

Optimising nutrition to prevent pressure ulcer development

Despite the inherent difficulties of measuring nutritional status, the evidence strongly suggests that malnutrition does in fact predispose to pressure ulcer development. Fortunately, malnutrition is a reversible risk factor and a recent Cochrane review concluded that older patients in the acute phase of an illness who are provided with nutrition support are significantly less likely to develop a pressure ulcer. The key is the early identification of the patients who are at the greatest risk through nutritional screening so that interventions can be initiated when it is most likely to have maximum impact.

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KEY WORDS

Pressure ulcer
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Pressure ulcers are common in most healthcare settings. Precise estimates of incidence or prevalence are unknown, but figures quoted include prevalence rates of 3–10% and 3–23% depending on the setting (European Pressure Ulcer Advisory Panel [EPUAP], 2002). A pressure ulcer is defined as an 'area of localised damage to the skin and underlying tissue caused by pressure, shear, friction and/or a combination of these' (EPUAP, 1999). Pressure, friction and shear are considered to be extrinsic factors that impinge upon the surface of the skin. In addition, several intrinsic factors can contribute to the development of pressure ulcers. Intrinsic factors alter the structural components of, and the blood supply to, the tissues or reduce the sensation or perception response mechanism, for example diabetic neuropathy where a patient may not feel the stimulus to change

position when pressure is applied. Poor nutritional status is frequently cited as one of these intrinsic factors (Breslow, 1991; Bergstrom et al, 1992; Breslow and Bergstrom, 1994; Selvaag et al, 2002). Although, the exact mechanism by which it contributes to pressure ulcer development is poorly understood.

There is a lack of research of sound methodological quality that relates to malnutrition and pressure ulcers. Nevertheless, healthcare providers commonly associate poor nutrition with the development of pressure ulcers and also with poor healing if a pressure ulcer does develop. Malnutrition is frequently found in these patients, and up to 50% have been found to be malnourished on admission to hospital (Stratton et al, 2003).

Pressure ulcers result in a huge strain on resources for the health service, which is arguably 95% preventable (National Pressure Ulcer Advisory Panel [NPUAP], 2001). Not to mention the human cost in terms of reduced quality of life, pain and suffering (Strauss and Margolis, 1996). The exact pathophysiology of pressure ulcer formation is not fully understood. Theories include cell deformation theory, ischaemia-reperfusion injury and cell-to-cell contact theory (Bouten et al, 2003). Research in animals has found that typical ischaemia-reperfusion damage can be prevented in part by

treatment with vitamin E (Houwing et al, 2000). However, further research is necessary to determine whether these results can be extrapolated to humans.

This review seeks to investigate the role of nutrition in the prevention of pressure ulcers and will focus on the effect of mixed nutritional support on pressure ulcer development in at-risk groups and on nutritional status as a predictor of pressure ulcer development with specific reference to albumin. Low serum albumin is frequently cited as a nutritional risk factor for pressure ulcer development, however, other aetiologies of hypoalbuminaemia are often not considered which has resulted in confusion and controversy (Finucane, 1995; Gilmore et al, 1995; Anthony et al, 2000).

The literature accessed for this review was obtained using CINAHL and Medline. Key terms to facilitate the search were 'pressure ulcer', 'decubitus ulcer', 'pressure sore', 'bed sore', 'malnutrition', 'nutritional status' and 'nutrition support'. Hand searching also supplemented the evidence base. A paucity of good quality clinical trials was found and several of the studies cited are from before 2000.

Effect of nutritional status on pressure ulcer development

Allman et al (1995) conducted a prospective cohort study of pressure

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ulcer risk factors among 286 patients admitted to an acute hospital. Inclusion criteria included that they were expected to be chair- or bed-bound for at least five days or had a hip fracture and were over 55 years of age. Of the subjects, 12.9% developed a grade 2 pressure ulcer or above. They found after multivariate Cox regression analysis that lymphopenia and decreased body weight were independent nutritional risk factors. Lymphopenia, defined as lymphocyte counts less than $1.5 \times 10^9/L$, resulted in almost five times the risk of developing a grade 2 pressure ulcer or higher. A body weight in the lowest quartile of baseline weight ($<58\text{kg}$) increased pressure ulcer risk two-fold.

Haemoglobin level, food intake, hypoalbuminaemia, nurse assessment of patient build, and reduced protein or calorie intake were not found to be associated with pressure ulcer development.

Conversely, Anthony et al (2000) suggested that albumin levels are an accurate predictor of pressure ulcer risk and recommended that risk assessment of pressure ulcers can possibly be improved by adding serum albumin to one of the pre-existing pressure ulcer risk assessment tools such as the Waterlow score. Using logistic regression they found hypoalbuminaemia to be predictive of pressure ulcers ($P=0.009$). However, only 6.3% of the 773 patients studied actually had a low serum albumin and the effect of confounding factors such as hepatic disease, renal disease, hydration status or diuretic usage on serum albumin was not taken into account (Thompson and Fuhrman, 2005).

