

# Details of an MRSA Outbreak in an Internal Ward of General Hospital

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## **Introduction**

Nosocomial infections are infections acquired directly or indirectly in a hospital or healthcare services. The term is usually used for the infections that emerge during the hospital admission or up to one month after discharge from hospital. Nosocomial infections are very challenging in the current century due mainly to the resistance of some dangerous microorganisms to previously effective wide spectrum antibiotics. There are many microorganisms that cause hospital acquired infection, the most important of which is *Staphylococcus aureus* causing significant infections in hospital and healthcare facilities worldwide. *Staphylococcus aureus* is resistant to most effective antibiotics so far present on the market, among which methicillin resistant *Staphylococcus aureus* (MRSA) and vancomycin resistant *Staphylococcus aureus* (VRSA) are very critical. According to CDC (2007), MRSA is becoming more prevalent in healthcare settings and its antimicrobial resistance ability is growing very rapidly over time; for instance, in 1974, MRSA infections constituted two percent of the total number of staphylococcal infections; in 1995 it was 22%; while in 2004 it was some 63%.

The importance and significance of multi-drug resistance organisms (MDROs) in the hospital setting is crucial because when an epidemic of one of these organisms occurs in health care facilities, very limited choices are available to treat, eradicate and prevent the recurrence of the infection. Considering this, nowadays, there are efforts to target these infections by implementing and applying preventive and control measures in order to prevent or reduce the chance of emergence or the recurrence of these infections in a hospital setting. Therefore, emergence of an outbreak of MDROs in a hospital or health care facility needs to be fully investigated and all aspects of intervention such as identification of causative pathogen, isolation, preventive measures and treatment, as well as other necessary steps, should be thoroughly investigated, defined and implemented.

## **Statement of a fact, explaining an outbreak in a general hospital**

In a general hospital containing 500 beds and different wards, on Monday the 21<sup>st</sup> of May 2007, a patient in the respiratory section of internal ward developed skin problem resembling a spider bite; the next day, Tuesday the 22<sup>nd</sup> of May 2007, two more patients in the internal ward respiratory section showed the same type of skin infections which started out as painful pimples, resembling spider bites. Consequently, the ward manager decided to ask for a pest control

inspection. The pest control inspectors inspected the respiratory section and entire ward on 22<sup>nd</sup> and 23<sup>rd</sup> of May but no evidence of spider was found and, therefore, no action was taken. On the next day, the 24<sup>th</sup> of May, when doctors visited the same patients they realized that the skin infection had developed into boils and draining sores. These symptoms raised the idea of an outbreak of infection for not only had all nine patients shown almost the same kind of skin infection, signs and symptoms, but also, over the last two days, six more patients had developed the condition, so the number grew to nine, (appendix 1).

The signs and symptoms of all nine patients were mostly similar, including redness, warmth, swelling, tenderness of skin, boils, blisters, folliculitis and impetigo; in addition, some of the patients also had fever and chills.

The hospital administration appointed an executive group to fully investigate the outbreak, to identify the causative agent, to establish preventive and control measures, and to then implement appropriate treatment. In addition, the executive group was assigned to direct staff, allocate funds and review progress daily. The group consisted of chief executive, the director of clinical services, the director of nursing services, the chief executive of another hospital (because of his previous experience in outbreak management), a clinical microbiologist and an infection control nurse. To identify what was the causative agent of this infection, samples collected from all nine patients were sent to the microbiology laboratory for culture, identification and antibiotic susceptibility testing on Thursday the 24<sup>th</sup>.

The executive group decided to meet daily and to make urgent plan for conservative treatment and preventive and control measures to stop the spread of the infection to other yet uninfected patients and staff until the arrival of the final identification of the causative pathogen, and to establish organism-specific appropriate treatment and control measures based on causative pathogen once it is identified. Listed below are the conservative interventions urgently applied until the laboratory results for identification and antibiotic susceptibility tests arrived.

