



Spinal epidural abscess: Does social deprivation influence 1-year mortality?

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ABSTRACT

Background: Spinal epidural abscess are difficult to diagnose due to its non-specific clinical presentation. Spinal epidural abscess is identified late or missed in almost half of patients. To better assist clinicians in making this diagnosis there needs to be a better appreciation of at risk patient profile.

Methods: We identified all patients who were admitted to a tertiary referral centre with a diagnosis of spinal epidural abscess between February 2009 and January 2022. Demographic details, presentation details, past medical history, concurrent infection, and social factors were recorded for all patients. The primary aim of our study was to examine if there is a relationship between social deprivation and risk of mortality at one year in patients with spinal epidural abscess.

Results: There were 140 eligible patients associated with 146 hospital encounters for spinal epidural abscess. Eighteen patients died at ≤ 365 days post discharge, while 122 patients were alive at >365 days post discharge. There were 86 males and 54 females. The average age was 59.9 years with an IQR of 19.3. There were 28 % Māori, 58 % European and 12 % of all other ethnicities. In the most deprived quintile there were 34.5 %, compared to 7.5 % in the least deprived quintile.

Multivariate stepwise regression analysis found that age, mean neutrophil count and congestive heart failure all showed a statistically significance association with mortality at one year. There was no association between one year mortality and these variables: deprivation status, rurality or Māori ethnicity.

Conclusion: Mortality at one year appears to be significantly associated with age, mean neutrophil count and congestive heart failure. There was no obvious association between deprivation and mortality at one year. Identifying patients at the greatest risk of mortality remains a complex challenge.

1. Introduction

Spinal epidural abscess (SEA) are a rare (2 to 12.5 per 10,000) but serious condition [1]. SEA can have serious consequences such as irreversible neurological impairment and carries a mortality risk of about 15 % [2,3].

SEA may be treated with medical therapy alone or in combination with surgical intervention. Traditionally SEA was considered a surgical emergency, however a 2014 systematic review found no significant difference in motor outcomes between those medically managed compared to those surgically managed [1]. A later systematic review in 2016 concluded that in neurologically symptomatic patients, surgery with adjuvant antibiotics remains the optimal management. It would appear that the optimal SEA management regimen remains a topic of controversy [4].

SEA is often difficult to diagnose, it is identified late or missed in up

to 50 % of patients [5]. The main symptoms associated with SEA are back pain, fever and altered neurology, however, this presentation is often seen in many other soft tissue conditions such as osteomyelitis. Indeed, Hunter et al. found that the “triad” of back pain, fever, and altered neurology was only identified in 7 % of the patients that they studied [5]. Commonly associated patient factors are: age, diabetes mellitus, peripheral vascular disease, renal failure, malignancy, use of immunosuppressive medications, cardiac disease, liver disease, endocarditis, intravenous drug use and alcohol abuse [4].

Although there is some appreciation of at risk patient profile [1,4], numerous variables have not been studied. In 2021 Hunter et al. noted that multifocal sepsis, Māori ethnicity and elevated white cell count are all predictors for failing SEA medical management. They acknowledged that social deprivation, distance from treatment facility, body mass index (BMI) and smoking status are likely implicated in the prognosis of SEA [5]. Social deprivation is an often a neglected variable when

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considering the prognosis of various diseases.

Therefore, the primary aim of our study was to examine if there is a relationship between social deprivation and risk of mortality at one year in patients with SEA. To our knowledge this will be first study to examine this relationship. The secondary aims of this study were to examine if mortality at one year is associated with: rurality (geographical location), ethnicity and comorbidity (defined by the Charlson Comorbidity Index [CCI]). We hypothesise that a higher deprivation score will be associated with a higher mortality rate at one year.

2. Methods

Approval for this audit was granted by the Institutional Review Board (# 4498P). Permission was also granted for a retrospective outcome analysis and on this basis the need for patient consent was waived.

Using hospital coding, we identified all patients who were admitted with a diagnosis of SEA between February 2009 and January 2022. These years were selected in order to examine a full 10 year period and to allow us to follow up mortality at one year.

The study was conducted at a Level 1 trauma centre and tertiary referral spine centre for a population of just over 900 000 [6]. All pyogenic spinal column infections are managed by the Orthopaedic Spinal Service in conjunction with the specialist Infectious Disease service.

