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HEALTHCARE ASSOCIATED INFECTIONS: A HAZARD TO PUBLIC HEALTH

Dr. Laigy Mariam Mathias, Dr. Leemol Lalu and Dr. Mable Marion*

Department of Pharmacy Practice, Bapuji Pharmacy College, Davangere, Karnataka.

***Corresponding Author: Dr. Mable Marion**

Department of Pharmacy Practice, Bapuji Pharmacy College, Davangere, Karnataka.

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ABSTRACT

Healthcare associated infection (HAI) is localized or systemic condition resulting from adverse reaction to the presence of infectious agent or its toxins acquired from healthcare settings that was not incubating or symptomatic at the time of admission to the healthcare facility. HAI is increasingly becoming a major global public health problem posing great threat to patient safety and wellbeing of healthcare providers. HAIs include Surgical Site Infections(SSI), Ventilator Associated Pneumonia(VAP), Central Line Associated Blood Stream Infections (CLABSI), Catheter Associated Urinary Tract Infections(CAUTI), Methicillin Resistant S. aureus (MRSA), clostridium difficile infections. HAIs have significant consequences on patients, their families, and the community as a whole. The most common consequences of HAIs are increased morbidity, mortality, and length of hospitalization. Such consequences contribute substantially to raise both the direct and indirect cost of the health care services, which result in additional costs to treat infected cases. Infection control in hospitals and their ICUs is extremely challenging. Infection control professionals (ICPs) play a critical role in leading HAI-reduction interventions, and are responsible for the implementation and ongoing management of such interventions across hospitals and their ICUs. Proper education and training of health care workers increases compliance with and adoption of best practices to prevent HAIs. Despite the development of many hi-tech methods, hand washing with soap and water or alcohol rub is still the most important means of maintaining personal hygiene and preventing HCAIs. We performed a MEDLINE search using combination of keywords such as hospital associated infection, ventilator associated pneumonia, surgical site infections, central line associated blood stream infections, intensive care units. We reviewed the relevant publications with regard to VAP, SSI and CLABSI. We selected 35 relevant publications with some important cross references from the list of publications. The aim of this review conducted was to estimate the incidence, the major related risk factors, outcomes for acquiring VAP, SSI and CLABSI in various hospitals. This helps to provide a standardized tool for hospitals to identify target areas for quality improvement.

KEYWORDS: HAI is increasingly becoming a major global public health problem posing great threat to patient safety and wellbeing of healthcare providers.

INTRODUCTION

HAIs accounts for a large proportion of damages caused by healthcare processes in both developed countries and low income settings.[1] The impact of HAI implies prolonged hospital stay, long-term disability, increased resistance of micro-organisms to antimicrobials, a massive additional financial burden for health systems, high costs for patients and their families, and excess deaths.[16] HAI affects about 7.6% of patients in regular wards and at least half of those admitted to intensive care units (ICU) in developed countries. The magnitude of the problem in the low-income settings remains largely unknown and in most cases underestimated due to the complex nature of its diagnosis and lack of proper surveillance. Studies conducted in low-income settings showed that hospital-wide prevalence of HAI is about 15.5 per 100 patients which is much higher than reports from Europe and North America. As a matter of fact, most of the global reports of HAIs are focused on prevalence and there are only very few reports about the incidence rate of HAIs.[1] The Centers for Disease Control (CDC) estimates that HAIs account for an estimated 1.7 million infections and 99,000 associated deaths each year. Of these infections: 22 percent are surgical site infections, 15 percent are ventilator associated pneumonia & 14 percent are bloodstream infections. The surveillance of HAI is regarded as an essential part of infection control and prevention.[10] The most common pathogens isolated from the published studies were Pseudomonas auruginosa, S. aureus, E.coli, Acinetobacter, Klebsiella, Enterobacter and Citrobacter . Although eradication of HAI is impossible, a well-conducted surveillance and prevention program may significantly reduce HAI and associated costs. Continuous prospective surveillance for HAIs is the gold

standard, this approach requires comprehensive resources.[15]

VENTILATOR ASSOCIATED PNUEMONIA DEFINITION

VAP is defined as pneumonia that occurs 48h or more after endotracheal intubation or tracheostomy, caused by infectious agents not present or incubating at the time mechanical ventilation was started. It can be of two types: (i) early-onset VAP which is defined as VAP that occurs within the first 4 days of ventilation, and (ii) late-onset VAP which is defined as VAP that occurs more than 4 days after initiation of mechanical ventilation.[3] VAP is the most frequent HAI in the intensive care unit (ICU). Between 10% and 20% of patients receiving 48 hrs of mechanical ventilation will develop VAP.[31] VAP is associated with prolonged hospital stay increased cost, and an excess risk of mortality.[3,12] The most important mechanism of infection is aspiration of oropharyngeal organisms into the distal bronchi, followed by bacterial proliferation and parenchymal invasion. Inflammation of the bronchial wall involves the alveolar septa and air spaces leading to bronchopneumonia.[31,34] Symptoms include at least two of the following appearing during hospitalization: cough, purulent sputum, new infiltrate on chest radiograph consistent with infection.[1]

