

Original Article

Antibiotic susceptibility of wound isolates in plastic surgery patients at a tertiary care centre

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ABSTRACT

Context: Wound infection increases the hospital stay and adversely affects the recovery of patients. Culture and sensitivity of wound isolates help in proper diagnosis and management of these patients. **Aim:** To identify common bacteria causing wound infections and their antibiotic sensitivity pattern. **Study Design:** A cross-sectional study. **Place and Duration of Study:** Government Medical College and Hospital, Nagpur from October 2013 to October 2015. **Materials and Methods:** Pus samples were collected by doctors in ward using Sterile Swab Sticks. Bacterial isolates were identified and cultured, and antibiotic culture sensitivity tests were performed. **Statistical Analysis Used:** Chi-square test. **Results:** There were a total of 150 patients with infected wounds. Most common organism isolated was *Pseudomonas* followed by *Klebsiella* and *Staphylococcus aureus*. All of these organisms were resistant to most routine antibiotics. **Conclusion:** We suggest a multidisciplinary approach to wound management, rational drug use, routine microbiological surveillance of wounds and institution of hospital infection control policy.

KEY WORDS

Bacteria; culture and sensitivity; wound infection

INTRODUCTION

A wound is the breakdown in the protective function of the skin, loss of continuity of epithelium with or without loss of underlying connective tissue. Wounds can be accidental, pathological or post-operative. All wounds contain bacteria, but the majority of the wounds do not get infected.^[1] Infection is believed to occur when virulence factors expressed by one or more microorganisms in the wound outcompete the host's natural immune system.^[2]


Wound infection is the most common nosocomial infection and cross infection is common.^[3] Wound infections adversely affect morbidity and mortality, delay wound healing, cause wound breakdown and are also associated with longer hospital stay and increased the cost of health care.^[4]

With an extra hospital stay of 6–14 days at as much as 180 American dollars, aseptic surgical wound is considered 'a remarkable expensive luxury'.^[5]

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The era of chemotherapy is about 60 years old; however, surgery without sepsis remains an unfulfilled goal. The widespread and indiscriminate use of antibiotics has led to the progressive development of resistance to penicillin and many other antibiotics.

'We are fighting an escalating war against the microbes, and it is felt that we are the losers'. Antibacterial susceptibility pattern for microorganisms isolated from hospitalised patients with wound infection is continuously evolving.^[6] Unavailability of appropriate alternative antibiotics may be life-threatening to some hospitalised patients.^[7] Determining the sensitivity of bacterial isolates to antibiotics can help the clinician choose the appropriate antimicrobial.

Very few studies of this kind have been carried out in India, and this issue was largely unexplored in an apex teaching medical institution at Nagpur.

A prospective observational analysis study was thus carried out to elucidate the extent of the problem, the bacteriology and factors associated with wound infection, the antibiotic sensitivity and resistance pattern and to provide baseline estimate for subsequent comparison and to guide hospital infection control and antibiotic usage policy.

MATERIALS AND METHODS

Study design

This is a prospective cross-sectional study of isolates from the Plastic Surgery Unit of Government Medical College and Hospital (GMCH), Nagpur, from October 2013 to October 2015.

Study area

GMCH, Nagpur, is the largest apex teaching medical institution in the Vidarbha region of Maharashtra catering to 2.4 million inhabitants of Nagpur and surrounding districts.

Inclusion/exclusion criteria

All patients admitted to plastic surgery ward with the raw area and suspected wound infection were enrolled in the study. Wound infection was suspected if the wound was exuding pus or fluid, not healing well or associated with signs and symptoms of inflammation. Very ill patients and those undergoing antibiotic therapy 2 weeks before the study were excluded from the study. Patient's age, sex, type, site, duration and aetiology of the wound were noted.

Ethical committee clearance for the study was granted by the Ethics Review Board of GMCH.

Sampling procedure

A questionnaire was used to obtain data from the patient after obtaining informed consent from the patient and guardians. Sample collection was performed from open wound in the ward by the resident surgeon using Sterile Swab Sticks as per existing departmental guidelines. Only one swab per patient was collected after carefully cleaning the wound and surrounding skin with saline to avoid surface contamination. The samples were then transferred to microbiology laboratory within 1 h of collection using airtight sterile vial.

Culture and identification

Swabs collected were streaked on blood agar and MacConkey agar by sterile inoculation loop. Agar plates were incubated at 37° for 24–48 h. Bacterial colonies on the agar plate were then Gram stained and subjected to biochemical tests for identification and classification.

