Ad-dressing the wound

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A multitude of dressings and a plethora of review articles dealing with the benefits of one dressing technique over another is now available. When dealing with wounds, there is a lot more to consider than just what dressing is best. One must first have a clear understanding of the etiology as well as the existence of complicating factors. It is more important to address causation than to simply cover the wound.

In regards to leg ulcers, the prevalence in Australia is estimated to be 1.1 per thousand people, with an increased prevalence of 3.5 per cent for people over the age of 65 years [1]. It is also a costly exercise; in 1995 in Wales, approximately £5.5 million per annum is spent on wound management materials alone, a substantial capital outlay [2]. It is clear then that the problems surrounding wound healing are not insignificant.

Wound healing is influenced firstly by the cause of the wound and secondly by the presence of intrinsic and extrinsic factors that delay or complicate wound healing. Primary or causative factors are those disease processes and conditions that lead to tissue breakdown and wound formation such as pressure, venous disease, arterial disease, neuropathy, neoplasia, vasculitis and trauma. The secondary influences are contributing factors; those processes which retard healing or increase the risk of tissue breakdown when associated with the causative factors. Examples of these are nutrition, infection, ischaemia, systemic illness such as diabetes, steroid excess, uremia and hepatic failure and social causes.

This article will discuss each of these factors in a simple but clinically relevant manner.

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Primary/causative factors

Pressure related damage

Pressure ulcers are said to cause problems in the range of 5.4-10.6 per cent of our hospitalised population [3-7]. Twenty three per cent of nursing home residents are said to suffer with a pressure area [8-9]. In one study, 66 per cent of people over 65 years of age with a fractured neck or femur developed a pressure area [10].

Capillary closing pressure has been shown to be 32mm of mercury. Pressures greater than this are therefore said to lead to capillary closure and ultimately tissue ischaemia and ulceration. Pressure tends to occur mostly over bony prominences and the time required for pressure ulcer development maybe as little as 2 hours [11]. Shearing and friction forces may also lead to ulceration and these forces tend to occur when patients slide against the bed surface. This is to be expected at any situation in which the patient is dragged against the bed, either by staff or by their own action.

Mobility is also extremely important. Any reduction in a patient's mobility would place that person at risk of developing pressure areas. It is important to understand that any patient in a narrow bed or with bed rails up is at increased risk as this will limit their ability to mobilise both within and outside the bed. Also any patient with an IV line, a catheter or attached to a monitor is basically restrained and therefore at increased risk.

The treatment for a patient at risk of a pressure area is to address the underlying cause. Pressure relief should be provided. This includes positioning the patient correctly and using the correct bed surface. The positioning of the patient is critical. A patient sitting out of bed is placing a great amount of weight over a small surface area and is therefore at higher risk of pressure ulceration than if lying in bed. This does not necessarily mean that the patient should not be sat out of bed but rather that having sat a patient out of bed, one should understand the risks involved. If the patient is to remain in bed, they should try to keep off their bony prominences. Positioning with pillows under the left or right whilst lying on their back or front will assist in moving the patient off their bony prominences.

It is also important to understand that patients sitting at an angle higher than 30 degrees head up are likely to slide down the bed – leading to increase shear and friction forces. Regular repositioning of individuals identified at risk should be as frequent as their skin's tolerance to pressure dictates. The patient should also be mobilised as early as possible.

Venous disease

Venous disease of the lower limb, which may be due to incompetent perforators or noncompliant valves in the veins, results in oedema and may frequently lead to ulceration. Often there is a past history of deep venous thrombosis, varicose veins or neurological or musculoskeletal conditions which reduce the effectiveness of the calf muscle pump. The patient who cannot flex at the ankle joint is unlikely to be using their calf muscle pump efficiently and is therefore likely to develop venous oedema.

The treatment of venous disease is elevation and compression therapy. If an ulcer exists, the patient should wear some form of compression bandaging, either a high stretch, low stretch or four layer bandage. Once the ulcer is healed, a compression stocking should be worn, preferably with more pressure than that provided by an antiembolic stocking. A pressure of greater than 30mmHg at the ankle is recommended [12]. If compliance is a problem, one could opt for the use of a compression pump for 1 hour in the morning and 1 hour in the evening to each leg. A recent study has shown this to be as affective as the use of bandages [13].

