ORIGINAL ARTICLE

Risk factors for 90-day and 180-day mortality in hospitalised patients with pressure ulcers

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Abstract

An understanding of risk factors associated with mortality among pressure ulcer patients can inform prognostic counselling and treatment plans. This retrospective cohort study examined associations of comorbid illness, demographic characteristics and laboratory values with 90-day and 90- to 180-day mortality in adult hospitalised patients with pressure ulcers. Data were extracted from hospital databases at two academic urban hospitals. Covariates included mortality risk factors identified in other populations, including demographic and laboratory variables, DRG weight, 'systemic infection or fever' and comorbidity categories from the Charlson comorbidity index. In adjusted Cox proportional hazards models, diabetes, chronic renal failure, congestive heart failure and metastatic cancer were significantly associated with mortality in both time frames. There was no significant effect on mortality from dementia, hemiplegia/paraplegia, rheumatic disease, chronic pulmonary disease or peripheral vascular disease. Myocardial infarction, cerebrovascular disease, liver disease and human immunodeficiency virus/AIDS were associated with mortality in the 90-day time frame only. 'Systemic infection or fever' was associated with mortality in the 90-day time frame but did not show a confounding effect on other variables, and the only significant interaction term was with metastatic cancer. Albumin was the only studied laboratory value that was strongly associated with mortality. Understanding the context of comorbid illness in pressure ulcer patients sets the groundwork for more robust studies of patient- and population-level outcomes, as well as study of heterogeneity within this group.

Introduction

Pressure ulcers are a common disease in hospitalised patients: prevalence ranges from 10% to 18% (1). While risk factors for mortality have been explored for populations such as patients of general medical inpatient services and geriatric patients (2), studies of mortality have not previously looked at hospitalised patients with pressure ulcers as a unique population. Patients with pressure ulcers may have different predictors of mortality than a general inpatient population (3–5), given their high burden of severe acute and chronic illness, impaired functional status and elevated risk of sepsis because of wound infections. This epidemiologic perspective is of importance to wound clinicians who treat patients with pressure ulcers in the hospital and after hospital discharge.

Key Messages

- understanding the association of comorbid conditions and laboratory values with mortality in pressure ulcer patients can help providers to tailor treatment plans to individual clinical scenarios
- the population of patients with pressure ulcers has unique characteristics. Therefore, conditions identified as associated with mortality in general populations may not have the same relationship in this population
- diabetes, chronic renal failure, congestive heart failure and metastatic cancer were significantly associated with 90-day and 90- to 180-day mortality, as was albumin

- level. Myocardial infarction, cerebrovascular disease, liver disease and HIV/AIDS were associated with mortality in the 90-day time frame only
- dementia, hemiplegia/paraplegia, rheumatic disease, chronic pulmonary disease and peripheral vascular disease are not significantly associated with 90-day or 90- to 180-day mortality in hospitalised patients with pressure ulcers
- 'systemic infection or fever' was associated with 90-day mortality, but it did not have confounding effects with other comorbidity variables, and it interacted only with metastatic cancer

For inpatients with pressure ulcers, diseases other than the ulcer itself will often be the most likely aetiology of death. The wound healing clinician will therefore need to consider the context of the overall medical prognosis when planning for how aggressively or how conservatively to treat the wound in any given patient. Identifying a comorbid disease or laboratory marker as indicative of increased mortality in the pressure ulcer population may assist practitioners in counselling patients and families about prognosis and designing patient-centred strategies for pressure ulcer treatment. In cases where patients have poor overall prognosis based on their underlying comorbidities, the treating team may wish to set goals of care focusing primarily on minimisation of ulcer-related morbidity rather than pursue aggressive therapies aimed at healing, such as operative debridement of non-healing tissue in an otherwise stable and uninfected wound (6,7). On the other hand, if increased mortality is attributable to sepsis from the pressure ulcer itself, the treating team may consider more aggressive pressure ulcer treatment protocols to control this risk.

