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Original Research Article

Antibiogram of burn wound isolates at Masina hospital, Mumbai, India: A 12-year descriptive cross sectional study

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ABSTRACT

Aim: To study the antibiogram of Klebsiella, Pseudomonas and Staphylococcus and its change in sensitivities with time.**Materials and Methods:** 4909 swabs were taken from 790 of patients admitted to the Burns unit of Masina Hospital, Mumbai, over a period of 12 years (2008–2019). The swabs were cultured and percentage antibiotic sensitivity of 6835 predominate isolates to different class of antibiotic was determined and reviewed.**Results:** Klebsiella was the predominant organism in our set-up, followed by Pseudomonas and Staphylococcus aureus. The antibiotic sensitivities of the most predominant organisms are discussed in detail in this article.**Conclusion:** Gram negative nosocomial infection predominate a burn injury. Knowing the predominant target pathogens and their sensitivity pattern towards different antibiotics will avoid misuse of antibiotic, contribute to prescribing the correct antibiotics and timely clinical treatment. A routine microbiological surveillance prior to administering an antibiotic, a well established infection control department and regular reporting of changing antibiotic trends will help us overcome our battle against emerging multi drug resistant organism, thereby having more successful treatment outcome in burn patients.This is an Open Access (OA) journal, and articles are distributed under the terms of the [Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License](https://creativecommons.org/licenses/by-nc-sa/4.0/), which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.For reprints contact: reprint@ipinnovative.com

1. Introduction

A burn injury is not localized to the skin. It is a trauma that involves the entire human body. Survival rate of patients correlates to many factors, burn wound infection being the leading cause of morbidity and mortality.¹ The most feared complication is the development of systemic infection. Increased risk of infection can be due to prolong hospital stay. At one time, the use of prophylactic antibiotics was considered imperative to the treatment of burn patients, however, research has shown that use of prophylactic antibiotics offers no protection

against development of burn wound sepsis and only aids in creation of antibiotic resistant bacteria.² The burn wound is sterile immediately after a burn, however after some time it becomes rich in organisms that are mainly transferred from the environment. Burn patient have a long recovery period resulting in more exposure to hospital environment leading to nosocomial infection. The GI tract is another important source of organisms in burn patients and these can get transmitted to the surface by fecal contamination of wounds.³ Systemic review and Meta analysis has shown that gram negative organisms predominates a burn wound. It establishes that burn wound infection does not differ significantly between burn centers. Whilst burn wound infection is not exclusive to these bacteria, it is hoped

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that reporting the presence of common group of organisms termed as 'target organism' facilitates clinical practice and targets research towards a defined clinical demand. *Pseudomonas*, *Klebsiella*, *E. coli*, *Proteus*, *Acinetobacter*, *Staphylococcus* are commonest group of organisms isolated from clinically infected burn wounds regardless of the centers. It is however acknowledged that there could be other bacteria that could infect burn wounds and it is important to monitor emerging infections. Knowing the target organisms and their timely surveillance of resistance pattern to various classes of antibiotics may contribute to timely clinical treatment by prescribing the right antibiotics and to facilitate a rationalized, targeted and expedited antimicrobial development.^{4,5} Global antibiotic resistance partnership (GARP) recommends national surveillance of antibiotic resistance and antibiotic use which will give better information to underpin decisions on standard treatment guidelines and monitor changes over time.⁶

2. Materials and Methods

As a standard of care, wound swabs are collected of patients admitted in the burns unit of Masina on admission and thereafter weekly, to assess the antibiotic sensitivity of the burn wound isolates. From 2008 to 2019, 790 patients were admitted to the Burns unit. A total of 4909 wound swabs were collected, processed and their antibiotic sensitivity was recorded. Antibiotic sensitivity was tested by the disc diffusion method of Kirby-Bauer.

3. Results

Among 4909 microbiological samples which were taken during the study period, 6835 predominant bacterial strains were isolated. The most predominate organisms in our centre was found to be *Klebsiella* having an average percentage of 50.34%, *P. aeruginosa* at 39.79 % and *Staphylococcus* lagged at 9.73% (Table 1)

The antibiotic sensitivities for individual organisms are shown in Tables below.

