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EMPIRICAL STUDIES

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Sepsis Episodes Caused by Pressure Injuries in Critical Illness: A Retrospective Observational Cohort Study

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Abstract

BACKGROUND: Critically-ill patients (CIPs) with pressure injuries (PIs) may develop bloodstream infections (BSIs). **PURPOSE:** To identify predisposing factors and discuss diagnosis and management of sepsis-related PIs in CIPs. **METHODS:** The records of CIPs in the intensive care unit (ICU) between January 1, 2014, and January 1, 2020, with PI with sepsis-diagnoses and with different site cultures that were positive concurrent with bloodstream-cultures were retrospectively reviewed. **RESULTS:** Ninety-one sepsis episodes were included in the study. Low albumin level (U = 382.00, P = .006), renal failure (odds ratio [OR], 0.108 [95% CI, 0.015-0.783]; P = .025), and length of ICU stay (U = 130.00, P < .001) were identified as risk factors of BSIs due to PIs. The probability of BSI during a sepsis episode was lower in CIPs with PIs with higher C-reactive protein levels (U = 233.00, P < .001) and whose injury resulted from trauma or surgery (OR, 0.101 [95% CI, 0.016-0.626]; P = .014). The mortality was higher in CIPs with PI-induced BSIs (OR, 0.051 [95% CI, 0.008-0.309]; P = .001). **CONCLUSIONS:** Pressure injury-induced sepsis was associated with a high risk of 28-day mortality. The findings suggest that CIPs with PI are at increased risk of BSIs if they have low albumin levels, renal-failure, and prolonged ICU stay during sepsis episodes.

Introduction

Pressure injuries (PIs) are common in critically ill patients (CIPs). These injuries consist of tissue necrosis that causes undesirable conditions such as pain, the need for surgery, prolonged stay in the intensive care unit (ICU), financial cost, and increased morbidity and mortality.[1,2] In addition, bacteremia associated with infected PIs leads to an increased severity of all these undesirable conditions.[2] Several epidemiologic studies of hospitalized patients or CIPs have shown that approximately 7.5% to 9% of septic episodes are caused by PIs.[3] Surprisingly, few published articles have reported on either predisposing factors for PI infections or clinical and epidemiologic factors that allow reliable diagnosis and antibiotic resistance in CIPs.

Pressure injury infections are associated with a high risk of mortality,[4] and source control in sepsis is crucial. Therefore, it is important to recognize and diagnose infected PIs during sepsis episodes. Diagnosis of infected PIs involves identification of the bloodstream infection via blood culture and identification of PI infection via wound cultures. These cultures should have the same microorganisms, and both must have the same antimicrobial resistance. Sepsis is life-threatening, and predicting PI infection or considering another source of sepsis before obtaining the culture results is crucial in the care of CIPs.

Antibiotic resistance is increasing owing to the long-term use of broad-spectrum antibiotics in CIPs.[3,5] Although PIs are common in CIPs, the diagnosis of PI infection is rare. The epidemiology of infection in PIs in critical illness has not been well described.[6] The reported rate of PI infections among all patients with PIs was 6% in nursing home residents.[7] A cross-

sectional 24-hour point prevalence study of CIPs reported the prevalences of all infections, and skin infection prevalence was reported as 6.4%.[5] Notably, there are no guidelines for PI infections that use the best available evidence for managing conditions such as ventilator-acquired pneumonia. Also, predisposing factors, reliable diagnosis, and treatment of sepsis-related PIs are unclear. In the clinical experience of the authors of the present study, it is even more difficult to determine the source of sepsis unless it is obvious in CIPs with PIs whether the source of sepsis is related to PIs. In addition, it is difficult to identify factors associated with sepsis with PIs in CIPs.

The goal of the present study was to identify predisposing factors and discuss the diagnosis and management of sepsis-related PIs in CIPs. Demographics, medical characteristics, scoring systems, laboratory findings, clinical status, antibiotic use, surgical and vasopressor treatments, PI stage and location, and types of microorganisms and their antimicrobial resistance profiles isolated from CIPs with PIs during sepsis episodes were compared.

