Heel damage and epidural analgesia: is there a connection?

• **Objective:** To determine heel pressure injury prevalence in surgical patients prescribed epidural analgesia and identify intrinsic and extrinsic factors associated with heel damage in this patient cohort.
• **Method:** A descriptive study was used to investigate intrinsic and extrinsic variables associated with recorded heel observations at a private hospital in Victoria, Australia. We recruited a sample of 29 consecutive non-emergency participants undergoing general anaesthesia for major surgery, who were prescribed epidural analgesia postoperatively. A total of 58 heel observations were made.
• **Results:** Heel damage prevalence in the study sample was 13.8% (n=8).
• **Conclusion:** Intrinsic factors associated with this complication included intra- and postoperative hypotension, vascular disease, smoking, chronic obstructive pulmonary disease, and multiple disease burdens. Extrinsic factors associated with heel damage included thromboembolic deterrent stockings and the postoperative ward use of heel protectors designed for use in operating theatres. It is hoped that the study findings will assist improvements in the assessment of heel damage risk and promote the pressure prevention strategies required by this patient cohort.
• **Conflict of interest:** Four pairs of each type of heel protector were donated by the manufacturing companies. One year after the study's conclusion, financial assistance was provided by Action Products Inc. and Edwards Medical to present the research findings at the AORN conference in Denver, Colorado.

pressure ulcers; intrinsic and extrinsic variables, heel protectors; epidural anaesthesia

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Heels are especially prone to pressure damage.[1] In the normal standing position, a thick mass of musculature over the medial calcaneal tuberosity, the dorsal aspect of the calcaneum, protects them from pressure. However, when lying supine on support surfaces, the pressure on patients' heels is concentrated over the calcaneal tuberosity, the posterior protrusion of the calcaneum.[2,3] This bony prominence, covered with fibrous tissue, has only a thin layer of subcutaneous tissue, which is poorly vascularised and without musculature.[4] It is clinically important to recognise that hypoperfusion from hypotension initially occurs in the subcutaneous tissue.[5]

Pressure damage can occur in as little as 30 minutes, yet in surgical patients signs may not appear for up to 24 hours.[6-8] The development of PUs at bone rather than skin may be a reason for this late response.[9] Historically, damage has been thought to occur with capillaries closing at interface pressures of 32mmHg.[10] However, in healthy individuals, interface pressures of 20mmHg are sufficient to close heel capillaries.[11] Surgery and/or disease burdens have the potential to increase the risk of heel damage at pressures <20mmHg.[11]

Epidural analgesia is used to minimise pain caused by complex surgical procedures. Two common adverse consequences of epidural analgesia relate to pressure injuries:
• Immobility (secondary to motor block)
• Hypotension secondary to autonomic blockade.[12,13]

The combination of sensory and motor deficits prevents awareness of damaged areas, and stops patients shifting their weight and relieving these unrecognised sources of pressure.[14] When compounded with hypotension, vascular perfusion is reduced, which affects the innate ability to endure pressure.[11] The density of motor block is measured according to the Bromage scale, which allows the assessment of motor deficits (Box 1).

The literature linking heel damage and epidural analgesia is scarce. Two reports have acknowledged an association between postoperative epidural analgesia and heel damage. Shah[15] identified three patients who underwent major gynaecological surgery for carcinoma. All received general anaesthesia and postoperative epidural analgesia. In addition, all experienced hypotension and motor deficits, followed by heel damage. Angel et al.[16] addressed the issue directly, linking pressure damage with motor and sensory deficits. Their case report described a patient undergoing leg amputation for peripheral arterial occlusive disease. Both during and after surgery, a continuous epidural infusion was prescribed, which was complicated by hypotension. A heel PU developed, which was successfully treated using a low air-loss mattress and wound management interventions. The authors proposed that hypoperfusion occurs when motor and sensory deficits present together with hypotension, and it was recommended that further research be undertaken to identify appropriate PU prevention strategies for this patient population.[16]

Box 1. Bromage scoring system.

