International Journal of Gerontology 19 (2025) 115–120
https://doi.org/10.6890/IJGE.202504_19(2).0010

International Journal of Gerontology

journal homepage: http://www.sgecm.org.tw/ijge/

Original Article

Determinants of Mortality in Geriatric Palliative Care Patients with Pressure Injuries

Mursel Karadavut [a], Busra Akpınar [a], Pınar Tosun Tasar [a *], Omer Karasahin [b], Mustafa Utlu [a], Taha Burak Urvasızoglu [c], Mehmet Batuhan Duman [c]

[a] *Division of Geriatrics, Department of Internal Medicine, Ataturk University Hospital, Erzurum, Turkey,* [b] *Infectious Diseases Clinic, Erzurum Regional Training and Research Hospital, Erzurum, Turkey,* [c] *Department of Internal Medicine, Ataturk University Hospital, Erzurum, Turkey*

ARTICLE INFO

Accepted 28 November 2024

Keywords:
palliative care,
pressure injury,
mortality

SUMMARY

Background: Our study aimed to identify factors associated with mortality in geriatric palliative care patients with pressure injuries (PI).
Methods: This prospective observational study included patients with PI who were hospitalized in a palliative care unit. Demographic characteristics, chronic diseases, and number of diseases were recorded, and each patient was assessed using the Barthel Index (BI) and Mini Nutritional Assessment (MNA). All patients were also staged according to the Braden scale, Norton scale, and European Pressure Ulcer Advisory Panel.
Results: A total of 92 patients who were treated in the palliative care unit and had PI during the study period were evaluated. Their mean age was 74 years and 53.3% were male. Forty-two patients (45.7%) died during hospital follow-up, and 14 patients (15.2%) died within the first 30 days. In the adjusted Cox proportional hazard analysis, thrombocytopenia, anemia, elevated C-reactive protein, and elevated procalcitonin were significantly associated with increased mortality. Being single or widowed, having diabetes mellitus, and the presence of thrombocytopenia were found to be independent risk factors for mortality, while receiving antibiotic therapy was found to be a protective factor.
Conclusion: Diabetes mellitus, marital status and thrombocytopenia were identified as independent risk factors for mortality in patients hospitalized for PI in the palliative care unit.

1. Introduction

Life expectancy has gradually increased worldwide.[1] Longer life expectancy brings increased frequency and duration of hospitalization and a greater need for palliative care centers.[2]

Palliative care patients are exposed to various factors that lead to deterioration of physiological function and autonomy during the course of their disease.[3] These people often become bedridden at the end of life due to required positioning and sedative treatment, all of which lead to an increase in pressure injuries (PI).[4]

In addition, xeroderma, malnutrition, and end-stage disease also contribute to the development of PI.[3] PI can cause many complications such as pain, depression, and infection. The presence of these complications leads to further deterioration of health, prolonged healing and hospital stays, increased health expenditures, and early mortality.[5]

In our palliative care center, all patients and families are informed about PI regardless of whether the patient has PI at admission. One of the most important PI prevention strategies is to evenly distribute and reduce the pressure on the patient. The most effective method to reduce the intensity and duration of pressure is positioning, and specialized pads that reduce or relieve pressure can be used. The pressure exerted by medical devices such as masks, tubes, and catheters can be reduced by changing their position and applying prophylactic wound dressing where there is skin contact. Water-based moisturizers should be applied at least once a day, especially for patients with very dry skin, and a barrier cream can be used with older patients at risk of moisture-related skin damage. Patients with incontinence should be reminded to go to the toilet at regular intervals, and for patients using diapers, the perineal and sacral areas should be cleaned carefully. After the development of PI, care is based on the use of appropriate wound care products and pressure reduction. Isotonic saline solution is often utilized in wound care, while strong wound cleansers such as betadine and hydrogen peroxide are not used. In the presence of necrotic tissue, debridement is beneficial.

The incidence of PI among hospital inpatients varies between 4% and 38%; the rate of mortality associated with PI depends on existing comorbidities, especially in older patients, and can be up to 68%.[6] The presence of PI in ventilator-dependent patients in the intensive care unit is an independent risk factor for mortality.[7] The most important predictors of PI are neurodegenerative conditions such as dementia and stroke, and other chronic diseases such as cardiovascular disease and diabetes mellitus (DM). The most common factors precipitating PI are low body mass, high inflammatory biomarkers, low cardiac output, immobilization, and hemodynamic instability such as hypotension.[8,9]

* Corresponding author. Division of Geriatrics, Department of Internal Medicine, Ataturk University Hospital, Izmir, Turkey.
E-mail address: pinar.tosun@gmail.com (P. Tosun Tasar)

Although the prevention of PI is a very important goal, it is often unavoidable in patients receiving palliative care.[10,11] In patients for whom wound healing is not fully possible, the aim should be to stabilize and prevent further worsening of the wound.[12,13] In addition, assessing risk factors to predict the prognosis of patients with PI will play a key role in selecting an appropriate treatment and care strategy.[12,14,15] Therefore, our study aimed to determine the factors associated with mortality in geriatric patients with PI receiving palliative care.

