Case study: A pressure wound or a wound as the result of an internal calcifying disorder?

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ABSTRACT

Pressure wounds are not uncommon in persons with spinal cord injury. This study of an ischial tuberosity pressure wound featured an unexpected finding in the wound bed — calcified material. Following internal imaging and histopathology of the material, "Fat necrosis with dystrophic calcification" was reported. The wound was then deemed not to be a pressure wound, but due to its anatomical position it was conservatively managed as such, when surgical intervention was declined by the patient. This time-line case study depicts holistic management of a patient with tetraplegia at home with a slow healing wound. Strategies to reduce external pressure forces on his wound are discussed; the healing was most likely impeded by internal forces from a calcifying disorder. With regular monitoring and care, the wound appeared healed a year after first sighting the calcified material. With no medical therapy prescribed and no confirmation that the calcification disorder is no longer an issue, the question arises as to how long the skin will remain intact.

Keywords: Dystrophic calcification, spinal cord injury, wound management.

INTRODUCTION

There are an estimated 12,000 people living in Australia with a spinal cord injury (SCI)[1]; (2008 is the latest year from which data from the Australian Spinal Cord Injury Register is available). With a pressure injury prevalence rate of 30% at 20 years following SCI[2], approximately 3600 of these persons will have a pressure injury at any one time; limited mobility and/or limited sensation remaining the highest risks for pressure injury development[3]. Amongst the pressure wound literature[4,5], there are specific management guidelines[6,7], models of care[3], a data set[8] for persons with SCI, and a paper written on physiological changes possibly affecting wound healing in persons with SCI[9].

This case study is of Mark (pseudonym), a person with SCI with what was initially thought to be a pressure wound, but Mark's case is unusual in that material was found in the wound bed as a result of an internal calcifying disorder. This paper highlights the ramifications of SCI on body systems, in particular the integumentary system, below the level of injury. Throughout this study, Mark was actively engaged in making informed choices managing his wound towards healing; the conservative strategies are discussed.

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MARK'S HISTORY

Mark, now aged 60, sustained SCI from a motor vehicle accident in 1986. His injury is described as cervical 5 vertebrae level tetraplegia; he is dependent on others in all activities of daily living. Tetraplegia is an impairment of all limbs, trunk and pelvic organs and a compromise to respiration. Mark's extent of impairment is incomplete, which indicates that there is some sparing of sensory and motor nerves below the level of his injury. Following admission to a spinal unit and time in a rehabilitation unit, Mark was discharged home and has lived as full a life as he possibly could with his level of injury. Mark lives with his mother in a large house divided into two self-contained units. His mother attends meal preparation; all domestic and Mark's personal care is attended by private carers.

Mark's medical history includes a myocutaneous flap for a pressure wound on his right ischial tuberosity (IT) in 2000. In an erect sitting position the body rests on the right and left tuberosity of the ischium. Other medical history includes insertion of a pacemaker in 2008 for cardiac arrhythmia, obstructive sleep apnoea treated with continuous positive airway pressure via nasal prongs overnight, renal impairment and a split skin graft for a burn to the left foot in 2015. He has central adiposity, does not have diabetes, does not smoke and partakes of a normal diet. A neurogenic bladder is managed by an indwelling catheter. He has no known allergies. Medications included oxybutynin, thiamine, baclofen, fludrocortisone, diazepam and multivitamin. For chronic neuropathic pain he is prescribed gabapentin.

Mark's wound, pertinent to this study, was situated between his right peri-sacral area and his right IT. When dependent on

a wheelchair for mobility, skin in these areas is at high risk, as the region experiences constant pressure between the underlying IT bony prominences and sitting surfaces. Due to its close proximity to the IT, the wound was initially described and managed as an IT pressure wound.

The 12-month history of Mark's skin in this region was that it would break down, be treated at home by community nurses, heal but not stay healed. Due to interference of the catabolism and biosynthesis of neurologically impaired skin, there is a decrease in skin elasticity, which, in turn, decreases the skin's tensile strength below the level of injury, making the skin more susceptible to injury[6]. Mark's skin over his right IT was described as fragile and dusky coloured, which would signify poor oxygenation/capillary supply, although the skin blanched readily. Individuals with SCI have a reduced blood supply below the level of injury, which results in a reduction of nutrients to the skin and surrounding tissues and a reduction in oxygen supply, increasing the possibility of ischaemia[6,9]. Muscle hypertrophy was apparent in Mark's buttocks, which is common in persons with high-level SCI who have been wheelchair-dependent for as many years.

In June 2016, Mark estimated that over the previous 12-month period the longest he had been free of wound care for skin breakdown in the right IT/peri-sacral area was one period of eight weeks. To manage skin breakdown, other than being out of bed for bowel care and showering, he had hired an alternating pressure air mattress replacement and put himself on intermittent bed rest, periodically lying on his left side to totally relieve pressure over the area of concern. Evidence does not support full-time bed rest to treat pressure injuries in persons with SCI[7]; modified sitting protocol with intermittent bed rest is the preferred option. As the individual will possibly experience respiratory compromise, full-time bed rest increases the risk of chest infection, furthers psychosocial isolation and has deleterious effects of deconditioning[3].

