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Original Article

"Bacteriological Status of Pressure Sore - A Study of 50 Cases"

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Abstract:

Background of the study: *Pressure sores are major cause of morbidity and mortality in the patients of the long term care facility. Infected pressure sores are very difficult to treat. Managing pressure sore needs care and expertise.*

Objectives: *To study the bacteriological status of pressure sore by qualitative and quantitative culture and to find out the sensitivity pattern of the isolated bacteria to the various antibiotics.*

Methods: *50 patients were included in this study. Wound swabs were collected from pressure sore and deep tissue specimen sampled from pressure sore for quantitative culture in 1st and 3rd visit at 20 days interval. Patients with pressure sore were followed up for healing and their wound healing rate according to PUSH Tool 3.0 is correlated with the bacterial load in the pressure sore. Results were summarized in data table and analyzed.*

Results: *Pseudomonas species were found to be most frequent bacterial isolate followed by E.Coli. Next leading isolated bacteria were Staph. Aureus and Proteus. Ceftazidime, Amikacin, Ciprofloxacin and Gentamycin showed higher percentage of sensitivity and organisms mostly resistant to Ampicillin,Amoxycillin, Co trimoxazole,Flucloxacillin, Ceftriaxone. Quantitative culture of the pressure sore revealed that 40.5% of the sore had bacterial load >10^5 CFU/ gm of tissue and 59.5% had bacterial load <10^5 CFU/gm of tissue on 1st visit. On 3rd visit quantitative culture of the pressure sore after 20 days showed decrease in frequency of >10^5 CFU/ gm of tissue to 21(28.37%). No statistically significant decrease of bacterial load from 1st to 3rd visit noted. No significant difference in healing also noted in between two groups and in different bacterial species.*

Introduction:

Pressure sore is defined as soft tissue injuries resulting from unrelieved pressure over a bony prominence[1]. Traditionally, the problem has been left to nursing staff to identify and manage, but many of the predisposing factors are probably medical in origin and there has been little interest in identifying or addressing them[2]. The exact incidence of pressure sores is uncertain, but is between 3% and 10% in the general hospital population and almost certainly higher in the critically ill[3]. Approximately 9% of all hospitalized patients develop pressure sore[1].

The single most important factor in the development of pressure sore is excessive and prolonged pressure[4]. The etiology of pressure sores is multifactorial. Malnutrition, local perfusion, friction, shearing, infection, drugs administered and other factors may also be involved. In 1975, Darrell Shea, an orthopedic surgeon at the university of Miami, published a method for classifying pressure sores[5]. Shea pressure sore grading system: stage I: the ulcer is confined to the epidermis and superficial dermis, stage II : The ulcer extend through the skin and subcutaneous tissue, stage III : The ulcer extends to the muscle, Stage IV : The ulcer has invaded bone or joint. Each stage was defined simply by anatomic depth of tissue damage; the etiology, the presence of osteomyelitis and the rates of recurrence were not considered. But with minor change this is by far the most acceptable grading system for pressure sore.

Infection in pressure sore by bacteria is major cause that hampers healing of pressure sore[1]. Identifying the major pathogenic bacteria in pressure sore will help us treating the condition. Now a days resistance of organism to antibiotics is another common problem. Sensitivity pattern of bacteria isolated from pressure sore to the antibiotics will be studied and will give us a comprehensive data for prescribing antibiotics in pressure sore. Several study shows that wound biofilm[17] which is newer concept creates a layer of organisms on the surface of the wound that prevents healing of the chronic wound like pressure sore.

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Materials and Methods:

It was a prospective observational study carried out between July 1, 2009- January 31, 2011 in the Department of Plastic Surgery, Dhaka Medical College Hospital.

A total of 50 patients admitted or referred to Department of Plastic Surgery, Dhaka Medical College Hospital with pressure sore were included by purposive sampling, based on some inclusion & exclusion criteria.