Antibiotic sensitivity

Antibiotic susceptibility was done by Kirby–Bauer disc diffusion method and interpreted according to the recommendations of the National Committee for Clinical Laboratory Standards.^[8] The drugs tested for both Gram-positive and Gram-negative bacteria were ampicillin, amoxicillin-clavulanic acid, amikacin, cefotaxime, ceftriaxone, ciprofloxacin, gentamicin, imipenem, piperacillin-tazobactam and levofloxacin. The antibiotics selected were based on the availability and prescription frequency of these drugs in the study area.

Statistical analysis

Chi-square test was employed to compare the association of the sociodemographic data, wound type, location, duration, with wound infection status of the patient. $P < 0.05$ was considered statistically significant.

Ethics

Written informed consent was obtained from all study participants.

RESULTS

A total of 150 wound swab samples were collected from the patients admitted to the Plastic Surgery Unit from October 2013 to October 2015. The subjects included 94 (62.6%) males and 56 (37.4%) females. The ages of the patients ranged from 6 months to 80 years with a mean age of 33.6 years [Table 1].

Sixty-six (4%) samples were obtained from the leg, whereas 25 (16.7%) samples were obtained from the hand.

Seventy-one (47.3%) wounds were post-traumatic, whereas 48 (32%) were post-cellulitic (cellulitis, abscess, necrotising fasciitis and Fournier's gangrene) [Table 2].

Duration of the presentation was calculated as time from injury, wound to first seen by us. Eighty-seven (58%) wounds presented to us in the 1st week, whereas 24 (16%) wounds presented in the 2nd week [Table 3].

Bacteriology

Of the 150 wounds examined, 129 (86%) were culture positive, whereas 12 (9.3%) were polymicrobial in nature. One hundred and forty-three bacterial isolates were obtained in a total of which 111 (78%) were Gram-negative and 32 (22%) were Gram-positive.

Pseudomonas aeruginosa was the most common bacteria isolated 46 (32.2%) followed by *Klebsiella* 28 (19.6%), *Staphylococcus aureus* 24 (16.8%), *Proteus* species 22 (15.4%), *Escherichia coli* 15 (10.5%) and coagulase negative staphylococci (CONS) 8 (5.5%) [Table 4 and Figure 1].

Antibiotic susceptibility pattern of bacterial isolates

All the bacterial isolates were tested for antibiotic sensitivity by the Kirby–Bauer method against selected 10 antibiotics. For *Pseudomonas* stain, piperacillin was included [Table 5].

The Gram-negative isolates were fairly resistant to commonly used antibiotics such as ampicillin (70.3%), metronidazole (46%) and ciprofloxacin and gentamicin (43.2%). About 76.1% *Pseudomonas* isolates were sensitive to piperacillin [Table 6].

The Gram-positive isolates showed 100% sensitivity to amikacin, imipenem and levofloxacin and were strongly sensitive to 3rd generation cephalosporin such as cefotaxime (93.8%) and ceftriaxone (90.6%). Seventy-five percent of the isolates were resistant to ampicillin [Figures 2, 3 and Table 7].

The Gram-positive bacteria, *S. aureus* and CONS decreased dramatically as the duration of the wound increased. All the *Pseudomonas* isolates were resistant to ampicillin but were fairly sensitive to piperacillin (76.1%), amikacin and ceftriaxone (71.7%).

Table 1: Wound infection and demographic characteristics

| Demographic character | Infected, n (%) | Not infected, n (%) | Total |
|-----------------------|-----------------|---------------------|-----------|
| Sex | | | |
| Male | 82 (87.2) | 12 (12.8) | 94 (62.6) |
| Female | 47 (83.9) | 9 (16.1) | 56 (37.4) |
| Total | 129 (86) | 21 (14) | 150 (100) |
| Age (years) | | | |
| ≤10 | 7 (70) | 3 (30) | 10 (6.7) |
| 11-20 | 13 (81.3) | 3 (18.7) | 16 (10.7) |
| 21-30 | 21 (91.3) | 2 (8.7) | 23 (15.3) |
| 31-40 | 36 (81.8) | 8 (18.2) | 44 (29.3) |
| 41-50 | 29 (85.3) | 5 (14.7) | 34 (22.7) |
| 51-60 | 12 (80) | 3 (20) | 15 (10) |
| ≥60 | 6 (75) | 6 (25) | 8 (5.3) |
| Total | 124 (82.7) | 26 (17.3) | 150 (100) |