2. Materials and methods

This prospective observational study included patients with PI who were hospitalized in the palliative care unit of a tertiary university hospital between January 1, 2021 and June 1, 2024. Inclusion criteria were being a geriatric patient, being admitted for PI, and staying in the unit for 24 hours or more. Exclusion criteria were being hospitalized for less than 24 hours and repeated admission to the palliative care unit.

The patients' demographic characteristics, chronic diseases, number of diseases, medications used, where the patients were before admission to palliative care, their height, weight, and body mass index (BMI) at admission to palliative care, mode of nutrition intake, and presence of urinary incontinence and urinary catheter were recorded.

The Barthel Index (BI) was also calculated at the time of hospital admission. The BI was first applied by Mahoney and Bartel in 1965.[16] The Turkish validity and reliability studies of the BI were conducted by Küçükdeveci et al. in 2000 in patients with stroke and spinal cord injuries.[17]

The nutritional status of the patients was evaluated using the Full Mini Nutritional Assessment (MNA).[18] Patients with PI were staged according to the Braden scale, Norton scale, and European Pressure Ulcer Advisory Panel (EPUAP). The Braden scale comprises 6 subheadings: sensory perception, humidity, mobility, physical activity, nutrition, friction, and shearing.[19] In the Norton scale, 5 risk factors (physical status, mental status, activity status, mobility, and incontinence) are rated between 1 and 4, with a total score of 5–20.[20]

According to the EPUAP grading system, PIs have four stages. In stage 1, the skin is intact with non-blanchable erythema, usually over a bony protrusion. In stage 2, there is partial loss of the dermis, with a pink/red wound bed and a superficial ulcer without necrosis. In stage 3, there is full-thickness skin loss and exposed subcutaneous adipose tissue but no exposure of bone, tendon, or muscle. In stage 4, there is full-thickness tissue loss with exposed bone, tendon, or muscle.[4]

Blood, urine, superficial scrape samples, and especially deep tissue samples obtained from wounds that underwent surgical debridement were obtained for culture upon admission to the unit. Biomarker results obtained on the first day of hospitalization, PI location, treatments received while hospitalized, length of hospital stay, and outcome were recorded.

2.1. Statistical analysis

Descriptive statistics are presented as median and range for nonparametric continuous data. Categorical data were presented as frequency and percentage and were compared using the chi-square test. The Mann-Whitney U test was used to evaluate differences in score values. A probability (p) value of less than 0.05 was considered statistically significant. Additional adjusted curves were plotted for risk factors associated with mortality. Cox proportional hazards analysis with and without adjustments for age and sex was used to determine the hazard ratio for mortality in individuals with PI. A multivariate Cox regression model was created with variables found to be significant in the adjusted Cox proportional hazards analysis: being single/widowed, comorbid DM, antibiotic use, hospital onset of PI, thrombocytopenia, anemia, C-reactive protein (CRP) elevation, and procalcitonin elevation. To estimate mortality risk, a survival curve was first obtained by the Kaplan-Meier method. Cut-off values for biomarkers were obtained from the literature.[21] ROC analysis was performed to determine cut-off points for biomarkers if not included in the literature. The cut-off value with the highest sensitivity, specificity, and diagnostic power were determined using the Youden J index. To identify independent factors associated with mortality in our patient group, all predictive variables with a p-value of < 0.005 were entered into the proportional hazards model. All statistical analyses were performed using SPSS 21.0.

Approval to conduct the study was obtained from the Atatürk University Faculty of Medicine Clinical Research Ethics Committee (date: 17/12/2020, meeting no: 10, decision no: 21).

3. Results

A total of 92 patients were evaluated. Their mean age was 74 (65–99) years and 49 (53.3%) were male. The mean length of hospital stay was 41 (2–550) days. Sixty-five patients (70.7%) had a single PI and 27 patients (29.3%) had PI at multiple sites. The most common site was the sacrum (n = 88, 95.4%). The distribution of PI locations is presented in Figure 1.

Mortality occurred in 42 patients (45.7%) during the hospital follow-up period, of which 14 patients (15.2%) died within the first 30 days. The sociodemographic and clinical characteristics of the patients with and without 30-day mortality are presented in Table 1. In the adjusted Cox proportional hazard analysis, being single or widowed and having DM increased the risk of mortality, while antibiotic use and in-hospital onset of PI reduced the risk (Table 1). In addition, 40 (43.5%) of the patients had PI infection, and polymicrobial infection was detected in 19 patients (19.6%). The most frequently isolated microorganisms were *Klebsiella pneumonia* (n = 15, 16.3%), *Pseudomonas aeruginosa* (n = 8, 8.6%), and *Escherichia coli* (n = 7, 7.6%).

