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A Comparative Study of Iraqi Patients with Bedsore and other Inpatients as a Control Group

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Abstract
Bedsore (pressure ulcers)is a major nurse-careoutcome. There are many influences that can contribute to the progress of pressure ulcers, but the final common pathway to ulceration is tissue ischemia. This study aimed to detect some etiological factors that predispose to bedsore. The study was conducted on patients attending intensive care departments. Out of 82 inpatients who suffered from pressure ulcer, 54were males and 28were females. Specimens were collected during the period of September, 2019 –February, 2020 from different Hospitals (Alkadhimiya Teaching Hospital, General Hospital of AlshaheedAl-Sadr, and Medical City/ Ghazi Al-Hariri hospital) in Baghdad, Iraq. Simultaneously, other 20 specimens were collected as control frominpatientswho did not suffer from any skin lesion.The patients were categorizing according to the causative agent into 48 patients with chronic diseases (58.6%), 26 with accidental and surgical processes (31.7%), and 8 dehydrated cases (severe diarrhea) (9.7%). The demographic results showed that malesconstituted a percentage of 65.9% of patients, while 34.1% were females. The majority of the patients wereat an age range of 36-65 years. The study also revealed that smoking did not affect significantly bed sore development. In addition, most patients were under chronic disease, and diabetes mellitus incidence significantly varied among different age groups. The results of the microbiological study showed that the most prevalent microbial isolates followed the order Staphylococcus spp.>Enterobacteriaceae spp. > Fungi >Acinetobacter spp. > Bacillus spp. > Pseudomonas spp. and Streptococcus spp. >Corynobacterium. The resultsrevealed no significant differences in microbial distribution between patients and control group.

Keywords: Bed sore, Pressure ulcer, Etiology study of bed sore

دراسة مقارنة بين مرضى عراقيين مصابين بقرحة الفراش وغيرهم من المرضى الراقدين في المستشفى كمجموعة سيطرة

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الخلاصة
قرحة الفراش (قرح الضغط) هي نتيجة رئيسية للرعاية التمريضية. هناك العديد من العوامل التي يمكن أن تسهم في ظهور تقرحات الفراش ، ولكن المسار المشترك النهائي للتقرح هو نقص تروية الأنسجة. هدفت هذه الدراسة إلى الكشف عن بعض العوامل المسببة التي تؤهل لقرحة الفراش. أجريت هذه الدراسة على مرضى يرقدون في

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قسم العناية المركزة ، من بين 82 مريضا داخليا. عانى 54 من الذكور و 28 من الاناث من قرحة الفراش. تم جمع العينات خلال الفترة من ايلول \ 2019- شباط \ 2020 منمختلفالمستشفيات (مستشفىالكاظميةالتعليمي،مستشفىالشهيدالصدرالعامومدينة الطب/ مستشفى غازي الحريري) في بغداد / العراق. في نفس الوقت ، تم جمع 20 عينة أخرى من السيطرة في المستشفى من المرضى الداخليين الذين لم يعانوا من أي آفة جلدية. صنف المرضى حسب العامل المسبب ، الأمراض المزمنة 48 (58.6٪) والعمليات الجراحية والحوادث 26 (31.7٪) والجفاف (الإسهال الشديد) 8 (9.7٪). بينت النتائج أن نسبة المرضى الذكور 65.9٪ مقابل 34.1٪ للإناث ومعظمهم دون 36-65 سنة. أظهرت هذه الدراسة أن التدخين لم يؤثر بشكل كبير على تكون قرح الفراش. معظم المرضى يعانون من مرض مزمن ، وكان داء السكري متغيرًا بشكل كبير بين مختلف الفئات العمرية للمرضى. أظهرت نتائج الدراسة الجرثومية أن معظم العزلات الجرثوميةالملوثة لقرحة الفراش هي

Bacillus <Acinetobacter spp.<Fungi<Enterobacteriaceae spp.<Staphylococcus spp. Corynobacterium spp.<Streptococcus spp. وPseudomonas spp. <spp.

والنتيجة أوضحت عدم وجود فرق كبير معنوي بين المرضى ومجموعة السيطرة.