Table 2: Wound type and location

| | Number(%) |
|-----------------------|-----------|
| Wound type | |
| Post-traumatic | 71 (47.3) |
| Post-cellulitic | 48 (32) |
| Post-operative | 18 (12) |
| Burn wound | 4 (2.7) |
| Diabetic foot ulcers | 9 (6) |
| Total | 150 (100) |
| Wound location | |
| Leg | 66 (44) |
| Hand | 25 (16.7) |
| Genitals | 14 (9.3) |
| Breast/chest | 12 (8) |
| Abdomen | 11 (7.3) |
| Scalp | 9 (6) |
| Buttocks | 8 (5.3) |
| Head and neck | 3 (2) |
| Back | 2 (1.3) |
| Total | 150 (100) |

Table 3: Duration of presentation

| Duration of wound (days) | n (%) |
|--------------------------|-----------|
| 0-7 | 87 (58) |
| 7-14 | 24 (16) |
| 14-21 | 17 (11.3) |
| >21 | 22 (14.7) |

Table 4: Frequency of bacteria isolated

| Bacteria isolated | n (%) |
|------------------------|-----------|
| <i>P. aeruginosa</i> | 46 (32.2) |
| <i>K. pneumonia</i> | 28 (19.6) |
| <i>S. aureus</i> | 24 (16.8) |
| <i>Proteus</i> species | 22 (15.4) |
| <i>E. coli</i> | 15 (10.5) |
| CONS | 08 (5.5) |

CONS: Coagulase negative staphylococci, *P. aeruginosa*: *Pseudomonas aeruginosa*, *K. pneumonia*: *Klebsiella pneumonia*, *S. aureus*: *Staphylococcus aureus*, *E. coli*: *Escherichia coli*

Table 5: Antibiotic sensitivity of Gram-negative isolates

| Gram-negative isolates (number) | Reaction | Antibiotics, n (%) | | | | | | | | | |
|---------------------------------|----------|--------------------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|
| | | A | AK | C | T | Cx | G | I | L | M | P |
| <i>Pseudomonas</i> (46) | S | - | 33 (71.7) | 26 (56.5) | 31 (67.4) | 33 (71.7) | 28 (60.9) | 39 (34.8) | 30 (65.2) | 24 (52.2) | 35 (76.1) |
| | R | 46 (100) | 13 (28.3) | 20 (43.5) | 15 (32.6) | 13 (28.3) | 18 (39.1) | 07 (15.2) | 16 (34.8) | 22 (47.8) | 11 (23.9) |
| <i>Klebsiella</i> (28) | S | 11 (39.3) | 18 (64.3) | 16 (57.1) | 22 (78.6) | 22 (78.6) | 14 (50) | 26 (92.9) | 28 (100) | 12 (42.9) | - |
| | R | 17 (60.7) | 10 (35.7) | 12 (42.9) | 06 (21.4) | 06 (21.4) | 14 (50) | 02 (7.1) | - | 16 (57.1) | - |
| <i>Proteus</i> (22) | S | 12 (54.5) | 14 (63.6) | 10 (45.5) | 10 (45.5) | 14 (63.6) | 11 (50) | 19 (86.4) | 18 (81.8) | 14 (63.6) | - |
| | R | 10 (45.5) | 08 (36.4) | 12 (54.5) | 12 (54.5) | 08 (36.4) | 11 (50) | 03 (13.6) | 04 (18.2) | 08 (36.4) | - |
| <i>E. coli</i> (15) | S | 10 (66.7) | 12 (80) | 11 (73.3) | 11 (73.3) | 13 (86.7) | 10 (66.7) | 14 (99.3) | 13 (86.7) | 10 (66.7) | - |
| | R | 05 (33.3) | 03 (20) | 04 (26.7) | 04 (26.7) | 02 (13.3) | 05 (33.3) | 01 (6.7) | 02 (13.3) | 05 (33.3) | - |
| Total (111) | S | 33 (29.7) | 77 (69.4) | 63 (56.8) | 74 (66.7) | 82 (73.9) | 63 (56.8) | 98 (88.3) | 89 (80.2) | 60 (54) | 35 (76.1) |
| | R | 78 (70.3) | 34 (30.6) | 48 (43.2) | 37 (33.3) | 29 (26.1) | 48 (43.2) | 13 (11.7) | 22 (19.8) | 05 (46) | 11 (23.9) |

S: Sensitive, R: Resistance, -: Zero, A: Ampicillin, AK: Amikacin, C: Ciprofloxacin, T: Cefotaxime, Cx: Ceftriaxone, G: Gentamicin, I: Imipenem, L: Levofloxacin, M: Metronidazole, P: Pipracillin, *E. coli*: *Escherichia coli*