4. Discussion

Penicillin & Carbapenems (Tables 2, 3 and 4): Amoxicillin + Clavulanic acid showed a significant increase in sensitivity to *Staphylococcus*. Piperacillin + Tazobactam shows good sensitivity to gram positive organisms. In case of *Klebsiella* it has considerably dropped over time. *Pseudomonas* does show some improvement in its sensitivity to Piperacillin + Tazobactam in 2018-19 after a drop in the intermittent years.

Meropenem continues to show good sensitivity to all our three predominate isolates. At our unit the carbapenems did not show a drastic drop in its sensitivity pattern. Zetal and Piri et al have stated that in order to prevent the incidence of Imipenem resistance, usage of broad spectrum

antibiotics especially carbapenems should be restricted. Presently Imipenem, Vancomycin, Netilmicin and Linezolid are effective for drug resistant pathogen, however there must be controlled use of these resourceful antibiotics.⁷⁻⁹ Potz et al study stated that, while resistance to multiple antibiotics limits the therapeutic options for infections with ESBL-producing organisms, none of the isolates in their study showed resistance to Imipenem or Meropenem. It is therefore comforting to observe the continuing efficacy of the carbapenems against problematic isolates.¹⁰ However overuse can change this trend. Hence we must be carefully in using this group of antibiotics.

Aztreonam did start with a good response however dropped as years passed by.

Cephalosporins (Tables 5, 6 and 7) The initial years showed a good sensitivity however even the 4th generation cephalosporins and the combination with sulbactam and tazobactam could not enhance the sensitivity of this group of antibiotic as expected. In case of gram positive *Staphylococcus* this group still shows some hope. The first prospective study of ESBLs in clinically significant Enterobacteriaceae in the UK stated that almost half the isolates that were cephalosporin resistant produced CTX-M enzymes & high-level AmpC β -lactamase. These isolates now have a wide distribution and dominance among cephalosporin-resistance mechanisms amongst the Enterobacteriaceae. Over 93% of cephalosporin-resistant *Klebsiella* harboured ESBLs, and CTX-M ESBLs outnumbered non-CTX ESBLs by more than 7:1 in this species.¹⁰ Our data proves the same.

Aminoglycosides (Tables 8, 9 and 10): A stagnant graph in case of Gentamycin & Tobramycin against *Klebsiella* & *Pseudomonas* was observed. 50% sensitivity to *Staphylococcus* still persists.

Amikacin & Netilmicin showed a gradual increase in the sensitivity to *Staphylococcus* & *Pseudomonas*.

Aminoglycoside (AG) antibiotics are used to treat many Gram-negative and some Gram-positive infections and, importantly, multidrug-resistant tuberculosis and we must not lose its effectiveness.

Sylvie Garneau-Tsodikova in 2016 found that among various bacterial species, resistance to AGs arises through a variety of intrinsic and acquired mechanisms. The bacterial cell wall serves as a natural barrier for small molecules such as AGs and may be further fortified via acquired mutations. Efflux pumps work to expel AGs from bacterial cells, and modifications here too may cause further resistance to AGs. Mutations in the ribosomal target of AGs, while rare, also contribute to resistance. Rapid detection and identification of resistance genes can allow tailored therapy with these antibiotics. This will not only be more effective at fighting each resistant bacterial infection but also prevent unnecessary use of irrelevant antibiotics.¹¹

Table 1: Bacteriological studies

Year	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19	Total
Total isolates	889	1238	1588	1286	849	985	6835
Organisms	%	%	%	%	%	%	%
Klebsiella	39.37	43.53	48.29	52.26	53.12	65.48	50.34
Pseudomonas	42.96	44.26	47.17	42.53	36.75	25.07	39.79
S. aureus	16.87	12.19	4.53	5.21	10.12	9.44	9.73

Table 2: Antibiotic sensitivity of Klebsiella- Penicillins

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Ampicillin	0.60	1.11	NT	NT	NT	NT
Cloxacillin	0.60	1.11	NT	NT	NT	NT
Piperacillin	17.37	3.34	NT	NT	NT	NT
Amoxicillin+Clavulanic acid	NA	NA	1.04	3.27	7.54	5.89
Piperacillin+Tazobactam	95.21	28.57	5.35	6.85	11.09	18.76
Ticarcillin+Clavulanic acid	12.57	1.86	1.96	3.72	7.76	5.12
Meropenem	41.92	60.85	23.99	36.76	66.08	21.24
Imipenem	95.81	62.71	28.16	40.03	62.31	25.12
Ertapenem	NA	NA	6.13	13.99	32.82	21.40
Faropenem	NA	NA	7.43	5.65	19.73	20.19
Doripenem	NA	NA	7.00	13.24	19.73	25.27
Aztreonam	2.40	6.31	5.87	6.99	12.42	5.58

