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Incidence, Bacterial Growth Pattern and Antibiotic Sensitivity of Patient With Pressure Ulcer in Dr Soetomo General Hospital Surabaya

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Abstract— Pressure ulcer is a condition often found in chronically ill patients with prolonged immobilization. Infected pressure sore can inhibit wound healing, wound treatment and even worsen the patient's condition. Wound infections in hospitals are commonly related to nosocomial infection and antibiotics resistance. This research evaluated the incidence, bacterial growth pattern and antibiotics sensitivity of patients with pressure ulcer in Soetomo General Hospital Surabaya. A total of 35 patients were admitted to RSDS and 14 patients fulfilled the requirements to be analyzed. We analyzed 14 patients' medical records admitted to Soetomo Hospital from 1 October 2019–31 January 2020, including demographic data, diagnosis, decubitus site and stages, bacterial culture and antibiotic sensitivity. 71.5% of the patient were male and 57.1% categorized as old adult. Encephalopathy was the most common diagnosis found in pressure ulcer patients. A total of 85.7% of pressure ulcers were developed in the sacral region. Out of 19 isolates, the most common bacteria found is *E.coli, followed by E. faecalis, P. aeruginosa, and A. baumanii*. Amikacin demonstrated a high sensitivity againstthe majority of gram-negative bacteria. *E. coli* were found very sensitive to amikacin, imipenem and meropenem. Cefepime, ceftazidime, gentamycin, imipenem, and piperacillin-tazobactam were other antibiotics that showed 100% sensitivity to *P. aerugionsa*. Cefoperazone-Sulbactam was the only antibiotic found to be very sensitive to *A. baumanii*.

Keywords- Pressure ulcer, decubitus ulcer, bacterial, antibiotic, chronic wound

1. Introduction

Pressure ulcer or decubitus ulcer is common in the patient with chronic injury and long term care. It is an injury of the skin and underlying tissue that occurs due to lack of blood flow and irritation of the skin covering a protruding opening, where the skin is subjected to pressure from a bed, chair, cast, splint, or other hard objects in a long period. This damage usually develops in a bony prominence area. Diseases or conditions associated with immobilization or limited mobility have a significant role in the occurrence of pressure ulcers [1].

According to data from the Department Of Plastic Surgery Reconstruction and Aesthetics of Soetomo Hospital (RSDS), there were 181 hospitalized patients with a pressure ulcer from January 2011 to December 2013 at RSDS, whereas 65% of the total cases are stage III pressure ulcers [2]. A pressure ulcer in stage III or above severity is an open wound that leads to the potential of infection. An infection in pressure ulcer worsen the patient's prognosis and increase the period of treatment.

The appearance of pathogenic bacteria resistant to certain antibiotics, called multi-drug resistant bacteria, complicates the treatment process and prolongs wound recovery. Moreover, nosocomial infections are common in hospitals. Controlling the spread of multi-drug resistant bacteria is important for optimal antibiotic treatment. Therefore, regular testing of bacterial specimens is necessary.

Complications arising from pressure ulcers are associated with significant morbidity and mortality. Prior study at Wahidin Sudirohusodo Hospital reported that the most dominant bacterial pathogens in pressure ulcer specimens were *Pseudomonas aeruginosa*, followed by *Acinetobacter baumanii* and *Staphylococcus aureus* [3]. On the other hand, Setiani et al. reported *Acinetobacter sp* species as the most common bacterial pathogen found in pressure ulcer in AW Sjahranie Hospital [4].

There was still no report of bacterial growth pattern and antibiotics sensitivity in the pressure ulcer patients from RSDS. This study purposed to obtain a picture of the incidence, bacterial growth pattern in patients with a pressure ulcer and the latest antibiotic sensitivity tests to help direct the administration of antibiotics empirically to be faster and more precise.

2. Methods

This research is a prospective study employing a descriptive observational method. A total of 35 patients with a pressure ulcer were admitted to RSDS from October 2019 to January 2020. We collected data from patients' medical records, including demography data, diagnosis of patients, location and stages of the pressure ulcer, wound swab of isolated bacteria, and antimicrobial sensitivity

3. Results

3.1. Demograpic data of the patients

14 out of 35 patients with pressure ulcers admitted to RSDS between October 2019 to January 2020 had a wound swab and complete medical records to be analyzed. The patients with pressure ulcer were predominantly male (71.5%) and older age (57.1%) based on the obtained data in table 1.