### **Empirical treatment**

On the first day (the 24<sup>th</sup> of May), the laboratory reported that the causative agent of the outbreak of skin infection in the respiratory ward was a Gram positive coccus, but its genus and species were yet to be determined by more laboratory examinations. Based on this information, it was

decided to start a wide spectrum anti Gram positive cocci antibiotic. Since cephalosporins are agents of  $\beta$ -lactam antibiotics, which have a wide spectrum bactericidal activity, and first generation cephalosporins are good choices for Gram positive microorganisms, cefazolin (a first generation cephalosporin antibiotic) was started to be given intramuscularly to all patients who had developed skin infections (appendix 1). According to Rossi (2006), cefazolin is an effective drug against skin infections, for example, cellulites and abscesses and is applied parenterally. Cefazolin was administered to the patients for two days until the result of antibiotic susceptibility test arrived, but apparently there was no evidence of improvement.

### **Conservative preventive and control measures**

In order to prevent the spread of the outbreak to uninfected patients, some intensive control measures were planned and applied to ensure the risk of outbreak dissemination could be eliminated or reduced. These control measures were as follows:

1. Isolation measures,
2. Hand hygiene,
3. Barrier precautions: gloves, masks, gowns,
4. Room disinfection,
5. Appointing some dedicated medical and nursing staff for the isolated patients.

These measures along with other appropriate interventions will be discussed in details later.

### **Identification of the causative agents**

As was discussed earlier, in order to identify the causative pathogen involved in the emergence of the skin infection outbreak, and perform the antibiotic susceptibility testing, samples and swabs collected from all nine patients were sent to microbiology laboratory. There are several methods of detecting the pathogen. The laboratory first performed the Gram stain in order to identify whether the microorganism was Gram positive or Gram negative and whether it was coccus or bacillus. As was reported earlier, it was shown that the pathogen was a member of Gram positive cocci. So, further testing was necessary to identify if this Gram positive coccus was staphylococcus, streptococcus or enterococcus. To do this, first, a catalase test was performed in order to differentiate between staphylococci and streptococci or enterococci. According to Murray et al. (2003, 396), staphylococci are catalase positive while, streptococci and enterococci

are catalase negative. Via the catalase test, it was found that the causative pathogen was catalase positive, which shows that the causative pathogen was a member of staphylococcus species.

In order to confirm the exact species of the causative pathogen, a coagulase test was performed to discriminate between *staphylococcus aureus*, which is coagulase positive, and coagulase negative staphylococci. The result for the coagulase test was positive (coagulase +) which is a marker of *staphylococcus aureus*. But identification was needed to be confirmed by performing either DNase or Chromagar tests. The results of DNase test was also positive (DNase +) which eventually confirmed the identification of *staphylococcus aureus* as the causative pathogen (appendix 1). Generally, according to Costantino (2007, 30), when a Gram positive coccus is both catalase and coagulase positive and the result of DNase test is also positive for this Gram positive coccus, the bacterium is confirmed to be *staphylococcus aureus*.

Agglutination kits were also available for the detection of the causative pathogen, and they were used in parallel to the method described previously after it was shown that the bacterium was a Gram positive coccus. Murray et al. (2003, 222) mention that there are some agglutination kits available which can be used to confirm *Staphylococcus aureus* by detecting protein A and clumping factor, although some strains of MRSA have low levels of these proteins. Newer kits now work by also detecting surface antigen. Other latex kits detect PBP2a (Penicillin Binding Protein 2a) which occurs within the cell membrane and requires lysis of the cells for detection. There are some other methods for the identification of a bacterium too, but methods that were used for this outbreak were the ones discussed previously. On the 25<sup>th</sup> of May, the final identification of causative pathogen was reported to the executive group, but antibiotic susceptibility test was to be done and reported the next day.

The result of the antibiotic test shocked the executive group because on the 26<sup>th</sup> of May, the laboratory reported that the strain of *Staphylococcus aureus* that was causative pathogen of the outbreak was resistant to co-trimoxazole (SXT), tetracycline, methicillin, erythromycin, chloramphenicol and cefazolin which, as Khawcharoenporn (2006, 290) states, are the effective drugs against *Staphylococcus aureus*. This was why the conservative treatment by cefazolin applied before arrival of the final report was not successful. Now it was clear that an outbreak of MRSA had occurred in the respiratory section, which limits the choices of treatment. But,

thankfully, the laboratory report stated that the strain of MRSA involved in this outbreak was susceptible to vancomycin (appendix 1). The results of the laboratory identification and antibiotic susceptibility tests arrived on Sunday the 27<sup>th</sup>.