- Patients with pressure sore were evaluated by relevant history, clinical examination.
- The pressure sore was sampled with a cotton swab within 48 hours of admission/referral. Sore was swabbed according to Gloucestershire protocols for taking samples using wound swabs
- Deep tissue specimens for quantitative culture were taken from the area of sore where clinical signs of infection were evident Biopsy was taken under local anesthesia (If needed) by stabbing with number 11 blade. Biopsy specimen then put into pre weighted saline (2ml) containing container and send to laboratory for quantitative study.
- Wound swabs for qualitative study were taken at 10 days interval in 1st, 2nd and 3rd visit and quantitative culture of the pressure sore from deep tissue specimen was done during 1st and 3rd visit. Similar collection methods used during swab and deep specimen collection.
- All the patients managed by regular alternate day dressing by 1:1 mixture of normal saline and povidone iodine, measurement of reliving pressure by changing posture 2 hourly, improvement of nutrition and antibiotics administered according to 1st culture report and continued up to 14 days.
- Laboratory investigation revealed bacterial growth and the sensitivity pattern of the isolated bacteria to the antibiotics. Quantitative culture revealed bacterial infective status.
- Data obtained were then presented and analyzed with a view to correlate bacterial species and bacterial load and its effect on the healing of the pressure sore. According to the quantitative culture pressure sores and were divided into two groups. Wounds that had 10^5 CFU/ gm of tissue or more designated for Group- A and wounds having $<10^5$ CFU/ gm of tissue designated in Group- B. Pressure sores were then evaluated for healing of the wound.
- **Evaluation Procedure:** Healing of ulcer was measured using the PUSH 3.0 Tool.[10]

Results and discussion:

Pressure sore is the one of the leading cause of debility among the hospital admitted patients. We studied 50 patients admitted in the Dhaka Medical College Hospital for their bacteriological status. Most of the patients in our study were distributed in middle age group. Mean age of the patients were 47.44 ± 13.30. Among the patients with pressure sore 72% were male and 28% were female. (Figure:1, 2). In our study group male were predominant. Male female ratio was 2.57: 1. In 2009 Sciffmen J. et al.[20]60 patients in wound healing inpatient unit were reviewed, from the Wound Electronic Medical Record, The mean age of the patients

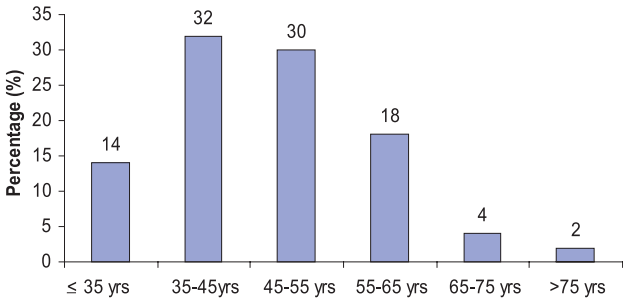

Fig.-1: *Bar diagram showing age distribution of the patients.*

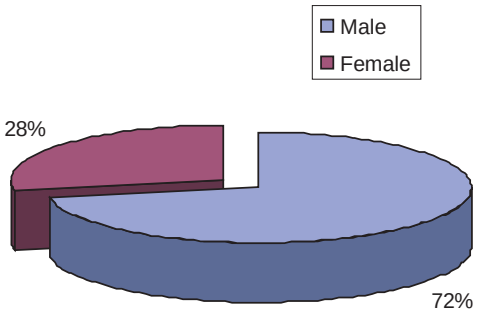

Fig.-2: *Pie chart showing sex distribution of the patients with pressure sore*

was 73.1 years, and 45% were men. In contrast to that study mean age is lower in our study group. Diseases that influenced pressure sore noted in table-I. In our study group sacrum were the most affected pressure sore site (56.7%) followed by trochanter (36.5%). Ischium was affected in 2 patients (2.7%) (Table-II) 16 patients pressure sore in two sites and 4 patients had pressure sore in three sites. In our study group most of the pressure sore in stage III (48.65%) followed by stage IV (39.19%). In 2009 Sciffmen J.et al.[6]found that most wounds (53%) were located on the hip (ischial or trochanteric); others were on the sacrum (32%) and the heels. In 2005, Matthias T [7] studied management of complicated skin and soft tissue infections and isolated Staphylococcus aureus 48.% Pseudomonas aeruginosa 10.8% Enterococcus sp. 8.2% Escherichia coli 7.0% Entereobacter spp. 5.8% Klebsiella spp. 5.1% of cases. In our study all the organism that isolated described in Table III.

