Does a single dose of intravenous dexamethasone reduce Symptoms in Emergency department patients with low Back pain and RA diculopathy (SEBRA)?

A double-blind randomised controlled trial

Ravichandra Balakrishnamoorthy,1,2 Isabelle Horgan,1,3 Siegfried Perez,4 Michael Craig Steele,5,6 Gerben B Keijzers4,7,8

ABSTRACT

Objective To assess the effect of a single dose of intravenous dexamethasone in addition to routine treatment on visual analogue scale (VAS) pain scores at 24 h in emergency department (ED) patients with low back pain with radiculopathy (LBPR).

Methods Double-blind randomised controlled trial of 58 adult ED patients with LBPR, conducted in one tertiary and one urban ED. The intervention was 8 mg of intravenous dexamethasone (or placebo) in addition to current routine care. The primary outcome was the change in VAS pain scores between presentation and 24 h. Secondary outcomes included VAS pain scores at 6 weeks, ED length of stay (EDLOS), straight leg raise (SLR) angles and Oswestry functional scores.

Results Patients treated with dexamethasone had a 1.86 point (95% CI 0.31 to 3.42, p=0.019) greater reduction in VAS pain scores at 24 h than placebo (dexamethasone: −2.63 (95% CI −3.63 to −1.63) versus placebo: −0.77 (95% CI −2.04 to 0.51)). At 6 weeks, both groups had similar significant and sustained decrease in VAS scores compared with baseline. Patients receiving dexamethasone had a significantly shorter EDLOS (median: 3.5 h vs 18.8 h, p=0.049) and improved SLR angle at discharge (14.7°, p=0.040). There was no difference in functional scores.

Conclusions In patients with LBPR, a single dose of intravenous dexamethasone in addition to routine management improved VAS pain scores at 24 h, but this effect was not statistically significant at 6 weeks. Dexamethasone may reduce EDLOS and can be considered as a safe adjunct to standard treatment.

Trial registration number ACTRN12611001020976.

INTRODUCTION

Each year, 15%–45% of adults suffer low back pain, and 1 in 20 people present to a healthcare professional with a new episode.2 Based on the 2004–2005 National Health Survey conducted in Australia, 15% of the population reported having back problems, leading to a total expenditure of A$567 million (US$∼630 million) in the treatment of back pain.3 In the USA, back pain is the fifth most common reason for all physician visits.4 In 2006, total healthcare costs associated with low back pain in the USA exceeded US$100 billion per year.5 Some causes of acute and chronic low back pain, such as lumbar stenosis or disc herniation causing nerve root compression, can be associated with radiculopathy.6 For these patients, the current mainstay of emergency department (ED) treatment includes appropriate analgesia and physiotherapy when available.

Although some studies have assessed systemic corticosteroid treatments in back pain, they were performed in settings outside the ED,6 used tapered dosing for at least 7 days7 or included patients without radiculopathy.8 Hence, there is little evidence that single-dose corticosteroids provide additional symptom relief for ED patients with low back pain with radiculopathy (LBPR).

Back pain meets the broad criteria for National Health Priority Area in Australia, in which outcomes can be improved with an effective treatment strategy.9 Among patients treated for back pain in the emergency setting, almost half still experienced symptoms at 3 months postdischarge,10 with another cohort study suggesting that nearly one-third of individuals did not achieve a full recovery at 12 months.11

In 2010, back pain with radiculopathy accounted for approximately 1% of ED diagnoses in the health district where this study took place. Pain leading to the inability to mobilise appears to be the largest factor preventing discharge after initial management. This poses both a medical and logistical challenge. Thus, improving the management of low back pain has the potential to benefit both patients and the healthcare system. Despite the lack of high-quality evidence, it is not unusual for clinicians to prescribe single-dose parenteral steroids for patients with LBPR, highlighting the need for further data to bridge this evidence-practice gap.

Key messages

What this study adds
In emergency department (ED) patients with low back and radiating features, a single dose of 8 mg intravenous dexamethasone improved pain scores at 24 h more so than placebo. This study also suggests it may reduce ED length of stay.

What is already known on this subject
Some studies have assessed systemic corticosteroid treatments in back pain, but these were performed in settings outside the ED, used tapered oral dosing or included patients without radiculopathy.

