Surgical wound infections in King Fahad Hospital at Al-Baha

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The risk of infection is generally based on the susceptibility of a surgical wound to microbial contamination. Clean surgery carries a 1-5% risk of postoperative wound infection, and in dirty procedures that are significantly more susceptible to endogenous contamination, 27% risk of infection has been estimated.[1] The Guideline for Prevention of Surgical Site Infection, 1999 issued by the Centers for Disease Control and Prevention (CDC) classified surgical wound infections as being either incisional (involving skin, subcutaneous tissue, or deeper fascia, and muscle tissue) or organ/space, involving any internal organs or anatomical spaces.[2] Despite the frequency, and prevalence of endogenous anaerobes in surgical wound infections, the CDC guideline for the prevention of surgical site infection (SSI) has recognized *Staphylococcus aureus* (*S. aureus*), coagulase negative *Staphylococci*, *Enterococcus species (Enterococcus spp.*), *Escherichia coli* (*E. coli*), *Pseudomonas aeruginosa* (*P. aeruginosa*), and *Enterobacter species (Enterobacter spp.*) as the most frequently isolated pathogens.[2]

The aim of this study was to determine the etiologies of wound infections, and their susceptibility against antibiotics that used as a prophylaxis in our hospital. This study was carried out at King Fahad Hospital, Al-Baha, Kingdom Saudi Arabia of 694 patients' swabs who undergone surgical treatment (abdominal, vascular, orthopedic, and reparative surgery) from September 2003 to August 2004. All deep wounds processed in aerobic and anaerobic conditions. The age of patients was between 1-120 (average 45) years old. The cultures were evaluated by standard microbiological methods. The identification, and susceptibility tests of microorganisms was performed by either of Microscan (Dade Behring), and BD Phoenix [BD Diagnostic Systems, Sparks, Md. (Becton Dickinson)] automated systems and API (Analytical Profile Index, bioMeriuex, France) systems with standard diagnostic microbiological laboratory methods (for example, coagulase, oxidase, catalase). Disc diffusion method as described by National Committee for Clinical Laboratory Standards (NCCLS).[3] In disc diffusion method, all organisms were tested on Muller-Hinton Agar (Becton, Dickinson, USA), and the following discs were used (obtained from Oxoid, UK): oxacillin (one µg), penicillin (10 U), ampicillin (10 µg), erythromycin (15 µg), clindamycin (2 µg), tetracycline (30 µg), vancomycin (30 µg), amoxicillin clavulanic acid (20/10 µg), cephalothin (30 µg), cefoxitin (10 µg), (30 µg), ceftazidime (30 µg), trimethoprim sulfamethoxazole (1.25/23.75 µg), fucidic acid (10 µg), metronidazole (5 µg), gentamicin (10 µg), amikacin (30 µg), aztreonam (30 µg), imipenem (10 µg), piperacillin (100 µg), ciprofloxacin (5 µg), and norfloxacin (10 µg). Interpretation of zone diameter was based according to the NCCLS guidelines.[5] *Escherichia coli* ATCC 25922, *S. aureus* ATCC 29213, and *P. aeruginosa* ATCC 27853 were included as control. In the 6433 surgical procedures performed in the hospital, 670 (10.4%) patients developed an infection. The 374 (6%) of the 6433 patients was positive culture. On the other hand, 374 (56%) of 670 patients, had positive cultures included different types of microorganisms as seen in **Table 1**. No agent was identified in 153 (23%) of 670 patients, and contaminated samples were 143 (21%), and reported as mixed growth. From positive cultures *S. aureus* (31%) (116 isolate) was the most common cause of wound infections; *P. aeruginosa* (14%) was the second, and followed by *E. coli* (9%), and *Enterobacter spp.* (9%). Methicillin resistance *Staphylococcus aureus* (MRSA) was observed in 19 (16%) of 116 *S. aureus* isolates. *Enterococcus sp.* (5%), coagulase negative *Staphylococcus* (5%), and *Streptococcus* group (3%) were the other large groups isolated in this study. Methicillin resistance was observed in 7 (37%) of 19 coagulase negative *Staphylococcus*. While *Proteus mirabilis* (3.5%), *Klebsiella pneumoniae* (2.4%), and *Acinetobacter baumannii (A. baumannii)* (2%) were other gram negative rods isolated in this study. Five (1.3%) was *Candida spp*, and 4 of them were *Candida albicans*. Ten (2.7%) of the 374 isolates was anaerobic bacteria, and 6 of them was *Bacteroides fragilis (B. fragilis)*. One patient had *Clostridium butilinum* confirmed by second culture. Eight (2%) patient had multiple agents, predominantly *P. aeruginosa* (**Table 2**). Three (<1%) patients had a pure culture of diphteroids with double culture, and polymorph nucleolar leukocytes with gram positive rods seen by gram stain. on the other hand, a small numbers of gram negative rods isolated in this study after confirmed by second culture, like one isolate of *Salmonella* group D, *Hemophylus influenzae*, *Morganella morganii*, and one isolate also for *Flavibacterium spp*, *Alcaligenes feacalis*, *Aeromonas hydrophila*, *Pseudomonas stutzerii*, *Pseudomonas fluorescens*, and *Pseudomonas spp.* Most *S. aureus* isolates were susceptible to all anti-gram positive panels except penicillin, and ampicillin. Methicillin resistant strains (16%) were mostly susceptible only for vancomycin. Most *P.*

