Pressure sores with associated spasticity: a clinical challenge

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Atiyeh BS, Hayek SN. Pressure sores with associated spasticity: a clinical challenge. Int Wound J 2005;2:77–80.

ABSTRACT

Paraplegic and quadriplegic patients particularly those suffering from spinal cord injuries are at a high risk of developing pressure ulcerations. Unlike pressure ulcers in geriatric patients, which usually can be controlled with pressure relieving devices and local wound care, pressure ulceration complicating spinal cord injuries should be viewed from another perspective. Clinical management is also more complex because of the associated spasticity. Although it is now recognised that spasticity control is critical for management of patients with cerebral or spinal cord diseases or injuries, published risk assessment studies and risk assessment pressure sore scales fail to recognise spacticity as a major risk factor. Identification of spasticity should heighten the awareness of medical and paramedical personnel and have a positive impact on prevention as well as on treatment of pressure sores in this particularly difficult group of patients. We present our experience with a young quadriplegic patient with severe spasticity presenting with a large infected ischial pressure sore. All surgical as well as conservative attempts to achieve healing failed because of our failure to recognise the importance of spasticity control in the overall treatment scheme. Spasticity control should be included as a prerequisite for any treatment protocol of such patients.

Key words: Pressure sore • Spasticity • Paraplegic

INTRODUCTION

Pressure ulcers are defined as lesions caused by unrelieved pressure, resulting in damage to the underlying tissues. They usually occur over bony prominences and are classified as stages by the degree of tissue damage observed (1,2). The aetiology of pressure ulcers, however, is usually multidimensional. Pressure, shear, friction, moisture and poor nutrition contribute directly to the development of pressure ulcers (1). Other factors associated with the development of pressure ulcers include immobility and psychosocial factors, such as inadequate personal and financial resources and non compliance with acknowledged preventive behaviours (1,3).

Two fundamental aspects are of great importance for the development of pressure ulceration: the duration and intensity of pressure and the tissue tolerance to pressure (2). Tissue oxygenation is drastically decreased whenever external pressure exceeding capillary filling pressure is applied (loaded position). Tissue perfusion is interrupted, and tissue necrosis may develop if this interruption is prolonged beyond a critical tolerance period relative to the specific tissues. Pressure relief or unloading is required to raise tissue oxygen to unloaded levels (4). Pressure-relieving devices have become standard practice for the treatment as well as for the prevention of pressure ulceration (5).

Pressure ulcers are a serious and frequent life-long complication of spinal cord injury (SCI) and have a significant effect on general health and quality of life (1,6). Problems with

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Key Points

- pressure, shear, friction, moisture and poor nutrition contribute to the development of pressure ulcers
- pressure relieving or unloading is required to raise tissue oxygen
- pressure ulcers are a serious and frequent life-long complication of spinal cord injury

Key Points

- in the USA alone some 40,000 SCI patients develop new pressure ulcers per year
- good assessment and management can either prevent or treat pressure ulcers
- a 26 year old SCI patient developed multiple pressure ulcers
- despite good care and offloading patient continued to develop ulcers

pressure ulcers are also associated with significant costs for society (7,8). In United States of America (USA) alone, more than 40 000 SCI patients develop new pressure sores every year, and management of these ulcers can require weeks of complex treatment (9,10). Twenty-seven per cent of SCI patients transferred to special care centres present with a pressure ulcer on admission (11). Whether treated with surgery or by conservative, non surgical measures, pressure ulcers recur in 5–91% of spinal cord injured patients (12).

The risk assessment pressure sore scale generally applied to evaluate the risk of pressure ulcer development is composed of risk factors included in the Norton scale, the remodified Norton scale and the Braden scale. It includes 12 variables: general physical condition, activity, mobility, moisture, food intake, fluid intake, sensory perception, friction and shear, skin type, bodily constitution, body temperature and serum albumin level (2). Wound healing can be achieved by addressing these risk factors early and providing appropriate treatment of the ulcers (6). To adequately evaluate the impact of these risk factors on wound healing and success of the surgical procedure for patients undergoing surgical treatment, further analysis needs to be performed (2). Although spasticity is not considered as such as one of the risk factors, it is obvious that it has a direct impact on activity, mobility and friction and shear. Mechanical stress as well as difficulty of skin inspection and modification of weight distribution secondary to spasticity are definitely key factors in pressure ulceration and are detrimental to wound healing. Moreover, spasticity makes proper limb positioning following surgery extremely difficult to achieve (13).