Bromage score 0
Able to move fully (no motor block)
Bromage score 1
Able to move feet and knees only (slight motor block)
Bromage score 2
Able to only move feet (significant motor block)
Bromage score 3
Complete immobility of lower limbs

A PU audit undertaken at our research venue found that the prevalence of all PUs was 18.31% (n=13). However, the vast majority (14.08%, n=10) of ulcers were associated with postoperative epidural analgesia in surgical patients. The audit did not record the injury site, but anecdotal evidence identified that two surgical patients receiving epidural analgesia suffered heel damage severe enough to warrant surgical intervention. Standard care at the time of this audit included 2-hourly repositioning regimes, in-house risk assessment[17] and mandatory heel protection for patients receiving epidural analgesia. The latter comprised two types of heel protectors:

- Action heel cups made of Akton polymer (Action Products Inc.), colloquially referred to as 'heel pads'
- Repose Inflatable heel protectors (Frontier Medical Group), colloquially known as 'inflatable booties'.

The heel pad is a block of synthetic rubber with a scalloped area upon which to rest the heel. The pad redistributes point pressure impact on the calcaneal tuberosity across a larger surface area. Despite the manufacturer's recommendations that they be used in operating theatres, the heel pads were also available and used on all wards.

Inflatable booties are static devices. When inflated, air-filled chambers run lengthways from the toes to the upper calf, leaving a pressure-free area for heel suspension. These were available in limited supply on wards for all surgical patients.

One pair of these two types of heel protection device, depending on availability or their proximity, was routinely placed under both heels. Selection was indiscriminate because there is little evidence on their respective efficacy.[18,19]

Spurred by the audit findings and the need to prevent heel damage, we set out to:

- Investigate the prevalence of heel damage in surgical patients receiving epidural analgesia
- Investigate which intrinsic and extrinsic factors influence heal ulceration in the above cohort.

Method

Sample

A convenience sample was selected from consecutive elective adult patients undergoing major surgery, where postoperative epidural analgesia was often prescribed. Patients were recruited into the study at the pre-admission clinic. The inclusion and exclusion criteria are listed in Box 2. Surgical procedures undertaken at this facility following which postoperative epidural analgesia is often prescribed include orthopaedic, abdominal, renal and urological surgeries. All participants in the sample group belonged to these surgical groups. Premixed solutions of Naropin 0.2% and fentanyl 400µg were exclusively used for adult patients at the research venue. Therefore, only adults participated.

Box 2. Inclusion and exclusion criteria

Inclusion criteria
Participant must give informed non-binding consent
Participant must be over 18 years of age as only adults are prescribed epidural analgesia
Postoperative pain management was to comprise continuous epidural infusion
Elective admission
Exclusion criteria
Signed withdrawal of consent form
Previous participant of current trial (duplicate results)
Past history of lower limb amputation
Requires power of attorney to consent
Non-English speaking background (due to resource constraints)
Pre-existing heel ulcer

Ethics approval

Ethics approval was granted by the hospital's ethics committee (SJOGHCEC reference no. 248) and the university's ethics committee (reference no. FHEC06/124). Both the ethics committees and hospital management were kept informed of the study's progress and completion.

Recruitment and data collectors

All surgical nurses involved in recruitment and data collection received training on participant safety and how to fill in the data collection sheets. Data collection accuracy was evaluated by questionnaire, with all achieving 100%. To minimise bias, the researcher was not involved in either recruitment or data collection.

Procedure

Intraoperatively, all heels were protected using heel pads. On arrival at the post anaesthetic care unit (PACU), each participant's left heel was rested on a heel pad and the right heel on an inflatable bootie, maximising internal validity. Thromboembolic deterrent stockings (TEDS) worn by participants extended from toes to knees.