Historically when each wound healed, a gradual return to the sitting program tailored to Mark commenced. Consideration needed to be given to the healed area's location, carer hours available, Mark's weight-shifting ability and his transfers[7]. The goal of a sitting program is to attempt to promote skin tolerance to withstand a minimum of eight hours seated in a wheelchair on a specialised pressure-relieving cushion without detriment to skin integrity; once achieved, the skin should be able to tolerate time on a commode for bowel care and a normal day of activities in a wheelchair[7]. Mark's wheelchair seat was a custom-made air cell cushion relying on immersion and envelopment. With the completion of each sitting program, Mark was up all day in his wheelchair, but within weeks the right IT region would break down and the scenario repeated.

Variables such as Mark's sitting and lying interface pressures were regularly mapped by his occupational therapist and seating technician. Care/body sling transfers/mobilising techniques were regularly reviewed to ensure all techniques were not putting extra stretch, friction or pressure on the area.

Mark's mood was low at times; his frustration was with the failure of the wound to progress towards healing, despite his readiness to follow prescribed treatment, other than refusing professional counselling. Low self-esteem and life satisfaction have been found to be associated with pressure injury development in persons with SCI[3].

WOUND ASSESSMENT

An unusual finding, 8 June 2016

The wound was recorded as an IT pressure injury, width at surface 10 mm x10 mm and 30 mm in depth. The wound was free of slough, with no signs of granulation and was dressed with a Hydrofibre® under a hydrocolloid dressing. Due to intricate secondary changes in the way pain is transmitted and processed via the central nervous system after SCI, Mark was unable to relate to specific pain at the wound site. No oedema or signs of clinical infection were noted. An increase in haemoserous exudate had been recorded the previous week and maceration of the peri-wound was evident. On this occasion when probing the wound for possible tracking or the presence of a collection, it was noted that there was cream-coloured material in the wound bed. On initial sighting it was thought to be a plug of slough, but on investigation it was found to be hard and able to be removed from the dermis with forceps. The material piece was 15 mm x 10 mm x 5 mm, with striations and jagged edges. At this time, the secondary dressing was changed to an absorbent dressing pad to manage the increased exudate and barrier film wipes were introduced.

The community nurse reported the finding to the general practitioner (GP) who, in turn, ordered full blood count, pelvic x-ray and ultrasound. The results of these investigations reported "no frank bony destruction" and the blood results were within normal limits. Mark was subsequently referred to the local spinal plastics clinic for assessment and management advice.

While waiting for the clinic appointment, other than feeling frustrated with the repeating pattern, Mark was well. His wound care was attended as required and any hard material was removed from the wound bed and placed in a specimen container.

Spinal plastics clinic, July 2016

On the appointed day, Mark presented himself to the clinic's multidisciplinary team. The team undertook a comprehensive assessment. After observing the wound and reading the results of the GP's investigations, the plastic surgeon ordered a computerised axial tomography scan of the area and an inflammatory marker C–reactive protein (CRP) level. The specimen container of hard material pieces ranging from 10 mm x 9 mm to the size of grains of sand was sent for histopathology.

The clinic's spinal rehabilitation physician discontinued fludrocortisone, which had been prescribed in 2013 for low sodium count and syncope episodes. Mark's weight was recorded as 100.5 kilograms, BMI 30. To assist in collagen growth and protein accumulation at the wound site, the dietitian advised an arginine-containing supplement twice-daily. No management advice, different to what was already in place at home, could be offered by the occupational therapist or the clinic's nurse. Mark returned home to await the results and to continue intermittent bed rest and wound dressings.

RESULTS

The scan of the area identified "soft tissue stranding with central calcifications and gas abutting the right IT measuring approximately 46 mm x 44 mm" and "if there is clinical concern of osteomyelitis in the region, a bone scan may be helpful". The CRP was recorded as low-grade elevation. The plastic surgeon did not request a bone scan.

Histopathology reported on a piece of material, measuring 10 mm x 9 mm x 4 mm — "sections show necrotic adipose tissue and fibrocollagenous tissue in which dystrophic calcification is prevalent. Viable tissue is not seen"; the diagnosis being "Fat necrosis with dystrophic calcification".

Dystrophic calcification is one of the cutaneous calcification categories[9,10]; the most common form results from local trauma and motion or in areas subject to trauma[10] such as in Mark's case, around his IT bony prominences. It is reported that soon after bed rest begins in persons with tetraplegia, cutaneous dystrophic changes occur[9]. Under normal physiological circumstances, when the body undergoes injury, innate protection mechanisms inhibit the formation of calcification within soft tissue, but when these biological mechanisms are insufficient, soft tissue can develop persistent calcification[11]. As the calcifications enlarge they may ulcerate, form sinuses and exude chalk-like material[12].