Methods

Ethics approval. This retrospective observational cohort study was approved on February 19, 2020, by the ethics committee at Bursa Uludag Medical Faculty (IRB 00004769, decision number: 2020-3/11). No study-related interventions were performed on human subjects. During admission to the ICU, all patients or a designated relative provided informed consent to use their deidentified clinical information as well as laboratory and radiological findings in scientific publications. The need for informed consent was waived by the ethics committee.

Study design and patient selection. Data from adult patients who were treated at Bursa Uludag University tertiary care ICU between January 1, 2014, and January 1, 2020, and who met the study criteria were retrospectively evaluated. The present study used the definition of sepsis agreed on at a consensus meeting that was published in 1992[8] and endorsed in 2003.[9] If sepsis diagnosis was present, definitive or probable infections (defined according to the International Sepsis Forum[10]) were included in the study in CIPs with PIs. Definitive infection was confirmed by positive culture results and probable infection was confirmed with laboratory findings and clinical conditions such as fever.[10] Adult patients admitted to the ICU with PIs and who were diagnosed as having sepsis were included, as were those with proven or suspected PI infection.

Critically-ill patients with proven PI infections were included in study group 1, and CIPs with suspected PI infections were included in study group 2. The criteria for group 1 included all of the following: 1 or more true-positive blood cultures, the presence of 1 or more PIs, and positive culture from the PI, including at least the microorganism or microorganisms isolated in the blood culture with an identical resistance phenotype. The criteria for group 2 included all of the following: 1 or more true-positive blood cultures, the presence of 1 or more PIs, a negative culture from the PI or colonization, and an identified source of the bacteremia other than the PI.

Data collection. Data on demographics (ie, age, sex) and comorbidities were collected. Acute renal failure was diagnosed according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines.[11] Acute Physiology and Chronic Health Evaluation (APACHE) II scores and Sequential Organ Failure Assessment (SOFA) scores (calculated using the worst values observed within 24 hours after positive wound culture collection) were noted. Additional information collected included whether the patient needed a vasopressor within 24 hours after positive wound culture collection, PI stage and location according to National Pressure Injury Advisory Panel (NPIAP) criteria (Stage 1, non-blanchable erythema of intact skin; Stage 2, partial-thickness skin loss with exposed dermis; Stage 3, full-thickness skin loss; Stage 4, full-thickness skin and tissue loss; Unstageable PI, obscured full-thickness skin and tissue loss; Deep Tissue PI, persistent non-blanchable deep red, maroon or purple discoloration), laboratory test results within 24 hours after positive wound culture collection (ie, white blood cell [WBC] count, procalcitonin [PCT], C-reactive protein [CRP], albumin level), and the presence of fever (core temperature > 38.3°C) or hypothermia (core temperature < 36.5°C).

Results

Statistical analysis. Data analysis was conducted using SPSS Statistics version 23.0 (IBM Corporation). A *P* value of less than .05 was considered statistically significant.