Goode et al (1992) investigated the contribution of specific nutritional deficiencies (as indicated by zinc, vitamin A, C and E, albumin and haemoglobin concentrations) to the risk of developing pressure ulcers in 21 older patients with fractured neck of femur in an observational cohort study. They concluded that low concentrations of leucocyte vitamin C are a risk factor for pressure ulcer development in this patient group.

Due to the very small sample size the results of this study are not statistically significant, nevertheless, this research warrants further investigation in a much larger study.

Ek et al (1991) investigated the development of pressure ulcers in a cohort of 501 patients admitted to a long-term medical ward and who remained in hospital for more than three weeks. From this they found 28.5% of subjects to be malnourished. Furthermore they found the pressure ulcer frequency was higher among the malnourished group with 34.8% of them developing pressure ulcers compared with 20.6% in the non-malnourished group ($p<0.01$). Following multiple regression analysis, serum albumin ($p<0.001$), mobility ($p<0.001$), and activity and food intake ($p<0.05$) were found to be the most useful predictors of pressure ulcer development.

However, a description of a power calculation was not cited in the study report. Therefore, it is questionable that these results reached true statistical significance. Additionally, only about one-third of patients initially included in the study could be followed up for the full study period of 26 weeks due to earlier discharge or death. The assessment of food intake was vague and subjective. Intake is simply described as 'normal, insufficient, parenteral or no intake'. This method gives no indication of the nutrient quality of the diet consumed. Albumin, yet again is found to be a predictor of pressure ulcers. However, was the low albumin due to malnutrition or was it due to other disease processes which were not controlled for in this study? Despite these limitations this study supports the evidence that malnutrition as measured by traditional assessment techniques is a contributing factor to pressure ulcer development.

Pinchcofsky-Devin et al (1986) concluded in their cross-sectional study of 232 nursing home patients that serum albumin and total lymphocyte count were accurate predictors of pressure ulcer development and this was because they are accurate markers of malnutrition. This study

is frequently cited as evidence that malnutrition contributes to pressure ulcer development (Bergstrom et al, 1992; Finucane, 1995; Lewis, 1998). They suggested that if the serum albumin is below 3.3g/dL and the total lymphocyte count is below $1,220\text{mm}^3$, nutritional intervention should be implemented to prevent pressure ulcer development. They disregarded the fact that the other biochemical measures of nutritional status, serum prealbumin and retinol binding protein were not significantly lower in the group with pressure ulcers compared with the group without despite these parameters being measured. Also, they did not cite the anthropometric measures of either group although several measures were taken. Therefore, they based their nutritional assessment on only two parameters, which are neither highly sensitive nor specific to malnutrition (Thompson and Fuhrman, 2005).

In addition, they extrapolated that as severe malnutrition (as they defined it) was present in all the patients who already had pressure ulcers, that it was a risk factor for their development. They did not consider that the pressure ulcer could have caused the malnutrition. Albumin levels could have been lowered due to an acute inflammatory response, infection or losses through exudates (Berlowitz and Wilking, 1989; Thomas, 2001). They discovered an inverse relationship between the grade of the ulcer and the serum albumin, deducing that as the ulcer worsened the patient became more malnourished. Albumin may be a good marker of disease severity but this is not necessarily related to malnutrition (Thompson and Fuhrman, 2005).

Berlowitz and Wilking (1989) highlighted the pitfalls of relying too much on information gleaned from cross-sectional studies. They undertook a comparison of a cohort study with a cross-sectional study in the same population at a chronic care hospital. First, a cross-sectional analysis identified factors associated with the presence of pressure sores. Then cohort methodology was used to identify factors associated with the future development of a pressure sore.

Following multivariate analysis of the cross-sectional data ($n=299$), the odds of having a pressure ulcer were almost twice as much for those with impaired nutritional intake (odds ratio [OR]=1.9) and for every 10mg/ml decrease in the serum albumin, the odds of having a pressure ulcer doubled.

Twenty of the 185 patients in the cohort analysis developed a pressure ulcer (11%). Once again impaired nutritional intake was found to be a significant ($p<0.05$) independent predictor for the future development of a pressure ulcer (OR=2.8). However, hypoalbuminaemia was not associated with pressure ulcer development. This illustrates the importance of interpreting cross-sectional studies carefully. They can be subject to considerable bias. For example, albumin was not routinely measured but was more frequently measured in the patients with a pressure ulcer present on admission and on all those that developed a pressure ulcer. Several cross-sectional studies have found that patients with pressure ulcers have low serum albumin. This study supports the theory that the low serum albumin associated with pressure ulcers is a secondary factor. The pressure ulcer causes the low serum albumin rather than vice versa.