Table-I

Causative Disease of the patients of pressure sore.

Causative Disease	Frequency	Percent
Cerebrovascular disease	28	56.0
Unconsciousness due to other medical disease (Meningitis/ Septicaemia)	12	24.0
Spinal Trauma	7	14.0
Spinal Tumour	2	4.0
Spinal TB	1	2.0

Table-II

Site of pressure sore among the patients.

Site	Frequency	Percent
Sacrum	42	56.7
Rt. Trochanter	16	21.6
Lt. Trochanter	11	14.9
Ischium	2	2.7
Illiac crest	2	2.7
Heel	1	1.4
Total	74	100.0

Table-III

Distribution of isolated bacteria among the patients

Isolated bacteria	(1st visit)		(2nd visit)		(3rd visit)		P
	Frequency	Percent	Frequency	Percent	Frequency	Percent	value*
Pseudomonas species	28	34.6	22	28.20	21	27.63	0.852
E.Coli	23	28.4	20	25.64	17	22.36	0.902
Proteus	9	11.1	8	10.25	8	10.52	0.882
Klebsiela	3	3.7	3	3.84	2	2.63	0.765
Staph. Aureus	10	12.34	8	10.25	9	11.84	0.753
Bacteroides Sp.	3	3.7	3	3.84	2	2.63	0.754
Candida species	3	3.7	2	3.84	2	2.63	0.754
No growth	2	2.5	12	15.38	17	22.36	0.033

#Multiple organism in few wounds

*Chi square test was done to measure the level of significance between 1st and 3rd visit.

Table-IV

Antibiotics sensitivity of the pressure sore patients wound swab.

Drug	1st visit		2nd visit		3rd visit	
	Sensitive	Resistant	Sensitive	Resistant	Sensitive	Resistant
Ceftazidime	59(77.63)	17(22.37)	51(79.68)	13(20.32)	46(83.63)	9(16.36)
Amikacin	54(71.05)	22(28.95)	47(73.44)	17(26.56)	42(76.36)	13(23.64)
Gentamycin	43(56.58)	33(43.42)	35(54.68)	19(45.32)	33(60)	22(40)
Ampicillin	4(5.26)	72(94.74)	3(4.68)	61(95.32)	3(5.45)	52(94.55)
Ceftriaxone	33(43.42)	43(56.58)	25(39.06)	39(60.94)	23(41.81)	32(54.18)
Amoxycillin	7(9.22)	69(90.78)	6(9.38)	58(90.62)	4(7.27)	51(92.72)
Flucloxacillin	11(14.47)	65(85.53)	8(12.5)	54(87.5)	6(10.90)	49(89.09)
Ciprofloxacin	55(72.33)	21(27.67)	42(65.63)	22(34.37)	38(69.09)	17(30.90)
Co trimoxazole	20(26.32)	56(73.68)	14(21.88)	40(78.12)	22(40)	33(60)
Cephradine	23(30.27)	53(69.73)	19(29.68)	35(70.32)	16(29.09)	39(70.91)
Doxy cycline	30(39.47)	46(60.53)	23(35.94)	41(64.06)	21(34.18)	34(61.82)

*Figure within parentheses indicates in percentage

Table-V

Quantitative culture of the pressure sore.

	1st visit		3rd visit		P value*
	Frequency	Percent	Frequency	Percent	
Group A(10^5 CFU/gm of tissue or more)	30	40.5	21	28.37	0.452
Group B(<10^5 CFU/gm of tissue)	44	59.5	53	71.63	0.442
Total	74	100.0	74	100.0	

*Chi square test was done to measure the level of significance.