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Table 1. Heel number prevalence

Heel no.	Prevalence (%)	Total no.
0	74.1	43
1	12.0	7
2	3.45	2
3	10.34	6

Data collection and retrieval

Baseline data of heel observations, Bromage score, use of TEDS and risk assessment[17] were recorded on data collection sheets in the PACU. Heel observations were assigned a numerical value for documentation purposes:

- Heel number 0: no signs of tissue damage
- Heel number 1: blanching erythema, which was considered an indicator of potential damage, warranting prompt intervention to prevent progression to actual damage
- Heel number 2: non-blanching erythema, a valid indicator of PU formation that is reversible with early intervention.[20-22]
- Heel number 3: this comprised any safety concerns, based on the surgical nurses' clinical judgement. These concerns were described as pain or altered tissue texture without any visible signs of erythema.

These data were entered onto the same sheets in the surgical wards during the 2-hourly routine pressure prevention regimen. This process continued for 24 hours,[8] but was stopped earlier if required. Reasons for patient withdrawal before 24 hours included removal of the epidural infusion and compromised patient safety through potential or actual damage (i.e. heel observation numbers 1–3). In such circumstances, a pair of the more effective heel protectors for that individual was placed under both heels. It was stipulated that medical staff should be contacted if neither device proved effective, although this situation never arose.

To avoid bias, the researcher documented each patient's age, weight, past history and duration of surgery after the heel assessment. (We had intended to calculate body mass index but due to missing data had to record weight in kilograms instead.) In this way, both the data collectors and the researcher were blinded to any emerging trends.

Data analysis

All heel observation entries were included in the analysis. Given the small sample and descriptive nature of the study, data analysis was limited to frequency distribution, mean and standard deviation. Comparisons between intrinsic and extrinsic variables with heel numbers were conducted using odds ratios (OR). When the OR is greater than 1.0, the chance of the adverse event occurring is increased. Where the OR is less than 1.0, the chance of the adverse event occurring is reduced.[23] The sample size of this study was considered too small to analyse statistical significance.

Results

In all, 29 participants were recruited into the study, allowing 58 heel observations. The mean age was 71.9 years (± 20, range 54–94); 62% (n=18) were aged between 60 and 79 years. Thirteen were male and 16 female.

The 58 heel observations revealed a total prevalence of heel damage (heel observation numbers 2 and 3) of 13.8%. The prevalence of potential heel damage (heel observation number 1) was 12.0%. The percentage of damage-free heels (heel observation number 0) was 74.1%. Full results are given in Table 1.

Correlating heel damage with gender revealed that five of the damaged heels (62.5%) occurred in females, compared with three (37.5%) in males. Six cases of heel damage (75%) occurred in the 60–79 year age group.

Body weight ranged between 51kg and 125kg, although 19 patients (65.5%) weighed between 70kg and 89kg. Six patients (75%) with heel damage were in the 70–89kg weight range.

Two participants had no health complications and did not experience heel damage. The remaining 27 (93.1%) had co-existing health variables that are considered to be significant indicators of pressure ulceration.[21,24,25] Four of the cases of heel damage (50%) occurred in patients who had intra- and post-operative hypotension (Table 2).

Investigation of the heel damage associated with comorbidities was the only analysis that grouped potential damage and actual damage together (heel numbers 1–3). Allowing participants who had comorbidities and potential heel damage to remain in the study increased their risk of developing actual damage. This potential and actual heel damage was grouped together for analysis by OR for distinct and clustered comorbidities.

The OR of developing potential or actual heel damage associated with vascular disease was 4.0. All of the other health variables analysed distinctly yielded an OR of 1.0 or less: the OR for type 2 diabetes mellitus was 0.5, that for renal disease was 1.0, and for cardiac disease was 1.0. When analysed in clusters, participants with three or more coexisting health conditions yielded an OR of 1.4. Only participants who had smoked for decades (n=4) with or without a history of chronic obstructive pulmonary disease (COPD, n=3) developed specific heel damage with pain or altered tissue texture and without any visible sign of erythema (heel number 3) (Table 3).

Correlation between heel damage and Bromage score found that five of the patients with heel damage (62.5%) did not have any motor deficits (a Bromage score of 0). Only two heels (25%) correspond-

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ed to a Bromage score of 2, and one (12.5%) had a Bromage score of 3.