Risk scores and biomarker levels at admission in patients with and without 30-day mortality are presented in Table 2. In the adjusted Cox proportional hazard analysis, thrombocytopenia, anemia, elevated CRP, and elevated procalcitonin were significantly associ-

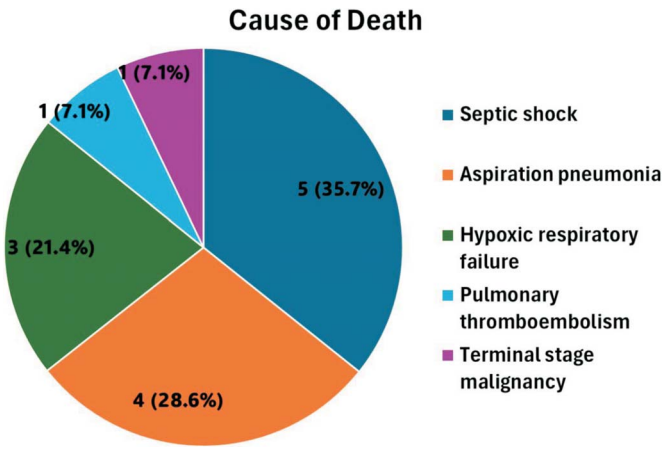

Figure 1. Causes of death.

Table 1
Comparison of patient characteristics and comorbidities according to 30-day mortality.

	30-day mortality		Unadjusted hazard ratio (95% CI)	p value	Adjusted hazard ratio (95% CI)[a]	p value
	Yes (n = 14)	No (n = 78)				
Age (years), median (range)	71 (66–92)	75 (65–99)	0.97 (0.92–1.04)	0.519	0.98 (0.91–1.03)	0.353[b]
Gender, n (%), female	9 (64.3)	34 (43.6)	2.28 (0.76–6.80)	0.140	2.49 (0.82–7.35)	0.104[c]
Body mass index, n (%), ≥ 25 kg/m^2	6 (42.9)	22 (28.2)	1.77 (0.61–5.12)	0.290	1.40 (0.45–4.32)	0.550
Marital status, n (%)						
Married (reference)	3 (21.4)	48 (61.5)				
Single/widowed	11 (76.6)	30 (38.5)	5.64 (1.56–20.30)	**0.008**	6.60 (1.69–25.63)	0.006
Place of residence, n (%)						
Home (reference)	13 (92.9)	74 (94.9)				
Nursing home	1 (7.1)	4 (5.1)	0.94 (0.12–7.13)	0.954	1.33 (0.17–10.60)	0.784
Where PI occurred, n (%)						
Home (reference)	10 (71.4)	25 (32.1)				
Nursing home	-	5 (6.4)	0.01	0.987	0.01	0.981
Hospital	4 (28.6)	48 (61.5)	0.20 (0.06–0.65)	**0.008**	0.17 (0.05–0.57)	0.004
Comorbidities, n (%)						
HT	11 (78.6)	39 (50.0)	3.18 (0.88–11.42)	0.076	2.86 (0.78–10.43)	0.110
DM	9 (64.3)	19 (24.4)	5.44 (1.81–16.29)	**0.002**	5.03 (1.66–15.23)	0.004
COPD	4 (28.6)	11 (14.1)	2.50 (0.78–8.02)	0.122	2.22 (0.69–7.17)	0.182
CKD	2 (14.3)	4 (5.1)	3.10 (0.68–14.03)	0.141	3.24 (0.69–15.14)	0.135
CVD	3 (21.4)	36 (46.2)	0.35 (0.99–1.27)	0.114	0.32 (0.08–1.19)	0.090
Dementia	5 (35.7)	32 (41.0)	0.72 (0.24–2.15)	0.557	0.73 (0.24–2.21)	0.586
Parkinson's	2 (14.3)	7 (9.0)	1.33 (0.29–5.96)	0.706	1.61 (0.35–7.44)	0.536
Postoperative immobilization	2 (14.3)	10 (12.8)	1.01 (0.22–4.51)	0.993	0.92 (0.20–4.23)	0.923
Malignancy	1 (7.1)	20 (25.6)	0.23 (0.03–1.82)	0.167	0.23 (0.03–1.76)	0.157
Mode of nutrition, n (%)						
Parenteral	3 (21.4)	10 (12.8)	1.53 (0.42–5.53)	0.509	2.20 (0.56–8.53)	0.254
Oral	13 (92.9)	51 (65.4)	6.61 (0.86–50.64)	0.069	6.00 (0.72–49.40)	0.096
PEG tube	-	23 (29.5)	0.29 (0.01–4.19)	0.163	0.17 (0.02–1.35)	0.095
Infectious disease, n (%)						
Bloodstream infection	2 (14.3)	7 (9.0)	1.63 (0.36–7.32)	0.519	1.94 (0.42–8.93)	0.392
Urinary tract infection	6 (42.9)	19 (24.4)	2.15 (0.74–6.20)	0.156	2.44 (0.83–7.17)	0.104
PI infection	4 (28.6)	36 (46.2)	0.45 (0.14–1.46)	0.187	0.53 (0.16–1.71)	0.284
Polymicrobial PI infection	4 (28.6)	14 (17.9)	1.62 (0.50–5.20)	0.495	1.64 (0.51–5.27)	0.400
Treatments received, n (%)						
Surgical debridement	1 (7.1)	19 (24.4)	0.23 (0.03–1.79)	0.161	0.26 (0.03–2.02)	0.199
Negative pressure wound dressing	1 (7.1)	21 (26.9)	0.19 (0.02–1.48)	0.113	0.21 (0.03–1.67)	0.142
Antibiotic therapy	12 (85.7)	77 (98.7)	0.09 (0.01–0.42)	**0.002**	0.05 (0.02–0.44)	0.004
Antipseudomonal beta lactam	67 (85.9)	7 (50.0)	0.16 (0.05–0.56)	**0.004**	0.20 (0.07–0.61)	0.005
Carbapenem	53 (67.9)	6 (42.9)	0.35 (0.11–1.13)	0.079	0.40 (0.13–1.12)	0.121
Glycopeptide	14 (17.9)	-	-	-	-	-
Aminoglycoside or polymyxin	6 (7.7)	-	-	-	-	-
Red cell transfusion	8 (57.1)	50 (64.1)	0.59 (0.20–1.73)	0.340	0.66 (0.22–1.97)	0.465