It is also important to understand what comorbidities do not increase mortality in this population. On the basis of data from other populations, health care providers may assume that a patient with a certain comorbid condition will have a shorter life span than other pressure ulcer patients and therefore be a poorer candidate for aggressive therapies aimed at healing. These beliefs should be tested to avoid incorrect assumptions on the part of providers about patients' longevity and consequent healing potential. Patients whose comorbid disease will not increase their risk of dying in a short- or intermediate-term time frame may require advocacy from wound healing clinicians who recommend aggressively pursuing healing in these patients.

As pressure ulcers attain increasing visibility in regulatory and quality measures, studies of this group of patients as a well-defined population become increasingly important. Any group of patients, whether this may be defined as patients with heart failure, patients admitted with sepsis to the intensive care unit or patients over 65 years old, has a heterogenous variety of concomitant health conditions and other prognostic factors. Nonetheless, progress in understanding and managing these conditions has been made by studying these entities as unified groups to understand their clinical characteristics. To understand the disease and its prognosis, pressure ulcers can benefit from the same descriptive epidemiological approach, that is, to carefully describe the patient population so as to set the groundwork for more robust studies of patient- and population-level outcomes.

In this study, we examine the association of mortality in pressure ulcer patients with comorbid diseases. The comorbidities selected were based on those included in the Charlson comorbidity index (8) and other patient characteristics and laboratory values shown elsewhere to increase mortality in populations such as medical inpatients and geriatric patients (9). 'Systemic infection or fever' was also included as a covariate in order to examine its independent effect on mortality and its confounding effects or interactions with other variables.

Methods

Setting and study population

This historical cohort study included patients aged 21 years and above admitted to either of the two urban academic hospitals between 1 January 2000 and 1 April 2008. Subjects were included if they had an ICD-9 discharge diagnosis of pressure ulcer (707·0), a recorded race in the hospital database, a recorded Social Security Number and a complete blood count or basic metabolic panel drawn during the hospital stay. For subjects with multiple admissions, only the earliest admission was considered as an index hospitalisation.

Data collection

Data were extracted from hospital databases.

Charlson comorbidity index disease categories were included as covariates (10). ICD-9 sets were identical to Charlson categories except for renal disease, diabetes and liver disease. For renal disease, a new ICD-9 set was built to include only chronic (not acute) renal disease,* as renal function on admission was separately examined based on laboratory values. The Charlson categories of 'complicated' and 'uncomplicated' diabetes were merged into a single category (11), as were 'mild' and 'moderate-to-severe' liver disease. For the Charlson categories, as for all diagnostic categories in this study, ICD-9 diagnostic codes were extracted as recorded in the hospital database for the index hospitalisation.

Where possible, laboratory data were used to identify comorbid disease in subjects not captured via ICD-9 coding. The diabetes variable was enriched by including subjects with HbA1c >6·5 (12) 365 days prior to or after the date of admission. Chronic renal disease included subjects whose highest estimated glomerular filtration rate (eGFR) within 90 days before or after admission was under <60 ml/min/1·73 m^2. Congestive heart failure included patients with ejection fraction of <50% during the hospital stay. Liver disease included patients with detectable viral loads for hepatitis B or C, a

*ICD-9 codes 250·4, 250·40, 250·41, 250·42, 250·43, 285·21, 361·04, 403, 403·00, 403·01, 403·11, 403·9, 403·90, 403·91, 404, 404·0, 404·00, 404·01, 404·02, 404·03, 404·1, 404·10, 404·11, 404·12, 404·13, 404·9, 404·90, 404·91, 404·92, 404·93, 458·21, 572·4, 581, 581·0, 581·1, 581·2, 581·3, 581·8, 581·81, 581·89, 581·9, 585, 585·1, 585·2, 585·3, 585·4, 585·5, 585·6, 585·9, 586, 587, 588, 588·0, 588·8, 588·81, 588·89, 588·9, 753·12, 753·13, 753·14, 792·5, E879·1, V45·1, V45·73, V56, V56·0, V56·1, V56·2, V56·31, V56·32, V56·8, 249·40, 249·41, V45·11, V45·12.

genotype for hepatitis C or positive hepatitis B surface antigen. Human immunodeficiency virus (HIV) included patients with a positive HIV screening test or detectable HIV viral loads. Laboratory values were screened for time periods both before and after the index admission to increase the likelihood of detecting a diagnosis that was missed in ICD-9 coding.

