When is a pressure ulcer not a pressure ulcer?

KEY WORDS
- Pressure ulcer
- Suspected deep tissue injury
- Device-related pressure ulcer
- Wound classification

The current attention on pressure ulcer (PU) prevention has been welcomed by tissue viability nurses in the UK. However, it has also created a need for clarity and a deeper understanding around what is, and what is not, a PU as well as a need to define when PU development may be unavoidable. Two areas in PU development that remain difficult to prevent and classify as avoidable or unavoidable are suspected deep tissue injuries (SDTIs), and device-related pressure ulcers (DRPUs). The aim of this article is to provoke debate around these two complex areas of skin damage. In terms of serious incident reporting, and associated financial penalties or reimbursements, it is important to distinguish correctly if SDTIs and DRPUs are PUs, or something quite different. Therefore, it is imperative to investigate, define, and classify these lesion types correctly, based on robust evidence.

HEIDI GUY
Tissue Viability Clinical Nurse Specialist, East & North Herts NHS Trust; Honorary Lecturer, University of Hertfordshire, Hatfield

PAULINE GILROY
Senior Tissue Viability Clinical Nurse Specialist, West Hertfordshire Hospitals NHS Trust, Watford

FIONA DOWNIE
Nurse Consultant Tissue Viability, Papworth Hospital NHS Foundation Trust, Cambridge; Senior Lecturer in Tissue Viability, Anglia Ruskin University, Cambridge

Over the past few years in the UK, there has been an increasing focus on the prevention of pressure ulcers (PUs) because they have come to be recognised by the Department of Health as a preventable harm (Stephen-Haynes, 2011). Targets for their reduction have been advised nationally via Safety Thermometer (Health and Social Care information Centre, 2013), and set locally by commissioners.

This focus on the prevention of PUs has been welcomed by tissue viability nurses, who have persistently strived to introduce strategies that will prevent PU development. However, it has also created a need for clarity and deeper understanding around what is – and what is not – a PU as well as a need to define when PU development may be unavoidable.

Two areas in PU development that remain difficult to prevent and classify as avoidable or unavoidable are suspected deep tissue injuries (SDTIs; European Pressure Ulcer Advisory Panel [EPUAP]–National Pressure Ulcer Advisory Panel [NPUAP], 2009), and device-related pressure ulcers (DRPUs). In this article, the authors aim to provoke a debate in relation to both these areas.

BACKGROUND

EPUAP–NPUAP (2009) define a PU as an area of localised damage, usually over a bony prominence, that occurs as a consequence of pressure or pressure in combination with shear. PUs are usually associated with a period of immobility, leading to pressure from the weight-bearing point on the skeleton causing partial occlusion in the blood flow to the local tissue. However, they can also occur as a result of pressure from an external device or object pressing against the skin, and in these instances may not be related to a bony prominence.

PUs present at varying depths. There have been several attempts to produce tools to aid in the staging of PUs based on depth, the EPUAP–NPUAP (2009) tool being one example. In 2005, NICE recommended that the EPUAP grading tool be adopted nationally, thereby promoting a national standard for defining pressure ulcer damage levels. In 2009, the EPUAP in conjunction with the NPUAP redefined the staging of pressure ulcers to produce the current national grading tool. The NPUAP (2009) were already considering additional definitions (*Box 1*) of damage – possibly because of the way finance for health care is distributed. The case was different in Europe

Box 1. Pressure ulcer classification additional categories for the USA (NPUAP–EPUAP, 2009)

Unstageable/ Unclassified: Full-thickness skin or tissue loss – depth unknown

Full thickness tissue loss in which actual depth of the ulcer is completely obscured by slough (yellow, tan, gray, green or brown) and/or eschar (tan, brown or black) in the wound bed. Until enough slough and/or eschar are removed to expose the base of the wound, the true depth cannot be determined; but it will be either a Category/Stage III or IV. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as "the body's natural (biological) cover" and should not be removed.

Suspected Deep Tissue Injury – depth unknown

Purple or maroon localized area of discoloured intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue. Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid exposing additional layers of tissue even with optimal treatment.

because financial reimbursement or penalty was not attributed to pressure damage.

The scene has now changed again in the UK, and there is a need to differentiate between the following:

- The levels of damage caused by pressure/shear.
- When this damage is unavoidable.
- When pressure/shear is not the cause.