CI, confidence interval; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; CVD, cerebrovascular disease; DM, diabetes mellitus; HT, hypertension; PEG, percutaneous endoscopic gastrostomy, PI, pressure injury.
Antipseudomonal beta lactam (meropenem, imipenem, piperacillin tazobactam, ceftazidime, cefepime), Carbapenem (imipenem, meropenem, ertapenem).
[a] Adjusted for age and sex. [b] Adjusted for sex. [c] Adjusted for age.

Table 2
Risk scores and biomarkers levels at admission according to 30-day mortality.

	30-day mortality		Unadjusted hazard ratio (95% CI)	p value	Adjusted hazard ratio* (95% CI)	p value
	Yes (n = 14)	No (n = 78)				
Biomarker, n (%)						
Hypoalbuminemia (albumin < 2.5 g/dL)	7 (50.0)	30 (38.5)	1.55 (0.54–4.44)	0.407	1.52 (0.53–4.36)	0.428
Creatinine > 1.2 mg/dL	12 (14.0)	5 (35.7)	2.84 (0.95–8.50)	0.061	2.36 (0.76–7.26)	0.134
Anemia (Hg < 12 g/dL)	13 (92.9)	51 (65.4)	6.52 (0.85–50.07)	0.071	8.77 (1.12–67.31)	**0.038**
Neutrophilia (neutrophils > 11 × 10^3/μL)	5 (35.7)	18 (23.1)	2.10 (0.67–6.04)	0.210	2.56 (0.82–8.00)	0.105
Lymphopenia (lymphocytes < 1.5 × 10^3/μL)	11 (78.6)	49 (62.8)	2.35 (0.64–8.77)	0.192	2.63 (0.71–9.72)	0.145
Thrombocytopenia (platelets < 150 × 10^3/μL)	5 (35.7)	9 (11.5)	3.42 (1.14–10.24)	**0.028**	3.81 (1.26–11.55)	**0.018**
CRP > 79.75 mg/dL	12 (85.7)	41 (52.6)	4.41 (0.98–19.73)	0.052	4.89 (1.07–22.16)	**0.040**
PCT > 0.32 (ng/mL)	11 (78.6)	34 (43.6)	4.20 (1.16–15.09)	**0.028**	4.69 (1.25–17.53)	**0.022**
Risk scores, n (%)						
MNA score ≤ 7 (malnourished)	9 (64.3)	65 (83.3)	0.41 (0.13–1.23)	0.114	0.53 (0.17–1.69)	0.289
Braden score ≤ 12 (high-risk group)	10 (71.4)	47 (60.3)	1.50 (0.47–4.80)	0.490	1.96 (0.59–6.46)	0.266
Barthel index < 40 (very dependent)	13 (92.9)	72 (92.3)	1.05 (0.14–8.09)	0.961	1.43 (0.18–11.34)	0.732
Norton score < 10 (very high risk)	8 (57.1)	53 (67.9)	0.63 (0.22–1.83)	0.403	0.77 (0.26–2.29)	0.644
EPUAP PI stage, n (%)						
Stage 2 (reference)	8 (57.1)	28 (35.9)				
Stage 3	3 (21.4)	15 (19.2)	0.77 (0.20–2.96)	0.712	0.64 (0.16–2.49)	0.644
Stage 4	3 (21.4)	35 (44.9)	0.30 (0.81–1.15)	0.081	0.31 (0.08–1.18)	0.087

CI, confidence interval; CRP, C-reactive protein; EPUAP; European Pressure Ulcer Advisory Panel; Hg, hemoglobin; MNA, Mini Nutritional Assessment, PCT, procalcitonin; PI, pressure injury.
* Adjusted for age and sex.

ated with increased mortality. Receiving antibiotic treatment was an independent protective factor for mortality. The Cox proportional hazard analysis model for mortality in patients with PI is presented in Table 3 (Figure 2).