To cite:
We aimed to assess the effect of a single dose of 8 mg intravenous dexamethasone compared with placebo in addition to standard management (analgesia and physiotherapy referral) in ED patients with LBPR.

We hypothesised that this intervention (compared with placebo) would lead to a greater reduction in the primary outcome: visual analogue scale (VAS) pain scores at 24 h. We also hypothesised that the intervention would improve secondary outcomes including pain scores at 6 weeks, functional scores at 24 h and 6 weeks, ED length of stay (EDLOS) and straight leg raise (SLR) angle.

**METHODS**

**Study design**

This was a double-blind randomised controlled trial approved by the Health District’s Human Research and Ethics Committee and registered prior commencement with the Australia and New Zealand Clinical Trials Register (no.: 12611001020976) without any protocol changes. We adhered to the CONSORT statement (http://www.consort-statement.org).

**Setting**

The trial was conducted between November 2011 and November 2012 in the EDs of the two public hospitals within the same Health District in Southeast Queensland, Australia. The main campus is a 570-bed major metropolitan teaching hospital and the second campus (located 12 km from the main campus) is an urban district hospital with 200 beds. The ED census in 2012 was 67 000 and 50 000, respectively.

**Selection of participants**

Patients who presented with low back pain and leg radiation at triage were identified as potential participants in this trial. Further inclusion criteria were age between 18 and 55 years, a positive SLR test and difficulty mobilising. Exclusion criteria were likely alternative diagnosis or ‘red flags’ (fever, recent trauma, history of malignancy), pregnancy, known allergy to dexamethasone, current use of glucocorticoids, history of lower back surgery and inability to provide consent. Eligible patients were recruited by the triage nurse, treating staff or research nurse according to these inclusion and exclusion criteria. Written informed consent from eligible participants was obtained before randomisation.

**Measurements: straight leg raise test**

An inclusion criterion was to have a positive SLR test, as assessed by either a doctor or a physiotherapist. An SLR test was positive if pain was reproduced by passive movement of the hip joint between 0° and 70° as measured with a goniometer. The SLR test is highly sensitive for detecting LBPR and assessing the effect of treatment. The average angle of passive hip flexion from three attempts was calculated and documented at initial presentation and again upon discharge.

**Interventions**

After being consented, eligible participants were randomised to either treatment (8 mg intravenous dexamethasone in 2 mL) or control (2 mL intravenous 0.9% sodium chloride) group. Syringes for both treatment and control were identical except for unique sequence numbers. The allocated treatment was in addition to current routine care for LBPR. Both groups received a standardised regimen of regular analgesia, physiotherapy referral and education. Standardised analgesia included regular oral acetaminophen/codeine, ibuprofen and oral oxycodone as required.

**Randomisation and allocation concealment**

The randomisation was overseen by an independent statistician using computer-generated block randomisation with blocks of 10 for each hospital. Allocation concealment was maintained using individual sequentially numbered opaque sealed envelopes. These envelopes were opened after the patient had provided written consent.

A study nurse (not involved with recruitment, consenting or the participant’s care) opened the next available sealed envelope (containing the patient allocation) and would ask the treating nurse to administer the allocated treatment from the syringe matching the sequence number in the envelope. Nurses and doctors involved with patient care and data collection, participants and the analysing statistician were blinded to the group allocation.

**Outcome measures and data collection**

The primary outcome measure was the change in level of pain, documented using a VAS. A 10 cm line ranging from ‘no pain’ to ‘worst pain ever’ was presented. Patients were instructed to mark an ‘X’ on any point of the line that best described their current level of pain. The VAS is a validated, reliable and easy means of measuring pain in the acute medicine setting with a change of between 1.3 and 2.0 points generally accepted as the minimum clinically meaningful effect. We did not pre-specify patient position for VAS measurement. The VAS pain score was collected at four distinct points: on presentation, on discharge, 24 h after randomisation and 6 weeks postinitial presentation to ED.

Secondary outcome measures included (1) EDLOS (defined as triage time to time of discharge), (2) time to return to normal activities (in days), (3) SLR range of motion (degrees) as described above, (4) analgesia requirements and proportion of adverse effects and (5) the Oswestry Disability Index (ODI). This index is a validated outcome measure in assessing a patient’s level of disability status as a result of spinal disorders. The questionnaire consists of 10 multiple choice questions worth up to 5 marks each, giving a scoring range of 0–50, with a lower score representing a lower degree of disability. The score is then used to calculate (in percentage) the level of a patient’s disability. Participants’ scores were collected at three times: on presentation, at 24 h and 6 weeks.