Table 6: Antibiotic sensitivity of Gram-positive isolates

| Gram-positive isolates | Reaction | Antibiotics | | | | | | | | | |
|------------------------|----------|-------------|----------|-----------|-----------|-----------|-----------|----------|----------|-----------|--|
| | | A | AK | C | T | Cx | G | I | L | M | |
| <i>S. aureus</i> (24) | S | 04 (16.7) | 24 (100) | 21 (87.5) | 22 (91.7) | 21 (87.5) | 18 (75) | 24 (100) | 24 (100) | 15 (62.5) | |
| | R | 20 (83.3) | - | 3 (12.5) | 02 (8.3) | 03 (12.5) | 06 (25) | - | - | 09 (37.5) | |
| CONS (08) | S | 03 (37.5) | 08 (100) | 06 (75) | 08 (100) | 08 (100) | 05 (62.5) | 08 (100) | 08 (100) | 04 (50) | |
| | R | 05 (62.5) | - | 02 (25) | - | - | 03 (37.5) | - | - | 04 (50) | |
| Total (32) | S | 08 (25) | 32 (100) | 27 (84.4) | 30 (93.8) | 29 (90.6) | 23 (71.9) | 32 (100) | 32 (100) | 19 (59.4) | |
| | R | 25 (75) | - | 05 (15.6) | 02 (6.2) | 03 (9.4) | 09 (28.1) | - | - | 13 (40.6) | |

S: Sensitive, R: Resistance, -: Zero, A: Ampicillin, AK: Amikacin, C: Ciprofloxacin, T: Cefotaxime, Cx: Ceftriaxone, G: Gentamicin, I: Imipenem, L: Levofloxacin, M: Metronidazole, *S. aureus*: *Staphylococcus aureus*, CONS: Coagulase negative staphylococci

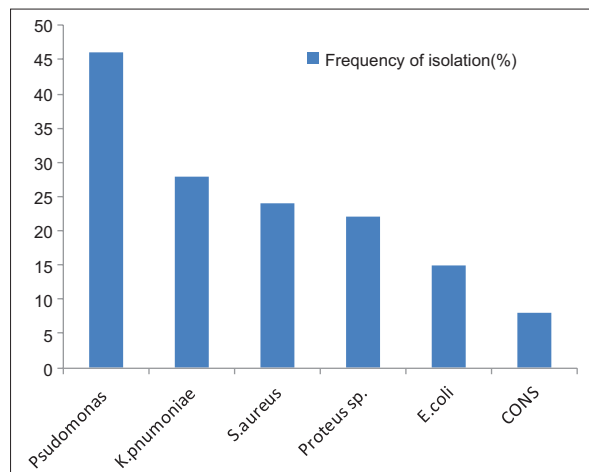


Figure 1: Frequency of bacteria isolated. CONS: Coagulase-negative staphylococci. *S. aureus*: *Staphylococcus aureus*

Eighty-four percent of the *Staphylococcus* isolates, on the other hand, were resistant to ampicillin but were highly sensitive to amikacin, imipenem and levofloxacin (100%).

Beyond the 3rd week, all *Klebsiella* isolates were resistant to all the routinely used antibiotics except for imipenem and levofloxacin.

The sensitivity of *S. aureus*, *Pseudomonas* and *Klebsiella* gradually decreased as the duration of wound increased to all the routinely used antibiotics.

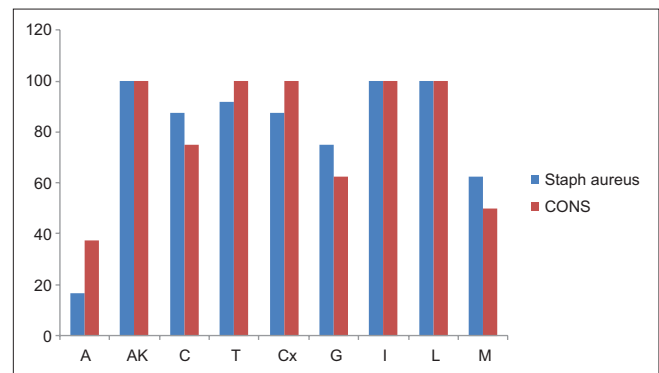


Figure 2: Antibiotic sensitivity in Gram-positive bacteria. A: Ampicillin, AK: Amikacin, C: Ciprofloxacin, T: Cefotaxime, Cx: Ceftriaxone, G: Gentamicin, I: Imipenem, L: Levofloxacin, M: Metronidazole, CONS: Coagulase-negative staphylococci

Imipenem and levofloxacin were the most efficient antibiotics in our study, followed by amikacin and 3rd generation cephalosporins [Figure 4].