Table 1 - Different microorganisms isolated from wound infections.

Aerobic and facultative bacteria	n
*Staphylococcus aureus**	116
Pseudomonas aeruginosa	54
Other *Pseudomonads*	3
Acinetobacter baumannii	8
Escherichia coli	35
Enterobacter cloacae	27
Enterobacter sakazakii	4
Enterobacter species	4
Enterococcus species	13
Enterococcus feacalis	4
Enterococcus feacium	2
Proteus mirabilis	13
Klebsiella pneumoniae	9
Klebsiella oxytoca	2
Citrobacter frundii	3
Serratia marescense	3
Other gram(-) *bacilli***	7
Coagulase negative *Staphylococcus* †	19
Streptococcus	20
Streptococcus pneumoniae	2
Candida species	5
Diphteroids	3
Bacteroides fragilis	3
Bacteroides caccae	1
Bacteroides species	2
Provetella disiens	1
Fusobacterium species	1
Peptostreptococcus species	1
Clostridium butilinum	1
Total	**366**

* - 19 methicillin resitance *Staphylococcus aureus*, ** - One isolate for each; *Alcaligenes feacalis, Aeromonas hydrophila, flavibacterium species, Morganella morganii, Serratia odorifera, Hemophylus influenzae*, and *Salmonella group D*,
† - Methicilin resistant *Staphylococcus epidermis* (MRSE).

Table 2 - Multiple agents isolated from 8 patients.

Multiple bacteria	n
Pseudomonas aeruginosa + *Klebsiella ozanae*	1
Pseudomonas aeruginosa + *Enterobacter cloacae*	2
Pseudomonas aeruginosa + *Staphylococcus aureus*	1
Pseudomonas aeruginosa + *Enterococcus species*	1
Staphylococcus aureus + AGBHS	1
Acinetobacter baumannii + MRSA	1
Acinetobacter baumannii + *Klebsiella pneumoniae*	1
Total	**8**

MRSA - methicillin resitance *Staphylococcus aureus*, AGBHS - A Group Beta Hemolytic *Streptococcus.*

aeruginosa isolates were susceptible to amikacin (93%), piperacillin (85%), ceftazidime (87%), ciprofloxacine (91%), imipenem (96%), netilmicine (92%), tobramycin (92%), and gentamycin (87%). Only 3 isolates were multi resistant, one of these isolates was sensitive to amikacin, imipenem, and other 2 isolates were sensitive to imipenem, and ciprofloxacine only. Most of other microorganisms were multi-sensitive to the antibiotics that was tested in this study except some isolates of *Acinetobacter baumannii*, which were multi-resistant to all drugs except imipenem. Anaerobic species (10 strains) were isolated from different patients **(Table 1)**. Overall, the anaerobic gram positive cocci (one isolates) was susceptible to all the drugs tested, and gram positive bacilli (one isolate) was susceptible to all drugs except trimethoprim/sulfamethoxazole, and clindamicin while the gram-negative isolates (6 *Bacteroides spp*. one *Prevotella spp*., and one *fusibacterium spp*) were shown to be resistant to ampicillin, and cefazolin.