Census data from 2023 shows a diverse demographic for the study region with 71.7 % New Zealand Europeans and 25.2 % Māori; there is a relatively higher proportion of Māori in our region compared to New Zealand as a whole (17.8 %) [7]. In the Waikato Hospital region this is the percentage breakdown of residents in each of the deprivation deciles, from 1 to 10 respectively: 6.4 %, 6.3 %, 7.5 %, 8.0 %, 8.6 %, 10.3 %, 12.6 %, 12.8 %, 14.6 %, 13.0 %. In general Waikato has a greater portion of residents in the most deprived deciles compared to the general New Zealand population [8].

Inclusion criteria is all patients (any age) who were discharged from Waikato Hospital with the diagnosis of SEA. Post operative epidural abscess surgical site infections were excluded. Diagnoses were confirmed based on radiological evidence with magnetic resonance imaging (MRI).

For the purposes of this study the main outcome of interest was mortality at one year post-discharge. Therefore patients were divided into two groups. Group 1 refers to the patients allocated to the “mortality in ≤ 365 days” group. These were patients who died ≤ 365 days post discharge. Group 2 refers to the “no mortality in ≤ 365 days” group, these are patients who did not die ≤ 365 days post discharge.

2.1. Data collection

Explanatory variables and outcomes were collected retrospectively. Demographic details, presentation details, past medical history, concurrent infection, and social factors were recorded. Details included: age, sex, ethnicity, weight, height, smoking status and comorbidities (as defined by the CCI) (see [appendix 1](#)) [9].

Information was collected on presentation details. Admission laboratory results were collected including: C-reactive protein ([CRP], mg/L), haemoglobin ([Hb] g/L), albumin ([Alb], mg/L), White Blood Cell ([WBC] $\times 10^9/L$), Neutrophils ([NEUT] $\times 10^9/L$), Lymphocytes ([LYMPH] $\times 10^9/L$) and Platelets ([PLT] $\times 10^9/L$). For the purposes of this study, “sepsis” was defined by the SIRS criteria [10].

Microbiology was reviewed for: surgical cultures, blood cultures and urine cultures during the admission period. Number of positive cultures and isolated organism were recorded.

MRI reports and images were obtained to quantify the level and position of the abscess relative to the thecal sac. Information regarding the clinical management of the patient was also collected including; name of antibiotic, route of antibiotic, dose and duration of the

inpatient/outpatient antibiotics administered and type of surgical management. Post op Intensive Care Unit (ICU) admission was recorded.

Length of stay (defined by time from MRI diagnosis to discharge from hospital) was calculated; readmission to hospital within 30 days were also recorded.

Past medical history, including previous surgeries in the past 3 months and evidence of prednisone use prior to admission were recorded. Location of any concurrent infections were also recorded.

Information on social factors were recorded, namely NZ Deprivation 2018 (NZDEP2018) score (scale from 1 to 10, with 1 being least deprived and 10 being the most deprived) [11] and Geographical Classification of Health (GCH) score (scale with the following measurements U1, U2, R1, R2, R3 with U1 being the least rural and R3 being the most rural) [12].

2.2. Statistical analysis

The 2 cohorts were represented by descriptive statistics; relative frequencies, percentages, interquartile range and means were calculated. Univariate analysis for descriptive statistics was either *t* test (numerical variables) or chi-squared test (categorical variables).

Given that the focus of this paper was on social deprivation we conducted a univariate subgroup analysis to see if any of our variables were correlated with social deprivation. We then performed a multivariate stepwise regression analysis to identify potential predictors of death at ≤ 365 days post discharge. Statistical significance was accepted if $p < 0.05$.

We used Jamovi software to run a “independent samples *t*-test” power analysis in order to evaluate the sensitivity of our study. We found that with group sizes of at least 18 and 126 we can reliability detect with a probability > 0.8 and an effect size ≥ 0.711 , which would be considered as a “large effect”. We have assumed a two sided criterion for detection that allows for a maximum type 1 error rate of $\alpha = 0.05$.

3. Results

3.1. Demographics

Over the 12-year period, there were 140 patients associated with 146 hospital encounters with SEA. There were 86 males and 54 females; average age was 59.9 years with an IQR of 19.3. There were 28 % Māori, 58 % for New Zealand European and 12 % for all other ethnicities; these figures closely align with ethnic breakdown for the Waikato region [7]. The most deprived quantile contained 34.5 % of patients, compared to 7.534 % in the least deprived quantile. The mean BMI for the cohort was 40.4 with a SD of 92.4 ([Table 1](#)).