DISCUSSION

Wound infection is a major cause of concern among both patients and health-care providers. Not only does it add to the physical and psychological morbidity but also it increases the patient's financial burden and prolongs hospital stay.^[4] In the present study, an attempt has been made to know the various

Table 7: Correlation between duration of wound bacteriology and antibiotic resistance

| Duration of wound (days) | Number of patients | Bacteria isolated | Number | Antibiotic resistance | | | | | | | | | |
|--------------------------|--------------------|--------------------|--------|-----------------------|------|------|------|------|------|------|------|------|------|
| | | | | A | AK | C | T | Cx | G | I | L | M | P |
| 0-7 | 87 | <i>S. aureus</i> | 10 | 80 | - | 10 | - | - | 10 | - | - | 40 | NT |
| | | <i>Klebsiella</i> | 8 | 37.5 | - | 12.5 | - | - | 37.5 | - | - | 50 | NT |
| | | <i>Pseudomonas</i> | 5 | 100 | 40 | 60 | 20 | - | 60 | - | - | 20 | - |
| | | <i>E. coli</i> | 2 | 50 | - | - | 50 | - | 50 | - | - | 50 | NT |
| | | CONS | 4 | 50 | - | 25 | - | - | 50 | - | - | 50 | NT |
| | | <i>Proteus</i> | 3 | 66.6 | - | 66.6 | 33.3 | - | 33.3 | - | - | 66.6 | NT |
| | | N | 7 | | | | | | | | | | |
| 7-14 | 24 | <i>S. aureus</i> | 8 | 75 | - | - | 12.5 | 12.5 | 12.5 | - | - | 25 | NT |
| | | <i>Klebsiella</i> | 12 | 50 | 33.3 | 41.7 | 33.3 | - | 33.3 | - | - | 33.3 | NT |
| | | <i>Pseudomonas</i> | 11 | 100 | 27.3 | 36.4 | 18.2 | 18.2 | 27.3 | - | 9 | 27.3 | 9 |
| | | <i>E. coli</i> | 5 | 20 | 20 | 20 | 20 | 40 | 20 | - | - | 20 | NT |
| | | CONS | 2 | 100 | - | 50 | - | - | - | - | - | 50 | NT |
| | | <i>Proteus</i> | 4 | 50 | 25 | 25 | 50 | 50 | - | 25 | - | - | NT |
| | | N | | | | | | | | | | | |
| 14-21 | 17 | <i>S. aureus</i> | 4 | 100 | - | - | - | 50 | 50 | - | - | 50 | NT |
| | | <i>Klebsiella</i> | 6 | 100 | 66.6 | 66.6 | 33.3 | 66.6 | 83.3 | 33.3 | - | 100 | NT |
| | | <i>Pseudomonas</i> | 14 | 100 | 28.6 | 35.7 | 28.6 | 35.7 | 21.4 | 7.1 | 37.5 | 14.3 | 14.3 |
| | | <i>E. coli</i> | 3 | 33.3 | 33.3 | 66.6 | 33.3 | - | 33.3 | - | - | - | NT |
| | | CONS | 2 | 50 | - | - | - | - | 50 | - | - | 50 | NT |
| | | <i>Proteus</i> | 9 | 66.6 | 44.4 | 44.4 | 55.5 | 33.3 | 66.6 | - | 33.3 | 22.2 | NT |
| | | N | 8 | | | | | | | | | | |
| >21 | 22 | <i>S. aureus</i> | 2 | 100 | - | 100 | 100 | - | 100 | - | - | 50 | NT |
| | | <i>Klebsiella</i> | 2 | 100 | 100 | 100 | 100 | 100 | 100 | - | - | 100 | NT |
| | | <i>Pseudomonas</i> | 16 | 100 | 37.5 | 37.5 | 50 | 37.5 | 56.3 | 37.5 | 56.3 | 100 | 50 |
| | | <i>E. coli</i> | 5 | 40 | 20 | 20 | - | - | 40 | 20 | 40 | 60 | NT |
| | | CONS | - | - | - | - | - | - | - | - | - | - | - |
| | | <i>Proteus</i> | 6 | - | 50 | 83.3 | 66.6 | 50 | 66.6 | 33.3 | 16.6 | 66.6 | NT |
| | | N | 6 | | | | | | | | | | |

S. aureus: *Staphylococcus aureus*, *E. coli*: *Escherichia coli*, CONS: Coagulase negative staphylococci, N: No growth, A: Ampicillin, AK: Amikacin, C: Ciprofloxacin, T: Cefotaxime, Cx: Ceftriaxone, G: Gentamicin, I: Imipenem, L: Levofloxacin, M: Metronidazole, P: Piperacillin, NT: Not tested