Table-VI

Push Tool score of pressure sore (All bacterial species)

Bacteria	Pseudomonas	E coli	Staph Aureus	Proteus	Klebsiela	Bacteroides	P value
1st visit	13.16±1.10	13.15±0.95	12.11±0.25	12.15±0.25	12.25±0.25	11.75±0.35	0.987
2nd visit	12.67±1.56	11.90±0.88	11.25±0.15	11.35±0.35	11.55±0.75	10.50±0.75	0.876
3rd visit	11.75±1.45	11.05±0.75	10.35±0.30	10.55±35	10.85±0.25	10.25±0.45	0.854
P value	0.643	0.675	0.588	0.650	0.545	0.667	

* t test done to measure the level of significance.

In 2002 Nigel J L[8] described a study of 23 consecutively evaluated patients, bacteriological findings for clinically infected pressure ulcers were assessed by both aerobic and anaerobic culture techniques and specialized specimen transport . An average of 4 isolates (3 aerobes and 1 anaerobe) was recovered. Bacteremia was extremely prevalent (79%) among these patients presenting with sepsis manifestations. Aerobes were more commonly isolated from the ulcers than were anaerobes, but twice as many anaerobes were recovered from cultures of blood samples obtained from 19 patients with bacteremia. Isolates recovered from the ulcer included Proteus mirabilis, Escherichia coli, enterococci, staphylococci, and Pseudomonas species. Anaerobic isolates included Peptostreptococcus species, Bacteroides fragilis, and Clostridium perfringens. The predominant bacteremic isolates were B. fragilis, Peptostreptococcus species, P. mirabilis, and Staphylococcus aureus. In 41% of cases, the bacteremia was polymicrobial.

Bacterial sensitivity to antibiotics were plotted in table-V. Quantitative culture of the pressure sore reveals that 40.5% of the sore had bacterial load e"10^5 CFU/gm of tissue and 59.5% had bacterial load <10^5 CFU/gm of tissue in 1st visit. At 3rd visit quantitative culture of the pressure sore after 20 days shows decreases in frequency of >10^5 CFU/ gm of tissue to 21(28.37%). P value was statistically insignificant. Percentage of sore in group A decreases but No statistically significant decrease of bacterial load from 1st to 3rd visit noted (Table V).

Pressure sores were then evaluated for healing of the wound. Healing of ulcer was measured using the PUSH 3.0 Tool. No bacterial species is identified that hampers healing significantly (Table VI).

Conclusion

Pressure sores are probably an under-rated medical problem. There are major medical and financial implications arising from their development. During management of pressure sore patients most difficult is management of pressure sore. Wound debridement along with other management is key principle Pseudomonas species were most frequent bacterial isolated followed by E.Coli.. Next leading isolated bacteria were Staph. Aureus and Proteus. A few sores showed growth of candida and anaerobic bacteroides species. Ceftazidime, Amikacin, Ciprofloxacin and Gentamycin shows higher percentage of sensitivity in 1st visit .Organisms isolated from pressure sore were mostly resistant to Ampicillin,Amoxycillin, Co trimoxazole,Flucloxacillin, Ceftriaxone. In 2nd and 3rd visit similar results were noted. Quantitative culture of the pressure sore reveals that 40.5% of the sore had bacterial load e"105 CFU/gm of tissue and 59.5% had bacterial load <105 CFU/gm of tissue in 1st visit. At 3rd visit quantitative culture of the pressure sore after 20 days shows decreases in frequency of >105 CFU/ gm of tissue to 21(28.37%). P value was statistically insignificant. Percentage of sore in group A decreases but No statistically significant decrease of bacterial load from 1st to 3rd visit

noted. No significant difference in results noted in between two groups and in different bacterial species in relation to healing. Wound biofilms concept clarifies that bacterial colonization that reside in biofilms delays healing. Dressing and antibiotics can make the wound free from bacterial growth but biofilm that make the wound chronic and hampers healing.

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