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EVALUATION OF INFECTION AGENT AND ANTIBIOTIC RESISTANCE DISTRIBUTION IN PALLIATIVE CARE PATIENTS WITH PRESSURE ULCERS

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Abstract

Objectives: In our study, it was aimed to examine the distribution of infectious microorganisms, and antibiotic resistance status in palliative care patients with pressure ulcers followed in Ankara Polatlı Duatepe State Hospital Palliative Care Service in 2019- 2020.

Materials and Methods: The sex, age, and detected diseases of a total of 178 palliative care patients included in our study were analyzed retrospectively. For determining the causative agents of pressure ulcer infections in these patients, Gram staining was performed on the bacterial cultures that developed in the wound samples, and the Vitek-2 (bioMérieux, France) automatic test device was used to identify these cultures and determine their antibiotic susceptibility.

Results: It was observed that the single-agent microorganism grew in 26 of the cultures. When the 26 active microorganisms we detected in the wound culture growths were examined; it was observed that *Escherichia coli* (n=9, 34.62%) and *Proteus mirabilis* (n=3, 11.54%) grew more frequently in enteric bacteria and *Pseudomonas aeruginosa* (n=3, 11.54%) in non-fermentative bacteria. In our study, the absence of antibiotic resistance in *Pseudomonas aeruginosa* isolates was considered remarkable. In our study, 100% resistance was found to antibiotics such as Ampicillin, Cefepime, Ceftriaxone, Ciprofloxacin, Amoxicillin-clavulanate, and Gentamicin in gram (+) bacteria, while 100% resistance was found against antibiotics such as Ceftriaxone, Ciprofloxacin, and Trimethoprim/sulfamethoxazole in gram (-) bacteria.

Conclusion: In the treatment of infection pressure ulcers, starting antibiotic therapy at the appropriate time and choosing the right antibiotic is one of the most important factors that determine the success of treatment.

Keywords: Pressure ulcer, palliative care, bacteria, antibiotic resistance.

Introduction

Pressure ulcers are defined as localized tissue damage to the skin and/or subcutaneous tissue, often by pressure on bony prominences or by friction with pressure.[1] Among the body regions where pressure ulcers are most common are sacrum, hip, heel, leg, rib, and scalp.[2-4] Pressure ulcers are an important health problem that increases morbidity and mortality, prolongs hospitalization, and increases the cost of treatment, especially in bedridden patients with limited mobility and in the elderly. 70% of pressure ulcers are seen in people over 65 years of age, who have long periods of inactivity and have neurological or vascular diseases.[5]

Pressure ulcer infections are usually polymicrobial. These infections can cause more serious infections such as cellulitis, osteomyelitis, and sepsis. Although many agents are blamed as causative agents in pressure ulcer infections, the most commonly isolated aerobic bacteria are; staphylococci, enterococci, *Proteus mirabilis*, *Escherichia coli*, *Pseudomonas aeruginosa*, anaerobic bacteria; *Peptosterptococci*, *Bacteriodes fragilis,* and *Clostridium spp*.[6, 7]

In the treatment of pressure ulcer infections, "knowledge of the causative agent" is decisive in the selection of antibiotics and the duration of treatment. According to the results of the culture antibiogram, starting the treatment by considering the antibiotic resistance increases the success of the treatment.

In this study, it was aimed to examine the distribution of causative microorganisms and antibiotic resistance in pressure ulcer infections developed in palliative care patients followed in the Palliative Care Service of our hospital in 2019-2020.

Materials and Methods

Our study included 178 patients who were hospitalized with various diagnoses in Ankara Polatlı Duatepe State Hospital Palliative Care Service from 1st January 2019 to 31th December 2020 and had clinical signs of pressure ulcer infection during the hospitalization. In the staging of pressure ulcers, the classification specified in the guideline of the European Pressure Ulcer Advisory Panel (EPUAP), which provides recommendations for the prevention and treatment of pressure sores, was used.[8] According to EPUAP staging; in stage 2, there was partial depth tissue loss affecting the epidermis and/or the upper layer of the dermis. Clinically, peeling and blistering of the skin were observed, and the wound was superficial. In stage 3, there was full-depth tissue loss or necrosis, including all tissue from the epidermis to the upper fascia. In stage 4, there was full-depth tissue loss as in stage 3. Tissue loss and necrosis had progressed below the fascia, into bone tissue, and supporting structures such as tendons and joint capsules. In patients who developed pressure ulcers, findings such as tenderness, temperature increase, discharge, and redness, which are signs of local infection, and swab cultures

were evaluated together, and the diagnosis of active infection or colonization was made. The distribution of 11 causative microorganisms isolated from 26 of these patients with growth in their wound swab cultures and their antibiotic resistance status were examined. Results considered as colonization and/or contamination were excluded from the study.