Introduction

Skin is the body's largest organ, extending continuously with the mucous membranes that are lining the body surface [1]. Skinconsists of three layers: the epidermis, dermis, and subcutaneous tissue [2]. Bacteria, fungi, viruses, and parasites may act as normal microbiota or may cause skin diseases and injures [3]. Pressure ulcers (PUs) are one of the most prevalent skin-related diseases, which is prone to microbial contamination[4]. Pressure ulcers are also known as bedsores, decubitus ulcers, and pressure injuries, which are representinga major problem for hospital patients [5].PUs are characterized as degenerative skin and subcutaneous tissue changes [6]. Ulcer is caused when an area of the skin and the tissues below is damaged because it is put under sufficient pressure to impair blood supply [7]. Bedsore most often develops on the skin that is covering bony body areas, such as heels, ankles, hips, back of the head or its sides, and tailbones [8]. The symptoms of pressure ulcers include unusual skin colorvariations or texture changes, swelling, draining-like pus, and a skin area that feels fresher or warmer to the touch than other areas [9]. There are three main key factors leading to bedsores, which are pressure, friction and shear [10]. Risk factors of pressure ulcers include immobility, lack of sensory perception, poor nutrition and hydration, medical conditions which affect the flow of blood, age, low tissue oxygen tension, and moisture [11, 12]. PUscan progress in four stages, depending on the degree of tissue damage [13]. Stage one is the mildest stage, characterized by color change in the upper layer of skin, usually to a deep red,when the wound has not yet opened [14]. In stage two; ulcer may appear as a blister filled with serum (clear to yellowish fluid) that may or may not have burst [15]. In stage three, ulcer could look like a crater that has bad smell. It is essential to look for signs of infection at this point, including bad odor, pus, redness, and discolored drainage [16]. Stage four is the most sever one, where sores reach into deep tissues, including muscles, tendons, and ligaments below the subcutaneous fat,and, in more extreme cases, can stretch as far down as the cartilage or bone. Throughout this point, there is a high risk of infection [17]. Various aspects act together to cause bedsore in the functionally compromised older population. The pathogenesis of bedsore is includes hormonal changes, decreased immune protection, inflammatory factors, diminished blood perfusion, and degenerative changes [18]. A complete approach is required for functionally reduced older patients with co-morbidities to prevent progress of bedsore[19]. Therefore, because PUs act as a portal of microbial entry to the human body,this study aimed to detect some etiological factors that predispose bedsore.

Materials and Methods

Samples collection

of the study involved 82 inpatients (54 males and 28 females) who suffered from pressure ulcer.From each patient, 2 swab specimens were collected; one was from the affected site (ulcer) and the second was from a not affected site. Also, 1 swab specimen was collected from the skin of each control subject (the specimen of control organ is similar to that of patient organ).All subjects had an age range of 2- 95 years. Depending on the age, the bedsore patients and hospitalized control subject were classified into three groups (0-35, 36-65 and 66-95 years). The patients were also classified according to the causal agents of ulcer, which included chronic disease (diabetes mellitus, different heart diseases, and both), accidental or surgical processes, and severe dehydration. All these data were included in a questionnaire form. The specimens were collected during the period of September, 2019 –February, 2020 from different Hospitals (Alkadhimiya Teaching Hospital, General Hospitalof Al-ShaheedAl-Sadr, and Medical City/ Ghazi Al-Hariri Hospital) in Baghdad, Iraq. The hospitalized control subjects included17 male and 3 female inpatient who did not suffer from any skin lesion, with an age range of 21-85 years.

Isolation and identification of microbes

After swabbing the normal tissue and the ulcerative lesion in sterile conditions, eachswab specimen was put in a sterile plane tube containing brain heart infusion broth(Himedia/India) and incubated for 24 hours at 37°C.Aloopful of cultured broth was streaked on blood agar, brain heart infusion agar, MacConkey agar and Mannitol salt agar (Himedia/India) and incubated for 24 hours at 37°C.

Depending on a previously described method [20], identification of each isolate was achieved according to the colony morphology, colony color, Gram stain reaction, and biochemical tests,which included Urease, Voges-Proskauer, Methyl red, Indole, Catalase, Oxidase, and Coagulase.

Statistical analysis

Data analysis was performed by utilizing SPSS for Windows, version 22 (SPSS Inc. Chicago, Illinois, United States). Categorical variables were expressed as frequencies and proportions. Proportions were compared using the Chi-square test (χ^2).Differences with a *p* value lower than 0.05 were considered as statistically significant [21].