### **Locating the source of infection**

After the causative pathogen was identified, it was the time to see what or who was the source of infection. A full investigation and examination of the infected patients, as well as HCWs (Health Care Workers) who were in close contact with the infected patients shortly before and during the outbreak, was carried out. Finally, it was seen that the first patient who developed the infection, had a cut on his skin in the axillar region when admitted to hospital on Wednesday the 16<sup>th</sup>. Since *Staphylococcus aureus* is one of the commensal microorganisms on the skin of ordinary people without causing any problem, waiting for a route of entry usually prepared by the break of skin such as cut, injury, surgery, wounds etc, it was confirmed that the cut on the axillar region of the patient paved the way for *Staphylococcus aureus* to cause the infection, and when doctors were visiting him and performing the physical examination they spread the infection to other parts of the chest and abdomen; other patients were infected when they were examined with the same contaminated stethoscope and other medical tools. The other issue was that doctors and other HCWs were not washing their hands after examining the infected patient and before examining others because they were unaware of the infection in the first stage. But the good news was that doctors and other HCWs who were working in the respiratory section were not responsible to examine the patients in other wards, otherwise the infection would have rapidly spread to other sections of the internal ward and eventually throughout the whole hospital.

### **Virulence factors**

In order to react appropriately and efficiently against MRSA in this outbreak, it was important to understand and recognize the virulence factors of this specific microorganism, because MRSA has some virulence factors which are highly potential to cause more serious symptoms and diseases if not treated in the first stage. The whole virulence factors of MRSA are detailed below. It should be noted that not all strains of MRSA have all of the virulence factors, but since determining the virulence factors of each strain of MRSA takes a long time to be determined, the

executive group decided to bring all virulence factors of MRSA into consideration and plan for preventive measurements to avoid any other problem potential to MRSA. According to Todar (2006), *staphylococcus aureus* has many virulence factors, such as:

**A. Surface protein: (Improves colonization of host tissues)**

Surface proteins promote the attachment of *staphylococcus aureus* to host proteins, such as laminin and fibronectin, which form the extracellular matrix of epithelial and endothelial surfaces. Also, some strains express a fibrin- or fibrinogen-binding protein (clumping factor) that enhance attachment to blood clots and traumatized tissue. In addition, adhesin promotes attachment to collagen, causing osteomyelitis and septic arthritis.

**B. Avoidance of host defenses (capsule polysaccharide, protein A)**

*Staphylococcus aureus* also expresses a number of factors that interfere with the defense mechanism of host cells. These factors include:

**Capsular Polysaccharide:** *Staphylococcus aureus* expresses a polysaccharide capsule in its surface. This capsule can prevent phagocytosis in the absence of the complement and avoid lysis of bacterium by bacteriophages.

**Protein A:** *Staphylococcus aureus* has protein A on its surface, the function of which is to bind to IgG molecules in their Fc regions. As a result, the bacteria bind to IgG in an improper orientation on their surface disrupting opsonization and phagocytosis.

**C. Biochemical properties that enables *S. aureus* to survive in phagocytosis (Enzymes)**

*Staphylococcus aureus* has some enzymes having various biochemical properties and functions. For example, Coagulase is an extracellular protein whose function is to bind to prothrombin of the host to form a complex called staphylothrombin. As a result of the protease activity of thrombin, fibrinogen is converted to fibrin. As it was discussed in the identification section, coagulase is a traditional marker for identification of *Staphylococcus aureus* in the clinical microbiology laboratory. Staphylokinase is another enzyme present in *staphylococcus aureus*, the function of which is lysing fibrine. *Staphylococcus aureus* also produces some other extra-cellular enzymes such as DNase lysing DNA of host cell, fatty acid modifying enzyme, FAME, lipase that helps MRSA to spread deep into cutaneous and subcutaneous tissue, catalase that

helps microorganism to resist phagocytosis, fibrinolysins that breaks down fibrin and helps to invade tissue, hyaluronidase that helps with the invasion of the tissue, and protease the function of which is clear from its name.