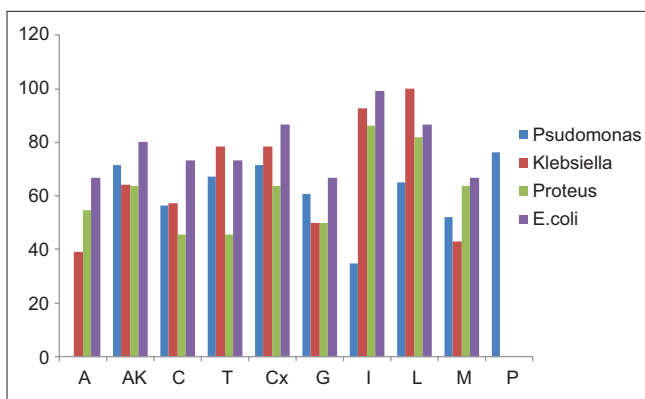


Figure 3: Antibiotic sensitivity in Gram-negative bacteria. A: Ampicillin, AK: Amikacin, C: Ciprofloxacin, T: Cefotaxime, Cx: Ceftriaxone, G: Gentamicin, I: Imipenem, L: Levofloxacin, M: Metronidazole, P: Piperacillin

bacterial flora responsible for wound infections, their antibiogram and to correlate the organisms with risk factors.

Out of the 150 wound swabs collected, 129 (86%) were confirmed to be infected by bacteriological study. The incidence of wound infection was slightly more common

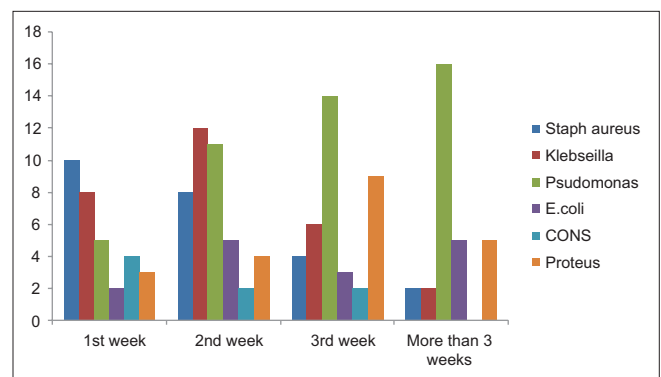


Figure 4: Frequency of bacterial isolates (Y axis) with time (X axis). CONS: Coagulase negative staphylococci

in males (87.2%) than in females (83.9%). This is in keeping with studies done in different parts of the world.^[9-11] This might be explained by traditional male dominance in occupations where trauma is common, namely, farming and industry.

This also accounts for the fact that majority of the infected wounds were seen in the working age group of

20–30 years (91.3) and that the most common wound type was post-traumatic (47.3%) with the legs (44%) and hands (16.7%) being the most commonly affected sites for wound infection.

These findings compare with those done by Pondei *et al.*, 2013.^[12]

Age obviously is an immutable patient characteristic and even if it is a risk factor for wound infection, it appears to be at most a modest one. In our study, the overall culture positivity rate was 86%, whereas 14% of the samples studied were culture negative. A common problem faced by health-care practitioners is managing a patient with all the symptoms and signs of wound infection but with 'no bacterial growth' on the culture report. The incidence of such culture-negative infections can be as high as 30% based on published studies.^[13,14]

Causes of culture negative wound infections:

- Most frequent cause is thought to be culturing the infected site after commencement of antibiotics
- Atypical organisms do not grow on standard culture media. Furthermore, they may grow rather slowly, and the culture plates are discarded before any growth becomes apparent
- Organisms incorrectly dismissed as contaminants (*Staphylococcus epidermidis*, *Corynebacterium* species) when in actual they may be the actual cause of wound infections.

Microbes causing culture negative wound infection and which do not exhibit growth up to 3 days on routine culture media are:^[13]

- Atypical mycobacteria
- Mycoplasma and ureaplasma
- Legionella
- Small colony variant *S. aureus*
- Anaerobes.

Managing wound infections, when microbiological diagnosis is not forthcoming, are a common and challenging clinical problem in surgical practice. Steps to reduce the incidence of culture negative wound infections can be:

- Standard plates to be incubated for an additional 5–7 days, which allows the 'slow growers' to be identified
- Subculture the broth even in the absence of visible growth

- Repeat swabs if diagnosis is doubtful with a request for an additional diagnostic tests, special culture media and repeat cultures
- Include anaerobic media to detect anaerobic pathogens
- Institute appropriate media to detect anaerobic pathogens.