'Systemic infection or fever' was defined as ICD-9 codes for sepsis, septicaemia, bacteraemia and/or fever[†] on the index admission.

Data on all-cause mortality were drawn from hospital databases and the Social Security Master Death Index for 180 days after the admission date, regardless of whether death occurred before or after discharge. Social Security Administration mortality data have an estimated sensitivity of 83% (13).

Additional covariates were drawn from a literature review of potential risk factors for mortality in hospitalised patients, geriatric patients and patients with sepsis. These covariates included age (14), sex (15), race (16,17), Diagnosis Related Group (DRG) weight (a marker of resource intensity of the acute treatment) and key laboratory markers.

The first laboratory result from the index admission was obtained for albumin, sodium, white blood cell (WBC) count, haematocrit and creatinine. The eGFR for kidney function on admission and for chronic kidney function was calculated using the Modification of Diet in Renal Disease equation (18).

Excluded from analysis were covariates that were initially considered but for which fewer than 75% of subjects had data: ethnicity (as distinct from race), malnutrition diagnosis, C-reactive protein, troponin and total and HDL cholesterol. Data on functional status (2,19), presence of ulcer on admission and ulcer stage were not available in our database.

Statistical analysis

Analyses were performed in Stata 10·0. Univariate description of the population was performed for comorbidity and laboratory variables to determine prevalence and check for missing values.

Cox proportional hazards models were developed for mortality at 90 days and at 90- to 180-days. The decision to build separate models for these time periods was based on the observation that the graph of Kaplan–Meier survival estimates had a decreasing slope over time, and on the hypothesis that mortality risk factors (such as systemic infection or fever on admission) may differ during and in the months immediately after hospitalisation, compared with an intermediate-term timeframe. In all analyses, the models for 90- to 180-day mortality were limited to patients who survived beyond 90 days. The Efron method was used for instances where multiple subjects died on the same day (20). Variables initially included in the models included age, sex, race and all variables with Wald statistic $P < 0·20$ on bivariate survival analyses. The models were refined by comparing models after removing each variable with $P \geq 0·05$ and after adding back each variable removed in the first steps. For each variable removed or added,

[†]ICD-9 codes 038, 038·0, 038·1, 038·11, 038·19, 038·2, 038·3, 038·4, 038·40, 038·41, 038·42, 038·43, 038·44, 038·49, 038·8, 038·9, 780·6, 785·52, 790·7, 995·90, 995·91, 995·92.

the effects on log likelihood ratio, Wald statistics, hazard ratios and confidence intervals were manually reviewed. Variables were left in the models if the Wald statistic or likelihood ratio test showed $P < 0·05$, if the hazard ratio changed by at least 10%, if the P values of the other variables changed by 0·02 or by 10% (whichever was more) or the P value crossed the threshold of 0·05. Age, sex and race were left in all models as key variables. Proportional hazards assumptions were tested with Schoenfeld residuals and log–log plots. For variables that did not meet the proportional hazards assumption, the model was stratified by that variable after confirming that there were no interaction effects for that variable. Specifically, the 90-day model was stratified for malignancy and for sodium level. To assess for confounding effects between variables, each variable was removed from the model to assess for changes in the hazard ratios and P values of the covariates and to assess the likelihood ratio compared with the original model. In the case (the 90-day model) in which 'systemic infection or fever' was included in the final model, interaction effects were explored for 'systemic infection or fever' with each comorbid disease variable and with albumin level. Interaction effects leading to a likelihood ratio of <0·05 were kept in the final model.