Risk Factors of Vap

Multiple factors have been identified as potential causes for VAP which. The duration of mechanical ventilation is an important risk factor for VAP.[2] We observed that the incidence of VAP increased in patients who were on mechanical ventilation for >15 days as compared to those who were ventilated for <15 days.[3] Supine position of mechanically ventilated patients increases the risk of VAP as compared to patients in semi-recumbent position.[35] Comatosed state was found to be a risk factor to develop VAP in ventilated patients.[2] Discontinuation and reinstitution of mechanical ventilation and tracheostomy was proved to be an independent risk factor of VAP in various studies. Many studies have shown that traumatic patients are at increased risk of developing VAP compared to medical patients.[3] The higher incidence of VAP in comatosed, supine positioned, traumatic, reintubated patients who are ventilated for a prolonged period of time may be due to increased the risk of aspiration.[2] Stress ulcer prophylaxes such as PPIs and H2 receptor antagonists were found to predispose the ventilated patients to develop VAP.[26] Sucralfate appeared to have a small protective effect against VAP because it doesn't not raise the gastric pH like PPIs and H_2 receptor antagonists. Therefore whenever feasible, Sucralfate should be used instead of H_2 receptor antagonists.[2] Another risk factor which was evaluated from the studies was the administration of broad spectrum antibiotics in the preceding seven days. This may be due to the development of subsequent colonization with resistant pathogens responsible for super infection.[3] A high incidence of VAP was due to lack of adequate nursing staff (which should be ideally 1:1) which may have adversely affected the quality of care given to patients. Comorbid conditions like hypertension, diabetes, immunosuppressed state (smoking and alcoholism) which were associated with older age were found to risk factors of VAP in most of the studies.

Diagnosis of Vap

Certain diagnostic criteria were used in the published studies. A diagnosis of ventilator-associated pneumonia was considered only in patients who were receiving mechanical ventilation or who stopped receiving ventilation for less than 48 hours.Each case of pneumonia was classified according to four additional definitions: 1) the bedside clinician's diagnosis 2) the modified Centers for Disease Control and Prevention criteria, which require a new radiographic infiltrate persistent for 48 hours or more plus a body temperature greater than 38.5 °C or less than 35.0 °C, a leukocyte count of more than 10 cells x 109/L or less than 3 cells x 109/L, purulent sputum or change in character of sputum, or isolation of pathogenic bacteria from an endotracheal aspirate 3) a Clinical Pulmonary Infection score of 6) a positive culture from bronchoalveolar lavage (>104 colony-forming units/mL) or protected specimen brush (<10 colony-forming units/mL)[26,27]

Microbiology of Vap

Awareness of the microbiology of VAP is essential for selecting optimal antibiotic therapy and improving these outcomes. The specific microbial causes of VAP are many and varied. Most cases of VAP are caused by bacterial pathogens that normally colonize the oropharynx and gut, or that are acquired via transmission by health-care workers from environmental surfaces or from other patients.[34] Pseudomonas aeruginosa was the most common Gram-negative bacterial pathogen isolated from therespiratory tract among infected patients with VAP for both survivors and non-survivors. Staphylococcus aureus was the most common Gram-positive bacterial pathogen associated with VAP.[27] Other common pathogens isolated in most studies were E.coli, Acinetobacter, Klebsiella, Enterobacter and Citrobacter.[3,35]

Outcomes of Vap

The mortality rate of patients developing VAP was significantly greater than the mortality rate of patients without VAP. Patients who died during their hospitalization werestatistically older, had greater APACHE II scores, lower Pao2/Fio2 ratios, were more likely to have bacteremia, be immune-compromised, develop acute renal failure, have congestive heart failure, compared to patients who survived their hospitalization. The patients who diedmore frequently underwent dialysis, required multiple central venous lines, and received treatment lessfrequently with histamine type-2 receptor antagonists and antacids. Similarly, the duration of mechanical ventilation was significantly longer among hospital non-survivors. Secondary Clinical Outcomes

included significant longerlengths of stay in the ICU and in the hospitalcompared topatients without VAP and increased healthcare cost.[1,27]

Prevention of Vap

A prevention policy aiming to reduce VAP remains an important element of the management for patients admitted to ICUs and requiring mechanical ventilation. The current preventive strategies for VAP are mainly directed at colonization and aspiration modification, such as avoiding intubation oral care assessing for early weaning and mobility and prophylactic probiotics. Hand washing is widely recognized as an important but underused measure to prevent nosocomial infection. Hands should be washed before and after patient contact and in between patient contact.[2]

Minimize Ventilator Exposure: The first choice for lowering VAP risk is minimizing a patient's exposure to mechanical ventilation. The use of non-invasive ventilation approaches is encouraged, such as bi-level positive airway pressure (BiPAP) or continuous positive airway pressure (CPAP). If mechanical ventilation can't be avoided, work to minimize its duration. Ventilator weaning protocols (for example, daily interruption of sedation and coordination with a spontaneous breathing trial) or evidence-based care bundles can be effective in shortening mechanical ventilation duration.