The surgical patient is colonised by microorganisms during his/her stay in the hospital. It is a common observation that chronic illness and a longer hospital stay are associated with wound infection which was evident in our study.

In our study, the sensitivity of *S. aureus*, *Pseudomonas* and *Klebsiella* gradually decreased to routine antibiotics as duration of the wound increased. Beyond the 3rd week, all *Klebsiella* isolates were resistant to all our commonly used antibiotics (except imipenem and levofloxacin).

Longer wound duration may be due to the lack of health-care facilities, failure to report wound infection, fallacies in the diagnosis and management of wounds, longer pre-operative hospitalisation, poor general condition or complexity of the patient's illness. Pre-existing illness may lower the immune status of the patient and may itself contribute to wound infection.

These findings are in accordance with those done by Anvikar *et al.*^[15] In this study, 90.7% of the culture positive wound showed monomicrobial growth and 9.3% were polymicrobial in nature. Fourteen percent had no growth.

Similar high percentage of monomicrobial growth has been reported in different parts of India (86–100%) and Pakistan (98%).^[16-19]

The present study shows the emergence of Gram-negative *bacilli*, accounting for 78% of the isolates, as the principle offenders of wound infection. There is a change in the bacterial aetiology of wound infection from time to time. A century ago, the most feared and frequent pathogen was *Streptococcus*, 20 years back, the coagulase positive *Staphylococcus* was the principle offender, whereas Gram-negative *bacilli* are now replacing *Staphylococcus*.

Our study showed *Pseudomonas* to be the principle cause of wound infection (32.2%) followed by *Klebsiella* (19.6%), *S. aureus* (16.8%), *Proteus* (15.4%), *E. coli* (10.5%) and CONS (5.5%). Our observation of *Pseudomonas* as the most common pathogen in wound infection differs from other

studies in India and rest of the world showing *S. aureus* to be predominant.^[9,15,19-22]

This difference could be attributed to the regional variability of endemic bacterial flora warranting each institution to determine its own set of prevalent microorganisms and associated indices. The frequency of *Pseudomonas* isolates from infected wounds continues to rise with duration of the wound [Figure 4]. On the contrary, the Gram-positive bacteria (*S. aureus* and CONS) showed a decline with duration of the wound.

While *S. aureus* and *Klebsiella* were the most common pathogens in the first 2 weeks, *Pseudomonas* and *Proteus* were most common thereafter.

Antibiotic resistance by the isolates to commonly prescribed and routinely used antibiotics was alarmingly high. The Gram-negative isolates were fairly resistant to commonly used antibiotics such as ampicillin (70.3%), metronidazole (46%), ciprofloxacin and gentamicin (43.2%). Seventy-five percent of Gram-positive isolates were resistant to ampicillin.

All the *Pseudomonas* isolates and 84% of *Staphylococcus* isolates were resistant to ampicillin. Beyond the 3rd week, all the *Klebsiella* isolates were resistant to all routinely used antibiotics (except imipenem and levofloxacin).

The high degree of resistance may be attributed to the widespread abuse of antibiotics, practicing self-medication, indiscriminate use of antibiotics as oral prophylaxis, lack of laboratory services and guidelines/protocols regarding the selection of antibiotics.^[23]

This pattern of antibiotic sensitivity correlates with the study of Anvikar *et al.*^[15] who has reported that organism causing wound infections are resistant to commonly used antibiotics. The increased emergence of penicillin and methicillin resistance of *S. aureus* stains worldwide is reflected in this study where 84% of the *S. aureus* stains were resistant to ampicillin.^[9,15,23]

Imipenem and levofloxacin were the most efficient antibiotics in our study, followed by amikacin and 3rd generation cephalosporins.

CONCLUSION

The most common isolate in wound infection was *Pseudomonas*, followed by *Klebsiella*, *S. aureus*, *Proteus*, *E. coli* and coagulase negative *S. aureus*. These isolates showed a high degree of resistance to ampicillin, metronidazole, ciprofloxacin and gentamicin.