This study was approved by the Institutional Review Board of Montefiore Medical Center.

Results

Univariate and bivariate analyses

Inclusion criteria were met by 6296 patients. Cumulative mortality after the index admission date was 44·6% at 90 days and 54·4% at 180 days. Of patients who survived 90 days, the cumulative mortality at 180 days was 17·8%.

Demographic variables, comorbid conditions and laboratory values are shown in Table 1. Despite the wide age range, 78·2% of subjects were aged 65 years and above.

Unadjusted (bivariate) associations of each variable with 90-day and with 90- to 180-day mortality are shown in Table 2.

Multivariate models for 90-day mortality and for 90- to 180-day mortality

The adjusted Cox proportional hazards models for 90-day mortality and for 90- to 180-day mortality are shown in Table 3.

Comorbid conditions that were not significantly associated with mortality in either model included dementia, hemiplegia/paraplegia, rheumatic disease, chronic pulmonary disease and peripheral vascular disease. In addition, peptic ulcer was significant only in the 90- to 180-day model, and in this case with a P value of 0·045. This was not a strong indicator of significance in this study with multiple comparisons. Black race, glucose and GFR on admission were also not significant for either time frame.

Variables significant in both models included age, sex, Hispanic race, diabetes, chronic renal failure, congestive heart failure, metastatic cancer and albumin level. Variables significantly associated with mortality in the shorter (90-day)

Table 1 Population characteristics (median and range for continuous variables; percentage for categorical variables)

Variable	N= 6296
Demographic characteristics	
Age (years)	78 (20–108)
Sex	
Female	59·2%
Male	40·8%
Race	
White	42·2%
Black	36·0%
Hispanic	16·3%
Other race	5·6%
Clinical characteristics	
DRG weight (2007)	1·58 (0·51–23·11)
Systemic infection or fever during index admission	40·0%
Laboratory values	
Albumin (g/dl)	3·1 (0·7–5·4)
White blood cells (k/μl)	11·5 (0·2–313·2)
Haematocrit (%)	34·0 (9·4–65·0) 138
Sodium (mEq/l)	(95–194)
Glucose (mg/dl)	128 (1–2307)
Estimated glomerular filtration rate on admission (ml/min/1·73 m^2)	55·2 (1·67–1362·8)
Comorbid disease	
Diabetes	92·8%
Congestive heart failure	40·6%
Chronic renal failure	31·4%
Any liver disease	11·0%
Human immunodeficiency virus/AIDS	3·4%
Dementia	12·9%
Cerebrovascular disease	18·3%
Hemiplegia or paraplegia	5·3%
Myocardial infarction	10·1%
Peripheral vascular disease	12·3%
Chronic pulmonary disease	21·6%
Rheumatologic disease	2·3%
Peptic ulcer disease	3·3%
Any malignancy	12·3%
Metastatic solid tumour	6·8%

time frame only included systemic infection or fever, myocardial infarction, cerebrovascular disease, liver disease, HIV, WBC count and haematocrit. The haematocrit variable showed increasing mortality with increasing haematocrit, which is counterintuitive. The hazard ratio very close to 1·00 suggests that this relationship is not significant. Variables significantly associated with mortality only in the longer (90- to 180-day) time frame included DRG weight.

When checking for confounding effects in the 90-day model, removal of the age variable resulted in HIV becoming non-significant. Removal of 'systemic infection or fever' caused a decrease in the P value for WBC count from 0·028 to <0·001, with a similar hazard ratio. Removal of the albumin variable resulted in haematocrit becoming protective instead of increasing mortality, but hazard ratios were very near 1·00; in addition, the hazard ratio for HIV increased to 1·57 with $P < 0·001$. Removal of congestive heart failure made the P value for WBC count non-significant (from 0·028 to 0·073),

with a similar hazard ratio. Removal of chronic renal disease increased the P values for HIV from 0·007 to 0·028 and for WBC count from 0·028 to 0·093, both with similar hazard ratios.