NA- Not Applicable; NT- Not tested

Table 3: Antibiotic sensitivity of Pseudomonas- Penicillins

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Ampicillin	2.33	0.00	NT	NT	NT	NT
Cloxacillin	1.74	0.55	NT	NT	NT	NT
Piperacillin	12.79	23.18	NT	NT	NT	NT
Amoxicillin+Clavulanic acid	NA	NA	1.20	1.10	8.01	15.79
Piperacillin+Tazobactam	70.35	57.12	23.10	14.08	30.45	66.80
Ticarcillin+Clavulanic acid	8.72	3.65	2.54	9.51	14.74	19.03
Meropenem	41.86	50.00	28.60	51.19	63.78	16.19
Imipenem	66.86	50.91	33.11	54.11	40.71	16.60
Ertapenem	NA	NA	3.47	16.64	21.15	18.62
Faropenem	NA	NA	1.87	13.71	21.79	36.64
Doripenem	NA	NA	6.94	47.71	64.10	63.56
Aztreonam	8.72	26.09	5.47	7.88	20.83	9.72

NA- Not Applicable; NT- Not tested

Table 4: Antibiotic sensitivity of Staphylococcus- Penicillins

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Ampicillin	35.80	17.22	NT	NT	NT	NT
Cloxacillin	35.80	23.18	NT	NT	NT	NT
Piperacillin	49.38	25.83	NT	NT	NT	NT
Amoxicillin+Clavulanic acid	NA	NA	66.67	52.24	50.00	52.69
Piperacillin+Tazobactam	40.74	49.67	73.61	53.73	54.65	78.49
Ticarcillin+Clavulanic acid	60.49	30.46	69.44	55.22	54.65	77.42
Meropenem	59.26	78.81	76.39	59.70	59.30	69.89
Imipenem	77.78	71.52	66.67	56.72	68.60	74.19
Ertapenem	NA	NA	72.22	56.72	56.98	37.63
Faropenem	NA	NA	65.28	43.28	44.19	60.22
Doripenem	NA	NA	26.39	47.76	67.44	96.77
Aztreonam	32.10	17.22	40.28	40.30	32.56	24.73

NA- Not Applicable; NT- Not tested

Table 5: Antibiotic Sensitivity of Klebsiella- Cephalosporin

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Cefazolin	1.8	0.93	NT	NT	NT	NT
Cefuroxime	4.79	3.71	1.96	2.23	4.66	2.17
Ceftizoxime	32.34	15.03	4.17	3.42	4.43	0.31
Cefixime	1.20	6.68	NT	NT	NT	NT
Cefixime + Clavulanic acid	NA	NA	3.00	2.98	6.87	2.33
Cefoperazone	19.16	4.27	NT	NT	NT	NT
Cefoperazone+ sulbactam	84.43	38.96	7.69	15.18	19.73	9.15
Cefotaxime	19.16	7.98	NT	NT	NT	NT
Cefotaxim+sulbactam	85.63	39.15	3.00	2.83	2.22	3.72
Ceftazidime	8.38	7.66	NT	NT	NT	NT
Ceftazidime+Tazobactam	NA	NA	2.35	2.99	5.54	5.43
Ceftriaxone	26.35	7.98	NT	NT	NT	NT
Ceftriaxone+ sulbactam	68.86	24.30	3.78	4.32	12.64	6.82
Ceftriaxone+Tazobactam	NA	NA	4.69	2.83	3.10	12.87
Cefpirome	18.56	7.98	NT	NT	NT	NT
Cefepime	28.14	7.98	NT	NT	NT	NT
Cefepime+Tazobactam	NA	NA	12.91	24.7	20.13	23.72