Data sheets were deposited in a locked box. A single research nurse collected these and was responsible for data entry.

**Follow-up procedure**

The treating staff (medical and physiotherapy) discharged patients from ED based on their clinical judgement, with ability to mobilise safely a compulsory component. Participants were given follow-up questionnaires (including VAS and ODI) and instructions to self-complete and return them by reply-paid envelopes at 24 h and 6 weeks.

**Primary data analysis**

A power calculation was performed based on the assumption that in the placebo group (routine management only) the VAS pain score would improve by 1.5 points. This was based on local audit data of all patients in 2010 with LBPR, where VAS scores decreased from 7.5 to 6 with routine management. We hypothesised that the addition of dexamethasone would decrease the VAS pain score by a further 1.5 points (7.5 to 4.5),
which we deemed clinically significant. All VAS scores were assumed to have an SD of 2 points. We calculated a required number of 44 patients (22 per group) to have 80% power to detect this difference ($\alpha=0.05$, two-sided). To account for loss to follow-up and to have complete follow-up for the secondary endpoint of VAS at 6 weeks, we aimed to enrol 70–100 patients. Analysis was by intention to treat. No interim analyses were performed.

Data taken from completed questionnaires were collated using Microsoft Excel spreadsheet software and then coded prior to transfer to the Statistical Package for the Social Sciences (V17.0, SPSS, Chicago, Illinois, USA) for statistical analysis. Before analysis, all variables were reviewed for accuracy of data entry, missing values and outliers using SPSS. No modifications were made for missing data. For continuous variables, we used an independent t test or Mann–Whitney U test (if variables were not normally distributed) to compare treatment groups. We conducted a regression analysis as a secondary analysis to account for baseline imbalance, with change in VAS as dependent variable and baseline VAS, amount of oxycodone and intervention (placebo/dexamethasone) as independent variables. For categorical variables, the $\chi^2$ test was used. An $\alpha$ of 0.05 was statistically significant.

**RESULTS**

Fifty-eight patients were randomised and had a mean age of 38 years, with even distribution of gender. [Figure 1](#) shows patient eligibility, allocation and follow-up. Of 69 patients who met eligibility criteria, 11 did not consent to randomisation. Of the remaining 58 patients, the primary outcome was available for 48 patients. Table 1 summarises the patient baseline characteristics.

Primary outcome analysis showed that, at 24 h, patients treated with a single dose of intravenous dexamethasone had a 1.86 point (95% CI 0.3 to 3.4, $p=0.02$) greater reduction in pain scores compared with patients receiving placebo (see [Table 2](#) and [Figure 2](#)). The dexamethasone group improved from 8.08 to 5.45 on the VAS (2.63 point reduction, 95% CI 1.6 to 3.6, $p<0.001$) compared with a decrease from 7.02 to 6.25 (0.77 point reduction, 95% CI $-0.5$ to $2.0$, $p=0.224$). Both groups showed a similar significant reduction in VAS pain score between presentation and discharge ($-3.00$ and $-2.32$ points). At 6 weeks, both groups had significant and sustained decrease in VAS scores compared with baseline (dexamethasone: $-4.28$ (95% CI $-6.02$ to $-2.54$), $p<0.001$ vs placebo: $-2.83$ (95% CI $-4.37$ to $-1.28$), $p<0.001$), but the reduction in the

![Flow diagram](#)

*Figure 1  Flow diagram. ED, emergency department; IV, intravenous; SLR, straight leg raise test.*
dexamethasone group was not significantly greater (difference of the mean VAS at 6 weeks: 1.45 points (95% CI 0.83 to 3.74, p=0.204)).

Using regression analysis, controlling for baseline VAS reduced the estimated treatment effect at 24 h by 28% from 1.86 to 1.33 (95% CI −0.2 to 2.8, p=0.079) and controlling for oxycodone use reduced the estimated treatment effect at 24 h by 6%, from 1.86 to 1.75 (95% CI 0.2 to 3.3, p=0.031). Controlling for baseline VAS reduced the estimated treatment effect at 6 weeks by 48%, from 1.45 to 0.76 (95% CI −1.3 to 2.8, p=0.46). Controlling for the amount of oxycodone reduced the estimated treatment effect at 6 weeks by 11%, from 1.45 to 1.29 (95% CI −1.0 to 3.6, p=0.27).