4. Discussion

Preventing or managing PI involves not only appropriate assessment tools but also the development of preventive strategies and the education of family and staff.

Palliative patients with PI often have delayed healing because of immune dysfunction, biochemical abnormalities, physiological stress, systemic and local hypoxia, and critical or end-stage disease. In addition, these patients commonly receive steroids for symptomatic treatment and chemotherapeutics for malignancy.[22] Hypotension or dehydration may also impair blood circulation to the PI, and excessive humidity and incontinence increases the risk of PI infection.[23] These are all important precipitating factors of mortality in palliative care units, where PI is a common struggle.

In a study of geriatric patients hospitalized for PI, the mortality rate was shown to be 15.2%.[24] A 2019 meta-analysis by Song et al.[25] showed that mortality was twice as high in patients with PI compared to those without. In a study including 684 patients admitted to a geriatric unit, during 12 weeks of follow-up the prevalence of PI was found to be 15.5% and the mortality rate was 66%.[21] In our sample of 92 geriatric patients with PI treated in a palliative care unit, 45.7% died during hospital follow-up and 15.2% died within the first 30 days of admission to palliative care. Being single or widowed, having DM, and the presence of thrombocytopenia were identified as independent risk factors associated with increased mortality.

The prevalence of DM has steadily increased in recent decades. Sensory loss in diabetic neuropathy is a risk factor for diabetic foot and PI. Diabetic neuropathy can also lead to Charcot foot, which causes bone destruction, deformity, and infection.[26] With longer duration of DM, there is increased glycosylation in the vessel walls, leading to impaired perfusion of the skin and local ischemia. Most diabetic patients develop vascular complications that lead to damage in the target organs.[27] Oral antidiabetic drugs that cause hypoglycemia result in decreased physical activity and often lead to weight gain. The risk of infection is also increased in diabetic patients. In the literature, DM is the most commonly identified disease in patients with PI, and DM has been clearly shown to increase the risk of developing PI. Lyder et al.[28] detected diabetes in 42.2% of 2313 patients with PI, while Margolis et al.[29] reported that patients with DM had a 1.75 times higher risk of developing PI. Other contributors to PI in the feet of diabetic patients include dry skin[30] and hard, thinner tissue.[31] Moreover, diabetes is associated with obesity, and hospital inpatients with morbid obesity were shown to be at higher risk of PI.[32] The direct relationship between the presence of DM and organ damage and infection is also expected to increase the risk of mortality in these patients. In the present study, the presence of DM was an independent risk factor associated with a 7.48-fold increase in mortality.

Care services consist of formal care provided by public and private institutions, as well as the informal care provided by family, neighbors, and friends.[33] In the literature, higher mortality has been

Table 3
Cox proportional hazard analysis model for mortality in patients with pressure injury.

Variable	Hazard ratio (95% CI)	p value
Marital status: single/widowed	9.305 (1.305–45.261)	**0.006**
Where PI occurred: hospital	0.200 (0.045–0.895)	0.155
Presence of DM	7.476 (1.657–33.724)	**0.009**
Treatments received: antibiotic therapy	0.010 (0.001–0.250)	**0.005**
Anemia (Hg < 12 g/dL)	8.031 (0.834–77.353)	0.071
CRP > 79.75 mg/dL	2.085 (0.386–11.252)	0.393
Presence of thrombocytopenia	5.641 (1.259–25.265)	**0.024**
PCT > 0.32 (ng/mL)	11.253 (1.173–107.959)	0.056