Results and Discussion

Age and gender of patients with pressure ulcers

Out of 82 inpatients, 54(65.9%) were males and 28(34.1%) were females, as shown in table (1). Sex-related demographic results showed thatyounger persons suffering from bedsore are males. This might suggest thata greater number of males who are suffering from traumatic spinal cord injuries. Another factor can be the biological effects of intrinsic aging, which alters the skin's mechanical environment and predispose it to wound healing delays and/or impairment. Cell signaling in aged skin is impaired by reduced collagen deposition and increased cross linking, resulting in prolonged inflammation, delayed migration / proliferation, and inadequate remodeling. Whereas our results demonstrated that, in the older subjects, most bedsore patients are women, as a consequence of their survival advantage over menand hormonal changes[22].Also, the result in Table 1 showsthat most patients belong to the second and third groups of age.However, no significant differences were found among age groups. In the control group, the number of male inpatients was 17 (85%) while females were 3 (15%).

Table 1-Patients distribution according to age groups and gender

	Age categories			Total
	(0-35)y	(36-65) y	(66-95)y	
Gender				
Male	10(55.60%)	28(70.00%)	16(66.70%)	54(65.90%)
Female	8(44.40%)	12(30.00%)	8(33.30%)	28(34.10%)
Total	18(22.00%)	40(48.80%)	24(29.30%)	82(100%)
χ^2	1.08	0.59	0.01	__
*p*value	0.29	0.44	0.92	__

Some global researches indicated the prevalence of bedsorein a twice modal age distribution. The first peak was related to the third decade of life and was a small peak, indicating ulceration in those with traumatic neurologic injury. Immobility with no sensation makes these patients susceptible to progressive bedsore[22].

Blanc and coworkers [23]reported that theage of 65 years and above is a risk factor for ulcer development.Barbicel *et al.*[24] reported that two thirds of bedsore cases occurred in patients older than 70 years**,** because the aged skin displays a higher Young's modulus value, reduced viscoelastic recovery, and lower tensile strength than young skin, rendering it vulnerable to traumatic and pressure-related injury [25].

In 2015, Retie and Fisher [26]clarified that these age changes lead to clear visual changes and physical deficiencies that render the skin more vulnerable to injury and can lead to delays or impairments in the healing process. As a consequence, older people are predisposed to wound infection, trauma, and chronic wound formation. Thus, age-related demographics in the present results are in an agreement with global researches.

Another demographic parameter investigated is smoking. The results of this study show that smoking does not act as the main reason for bedsore occurrence (Table 2).However,Lane *et al*. [27] reported that smoking cessation intervention improved quitting rates among patients with spinal cord injuries and was related to changes in wound healing of pressure ulcers. Smoking has a detrimental impact on general health and secondary spinal cord injury complications, including the creation of pressure ulcers and slow wound healing. AlsoVie beckand coworkers[28]elucidated that smoking has a vase- constrictive effect on the small vessels at the dermal level, which reduces the amount of oxygenated blood that provides nutrition to the tissues, further retarding the healing process of bedsore and surgical wounds. Mohammad *et al*.[29]documented that smoking and the duration of cigarettes smoking significantly affect ulcer formation and the degree of damage to the tissues.

Table 2-Distribution of bedsore between smoking and non-smoking patients.

	Age categories			Total		
	(0-35)y	(36-65) y	(66-95)y			
Smoking habit						
Yes	3(16.70%)	12(30.00%)	5(20.80%)	20(24.40%)	1.42	0.48
No	15(83.30%)	28(70.00%)	19(79.20%)	62(75.60%)		
Total	18(22.00%)	40(48.80%)	24(29.30%)	82(100%)	__	__
$\chi 2$	0.74	1.33	0.23	__		

Causes of pressure ulcers

The 82 inpatients were distributed according to the causative agent into those with chronic diseases (48, 58.6%), accidental and surgical processes (26, 31.7%) and dehydrated cases (severe diarrhea; 8, 9.7%).