When checking for confounding effects in the 90- to 180-day model, removal of the albumin variable increased the hazard ratio of metastatic cancer from 1·53 to 1·69, decreasing the P value from 0·024 to 0·004. Not surprisingly, a relationship was also noted between 'any malignancy' and metastatic cancer, with increase in the hazard ratio and decrease in the P value for each when the other was removed. The P value for peptic ulcer crossed above the threshold of $P = 0·050$ when age, race, DRG weight or chronic renal disease variables were removed; however, as the original P value was 0·045, this was not considered of statistical importance. Removal of the age variable increased the P value of metastatic cancer from 0·024 to 0·050 and decreased that of black race from 0·727 to 0·177; again, this was of no evident clinical importance.

'Systemic infection or fever' was included in the final model for 90-day mortality, and interaction effects were explored between this variable and each comorbid disease, as well as with albumin. The only interaction effect with P values from the Wald statistic and likelihood ratio <0·05 was that between sepsis and metastatic cancer, which was included in the model. Including this interaction effect created only negligible changes in the hazard ratios and P values of the other variables, compared with the model without interaction effects.

Discussion

In pressure ulcer patients, survival time may affect the risk–benefit balance for interventions that improve wound healing at the cost of procedure-related morbidity and potential adverse effects. In this population with high overall mortality rates, an epidemiologic perspective on the role of comorbid disease and other patient characteristics in mortality can inform clinical decision making and counselling of patients and families.

The prevalence of comorbid disease in pressure ulcer patients may vary greatly between settings – for example, outpatients in a spinal cord centre may have less comorbid disease than this inpatient cohort and a much lower overall mortality rate. Therefore, clinical research on pressure ulcer patients should be conducted and interpreted with care to avoid inappropriate extrapolation of conclusions between very different types of patients. Our study sheds light on mortality in hospitalised patients with pressure ulcers, but may not be generalisable to pressure ulcer patients identified in other settings, such as nursing homes or home care.

Our analysis shows that not all variables associated with mortality in other populations are associated with mortality in the pressure ulcer population. For example, hemiplegia/paraplegia and dementia did not impact mortality at 90 days or 180 days in adjusted analyses. This suggests that – in the absence of cerebrovascular disease, which was controlled for – neurologic lesions alone do not affect mortality within 180 days. The same holds true for chronic pulmonary disease, rheumatic disease and peripheral vascular disease. On hospital

Table 2 Unadjusted mortality risk at 90 days and at 90- to 180-days from admission for inpatients with pressure ulcers