3.2. Microbiology

Positive surgical specimen cultures were recorded in 64 % of episodes, 50 % of which were positive for *S. aureus*. Positive blood cultures were recorded in 55 % of episodes, 41 % of which were positive for *S. aureus*.

Abscess location was predominantly dorsal (54 %), compared with 28 % being ventral. Ninety one patients had an abscess in the lumbar region (62 %), 43 were thoracic (29 %), 37 were cervical (25 %), 26 were sacral (17 %).

For those patients with concurrent infection, 44 had a concurrent musculoskeletal infection, while 54 had a concurrent non-musculoskeletal infection. There were 21 psoas infections, 9 knee infections, 8 hip infections and 8 shoulder infections. In comparison to the non-musculoskeletal infections 23 were lung infections and 19 were genitourinary tract infections.

Table 1

Demographic characteristics of the all 140 with spinal epidural abscess presenting to Waikato Hospital between February 2009 and January 2022, split by mortality outcome at 1 year.

Demographic variable	Death at <=365 days post discharge (N = 18)	Alive at > 365 days post discharge (N = 122)	Combined N = 140	P values (univariate) T test/ Chi squared
Mean age (IQR ^a)	75.722 (12.75)	57.672 (18.75)	59.993 (19.250)	<0.001*
Male Gender	13 (72.222 %)	73 (59.836 %)	86 (61.429 %)	0.314
Māori	4 (22.222 %)	36 (29.508 %)	40 (28.571 %)	0.523
NZ European	11 (61.111 %)	71 (58.197 %)	82 (58.571 %)	0.815
All other ethnicities	3 (16.667 %)	15 (12.295 %)	18 (12.857 %)	0.605
Details of the encounters				
	Death at <=365 days post discharge (N = 18)	Alive at > 365 days post discharge (N = 128)	Combined (N = 146)	P values (univariate) T test/ Chi squared
Smoking	3 (16.667 %)	25 (19.531 %)	28 (19.178 %)	0.773
Mean BMI ^b (SD)	28.760 (7.708)	41.976 (98.429)	40.390 (92.421)	0.605
Sepsis	13 (72.222 %)	60 (46.875 %)	73 (50 %)	0.044*
MRC Muscle Strength (Grade 4-5)	15 (83.333 %)	102 (79.688 %)	117 (80.137 %)	0.717
MRC Muscle Strength (Grade 1-3)	3 (16.667 %)	26 (20.313 %)	29 (19.863 %)	0.717
NZDEP2018 quantile				
Quantile 1 (NZDEP2018 1-2) [Least deprived]	2 (11.111 %)	9 (7.031 %)	11 (7.534 %)	0.549
Quantile 2 (NZDEP2018 3-4)	2 (11.111 %)	13 (10.156 %)	15 (10.274 %)	0.910
Quantile 3 (NZDEP2018 5-6)	2 (11.111 %)	28 (21.875 %)	30 (20.548 %)	0.287
Quantile 4 (NZDEP2018 7-8)	5 (27.778 %)	34 (26.563 %)	39 (26.712 %)	0.929
Quantile 5 (NZDEP2018 9-10) [Most deprived]	7 (38.889 %)	43 (33.594 %)	50 (34.247 %)	0.677
Not from NZ	0 (0 %)	1 (0.781 %)	1 (0.685 %)	NA
GCH				
U1	6 (33.333 %)	41 (32.031 %)	47 (32.192 %)	0.930
U2	5 (27.778 %)	24 (18.750 %)	29 (19.863 %)	0.382

Table 1 (continued)