3.2. Patients' Diagnosis

The most common diagnosis in pressure ulcer patients were encephalopathy, hospital-acquired pneumonia, inferior paraplegia, anaemia, sepsis, and hypoalbuminemia (Table 2).

3.3. Location and Stage of Pressure Ulcer

The majority of the patients had pressure ulcers in the sacral area (85.7%), while half of the patients had multiple site decubitus (Table 3). There was a total of 24 decubitus sites that developed among the patients, with stage IV pressure ulcers as the predominant ulcer. 10 out of 12 sacral ulcers were in stage III and IV severity. Moreover, pressure ulcers in the sacral region appear at a higher stage than in the other region (Table 4).

3.4. Isolated Bacteria on Pressure Ulcer

Polymicrobial colonization was found in all of the ulcers. The total of bacteria isolated from the ulcers was 19 bacteria. E. Coli 6 (31.6%) were identified as the predominant isolates, followed by Enterococcus faecalis 3 (15.8%), *Pseudomonas aeruginosa* 3 (15.8%), *Acinetobacter baumanii* 3 (15.8%), *Corynebacterium amycolatum* 1 (5.25%), *Corynebacterium striatum* 1 (5.25%), *Klebsiella Pneumoniae* 1 (5.25%), and *Proteus mirabilis* 1 (5.25%) (Table 6).

3.4. Antibiotic Senxitivity Test

Antibiotic sensitivity test reported that all types of isolated gram-negative bacteria were multi-drug resistant. Amikacin was found effective against 4 gram-negative bacteria except for *K. pneumoniae*. On the other hand, *E.coli* and *P. aeruginosa* were sensitive to imipenem and meropenem. *Acinetobacter baumanii* demonstrates a high resistance rate. Only cefoperazone-sulbactam was the only antibiotic that had a high sensitivity to this bacteria (Table 7).

4. Discussion

The majority of the pressure ulcer patients are males (71.5%), contrary to the Azevedo et al. and Mutia et al. Nevertheless, gender was not a significant predictor of pressure ulcers [5]. Moreover, elderly patients are dominant in our study (57.1%), similar to previous studies [6],[7]. Old age is a risk factor for pressure ulcers due to decreased skin integrity. A decrease in the macrophage's functioning leads to a delayed inflammatory response, delayed collagen synthesis, and slower epithelialization [8],[9].

The patients' diagnosis in the medical records was studied to determine the conditions that lead to pressure ulcers. Table 2 described the diagnosis of patients that was dominated by encephalopathy. Encephalopathy results in decreased cognitive function and consciousness, until seizures. Decreased consciousness will result in prolonged

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bed rest and make the patient's fulcrum hold weight for longer, and trigger pressure ulcers. Pneumonia diagnosis was also found in 50% of the patients in this study. The presence of pneumonia in patients leads to reduced oxygen intake, imbalance of inflammatory response, and tissue tolerance to the pressure that can trigger and accelerate the occurrence of pressure ulcers [10].

Immobilization and paralysis are among the strongest factors in pressure ulcer formation [1]. This study found several musculoskeletal system cases i.e muscular atrophy, inferior paraplegia, tetraplegia, TB spondylitis, bone tumor, and posterior pelvic dislocation. These conditions cause a decrease or loss of the patient's ability to move and change positions periodically. Cowan showed that groups of pressure ulcer patients with paralysis treated in long-term care units had higher stages thangroups without paralysis, either tetraplegia or paraplegia [11].

Sepsis was diagnosed in 6 out of 14 patients. With the increase of pressure ulcer stage, the wounds will get deeper. Those opened wounds allow bacteria to enter and cause the infection Cellulitis and osteomyelitis are diagnosed if the infection reaches the inside of the skin and bone respectively. Finally, sepsis is the most fatal complication if the infection reaches the vascular system [12].