Mixed nutritional supplementation and pressure ulcer development

A Cochrane review has found the evidence to support the use of nutritional supplementation in the treatment of pressure ulcers to be inconclusive due to the poor methodological quality of the research undertaken so far (Norris and Reynolds, 1971; Taylor and Rimmer, 1974; Chernoff et al, 1990; ter Riet et al, 1995; Langer et al, 2003). However, the review found weak evidence to support the use of mixed nutritional supplements in the prevention of pressure ulcers (Langer et al, 2003).

Hartgrink et al (1998) undertook a randomised controlled trial in 140 patients who had a fractured hip and were at high risk of developing a pressure ulcer. The experimental group received the standard hospital diet and an additional one litre of nasogastric overnight tube feeding providing

1,500kcal and 60g protein. The control group received the hospital diet alone. Of the 62 tube-fed patients, only 25 accepted their tube for more than one week and 16 patients for two weeks. Therefore this trial was too small to detect statistically significant differences.

The authors concluded that the nasogastric feeding was poorly tolerated and although the incidence and severity of pressure ulcers decreased in the tube-fed group, this difference was not significant. However, energy and protein intake, total serum protein, haemoglobin and serum albumin were significantly higher in the group receiving tube feeding after one and two weeks.

There were several limitations to this study: there was no blinding as a placebo was considered unethical; despite the high attrition rate, no intention to treat analysis was performed; and the method of randomisation was not described. Nonetheless this study was useful in that it demonstrated that nasogastric feeding is not well tolerated in this patient group and other modes of nutritional support should be considered in future research.

Houwing et al (2003) undertook a double-blind, randomised controlled trial of 103 hip fracture patients. The intervention group received the standard hospital diet and two 200ml oral supplements providing a total of 500kcal, 40g protein, 6g L-arginine, 20mg zinc, 500mg vitamin C, 200mg vitamin E and 4mg carotenoids. The control group received the standard hospital diet and two 200ml non-calorific water-based placebo drinks. Patients were followed up for 28 days. They found that 59% of the placebo group developed a Grade 1–2 pressure ulcer compared with 55% in the intervention group ($P=0.420$). The incidence of grade 2 pressure ulcers alone was 18% in the intervention group and 28% in the control group ($P=0.345$). Time of onset showed a trend ($P=0.090$) towards a delayed onset of pressure ulcer in the supplemented group (3.6 ± 0.9 days) compared with the placebo group (1.6 ± 0.9 days).

The authors concluded that nutritional supplementation of this group

may delay the onset and progression of pressure ulcers. If the supplementation had been started earlier (median start day was two days after surgery and 57% of the group developed their pressure ulcer in the first two days and 76% by the fourth day after surgery), it may have been able to reduce the pressure ulcer incidence significantly. It is well documented that a significant amount of damage is done before signs show on the skin surface (Bouten et al, 2003).

Yet again, the study was far too small to detect any clinically important difference as statistically significant. A power calculation showed that 350 patients would be needed per group in order to detect a 25% difference in pressure ulcer incidence (Langer et al, 2003).

Delmi et al (1990) undertook a randomised, controlled trial involving 59 patients with femoral neck fractures in an orthopaedic unit of a hospital in Geneva. The intervention group ($n=27$) received a daily 250ml oral nutrition supplement providing 254kcal, 20.4g protein, 29.5g carbohydrate and 58g fat. It also contained an extensive range of vitamins, minerals and trace elements. They received this in addition to the standard hospital diet. The control group ($n=32$) received the standard hospital diet only. Groups were followed up for up to six months.

The authors suggest that nutritional supplementation reduces pressure ulcer incidence as well as several other complications. Again, this study was not large enough to provide conclusive evidence and had several flaws in its methodology.

Despite a lack of statistical significance due to small sample sizes all the studies detailed above reported a lower incidence of pressure ulcers in the supplemented groups.

In an additional part to Ek et al's study (1991), a randomised, controlled trial investigating the effect of nutritional supplementation on the development of pressure ulcers in the same patient group was undertaken. Subjects were

randomised into either the intervention group (who received two 200ml supplements each containing 8g protein, 8g fat, 200kcal and 24g carbohydrate in addition to the standard 2,200kcal per day hospital diet) or the control group (who received the hospital diet alone).

They found no statistically significant difference in pressure ulcer development between those who received nutritional support and those who did not. Perhaps no reduction in pressure ulcer development was noted in the group who received supplements because there was poor compliance with the sip feeds or because they reduced voluntary oral intake. Unfortunately we cannot tell this from the study.