There is an urgent need to curb further spread of antimicrobial resistance, and we recommend the institution of a multidisciplinary approach to wound management, rational drug use, routine microbiological surveillance of wounds and rigorous infection control policies for the same.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Vowden P, Cooper RA. An integrated approach to managing wound infections. European wound management association position document. Management of wound infection. London: Published by MEP; 2006. p. 2-6.
2. Alexander FM. Wound Infection: Nursing Practice Hospital and Home, the Adult. New York: Churchill Livingstone; 1994.
3. Dionigi R, Rovera F, Dionigi G, Imperatori A, Ferrari A, Dionigi P, *et al.* Risk factors in surgery. J Chemother 2001;13:6-11.
4. Sule A, Thanin L, Sule Odu O, Olusanya O. Bacterial pathogens associated with infected wounds in Oguo State University teaching Hospital, Sagon, Nigeria. Afr J Clin Exp Microbiol 2002;3:13-6.
5. Howard JR. Principles of surgery. In: Schwartz S.L., editor. Surgical Infections. USA: McGraw Hill Publishers Inc.; 2010. p. 143-75.
6. Japoni A, Kaloni M, Fenshed SH, Ziyaeyon M, Albuzi A, Mehrabani D, *et al.* Antibiotic resistant bacteria in hospitalised patients with bloodstream infections; analysis of some associated factors. Iran Red Crescent Med J 2010;12:164-72.
7. Noorbaksh Sabet N, Japoni A, Mehrabani D, Japoni S. Multidrug resistant bacteria in Qoui Hospitals, Central Iran. Iran Red Crescent J 2010;12:501-3.
8. National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial susceptibility testing. 11th ed. Wayne, PA: CLSI; 2001.
9. Goswami N, Trivedi HR, Goswami AP. Antibiotic sensitivity profile of bacterial pathogens in postoperative wound infections at a tertiary care hospital in Gujarat, India. J Pharm 2011;2:158-64.
10. Ohalet CN, Obi RK, Emeakoroha MC. Bacteriology of different wound infections and their antibiotic susceptibility patterns in Imo state, Nigeria. World J Pharm Sci 2012;1:1155-72.
11. Amoran OE, Sogebi AO, Fatugose OM. Rates and risk factors

- with SSI in Tertiary care center in South West Nigeria. *Int J Trop Dis Health* 2013;3:25-36.
12. Pondei K, Fente BG, Oladapo O. Current microbial isolates from wound swabs, their culture and sensitivity pattern at the Niger delta university teaching hospital, Okolobiri, Nigeria. *Trop Med Health* 2013;41:49-53.
 13. Poulsen KB, Wachmann CH, Bremmelgaard A, Sørensen AI, Raahave D, Petersen JV. Survival of patients with surgical wound infection: A case-control study of common surgical interventions. *Br J Surg* 1995;82:208-9.
 14. Sonnerwirth AC, Jarret L. Gradwohls, *Clinical Laboratory Methods and Diagnosis*. 8th ed., Vol. 2. Missouri, USA: Mosby Year Book; 1980.
 15. Anvikar AR, Deshmukh AB, Karyakarte R-P, Damle A-S, Patwardhan N-S, Malik A-K, *et al.* A one year prospective study of 3280 surgical wounds. *IJMM* 1999;17:129-32.
 16. Suchitra JB, Lakshmeidei N. Surgical site infections: Assessing risk factors, outcomes and antimicrobial sensitivity patterns. *Afr J Microbiol Res* 2009;3:175-9.
 17. Bibi S, Channa GA, Siddiqui TR, Ahmed W. Pattern of bacterial pathogens in post operative wounds and their sensitivity patterns. *J Surg Pak (Int)* 2012;17:164-7.
 18. Sanjay KR, Prasad MN, Vijay Kumar GS. A study on isolation and detection of drug resistance in gram negative *bacilli* with special importance to post operative wound infection. *J Microbial Antimicrob* 2010;2:68-75.
 19. Basu S, Ramchuran Panray T, Bali Singh T, Gulati AK, Shukla VK. A prospective, descriptive study to identify the microbiological profile of chronic wounds in outpatients. *Ostomy Wound Manage* 2009;55:14-20.
 20. Bhatt C, Lakhey M. The distribution of pathogens causing wound infections and their antibiotic susceptibility pattern. *J Nepal Health Res Counc* 2006;5:22-6.
 21. Mulu A, Moges F, Tessema B, Kassu A. Pattern and multiple drug resistance of bacterial pathogens isolated from wound infection at University of Gondar Teaching Hospital, Northwest Ethiopia. *Ethiop Med J* 2006;44:125-31.
 22. Manikandan C, Amsath A. Antibiotic susceptibility of bacterial stains isolated from wound infection patients in Pattukkottai, Tamil Nadu, India. *Int J Curr Microbiol Appl Sci* 2013;2:195-203.
 23. Hart CA, Kariuki S. Antimicrobial resistance in developing countries. *BMJ* 1998;317:647-50.