Ten top tips: mitigating deep tissue pressure injury

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Deep tissue pressure injury (DTPI) was first identified as a type of pressure ulcer 20 years ago (Black, 2003). The US National Pressure Injury Advisory Panel started to formally study DTPI a few years later (Ankrom et al, 2005). While it might be believed that DTPI is an atypical pressure injury compared with the counterparts of stage 1–4 and unstageable pressure ulcers, it is likely the cause of full-thickness pressure ulcers (Berlowitz and Brienza, 2007).

For many years, it was taught that pressure ulcers start on the skin from occlusion of blood flow and the superficial wound progresses to a full-thickness wound over a fairly long time period (outside to inside development). The idea of an 'inside to outside' injury was seldom considered as a cause of pressure ulcers. However, DTPI is injury to muscle tissue from high magnitude pressure. DTPI can develop on any body part that is exposed to pressure from the underlying surface or overlying medical device. The intense pressure deforms and eventually destroys cell membranes in muscle, fascia and subcutaneous fat. The skin, fed through perforating vessels, remains intact due to its relatively low metabolic rate.

The first clinical sign of DTPI is a purple or maroon area of skin appearing around 48 hours after the pressure event *[Figure 1a]*. Due to the fact that the injury is in the muscle and other soft tissues, DTPI rapidly evolves into large full thickness, cavernous wounds *[Figure 1b and c]*. The direct injury also leads to ischaemia of distal tissues and an inflammatory response. Like other ischaemic problems, the continued poor perfusion extends the wound beyond the original damaged cells (Collard and Gelman, 2001). DTPI can, and often do, take months or years to fully close and come with an increased risk of sepsis and osteomyelitis due to the advanced necrosis of subdermal tissue with exposure of bone.

Due to these wounds having such a rapid pathogenesis, it is imperative every clinician knows how to recognise DTPI while there is a chance to mitigate the harm to the patient. The following 10 tips are aimed at assisting any clinician in recognising the signs of an impending DTPI and helping to avoid significant deterioration of the patient and the wound.

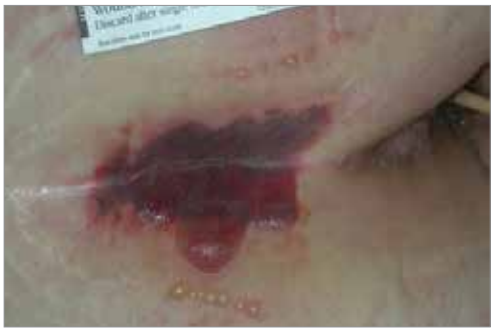

Figure 1a. *First appearance of deep tissue pressure injury as purple intact skin.*

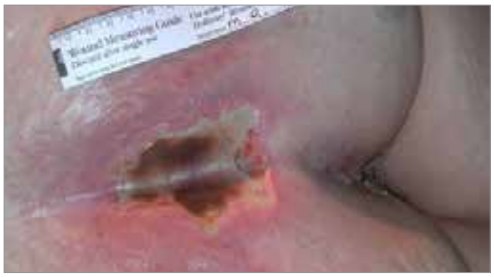

Figure 1b. *Evolution of the wound to an unstageable pressure injury.*

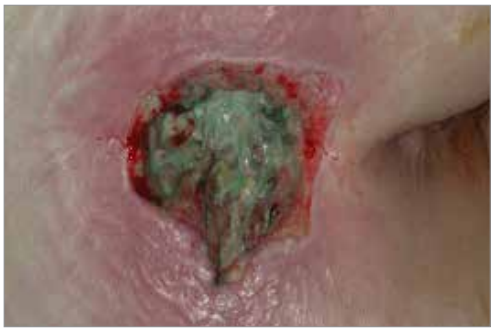

Figure 1c. *Fully necrotic wound bed.*

1 Identify those at high risk of DTPI: Traditionally, nurses have relied on using evidence-based Pressure Ulcer Risk Assessment Tools, such as the Braden scale or Norton scale to identify those at risk for a pressure ulcer. However, these tools are not as specific to DTPI risk as they perhaps could be as they were developed when the underlying pathophysiology of pressure ulcers was understood to be ischaemia alone and, therefore, mitigatable with turning, skin moisture management and improving nutrition. Therefore, it is important to have a high index of suspicion for patients at risk for evolving DTPI. The aetiology for a DTPI is often relatively short