Demographic variable	Death at <=365 days post discharge (N = 18)	Alive at > 365 days post discharge (N = 122)	Combined N = 140	P values (univariate) T test/ Chi squared
R1	4 (22.222 %)	37 (28.906 %)	41 (28.082 %)	0.546
R2	2 (11.111 %)	19 (14.844 %)	21 (14.384 %)	0.667
R3	1 (5.556 %)	6 (4.688 %)	7 (4.795 %)	0.879
Not from NZ	0 (0 %)	1 (0.781 %)	1 (0.685 %)	NA
Laboratory Values				
Mean Platelets (IQR)	226.611 (124.000)	287.641 (201.000)	280.117 (201.500)	0.118
Mean white cell count (IQR)	15.833 (7.008)	12.644 (6.485)	13.037 (6.752)	0.032*
Mean Neutrophils (IQR)	13.986 (5.973)	10.321 (5.800)	10.773 (6.547)	0.007*
Platelet: Lymphocyte Ratio	226.611: 0.846	287.641: 1.335	280.117: 1.275	0.098
Microbiology				
Positive Surgical specimen cultures	7 (38.889 %)	87 (67.969 %)	94 (64.384 %)	0.016*
Positive Surgical specimen positive for S. aureus	7 (38.889 %)	67 (52.344 %)	74 (50.685 %)	0.285
Positive Blood cultures	13 (72.222 %)	68 (53.125 %)	81 (55.479 %)	0.127
Blood cultures positive for S. aureus	9 (50 %)	52 (40.625 %)	61 (41.781 %)	0.450
Abscess characteristics				
Cervical	4 (22.222 %)	33 (25.781 %)	37 (25.342 %)	0.745
Thoracic	5 (27.778 %)	38 (39.688 %)	43 (29.452 %)	0.868
Lumbar	13 (72.222 %)	78 (60.938 %)	91 (62.329 %)	0.868
Sacral	3 (16.667 %)	23 (17.969 %)	26 (17.808 %)	0.892
Ventral	8 (44.444 %)	34 (26.563 %)	42 (28.767 %)	0.118
Dorsal	7 (38.889 %)	73 (57.031 %)	80 (54.795 %)	0.150
Circumferential	3 (16.67 %)	21 (16.406 %)	24 (16.438 %)	0.978
Medical co-morbidities				
MI ^c	4 (22.222 %)	14 (10.938 %)	18 (12.329 %)	0.173

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Table 1 (continued)

Demographic variable	Death at <=365 days post discharge (N = 18)	Alive at > 365 days post discharge (N = 122)	Combined N = 140	P values (univariate) T test/ Chi squared
CHF ^d	7 (38.889 %)	21 (16.406 %)	28 (19.178 %)	0.023*
Peripheral vascular disease	2 (11.111 %)	0 (0 %)	2 (1.370 %)	<0.001*
CVD ^e	2 (11.111 %)	8 (6.25 %)	10 (6.849 %)	0.445
Peptic ulcer disease	4 (22.22 %)	42 (32.813 %)	46 (31.507 %)	0.365
Diabetes	7 (38.889 %)	25 (19.531 %)	32 (21.918 %)	0.063
Moderate or severe renal disease	9 (50 %)	33 (25.781 %)	42 (28.767 %)	0.034*
Diabetes with end organ damage	10 (7.813 %)	4 (22.222 %)	14 (9.589 %)	0.052
Mean Charlston comorbidity index (IQR ^a)	3.611 (3.220)	1.875 (1.94)	2.089 (3)	0.038* (Welch's)

^a IQR, Interquartile range.

^b BMI; Body Mass index.

^c MI, Myocardial Infraction.

^d CHF, Congestive Heart Failure.

^e CVD, Cerebrovascular Disease.

* significant.

3.3. Comorbidity

The most prevalent comorbid conditions were congestive heart failure (n = 28, 19 %), peptic ulcer disease (n = 46, 31 %), diabetes (n = 32, 21 %) and moderate or severe renal disease (n = 42, 28 %).

3.4. Univariable analysis

Group 1, had a significantly higher mean age (p = 0.0001). The rates of sepsis were also significantly different. Sepsis was seen in 72 % of Group 1 compared with only 46 % in the other group (p = 0.044) (Table 1).

Mean White Cell Count (IQR) and Mean Neutrophil Count (IQR) were significantly higher in Group 1 compared to Group 2; 15.833 (7.008) and 13.986 (5.973) vs 12.644 (6.485) and 10.321 (5.8) respectively (Table 1).

The prevalence of congestive heart failure (p = 0.023), peripheral vascular disease (p = 0.0001463) and moderate or severe renal disease (p = 0.034) were different between the two groups. The mean CCI is significantly higher in the Group 1 (p = 0.038) (Table 1).

3.5. Subgroup analysis

We conducted a subgroup univariate analysis to see if any of our variables were correlated with social deprivation. We found that Māori ethnicity (p < 0.001), New Zealand European ethnicity (p < 0.001), having moderate or severe renal disease (p = 0.022) and having a high CCI score (p = 0.025) were all significantly correlated to social deprivation. There was no association between social deprivation and any of the laboratory values (Table 2).

3.6. Multivariate analysis

We conducted a multivariate stepwise regression analysis and found that age, mean neutrophil count and congestive heart failure showed a

statistically significance difference (Table 3).

The Nagelkerke R Square calculated for this study was 0.454. Nagelkerke R Square ranges from 0 to 1, with values closer to 1 indicating that the model explains the data perfectly. It is generally accepted that a value of 0.4 or higher means that the variables fit the model well. Therefore the variables of; age, mean neutrophil count, congestive heart failure and gender appear to be significant predictors of mortality at one year.