In particular, research showed that PU prevalence is still disturbingly high in older adults, especially those with diabetes mellitus, heart diseases, frailty, and high sensitivity to prevention and therapeutic interventions [30].Heart diseases are one of main causative factors that may lead patients to form bedsores, especially those lying in intensive health care units for long time. Efraim *et al*.[31] elucidated that in hospitals, community settings, and nursing homes, there are persistent and complicated factors related to immobility, tissue ischemia, and under-nutrition.

The results in Table 3 show that there are no significant differences among different age groups in causing bedsore, but there is a significant difference between males and females in the second age group ($p \leq 0.01$). However, the number of male patients was higher in the second and third age groups than females. Diabetes mellitusrecorded significant ($p \leq 0.05$) between different age groups.

Efraim*et al*, [31] stated that chronic illnesses such as DM, stroke, and advanced dementia seem to be strongly linked withbed sore progress. Also, Kaitani and coworkers[32] reported that hypotension, reduced blood perfusion and peripheral ischemia result in low-cardiacoutput, heart failure, and degraded oxygenation, leading to PU appearance. This will lower blood infusion into vital organ targets,includeheart, brain, eye retina, and legs.The skin of soft tissues will be predisposed to local ischemia. Lyder and his colleagues [33] reported that most of the hospitalized patients who had pressure ulcers also suffered from diabetes. On the other hand, cell metabolism and wound healing are disrupted by dehydration. In order to sustain blood flow to wounded tissues and to avoid further skin breakdown, sufficient fluid intake is required. Iizaka *et al*. [34] found that malnutrition is one of the predisposing factors for PU in patients at 65 years of age or older who were receiving home treatment. During acute hospitalizations, many cases of pressure ulcers were developed in one third of the elderly hospitalized patients who were undercognitive disability.

Table 3-The general characteristics of bedsore patients with chronic diseases

	Age categories			Total	χ^2	*P* value
	(0-35)y	(36-65) y	(66-95)y			
Gender						
Male	1(50.00%)	19(70.40%)	11(57.90%)	31(64.60%)	0.95	0.62
Female	1(50.00%)	8(29.60%)	8(42.10%)	17(35.40%)		
Total	2(4.17%)	27(56.25%)	19(39.58%)	48 (100%)	__	__
χ^2	0.19	17.77	0.61	__		
P value	0.66	0.00	0.43	__		
Chronic disease						
DM	2(100%)	9(33.30%)	3(15.80%)	14(29.20%)	6.73	0.03
Heart disease	0(0.00%)	9(33.30%)	6(31.60%)	15(31.30%)	0.96	0.61
Both	0(0.00%)	9(33.30%)	10(52.60%)	19(39.60%)	3.10	0.21
Total	2(4.17%)	27(56.25%)	19(39.58%)	48 (100%)	__	__
$\chi 2$	5.06	1.06	3.28	__		
P value	0.08	0.58	0.19	__		

Stages of pressure ulcer

Four pressure ulcer stages which are reflecting the severity of bedsore and intensity of spinal cord injuries were significantly different between the groups. Also, age, time of stay, fecal incontinence, diabetes mellitus, anemia and trauma were significantly linked toseverity of bedsores. The results in Table 4 reveal significant differences among stages of bedsores in the first and third age groups. The highest percentage of patients in the first age group is found in stage 4. This result suggests that this age group is more susceptible to accidental and surgical processes that lead to spinal cord injuries. The last age group recorded significant variations among different bedsore stages that may be related to age and health condition. Despite that the second age group recorded the highest percentage of patients; no significant variation was recorded among different stages, because this group included an age range that extended from youth to elderly. Beside that, all age groups recorded significant variation among them in both 1st and 4th stages of bedsore. These results suggest that bed sore occurrence does not recognize between small and old subjects. Curing and/or treatment are dependent mainly on nursing care medical care protocols for out / inpatients.

Table 4-Numbers and frequencies of long term hospitalized inpatients from different age categories based on the stage of bedsore

Stages	Age categories			Total	χ^2	*P* value
	(0-35)y	(36-65)y	(66-95)y			
1	1(9.10%)	3(27.30%)	7(63.60%)	11(13.40%)	7.28	0.02
2	2(8.70%)	15(65.20%)	6(26.10%)	23(28.10%)	4.44	0.10
3	5(22.70%)	10(45.50%)	7(31.80%)	22(26.80%)	0.14	0.93
4	10(4.55%)	12(46.20%)	4(15.30%)	26(31.74%)	7.28	0.02
Total	18(22.00%)	40(48.80%)	24(29.30%)	82(100%)	__	__
χ2	7.56	4.69	8.87	__		
*p*value	0.05	0.19	0.03	__		

Identification of bacterial species

All collected isolates were identified manually according to Holt *et al* [35],based on colony morphology, Gram stain and biochemical tests.