All pressure ulcer infections included in the study were clinically evaluated by the same physician. Topical antibiotics were used 3-5 days before sample collection was discontinued. After the swab sticks were moistened with sterile saline, the samples were taken by sufficiently pressing and rotating 360 degrees in 1 cm^2 area of the wound bed and placed in a Carry-Blair transport medium. Wound culture samples in transport medium were inoculated on 5% sheep blood agar and eosin methylene blue (EMB) agar medium. Preparations of the samples were stained with gram stain. Identification of growing bacteria and antimicrobial susceptibility tests were performed using the Vitek-2 (bioMérieux, France) automated system.

The leukocyte-white blood cell (WBC) (10^3/mL), C-reactive protein (CRP) (mg/dL), and erythrocyte sedimentation rate (ESR) (mm/h) measurements in the blood samples were taken from these patients were made in an automatic blood count device.

Before starting the study, local ethics committee approval was obtained. Data obtained from the hospital automation system and patient files (The demographic characteristics of the patients, underlying diseases, hospitalization history, etc.) were evaluated retrospectively.

Statistical Analysis

All statistical analyses were performed by using the statistical package SPSS for Windows, version 22.0 (SPSS, Chicago, Illinois, USA). Descriptive statistics such as frequency, percentage, and ratio were used to evaluate the data, and Student's t-test, and chi-square test were used for comparisons. The value of $p<0.05$ was considered statistically significant.

Results

Of 178 patients sampled from pressure ulcer infections, 72 (40.45%) were male, 106 (59.55%) were female, and the mean age was 76.79±12.04 (40-101) years. While there was no growth in 152 (85.39%) of the patients, growth was detected in the culture in 26 (14.61%), and a total of 11 bacteria grew. While no microbial growth was observed in 59 (81.94%) of male patients' wound cultures, growth was observed in 13 (18.05%) cultures. There was no microbial growth in 93 (87.7333%) of the female patients, while growth was observed in 13 (12.26%) of the female patients. The most common diagnoses were Alzheimer (37.64%, n=67), Malignancy

(20.22%, n=36), Diabetes mellitus (DM) (11.80%, n=21), and Cerebrovascular disease (CVO) (11.24%, n=20). Among the evaluated pressure ulcers. Eight of them (4.49%) were stage 4; 84 (47.19%) were stage 3, and 86 (48.31%) were stage 2.

It was observed that the single-agent microorganism grew in 26 of the cultures. When the 26 active microorganisms we detected in the wound culture growths were examined; it was observed that *Escherichia coli* (n=9, 34.62%) and *Proteus mirabilis* (n=3, 11.54%) grew more frequently in enteric bacteria and *Pseudomonas aeruginosa* (n=3, 11.54%) in non-fermentative bacteria.

The distribution of antibiotic resistance rates of an enteric, non-fermentative, gram (+), and gram (-) bacteria in which growth was detected in the culture are shown in Tables 1, 2, and 3; respectively.

In our study, the most effective agents against enteric bacteria were found to be Tobramycin, Meropenem, Imipenem, Tigecycline, Colistin, Amikacin, Cefazolin, Gentamicin, Ceftriaxone, Cefoxitin, Netilmicin, Levofloxacin, Cefuroxime, Cefixime, and Cefuroxime axetil, while in *Proteus mirabilis*, Tobramycin, Meropenem, Imipenem, Tigecycline, Colistin, Ertapenem, Cefuroxime, Cefixime, Cefuroxime axetil, Levofloxacin, Piperacillin/tazobactam, Trimethoprim/sulfamethoxazole, Aztreonam, Cefoxitin, and Cefepime. It was observed that no antibiotic group came to the fore in terms of resistance level.

In our study, the absence of antibiotic resistance in *Pseudomonas aeruginosa* isolates was considered remarkable.

In our study, 100% resistance was found to antibiotics such as Ampicillin, Cefepime, Ceftriaxone, Ciprofloxacin, Amoxicillin-clavulanate, and Gentamicin in gram (+) bacteria, while 100% resistance was found against antibiotics such as Ceftriaxone, Ciprofloxacin, and Trimethoprim/sulfamethoxazole in gram (-) bacteria.

In our study, the WBC (11.29±6.38) (10^3/mL), CRP (13.15±6.50) (mg/dL), and ESR (92.38±20.64) (mm/h) were found to be higher than the reference ranges ((4-10), (0-0.50), and (0-30); respectively) in patients with growth in wound culture (n=26).