Secondary outcome analysis demonstrated that patients in the dexamethasone group had significantly shorter EDLOS (median 3.5 vs 18.8 h, p=0.049) and on average 14.7 (95% CI 1.3 to 34.3, p=0.040) degrees greater improvement in SLR test from presentation compared with discharge (improvement in dexamethasone group 20.2°, p=0.008 vs 5.5° in placebo, p=0.151; see table 3).

There was no significant difference in improvement in Oswestry scores between dexamethasone and placebo at either 24 h (0.5% improvement, p=0.52 vs 4.6% improvement, p=0.63) or 6 weeks (23.8% improvement (95% CI 12 to 37, p<0.01) vs 21.9% improvement (95% CI 5 to 31, p<0.01)), respectively. There was also no statistical difference between groups in ability to return to their normal activities by 6 weeks (75% vs 60%, p=0.30) or analgesia requirements (table 3).

### Table 1 Baseline demographics

<table>
<thead>
<tr>
<th></th>
<th>Dexamethasone, n=29</th>
<th>Placebo, n=29</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females, n (%)</td>
<td>17 (58.6%)</td>
<td>13 (44.8%)</td>
<td>0.29</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>38.9 (9.1)</td>
<td>36.9 (9.9)</td>
<td>0.43</td>
</tr>
<tr>
<td>VAS pain score at presentation, mean (95% CI)</td>
<td>8.11 (7.4 to 8.8)</td>
<td>7.02 (6.2 to 7.8)</td>
<td>0.034</td>
</tr>
<tr>
<td>Oswestry score at presentation, % score (95% CI)</td>
<td>62.3 (54.9 to 69.7)</td>
<td>63.3 (55.1 to 71.6)</td>
<td>0.85</td>
</tr>
<tr>
<td>SLR angle (range of motion—hip joint), degrees (95% CI)</td>
<td>29.0 (22.3 to 35.7)</td>
<td>37.2 (30.9 to 43.5)</td>
<td>0.073</td>
</tr>
</tbody>
</table>

SLR, straight leg raise; VAS, visual analogue scale.

### Table 2 Pain visual analogue scores

<table>
<thead>
<tr>
<th></th>
<th>Dexamethasone</th>
<th>Placebo</th>
<th>Mean difference (placebo minus dexamethasone) (95% CI)</th>
<th>p Value of the difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>VAS at presentation, mean (95% CI), n=29</td>
<td>8.11 (7.4 to 8.79)</td>
<td>7.02 (6.22 to 7.82)</td>
<td>−1.10 (−2.11 to −0.08)</td>
<td>0.034</td>
</tr>
<tr>
<td>VAS at discharge, mean (95% CI), n=29</td>
<td>5.14 (4.00 to 6.28)</td>
<td>4.76 (3.56 to 5.96)</td>
<td>−0.38 (−1.98 to 2.23)</td>
<td>0.64</td>
</tr>
<tr>
<td>VAS at 24 h, mean (95% CI), n=26; 22</td>
<td>5.45 (4.29 to 6.61)</td>
<td>6.23 (5.21 to 7.25)</td>
<td>0.78 (0.74 to 2.30)</td>
<td>0.31</td>
</tr>
<tr>
<td>VAS at 6 weeks, mean (95% CI), n=20; 16</td>
<td>3.80 (2.27 to 5.33)</td>
<td>4.32 (3.03 to 5.61)</td>
<td>0.52 (−1.43 to 2.48)</td>
<td>0.59</td>
</tr>
<tr>
<td>Change in VAS between presentation and discharge, difference of the mean (95% CI), n=29</td>
<td>−3.00 (−4.33 to −1.66)</td>
<td>−2.32 (−3.43 to −1.21)</td>
<td>0.68 (−1.01 to 2.36)</td>
<td>0.42</td>
</tr>
<tr>
<td>Change in VAS between presentation and 24 h, difference of the mean (95% CI), n=25; 21</td>
<td>−2.63 (−3.63 to −1.63)</td>
<td>−0.77 (−2.04 to 0.51)</td>
<td>1.86 (0.31 to 3.42)</td>
<td>0.019</td>
</tr>
<tr>
<td>Change in VAS between presentation and 6 weeks, difference of the mean (95% CI), n=19; 16</td>
<td>−4.28 (−6.02 to −2.54)</td>
<td>−2.83 (−4.37 to −1.28)</td>
<td>1.45 (−0.83 to 3.74)</td>
<td>0.20</td>
</tr>
</tbody>
</table>

p Values are based on independent t test. VAS, visual analogue scale.