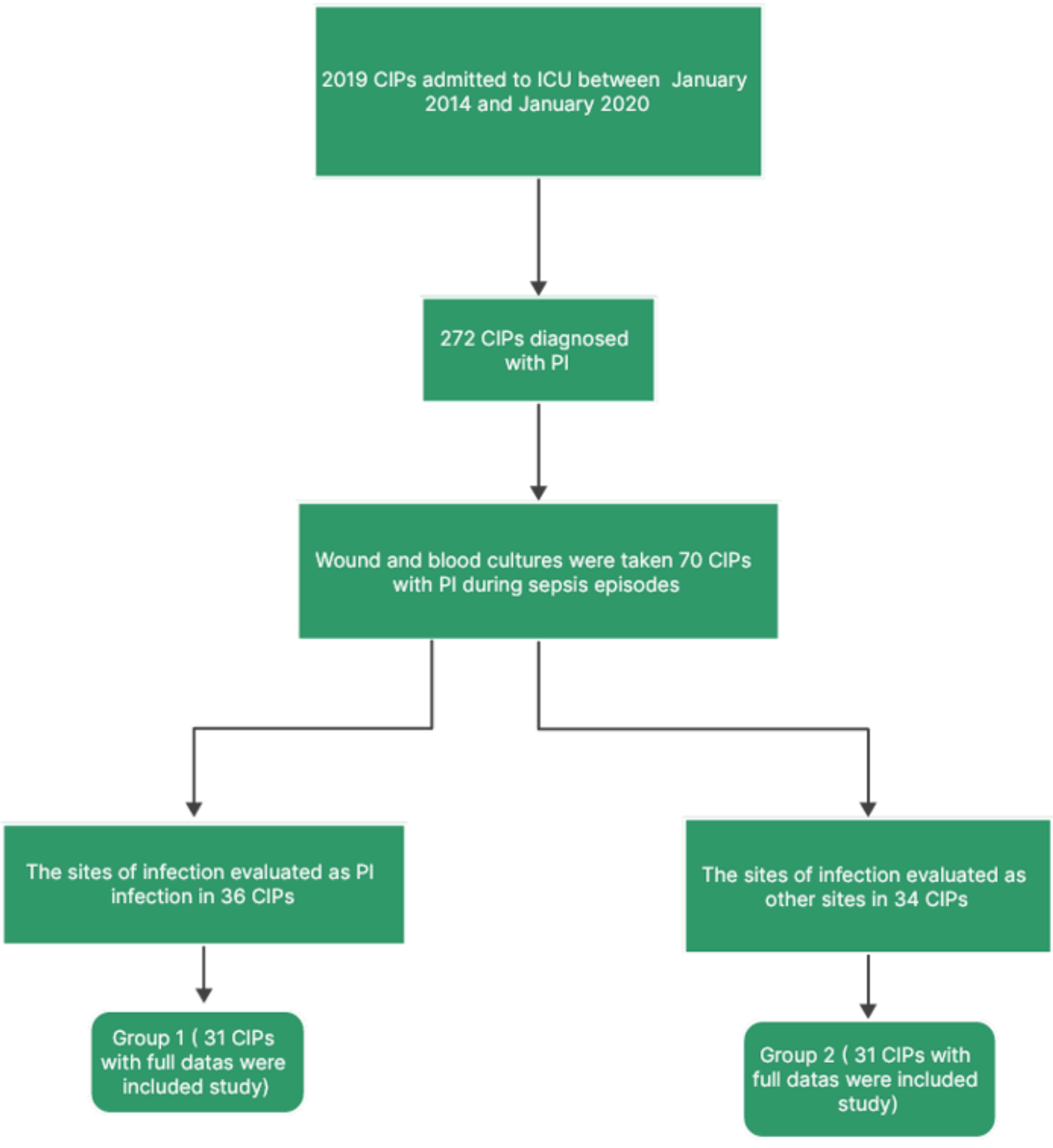


Figure 1. Flowchart of Patients Included in the Retrospective Study

Patient population and characteristics. Between January 1, 2014, and January 1, 2020, among 2019 CIPs, 272 patients in the adult ICU were diagnosed as having PIs. Of these, 91 patients had a diagnosis of sepsis. Pressure injury cultures were obtained from 70 patients with PIs who had been diagnosed with sepsis. The PI was identified as the site of infection in 36 of the 272 patients (13.2%) (group 1). In 34 patients, the site of infection was identified as a site other than the PI (group 2). Only 1 septic episode per patient was included in the study. A total of 62 patients, with 31 patients in each group, were included (**Figure 1**).

Table 1. Demographic Characteristics, Hospitalization, Scoring Systems, and Laboratory Findings in Patients With Pressure Injuries During Sepsis Episodes

Variable	Group 1 (n = 31)	Group 2 (n = 31)	*P* value
Demographic characteristic			
Age, mean (SD), y	65.65 (18.27)	63 (16.02)	.339
Sex, male (n = 40)	21	19	.956
Sex, female (n = 22)	10	12	.956
Scoring system, mean (SD)			
APACHE II	20.94 (6.62)	23.45 (9.26)	.142
SOFA	6.8 (2.6)	6.0 (3.6)	.159
Laboratory findings, mean (SD)			
WBC	11.737 (5901)	11.686 (6813)	.938
CRP (mg/L)	**50.33 (54.41)[a]**	**155.04 (156.24)**	**<.001**
Procalcitonin (µg/L)	2.4 (5.1)	12.92 (24.14)	.074
Albumin (g/dL)	**20.81 (5.88)**	**25.77 (4.8)**	**.006**
Fever (n=18)	8	10	.076
Trauma/Surgery (n=18)	**2**	**16**	**.014**
ICU-LOS, mean (SD)	**39 (25.64)**	**12.61 (11.33)**	**<.001**
Surgical debridement (n=18)	13	5	.078
28-day mortality (n=42)	**28**	**14**	**.020**
Vasopressor use (n=28)	17	11	.520
Sepsis source			
Ventilator-associated pneumonia	0	15	
Urinary infection	0	7	
Central venous catheter-related	0	6	
Bloodstream infection	0	3	
Intra-abdominal infection	0	0	
PI infection	31	0	