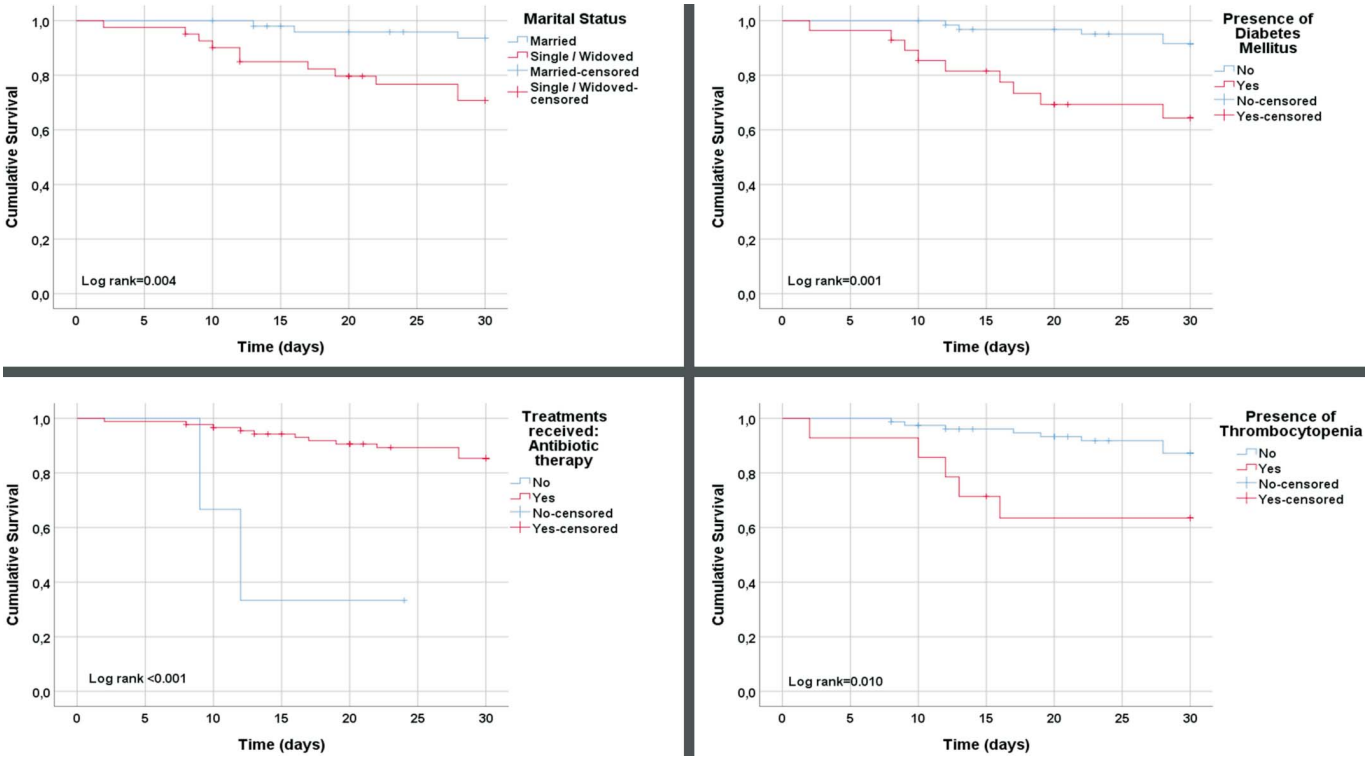

Figure 2. Kaplan-Meier survival curves according to mortality predictors for individuals with pressure injury.

reported in nursing home residents with PI due to immobilization, incontinence, and higher comorbidity.[34] In our study, there was no difference in mortality according to the patients' location before admission to the palliative care unit, which we attribute to the low number of patients coming from nursing homes. However, being single or widowed was found to be an independent risk factor for higher mortality, which may be related to a lack of social support. A study investigating informal caregivers' degree of relationship to patients showed that patients in Taiwan were mostly cared for by their spouses.[35] In contrast, studies by Tuna et al.[36] and Akgün[37] showed that in our country, children generally provide informal care. This can be explained by the traditional Turkish family structure, strong family ties, and the expectation that adult children care for their aging parents.

In patients with infected PI, neutrophil count increases secondary to acute infection. Previous studies have shown that neutrophilia is associated with recurrent ischemic events and vascular death. The authors of these studies suggested infection or systemic inflammatory conditions as possible causes of increased mortality. The higher mortality in patients with stage 4 PI may also be explained by the fact that these PI are more easily infected.[21] In our study, the presence of neutrophilia in patients with PI was not associated with mortality. Thrombocytopenia is an important laboratory marker of a severe systemic response secondary to infectious diseases such as sepsis. This marker can be considered a negative acute phase indicator, and has been identified as an independent mortality marker in multivariate analysis. In addition, it was revealed that platelet counts can be used as a prognostic factor to predict mortality among community-dwelling older people, regardless of the presence of PI.[38] In our study, thrombocytopenia was an independent factor that increased the risk of mortality by 5.64-fold.

Serum albumin levels are affected by inflammation, hydration, wound severity, and disease status.[39] Although hypoalbuminemia has been identified in the literature as a risk factor for PI,[21] no relationship was found between hypoalbuminemia and mortality in our study.

In our study, 43.5% of the patients had infected PI, and polymicrobial infections were detected in 19.6% of those patients. It was reported in the literature that mortality is 3.8 times higher in patients with multiple microorganisms in PI cultures.[40] However, this had no effect on mortality in our study. This difference was explained by the high rate of antibiotic use among our patients. In particular, the use of beta lactam drugs with antipseudomonal activity was protective in terms of mortality. When the causes of death are analyzed, it is seen that infectious diseases and related complications are remarkably high. This suggests that limiting the use of invasive devices and implementing infection control measures will be effective in reducing mortality. It also indicates that the protective effect of antibiotic use against mortality observed in our analysis is a reflection of infectious disease control.

Strengths of our study are that it was prospective and is one of the few studies conducted among patients receiving palliative care treatment in our country. Limitations of our study are that it was conducted in a single center and there was no control group. Furthermore, the duration and complications of comorbid diseases were not questioned, which may be particularly relevant in terms of diabetes control and renal function.