#### **D. Toxins (able to damage membrane and lyse eukaryotic cell wall)**

*Staphylococcus aureus* also produces toxins that directly act on polymorphonuclear leukocytes (leukocidin, leukotoxin). Since polymorphonuclear leukocytes are important for phagocytosis and phagocytosis is an important factor against *staphylococcus aureus*, leukocidin is a very important virulence factor. *Staphylococcus aureus* also produces some types of superantigens in form of enterotoxins (ET) causing diarrhea, epidermolytic toxins causing scalded skin syndrome and toxic shock syndrome toxin (TSST) which causes toxic shock syndrome. The exfoliatin toxin causes the scalded skin syndrome in neonates, which results in widespread blistering and loss of the epidermis. There are two antigenically distinct forms of this toxin, ET-A and ET-B. The toxins have esterase and protease activity and apparently target a protein which is involved in maintaining the integrity of the epidermis.  $\alpha$ -toxin ( $\alpha$ -hemolysin) is the strongest and best characterized toxin that damages cell membrane. It is expressed as a monomer which is able to bind to the membrane of susceptible cells. Platelets and monocytes are particularly sensitive to  $\alpha$ -toxin. Susceptible cells have specific receptors for  $\alpha$ -toxin which allows the toxin to bind with the cell and act on its target.  $\beta$ -toxin is a sphingomyelinase and can damage membranes that have much of this lipid.

*Staphylococcus aureus* expresses different types of protein toxins responsible for symptoms during infections. Those which damage the membranes of cells were discussed above. Some also lyse erythrocytes, causing haemolysis; leukocidin causes membrane damage to leukocytes. Staphylococcal enterotoxins cause emesis (vomiting) when ingested, and in fact, the bacterium is a leading cause of food poisoning.

Moreover, Miller et al. (2005, 1445) points out that MRSA also carries a genes coding for Penton Valentine Leukocidin (PVL), a toxin responsible for the lysis of white blood cells. PVL is proven to be responsible for severe haemolysis and necrotic pneumonia in children too.

### **E. Inherent and acquired antimicrobial resistance**

*Staphylococcus aureus* became resistant to methicillin by acquiring a gene called *mecA*. It is usually located on a large DNA sequence called staphylococcal cassette chromosome *SCCmec*. According to Guignard et al. (2005, 480), the presence of *mecA* reduces the affinity of PBP2a (Penicillin binding protein 2a) for binding with  $\beta$ -lactam antibiotics. Penicillin binding proteins are necessary for correct synthesis of the bacterial cell wall, so when these proteins are blocked by penicillins the cell walls are incorrectly formed and are easy to be lysed. However, in the case of MRSA, the presence of PBP2a allows the bacterium to correctly synthesize the cell wall in the presence of methicillin too. Some strains of MRSA also show  $\beta$ -lactamase, and therefore can resist all  $\beta$ -lactam antibiotics including all penicillins and cephalosporins.

As can be seen from the outline above about virulence factors of *Staphylococcus aureus*, this microorganism was able to generate a more serious outbreak if not treated in the earliest. Therefore, as soon as the final confirmed laboratory report arrived, treatment of infected patients started.

### **Treatment**

As was pointed out earlier, the laboratory antibiotic susceptibility results showed that the strain of MRSA responsible for this outbreak was susceptible to vancomycin. Therefore, the executive group decided to start the treatment of the infected patients with vancomycin. Vancomycin was indicated for the patients, 500 mg every six hours in a dilute solution intravenously. Each dose was administered over at least one hour (no more than 10 mg/minute) to prevent the incidence of pain and thrombophlebitis and avoid infusion reaction commonly known as 'red-man-syndrome'. Vancomycin was used until the infection was eliminated and colonization-site cultures confirmed eradication of the outbreak.

Moreover, in addition to using vancomycin, for patients who developed abscesses and purulent sores, health care staff performed incision and drainage (I & D) procedures in order to drain the pus from the infected area (appendix 1).