4. Discussion

The primary aim of this study was to examine if there is a relationship between the social deprivation of an SEA patient and their risk of mortality at one year. We had hypothesised that there was likely a strong relationship between level of social deprivation and 1-year mortality. This is the first study to our knowledge to examine the potential role of social deprivation in predicting mortality in SEA patients.

The data that we collected failed to show any significant association between the social deprivation of an SEA patient and their risk of mortality at one year. This surprisingly, led to a rejection of our initial hypothesis. Historical surgical data strongly indicates that more deprived peoples have worse surgical outcomes. A study of 9,034 patients treated in the United Kingdom National Health Service indicated that at 3 years post discharge from a surgical procedure, a greater number of patients from the most deprived quantile died compared to those in the least deprived quantile. The most deprived fifth of the population had a 40 % greater risk of dying after 3 years, compared to the least deprived fifth of the population [13]. In contrast, a Scottish cohort study of 1,477,810 general surgical patients found that mortality outcomes are not affected by deprivation, but greatly affected by comorbidity status. Patients who had a CCI > 4 had a 1-year mortality risk 90–96 times that of the comparison group [14].

The reason underpinning this association appears uncertain. It might be that lower socioeconomic people are more comorbid due to reduced access to primary care, which leads to later identification of disease and therefore delayed intervention. It may relate to relative income and access to basic food and medicines which can vary between geographical regions and health care systems. Patients with greater co-morbidity or more advanced diseases states (e.g., end-stage diabetes as opposed to well-controlled) are more likely to have poorer rates of post-surgical recovery, which may explain the higher mortality rate seen in lower socioeconomic communities [15].

Our study and others like it, have failed to demonstrate a linear relationship between social deprivation and mortality. Indeed a study conducted in the United States analysed a group of patients with 'proxy markers' for low socioeconomic status, such as being homeless, diagnosed with a substance use disorder or having a Medicaid benefit [16]. Brown et al. indicated that there was no difference in the in-hospital mortality rate or new paralysis rate between the proxy "deprived" group and the "non-deprived" comparison group. The authors of the study accept that this was a surprising result and suggest that it might have been due to the small sample size of their study [16].

Wan et al. suggests that the relationship between lower socioeconomic status and long term mortality outcomes should be considered as an "indirect" relationship. These authors indicate that lower socioeconomic peoples have a higher baseline comorbidity risk, which means that they are more likely to suffer a post-operative complication. It appears that "post-operative complication" is the factor directly correlated with mortality outcomes, rather than socioeconomic status itself. The data of their study appears to support this theory. In their study the post-op complication rate was 12.3 % in the most deprived quantile compared to 9.9 % in the least deprived quantile [13]. On multivariate analysis they noted that patients who had a post operative complication had a reduced 3 year survival compared to those who did not have a post-op complication. They concluded that the impact of post-operative complication on survival was much greater than the impact of

Table 2

Demographic, laboratory, microbiology, and comorbidity variables of the spinal epidural abscess encounters in comparison to deprivation quantile.