Current practice in the UK is the reporting of a grade 3–4 PU as a serious incident, and a root cause analysis (RCA) will ensue. Commissioning for Quality and Innovation (CQUIN) and quality targets are set locally by commissioning organisations who receive the results of the RCA. Correct diagnosis of lesions is, therefore, essential in the avoidance of financial penalties for lesions that are not grade 3–4 PUs, and more importantly that correct prevention strategies can be put in place. However, it is well recognised that clinicians have difficulty grading PUs (DeFloor et al, 2006; Kelly and Isted, 2011). This diagnosis becomes harder for the clinician to make where wound depth is unclear, because the lesion presents as an area of purple discolouration (i.e. an SDTI).

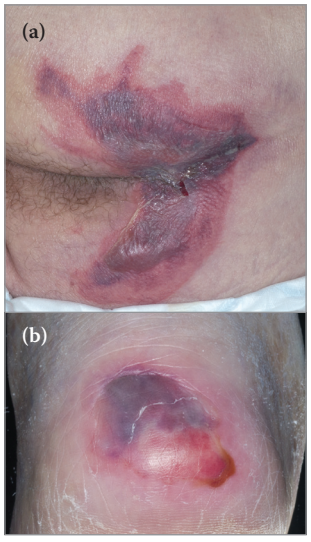

Figure 1. Examples of suspected deep tissue injury to the (a) sacrum and (b) heel. Note the purple colour of the tissue.

SUSPECTED DEEP TISSUE INJURY

The nature of deep tissue injury (DTI) has been debated in the literature for many years (Ankrom et al, 2005; Zulkowski et al, 2005; Salcido, 2006; Fleck, 2007; Briggs, 2011; Stewart and Salcido, 2012). Bliss (1992) informed us that Sir James Paget described DTI in 1862 as "purple or yellow discolouration from the excavation of blood or bloody fluid". Of course, we can be confident that in 1862 the population neither lived as long, nor with as many comorbidities, as it does today. Therefore, it is fair to speculate that skin in a patient dealing with disease processes/ medications that have the potential to affect tissue vascularisation/perfusion is more likely to fail and develop an ischaemic event far more rapidly than in a healthier, perhaps younger, patient. In addition, we are now in an age of advanced medicine and surgical techniques, and older patients are being exposed to – and surviving – complex surgeries and prolonged medical management. Interestingly, in a recent prevalence study by VanGilder et al (2010), patients with an SDTI had a mean age of 71.9 ± 1.03 years, higher than those who had grade 3–4 PUs.

It has been suggested that PU grading be kept simple – for instance, "superficial" and "deep" (Fletcher et al, 2008; Downie and Guy, 2012). This is easier to do when there is an open wound with visible tissue structures or devitalised tissue. However, on initial visual inspection it is not possible to determine the depth of some lesions (Briggs, 2011), or the underlying cause. These present as purple areas of discoloration (*Figure 1a*). Certainly, purple discolouration can be caused by pressure (*Figure 1b*), but it can also be due to other causes, idiopathic or known (e.g. haemorrhagic lesions, purpura [Khetan et al, 2012], or end-of-life skin changes [Sibbald et al, 2009]).

Smart (2013) has hypothesised, in her commentary on DTI, that in the metabolically unstable patient this injury is actually an ischaemia–reperfusion injury. As Smart (2013) reports this is a likely scenario in the critically unwell patient receiving life support who develops a SDTI. Anecdotally, the authors have witnessed such events in the intensive care setting where SDTIs have disappeared as the patient's general condition improves.

To confirm that an SDTI is pressure damage, it is necessary to refer back to the definition of a PU, localised and over a bony prominence (EPUAP–NPUAP, 2009). It could be argued that if the SDTI is neither localised nor over a bony prominence then an alternative aetiology needs to be considered to reflect the cause (*Figure 1a*). We should, therefore, debate that if it is not known

whether the underlying soft tissue is damaged (e.g. *Figure 2*), a period of observation is agreed prior to classifying. Briggs (2011) provides an example where deep tissue was proven not to be involved in an SDTI. Again anecdotally, Briggs' finding has been reflected in local practice; cases where an area of purple has disappeared one week after discovery, and either no damage or a superficial (grade 2) PU remains. There have been other cases where the area does not change for many months. Sometimes these areas are macular and small (i.e. 1 cm × 1 cm; Sibbald et al, 2009) over the heel or on the toe (*Figure 3*). They cause the patient no discomfort and fade over time. Perhaps they are some kind of isolated idiopathic or thrombocytopaenic purpura?