Exposed of subcutaneous tissue for a wide variety of microorganisms provides a favorable substratum to contaminate, colonize, and compromised the host immune response, and if the involved tissue is devitalized (for example, ischemic, hypoxic, or necrotic) the conditions become optimal for microbial growth. Wound contaminants are likely to originate from 3 main sources: (1) the environment (exogenous microorganisms in the air or those introduced by traumatic injury), (2) the surrounding skin (involving members of the normal skin microflora such as *Staphylococcus epidermidis*, *Micrococci*, skin diphtheroids, and *Propionibacteria*), and (3) endogenous sources involving mucous membranes (primarily the gastrointestinal, oropharyngeal, and genitourinary mucosa).[4] The normal microfloras of the oral cavity, gut, and the vagina are both various, and abundant, and these sources (particularly the oral, and gastrointestinal mucosa) supply the vast majority of microorganisms that colonize wounds. Complete microbiological analyses of wounds demonstrate close correlations between the species found in the normal flora of the gut or oral cavity, and microorganisms present in wounds in close proximity to those sites.[5] In our study, we found that a lot of endogenous microorganisms like *E. coli, Enterobacter spp., Enterococcus sp.,* and coagulase negative *Staphylococcus* were the causative agent of surgical wound infections, even most strains of *S. aureus*, and *P. aeroginosa*, which were multi-sensitive and which correlated with these findings. Our results were correlated with previous study, that *S. aureus* (33.5%), *P. aeruginosa* (14%), and *E. coli* (9%) were the top 3 organisms causing infections in our hospital. To summarize our findings of MRSA strains, 3 isolate of *P. aeruginosa* and 2 isolate of

Enterobacter cloacae were multi resistant, and these microorganisms may be acquired from the hospital (exogenous), and not affected by traditional prophylaxis drugs. The infection rate of our study was 6%, which correlated with other studies that showed rates ranged from 1.5-5.9%. A common mistake in different studies, and opinions is that the culture, and isolation of anaerobic bacteria was minimal or omitted, whereas when wounds are investigated by appropriate microbiological techniques, anaerobes are found to form a significant proportion of the microbial population in both acute, and chronic wounds.[6] Due to some anaerobes that are resistant to penicillin, treatment should also include appropriate coverage of those organisms. Surgical management, including drainage, is still the treatment of choice for SSI. The presence of penicillin-resistant anaerobic bacteria, however, such as the *B. fragilis* group, may warrant the administration of appropriate antimicrobial agents, such as clindamycin, cefoxitin, metronidazole, a carbapenem, or a combination of a lactamase inhibitor, and penicillin. In our study, we found that *B. fragilis* (mostly isolated), and other gram negative anaerobic bacilli were shown to be resistant to ampicillin, and cefazolin. Antimicrobial prophylaxis with agents, also effective against anaerobic bacteria (for example, cefoxitin, cefotetan) should be considered, and prospective studies to assess the aerobic, and anaerobic microbiology of postoperative infection are warranted. According to literature data, perioperative prophylaxis can decrease the incidence of wound infection. Cefazolin is the most used agent for surgical prophylaxis in our hospital but can be ineffective against the increasingly common wound pathogens methicillin-resistant *S. aureus*, methicillin-resistant coagulase negative *staphylococci*, *P. aeruginosa*, and other species of gram-negative rods.

In conclusion, this study highlights the polymicrobial nature of SSI and the importance of anaerobic bacteria in SSI's, at same, time the importance of updating surgery prophylaxis to add a stronger antibiotic that may decrease the multi-resistant bacterial infections like MRSA, and *P. aeruginosa*. This study is focused on the candidal infections that are increasing worldwide.[7]

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Prevention of restenosis following choanal atresia repair. *Description of new stent*

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Several surgical approaches for the repair of congenital choanal atresia have been described since its first correction by Emmert in 1854.[1] Stents are usually inserted in both nostrils following surgical repair to prevent the occurrence of postoperative stenosis.[2] However, there is no standard stent used, and all stents have to be fashioned at the time of surgery from soft, and hard materials. However, the most common is the preformed plain endotracheal tube.[3] Alternatives to stenting are serial dilation of the choanae once a week for 4-6 weeks, or regular bougienage every 2 months.

There are several problems associated with the current methods of preventing recurrence of stenosis. This may explain the high incidence of restenosis, which may reach 80%. Stents made of polyvinyl chloride (PVC) soften at body temperature, and may collapse under outside pressure.[4] Repeated anesthetics may unnecessarily subject the newborn to the hazards of anesthesia, and tracheal intubation. Those stents made of rubber or PVC may also induce localized tissue reaction. After the success of using a stent made of reinforced