Microbial isolates

Microbial isolates from skin (patient ulcers and normal skin) included different Gram positive and negative bacterial species (aerobes and some anaerobes), as well as fungal species, including Candida .

The results of the microbiological study showed that the most prevalent microbial isolates, based on total number of each species,in the ulcerative skinsamples included (from large to small number) *Staphylococcus spp*.75(40.50%) >*Enterobacteriaceae* spp.51(27.60) >*Fungi* 23(12.40%) >*Acinetbacter spp*. 11(5.90%) >*Streptococcus spp.* 9(4.90%) >*Pseudomonas spp*.8(4.30%) >*Bacillus spp*. 6(3.20%) >*Corynobacterium* 2(1.10%), as shown inTable 5.The results showed no significant differences between normal and ulcerative skin samples for the same patient. There were also no significant differences between ulcerative patients and hospital control subjects. Most bacterial isolates are considered as normal skin flora, but there is the opportunity that these bacterial isolates become pathogenic, especially in immune

compromised patients. All the patients in this study are considered to be under stress and immune depression. Dowd *et al.*[36] clarified that in addition to wound and associated microbiota, other causes can have the likely impact of bacterial bedsores.Chen and Tsao[37] investigated the microbiology of bedsores and noticed that the general populations are*S. aureus, S. epidermidis, P. mirabilis,* and *P. aeruginosa*.

Grice and co-workers [38]stated that the skin contains three main ecological areas, namely the sebaceous, the moist, and the dry. The key species found in the sebaceous areas are *Staphylococci* species. *Corynebacteria*species are dominant in moist places of the body along with *Staphylococci.* Ecologically speaking, sebaceous areas had higher scores than moist and dry ones. Location of bedding, for example, Bacterial colonization cannot be affected as many as physical harm factors.It has been shown that the functions and forms of bacteria in chronic wounds differ. Sapico*et al*. [39] reported that the most resident species in bedsores were *E.coli, Proteus spp.*, Group D *streptococci*, and anaerobic *streptococci,* while *S.aureus*and *P. aeruginosa*were more commonly separated from healing wounds.

The present results showed that the most dominant bacterial isolates belonged to *Staphylococcus* sp.(75, 40.50%), which included*S. aureus*(41, 22.20%) and *S. epidermidis*(34, 18.40%), with no significant differences between the two bacterial isolates occurrence in the PU patients group.Both bacterial isolates recorded increased colonization in patient groups compared with the control one (20; 46.50%), which indirectly reflect the possibility that these bacterial isolates belong to skin microbiota . Therefore, it can be suggested that the increased *S. epidermidis* colonization on skin did not counteract *S. aureus* foundation, and *vice versa*. These results disagreed with that documented by Nakatsuji*et al.*[40]who, in conclusive studies based on culture, demonstrated that *S. epidermidis* can inhibit the growth of *S.aureus* in the skin microbiome community.

Also, the present results revealed that*Enterobacteriaceae* members in ulcerative skin constituted the second largest bacterial group in the patients (51, 27.60 %) compared with the control (6, 14.00%). These members included mainly (from higher to lower numbers) are *Klebsellia spp.* 18(9.70%)>*E. coli* 16(8.60%)>*Proteus spp*. 12(6.50%)>*Serriatia spp.* 4(2.20%). This result is incompatible with those ofAli and William [41]who concluded,based on different global literatures (1966 to February 2014), that the Gram-negative bacteria include the greatest number of single bacterial genera (18 distinct genera), but *Proteus mirabilis* was the most common *Enterobacteriaceae* organism found in bedsores.Thus, the present results suggest that the skin microbiome is mixed with GIT normal flora in the patients group.These bacteria can be converted to infectious agents, particularly in open or ulcerative skin. The same review indicated that *Pseudomonas aeruginosa*and *Streptococcus sp.*,especially *S. fecalis*are, are more dominant in PU than other bacterial isolates [41].