Subglottic suctioning endotracheal tubes: It has been postulated that intermittently or continuously removing the secretions that pool above the ETT cuff may reduce the risk for aspiration and subsequent development of VAP. ETTs have therefore, been designed to accomplish this task through application of negative pressure to a separate port that opens above the ETT cuff.

Head of bed elevation: Elevation of the head of the bed is attempted to reduce aspiration of gastric content. The basis for this intervention comes from studies which have shown that aspiration of gastric contents occurs to a greater extent in supine patients than in patients in a semi-recumbent position.

Antimicrobial-coated endotracheal tubes: ETTs coated with antimicrobial substances have been studied as a means to decrease bacterial colonization and prevent biofilm production with the ultimate hope of reducing VAP rates. It is hypothesized that microorganisms reach the ETT either as a consequence of a contaminated oropharynx or reflux of gastric secretions. The use of a silver-coated ETT has been proposed as a method to reduce biofilm production. It is well known that silver has broad-spectrum antimicrobial activity, decreases bacterial adhesion in vitro and blocks biofilm formation.

Oral decontamination: Oral health quickly deteriorates in mechanically ventilated patients. Some patients sustain injuries to the oral mucosa during the intubation procedure, and after intubation, patients are prone to dry mouth. These factors, in addition to a severely compromised immune system, can cause an increase in bacteria colonization in the oral mucosa, with the endotracheal tube serving as a direct route to the lungs. Oropharyangeal decontamination with chlorhexidine solution has been shown to reduce the occurrence of VAP.[2]

Probiotics: A new strategy in the fight against VAP is the use of probiotics. Probiotic bacteria can decrease the development of VAP through local and systemic actions that improve intestinal barrier function, increase host cell antimicrobial peptides, regulate the composition of the intestinal flora, and reduce overgrowth of pathogenic bacteria and bacterial translocation.

Surgical Site Infection

Defnition

Surgical site infection previously termed as post-operative wound infection is defined as any infection presenting upto 30 days after a surgical procedure and up to 1 year if a prosthetic is implanted in the patient.[25] Surgical site infections are the third most commonly reported nosocomial infection that has an adverse impact on hospital as well as on the patient.[22] SSIs remain a major cause of morbidity and death among surgical patients.[28] It is also responsible for increasing the length of hospital stay which results in social and economic loss to patients and family.[22] Studies have found that rate of SSI are globally increasing even in hospitals with the most modern facilities and standard protocols of pre-operative preparation and antibiotic prophylaxis.[28] Rate of SSI can vary greatly worldwide from hospital to hospital.[11] Symptoms of surgical site infection include fever, pain at surgical site, swelling and pus from wound site.[9]

Risk Factors of Ssi

There are several risk factors for the development of surgical site infection likelevel of patient, procedure, hospital setting ,surgical team practice,wound class, previous surgery, age above 40 years, preoperative hospital stay more than 7 days, administration of antimicrobial prophylaxis before 1 hour of skin incision are all independent predictors of SSIs.[4] The mechanism of surgical site infection in elderly and general anaesthesia patients are found that they are unable to provide a robust defence against pathogens because of their weak or depressed immune system, under normal circumstances bacteria in abdomen maintain an ecological balance,abdominal surgeries could upset this balance and change proportion of bacteria thereby inducing an SSI.[23] Studies have shown that administering antimicrobial prophylaxis within 1 hour before skin incision and shortening of pre-hospital stay and duration of operation further decreased the incidence rate of SSIs.[4] Some other studies showed that rate of SSI was significantly higher in diabetic and female patients. Also contaminated operations contributed more number of SSIs than other procedures

conducted.[25] Prolonged duration of operation results in increased exposure of operation site to air, prolonged trauma, prolonged anaesthesia and blood loss can all cause infections. The use of drains in contaminated, dirty wounds and in emergency and prolonged operations has increased the probability of wound getting infected. Also certain underlying factors like anaemia, smoking, DM may alter or decrease the immune status thus significantly increasing the risk of SSI.[22] The most commonly isolated organism in most cases are Escherichia coli, Pseudomonas aeruginosa and S.aureus.[22] Other factors such as Body Mass Index, Video assisted procedures, blood transfusion, general anaesthesia, non-performance of pre-operative bath and pre-existing chronic diseases can all be considered as risk factors of SSI.[5]