Mark's skin in his right IT region had experienced much trauma, with the history of flap repair in 2000, and there had been multiple episodes of bed rest due to breakdown in skin integrity, particularly over the previous 12-month period. Those skin issues had been managed as pressure areas/wounds but, in hindsight, they could have been associated with internal disorders, although chalky material had not been reported previously in his wounds. A less common cause of dystrophic calcification is lobular panniculitis, an inflammatory process primarily affecting the subcutaneous fat lobules; the inflammation leading to frank fat necrosis, characterised by the formation of small, dull-white foci and dystrophic calcification[12].

Spinal plastics clinic, August 2016

Mark returned to the clinic to discuss the results. The wound showed no signs of infection and there was no change in wound dimensions. A reduced rate of haemoserous exudate had been noted and the surrounding skin had improved in condition. Periodically the community nurse had reported finding calcified material in the wound bed, ranging from the size of a grain of sand and up to 5 mm-sized pieces.

The spinal plastic surgeon explained that the response to treatment for dystrophic calcification was unpredictable and treatment carried a risk of related adverse effects[10-12]. Therefore his first offering for Mark's consideration was to do nothing, other than attend wound care and allow the wound to heal, with no guarantee that the skin would remain intact considering the previous 12-month history. The second offering for Mark's consideration was surgery. Surgical options included excision with/without a fasciocutaneous flap, or incision and drainage; the risks of recurrence and poor wound healing[10] were clearly stated. Mark had the capacity to be involved in his own decision making and he declined the offer of surgical intervention, opting to continue with conservative wound management.

Mark returned home to continue with wound dressings; he returned the hired powered mattress which he found uncomfortable and changed to a normal mattress with 15 cm foam overlay; his lying and sitting pressures were rechecked by his occupational therapist. Three months after first sighting calcified material in the wound, the GP requested a repeat computerised axial tomography scan and CRP level.

Scan results

The scan reported "the area superficial to the IT is associated with an underlying thick-walled cavity outlined by air, measuring 14 mm x 38 mm x 15 mm. Multiple high-density foci surround the cavity which may represent bone fragments or dystrophic calcification" and "the underlying IT is sclerotic with possible cortical erosion raising the possibility of osteomyelitis". The GP informed Mark of the scan results and no change in CRP level and advised him to attend the spinal plastics clinic to further discuss ongoing management.

Spinal plastics clinic, October 2016

The wound dimensions were unchanged and the wound edges were firm. Serous exudate was noted and the peri-wound was described as in good condition. Since mid-September no further calcified material had been sighted. Following discussion with Mark about the latest scan results, the plastic surgeon agreed to continue with conservative management, on the proviso that by the end of the year Mark remained well and the wound was showing signs of healing; otherwise surgical intervention would be advised.

On returning home, to reduce biofilm formation that may have been contributing to the wound, a 10-minute soak of betaine surfactant and polyaminopropyl biguanide (polihexanide) was commenced prior to packing with Hydrofibre underneath a hydrocolloid dressing.

Spinal plastics clinic, December 2016

Mark was clinically well. His wound depth was recorded as 25 mm with a 7 mm width, which was the first reduction in wound dimensions since June; the peri-wound was noted to be in good condition. A reduction in CRP level was recorded and surgery was not discussed. Mark returned home to continue conservative management.

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Further reduction in wound dimensions were noted, depth of 20 mm with 5 mm width Following a seating program, Mark was up daily using his power chair's tilt mechanism periodically to relieve all day sitting pressure on the one area. The wound continued to be dressed according to exudate level. Although the GP advised Mark to revisit the spinal plastic clinic for review, he declined.

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No further dressings were required. One year after first sighting the calcified material in the wound, the area appeared healed, indent-like. A scan of the area would have indicated whether or not the underlying calcifying disorder was still evident, but as Mark refused the scan it was an unknown entity.

CONCLUSION

Sensory loss, motor impairment and changes in skin physiology below the level of his SCI lesion made Mark vulnerable to pressure wound development. When wounds do occur, holistic management plans are imperative. Whether this particular wound was a pressure wound which happened to expose material from dystrophic calcification within the IT region, or the wound was an ulceration and sinus formed by forces exuding calcifying material is not known. With no medical therapy prescribed and Mark refusing surgical intervention, the wound appeared to be healed. To the best of his ability, Mark can eliminate external pressure forces, but internal forces possibly remain, which this author believes to have been the major contributing factor to an interesting wound.

At the time of submitting this case study, in mid-September 2017, there has been no further skin breakdown in the right IT region.

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CONFLICT OF INTEREST

None.

ETHICS

Northern Sydney Local Health District Human Research Ethics Committee assessed this study as low/negligible risk.

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