In addition, as a result of nosocomial infections, *Acinetobacterspp.* is gaining importance throughout its entire role in skin and soft tissue infections.The results in Table 5 revealed that there is no significant difference between the two types of patient specimens (11,7.40%and 11, 5.90%, respectively)and the control (3, 7.00%). This result was explained by Dubert*et al*. [42], whospeculated that four exclusive necrotizing features, related to skin and soft tissue infections caused by*Acinetobacterbaumannii*, are present in hosts with underlying co-morbidities, such as cirrhosis andtrauma, and frequently followed by bacteremias. Resistance to many medicaments and the recurrent existence of complicated co-pathogens were often reported. Most cases have been identified as surgical debridement and may lead to substantial mortality rates. Therefore, it can be suggested that *Acinetobacter* sp. in the present study was acquired from hospital environment through long periods of staying in intensive care departments.

Corynebacterium spp. is one of the most predominated bacterium found in individual wounds ,as Diez-Aguilar*etal.*[43] mentioned. While the present results recorded low

percentages found in patients' specimens(2, 1.30% and 2, 1.10%) and the control (1(, 2.30%).

Bacillus spp. is also considered as a contaminant that can cause major diseases and occurs in a number of syndromes, for example in localized infections and bacteremia. Any clinical manifestation, such as necrotic infections (e.g. fasciitis), could result from the release of exotoxins, such as phospholipases, proteases, and haemolysins, asBottone [44] mentioned. The results in Table 5 show no significant differences between both patient specimens (8, 5.40% and 6, 3.20%) and control (5, 11.60%), which may reflect the contamination of hospitals' environments with this genus of bacteria.

In addition,we did not record significant differences in fungus distribution between patient specimens (14, 9.40% and 13, 7.00%) and control (6, 14.00%). Chen and coworkers [45] demonstrated that fungal infections are one of the world's most serious health problems for immune compromised patients. Candidainfectionsof skin is the third most common long-term infection. Researchers from Pennsylvania and Iowa found that fungal communities in chronic wounds may form mixed bacterial-fungalbiofilms , and that these may be associated with poor outcomes, especially for diabetic foot ulcers, and with significantly more fungal infections in patients with higher glycosylated hemoglobin levels [46]. Thus, it can concluded that fungal skin infections might lead to further skin breakdown upon poor quality of nursing care.

Table 5-The percentages and frequencies of different bacterial species found in control and patients (healthy skin & pressure ulcer) groups

Microbial Types	Control (n=20)	patients (n=82)		Total	χ^2	*P*
		Healthy skin	pressure ulcer			
Staphylococcus spp.	20(46.50%)	78(52.30%)	75(40.50%)	173(45.90%)	4.64	0.09
Enterobacteriaceaesp p	6(14.00%)	30(20.10%)	51(27.60)	87(23.10%)	4.84	0.08
Pseudomonas spp	0(0.00%)	4(2.70%)	8(4.30%)	12(3.20%)	2.31	0.31
Streptococcus spp	2(4.70%)	1(0.70%)	9(4.90%)	12(3.20%)	5.04	0.08
Acinetobacterspp	3(7.00%)	11(7.40%)	11(5.90%)	25(6.60%)	0.28	0.86
Corynobacteriumspp	1(2.30%)	2(1.30%)	2(1.10%)	5(1.30%)	0.41	0.81
Bacillus spp	5(11.60%)	8(5.40%)	6(3.20%)	19(5.00%)	5.18	0.07
Fungi	6(14.00%)	15(10.10%)	23(12.40%)	44(11.70%)	0.69	0.70
Total	43 (100%)	149(100%)	185(100%)	377(100%)	__	__
χ^2	8.39	9.04	13.08	__		
P value	0.29	0.24	0.07	__		

Conclusions

From the outcomes of our investigation, it is possible to conclude thatbedsores develop primarily from shear and pressure. They are developed in natural situations and most commonly found in chair bound, bedridden or immobile people. They often progress in people who have been hospitalized for a long period for some diverse problems. The prevention of bedsore represents a marker of quality of hospitalization care. Because of their opportunity to compete with skin microbiota, these microorganisms can pose a great risk of causingbedsores, especially the open type.

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