Incontinence Associated Dermatitis: Update 2013

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Objectives

- Review etiology, epidemiology, pathophysiology of IAD
- Discuss differential diagnosis
- Define IAD and its relationship to pressure ulcer risk
- Outline options for prevention and treatment
Faculty disclosure: none
Functions of the Skin

- Thermoregulation
- Sensory organ/communication
- Immune functions; acts as a first line of defense
- Vitamin D metabolism
- **Barrier against toxins in external environment and against fluid & electrolyte loss from internal environment**


Figure: Verdier-Sevrain S, Bonte F. Journal of Cosmetic Dermatology 2007; 6:75.
**Definition:**

**Incontinence Associated Dermatitis (IAD)**

- Irritation and inflammation associated with exposure to stool or urine
- Often accompanied by erosion of the skin
- Sometimes accompanied by secondary cutaneous infection (i.e.: candidiasis)
- Distinct etiology and pathophysiology

Photograph courtesy Linda Bohacek
IAD: One Form of Moisture Associated Skin Damage (MASD)

- Definition: inflammation, erosion ± secondary infection associated with excessive exposure to body’s effluents including perspiration, urine, stool, exudate, effluent from ostomy or fistula

- Common Manifestations:
  - Incontinence Associated Dermatitis¹
  - Intertriginous dermatitis²
  - Periwound Maceration³
  - Peristomal moisture dermatitis⁴

**Etiologic Factors: Urine**

- **Water in urine**
  - ↓ skin hardness, rendering it more susceptible to friction and erosion\(^1-^3\)
  - Compromises barrier function of skin\(^4\)
    - ↑ permeability to pathogenic species
    - ↑ permeability to irritants in urine or stool
  - Effects exacerbated by presence of occlusive device such as warp around incontinence brief

Adverse Effects of Urine on Skin

- **Urinary pH and ammonia content**
  - Limited evidence suggests alkaline urine more damaging to skin than urine with lower pH\(^1,2\)
  - Ammonia inherent in urine and produced by conversion of urea in presence of Corynebacterium and fungal species such as candida albicans\(^1-3\)
  - No direct evidence ammonia damages intact skin; probably aggravates already compromised skin\(^1\)
  - Digestive enzymes active in more alkaline environment; may explain increased damae with double FI and UI

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Adverse Effects of Stool on Skin

- Fecal enzymes
  - Protease & lipase potentially break down both principal elements of moisture barrier
  - In vivo evidence shows that exposure to digestive enzymes in human skin led to
    - ↑ TEWL
    - ↑ pH
    - Visible damage only when occlusion present
    - Evidence of damage present after 12 days

**Associated Factors: Occlusion**

- **Use of absorptive containment devices**
  - Exacerbate overhydration by promoting perspiration & retaining urine and stool; *with padding alone*:
    - TEWL increases 3-4 fold within days
    - \( \text{CO}_2 \) emission increases > 4 fold
    - pH increases from 4.4 to 7.1 (*without incontinence*)

Figure 1  Aetiology of incontinence-associated dermatitis (based on Jeter & Lutz 1996 and Newman et al. 2007).
Associated Factors: IAD & Pressure Ulcers

- Association between these conditions is undeniable; nature of relationship remains a mystery
- IAD vs Stage II PU may be a problem with differential diagnosis?
- IAD impairs skin’s tolerance for pressure/shear
- Ongoing debate & controversy about nature of relationship reflects difficulty differentiating based on visual inspection alone
- FI and double incontinence strongly associated with PU risk, mixed evidence concerning UI alone²-⁶

# Epidemiology: Prevalence of IAD


<table>
<thead>
<tr>
<th>Reference</th>
<th>N</th>
<th>Health Care Setting</th>
<th>Incontinence Type</th>
<th>Method of Measurement</th>
<th>Prevalence, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Junkin and associates⁶</td>
<td>976</td>
<td>Acute care</td>
<td>Urinary and fecal incontinence</td>
<td>Direct observation</td>
<td>27</td>
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<tr>
<td>Bliss and associates⁴</td>
<td>10,215</td>
<td>Long-term care</td>
<td>Urinary and fecal incontinence</td>
<td>Review of electronic database</td>
<td>5.7</td>
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<tr>
<td>Defloor and associates⁵</td>
<td>19,964</td>
<td>Long-term care</td>
<td>Urinary and fecal incontinence</td>
<td>Direct observation</td>
<td>5.7</td>
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<tr>
<td>Arnold-Long and Reed¹⁰</td>
<td>171</td>
<td>Long-term acute care</td>
<td>Urinary and fecal incontinence</td>
<td>Direct observation</td>
<td>22.8</td>
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<tr>
<td>Beeckman and associates¹¹</td>
<td>141</td>
<td>Long-term care</td>
<td>Urinary and fecal incontinence</td>
<td>Direct observation</td>
<td>22.5</td>
</tr>
<tr>
<td>Junkin and Seleko²</td>
<td>608</td>
<td>Acute care</td>
<td>Urinary and fecal incontinence</td>
<td>Direct observation</td>
<td>20</td>
</tr>
</tbody>
</table>
**Epidemiology of IAD: Incidence**