	Quantile 1 (NZDEP2018 1–2) [Least deprived]	Quantile 2 (NZDEP2018 3–4)	Quantile 3 (NZDEP2018 5–6)	Quantile 4 (NZDEP2018 7–8)	Quantile 5 (NZDEP2018 9–10) [Most deprived]	P value (ANOVA/Chi squared)
Demographic variable						
Mean age (IQR ^a)	66.182 (13)	56.333 (18)	63.033 (23.25)	62.590 (18.500)	56.680 (16.000)	0.314
Gender (Female)	4 (36.364 %)	5 (33.333 %)	9 (30.000 %)	18 (46.154 %)	20 (40.000 %)	0.715
Māori	1 (9.091 %)	1 (6.667 %)	2 (6.667 %)	10 (25.641 %)	27 (54.000 %)	<0.001*
NZ European	9 (81.818 %)	12 (80.000 %)	25 (83.333 %)	21 (53.846 %)	18 (36.000 %)	<0.001*
All other ethnicities	1 (9.091 %)	2 (13.333 %)	3 (10 %)	8 (20.513 %)	5 (10.000 %)	0.607
Smoking	1 (9.091 %)	4 (26.667 %)	1 (3.333 %)	8 (20.513 %)	14 (28.000 %)	0.071
Mean BMI (IQR ^a)	27.610 (3.659)	31.076 (9.998)	32.926 (5.747)	29.134 (8.538)	59.340 (11.130)	0.6
Sepsis	6 (54.545 %)	7 (46.667 %)	14 (46.667 %)	20 (51.282 %)	26 (52.000 %)	0.983
Laboratory Values						
Mean Platelets (IQR ^b)	300.455 (118.500)	312.733 (171.000)	284.370 (228.250)	299.692 (222.000)	246.460 (180.000)	0.433
Mean white cell count (IQR ^b)	12.653 (5.110)	13.475 (6.740)	12.064 (6.815)	13.084 (6.700)	13.626 (6.375)	0.843
Mean Neutrophils (IQR ^b)	10.380 (4.890)	10.831 (6.820)	10.015 (6.217)	10.669 (6.845)	11.481 (7.150)	0.833
Platelet: Lymphocyte Ratio	300.455: 1.252	312.733: 1.354	284.370: 0.997	299.692: 1.403	246.460: 1.308	0.725
Microbiology						
Positive Surgical specimen cultures	2.000 (2.5)	1.400 (2.5)	1.800 (3)	2.436 (4)	2.680 (3.75)	0.389
Positive Surgical specimen positive for Staph Aureus	0.545 (0.522)	0.333 (0.488)	0.467 (0.507)	0.513 (0.506)	0.580 (0.499)	0.544
Positive Blood cultures	1.545 (2.5)	1.267 (2)	1.100 (2)	1.333 (2)	1.840 (2.75)	0.476
Blood cultures positive for Staph Aureus	0.364 (1)	0.400 (1)	0.367 (1)	0.359 (1)	0.520 (1)	0.539
Medical co-morbidities						
MI ^b	0 (0.000 %)	2 (11.111 %)	3 (16.667 %)	5 (27.778 %)	8 (44.444 %)	0.676
CHF ^c	2 (7.143 %)	3 (10.714 %)	5 (17.857 %)	5 (17.857 %)	13 (46.429 %)	0.620
Peripheral vascular disease	0 (0.000 %)	1 (50.000 %)	1 (50.000 %)	(0) 0.000 %	(0) 0.000 %	0.256
CVD ^d	0 (0.000 %)	2 (20.000 %)	1 (10.000 %)	5 (50.000 %)	2 (20.000 %)	0.271
Peptic ulcer disease	2.000 (4.348 %)	2.000 (4.348 %)	14.000 (30.435 %)	11.000 (23.913 %)	17.000 (36.957 %)	0.152
Diabetes	1.000 (3.125 %)	3.000 (9.375 %)	4.000 (12.500 %)	13.000 (40.625 %)	11.000 (34.375 %)	0.256
Moderate or severe renal disease	0 (0.000 %)	2.000 (4.762 %)	6.000 (14.286 %)	16.000 (38.095 %)	18.000 (42.857 %)	0.022*
Diabetes with end organ damage	0 (0.000 %)	1 (7.143 %)	0 (0.000 %)	7 (50.000 %)	6 (42.857 %)	0.094
Mean Charleston comorbidity index (IQR ^b)	0.636 (0.5)	1.400 (2)	1.733 (2.75)	2.692 (2)	2.400 (3)	0.025*

^a IQR, Interquartile range.

^b MI, Myocardial Infraction.

^c CHF, Congestive Heart Failure.

^d CVD, Cerebrovascular Disease.

* significant.

Table 3

Stepwise logistic regression analysis.

Variable	Estimate	Standard Error	Odds Ratio	95 % Confidence interval (odds ratio scale)	P value
Age	0.143	0.037	1.153	1.073, 1.240	0.0001118*
Neutrophils	0.196	0.059	1.217	1.084, 1.366	0.0008543*
CHF ^a	1.482	0.694	4.403	1.130, 17.163	0.033*
Gender	-1.099	0.683	0.333	0.087, 1.271	0.108

^a CHF, Congestive Heart Failure.

* significant.

deprivation [13]. Du et al. concur with this theory, believing that reducing post-operative cardiac and pulmonary complications would result in an associated decrease SEA mortality [17].

Using postoperative complications to predict mortality has been suggested previously in the spine literature. A cross sectional study of 4073 patients who underwent adult spinal deformity surgery noted that there was strong overlap between cardiac complications and mortality

[18]. If post-surgical complications can be accurately predicted then mortality prognostication would greatly increase, however as we see social deprivation is not an obvious variable to include [18].