Incidence of adverse effect reporting between groups was similar (18% vs 15%). One patient receiving dexamethasone reported a dexamethasone-specific adverse event directly after the intravenous bolus; peri-anal itching. All the adverse effects were mild (eg, nausea, mild headache, light-headedness), transient and none required treatment.

### DISCUSSION

We found that adding a single dose of 8 mg intravenous dexamethasone to current routine care significantly reduced pain at 24 h compared with placebo in ED patients with low back pain and radiating features. Pain scores at 6 weeks were further decreased in both groups, but the effect of dexamethasone over placebo was not sustained. SLR test (hip joint range of motion) at discharge improved significantly when dexamethasone was added. Dexamethasone appeared to contribute to early discharge and shorter median length of ED stay by around 15 h. These improvements may have a significant impact on patients as well as staff and the health system due to shorter stays allowing for greater availability of resources.

The rationale of systemic (intravenous, intramuscular injection (IMI) or oral) glucocorticoid use is its anti-inflammatory and oedema-reducing effect around the nerve root, resulting in pain reduction and hip mobility improvement. Using this rationale, the largest effect of glucocorticoids would be expected in patients with acute and severe pain where a discogenic origin is suspected. Only one other study investigated the use of intravenous steroids (500 mg methylprednisolone) for back pain with radiculopathy (discogenic sciatica). This study was not performed in an ED, but the pain was defined as acute if it had been present between 1 and 6 weeks. Consistent with our study, a statistically significant, although smaller and possibly not clinically significant (0.6 points), improvement in VAS pain scores was found within the first 24 h post-therapy. This effect did not persist beyond 3 days post-therapy and no functional score improvements were noted. Although similar in design, the smaller effect size may be explained by the subacute nature of the symptoms, whereas we included patients that likely had pain of a more acute nature.

Two studies assessed the effect of 160 mg IMI methylprednisolone in ED patients with back pain. The first study evaluated patients without radiculopathy and did not show improvement in pain or functional scores at 1 week or 1 month. The second study included patients with radicular symptoms and reported statistically non-significant improvement in the primary outcome of VAS pain scores at 1 week (1.1 points) and...
patients with radiculopathy may be more likely to benefit from glucocorticoids, possibly explaining the positive short-term effect in our population who required radiating features of back pain to be included. Furthermore, an intervention effect is often more pronounced at the severe end of the spectrum of disease. Difficulty mobilising was part of our inclusion criteria, potentially leading to inclusion of patients with more severe pain than previous studies, further explaining our more prominent effect.

Limitations
We used a real-world definition of ED patients with low back pain and radiating features, rather than more complex validated tools, and a hybrid definition of SLR test (combining reproduction of pain on SLR—on any angle—with the more narrow definition of reproduction of pain in sciatic distribution when raising 30°–70°). This has probably led to inclusion of a spectrum of disorders, not just patients with discogenic sciatica. Eighty-three potentially eligible patients were not considered for inclusion (figure 1). This was mainly due to other clinical priorities and the logistical challenges of an unfunded study. These missed eligible patients had similar demographics as the patients that were included, making selection bias unlikely. Patients did not receive any imaging as part of the study protocol to further delineate the cause of the pain. This is congruent with guidelines suggesting that acute imaging is not indicated unless cauda equina is suspected or severe motor function deficit is present. We did not distinguish between a first episode of acute back pain and exacerbations of chronic back pain. Although computer-generated randomisation occurred and allocation concealment was maintained, there was baseline imbalance on VAS pain score, with a higher score in the dexamethasone group. Since the rest of the baseline characteristics were similar and this was a small trial, we attributed this difference due to chance alone. A regression analysis controlling for baseline VAS imbalance on the treatment effect found that some (28%)—but not much—of the apparent treatment effect at 24 h was due to this baseline imbalance, with little impact (6%) of oxycodone use on the treatment effect, suggesting a genuine treatment effect of dexamethasone at 24 h. Furthermore, we are unable to comment on possible unmeasured confounders that may have had an effect on outcomes. Specifically, we did not measure physiotherapy compliance or