NA- Not Applicable; NT- Not tested

Table 6: Antibiotic Sensitivity of Pseudomonas- Cephalosporin

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Cefazolin	2.91	0.5	NT	NT	NT	NT
Cefuroxime	5.23	0.00	0.27	5.30	3.85	0.00
Ceftizoxime	39.53	7.12	1.74	6.76	2.88	0.00
Cefixime	4.65	1.09	NT	NT	NT	NT
Cefixime + Clavulanic acid	NA	NA	2.4	5.3	5.45	5.67
Cefoperazone	11.05	11.13	NT	NT	NT	NT
Cefoperazone+ sulbactam	90.12	43.43	9.21	12.25	18.91	12.55
Cefotaxime	13.95	12.59	3.07	14.26	12.18	13.36
Cefotaxim+sulbactam	88.95	22.26	NT	NT	NT	NT
Ceftazidime	7.56	5.75	NT	NT	NT	NT
Ceftazidime+Tazobactam	NA	NA	6.28	31.81	41.03	24.70
Ceftriaxone	19.19	2.92	NT	NT	NT	NT
Ceftriaxone+ sulbactam	58.14	10.77	4.54	13.35	24.36	27.94
Ceftriaxone+Tazobactam	NA	NA	24.41	24.13	39.10	56.68
Cefpirome	21.51	4.20	NT	NT	NT	NT
Cefepime	41.86	7.30	NT	NT	NT	NT
Cefepime+Tazobactam	NA	NA	20.29	21.94	22.99	25.1

NA- Not Applicable; NT- Not tested

Fluoroquinolones (Tables 11, 12 and 13): Fluoroquinolone class of antibiotics was introduced in 1986. Norfloxacin & Ciprofloxacin exhibited substantially greater potency against gram-negative bacteria. Subsequently other fluoroquinolones, such as levofloxacin and Moxifloxacin, were developed with enhanced activity against gram-positive bacteria. Because of their potency, spectrum of activity, oral bioavailability, and generally good safety profile, fluoroquinolones were used extensively for multiple clinical indications throughout the world. Although still clinically valuable, fluoroquinolone use has become limited in some clinical settings, as bacterial resistance has emerged over time.¹² At our unit, Gatifloxacin

showed good sensitivity however it was discontinued at our centre due to non availability and was replaced by Levofloxacin which shows some promise against burn wound isolates. Ofloxacin, Pefloxacin and Norfloxacin decreased in its response to burn wound isolates and hence was discontinued. Ciprofloxacin managed to maintain a 50% sensitivity to burn wound isolates.

Macrolides (Tables 14, 15 and 16): This group of antibiotics did not show a promising ability to fight gram negative burns wound infections however shows some sensitivity against Gram positive Staphylococcus.

In 2016, Fyfe et al stated that „Macrolide resistance mechanisms could be attributed to change in a 23S

Table 7: Antibiotic Sensitivity of Staphylococcus- Cephalosporin

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Cefazolin	43.21	14.57	NT	NT	NT	NT
Cefuroxime	37.04	17.22	59.72	46.27	37.21	40.86
Ceftizoxime	34.57	16.56	58.33	49.25	37.21	16.13
Cefixime	27.16	13.91	NT	NT	NT	NT
Cefixime+Clavulanic Acid	NA	NA	61.11	52.24	46.51	21.51
Cefoperazone	46.91	25.17	NT	NT	NT	NT
Cefoperazone+ sulbactam	80.25	46.36	63.89	56.72	56.98	79.57
Cefotaxime	44.44	49.22	55.56	56.72	40.70	58.06
Cefotaxim+sulbactam	56.79	14.57	NT	NT	NT	NT
Ceftazidime	32.10	11.92	NT	NT	NT	NT
Ceftazidime+Tazobactam	NA	NA	56.94	59.70	41.86	45.16
Ceftriaxone	40.74	20.53	NT	NT	NT	NT
Ceftriaxone+ sulbactam	40.74	20.53	54.17	59.70	46.51	64.52
Ceftriaxone+Tazobactam	NA	NA	69.44	58.21	47.67	38.71
Cefpirome	37.04	14.57	NT	NT	NT	NT
Cefepime	33.33	12.58	NT	NT	NT	NT
Cefepime+Tazobactam	NA	NA	62.5	44.78	45.35	58.06

NA- Not Applicable; NT- Not tested

Table 8: Antibiotic Sensitivity of Klebsiella-Aminoglycosides

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Gentamycin	13.17	25.79	3.52	11.76	29.93	11.47
Tobramycin	22.16	27.27	4.56	15.48	23.95	11.16
Amikacin	56.29	37.85	5.61	12.20	25.94	15.97
Netilmycin	50.90	34.69	11.34	15.33	23.95	8.37