One of the largest retrospective studies conducted by Schoenfeld and Wahlquist on SEA, noted that the inpatient mortality was 3 %, compared to complication rate of 26 %. The authors of this study did not follow up these patients over time, but had they done so it might have revealed this association between postoperative complications and long term mortality outcomes [19].

The literature seems to support the idea that variables other than socioeconomic status appear to be more strongly associated with mortality prognostication. The literature seems to suggest that variables such as comorbidity and postoperative complication are more closely associated with mortality outcomes.

It seems that the variables associated with mortality have not yet been clearly defined. Our study seems to suggest that perhaps the metric used to determine deprivation in New Zealand (NZDEP2018 score) is ineffective in determining mortality risk in SEA patients. The variables used to determine a NZDEP2018 score are: access to the internet, beneficiary status, income threshold, employment status, qualifications, living in own home, in a single parent family, bedroom occupancy level

and household dampness [11]. Wan et al. similarly failed to mention a strong association between the English Indices of Multiple deprivation 2019 (IMD 2019) and the long term mortality outcomes in general surgical patients. The seven domains included in the IMD 2019 are: income, employment, health and disability, education, skills and training, barriers to housing and other services, crime and living environment. Rather Wan et al. indicate that the statistically significant variables associated with long term mortality outcomes are: older age, male sex, ASA 2 to 4, metastatic cancer, lower preoperative haemoglobin and higher preoperative creatinine [13]. It appears that there the most useful measures for predicting mortality outcomes in SEA patients are not included in the NZDEP2018 scoring system.

We also hypothesised that perhaps ethnicity may be associated with 1-year mortality, given that Māori are over represented in lower deprivation quantiles, with 47.5 % of Māori being classified as quantile 5 (most deprived) compared to 21.4 % for European [8]. A national New Zealand audit published in 2021 of 876,976 acute surgical procedures found that the 30-day post-op mortality rate was highest among Māori peoples (age-standardised rate: 1.1/100), compared to any other ethnic group [20]. Māori were 14 % more likely to die within 30 days from any acute procedure compared to New Zealand Europeans, and 33 % more likely to die at 30 days post-op following an acute musculoskeletal operation [20]. Māori have a 30 % higher risk of post-op complication in comparison to Europeans, following an acute musculoskeletal procedure [20]. It is therefore reasonable to suggest that Māori, may have a higher mortality rate associated with SEA. However on analysis of our dataset we found being Māori was not associated with a higher mortality rate at 1 year.

We conducted an additional subgroup analysis and found that there was no statistically significant difference in mean CCI score between Māori and non-Māori ($p = 0.487$) (Table 4). The CCI is an easy way to compare the comorbidity status between two groups. It is believed having more co-morbidities reduces a patient immunocompetency, reducing a patients ability to fight an infection [21]. As a result patients with a higher CCI are more likely to have higher disease severity status and a higher risk of developing a post-operative infection. A 2024 New Zealand study of 150 SEA patients reported that a CCI score of 2.75 was associated with an increased 1-year mortality rate (OR = 1.34, CI 95 % [1.06–1.72], $p = 0.01$) [22]. In our subgroup analyses none of the groups had a mean CCI > 2.75. It is possible that mortality outcomes are in fact correlated with the CCI score. However it appears that the subgroups of Māori vs non-Māori did not have a high enough mean CCI score for a significant difference to be detected on subgroup analysis. Another study seems to support this theory: Lenga et al. found that a higher age-adjusted CCI score (OR = 1.8; 95 %CI 1.1–5.2; $p = 0.002$) was a significant predictor of mortality in SEA patients [21]. In their study the mean age-adjusted CCI score across groups ranged from 4.8 to 9.2. Again it appears that the population that we studied may not have had a high enough mean CCI score to detected a meaningful difference on subgroup analysis.

International data from Australia and Canada indicate that on average rural peoples have a higher mortality rate and lower life expectancies in comparison to their urban counterparts. Rural peoples of New Zealand have similarly high mortality rates. The way rural is defined in the healthcare context was recently redefined using the GCH, which is a novel classification tool that uses a mixed methods approach to define rural. It is based on; population size, density, drive time to medical services and access to 24-hour primary care/other prehospital

services [12]. Although the classification was only created in 2021, it appears to be a highly accurate tool [12]. Nixon 2023 concluded that on the basis of GCH for those aged < 60 years, mortality rates are higher in rural areas compared with major urban centres [23]. Nixon 2023 suggest that the largest urban–rural disparity is seen for injury-related deaths. In those aged < 60 years, injury-related mortality rates are markedly higher among rural populations compared to urban populations. Mortality rate ratios regarding injury-related mortality for non-Māori < 30 years, was 3.07; with a 95 % CI 2.09 to 4.51 [23].