## **Prevention and control measures**

Although before the confirmation of the causative pathogen some preventive and control measures were employed, after the identification of MRSA as the causative pathogen, specific preventive and control measures were planned and intensively implemented because MRSA is a very rapidly-spreading microorganism and there are so few choices for its therapy. In this case, the most important and significant issue is to control the spread of the infection from infected individual to uninfected patients and staff and undertake some preventive issues to prohibit the recurrence of the infection in the future. The following control measures were planned and implemented; the rationale of each control measure is explained based on very recent literature on the topic (appendix 1).

1. Screening and detection of MRSA carriers
2. Isolation measures,
3. Hand hygiene,
4. Barrier precautions: gloves, masks, gowns,
5. Room disinfection,
6. Covering of the infected site with dry bandage, and eliminating the removed bandages,
7. Decolonization of MRSA,
8. Surveillance of MRSA in the hospital, and
9. Reduction of the time of hospital stay.

### **1. Screening and detection of MRSA carriers**

Screening was the first step considered in the control and intervention measures. Screening of MRSA carriers, hospitalized patients and staff was needed to detect MRSA carriage. According to Nicole (2007, 7), screening of patients and staff is initially recommended in the outbreaks of MRSA and should be performed in outbreak situations, in patients transferring from another hospital, patients transferring from other units and patients transferring from nursing homes to find the source of infection. Screening the localization site of *staphylococcus aureus* is important and should be done on localization sites, such as the throat, axillae, nose, perineum, wounds etc. Screening of hospital staff on outbreak situation is encouraged and should be done for occupational health safety. Kluytmans (2007, 104) points out that generally, screening of patients and staffs helps to detect the infected patients as well as asymptomatic carriers and shows the

burden of the infection in the health care facility. The writer points out that, in the outbreak of MRSA, active screening is mandatory for the detection of the source of infection and establishing other control measures. Based on this information, all patients in respiratory sections and other patients accommodated in internal ward, as well as the staff who had close contact with them were screened to detect the unknown asymptomatic carriers.

## **2. Isolation measures**

As soon as the first case of MRSA was detected, a full screening of patients in the hospital was planned to be done to detect other unknown cases and to isolate the ward contacts and asymptomatic carriers. According to Kanerva (2007, 25), after the detection of the cases, there are two immediate steps to be considered.

1. Isolation of individual cases in private rooms to prevent the spread of infection to other people. Nursing staff that are examining or visiting the infected people should wash their hands thoroughly and change their gowns and gloves after each visit.
2. Curran et al. (2006, 376) suggests that if private room isolation was not possible because of any reason, cohorting of the infected patients in a special ward should be considered.

Therefore, all MRSA infected patients and asymptomatic carriers were cohorted in a multibed room in respiratory section (the infection was just limited in patients of respiratory section) because single bed room was not available. Isolation is very important in prevention of the spread of the microorganism from infected individuals to uninfected patients and staff and should really be considered the first step after detection of the cases. Furthermore, staff assigned to the cohort were only to work with cohort patients or in other words, crossover of the HCWs was avoided. All staff were to wash their hands thoroughly before leaving the cohort wards.

## **3. Hand hygiene**

The principle mode of transmission of MRSA in a hospital setting is considered to be from one infected or colonized individual to uninfected person via the hand of a HCW (Mayhall 2004, 476); in addition, one of the normal localization of *staphylococcus aureus* is skin; so cleaning hands thoroughly or hand hygiene is one of the most important issues that staff should consider. Hand hygiene was recommended to all staff attending the cohort of MRSA infected patients. Full

training sessions for all dedicated staff who were designated to work with cohort was established on the first day. Meanwhile, several preparations used for hand hygiene and easy accessibility to them were provided. As documented in WHO's (2002) guideline for hand hygiene in health-care settings, despite its very simplicity, washing hands with alcohol-based hand rubs, antimicrobial soap, antiseptic hand-wash, and even normal detergents is very effective in hand hygiene and consequently control of the infection. According to the report, several preparations used for hand hygiene, for example, plain or non-antimicrobial soaps, alcohol, chlorhexidine, chloroxylenol, hexachlorophene, iodine and iodophors, quaternary ammonium compounds, triclosan, and many more are available for hand hygiene, every one of which has special properties and purposes. A very important matter in this regard is that hand-washing materials should be easily and readily accessible for the caring staff, or otherwise, the adherence to the hand hygiene will come under question.