Table 1. Resistance rates to various antibiotics in enteric bacteria isolated from wound cultures

Antibiotic	Escherichia coli n (%) 7 (26.92%)	Escherichia coli (ESBL+) n (%) 2 (7.69%)	Proteus mirabilis n (%) 3 (11.54%)	Klebsiella pneumoniae ssp. n (%) 2 (7.69%)	Serratia marcescens n (%) 1 (3.85%)
Amikacin	1 (14.29)	0 (0)	2 (66.67)	1 (50)	0 (0)
Ampicillin	4 (57.14)	1 (50)	2 (66.67)	1 (50)	1 (100)
Cefazolin	0 (0)	1 (50)	2 (66.67)	1 (50)	0 (0)
Cefepime	4 (57.14)	1 (50)	1 (33.33)	1 (50)	0 (0)
Ceftriaxone	1 (14.29)	1 (50)	2 (66.67)	1 (50)	0 (0)
Ciprofloxacin	4 (57.14)	1 (50)	2 (66.67)	1 (50)	0 (0)
Cefoxitin	1 (14.29)	0 (0)	0 (0)	1 (50)	1 (100)
Netilmicin	1 (14.29)	1 (50)	2 (66.67)	1 (50)	0 (0)
Amoxicillin-clavulanate	2 (28.57)	1 (50)	2 (66.67)	1 (50)	0 (0)
Aztreonam	4 (57.14)	0 (0)	1 (33.33)	1 (50)	0 (0)
Levofloxacin	1 (14.29)	0 (0)	0 (0)	0 (0)	0 (0)
Ceftazidime	4 (57.14)	1 (50)	2 (66.67)	1 (50)	0 (0)
Cefuroxime	1 (14.29)	1 (50)	0 (0)	1 (50)	1 (100)
Cefixime	1 (14.29)	0 (0)	0 (0)	0 (0)	0 (0)
Cefuroxime axetil	1 (14.29)	1 (50)	0 (0)	1 (50)	1 (100)
Piperacillin/tazobactam	3 (42.86)	1 (50)	1 (33.33)	1 (50)	0 (0)
Piperacillin	2 (28.57)	1 (50)	1 (33.33)	0 (0)	0 (0)
Trimethoprim/sulfamethoxazole	7 (100)	0 (0)	1 (33.33)	1 (50)	0 (0)
Gentamicin	1 (14.29)	1 (50)	2 (66.67)	1 (50)	0 (0)
Ertapenem	2 (28.57)	1 (50)	0 (0)	1 (50)	0 (0)
Imipenem	1 (14.29)	0 (0)	1 (33.33)	0 (0)	0 (0)
Meropenem	1 (14.29)	0 (0)	0 (0)	1 (50)	0 (0)
Tigecycline	0 (0)	0 (0)	1 (33.33)	1 (50)	0 (0)
Tobramycin	1 (14.29)	0 (0)	0 (0)	0 (0)	0 (0)
Colistin	0 (0)	0 (0)	1 (33.33)	0 (0)	0 (0)

Table 2. Resistance rates to various antibiotics in gram (+) bacteria isolated from wound cultures

Antibiotic	Enterococcus faecalis n (%) 2 (7.69%)	Staphylococcus aureus n (%) 2 (7.69%)	Coagulase-negative Staphylococcus spp. n (%) 2 (7.69%)
Amikacin	0 (0)	N	N
Ampicillin	2 (100)	1 (50)	2 (100)
Cefepime	2 (100)	N	N
Ceftriaxone	2 (100)	N	N
Ciprofloxacin	2 (100)	1 (50)	2 (100)
Netilmicin	1 (50)	N	N
Amoxicillin-clavulanate	2 (100)	N	N
Aztreonam	1 (50)	N	N
Ceftazidime	1 (50)	N	N
Piperacillin/tazobactam	1 (50)	N	N
Penicillin G	0 (0)	1 (50)	N
Gentamicin	1 (50)	1 (50)	2 (100)
Ertapenem	0 (0)	N	N
Imipenem	0 (0)	N	N
Meropenem	0 (0)	N	N
Tigecycline	1 (50)	0 (0)	0 (0)

(N: Not tested.)