Although our study did not show a significant difference in mortality outcome for urban vs rural, this appears consistent with the literature. An earlier study that was conducted in the same region as our study indicated that the 90 day mortality rate for the “geographically remote” was 10 % [22]. This mortality rate appears to sit well within the generally reported range of 1–16 % [24].

Many demographic and etiological risk factors have been associated with the development of SEA. A 2000 systematic review, which is still quoted in recent literature indicates that there were 1005 individual risk factors associated with SEA [25]. Both this 2000 systemic review and a later one conducted in 2014 concluded that diabetes mellitus, increasing age, intravenous drug use and renal failure are all clearly associated with SEA [1,25].

Our study provides useful data, given that most of the available literature is focused on presenting data on the 30/90 day mortality risk or the in-hospital mortality risk. Shah et al., produced one of the largest retrospective studies which stated that there were 8 independent predictors of 90-day mortality, including: age greater than 65 years, diabetes, active malignancy, haemodialysis, pre-treatment motor deficit, endocarditis, pre-presentation duration of symptoms and leucocytosis [26]. Although this study was conducted across both regional and metro populations it helps to validate the results of our study. Shah et al. reports WBC count as an independent risk factor for 90-mortality, where as our study further specifies this by stating that perhaps it is the mean neutrophil count that most predictive variable of mortality. Close examination of the literature seems to support our previous idea that perhaps the factors which are most strongly correlated with mortality outcomes are not encapsulated within mainstream socioeconomic scoring scales [13].

We have identified that there are a few key gaps in the literature. It appears that the current literature is mainly focused on evaluating the short term mortality outcomes of SEA patients, generally reporting the 30 or 90 day mortality outcomes. There is little data examining the longer term outcomes, namely at one year. Our study has provided some evidence to help address this gap.

Within the SEA literature a number of outcomes are often measured. Mortality is often used given its dichotomous nature. It is likely that focusing on mortality alone will not capture the true impact that this condition has on patients. Mortality is a single outcome that should be considered alongside morbidity related outcomes including: new onset paralysis, readmission rate, sensory and motor function outcomes and self-care abilities [16,27]. As the profession moves towards become more patient focused, the above outcomes should be considered alongside, more subjective outcomes such as quality of life. Although these outcomes sit outside the scope our study it does illustrate the need for further research in this area.

A major limitation of this study is that the address used to calculate the NZDEP2018 score and GCH were based on the address at the time of admission to hospital which may not reflect where a person has lived for the majority of their lives. Another significant limitation is the relatively small sample size of our study. However given the rarity of SEA, this issue has been reported elsewhere in the SEA literature [16]. The overall small sample size resulted in small subgroups, which limits the power of our subgroup analysis. There was also a significant disparity in the sizes of the two subgroups, the “mortality at ≤ 365 days” group was much smaller than the size of the “no mortality at ≤ 365 days” group. However power analysis indicated that we could still get a good “size effect” from

Table 4

Mean (standard deviation) Charleson Comorbidity Index score for Māori ethnicity and deprivation quantile.

	Mean	SD	P value
Māori	2.293	2.648	0.487
Non-Māori	2.01	2.007	

this dataset despite these limitations. Small sample sizes will continue to effect study conducted on this topic, however this limitation might be overcome by an conducting an adequately powered multi-centre collaborative study. Finally, as with all retrospective studies, our study is also limited by inherent selection bias and is reliant on clinical documentation.

5. Conclusion

Despite a relatively small cohort, we found that when age, mean neutrophil count and congestive heart failure may be used to predict mortality at 1 year in those diagnosed with SEA. Age, mean neutrophil count and congestive heart failure have been identified in other studies as important variables and therefore should be considered in models used to diagnose SEA. We found that social deprivation, Māori ethnicity and geographical location are not associated with increased mortality outcomes at 1 year. Although this retrospective study provides useful insights it is critical that a large adequately powered, prospective study be conducted. A study such as this will be useful in further characterising the seemingly non-linear relationship between the variables of social deprivation, comorbidities and postoperative complications with mortality at various time points in patients with SEA.

CRedit authorship contribution statement

Eamon P.G. Walsh: Writing – review & editing, Writing – original draft, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation. **Joseph F. Baker:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Conceptualization.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jocn.2024.110890>.

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