Bourdel-Marchasson et al (2000) conducted a multicentre, randomised, controlled trial with 672 patients older than 65 years in the acute phase of a critical illness. Patients in the nutritional intervention group received two oral supplements per day in addition to the standard 1,800kcal per day hospital diet. The comparison group received the standard hospital diet alone. Each 200ml supplement provided 200kcal, 15g protein, 4.4g fat, 25g carbohydrate, 15mg vitamin C and 1.8mg of zinc. Patients were followed up for 15 consecutive days or until discharge if that came first.

At 15 days, the cumulative incidence of pressure ulcers was 40% (118/295) in the nutritional intervention group versus 48% (181/377) in the control group. This equates to a relative risk of developing an ulcer while taking oral nutrition support of 0.83 (95% CI: 0.70–0.99). The authors undertook multivariate analysis to account for differences in baseline comparability and found that patients receiving the intervention were significantly less likely to develop a pressure ulcer. Low serum albumin was also found to be an independent risk factor.

This is the largest randomised controlled trial investigating the effect of nutritional support on pressure ulcer incidence that is also of satisfactory methodological quality. It provides evidence that older people recovering from illness appear to develop fewer

pressure ulcers when given nutritional supplementation. Unfortunately, from this study we cannot deduce what part of the sip feed provided the benefit. Was it due simply to the extra energy or was it one of the micronutrients that protected the skin integrity?

Confusion exists surrounding the role of albumin as a predictor of pressure ulcer risk and as a marker of nutritional status. Serum albumin level can help to identify chronic malnutrition when used in conjunction with other nutritional parameters (Thompson and Fuhrman, 2005).

Conclusion

Confusion exists surrounding the role of albumin as a predictor of pressure ulcer risk and as a marker of nutritional status. Serum albumin level can help to identify chronic malnutrition when used in conjunction with other nutritional parameters (Thompson and Fuhrman, 2005).

Alone, it lacks the specificity and sensitivity required to accurately identify malnutrition. It has been hypothesised that low albumin is a risk factor for pressure ulcer development as it can cause cellular dehydration and interstitial oedema (Allman et al, 1995). However, from the evidence reviewed, low serum albumin appears to be a secondary factor in pressure ulcer development rather than a causative one and may act as a good marker of wound severity and wound progress. However, we cannot assume that the low albumin is due to malnutrition. But as hypoalbuminaemia has been found to be a marker of morbidity and mortality it could indirectly identify those at highest nutritional risk (Yearick et al, 1980).

There is evidence that elderly people recovering from illness benefit from oral mixed nutritional supplements, but the evidence to support their use in

Key Points

- ▶ Malnutrition is very unlikely to be the primary cause of a pressure ulcer. However, it can compromise tissue integrity and, when combined with other risk factors, may lead to a pressure ulcer in a person who otherwise would not have developed one.
- ▶ Serum albumin level can help to identify chronic malnutrition when used in conjunction with a range of other nutritional parameters. On its own it lacks the sensitivity and specificity required to accurately identify malnutrition. It may be a better marker of wound severity and wound progress.
- ▶ Provision of supplemental nutrition that provides balanced amounts of protein, energy, vitamins, minerals and trace elements can help prevent pressure ulcers in acutely ill older people.

other groups is inconclusive. That is not to say that the benefits do not exist. It may be just difficult to demonstrate due to the inherent limitations of nutrition research and it may reflect the poor methodological quality and small sample sizes of the studies undertaken to date.

The clinical trials tend to be heterogeneous with regard to patients and interventions, with different primary outcomes being evaluated resulting in it being difficult to come to a balanced conclusion. Adequately powered trials with rigorous methodology are necessary.

Pressure ulcers result from a complex interaction of factors of which malnutrition is just one. Malnutrition is very unlikely to be the sole cause but can compromise tissue integrity. When combined with other risk factors as well

as pressure, shear and/or friction, it may lead to a pressure ulcer in a person who otherwise would not have developed one.

There is evidence that elderly people recovering from illness benefit from oral mixed nutritional supplements, but the evidence to support their use in other groups is inconclusive. That is not to say that the benefits do not exist.

It is worth highlighting that however small a contributing factor malnutrition may be, unlike so many other risk factors, it is potentially reversible. As pressure ulcers tend to develop within the first few days of admission to hospital, early nutritional screening is essential to allow for early intervention. This could be included in a pressure ulcer risk assessment tool. A reliable and validated tool such as the Malnutrition Universal Screening Tool (MUST) should be used (Elia, 2003). Appropriate staff should be trained in nutritional screening and nutritional management of patients identified as at risk of malnutrition. Very high-risk patients and those with complex nutritional needs should be referred to a registered dietitian for a comprehensive nutritional assessment and an individualised dietetic treatment plan. **WUK**

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