Deep Tissue Pressure Injury: A Dangerous Form of Pressure Ulcers

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Deep Tissue Pressure Injury: Persistent non-blanchable deep red, maroon or purple discoloration

Intact or non-intact skin with localized area of persistent non-blanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood filled blister. Pain and temperature change often precede skin color changes. Discoloration may appear differently in darkly pigmented skin. This injury results from intense and/or prolonged pressure and shear forces at the bone-muscle interface.

DTPI Definition Continued

The wound may evolve rapidly to reveal the actual extent of tissue injury, or may resolve without tissue loss. If necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle or other underlying structures are visible, this indicates a full thickness pressure injury (Unstageable, Stage 3 or Stage 4). Do not use DTPI to describe vascular, traumatic, neuropathic, or dermatologic conditions.
Classify this pressure injury as a Deep Tissue Pressure Injury while it is intact discolored skin (Day 1) or discolored skin with epidermal blistering (Day 3). If the DTPI becomes necrotic, it is classified as unstageable (Day 10).

Evolution of DTPI

Pressure and ischemia

- Ischemia of tissue leads to injury/ulceration
  - Our original premise
- Cellular deformation until the cell membrane ruptures (Gefen)
- Repeated loading and unloading can lead to ulceration over time (Oomen)
- Repeated shearing extends ulcer (Takahashi)
- This differs from superficial ulcers which are due to moisture and friction (Bader)
- Combination of cellular deformation and ischemia creates full thickness injury faster

So depending on the duration and intensity of pressure....

- Pure ischemia
- Ischemia plus tissue destruction
- Tissue deformation
Zones of infarct, ischemia and injury?

- Area sustaining highest pressures is infarcted
  - Not recoverable
- Surrounded by rings of injured and ischemic tissue
  - Rescueable but also capable of evolving into full thickness injury
- Similar to pathophysiology of MI, stroke and burns

Tissue Ischemic Time

Reperfusion Injury: No Flow–Flow

Also similar to MI, stroke and burns
Adding insult to injury

- Initial injury from pressure evolves
  - Oxygen free radical release
  - Unpaired oxygen molecules
  - Injured tissue
- Laboratory data shows that muscle subjected to pressure stiffens over time
  - Inflammatory response
  - Tissue death – a local “rigor mortis” (Gefen, 2007)
- Shear forces on DTPI
  - Shear strain on the deformed tissue can extend the size of the wound

Shear extends the DTPI

- Obese patient developed massive DTPI on regular bed
- Placed in continuous lateral rotation
- Resultant wound with debridement of sciatic nerve, gluteal muscles
- Alive, but not able to rise from a chair, walk without aides or work

Identifying the High Risk Patient

- History of Confinement > 3 hrs (Black)
  - OR cases, ER/IR stays
  - Found down at the scene
  - Today, may be longer if DTPI due to ischemia rather than tissue deformation
- Other factors unknown
  - Anemia (Baharestani)
  - Anticoagulant Use (Richbourg)
  - Anemia and AC (Honaker)
Early presentation of purple-maroon tissue at 48 hrs

Blister phase at 72-96 hours

Necrotic phase at 7 days

- Develops on skin subjected to pressure
  - HOB up = sacral, upper buttocks at sacrum
  - Flat = buttocks tissue
  - Sitting erect = ischium
- Can develop under medical devices

- Epidermal loss with skin slippage
- Red blistered skin visible
- Commonly called a skin tear
Diagnosis of DTPI

- Usually by inspection
  - Issues with misidentification
- Ultrasound
  - Aoi, PRS, 2009
  - Aliano, 2014
- Elevated CPK
  - False positives
- Thermography
  - Bhargava, 2014

Recommended Treatment

- Relieve pressure completely from areas likely to have DTPI
  - Side to side turning
  - Support surfaces
  - Heels in boots
- Maintain perfusion
- Building evidence for noncontact low frequency ultrasound
  - Honaker Research
  - Sanada research on vibration
- Vasolex unknown
- No evidence today to support:
  - Early debridement
  - HBO

NCLFUS and DTPI

- NCLFUS reverses some DTPI pathophysiology
  - Nonthermal improvement in perfusion
  - Wave length reaches deeper tissues when injury occurred
  - High frequency US tends to stay at skin level
  - Production of cavitation (stress) on the tissues
  - Stimulates production of nitric oxide
  - Vasodilation and promotion of angiogenesis
  - Reverses the effect of oxygen free radicals
  - Removes cellular waste
Improvement of DTPI with noncontact, low freq ultrasound

- Retrospective chart review; March 09 – March 10
  - 127 sDTI’s
    - 63 were treated with SoC only
    - 64 with SoC and non contact low frequency ultrasound (NCUS)
- Inclusion consisted of patients identified with sDTI with in 4-5 days of onset.
- All patient received standard of care
- Treatment group received NCUS and standard of care

Honaker, 2011

When DTPI is POA

- Document the presence of DTPI “in evolution” in clinical records when
  - DTPI presents within 48 hours of admission
  - History of risk
  - Blistering DTPI presents within 72 hours of admission
  - History of risk and prior intact DTPI
- Coders have instructions that DTPI in evolution is a full thickness PI/PU POA

Two Extremes of Kozia’s Pressure Time Curve?
DTPI are dangerous forms of pressure ulcers

Be sure you and your staff can recognize the high risk patient and the onset for best outcomes