The patient should be informed that they must elevate their leg, preferably above the heart, whenever they are not mobilising. The patient should not be encouraged to sit rather than walk. Walking encourages the use of the calf muscle pump.

It is also important to inform patients that their condition is likely to be life-long. They will therefore require therapy for the rest of their lives.

Arterial disease

Arterial sclerosis is exacerbated by a number of factors including hypertension, smoking, hypercholesterolaemia, a family history of arterial disease and diabetes. Narrowing of the arteries leads to ischaemia and may result in ulceration of the lower limb.

Management should begin with assessment of the underlying condition and treatment of predisposing causes and conditions. The blood pressure needs to be adjusted with the preferable avoidance of Beta-blockade which can exacerbate peripheral ischaemia. Cessation of smoking should be encouraged. Cholesterol should be reduced. Aspirin and possibly other anti-platelet agents need to be considered.

The patient's arteries should be investigated initially with Doppler and Duplex ultrasound and, when surgery is considered, with arteriography. Therapy of the underlying arterial disease can be managed definitively with bypass operation, occasionally with sympathectomy and possibly by the use of medication, such as Oxpentifylline.

Neuropathy

Neuropathic ulcers tend to occur most commonly in peripheral neuropathy such as with diabetes and also with spinal disease and cerebral events such as stroke.

When a patient has a neuropathic condition, normal protective skin sensation is inhibited and there is an increased risk of mechanical, thermal or chemical trauma. The patient therefore requires significant education with particular reference to appropriate and correctly fitting footwear and avoidance of extremes of temperature such as heaters or hot baths. It is also important to treat skin trauma as soon as possible. Therefore a regular inspection regime is encouraged and this is to be done by both the patient and, where a patient is at particular risk, a podiatrist.

Neoplastic ulcer

Most common causes of neoplastic ulcers are sun exposure – some skin types are more susceptible than others. One also needs to consider neoplasia as a possible complicating factor in chronic ulceration.

The assessment of these lesions involves biopsy of any suspicious lesion. Surgical removal is the usual management for malignant neoplastic lesions.

Vasculitic ulcer

Vasculitic ulcers are usually associated with systemic auto-immune processes, such as rheumatoid arthritis, systemic lupus erythematosus and scleroderma. Occasionally a patient may develop vasculitic ulcers without any other systemic features.

Treatment normally involves the use of steroids or immune-modulators. Following a thorough history and examination, if a vasculitic ulcer is suspected, a biopsy needs to be performed. The patient is best referred to a specialist in this area such as a dermatologist.

Skin trauma

The type of traumatic ulcer depends on the type of trauma, the force used and the instrument, as well as on the patient's own skin fragility.

It is important to take extreme care with frail thin skin. Strong adhesive dressings are best avoided to prevent further trauma to potentially frail skin.

Secondary/contributing factors

Nutrition

When assessing a patient's nutritional status, one needs to be aware of multiple factors. In general, if a patient is cachetic then they might benefit from a broad approach. This would include increased protein, carbohydrate, fat, mineral and vitamins.

The most important three nutrients, however, appear to be: protein which particularly requires aggressive therapy when the albumin drops below 25g/L; zinc which is important in enzyme reactions and therefore important in tissue repair; and vitamin C which is important in wound healing.

Once a proper dietary assessment has been carried out, it is important to provide the patient and/or their carer with appropriate dietary advice in order to assist them correct any nutritional imbalances. Nutritional supplements may be required in some instances.

It is important to note that if a patient is not low in zinc then replacement can lead to toxicity. This can lead to diarrhoea as well as decreased absorption of other important minerals. Vitamin C, on the other hand, is water-soluble and therefore as long as the patient has reasonable urine output, they should not have complications with vitamin C replacement [14]. Good food sources of vitamin C include fruits and vegetables such as guava, strawberries, kiwi-fruit, oranges and other citrus fruits, capsicums, Brussels sprouts, broccoli and tomatoes. Sources of zinc include meat, hard cheeses, chicken drumsticks, nuts, dried beans, peas, lentils and wholemeal or wholegrain breads and cereals.