Abbreviations: APACHE, acute physiology and chronic health evaluation; CRP, C-reactive protein; d, day(s); ICU, intensive care unit length of stay until collection of wound culture; PI, pressure injury; SD, standard deviation; SOFA, sequential organ failure assessment; WBC, white blood cell count; y, year(s).
[a]Bold font used to indicate statistically significant variables.

Differences in laboratory findings, hospitalization time, and scoring systems between group 1 and group 2. Albumin levels were significantly lower (Mann-Whitney $U = 382.00$, $P = .006$) and length of stay in the ICU until wound culture was significantly longer ($U = 130.00$, $P < .001$) in group 1 compared with group 2. No significant difference was found between group 1 and group 2 in tems of mean age, APACHE II scores, SOFA scores, WBC counts, and PCT levels. C-reactive protein levels were significantly higher in group 2 than in group 1 ($U = 233.00$, $P < .001$). The medical and demographic characteristics of group 1 and group 2 are reported in **Table 1**.

Table 2. Comorbidities and Characteristics of Critically-Ill Patients With Pressure Injuries

Independent variables	B	SE	*P* value	OR	95% CI Lower Value	95% CI Upper Value
DM	.137	.969	.887	1.147	.172	7.658
COPD	-.159	1.035	.878	.853	.112	6.48
CAD/CHF	-2.230	1.013	.028	.108	.015	.783
CVD	.830	.798	.298	.436	.091	.091
Vasculitis	20.880	40192.9	1000	1169295254	.000	.000
Malignancy	-.716	.830	.389	.489	.096	2.487
Renal failure	**2.190**[a]	**.976**	**.025**	**8.937**	**1.320**	**60.495**
Trauma/surgery	**-2.294**	**.931**	**.014**	**.101**	**.016**	**.626**
Colostomy	-2.696	1.841	.143	.067	.002	2.493
PI stages[b]	.756	.711	.287	2.131	.529	8.585

Abbreviations: B, coefficient value of rank; CAD/CHF, coronary artery disease/congestive heart failure; COPD, chronic obstructive pulmonary disease; CVD, cerebrovascular disease; DM, diabetes mellitus; OR, odds ratio; PI, pressure injury; SE, standard error.
[a]Bold font used to indicate statistically significant variables.
[b]Binary logistic regression according to pressure injury stages.

Comorbidities and characteristics of CIPs with PI. Renal failure (chronic and acute) was the only common comorbidity in group 1. Renal failure in CIPs was associated with a significantly increased risk of PI infection (OR, 8.937 [95% CI, 1.320-60.495]; $P = 0.25$). However, age, sex, diabetes, congestive heart failure or coronary artery disease, vasculitis, and malignancy did not increase the risk of PI infection. The most affected anatomic sites were the sacrum (61%), heel (12%), and hip (10%). There were no significant differences between group 1 and group 2 according to PI location at the time of wound culture ($P > .21$ for all). In addition, the probability of PI-induced infection was lower in CIPs whose injury was caused by trauma or surgery (OR, 0.101 [95% CI, 0.016-0.626]; $P = .014$). No significant difference was found between CIPs with and without colostomy (OR, 0.067 [95% CI, 0.002-2.493]; $P = .143$). Of all patients with PIs, 29 had stage 4, 24 had stage 3, 7 had unstageable, and 2 had stage 2 PIs. However, PI stage was not statistically significant compared with the presence or absence of PI infection (OR, 2.131 [95% CI, 0.529-8.585]; $P = .287$) (**Table 2**).