Variables	90-day mortality risk Unadjusted hazard ratio	90- to 180-day mortality risk Unadjusted hazard ratio
Demographic characteristics		
Age (years)	1·02 (1·01–1·02), P < 0·001	1·01 (1·01–1·02), P < 0·001
Male sex	1·10 (1·02–1·18), P = 0·016	1·27 (1·09–1·49), P = 0·003
Black race*	0·86 (0·79–0·94), P = 0·001	0·92 (0·77–1·10), P = 0·341
Hispanic race*	1·12 (1·02–1·24), P = 0·024	1·60 (1·30–1·97), P < 0·001
Clinical characteristics		
DRG weight	1·02 (1·01–1·02), P = −0·001	1·06 (1·04–1·07), P < 0·001
Systemic infection or fever	1·66 (1·55–1·79), P < 0·001	1·37 (1·16–1·61), P < 0·001
Laboratory values		
Albumin (g/dl)	0·57 (0·54–0·60), P < 0·001	0·69 (0·61–0·78), P < 0·002
White blood cells (k/μl)	1·01 (1·01–1·01), P < 0·001	1·01 (1·00–1·01), P = 0·228
Haematocrit (%)	0·99 (0·98–0·99), P < 0·001	0·98 (0·97–1·00), P = 0·008
Sodium (mEq/l)	1·01 (1·01–1·01), P < 0·001	1·00 (1·00–1·01), P = 0·985
Glucose (mg/dl)	1·00 (1·00–1·00), P = 0·106	1·00 (1·00–1·00), P = 0·543
Estimated glomerular filtration rate on admission (ml/min/1·73 m^2)	1·00 (1·00–1·00), P < 0·001	1·00 (1·00–1·00), P = 0·049
Comorbid disease		
Diabetes	1·32 (1·13–1·54), P = 0·001	1·65 (1·16–2·34), P = 0·005
Congestive heart failure	1·54 (1·43–1·66), P < 0·001	1·46 (1·25–1·71), P < 0·001
Chronic renal failure	1·48 (1·37–1·60), P < 0·001	1·39 (1·18–1·65), P < 0·001
Any liver disease	1·30 (1·17–1·45), P < 0·001	0·89 (0·67–1·18), P = 0·408
Human immunodeficiency virus/AIDS	0·80 (0·64–0·99), P = 0·045	0·76 (0·48–1·21), P = 0·250
Dementia	1·27 (1·05–1·30), P = 0·003	0·98 (0·77–1·26), P = 0·892
Cerebrovascular disease	1·10 (1·00–1·21), P = 0·046	1·03 (0·84–1·27), P = 0·780
Hemiplegia/paraplegia	0·63 (0·52–0·77), P < 0·001	0·71 (0·49–1·02), P = 0·065
Myocardial infarction	1·41 (1·26–1·58), P < 0·001	1·22 (0·93–1·59), P = 0·154
Peripheral vascular disease	0·97 (0·87–1·10), P = 0·658	1·11 (0·88–1·40), P = 0·379
Chronic pulmonary disease	1·12 (1·02–1·22), P = 0·013	1·14 (0·94–1·38), P = 0·171
Rheumatic disease	1·06 (0·83–1·35), P = 0·621	1·35 (0·84–2·15), P = 0·214
Peptic ulcer	1·16 (0·96–1·41), P = 0·125	1·57 (1·06–2·33), P = 0·024
Any malignancy	1·60 (1·45–1·77), P < 0·001	1·81 (1·44–2·28), P < 0·001
Metastatic solid tumour	1·91 (1·68–2·16), P < 0·001	1·96 (1·43–2·67), P < 0·001

*Hazard ratios for race were compared with patients with 'White' or 'Other' race.

admission, glucose level (when controlling for diabetes) and renal function (when controlling for chronic renal disease) were not significant markers of mortality risk.

Comorbid diseases associated with increased mortality showed hazard ratios less than 2 (the higher hazard ratio for metastatic cancer in the 90-day model cannot be directly interpreted, as it is included in an interaction effect). In patients with multiple comorbidities, however, these risks would be multiplicative. Albumin level was a strong marker of mortality in patients with pressure ulcers. Although albumin level is not accurate in determining a patient's nutritional status (21), this laboratory value still may be important in the work-up of pressure ulcer patients as a marker of overall mortality.

'Systemic infection or fever' significantly affected 90-day mortality. Despite our expectation that there would be confounding or interaction effects between 'systemic infection or fever' and comorbid diseases such as diabetes or renal failure, this was not shown in our data. The sole interaction effect meeting the threshold of significance to P < 0·05 was with metastatic cancer. No confounding effects were noted. Previous publications have suggested that comorbid disease plays a role in differential mortality rates in patients with infections (22,23). However, data on comorbid disease and mortality in septic patients have been limited by use of unadjusted analyses (24,25) or by analyses examining only in-hospital or short-term mortality (26,27).

