

# Postoperative pressure ulcers in vascular patients after epidural analgesia: case reports

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## Abstract

Epidural analgesia is widely recognised as one form of postoperative pain relief. However, several authors have expressed concerns that there is an increased incidence of pressure ulcer development in patients with epidural analgesia. Patients with peripheral arterial occlusive diseases (PAODs) are already at risk of pressure ulcer development due to reduced peripheral blood supply, and the use of epidural analgesia will increase their risk unless appropriate preventative measures are taken at all levels of care. This case series demonstrates the potential complications associated with epidural analgesia in three postoperative patients with PAODs. Recommendations are made to try and reduce these complications. In addition, as there is limited low level evidence linking the development of pressure ulcers and epidural analgesia, further well-conducted research is needed.

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## Introduction

Pressure ulcers are defined as areas of localised damage to the skin and underlying tissue caused by pressure, shear and friction<sup>1</sup>. According to the National Pressure Ulcer Advisory Panel (NPUAP), pressure ulcers are classified into four stages<sup>2</sup>.

A review of the literature suggests that the prevalence of pressure ulcers range from 4-15.4%<sup>3-6</sup>. Pressure ulcers are one of the most common causes of morbidity and prolonged hospital stay. Studies have shown that the financial impact of treating pressure ulcers ranges from \$2.2-\$3.6 billion in the USA. This has been estimated to be in the excess of \$350 million in Australia<sup>4</sup>.

Epidural anaesthesia and analgesia provide a better outcome for postoperative pain relief and have become common practice after abdominal and other major surgical procedures<sup>8, 9</sup>. Several authors, however, report that side effects such as motor block and hypotension have contributed to the development of pressure ulcers<sup>7-12</sup>. The body's own defence mechanism against the development of a pressure ulcer is to change position regularly. Loss of this protective sensation induced by epidural analgesia and subsequent motor blockade may be sufficient to remove patients' reflexes to reposition<sup>7</sup>.

Reports of pressure ulcer development after epidural anaesthesia have described the presence of either motor block or hypotension during the intraoperative or postoperative period<sup>8-10, 13</sup>. Most of these reported cases were young patients who received epidural analgesia following obstetric or gynaecologic procedures. However, there may be unreported

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cases, in particular the elderly and patients with peripheral arterial occlusive disease (PAOD), who have had epidural anaesthesia and/or analgesia. This patient group must have preventative strategies implemented, such as regular turning regimes, pressure-relieving devices and, where possible, a reduction of risk factors<sup>14, 15</sup>.

The aim of this comprehensive preventative management plan is to maintain tissue tolerance to pressure and to protect the individual against the forces of pressure shear and friction<sup>16</sup>. There is, however, no literature, either research based or anecdotal, to substantiate whether the prevalence among the postoperative vascular patients is higher than other surgical patients after the use of epidural analgesia. This report describes three vascular cases that developed postoperative pressure ulcers following the use of epidural analgesia.

### Case one

A 58 year old male presented to accident and emergency with a critically ischaemic right leg. He had a past medical history of PAOD, ischaemic heart disease with previous coronary angioplasty, right internal iliac artery and right common femoral artery angioplasty and stent. He presented with 10 days' history of claudication at 20 metres and increasing rest pain in his right leg. On examination, he had absent femoral, popliteal and distal pulses on the right side, with reduced sensation and delayed capillary return. An angiogram demonstrated complete occlusion of the right iliac arteries.

A left to right femoro-femoral cross over bypass was performed which took 2.5 hours. An epidural catheter was inserted; Ropivacaine 2mg/ml with Fentanyl 2mcg/ml at 8mls per hour was started and remained *insitu* for a total of 17 hours. He had sensory level at T10 but no documentation of a motor block was made. After the epidural infusion was ceased the next morning, the patient complained of pain of the right buttock. Examination of the area demonstrated a Stage I pressure ulcer (Figure 1).

An ultrasound excluded an abscess or haematoma. The area was not considered to be a diathermia burn. The area was marked and the patient was placed on an low air loss overlay mattress in order to prevent any further tissue damage. Six days later the patient was discharged home without any further complications. On follow-ups 6 weeks later, the area had healed completely.

### Case two

A 64 year old man underwent right below knee amputation for a non-healing diabetic foot ulcer. Epidural anaesthesia and subsequent analgesia were used, consisting of Ropivacaine 0.2%/fentanyl 400mcg in premix bag; this remained *insitu* for a period of 4 days. At this point the patient was not identified as having any pressure ulcers. An angiogram of his left leg demonstrated a 10cm occlusion of the superficial femoral artery (SFA) and his ankle brachial index (ABI) was 0.63 for posterior tibial and 0.48 for dorsalis pedis.