Another area of debate around SDTI is skin failure. In 2009, the SCALE (Skin Changes at Life's End) consensus group (Sibbald et al, 2009) postulated that: "At the end of life, failure of the homeostatic mechanisms that support the skin can occur, resulting in a diminished reserve to handle insults such as minimal pressure". This paper describes sudden-onset skin injury occurring near to or within a few weeks of death. These lesions usually occur over the sacrum, and were described by Charcot as early as 1877 (Sibbald et al, 2009).

But what of those patients who are resuscitated from a dying state? Perhaps the homeostatic mechanisms that support the skin fail during this period and, as such, an SDTI occurs? This injury may be immediately visible, or there may be a delay in manifestation, which makes diagnosis problematic. For clinical practice to progress, clinicians need to observe and report SDTIs in detail.

LEARNING FROM WHAT WE KNOW

The deterioration of tissues as a result of cell death secondary to pressure follows a complex cascade of events (Stekelenburg et al, 2008; Leopold and Gefen, 2013) involving rigor mortis of muscle (Gefen, 2007), and eventually enzymatic breakdown of dead tissue resulting in a crater-like wound. The wound may extend down to bone, but will certainly extend to muscle (Leopald and Gefen, 2012). The time scale associated with this process is unclear, but may take a couple of weeks, and the skin may remain intact during this period. Forensic science may explain this process.

The skin is a more resilient tissue than muscle, ligament, or tendons, which all deteriorate in a corpse well before the skin (Farid, 2007). Therefore, if an area of purple discolouration on or under the skin is a DTI due to pressure, it could be expected that during a (presently-undefined) time period, it would progress to an eschar and

"The deterioration of tissues as a result of cell death secondary to pressure follows a complex cascade."

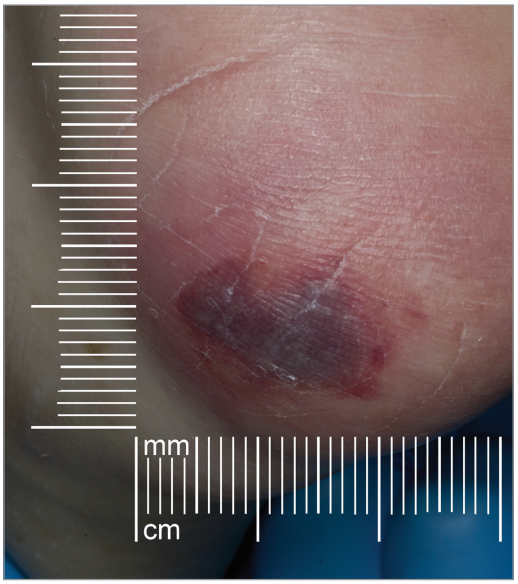

Figure 2. Examples of suspected deep tissue injury for which it is not possible to detect the extent of damage based on visual assessment.

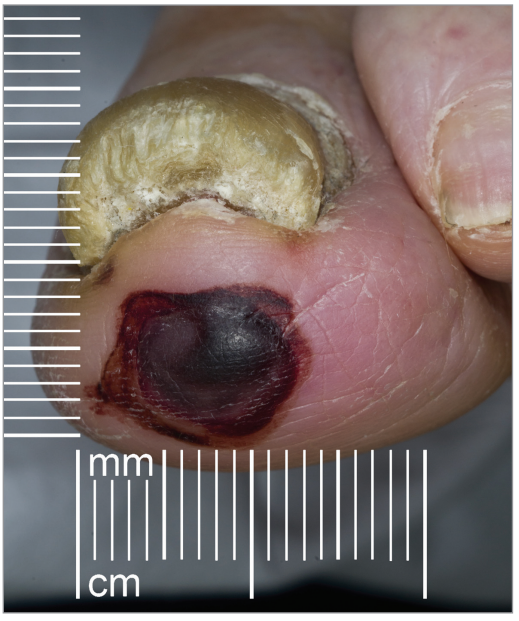

Figure 3. An example of suspected deep tissue injury to the apex of a toe.

"It is important for commissioners to understand that pressure ulcers initially reported as grade 3–4, may in fact not be grade 3–4 pressure ulcers – making associated financial penalties inappropriate."

eventually a cavity wound (Briggs, 2011). If this process has not occurred in a given SDTI, it is logical to conclude that perhaps the skin lesion was not due to pressure damage, and other causes should be sought.

It is interesting that this type of PU includes the word "suspected". This means that, when diagnosing a lesion based on what is seen, the clinician suspects that pressure has caused the damage. However, to suspect is no guarantee of guilt; the very term suggests it is necessary to further investigate the underlying aetiology of the lesion in question.