This study has limitations. Administrative data are prone to undercoding (28). This may have affected how representative our cohort was of all hospitalised patients with pressure ulcers, as providers and coders may be more likely to assign pressure ulcer codes to certain types of patients. In addition, comorbid diseases may have been underdetected in administrative data (29). For comorbid disease variables, undercoding in administrative data was mitigated when possible by using laboratory data to capture patients with specific comorbidities. Several potentially important variables, including functional status, presence of ulcer on admission and ulcer stage were not in the models because the data were unavailable. This dataset therefore could not be used to explore whether mortality predictors were influenced by ulcer stage or by whether the ulcer was hospital-acquired versus present on admission. The data could not show whether systemic infection or fever was related to a pressure ulcer or to another source. The absence of these variables impedes the assessment of generalisability of our results to other inpatient pressure ulcer populations which may have different distributions of these characteristics.

Despite these limitations, this study's results should be considered when counselling patients with pressure ulcers and

Table 3 Adjusted mortality risk at 90 days and at 90- to 180-days from admission for inpatients with pressure ulcers, by Cox proportional hazard analysis

Variables	90-day mortality risk Adjusted hazard ratio N = 5870	90- to 180-day mortality risk Adjusted hazard ratio N = 3204
Demographic characteristics		
Age (years)	1·02 (1·02–1·02), $P < 0.001$	1·02 (1·01–1·12), $P < 0.001$
Male sex	1·15 (1·06–1·24), $P = 0.001$	1·37 (1·16–1·62), $P < 0.001$
Black race*	0·92 (0·84–1·01), $P = 0.081$	0·97 (0·80–1·17), $P = 0.799$
Hispanic race*	1·13 (1·01–1·26), $P = 0.032$	1·61 (1·30–2·00), $P < 0.001$
Clinical characteristics		
DRG weight	–	1·06 (1·04–1·08), $P < 0.001$
Systemic infection or fever	1·50 (1·37–1·63), $P < 0.001$	–
Laboratory values		
Albumin (g/dl)	0·57 (0·53–0·60), $P < 0.001$	0·69 (0·61–0·79), $P < 0.001$
White blood cells (k/μl)	1·00 (1·00–1·01), $P = 0.046$	–
Haematocrit (%)	1·01 (1·01–1·02), $P = 0.001$	–
Sodium (mEq/l)**	–	–
Glucose (mg/dl)	–	–
Estimated glomerular filtration rate on admission (ml/min/1·73 m^2)	–	–
Comorbid disease		
Diabetes	1·31 (1·12–1·55), $P = 0.001$	1·63 (1·14–2·34), $P = 0.007$
Congestive heart failure	1·35 (1·25–1·47), $P < 0.001$	1·26 (1·07–1·50), $P = 0.007$
Chronic renal failure	1·46 (1·35–1·59), $P < 0.001$	1·34 (1·12–1·60), $P = 0.001$
Any liver disease	1·21 (1·07–1·36), $P = 0.002$	–
Human immunodeficiency virus/AIDS	1·40 (1·09–1·79), $P = 0.008$	–
Dementia	–	–
Cerebrovascular disease	1·17 (1·06–1·29), $P = 0.002$	–
Hemiplegia/paraplegia	–	–
Myocardial infarction	1·35 (1·20–1·52), $P < 0.001$	–
Peripheral vascular disease	–	–
Chronic pulmonary disease	–	–
Rheumatic disease	–	–
Peptic ulcer	–	1·50 (1·01–2·23), $P = 0.045$
Any malignancy**	–	1·62 (1·23–2·12), $P < 0.001$
Metastatic cancer	2·48 (2·06–2·99), $P < 0.001$	1·53 (1·06–2·22), $P = 0.024$
Interaction terms		
Systemic infection/fever – metastatic cancer	0·54 (0·41–0·72), $P < 0.001$	

*Hazard ratios for race were compared with patients with 'White' or 'Other' race. **90-day model: stratified for any malignancy and for sodium.

their families about the patient's overall prognosis and its relationship to pressure ulcer treatment options. Understanding the effects of comorbid disease on hospitalised pressure ulcer patients is part and parcel of caring for this complex population. Further study is required to understand heterogeneity within this population in order to tailor treatment plans to patients with different overall clinical scenarios.

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