Almost half (42.9%) of the pressure ulcer patients were diagnosed with anemia. A prior study confirmed that pressure ulcer incidence in patients with anemia was higher than those without anemia [13]. Low serum albumin levels (<2.8 g/dl) have a significant association with the incidence of pressure ulcers. Albumin plays a role in the wound healing process, thus maintaining more than 2.8 g/dl levels of albumin is the proper management and marker in handling pressure ulcers [14].

Sacral pressure ulcers developed in 12 out of 14 patients. The sacrum is part of the bone protrusion that withstands the patient's most significant load in bed rest for a long time. This part will experience greater pressure than other parts of the body. Suppose this suppression occurs for a long time and without a change in position. In that case, the tissues under the skin will experience ischemia, tissue necrotic, and may lead to a pressure ulcer formation [15]. Those describe the majority of sacral pressure ulcers in our study, similar to prior research that found decubitus ulcer patients were treated at RSDS in 2011-2013 and 2017 experienced the most ulcers in the sacral region [2],[6].

Stage IV ulcers developed in 54.2% of the total cases and were mostly found in the sacral region, which is the primary support for immobilized patients. The ulcer in this region will have the most pressure compared to other parts. If the patient is in a sleep position on his back, it worsens the ulcers condition [15].

Stage III and IV ulcers were also found in the trochanter and gluteal region. Pressure ulcer patients usually require a change in position periodically. The trochanter region is a part that supports the body at the time of the patient in a lateral position. On the other hand, the gluteus is one of the patient's supporting parts in the sleep position. Patients who come to RSDS were referred-patients who already had high degree ulcers or patients with immobilization or limited mobility who have never performed treatment for ulcers. Thus stages of ulcers found were mostly in the high stage.

Specimens obtained from the Department of Clinical Microbiology RSDS during the research period amounted to 19 specimens. The results of this study demonstrated that pressure ulcers had had various types of microorganisms. Distribution data found 5 (23.8%) gram-positive bacteria and 14 (66.7%) gram-negative bacteria. The most isolated gram-positive bacteria was *Enterococcus faecalis*. While the most isolated gram-negative bacteria was *Escherichia coli*, followed by *Pseudomonas aeruginosa* and *Acinetobacter baumanii*.

Most dominant bacteria on pressure ulcers in AWS Samarinda Hospital is *Acinetobacter spp* [4]. While another study at Wahidin Sudirohusodo Hospital Makassar obtained the two most dominant bacteria: *Pseudomonas Aeruginosa* and *Acinetobacter baumanii* [3]. In this study, *Escherichia coli* and *Enterococcus faecalis* are the most dominant bacteria. The number of *E. faecalis* and *E. coli* was directly proportional to the number of patients suffering from pressure ulcers in the sacrum. Patients with ulcers in the sacrum and surrounding areas can be infected with these types of bacteria from the stool coming out of the anus and affecting the ulcer's surface [16].

Enterococcus faecalis is an anaerobic-facultative bacteria commonly found in feces [17]. *Escherichia coli* is a gram-negative bacteria that live in the digestive tract as normal flora, but can also cause opportunistic infections of wounds. *Pseudomonas aeruginosa* bacteria is a pleomorphic-nonmotile gram-negative bacteria. It is one of the most common opportunistic pathogenic microorganisms found in chronic wound infections (including diabetic ulcers and ulcers) as well as burns [18]. *Acinetobacter baumanii* is also a gram-negative bacteria that is opportunistic pathogenic and widely found in hospitals, especially in patients treated for long periods [19]. These bacteria are known to have a broad spectrum of antibiotic resistance [20]. Thus it needs to be a concern because it can cause antibiotic-resistant nosocomial infections.

In antibiotic sensitivity tests, amikacin and meropenem were found to be more sensitive than other antibiotics. *E. coli, P. aeruginosa, and K. pneumoniae* were very sensitive to meropenem. Amikacin showed 100% sensitivity against *E. coli, P. aeruginosa, and P. mirabilis*.

Cefepime, ceftazidime, gentamycin, imipenem, and piperacillin-tazobactam were other antibiotics that showed 100% sensitivity to *P. aeruginosa*. Cefoperazone-Sulbactam was the only antibiotic found to be very sensitive to *A. baumanii* due to its wide spectrum of antibiotic resistance [20]. On the other hand, *E. faecalis*, as the most isolated gram-positive bacteria, was very sensitive to ampicillin, teicoplanin, and vancomycin.

