by Y® hydrating barrier cream if there are no local signs of fungal infection anymore.
COMBINING GLOBIAD AND MANAGEMENT

TREATMENT OF CAT. 2B IAD

• Apply the 2A IAD protocol
• Evaluate the infection according to table 1
  o In the case of local signs of bacterial infection:
    o Treatment of exudation lesions:
      ▪ Apply povidon iodine lotion
    o After desiccation of the exudation lesions:
      ▪ Replace the povidon iodine lotion by Y® hydrating barrier cream
  o Whenever there is a more profound ulceration: apply the wound care protocol for infected wounds
• In the case of local signs of fungal infection:
  • Treatment of exudation lesions:
    • Apply povidon iodine lotion on the exudation lesions
  • After desiccation of the exudation lesions:
    • Apply a paste containing Miconazol on the surrounding skin, 1 to 2 times a day, if there are still local signs of fungal infection. The paste should not be removed before applying a new layer
    • Replace the povidon iodine lotion by Y® hydrating barrier cream if there are no local signs of fungal infection anymore
  • Whenever there is a more profound ulceration: apply the wound care protocol for infected wounds

TO CONCLUDE

• IAD is associated with weakened skin, moisture/maceration, bacterial growth, and friction leading to an inflammatory reaction and (in severe cases) skin loss
• Key risk factors in ICU patients with fecal incontinence include liquid stool, diabetes, fever, not using diapers, age, history/current smoking and reduced perfusion and oxygenation
• GLOBIAD is a valid tool to assess IAD severity, but training is needed
• Next step: monitoring and minimum data-set (in progress)


REFERENCES & RESOURCES


Beeckman D., Schoonhoven L., European Pressure Ulcer Advisory Panel. PuClas3 eLearning Module. University Centre for Nursing & Midwifery and European Pressure Ulcer Advisory Panel. 2015