In conclusion, marital status, DM, and thrombocytopenia were identified as independent risk factors for mortality in patients hospitalized for PI in the palliative care unit. These risk factors should be considered to develop an effective care plan to prevent negative outcomes and unnecessary hospitalizations and readmissions.

References

1. World Health Organization. *World health statistics 2016: monitoring health for the SDGs, sustainable development goals*. World Health Organization; 2016.
2. World Health Organization. Strengthening of palliative care as a component of integrated treatment throughout the life course. *J Pain Palliat Care Pharmacother.* 2014;28:130–134. doi:0.3109/15360288.2014.911801
3. Langemo D, Haesler E, Naylor W, Tippett A, Young T. Evidence-based guidelines for pressure ulcer management at the end of life. *Int J Palliat Nurs*. 2015;21:225–232. doi:10.12968/ijpn.2015.21.5.225
4. National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance. *Prevention and Treatment of Pressure Ulcers: Quick Reference Guide*. Cambridge Media; 2014.
5. Allman RM, Goode PS, Burst N, Bartolucci AA, Thomas DR. Pressure ulcers, hospital complications, and disease severity: impact on hospital costs and length of stay. *Adv Wound Care*. 1999;12:22–30.
6. Afzali Borojeny L, Albatineh AN, Hasanpour Dehkordi A, Ghanei Gheshlagh R. The incidence of pressure ulcers and its associations in different wards of the hospital: A systematic review and meta-analysis. *Int J Prev Med*. 2020;11:171. doi:10.4103/ijpvm.IJPVM_182_19
7. Moore ZE, Patton D. Risk assessment tools for the prevention of pressure ulcers. *Cochrane Database Syst Rev.* 2019;1:CD006471. doi:10.1002/14651858.CD006471.pub4
8. Manley S, Mitchell A. The impact of nutrition on pressure ulcer healing. *Br J Nurs.* 2022;31:S26–S30. doi:10.12968/bjon.2022.31.12.S26
9. Arai K, Yamamoto KH, Suzuki T, Mitsukawa N, Ishii I. Risk factors affecting pressure ulcer healing: Impact of prescription medications. *Wound Repair Regen*. 2020;28:409–415. doi:10.1111/wrr.12791
10. Trombley K, Brennan MR, Thomas L, Kline M. Prelude to death or practice failure? Trombley-Brennan terminal tissue injuries. *Am J Hosp Palliat Care*. 2012;29:541–545. doi:10.1177/1049909111432449
11. Edsberg LE, Langemo D, Baharestani MM, Posthauer ME, Goldberg M. Unavoidable pressure injury: state of the science and consensus outcomes. *J Wound Ostomy Continence Nurs*. 2014;41:313–334. doi:10.1097/WON.0000000000000050
12. Maida V, Ennis M, Corban J. Wound outcomes in patients with advanced illness. *Int Wound J.* 2012;9:683–692. doi:10.1111/j.1742-481X.2012.00939.x
13. Alvarez OM, Kalinski C, Nusbaum J, et al. Incorporating wound healing strategies to improve palliation (symptom management) in patients with chronic wounds. *J Palliat Med.* 2007;10:1161–1189. doi:10.1089/jpm.2007.9909
14. Maida V, Ennis M, Kesthely C. Clinical parameters associated with pressure ulcer healing in patients with advanced illness. *J Pain Symptom Manage.* 2014;47:1035–1042. doi:10.1016/j.jpainsymman.2013.07.005
15. Hendrichova I, Castelli M, Mastroianni C, et al. Pressure ulcers in cancer palliative care patients. *Palliat Med.* 2010;24:669–673. doi:10.1177/0269216310376119
16. Katz S, Ford AB, Moskowitz RW, Jackson BA, Jaffe MW. Studies of illness in the aged. The Index of ADL: A standardized measure of biological and psychosocial function. *JAMA*. 1963;185:914–919. doi:10.1001/jama.1963.03060120024016
17. Kucukdeveci AA, Yavuzer G, Tennant A, Süldür N, Sonel B, Arasil T. Adaptation of the modified Barthel Index for use in physical medicine and rehabilitation in Turkey. *Scand J Rehabil Med*. 2000;32:87–92.
18. Guigoz Y, Vellas B, Garry PJ. Assessing the nutritional status of the elderly: The Mini Nutritional Assessment as part of the geriatric evaluation. *Nutr Rev.* 1996;54:S59–S65. doi:10.1111/j.1753-4887.1996.tb03793.x
19. Oğuz S, Olgun N. Braden Ölçeği ile hastaların risklerinin belirlenmesi ve planlı hemşirelik bakımının bası yaralarının önlenmesindeki etkinliğinin saptanması. Article in Turkish. *Hemşirelik Forumu*. 1998;1:131–135.
20. Stoelting J, McKenna L, Taggart E, Mottar R, Jeffers BR, Wendler MC. Prevention of nosocomial pressure ulcers: a process improvement project. *J Wound Ostomy Continence Nurs*. 2007;34:382–388. doi:10.1097/01.WON.0000281654.40578.88
21. Khor HM, Tan J, Saedon NI, et al. Determinants of mortality among older adults with pressure ulcers. *Arch Gerontol Geriatr.* 2014;59:536–541. doi:10.1016/j.archger.2014.07.011
22. Langemo D. General principles and approaches to wound prevention and care at end of life: an overview. *Ostomy Wound Manage.* 2012;58:24–26, 28, 30 passim.
23. Enoch S, Grey JE, Harding KG. ABC of wound healing. Non-surgical and