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immobility on a hard surface. Scenarios that can lead to this can be:

1. a fall or found down at the scene for an unknown duration
2. lying on an operating theatre table or gurney for more than 3 hours
3. nerve blockade preventing awareness and capability to change positions
4. not turning the entire body while in a comatose state (iatrogenic or otherwise)
5. prolonged sitting in an improperly cushioned chair.

Risk stemming from the operating theatre and the paralysed patient have been addressed in previous Ten Top Tips articles (Black et al, 2014; Black and Hotaling, 2021).

Clinicians who have worked for many years appreciate the fact that DTPI was not seen or described decades ago, so questions such as "Why now?" "What is different?" have been raised. There is a small bit of research on the increased incidence of DTPI in patients with impaired coagulation (thrombocytopenia, anticoagulants or liver failure) and/or have anaemia (Richbourg et al, 2011; Honaker et al, 2014). The association of new forms of anticoagulation on the development of DTPI has not been elucidated.

2 Appreciate the natural history in presentation of DTPI: The pathophysiology of DTPI is tissue cell membrane destruction, akin to the zones of injury seen with thermal burns. Due to the underlying structural death, this wound is essentially full-thickness when it first appears as a bruise or purple/maroon area on the skin exposed to pressure. The area appears between 48 and 72 hours after the patient is relieved from the pressure and will fully evolve into a full-thickness wound within 7 to 14 days, even if everything is done correctly from a treatment perspective once it is identified. It is important for members of the care team to not blame another discipline for failing to prevent this evolution, especially when the exposure to pressure preceded hospitalisation.

DTPI does not have extensive work in diagnostic assessments. Most of the diagnoses stems from the history and rapid evolution of the wound. Long-wave infrared thermography (or thermal imaging) also aids in identification of DTPI. Human skin is a nearly perfect emitter of infrared radiation and relative temperature differences can be used to diagnose changes in perfusion and inflammation of tissue. DTPI can exhibit either increased or decreased skin temperature (Bhargava, 2014) and thermographic images can capture both. Thermal imaging is being done with increasing accuracy in diagnosing areas of ischaemia in normal appearing tissues. [Figure 2]. A study done to examine subepidermal moisture measurements (SEM) in known deep-tissue pressure injury visible as purple or maroon skin compared to stage 1 pressure ulcers indicated that the SEM values could not discriminate stage 1 and DTPI (Gershon and Okonkwo, 2021).

3 Prophylactically intervene for the high-risk patient: Medical providers have long identified the need for deep vein thrombosis and gastrointestinal prophylaxis in high-risk patients within hours of admission to an inpatient facility. DTPI prophylaxis needs to follow the same paradigm. While DTPI prophylaxis is not as easy as ordering an anticoagulant or proton-pump inhibitor, it is crucial to prevent patient harm. When the patient has a risk profile for DTPI, interventions should be put in place to improve and not impede perfusion.

One of the crucial components of DTPI prophylaxis is removing as much pressure as possible from the at-risk area. This may include a low-air-loss or level 2 mattress, prophylactic 5-layer foam dressings, heel offloading boots,