Correlation between heel damage and postoperative use of TEDS found that seven cases of damage (87.5%) were associated with wearing TEDS continuously. Only one damaged heel (12.5%) was associated with TEDS applied 6 hours postoperatively. No heel damage was found when TEDS were absent.

The duration of surgical procedures ranged from 2 to 5 hours. Four damaged heels (50%) were associated with 2 hours in surgery. Two (25%) were linked with a surgical time of 3 hours, and two others (25%) with 5 hours in surgery.

Heel damage was only found in heels that rested on heel pads postoperatively (i.e. were not used in accordance with the manufacturer's instructions). However, all heels that were protected intraoperatively with heel pads and then postoperatively with inflatable booties remained damage free.

It was originally intended to investigate the correlation between heel damage and the in-house risk assessment tool.[17] However, the data available for analysis was found to be either incorrect or incomplete, which rendered any findings useless.

Discussion

This study found heel damage prevalence to be 13.8% (n=8). If we had not withdrawn patients with blanching erythema (12.0%, n=7) from the study, then the prevalence would have been higher still: 25.8% (n=15).

Given the mean age of participants and high prevalence of comorbidities, it can be argued that our sample is a fair representation of an ageing surgical populations with multiple disease burdens, undergoing major surgery.[21,26]

Vascular disease was strongly associated with potential or actual heel damage (OR=4.0). This contrasts with the large study by Gilcreast et al.,[25] which reported an OR of 2.1 for the occurrence of pressure ulceration in patients with vascular disease. Differences between these findings might be attributable to sampling variance and our decision to cluster medical variables together and to consider potential and actual damage together, aiming to improve participant safety.

Moreover, during episodic hypotension, co-existing disease burdens increase the threat to heel perfusion.[11] The clusters of health variables analysed demonstrated the impact of participants with potential or actual heel damage suffering three or more comorbidities (OR=1.4). Little research is available to compare with the current findings. Nonetheless, this study identified that multiple disease burdens contributed to heel damage in this patient cohort. Further research is needed is need on this.

Surprisingly, the smokers (who had all smoked for decades) failed to show any erythematous heel changes, yet all demonstrated the symptoms of pain or altered tissue texture. Noble noted that there were far fewer signs of erythema in response to pressure in young subjects who had recently started smoking compared with their non-smoking peers.[27] The current study's small sample size prevents us from drawing any conclusions, although may provide the rationale for further studies in larger populations.

Hypotension impacts heel perfusion as the thin layer of poorly vascularised, subcutaneous tissue that separates the skin and calcaneal tuberosity is affected first,[4,5,11] and intraoperative hypotension is considered a precursor to tissue damage.[16] Epidural analgesia is acknowledged to produce episodic hypotension.[12] Fifty per cent (n=4) of patients with heel damage suffered hypotension. This is a preliminary indication that the risk of heel damage is increased in patients given epidural analgesia who experience hypotension.

Surprisingly, 62.5% (n=5) of participants with actual heel damage had no motor deficit. Patients prescribed postoperative epidural analgesia without any motor deficit remain at risk of heel damage. While postoperative patients may be physically capable of shifting heel pressure burdens, it cannot

Table 2. Heel numbers and medical history

Medical history	Heel 0	Heel 1	Heel 2	Heel 3
P/I hypotension	9	3	1	3
Hypertension	6	3	0	3
Cancer	6	3	0	3
Smoker	0	0	0	4
Varicose veins	0	3	0	1
COPD	0	0	0	3
Depression	0	0	2	1
AMI	2	1	1	0
IHD	1	0	1	1
T 2DM	2	2	0	0
CRF	1	2	0	0
PVD	0	3	0	0
CCF	1	2	0	0
DVT	1	0	0	2

P/I hypotension = postoperative and/or intra-operative hypotension; COPD = chronic obstructive pulmonary disease; AMI = acute myocardial infaction; IHD = ischaemic heart disease; T2DM = Type 2 diabetes mellitus; CRF = chronic renal failure; PVD = peripheral vascular disease; CCF = congestive cardiac failure; DVT = deep vein thrombosis