Infection

Infection is primarily diagnosed using clinical judgement. Routine wound swabs to diagnose infection are not warranted, as a significant proportion of chronic ulcers will yield a positive swab. However, if a patient has a red, painful wound with a fever, they are likely to have an infection and at that time a wound culture may be useful. The most accurate method for determining the presence of a wound infection is quantitative bacteriology which requires a biopsy. If one wanted to swab the wound, it would be important to remove the slough first and to swab the underlying ulcer surface.

Treatment of infection is with systemic antibiotics. Antiseptics can be considered for wounds which are heavily colonised but not infected. Reducing the bacterial load in the wound may stimulate healing, however, the use of antiseptics should be for short periods of time and not continuously, as they have been shown to reduce fibroblast activity. Avoidance of occlusive dressings has been recommended in a wound infected with anaerobic bacteria.

Ischaemia/Hypoxia

Any cause of decreased oxygen tension in the tissues is likely to retard healing. Therefore, peripheral vascular disease, respiratory illness with a reduced PO_2, and anaemia may all exacerbate tissue breakdown and retard wound healing. All of these conditions are treated by dealing with the underlying illness.

Systemic and social factors

One always needs to approach the patient holistically. There is a need to look beyond the wound. The patient may have other underlying diseases in which the disease process or therapy may interfere with the healing of the ulcer.

It is always important to take into consideration the patient's environment. Who is their carer, what is their living arrangement, what equipment do they have to aid them in mobilising or which might be leading to repeated episodes of trauma? Without having a clear understanding of a patient's circumstances, it is very hard to develop a holistic treatment regime.

Conclusion

This article does not attempt to provide a comprehensive list of courses and treatments of wounds. Its aim is to encourage health professionals to look first at treating the cause of a wound, even before using moist healing techniques.

In summary, the treatment of an ulcer is definitely not the dressing. The dressing only aids us whilst we treat the underlying condition. Without a proper diagnosis, we are only providing band-aid therapy.

References

1. Baker SR, Stacey M *et al.* Aetiology of chronic leg ulcers. Eur J Vasc Surg 1992; **6**:215-51.
2. Thomas S. The cost of wound care in the community. JWC 1995; **4(8)**:350-354.
3. Wright R & Tizianni A. Pressure ulcer point prevalence study. Prim Intention 1996; **(4)1**:18-23.
4. Girrin J & Griffiths-Jones A. Towards prevention. Nurs. Times 1989; **85(12)**:64-66.
5. Mechan M. Multisite pressure ulcer prevalence survey. Decubitus 1990; **3(4)**:14-17.
6. Martin R & Keenan A. The incidence and management of pressure ulcers in a metropolitan teaching hospital. Prim Intention 1994; **(2)2**:31-34.
7. Eves S. A pressure ulcer survey conducted at a private hospital. Prim Intention 2000; **(8)1**:14-20.
8. Langemo DK, Olson B *et al.* Incidence of pressure sores in acute care, rehabilitation, extended care, home health and hospice in one locale. Decubitus 1989 May; **2(2)**:42.
9. Young L. Pressure ulcer prevalence and associated patient characteristics in one long-term care facility. Decubitus 1989 May; **2(2)**:52.
10. Versluysen M. How elderly patients with femora fracture develop pressure scores in hospital. Br Med J (Clin Res Ed) 1986 May 17; **292(6531)**:1311-3.
11. Kosiala M. Etiology of decubitus ulcers. Archives of Physical Medium and Rehabilitation 1961; **42**:19-29.
12. Stemmer R, Marescaux J & Furderer C. Compression treatment of the lower extremities particularly with compression stockings. The Dermatologist 1980; **31**:355-365.
13. Rowland J. Intermittent pump versus compression bandages in the treatment of venous leg ulcers. Aust NZ J Surg 2000; **70(2)**:110-3.
14. Rivers JM. Safety of high-level vitamin C ingestion. International Journal for Vitamin & Nutrition Research – Supplement 1989; **30**:95-102.

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