<table>
<thead>
<tr>
<th>Reference</th>
<th>N</th>
<th>Health Care Setting</th>
<th>Incontinence Type</th>
<th>Method of Measurement</th>
<th>Period of Observation</th>
<th>Incidence, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bliss and associates(^9)</td>
<td>981</td>
<td>Long-term care</td>
<td>Urinary and fecal incontinence</td>
<td>Direct observation</td>
<td>6 weeks</td>
<td>3.4</td>
</tr>
<tr>
<td>Bliss and associates(^12)</td>
<td>45</td>
<td>Critical care</td>
<td>Fecal incontinence</td>
<td>Direct observation</td>
<td>Duration of stay in the critical care unit: median time to onset of 4 d</td>
<td>36</td>
</tr>
<tr>
<td>Driver(^8)</td>
<td></td>
<td>Phase 1: n = 131</td>
<td>Fecal incontinence</td>
<td>Direct observation</td>
<td>Phase 1: Duration of stay in critical care unit: &lt;14 d</td>
<td>Phase 1: 50</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Phase 2: n = 177</td>
<td></td>
<td></td>
<td>Phase 2: Duration of stay in critical care unit: &gt;14 d</td>
<td>Phase 2: 19(^a)</td>
</tr>
<tr>
<td>Arnold-Long and Reed(^10)</td>
<td>132</td>
<td>Long-term care</td>
<td>Urinary and fecal incontinence</td>
<td>Direct observation</td>
<td>Duration of stay: Median time to onset 13.5 d</td>
<td>7.6</td>
</tr>
</tbody>
</table>

\(^a\)Researchers implemented defined skin care regimen, using 3-in-1 washcloth with skin cleanser, moisturizers, and dimethicone-based skin protectant during phase 2 of the study.

IAD: Screening begins with CNA or other non-licensed care providers
IAD: Diagnosis

- Primarily based on visual inspection
  - Inflammation (bright red) in persons with lighter skin tones
  - Located in *skin fold* or *underneath containment device*
  - Borders are poorly demarcated & irregular
  - Surface of skin may "glisten" owing to serous exudate
IAD: Diagnosis in persons with Darker Skin Tones

- Inflammation not readily apparent (ie: not bright red); often seen as areas of hyperpigmentation or variable red tones
- Hypopigmented areas with chronic inflammation
- Pattern of skin damage does not vary
IAD: Diagnosis

- Inspect Skin Folds
  - Opposing skin surfaces trap & harbor moisture
  - Warm moist environment encourages bacterial and fungal colonization, overgrowth and infection
  - Friction occurs as skin folds rub against one another
IAD: Diagnosis

- Assess for skin erosion
  - Partial thickness erosion occurs with IAD
  - Necrotic tissue: eschar or slough, full thickness damage indicates pressure ulceration
IAD: Diagnosis

- Look for secondary cutaneous infection, especially candidiasis
  - Opportunistic infection with *candida albicans*
  - Thrives in warm, moist environment & damages stratum corneum
  - Seen in 18% of one group of 976 acute care inpatients

Junkin J, Selekof J. IAD prevalence in acute care. WOCN National Conference, June 2006 Minneapolis, MN.
IAD: Diagnosis

- Suspect PU when wound is
  - Over bony prominence
  - Full thickness
  - Necrotic tissue is present
  - Skin is dark to purplish red

Images: http://www.snjourney.com/ClinicalInfo/Systems/Intrgum/newstagepu.htm
       http://www.lhsc.on.ca/wound/p_chart.htm
Emerging evidence reminds us that isolated photographs are insufficient.

The biggest aid in this case is a thorough history.
### IAD vs Pressure Ulcer: Differential Diagnosis

<table>
<thead>
<tr>
<th>Factors</th>
<th>IAD</th>
<th>Stage I Pressure Ulcers</th>
<th>Stage II Pressure Ulcers</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of condition</td>
<td>Exposure to urine or stool</td>
<td>Exposure to pressure, shear, and/or microclimate from immobility or inactivity</td>
<td>Exposure to pressure, shear, and/or microclimate from immobility or inactivity</td>
</tr>
<tr>
<td>Location of affected skin</td>
<td>Skin folds in areas where urine or stool can accumulate</td>
<td>Skin usually over bony prominences or exposed to other external pressure (eg, medical device)</td>
<td>Skin usually over bony prominences or exposed to other external pressure (eg, medical device)</td>
</tr>
<tr>
<td>Color of wound bed</td>
<td>Shiny, red, glistening, no slough in wound bed</td>
<td>Nonblanchable erythema of intact skin</td>
<td>Shiny, pink, or red open wound, no slough in wound bed</td>
</tr>
<tr>
<td>Color of periwound tissue</td>
<td>Red, irritated, edematous</td>
<td>Normal for race/ethnicity, edema may be palpable</td>
<td>Normal for race/ethnicity, edema may be palpable</td>
</tr>
<tr>
<td>Characteristics of involved area</td>
<td>Blotchy, not uniform in appearance</td>
<td>Tend to be single areas of erythema</td>
<td>Tend to be single ulcers with distinct ulcer wound margin</td>
</tr>
<tr>
<td>Pain</td>
<td>Burning, itching, and tingling</td>
<td>Sharp pain, usually no itching; pain may intensify when patient is initially moved off of injured areas</td>
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</tr>
<tr>
<td>Odor</td>
<td>Urine, fecal odor</td>
<td>None</td>
<td>None unless infected and then may have odor of infecting organism</td>
</tr>
<tr>
<td>Other</td>
<td>Candidiasis common (seen as satellite lesions)</td>
<td>Redness tends to resolve with offloading or repositioning of device</td>
<td>Ulcer bed is shallow and heals through epithelialization</td>
</tr>
</tbody>
</table>

Abbreviation: IAD, incontinence-associated dermatitis.