The report adds that providing easy access to hand-hygiene materials is mandatory for appropriate hand-hygiene behavior and is achievable in the majority of health-care facilities. In particular, in high-demand situations (e.g. the majority of critical-care units), under hectic working conditions, and at times of overcrowding or understaffing, health care workers (HCWs) may be more likely to use an alcohol-based hand rub than to wash their hands. Further, using alcohol-based hand rubs may be a better option than traditional hand-washing with plain soap and water or antiseptic hand-wash, because they not only require less time, but act faster and irritate hands less often. According to CDC (2007) some specific indications for hand hygiene are before patient contact, before putting on gloves and urinary catheters, or any other invasive devices that don't require surgery, and after contact with a patient's skin, after contact with body fluids or excretion, non-intact skin, and wound dressings, and after removing gloves. All the above mentioned information was covered in the training sessions and, as was discussed earlier, easy access to hand-hygiene materials was provided for all staff.

#### **4. Barrier precautions: gloves, masks and gowns**

All HCWs and staff were recommended to wear gloves, gowns and masks while attending each infected patient. In fact, gloves are very important preventive measures because they prevent dissemination of infection in three ways: first they protect HCW's hand to be infected by infected patients; second they protect HCW's hand to be infected by infected equipment; and third, they

protect other uninfected patients to be infected by HCWs. It needs to be emphasized that it was extremely emphasized that all health care workers change their gloves after examining each patient and wash their hands thoroughly after each gloves removal.

Meanwhile, masks were important for the protection of health care workers themselves. These were compulsory to be worn during intensive patient contact and close contact of colonized sites; in such a case, it reduces the risk of transient carriage by 50% (Lacey 2001, 308). He also continues that wearing mask decreases cross-contamination of hands from nose and throat. Mayhall (2004, 476) reveals that health care workers are twice likely to be at least transiently colonized by MRSA from an infected patient, if they do not wear a mask when taking care of a patient with MRSA.

Gowns were also effective protective measures and were recommended to be changed after examining each infected patients. All provided gowns were long sleeve and impermeable in order for microorganism not to be able to cross the gown and infect the HCW and consequently the other uninfected people. Hand-washing and hand hygiene was highly recommended after changing the gowns.

## **5. Room disinfection**

Since the beginning of detection of MRSA until complete eradication of MRSA from hospital, standard cleaning of MRSA patients' rooms was implemented; in addition, during the epidemic of MRSA, some reinforced cleaning by detergents containing disinfectants were established and carried out daily and routinely. In addition to room disinfection, linen and waste were also carefully handled according to infection-control standards to avoid HCW and environmental contamination. CDC (2007) recommends that the use of specific bags, and common-sense hygienic practices for storage and processing of linen is sufficient and there is no need for rigid rules and regulations.

Precaution and room disinfection measures were continued, full-scale, until the eradication of the outbreak of MRSA.

## **6. Covering of the infected site with dry bandage and eliminating the removed bandage**

Some patients developed draining sores or purulent ulcers; thus, their draining sores were covered by dry bandages to prevent the dissemination of MRSA. Indeed, covering of the infected site with a dry bandage helped inhibit the spread of MRSA from the infected wound or skin infection to other material or people. According to WHO's (2007) guidelines, keeping the infection dry with a clean bandage is especially important for infections that continue to produce pus or are purulent. The removed covers or bandages were taken away from the ward and eliminated because they had the bacteria and could potentially spread the infection to others. Washing hands after removing bandage was absolutely recommended.