Table 3. Resistance rates to various antibiotics in gram (-) bacteria isolated from wound cultures

Antibiotic	Burkholderia cepacia n (%) 1 (3.85%)	Citrobacter freundii n (%) 1 (3.85%)
Amikacin	0 (0)	0 (0)
Ampicillin	0 (0)	0 (0)
Cefepime	0 (0)	0 (0)
Ceftriaxone	0 (0)	1 (100)
Ciprofloxacin	0 (0)	1 (100)
Netilmicin	0 (0)	0 (0)
Amoxicillin-clavulanate	0 (0)	0 (0)
Aztreonam	0 (0)	0 (0)
Ceftazidime	0 (0)	0 (0)
Piperacillin/tazobactam	0 (0)	0 (0)
Trimethoprim/sulfamethoxazole	1 (100)	0 (0)
Gentamicin	0 (0)	0 (0)
Ertapenem	0 (0)	0 (0)
Imipenem	0 (0)	0 (0)
Meropenem	0 (0)	0 (0)
Tigecycline	0 (0)	0 (0)

Discussion

Diagnosis of pressure ulcer infections is complex and should be evaluated together with clinical symptoms, condition of scar tissue and surrounding, markers of inflammation, microbiological examination of targeted specimens, and tissue biopsies. Changes in pain quality, crepitation, increased exudate, pus, serous exudate with inflammation, increased erythema, bad smell, edema, and local temperature increase in surrounding tissues suggest infection. Tissue biopsy culture is the gold-standard method, but it is an invasive method. It requires intensive work and experience, it is not applied due to difficulties in clinical use, cost, and the need for experienced personnel. Instead, local wound swab cultures, which are a more non-invasive method, are preferred as a more usable method when evaluated together with local signs of infection. Swab cultures may also be insufficient to distinguish between occasional colonization and active infection. Therefore, evaluation of the infection together with clinical findings such as tenderness, erythema, temperature increase, and discharge, which are local findings of infection, gives more accurate results.[7]

In the literature, it has been reported that gram (-) bacteria take the first place among bacteria isolated from pressure ulcer infections, and these species are mostly isolated from pressure ulcer infections in patients with spinal cord injury. This supports the view that the infection occurs due to colonization of the skin with the urogenital and digestive flora.[9]

Ozturk et al. determined that enteric bacteria reproduced most frequently (43.40%) in pressure ulcer infections, *Escherichia coli* in enteric bacteria, and *Pseudomonas aeruginosa* in non-fermentative bacteria grew more frequent.[2] These results are in agreement with the results of the present study.

In our study, when the 26 causative microorganisms we detected in the wound culture; it was observed that *Escherichia coli* (n=9, 34.62%) and *Proteus mirabilis* (n=3, 11.54%) grew more frequently in enteric bacteria and *Pseudomonas aeruginosa* (n=3, 11.54%) in non-fermentative bacteria.

Dundar et al. in the swab samples taken from the pressure sores of 68 patients who received home care service, determined, that 48% of the bacteria reproduced as non-fermentative, 38% as enteric, and 14% as gram-positive bacteria. The most common bacteria are *Pseudomonas aeruginosa* (n=23), *Proteus spp*. (n=20) and *Acinetobacter baumannii* (n=18).[10]

In a study conducted on 55 patients with spinal cord lesions, *Staphylococcus aureus* and *Escherichia coli* were found to be the most common pathogens.[7] Heym et al. in their study, isolated *Enterobacter* 29%, *Staphylococcus spp*. 28% and *Enterococcus faecalis* 16% in the deep tissue biopsy cultures of 101 patients with spinal cord injury and decubitus ulcer infection.[11]

Kilic et al. found pressure ulcer infection was in 13.80% of 2893 patients hospitalized in the rehabilitation center. They isolated *Staphylococcus aureus, Acinetobacter spp., Escherichia coli, and Pseudomonas spp*. as causative agents in order of frequency.[12]

Altan et al., in their study found that the five most commonly isolated microorganisms in wound cultures were *Acinetobacter spp*. (28%), *Pseudomonas spp*. (16.60%), *Candida spp*. (11.40%), *Escherichia coli* (9.30%), and *Enterococcus spp*.[13]

In many clinical studies, the most frequently isolated agents from wound cultures have been reported as *Coagulase-negative staphylococci*, *Escherichia coli, Staphylococcus aureus, Pseudomonas aeruginosa, Acinetobacter baumannii, and Enterococcus spp*.[1, 6] Again, it has been reported that *Coagulase-negative staphylococci* are the most common contaminating bacterium in wound cultures and they show a growth of over 20% in wound cultures.[7] In our study, *Escherichia coli*, 34.62%, *Pseudomonas aeruginosa* 11.53%, *Staphylococcus aureus*, and *Coagulase-negative Staphylococcus spp*. 7.70% were isolated, respectively.