Given the uncertainty and time-dependent nature of these lesions highlighted here, it is important for commissioners to understand that PUs initially reported as grade 3–4, may in fact not be grade 3–4 pressure ulcers – making associated financial penalties inappropriate. Likewise, these uncertainties call into question the practice of immediately grading SDTIs; a period of observation – alongside preventative measures – will determine whether a lesion manifests as deep damage, or not. Only a close understanding of the purple SDTI gained through continued research and reporting will elucidate their pathogenesis, and whether prevention is possible.

DEVICE-RELATED PRESSURE ULCERS

Little has been published on pressure damage that occurs in awkward areas where general prevention strategies are not always effective. PUs caused by medical devices fall into this category; the areas affected can include nose, lips, ears, neck, palms, anus, genitalia, and so on.

As medical technology advances there is an increasing range of medical devices being used in patient care, such as nasogastic tubes, nasal cannula, tracheostomy tubes, etc. These devices can threaten skin integrity for a number of reasons:

- The materials used to manufacture the devices (e.g. plastics, silicone, rubber) are generally quite rigid.
- The devices can cause direct pressure to the skin, not necessarily over bony prominences.
- Some devices require a tight seal and/or secure fixation to be effective, causing unavoidable and direct pressure to the skin.
- The presence of medical devices can affect the microclimate of the skin (Black et al, 2010).
- The device, or fixation required for its use, can restrict regular skin assessment and inhibit the early detection of tissue damage.

As clinicians, do we always recognise this type of tissue damage as pressure damage? Or are they referred to as "other" wounds (e.g. critical illness injuries, trauma wounds, etc)? Failure to identify the true aetiology of tissue damage can result in poor prevention and management strategies. The published literature identifies that the incidence of DRPUs is between 21% and 34.5% (Black et al, 2010; Apold and Rydrych, 2012; Jaul, 2013). A similar prevalence has been found by one of the authors in an unpublished audit undertaken in large district general hospital.

The audit identified a 31% incidence of DRPUs between November 2011 and January 2012. Despite the implementation of several local PU prevention initiatives, the incidence rose to 45% in the same time period one year later (albeit recording skin damage of a more superficial degree). The majority (88%) of the recorded DRPUs occurred on the head or neck. The reviewing of these data shows that, while progress in PU prevention is generally being made, the impact on DRPU prevention is not as evident.

So are DRPUs avoidable or unavoidable? It must be acknowledged that a large proportion of patients requiring a medical device are likely to be clinically unwell and have multiple comorbidities and existing risk factors for PU development. Equally, while increasing the risk of pressure damage, the devices used may be delivering life-saving treatment – making the resultant tissue damage, arguably, unavoidable. However, DRPUs can be related to the poor placement, incorrect selection of, prolonged inappropriate exposure to, or inadequate protection from, such devices – meaning that a proportion DRPUs are avoidable if effective strategies to protect the patient from device-related pressure are put in place.

Like SDTIs, DRPUs require more observation to increase our understanding of their development and prevention. In addition, the materials and product design of some of the devices causing pressure/friction may not have been reviewed for many years and it is essential that clinicians engage with manufacturers to promote tissue protection.

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CONCLUSION

The authors hope to have provoked debate in two complex areas of skin damage presently classified as PUs. The exact aetiology of SDTI is not known, but potential pathogenic mechanisms and pathways are discussed. How best to grade such lesions has financial repercussions and, more importantly, informs decisions about the management of the lesion and patient.

Clinical experience, and a review of the literature, suggest that to categorise all purple, discoloured areas of skin as DTIs is inaccurate. It is not always possible to ascertain depth, causation, or the natural history of a lesion by visualisation alone at a single point in time. As such, there is a gap between PU aetiology and what is clinically visualised and reported. This gap appears never bigger than when describing the purple SDTI.

In the case of DRPUs, the question remains: "Do all clinicians classify DRPUs as PUs and count them in their PU prevalence figures?" It may be that they are categorised as something else all together. And if the DRPU is not over a bony prominence, is it a PU? The authors suggest that it probably is a PU, but friction does need to be excluded from the cause.

In terms of serious incident reporting, and basing financial penalties or reimbursements on those reports, it is important to distinguish whether SDTIs and DRPUs are actually PUs, or something quite different. It is imperative that we define and classify both lesion types correctly, based on robust evidence. In addition, it is essential that collaborative working goes on between the clinicians reporting PUs and the commissioners setting the targets, and that they agree on reportable definitions and time frames (Briggs, 2011). WUK

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