## **7. Decolonization of MRSA**

Another important issue was to decolonize MRSA from normal colonization in order to prevent the spread of the infection from colonization areas to uninfected people and consequently eliminating outbreak. According to Byl (2003, 13), decolonization of MRSA carriers can be done by decontamination of patient carriers and staff by applying nasal mupirocine ointment, body wash with antiseptic soap, and general or local appropriate antibiotic treatment after culture and antibiotic susceptibility testing. This will eradicate the colonization of MRSA and subsequently reduces the amount of contact of uninfected individuals with the microorganism preventing the spread of outbreak. Although the effect of decolonization seems to be marginal, but there are some special circumstances that requires this attempt. WHO (2007) confirms that patients who are immunosuppressed and colonized, those who have the potentiality to gain MRSA from their normal flora, patients who are mentally retarded and are likely to spread the organism due to their behavior, and those who had the history of repeated infection by MRSA strains that they carry, are the important candidates of decolonization.

## **8. Surveillance of MRSA in the hospital**

Surveillance of MRSA in the hospital was undertaken to understand the burden of infection, the level of success achieved, the intensity of intervention required, and the point that intensive control measure should be decreased. Regarding this issue, Nicole (2007, 10) states that effective infection control programs including surveillance, control activities, and appropriate management decrease the frequency of endemic nosocomial infections by 30% to 50%. Carlene et al. (2003,

364) emphasize that surveillance should be done intensively and should include the detection of infected patients and carriers-by performing necessary laboratory tests and examinations, and culture of blood, wound secretion and normal colonization of *Staphylococcus aureus*. They argue that active surveillance cultures (ASCs) are very important in eradication of the outbreaks of MRSA. Surveillance gives a good understanding of the effect of intensive control measures and the level of the success achieved by using a lot of resources for this issue. In fact, surveillance directs and clarifies what important steps should be considered next, to completely eradicate the outbreak.

### **9. Reduction of the time of hospital stay**

The period of hospital admission was reduced for patients admitted to hospital and it was planned that patients should not stay unnecessarily in hospital for a long time because it could predispose them to the infection. According to Cosgrove et al. (2005, 170), the risk of getting bacteraemia in patients staying longer in the hospital is increasing significantly. They found out that MRSA bacteraemia is highly associated with long period of hospitalization and is 1.30 times more compared with the shortest necessary stay in hospitals. It is also associated with increased hospital charges and unnecessary financial burden.

Generally, to avoid the recurrence of the infection, all intensive control steps and preventive measures were continued until the confirmation of the eradication of MRSA from hospital (respiratory section of internal ward) by laboratory examinations and active surveillance cultures from patients and environment. Also, it was highly recommended that all visitors who tended to visit their MRSA infected patients wear gloves, mask and, if it were to last too long even impermeable gown to avoid being infected and carrying the infection out of the hospital. Every person, from HCWs to visitors were to abide by the critical intensive infection-control policies, especially hand hygiene after visiting or contacting an MRSA infected patient before leaving the cohort room.

### **Result**

Applying all intensive control and preventive measures and careful, observed treatment the outbreak eliminated in three weeks. It is worth mentioning that all the intervention measures

implemented against the outbreak cost the hospital 850,000 dollars. After the final screening and active surveillance culture, officially the eradication of the outbreak was reported to the hospital administration on Monday the 18<sup>th</sup> of June, 2007 (appendix 1).

### **Conclusion**

Nosocomial pathogens, which are pathogens involved in hospital acquired infections, are serious issues in hospital settings. Early detection, identification, and appropriate action against these pathogens are the key elements to the successful eradication of an outbreak caused by these microorganisms. Among nosocomial pathogens, MRSA, or methicillin resistant *Staphylococcus aureus* is very common. In fact, infection caused by *Staphylococcus aureus* has become an increasing problem in hospital settings. MRSA is also resistant to many other alternative drugs which are effective against ordinary *Staphylococcus aureus*; for example, penicillins and first generation of cephalosporins. In this essay, an outbreak of MRSA in a respiratory section of a 500 bed-hospital internal ward was explained. Generally, in the case of an infection outbreak in a hospital, the identification of the causative agent should immediately be started and until arrival of the result of final identification and antibiotic susceptibility tests, some conservative treatment, prevention and control measures should be planned and carried out. As soon as the causative agent is recognized and appropriate antibiotic is identified, the proper treatment and organism-specific prevention and control measures should be designed and implemented.

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Appendix 1:

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