Table 3: Secondary outcomes

<table>
<thead>
<tr>
<th></th>
<th>Dexamethasone</th>
<th>Placebo</th>
<th>Placebo minus dexamethasone (95% CI)</th>
<th>p Value</th>
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<tbody>
<tr>
<td>Baseline, n=29; 29</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SLR angle—range of motion at hip—baseline (degrees), mean (95% CI)</td>
<td>29.0 (22.3 to 35.7)</td>
<td>37.2 (30.9 to 43.5)</td>
<td>8.2 (0.8 to 17.1)</td>
<td>0.073</td>
</tr>
<tr>
<td>Oswestry score—baseline (%), mean (95% CI)</td>
<td>62.3 (54.9 to 69.7)</td>
<td>63.3 (55.1 to 71.6)</td>
<td>1.0 (−9.7 to 11.8)</td>
<td>0.85</td>
</tr>
<tr>
<td>Discharge, n=29; 29</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>SLR angle—range of motion at hip—discharge (degrees), mean (95% CI)</td>
<td>49.2 (32.7 to 65.7)</td>
<td>42.7 (33.7 to 51.6)</td>
<td>−6.5 (−23.0 to 10.0)</td>
<td>0.43</td>
</tr>
<tr>
<td>ED length of stay, hrs, median (IQR)</td>
<td>3.50 (1.7 to 20.1)</td>
<td>18.8 (3.0 to 32.2)</td>
<td></td>
<td>0.049*</td>
</tr>
<tr>
<td>24 h, n=26; 22</td>
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<td></td>
</tr>
<tr>
<td>Oswestry score—24 hours (%), mean (95% CI)</td>
<td>61.8 (53.0 to 70.6)</td>
<td>58.8 (50.0 to 67.5)</td>
<td>−3.0 (−15.1 to 9.1)</td>
<td>0.62</td>
</tr>
<tr>
<td>Total number of 5 mg Oxycodone tablets used—24 h, median (IQR)</td>
<td>2.0 (0.5 to 3.5)</td>
<td>2.0 (0.0 to 4.0)</td>
<td></td>
<td>0.76*</td>
</tr>
<tr>
<td>6 weeks, n=20; 16</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oswestry score—6 weeks (%), mean (95% CI)</td>
<td>38.5 (26.3 to 50.7)</td>
<td>41.4 (29.8 to 53.0)</td>
<td>2.9 (−13.4 to 19.3)</td>
<td>0.72</td>
</tr>
<tr>
<td>Total number of 5 mg Oxycodone tablets used—6 weeks, median (IQR)</td>
<td>7.0 (1.3 to 17.5)</td>
<td>3.0 (0.0 to 25.0)</td>
<td></td>
<td>0.73*</td>
</tr>
</tbody>
</table>

* Mann–Whitney U test, other p values are independent t test. ED, emergency department; SLR, straight leg raise.

Figure 2: Visual analogue scale pain scores.
analgesia use other than oxycodone. The study was conducted in the EDs of a tertiary and urban hospital to improve generalisability, although in the same health district. We recruited fewer patients than intended and registered. However, the study was powered for the primary outcome (VAS pain score at 24 h), but not for VAS pain scores at 6 weeks as intended. This was due to relevant staff relocating and the study being unfunded. Change in VAS scores had reasonably narrow 95% CI to suggest a fair accuracy of effect estimate. Primary outcomes at 24 h were not available for 10 randomised patients. Every effort was made to contact these patients, and we are unable to comment on the effect this may have had. We found a statistically significant and clinically important decrease in EDLOS. Although this was a secondary outcome to generate further hypothesis, similar effects of dexamethasone on EDLOS have been found in patients with sore throat.24

In summary, this study found that a single dose of intravenous dexamethasone in ED patients with low back pain and radicularpathy provided a statistically and clinically significant improvement in pain score at 24 h compared with placebo, when added to routine management. We conclude that a single dose of dexamethasone can be used as a safe adjunct for pain relief and may decrease EDLOS.

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Contributors RB, IH and GBK conceived the study and prepared ethics. RB, IH, SP and GBK were responsible for data collection. MCS performed the statistical analysis. RB, IH and GBK were responsible for the data interpretation and drafting of manuscript. All authors approved the final manuscript.

Competing interests None.

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