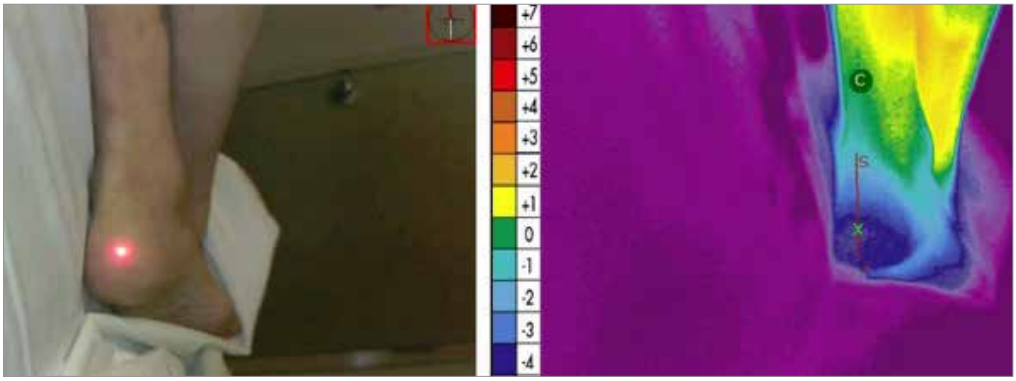

Figure 2. Thermographic image of the heel of a high-risk patient for DTPI shows an area of ischaemia, noted as blue area on thermographic scan. Note that the skin of the heel is intact and shows no signs of ischaemia.

and turning schedules avoiding the high-risk body part. The authors' clinical experience has demonstrated that upscaling the bed, implementing prophylactic dressings and offloading the affected area have been shown to reduce the severity of the DTPI.

4 Consider differential diagnoses for DTPI: Many clinical conditions can lead to purple skin; especially trauma and ischaemia. There are several important differential diagnoses for DTPI, all of which have different plans and treatments. The most common differential diagnoses are traumatic injury leading to haematoma, internal haematoma (Morel-Lavallée lesions) and chronic friction. Ischaemia stems from vasopressor use, warfarin necrosis and thrombotic events with COVID-19 (Black et al, 2020). The National Pressure Injury Advisory Panel (NPIAP) website has a document detailing examples of differential diagnoses for DTPI (NPIAP, 2021).

As in other medical diagnoses, it is imperative to determine the correct diagnosis to correctly treat or manage the disease. Interventions for one of these diseases will not be indicated or may possibly be contraindicated for another. Therefore, it is critical to determine what the most likely disease process is to avoid further harm to the patient with ineffective to inappropriate treatments.

5 Examine the patient's backside. If unable to turn, order an upscaled bed: The most important component in DTPI is to remove as much pressure from the wound as possible, which is done by turning the patient off of the wound. The easiest way to determine a patient's bed mobility is to ask the patient to roll onto a side so the backside can be examined. If the patient is unable to independently turn over in the bed, an upscaled bed needs to be ordered.

It is often thought that the bed is the only needed source of pressure relief. However, the support surface does not adequately relive pressure on an existing pressure injury. Many clinicians incorrectly conclude that the upscaled bed eliminates the need for turning schedules and offloading. Additionally, a bed that comes with a turn and reposition system also does not eliminate the need for additional turning from staff, but does reduce some of the burden on staff, so they can turn the patient the necessary 30 degrees lateral. Reducing the intensity of pressure on skin and soft tissue is aided by the support, but the only way to remove pressure from one body part is to shift it to another one by turning the patient.

6 Consider prolonged sitting as an aetiology of DTPI: Much effort has been made to mobilise patients early. While this is an important initiative, when the patient is slid onto a Geri chair or cardiac chair from the bed, the purpose of mobilisation is not met. In fact, the core support needed to maintain an upright posture is not strengthened while using these devices. Further, many patients require restraint in the chair to keep them from sliding which creates shear stress in the soft tissue. Finally, the padding of the chair is often quite thin. If patients are transferred to a bedside chair, the duration of sitting must also be controlled. Weak patients often slide down in the chair to rest on the sacrum, which is nearly the position they were lying in the bed. If there is minimal support from the cushion, one needs to be ordered promptly. If the chair cushion, the patient needs a temporary cushion and hourly repositioning in the chair, as it is unsafe to sit for prolonged periods without adequate pressure relief. Additionally, when returning back to bed from the seated position, it is important to provide sacral pressure relief by placing the patient on their sides, rather than supine.