Table 9: Antibiotic Sensitivity of Pseudomonas- Aminoglycosides

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Gentamycin	34.30	21.90	14.69	23.77	13.46	9.31
Tobramycin	30.81	11.86	10.95	24.86	21.79	14.57
Amikacin	44.19	51.09	28.70	29.98	33.01	40.49
Netilmycin	40.12	30.11	11.35	22.85	44.55	36.03

NA- Not Applicable; NT- Not tested

Table 10: Antibiotic Sensitivity of Staphylococcus -Aminoglycosides

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Gentamycin	43.21	21.19	48.61	43.28	53.49	48.39
Tobramycin	37.04	27.15	51.39	49.25	53.49	58.06
Amikacin	58.02	53.64	55.56	62.69	54.65	60.22
Netilmycin	80.25	52.98	45.83	58.21	48.84	75.27

NA- Not Applicable; NT- Not tested

Table 11: Antibiotic Sensitivity of Klebsiella -Fluoroquinolones

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Ofloxacin	66.47	45.27	NT	NT	NT	NT
Pefloxacin	26.35	14.29	NT	NT	NT	NT
Norfloxacin	15.57	16.51	NT	NT	NT	NT
Ciprofloxacin	69.46	56.77	5.87	27.83	54.10	24.96
Sparfloxacin	83.23	62.52	8.34	33.04	58.09	34.42
Lomefloxacin	37.72	41.74	2.74	29.46	43.68	34.11
Gatifloxacin	93.41	87.57	NT	NT	NT	NT

NA- Not Applicable; NT- Not tested

Table 12: Antibiotic Sensitivity of Pseudomonas -Fluoroquinolones

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Ofloxacin	73.26	7.30	NT	NT	NT	NT
Pefloxacin	33.72	3.83	NT	NT	NT	NT
Norfloxacin	38.95	19.89	NT	NT	NT	NT
Ciprofloxacin	59.88	54.56	25.50	46.98	77.56	45.34
Sparfloxacin	86.63	37.59	10.68	28.88	77.24	26.32
Lomefloxacin	59.88	6.93	2.27	7.50	60.58	23.08
Gatifloxacin	99.42	72.63	NT	NT	NT	NT

NA- Not Applicable; NT- Not tested

Table 13: Antibiotic Sensitivity of Staphylococcus-Fluoroquinolones

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Ofloxacin	82.72	7.30	NT	NT	NT	NT
Pefloxacin	40.74	3.83	NT	NT	NT	NT
Norfloxacin	44.44	19.89	NT	NT	NT	NT
Ciprofloxacin	59.26	54.56	66.67	67.16	51.16	49.46
Sparfloxacin	70.37	37.59	56.94	58.21	59.30	25.81
Lomefloxacin	49.38	6.93	58.33	61.19	40.70	45.16
Gatifloxacin	90.12	72.63	NT	NT	NT	NT

NA- Not Applicable; NT- Not tested

Table 14: Antibiotic Sensitivity of Klebsiella -Macrolides

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Erythromycin	28.74	2.41	1.30	11.61	24.61	9.61
Azithromycin	44.31	28.01	36.00	30.80	51.44	18.76
Roxithromycin	6.59	2.97	0.65	NT	NT	NT
Clarithromycin	7.78	3.15	1.83	24.70	60.53	18.45

NA- Not Applicable; NT- Not tested

Table 15: Antibiotic Sensitivity of Pseudomonas-Macrolides

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Erythromycin	12.21	5.11	1.47	32.72	36.86	14.57
Azithromycin	41.28	47.81	45.09	46.80	44.87	23.48
Roxithromycin	11.05	4.56	0.40	NT	NT	NT
Clarithromycin	13.95	2.01	0.27	24.68	37.18	14.57

NA- Not Applicable; NT- Not tested

Table 16: Antibiotic Sensitivity of Staphylococcus -Macrolides

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Erythromycin	30.86	19.87	44.44	52.24	37.21	31.18
Azithromycin	34.57	28.48	48.61	43.28	38.37	33.33
Roxithromycin	30.86	19.87	10.39	NT	NT	NT
Clarithromycin	32.10	13.25	47.22	34.33	34.88	32.26