In studies, the effectiveness of the Colistin agent in the treatment of non-fermentative bacteria such as *Pseudomonas aeruginosa* and *Acinetobacter baumannii* has been accepted.[2, 14] Ozturk et al. did not detect Colistin resistance in *Pseudomonas aeruginosa* and *Acinetobacter baumann*ii isolates.[2] Similarly, Colistin resistance was not detected in *Pseudomonas aeruginosa* isolates in our study.

Durmaz et al. in the study in which they evaluated a total of 137 *Pseudomonas aeruginosa* strains, the resistance rates in isolates were found against antibiotics; Amikacin 43%, Gentamicin 38%, Ceftazidime 42%, Cefepime 40%, Cefoperazone-sulbactam 44%, Ciprofloxacin 47%, Levofloxacin 47%, Piperacillin/tazobactam 71%, Imipenem 37%, Meropenem 37%, Colistin 7%, Netilmicin 28%, and Colistin has been reported as the most effective antimicrobial agent against *Pseudomonas aeruginosa strains*.[15]

Aktepe et al. reported that the most sensitive antibiotics in *Pseudomonas aeruginosa* strains were Amikacin and Tobramycin, and resistance was detected at a rate of 4.90%. Meropenem and Imipenem resistance were found to be 26% and 26.80%, respectively. While Ciprofloxacin resistance was determined as 33.30%, this rate was 70% and above for Cephalosporins. It has been stated that the resistance rates in Intensive Care Unit-derived strains increased to 46.40% for Carbapenems and 47.50% for Ciprofloxacin.[16] These results aren't in agreement with the results of the present study. In our study, no resistance was found to any of the antibiotics we used in *Pseudomonas aeruginosa* isolates. This may be since the patient population served by our hospital is not very large and it shows the importance of each hospital determining its resistance profiles.

Parlak et al. reported that they detected ESBL (extended-spectrum beta-lactamases) positivity at a rate of 48% in *Escherichia coli* strains and 67% in *Klebsiella pneumoniae* strains. It was stated that the most effective

antibiotics against isolated strains were the Carbapenem group, followed by Amikacin, Nitrofurantoin, and Cefoxitin in *Escherichia coli* strains, and Amikacin, Cefoxitin, and Ciprofloxacin in *Klebsiella pneumoniae* strains.[17] In our study, ESBL positivity was found 22.20% in *Escherichia coli* strains (2 of 9 strains), ESBL positivity was not found in *Klebsiella* strains. Especially *Escherichia coli* and *Klebsiella* strains showed 50% resistance to Ampicillin, Cefazolin, Cefepime, Ceftriaxone, and Ciprofloxacin.

Scivoletto et al. and Gürçay et al. found high WBC, CRP, and ESR in patients with pressure ulcer infection. [18, 19] In our study, we found that WBC, CRP, and ESR increased in patients with reproductive decubitus ulcer infection, which was consistent with the studies in the literature.

Pressure ulcer infections continue to be a health problem that reduces the quality of life in long-term care patients, despite the development of prevention and treatment methods.[20] It is natural to have superficial bacterial contamination in pressure ulcer infections. Patients with pressure ulcer infection often have an accompanying urinary system or respiratory tract infection, and if not treated, serious problems such as bacteremia and sepsis may occur, which can be fatal.[21]

When local swab cultures are evaluated together with the clinical findings of the infection in the diagnosis of pressure ulcer infection, it is still the most commonly used method. In pressure ulcer infections, aseptic conditions must be followed to prevent infection by colonized bacteria. When an infection develops, a culture should be taken, and antibiotic susceptibility testing should be performed.

In the treatment of infection, starting antibiotic therapy at the appropriate time and choosing the right antibiotic is one of the most important factors that determine the success of treatment. The distribution of infectious agents and the distribution of antibiotic resistance vary periodically and from clinic to clinic.

In our study, following the literature; in the diagnosis of pressure ulcer infection, local wound swab cultures should be evaluated together with the clinical findings of the infection, and we found that initiation of antibiotic therapy at the appropriate time and choosing the right antibiotic in the treatment of infection is one of the most important factors that determine the success of treatment.

Conclusions

As a result, determining the agent distributions and antibiotic resistance in patients with pressure ulcer infection, which we followed in our clinic, with the findings we obtained from our study, guided us in the treatment of our patients and the rational use of antibiotics. Since our study is single-centered, it provides limited information. It is aimed that our future studies will be carried out in a multi-center manner with clinics that periodically perform their surveillance work.

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Ethical Considerations: Ethics committee approval was obtained from Siirt University Non-Interventional Clinical Research Ethics Committee under the Declaration of Helsinki (Date: 06.12.2021, No: 1779, Decision No: 2021/11.01.04)

Conflict of Interest: The authors have no conflicts of interest to declare that are relevant to the content of this article.

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