7 Educate colleagues to promptly consult wound specialist in a high-risk patient with a new bruise or DTPI: Consultants play a significant role in the care of any patient. Because DTPI has not been known for long, wound care experts should be consulted early. Due to the natural history of DTPI with its rapid evolution, this type of wound needs close expert monitoring to evaluate for its progression and be able to adapt the treatment plan based on the wound status. Wound specialists, regardless of discipline, are taught to correctly identify/diagnose the wound, obtain appropriate evaluation and implement the appropriate treatment plan for this type of wound. In the age of telehealth technology, a wound specialist should not be out of reach. It may need some collaboration with the healthcare system to develop a contract or agreement with an outside wound group.

8 Consider the use of non-contact, low-frequency, ultrasonic Mist to reduce the severity of DTPI: Some research shows early resolution of DTPI with non-contact, low-frequency ultrasound (NCLFUS). Honaker and colleagues conducted a prospective study and reported that only 6% of patients with DTPI treated with NCLFUS developed full-thickness

pressure injury. This is in contrast to the control group having 64% of the patients developing full-thickness pressure ulcer when no NCLFUS was used (Honaker et al, 2016). Wagner-Cox (2017) studied 44 patients with both hospital and non-hospital acquired DTPI who were treated with NCLFUS and reported that all 44 patients had some resolution of the wound size and 10 patients completely resolved the DTPI with an average of 5.7 treatments (SD 3.4). Greer (2014) reported that 67% of DTPI resolved with NCLFUS, and of note, this study was conducted after hospitalisation, so those DTPI were more evolved. A Wounds UK meeting report details some of the other wounds that benefit from the debridement provided by the NCLFUS (MIST) system (Wounds UK, 2011). The 2019 Pressure Injury/Ulcer Clinical Practice Guideline contains a recommendation that NCLFUS be considered as an adjunct therapy for suspected DTPI (EPUAP et al, 2019).

9 **Examine the clinical evidence on the patterns of DTPI**: Most hospitals use a root cause analysis system of examining the circumstances that precede significant patient harm as part of a quality improvement programme. Because the timing of DTPI is consistent, the hospital quality team should be able to identify where the patient was at the time of DTPI started. This process is not to blame anyone for the events, but to explore the events so that they can be prevented in others; did most of the DTPI cases begin in surgery? Then examine the mattress in use and if the patient's risk was identified and properly attended to with preventive dressings or mattress overlays. Are most of the DTPIs stemming from critical care and appearing on the sacrum and medial buttocks? Then examine the use of preventive sacral dressings and the usual position of the head of the bed. Most hospital support surfaces can reduce the intensity of the pressure when the head of the bed is elevated 30 degrees, but once the head of the bed is higher, the ability of the support surface to mitigate shear and pressure is reduced. Are most of the DTPIs on the face in patients using oxygen delivery devices? Then examine the processes of care for moving the device or padding the skin.

This quality improvement process has its roots in the morbidity and mortality conferences which are designed to review adverse outcomes to prevent recurrence of errors and improve quality. Although it can feel demeaning to bring a case to this system level of review, a crucial component to improving healthcare systems is to identify weaknesses and areas for opportunity of improvement for future patients, as well as validate strengths. As DTPI is a complex phenomenon and multiple areas of care and opportunities for potential failure, it is crucial to bring these injuries forward for detailed examination. Both medicine and nursing disciplines need to determine what was successful, what needs to be re-evaluated, and what needs to be completely changed to prevent additional patients from succumbing to the same negative outcome as presented above.

10 **Advise the patient and family about the seriousness of DTPI:** The patient and family need to be informed that DTPI "will get a lot worse before it gets better". These patients realise that it did not take long to develop the pressure ulcer and expect it to get better quickly. Many of these patients require over a year to heal and multiple debridements, if not additional or more extensive surgery, such as flaps.

Conclusion

DTPIs start as an inconspicuous bruise that rapidly becomes a full-thickness and often necrotic wound, teaming with potential for harm, such as sepsis and osteomyelitis, pain and suffering, reduced quality of life, as well as the possible need for drastic surgical repair including muscle flaps and/or negative pressure wound therapy. Physicians need to identify these life-threatening wounds for what they are, consult the appropriate expertise to manage this significant wound, promote the highest level of function and capability, as well as the ongoing determination to mitigate harm to future patients by unsafe or inappropriate practice. WINT

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