Antibiotics are necessary to treat the infection, but debridement of the ulcer must be performed as primary treatment and leave all viable tissues. Antibiotics are adjunct to surgical debridement and not an alternative to it [15]. Otherwise, antibiotics alone will not clean up the ulcer.

5. Conclussion

Bacterial colonization was found in all pressure ulcer patients. Majority of the ulcers developed in the sacral region. Dominant isolates were *Escherichia coli*, commonly sensitive to meropenem, imipenem, and amikacin. All of the isolates showed multiple resistances, at least four or more antibiotics tested. Resulted data in this study could be considered as a guideline in emphyrical therrapy of pressure ulcer. In light of our findings, proper urinary and fecal control and regular antibiotic resistance test have to be done for each patient to prevent contamination and select an appropriate use of antibiotics.

6. References

- [1] NPUAP, PPPIA, EPUAP. Prevention and Treatment of Pressure Ulcers : Quick Reference Guide. Clinical Practice Guideline. 2014. P.1–75.
- [2] Saputro, I. Karakteristik Pasien Ulkus Dekubitus yang Dirawat oleh Departemen/SMF Ilmu Bedah Plastik Rekonstruksi dan Estetik RSUD Dr. Soetomo. Mimbar. (-). 2014. p.10-12
- [3] Cahyopoetro A, Sarimin S, Seweng A. Identifikasi Pola Kuman Dan Tes Resistensi Antibiotik Pada Penderita Ulkus Dekubitus Di Rs Wahidin Sudirohusodo. Jurnal Penelitian. Fakultas Kedokteran Universitas Hasanudin.2014.
- [4] Setiani D, Imamah I. N. Identifikasi Bakteri dan Faktor Risiko Kejadian Pressure Ulcer di RSUD AWS Samarinda. *Husada Mahakam: Jurnal Kesehatan*. 2019.
- [5] Anthony D, Reynolds T, Russell L. A regression analysis of the Waterlow score in pressure ulcer risk assessment. Clinical Rehabilitation. 2003;17(2):216-223.
- [6] Huvi I, Saputro I, Murtiastutik D. Profil Klinik Pasien dengan Ulkus Dekubitus di RSUD Dr. Soetomo Surabaya. Jurnal Profesi Medika : Jurnal Kedokteran dan Kesehatan. 2019;13(2).

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- [7] Mutia L, Pamungkas K A, Anggraini D. Profil Penderita Ulkus Dekubitus yang Menjalani Tirah Baring di Ruang Rawat Inap RSUD Arifin Achmad Provinsi Riau Periode Januari 2011- Desember 2013. *Jurnal Online Mahasiswa Fakultas Kedokteran Universitas Riau*, vol. 2, no. 2, Oct. 2015, p 1-11.
- [8] Azevedo Macena M, Costa Silva R, Dias Fernandes M, Almeida Medeiros A, Batista Lucio K, Carvalho Lira A. Pressure Ulcer Risk Evaluation in Critical Patients: Clinical and Social Characteristics. The Open Nursing Journal. 2017;11(1):91-97.
- [9] Potter P, Perry A, Stockert P, Hall A. Fundamentals of nursing. 9th ed. Missouri: Elsevier; 2017.
- [10] Krishnan S, Vodovotz Y, Karg P, Constantine G, Sowa G, Constantine F et al. Inflammatory Mediators Associated with Pressure Ulcer Development in Individuals with Pneumonia After Traumatic Spinal Cord Injury: A Pilot Study. Archives of Physical Medicine and Rehabilitation. 2017;98(9):1792-1799.
- [11] Cowan L, Ahn H, Flores M, Yarrow J, Barks L, Garvan C et al. Pressure Ulcer Prevalence by Level of Paralysis in Patients with Spinal Cord Injury in Long-term Care. Advances in Skin & Wound Care. 2019;32(3):122-130.
- [12] Flaherty E, Resnick B. Geriatrics Nursing Review Syllabus. New York, NY: American Geriatrics Society; 2014.
- [13] Bailey R, Reardon G, Wasserman M, McKenzie R, Hord R, Kilpatrick B. Association of Anemia with Pressure Ulcers, Falls, and Hospital Admissions among Long-term Care Residents. Health Outcomes Research in Medicine. 2011;2(4): e227-e240.
- [14] Sung Y, Park K. Factors Affecting the Healing of Pressure Ulcers in a Korean Acute Care Hospital. Journal of Wound, Ostomy and Continence Nursing. 2011;38(1):38-45.
- [15] Bhattacharya S, Mishra R. Pressure ulcers: Current understanding and newer modalities of treatment. Indian Journal of Plastic Surgery. 2015;48(01):004-016.
- [16] Boyko T, Longaker M, Yang G. Review of the Current Management of Pressure Ulcers. Advances in Wound Care. 2018;7(2):57-67.
- [17] Van Tyne D, Martin M, Gilmore M. Structure, Function, and Biology of the Enterococcus faecalis Cytolysin. Toxins. 2013;5(5):895-911.
- [18] Turner K, Everett J, Trivedi U, Rumbaugh K, Whiteley M. Requirements for Pseudomonas aeruginosa Acute Burn and Chronic Surgical Wound Infection. PLoS Genetics. 2014;10(7): e1004518.
- [19] Howard A, O'Donoghue M, Feeney A, Sleator R. Acinetobacter baumanii. Virulence. 2012;3(3):243-250.
- [20] Cerqueira G, Peleg A. Insights into Acinetobacter baumanii pathogenicity. IUBMB Life. 2011;63(12):1055-1060.