CASE PRESENTATION

Six years ago, a 29-year-old young man sustained a severe skiing accident. He had a cervical spine fracture at the level of C5–C6 that resulted in quadriplegia and spasticity in all four extremities. Subsequently, the patient developed sacral pressure sores, then right trochanteric pressure sores, which were treated by surgical debridement and myocutaneous flap reconstruction. Moreover, the patient developed repeated minor ulcerations over various pressure sites that healed with conservative local wound management and pressure relief. Despite the use of maximum doses of oral Lioresal®, control of spasticity was not successful. More recently, he presented with a right ischial pressure sore that has failed to heal over a period of 5 months with local care and adequate pressure relief. At examination, the small contracted ischial wound communicated with an infected and foul-smelling cavity extending from the ischium to the trochanter. Radical surgical debridement of the ulcer was performed excising completely the entire capsule and resecting the ischial tuberosity. The gluteus muscle having been used previously to cover an ischial ulcer, the wound was closed over a suction drain by a VY hamstring advancement myocutaneous flap. Postoperatively, following an episode of severe spasm, the patient developed a large haematoma that was evacuated from the pararectal medial suture line, keeping it open for drainage. However, this did not heal despite 3 months of adequate local wound care and appropriate antibiotics. The patient was taken back to the operating room, the wound edges were refreshened and the ulcer cavity was thoroughly irrigated and curetted, then closed over a suction drain. 10 days later, the wound dehisced. The patient was readmitted to the hospital. Re-excision of the ulcer capsule was performed, a gracilis myocutaneous flap was rotated to cover the pararectal open wound, the previously dissected hamstring flap was advanced further and a large chest tube was inserted in the cavity and with underwater seal was attached to continuous wall suction at a low pressure. One week later, following a severe episode of spasm, the patient developed a superficial mechanical dehiscence at the site of the hamstring flap that was treated in the operating room by debridement and secondary closure. Three days later, the wound dehisced again. Wound cultures grew *Pseudomonas aeroginosa* (heavy growth), *Klebsiella oxytoca* (light growth) and *Proteus morabilis* (heavy growth) sensitive to imipenem. Despite intravenous antibiotics and frequent wet-to-dry dressings, little improvement was noticed. Nanocrystaline silver dressing (Actisorb®, Johnson & Johnson, Gargrave, Skipton, UK) was then applied resulting in net clinical clearing of wound sepsis with good stimulation of granulation tissue formation. In order to control the severe spastic episodes that would occur spontaneously or

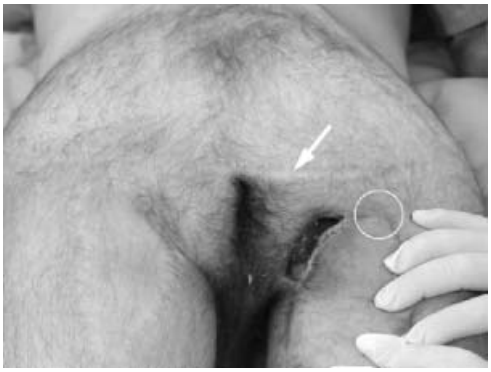

Figure 1. Wound dehiscence at the superior medial aspect of the VY hamstring myocutaneous flap. Site of original ischial pressure sore is indicated by a circle. Arrow indicates the scar of the healed gluteus maximus flap performed several years earlier to cover a sacral pressure sore.

whenever the patient is subjected to external stimuli, a total of 600 units of botulinum toxin were injected in the hip flexor muscles bilaterally (14). Partial improvement was observed. There was a marked decrease in spastic episodes at night. The patient was then discharged home on conservative wound management with Neugel® (Johnson & Johnson) which resulted in slow but gradual clinical decrease in the size of the cavity. The patient now is awaiting implantation of a pump to produce continuous dosing of Lioresal before any further therapy to his pressure ulcer is attempted (Figure 1).

DISCUSSION

Skin care and pressure ulcer prevention programs at present abound, although their content varies and their outcomes are often difficult to quantify (15). Pressure sores may heal with conservative management and local wound care, provided the site is relieved of pressure (16). Although totally acceptable in the geriatric or the terminally ill patients, conservative pressure ulcer management may not be justifiable for other patients. This mode of treatment is associated with prolonged immobilisation and is accompanied by a higher incidence of recurrence (16). Moreover, when associated with spasticity, conservative local treatment of the pressure ulceration is not usually successful (13).

The role of wound debridement with flap coverage in treating pressure sores has been clearly established (17). However, it is generally now accepted that spasticity control is an essential part of the preoperative management of patients with pressure ulcers (13). Severe spasticity can result in joint contractures, friction, shear and mobility impairment, which further impair function. Shearing movements due to spastic responses may precipitate skin breakdown. During the postoperative period, spastic patients are at risk of disrupting already repaired pressure sores resulting in wound dehiscence, flap loss, infection and haematoma (18–20). After surgery, prevention of recurrence includes avoiding pressure, optimising nutrition and local skin care, as well as reducing spasticity (18,21). Spasticity greatly interferes with mobility and self-care activities, which are critical for successful rehabilitative outcomes (19).

The present report illustrates the failure of surgical as well as conservative therapy of an ischial pressure ulcer because of underestimating the importance of spasticity in the generation of pressure ulcers and on the subsequent treatment interventions. Muscle spasm makes weight redistribution and frequent skin inspection rather difficult; moreover, proper positioning of the limb following surgery to relieve tension on the suture line is also difficult if not impossible. The fact that definitive treatment of pressure sores in spastic patients should involve not only debridement and flap coverage but also postoperative control of spasticity cannot be overstressed (18). Spasticity control should be an integral part of any algorithm for pressure ulcers treatment if the risk of complications is to be reduced and the chances of a favourable outcome optimised.

Key Points

- more specialised dressings were tried with some success
- it is generally accepted that spasticity control is an essential part of preoperative management of patients with pressure ulcers
- spasticity control should be an integral part of care

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