NA- Not Applicable; NT- Not tested

Table 17: Antibiotic Sensitivity of Klebisella- Others

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Colistin	49.70	77.18	13.82	39.29	77.61	59.07
Cotrimoxazole	24.55	16.70	6.91	10.27	17.52	6.20
Tetracycline	61.08	34.32	14.08	29.46	30.60	18.60
Chloramphenicol	34.13	23.93	11.08	38.39	60.75	19.07
Metronidazole	1.80	0.00	2.61	1.49	0.00	0.00
Clindamycin	1.20	1.67	0.00	0.00	0.00	0.16
Tigecycline	NA	13.36	22.29	40.63	71.40	59.06

NA- Not Applicable; NT- Not tested

Table 18: Antibiotic Sensitivity of Pseudomonas- Others

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Colistin	79.07	87.96	36.45	36.56	81.09	124.29
Co-trimoxazole	29.65	0.00	1.34	3.84	12.82	15.79
Tetracycline	62.79	6.75	11.62	31.81	13.78	21.86
Chloramphenicol	53.49	8.58	4.01	22.12	75.00	35.63
Metronidazole	0.00	0.55	0.00	0.00	0.64	4.05
Clindamycin	1.74	2.19	0.00	1.83	4.17	6.48
Tigecycline	NA	4.74	17.36	53.56	71.46	66.40

NA- Not Applicable; NT- Not tested

Table 19: Antibiotic Sensitivity of Staphylococcus - Others

	2008-09	2010-11	2012-13	2014-15	2016-17	2018-19
Co-trimoxazole	37.04	23.84	56.94	59.70	43.02	41.94
Tetracycline	51.85	38.41	56.94	58.21	55.81	75.27
Chloramphenicol	93.83	37.09	55.56	55.22	50.00	73.12
Metronidazole	17.28	4.64	0.00	0.00	4.65	9.68
Clindamycin	70.37	16.56	1.39	0.00	9.30	19.35
Tigecycline	NA	2.65	37.50	37.31	69.77	97.85

NA- Not Applicable; NT- Not tested

ribosomal RNA (rRNA) residue or a mutation in ribosomal protein L4 or L22 affecting the ribosome's interaction with the antibiotic, thus emphasizing that tailor made antibiotic therapies will prevent the birth and spread of resistant genes in bacterial isolates.¹³

Others (Tables 17, 18 and 19): Colistin managed to stay strong at 50% sensitivity against Pseudomonas & Klebsiella.

Tigecycline has shown promising ability to combat burn wound infection.

5. Conclusion

Ample evidence exists to support the notion that morbidity, mortality and quality of life outcome in burn patients is associated with organisms such as Klebsiella, Pseudomonas, E.coli & staphylococcus. Sepsis with these organisms is an independent indicator to mortality. These bacteria also promote failure of healing which is a major consequence to the management of extensive burn wounds. Specific risk factor associated to burn wound infection rates is increased resistance. In India, resistance to commonly used antibiotic prompts the use of newer generation of antibiotics. These newer antibiotics are expensive and not readily available for common man.⁹ This must be avoided and alternative and more sustainable methods to treat infections must be developed.

Following modification in the burn wound management practices may lower infection rates resulting in improved outcomes and curtailing the emergence of drug resistance.

Sensitivity can improve when there is less exposure to antibiotics, hence it is important to culture wounds on admission and at regular intervals of treatment and start

antibiotics based on the culture sensitivity reports. If the swab culture does not show any growth, and the patient is clinically stable, oral and/or intravenous antibiotics must be avoided. Patient must be preferably treated with topical antibiotics and burn wounds must be regular cleaned and dressed to avoid microbial colonization.

Regular reporting of the changing trends of antibiotic sensitivity is as important as restricted and targeted use of antibiotics. More and more data published from various burn centers will help formulate effective guidelines for therapy and will also be instrumental in forming strict antibiotic policies in various hospitals.¹⁴

The hospital infection control department must educate the staff in hand hygiene, isolation precautions and enhanced disinfection of patient room to further avoid any nosocomial infection thereby further reducing risk of burn wound infection and sepsis. Multidrug resistance once established in hospital environment can persist for months in a unit and can infect patients being treated there.⁸ Hence, having a well established infection control department in a hospital is a stepping stone towards curbing the birth of multi drug resistant strains.

To conclude, routine microbiological surveillance prior to administering an antibiotic, a well established infection control department and regular reporting of changing antibiotic trends will help us overcome our battle against emerging multi drug resistant organism, thereby having more successful treatment outcome in burn patients.

6. Conflicts of Interest

The authors declare no potential conflict of interest with respect to research, authorship, and/or publication of this

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