28-day mortality after sepsis diagnosis with PIs in CIPs. Sepsis-related PIs were associated with an increased risk of 28-day mortality after sepsis diagnosis in CIPs (OR, 0.051 [95% CI, 0.008-0.309]; $P = .001$).

Vasopressor treatments and body temperature. Of the 62 patients, 28 needed vasopressor treatment. The most commonly used vasopressor was norepinephrine (93%). In 18 patients, body temperature was above 38.3°C or below 36.5°C for at least 3 hours at the time of wound culture. However, no significant differences were found between group 1 and group 2 according to body temperature and vasopressor use at the time of wound culture ($P > .25$ for both).

Table 3. Types of Microorganism Isolated From Blood Cultures With Proven (Definitive) Pressure Injury Infection

Microorganisms	No. of cultures[a], n=73 (%)
Pseudomonas aeruginosa	14 (19.17)
Klebsiella pneumoniae	16 (21.92)
Acinetobacter baumannii	14 (19.17)
Escherichia coli	10 (13.69)
Enterococcus faecalis	1 (1.36)
Coagulase-negative staphylococci	7 (9.58)
Proteus mirabilis	9 (12.32)
Corynebacterium striatum	2 (2.73)

[a]Distribution of microorganisms grown in blood cultures belonging to Group 1.

Types of microorganisms isolated from PIs and their antimicrobial resistance profiles. *Klebsiella pneumoniae* (51.6%), *Acinetobacter baumanni* (45.2%), and *Pseudomonas aeruginosa* (45.2%) were the most frequently isolated microorganisms in blood cultures of CIPs with PIs with sepsis diagnoses (of group 1 and group 2; 62 patients). Types of microorganisms isolated from CIPs with PI infections (group 1) are shown in **Table 3**. The following percentages are the antibiogram results (resistance to antimictrobial agent) of 31 CIPs with PIs who had positive blood culture and definitive PI infections. Extended-spectrum β-lactamase (ESBL) positivity in *Escherichia coli* was 30% and in *K pneumoniae* was 56.25%. Carbapenem resistance in *K pneumoniae* was 62.5%, in *A baumanni* was 57.1%, and in *P aeruginosa* was 50%. Methicillin resistance in coagulase-negative staphylococci (CNS) was 9.3%. There was no vancomycin resistance in CNS and *Enterococcus faecalis*. In addition, there was no polymixin resistance in gram-negative bacteria. As a result of cultures and antimicrobial resistance profiles of microorganisms isolated from PIs, antimicrobial therapy was changed in 41.9% of patients.

Discussion

Patients with PIs may develop bloodstream infections with or without clinical signs of sepsis.[12] There are few published studies of bacteremia associated with PIs.[13] Moreover, clinical and epidemiologic factors that allow reliable diagnosis and antibiotic resistance in CIPs are unclear. Bacteremia is an infrequent complication but can lead to significant mortality in patients with PI.[14] The frequency of sepsis in patients with PI has been investigated in different patient populations, such as those with chronic spinal cord injury[15] or HIV,[16] with different results. In the Extended Study on Prevalence of Infection in Intensive Care III conducted on CIPs in 2017, sepsis caused by PIs accounted for 6.4% of all sepsis diagnoses.[5] To the authors' knowledge, the present study is the first to investigate the incidence of sepsis due to PI infection in CIPs. In the present study, which includes 6 years of data from CIPs with PIs, the incidence of sepsis due to PIs is 13.2%.

According to the authors' clinical experience, when sepsis is diagnosed in CIPs with a prolonged ICU stay, it can be difficult to determine whether a PI is the source of bacteremia. However, the specific risk factors that can make individuals susceptible to PIs have not been clearly described.[4] In the present study, low albumin levels, a longer length of stay in the ICU, and chronic and acute renal failure were identified as risk factors for sepsis due to PIs in CIPs. Host nutritional status, including malnutrition and hypoalbuminemia, has been well-documented in previous studies.[4,17] Albumin is a natural protein with beneficial physiologic functions, including maintaining colloid osmotic pressure, transporting metabolically active molecules, inhibiting oxidative stress, use as a surrogate marker of nutritional status, having an antithrombotic influence on platelets, being active in acid-base metabolism, and retaining vascular permeability.[17] Critical illness affects the rate of albumin synthesis and degradation, and it affects both transcapillary escape rates and lymph flow, resulting in hypoalbuminaemia.[18] Moreover, studies suggest that hypoalbuminemia could increase the frequency of PIs and the frequency of infections.[17] Similar to the findings from other studies, the current authors' analysis indicated that the probability of a sepsis source being a PI was higher in CIPs with lower albumin levels.