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Table 3. Odds ratio of health variables, both distinct and in clusters

Health variable	Heel stage 1–3	Heel stage 0	Odds ratio
Cardiac	8	8	1.0
Vascular	4	1	4.0
Depression	3	0	N/A
Cardiac and vascular	4	0	N/A
Diabetes	1	2	0.5
Diabetes and vascular	1	0	N/A
Diabetes and cardiac	1	2	0.5
Renal disease	1	1	1.0
Renal and vascular	1	0	N/A
Renal and cardiac	1	1	1.0
Smoking ± COPD	4	0	N/A
Three or more health variables	7	5	1.4

COPD = chronic obstructive pulmonary disease; N/A = not able to attend

Health variables:
Cardiac: congestive cardiac failure, ischaemic heart disease, acute myocardial infarction; vascular: peripheral vascular disease, varicose veins, deep vein thrombosis; renal: chronic renal failure; diabetes: type 2 diabetes mellitus

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be assumed that they will do so. Residual sedative effects of anaesthetic agents and opioid medication within the epidural infusions negatively affect self-initiated movement.[28,29] This study indicates that patients receiving epidural analgesia, regardless of Bromage score, are at risk of heel damage.

The use of TEDS postoperatively aims to minimise thrombosis, but it is recommended that they be replaced every 24 hours.[30,31] We found that their continuous use over 24 hours was detrimental to heels. Their efficacy relies on the application of 18mmHg ankle pressure,[32] although external pressures of 20mmHg[11] have been associated with localised heel ischaemia in healthy young study participants. Furthermore, patients undergoing surgical procedures, especially in the presence of hypotension and comorbidities may be vulnerable to even lower heel pressures.[11] TEDS must, therefore, be used with caution. The desired outcome of epidural analgesia is to reduce the sensation of pain; however, reduced sensation increases PU risk by 34 times.[28]

The current study found 50% (n=4) of heel damage was associated with a surgery duration of as little as 2 hours. Schoonhoven et al.[33] previously found operating room times of 5 hours or more were a precursor for pressure injuries (p=0.02). Participant numbers and sampling methods are probable influences to which these differences may be attributed. Further research is required to explore heel damage in postoperative patients receiving epidural analgesia and intraoperative times.

The hospital policy of the research venue mandates heel protectors for patients receiving postoperative epidural analgesia. Which product is chosen depends on the nurse and the product's availability. The researcher found no other studies investigating the efficacy of heel protectors from operating rooms on postoperative wards, and none specifically relating to epidural analgesia. This study's finding that heels remained damage free when protected intraoperatively by heel pads and postoperatively by inflatable booties is reasonable, given that heel pads are designed for use in operating theatres and inflatable booties are designed for ward use.

Limitations

- The major limitation is the small sample size and as such, the data must be interpreted with caution. Studies using larger numbers of participants may differ in findings
- The use of weight rather than BMI may have limited the study's ability to interpret data adequately
- Although heel damage was defined by a valid tool, potential and actual damage were grouped together in the analysis of existing comorbidities
- The inability to investigate in-house risk assessment for heel damage prevented further understanding of the risks associated with injuries.

Conclusion

Despite its small size, this study found that heels were vulnerable to damage in postoperative patients prescribed epidural analgesia. Heel damage prevalence was 13.8% (n=8), with all damage reversed by prompt intervention.

The prevalence rate might have been higher if patients with blanching erythema had been included in the study.

Data analysis confirmed vascular disease and multiple disease burdens were strongly associated with heel damage in these patients. Only participants who smoked and suffered COPD developed heel damage without visible colour changes.

These findings provide a preliminary indication that postoperative patients with pre-existing vascular disease, lung disease and multiple comorbidities given epidural analgesia might be at high risk of heel ulceration.

Finally, COPD, smoking and the continuous use of TEDS for more than 24 hours also markedly increased the risk of heel damage. While a further, larger study is required, the results of this evaluation will hopefully provide insight for clinicians and positive outcomes for this patient cohort. ■