7.	Tables

Table 1. Demographic data of patients

Variable	n	Percentage
Age		
< 20 years	4	28.6 %
20 – 29 years	0	0
30 – 39 years	2	14.3 %
40 – 49 years	2	14.3 %
50 – 59 years	3	21.4 %
> 60 years	3	21.4 %
Sex		
Male	10	71.5 %
Female	4	28.5 %

Table	2.	Diagnosis	of	patients
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Diagnosis	No. Of case
Neurology	
Encephalitis	1 (7.14%)
Enchepalopathy	7 (50.0%)
Loss of conciousness	3 (21.4%)
Spinal cord tumor	1 (7.14%)
Hydrocephalus	1 (7.14%)
Multiple Meningioma	1 (7.14%)
Respiratory	
CAP	2 (14.2%)
HAP	5 (35.7%)
Respiratory failure	2 (14.2%)
Pleural efusion	1 (7.14%)
Sputum retention	1 (7.14%)
Lower respiratory tract infection	1 (7.14%)
Musculoskeletal	
Mucle atrophy	1 (7.14%)
Scoliosis	1(7.14%)
Inferior paraplegia	3 (21.4%)
Tetraplegia	1 (7.14%)
Spondylitis TB	3 (21.4%)
Primary bone tumor	1 (7.14%)
Neglected posterior hip dislocation	1 (7.14%)
Osteomyelitis	1 (7.14%)
Haematology and Oncology	
Anaemia	6 (42.9%)
Sepsis	6 (42.9%)
Lung adenocarcinoma	1 (7.14%)
Genitourinaria	
AKI	2 (14.2%)
Fournier Gangrene	1 (7.14%)

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Hydronephrosis	1 (7.14%)
Immunology	
SLE	1 (7.14%)
Rheumatoid Arthritis	1 (7.14%)
Cardiology	
Hypertentsion	1 (7.14%)
Digestive	
Cholelitiasis	1 (7.14%)
Others	
Severe malnutrition	2 (14.2%)
Electrolite disorder	2 (14.2%)
Hypoalbuminemia	7 (50.0%)
Metabolic acidosis	2 (14.2%)

Regio	No of Incidence	Percentage
Sacral	12	85.7%
Thoracolumbal	2	14.2%
Trochanter	3	21.4%
Occipital	1	7.1%
Inguinal	1	7.1%
Gluteal	2	14.2%
Pedis	1	7.1%
Shoulder	1	7.1%
Brachii	1	7.1%

	Tabl	e 3.	Decu	bitus	site
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* There are several patients with multiple site decubitus

-	Tuble II Decubitus stuges				
Stage	No of Incindence	%			
Ι	0	0			
II	6	25.0			
III	5	20.8			
IV	13	54.2			