drug treatments. *BMJ.* 2006;332:900–903. doi:10.1136/bmj.332.7546.900
24. Abdi AH, Zeighami Mohammadi S, Hoseini M, Babagholzadeh S, Farmani P. Mortality and length of hospital stay in elderly with hospital – acquired pressure ulcer in alborz hospital of Karaj (2012). *Alborz University Med J*. 2018;7:15–23. doi:10.29252/aums.7.1.15
25. Song YP, Shen HW, Cai JY, Zha ML, Chen HL. The relationship between pressure injury complication and mortality risk of older patients in follow-up: A systematic review and meta-analysis. *Int Wound J*. 2019;16:1533–1544. doi:10.1111/iwj.13243
26. Rogers LC, Frykberg RG, Armstrong DG, et al. The Charcot foot in diabetes. *Diabetes Care.* 2011;34:2123–2129. doi:10.2337/dc11-0844
27. Graham ID, Harrison MB, Nelson EA, Lorimer K, Fisher A. Prevalence of lower-limb ulceration: a systematic review of prevalence studies. *Adv Skin Wound Care*. 2003;16:305–316. doi:10.1097/00129334-200311000-00013
28. Lyder CH, Wang Y, Metersky M, et al. Hospital-acquired pressure ulcers: results from the national Medicare Patient Safety Monitoring System study. *J Am Geriatr Soc.* 2012;60:1603–1608. doi:10.1111/j.1532-5415.2012.04106.x
29. Margolis DJ, Knauss J, Bilker W, Baumgarten M. Medical conditions as risk factors for pressure ulcers in an outpatient setting. *Age Ageing*. 2003;32:259–264. doi:10.1093/ageing/32.3.259
30. Farshchian M, Farshchian M, Fereydoonnejad M, Yazdanfar A, Kimyai-Asadi A. Cutaneous manifestations of diabetes mellitus: a case series. *Cutis*. 2010;86:31–35.
31. Gefen A. The biomechanics of heel ulcers. *J Tissue Viability.* 2010;19:124–131. doi:10.1016/j.jtv.2010.06.003
32. Ness SJ, Hickling DF, Bell JJ, Collins PF. The pressures of obesity: The relationship between obesity, malnutrition and pressure injuries in hospital inpatients. *Clin Nutr.* 2018;37:1569–1574. doi:10.1016/j.clnu.2017.08.014
33. OzyesiL Z, Oluk A, Cakmak D. The prediction level of caregiver burden on caregivers' state-trait anxiety levels. Article in Turkish. *Alpha Psychiat*. 2014;15:39–44. doi:10.5455/apd.39820
34. Keelaghan E, Margolis D, Zhan M, Baumgarten M. Prevalence of pressure ulcers on hospital admission among nursing home residents transferred to the hospital. *Wound Repair Regen*. 2008;16:331–336. doi:10.1111/j.1524-475X.2008.00373.x
35. Pang FC, Chow TW, Cummings JL, et al. Effect of neuropsychiatric symptoms of Alzheimer's disease on Chinese and American caregivers. *Int J Geriatr Psychiatry.* 2002;17:29–34. doi:10.1002/gps.510
36. Tuna M, Olgun N. The role of perceived social support on stroke patients' caregivers who have the burnout syndrome. *Hacet Univ Sağlık Bilim Fak Derg*. 2010;17:41–52.
37. Sahin Z. Kronik Obstrüktif Akciğer Hastalarına (KOAH) Bakım Veren Bireylerin Bakım Yükü ve Depresyon Düzeyleri. Article in Turkish. *Tıp Araştırmaları Dergisi*. 2014;12:113–119.
38. Tsai MT, Chen YT, Lin CH, Huang TP, Tarng DC; Taiwan Geriatric Kidney Disease Research Group. U-shaped mortality curve associated with platelet count among older people: a community-based cohort study. *Blood*. 2015;126:1633–1635. doi:10.1182/blood-2015-06-654764
39. Black JM, Cuddigan JE, Walko MA, Didier LA, Lander MJ, Kelpe MR. Medical device related pressure ulcers in hospitalized patients. *Int Wound J*. 2010;7:358–365. doi:10.1111/j.1742-481X.2010.00699.x
40. Tosun Taşar P, Karasahin O, Timur Ö, Sevinç C. Factors associated with mortality in patients with decubitus ulcers treated with negative pressure wound therapy. *Flora-Infek Hastalık*. 2019;24:377–383. doi:10.5578/flora.68955