The first barrier against pathogenic microorganisms is skin microbiota.[19] Prolonged immobilization, excessive perspiration contact with urine or feces, and transdermal water loss in CIPs lead to disruption of skin microbiota.[20] Maintaining optimal and balanced skin pH, skin microbiota, and hydration levels is more difficult with a prolonged ICU stay. Thus, host defense deficiency and unhealthy skin in all CIPs with prolonged ICU stay can become an "open door" proliferation to facilitate the entry of pathogenic microorganisms. In the present study, length of stay in the ICU was identified as a risk factor for sepsis due to PIs and was associated with the previously described factors.

The influence of comorbidities is controversial.[7] Studies suggest that chronic illnesses such as diabetes and obesity are associated with several health-related negative clinical outcomes.[21,22] These effects are related to neuropathy, impaired angiogenesis, and chronic low-grade inflammation.[21] In the present study, however, the prevalence of renal failure was higher in sepsis due to PIs compared with other comorbidities. Although diabetes has been identified as one of the most common comorbidities[4] and a risk factor for PI, in the present study there was no evidence that patients with diabetes and PIs were more likely than patients with other comorbidities to develop sepsis. The necessity of aseptic care, daily assessments, cleansing of exit sites, immobilization of catheters, and protection of exit sites and subcutaneous catheter tunnels from trauma limit the positioning of patients receiving renal replacement therapy. Malnutrition is a common problem in CIPs with renal insuffiency.[23] In the present study, the increased incidence of PI infections and renal failure was associated with malnutrition and immobilization.

Another perspective is that clinicians may not typically consider PIs in priority diagnosis as a source of sepsis in CIPs. In the present study, in CIPs with PIs with increased CRP levels in whom injury was caused by trauma or surgery, bacteremia was less likely to be caused by PIs. To the authors' knowledge, the present study is the first to investigate sources of sepsis in CIPs with PIs via proven cultures. The results of the present study showed that cultures taken from the PI were most likely negative when the cause of the injury was surgery or trauma, regardless of the location and degree of the injury. However, according to the culture results, CRP values were higher when the source of sepsis was not a PI.

One study showed that CRP is a marker of infection that also has a protective role against bacterial infections by creating complements.[24] The complement pathway is made up of plasma or membrane proteins, and it is an important system in immunity and the host defense against microbial infection.[24] Lower albumin levels, open door proliferation, and prolonged stay in the ICU may have disrupted the formation mechanism of the formation of CRP complement and caused low levels of CRP. In addition, this complement system may be better preserved in patients with PIs caused by trauma and/or surgery.

Recent studiesdescribe the microbial cultures of PIs as polymicrobial and diverse microorganisms, either aerobic or anaerobic bacteria or fungi.[4,5] These studies also demonstrated that PIs could be colonized by multiresistant species. Similar to the findings of other studies, the present study indicates the presence of polymicrobial and multiresistant microorganisms in positive wound cultures during septic episodes.

Limitations

The present study has limitations. The inclusion of CIPs with PIs with sepsis admitted to a single-referral university tertiary ICU limits the generalizability of the findings, especially regarding microorganisms isolated from cultures. In addition, not all septic episodes were included owing to the retrospective nature of the study, which may have affected the results.

Conclusion

The findings of this first-of-its-kind study of septic episodes in CIPs with PIs suggest that sepsis due to PIs can lead to significant mortality in CIPs. Therefore, it is crucial to identify the predisposing factors of sepsis due to PIs. Based on the findings of the present study, the authors urge health care providers to be more careful when correcting low albumin levels and with patient positioning during renal replacement therapy, especially in CIPs with PIs who have had a prolonged ICU stay.

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