 Table 4. Decubitus stages

Regio	Stage	Incidence	Percentage
	Ι	-	0
Sacral (n-12)	II	2	16.67%
Sacial (II-12)	III	3	25.00%
	IV	7	58.33%
	Ι	-	0
Thoracolumbar	II	2	100%
(n=2)	III	-	0
	IV	-	0
	Ι	-	0
Trochanter	II	-	0
(n=3)	III	1	33.34%
	IV	2	66.66%
	Ι	-	0
Occipital	II	-	0
(n=1)	III	1	100.00%
	IV	-	0
	Ι	-	0
Inguinal	II	-	0
(n=1)	III	-	0
	IV	1	100.00%
	Ι	-	0
Gluteal	II	1	50.00%
(n=2)	III	-	0
	IV	1	50.00%
	Ι	-	0
Malleolus	II	1	100.00%
(n=1)	III	-	0
	IV	-	0
	Ι	-	0
Shoulder	II	-	0
(n=1)	III	-	0
× /	IV	1	100.00%
	Ι	-	0
D 1''	II	-	0
Brachii	III	-	0
(n=1)	IV	1	100.00%

Table 5.	Details	of dec	ubitus	stages	based	on site
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Table 6. Bacterial pattern based on gram stain

Isolated Bacteria	n	Percentage
Gram-positive (n= 5)		
Enterococcus faecalis	3	15.8%
Corynebacterium amycolatum	1	5.25%
Corynebacterium striatum	1	5.25%
Gram-negative (n= 14)		
Escherichia coli	6	31.6%
Pseudomonas aeruginosa	3	15.8%
Acinetobacter baumanii	3	15.8%
Klebsiella pneumoniae	1	5.25%
Proteus mirabilis	1	5.25%

Table 7. Antibiotic sensitivity test

Antibiotics	E. coli n=6	P. aeruginosa n=3	A. baumanii n=3	K. pneumoniae n = 1	P. mirabilis n = 1
Amikacin	6 (100%)	3 (100%)	1 (33,3%)	-	1 (100%)
Amoxicillin- Clavulanic acid	3 (50%)	1 (33,3%)	-	-	-
Ampicillin-sulbactam	1 (16,7%)	1 (33,3%)	2 (66,7%)	-	-
Aztreonam	1 (16,7%)	2 (66,7%)	-	-	_
Cefepime	-	3 (100%)	1 (33,3%)	-	-
Cefoperazone- Sulbactam	4 (66,7%)	2 (66,7%)	3 (100%)	-	-
Cefotaxime	-	1 (33,3%)	-	-	-
Ceftazidime	2 (33,3%)	3 (100%)	1 (33,3%)	-	-
Ceftriaxone	-	1 (33,3%)	-	-	-
Cephazolin	1 (16,7%)	1 (33,3%)	-	-	-
Chloramphenicol	5 (83,4%)	-	-	1 (100%)	-
Ciprofloxacin	1 (16,7%)	2 (66,7%)	-	-	-
Gentamycin	5 (83,4%)	3 (100%)	1 (33,3%)	-	-
Imipenem	6 (100%)	3 (100%)	2 (66,7%)	-	-
Levofloxacin	1 (16,7%)	2 (66,7%)	-	-	-
Lipiarmycin	_	_	-	1 (100%)	_

Meropenem	6 (100%)	3 (100%)	2 (66,7%)	1 (100%)	-
Moxifloxacin	1 (16,7%)	-	-	-	-
Piperacillin	1 (16,7%)	2 (66,7%)	-	-	-
Piperacillin- tazobactam	5 (83,4%)	3 (100%)	1 (33,3%)	_	-
Tetracycline	1 (16,7%)	-	-	1 (100%)	-
Tigecycline	3 (50%)	1 (33,3%)	-	1 (100%)	-
Trimethoprimsulfa- methoxazole	2 (33,3%)	-	-	1 (100%)	1 (100%)

Antibiotics	Enterococcus faecalis n=3	Corynebacterium amycolatum n=1	Corynebacterium striatum n=1
Ampicillin	3 (100%)	-	-
Teicoplanin	3 (100%)	-	-
Vancomycin	3 (100%)	1 (100%)	-
Linezolid	-	1 (100%)	1 (100%)
Nalidixic acid	-	-	1 (100%)