Sixteen days after his initial operation, a left below knee femoro-popliteal bypass was performed. The length of the procedure was 4 hours and epidural analgesia was commenced using Ropivacaine 0.2% with Fentanyl 400mcg in a premix bag, at a rate of 8mls per hour. Following an assessment on return to the ward, the epidural infusion was withheld for 45 minutes due to hypotension and motor block, and restarted at the rate of 6mls/hour. Thirteen hours after the operation, the epidural infusion was ceased due to persistent motor blockade.

Assessment of his pressure areas the next day in view of the prolonged motor blockade revealed a Stage II pressure ulcer on his sacrum. This was not identified as being present prior to surgery. The pressure ulcer progressed to Stage IV over the next 5 days despite appropriate dressings and standard pressure area care, including the use of a low air loss overlay mattress. The necrotic area was debrided (Figure 2) and a vacuum assisted closure (VAC)<sup>TM</sup> dressing was applied; this was complicated by episodes of bleeding and therefore ceased. The use of an alternative dressing was applied in its place. The patient was transferred to a peripheral hospital for rehabilitation and, at 3-month follow-up, the ulcer had almost completely healed.

### Case three

A 50 year old male patient presented to accident and emergency with an ischaemic left leg and extensive tissue loss. He had no co-morbidities. On examination, he had a femoral pulse, but absent popliteal and pedal pulses. ABIs were not detected. Angiography demonstrated patent iliac, femoral and popliteal arteries. All the three run off vessels were occluded in the mid-calf. The patient was offered an amputation, which he initially declined. Ten days later he agreed to the procedure and the operation was performed. The length of the procedure was one hour.

An epidural was inserted pre-operatively, Ropivacaine 2mcg/ml with Fentanyl 2mcg/ml at 8mls per hour; this remained *insitu* for 3 days at the same rate. He was hypotensive 95/55-88/50 whilst the infusion was in progress there is no documentation to substantiate the presence of a motor block. On the third postoperative day, a Stage II (blister) pressure ulcer was identified on the patient's left heel. This was not present prior to surgery. The patient was placed on a low air loss overlay air mattress; the blister was left intact and protective dressing (Allevyn heel<sup>TM</sup>) applied.

The patient made an uneventful recovery and was transferred to a rehabilitation hospital. One month later at outpatient follow up, the pressure ulcer was resolving with superficial tissue loss (Figure 3).

## Discussion

Pressure ulcers are among the most common causes of significant morbidity and put a considerable pressure on health services' budgets. Despite increased awareness of pressure ulcers and improved wound management, there have not been significant changes in the incidence and prevalence over the last 2 decades<sup>3, 4, 6</sup>.

Most authors believe that there is a relationship between the use of postoperative epidural analgesia and development of pressure ulcers<sup>8, 10, 13</sup>. However, the exact cause-effect relationship between epidural analgesia and pressure ulcer development is yet to be determined. It is generally well understood that patients with PAOD are prone to the development of pressure ulcers because of poor circulation. This problem can be made worse by the use of epidural anaesthesia due to complications such as hypotension and motor block. There is, however, insufficient published evidence to substantiate this and further well designed studies are recommended.

Motor block and hypotension were identified in two of our three cases. This is consistent with other reports<sup>8-11</sup>. Nursing and medical staff dealing with postoperative patients should be familiar with pressure ulcer prevention protocols. Individuals identified as being at risk for pressure ulcer development on admission or thereafter should have preventative management plans instigated to prevent the development of a pressure ulcer.

In order to significantly reduce the incidence and prevalence of pressure ulcers, it is important to adhere to the skin care needs of patients at risk. There is sufficient evidence to directly link surgery and sensory loss with the development of pressure ulcers. In both instances, the individual is unable to respond to the stimulus of prolonged, intense, localised pressure, which is the body's own defence mechanism against pressure ulcer development<sup>16</sup>.

All patients were placed on low air-loss overlay mattresses after developing pressure ulcers. According to a recent report by the Cochrane Collaboration, the use of low air-loss mattresses appear to be effective in preventing and treating pressure ulcers compared to foam mattresses<sup>15</sup>. However, the use of pressure relieving support surfaces must not negate good clinical practice, including regular turning regimens and skin inspection. As demonstrated in case two of our reports, the patient was initially identified as having a Stage II pressure ulcer which, despite the use of a pressure relieving mattress, rapidly progressed to a Stage IV requiring surgical intervention.

Patients with PAOD are at a higher risk of developing a pressure ulcer than non-vascular patients due to reduced peripheral blood supply. This patient group must have preventative strategies implemented that aim to improve the tissue's tolerance to pressure<sup>16</sup>.



Figure 1



Figure 2



Figure 3

## Conclusion

These case studies illustrate the need for specific protocols and guidelines to be developed in order to prevent or minimise the risk of pressure ulcer development associated with epidural therapy. Preventative measures need to be applied before the development of pressure ulcers. Epidural protocols should include pressure area assessment and a specific plan of action in the presence of hypotension or motor block. There is insufficient research on the mechanisms of pressure ulcer development in vascular patents after the use of epidural anaesthesia and further research is recommended to address this problem – prevention is better than cure.

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