Diagnosis of Ssi

A surgical site infection can be primarily diagnosed from the appearance of wound,it includes at least one of the symptoms like purulent discharge,tenderness, swelling followed by clinicians diagnosis,organism isolated from the organ space by aseptic culturing technique, abscess formation identified during direct examination or during histopathologic examination.[9] Exudates were collected from wounds using sterile swabs which were transported at room temperature to lab within 15 min of collection and these swabs will be inoculated on chocolate, blood, and Macconkey agar and incubated for about 48-72 hours and gram stain procedure performed on culture growth to report organism. Susceptibility testing can be done by Kirby-Bauer disk diffusionmethod.[28]

Microbiology of Ssi

SSIs are preventable complications which can also increase patient burden, morbidity, mortality and increase the cost of treatment.[4] Cause of SSIs may differ from patient to patient but the most common cause is due to microorganisms.[22] The most commonly isolated organism in most cases are Escherichia coli, Pseudomonas aeruginosa and S.aureus. Multi drug resistance is a dreaded problem in infection it happens because prophylactic antimicrobials are given to patients who are at poor risk and they develop tolerance. Studies showed that Enterobacters show highest sensitivity to amikacin followed by gentamycin and very low sensitivity is noted with cephalosporins and fluoroquinolones. Vancomycin and Linezolid have shown good sensitivity Staphylococcus aureus.[22]

Outcome of Ssi

SSIs can be the primary cause of morbidity and death among operated patients.[28] Patients who develop SSIs are 60% more likely to spend time in an intensive care unit, 5 times more likely to be readmitted to hospital and 2 time more likely to die compared with patients without SSIs.[4] Studies conducted in western countries suggest that 2 to 5% of patients undergoing clean, contaminated surgeries and upto 20% undergoing intra-abdominal surgery develop SSIs.[4] The risk of SSIs can continue even after discharge as they develop in almost 2 percent of patients after discharge and are two to five times more likely to be readmitted to the hospital.[28] In short surgical site infections can increase morbidity, mortality, increased hospital stay and increased cost.[4,22]

Prevention of Ssi

From the studies it is found that SSIs are the second most common infections occurring in hospitalized patients following surgery hence SSI surveillance is integral to the hospital infection control and quality improvement programs. Prevention measures that are mentioned in studies are.

- Efforts to decrease the duration of surgeries performed without compromising patient's safety and beneficial outcome.
- Intra-medication can be promoted to avoid SSI in surgicalpatients, it can be defined as prophylactic administration of antibiotics within 1 hour and 24 hours after an operation.
- Regular and intensive drain care.
- Good post-operative care and efforts to boostpatient's immunity would help in decreasing further occurrence of SSI.
- Identify poor risk patients and ensure proper management to prevent from infections.
- Conduct periodic surveillance to keep a check on SSI.
- Improvements in operating room practices, instrument sterilization and administration of first dose of antimicrobial prophylaxis within 1 hour before skin incision are found to reduce the rate of SSIs.
- Shortening the pre-operative hospital stay can also help in reducing occurrence of SSIs.[22]

Central Line Associated Bloodstream Infection

Definition of Clabsi

The central line associated bloodstream infections (CLABSI) are the bloodstream infections confirmed by laboratory where central line was in situ for more than 48 hours from the time of event and the line was in place on the date of event or before.[8] Patients with any of the following signs and symptoms: fever (>38 C), chills/rigors or hypotension and at least one positive blood culture not related to contamination are recognized as CLABSI.

Central lines are of two types: (1) tunneled catheters are implanted surgically (by creating a subcutaneous track before entering vein) into the internal jugular, subclavian, or femoral vein for long-term (weeks to months) indications such as chemotherapy or hemodialysis and (2) non-tunneled catheters, the most commonly used central venous catheters, that are per cutaneously inserted and account for most CLABSIs. Within 7 to 10 days of central venous catheter placement, bacteria on the skin surface migrate along the external surface of the catheter from the skin exit site towards the intravascular space. The absence of tunnel (a

10 subcutaneous portion of the catheter contains a cuff that causes a fibrotic reaction around the catheter, creating a barrier to bacterial migration) places non-tunneled catheters at higher risk for CLABSIs. CLABSIs that occur beyond 10 days are usually caused by contamination of the hub (intraluminal) typically from a health care provider's contaminated hands but rarely from a host and often due to a breach of standard aseptic precautions to access hub. Less common mechanisms include hematogenous seeding of bacteria from another source or from a contaminated infusate.

Risk Factors of Clabsi

Several risk factors have been identified for CLABSIs in patients such asprolonged duration of catheterization, microbial colonization at the insertion site and of the catheter hub, and inadequate care/maintenance of the CVC after insertion.[20] Potential risk factors for CLABSI include underlying disease, method of insertion, site, duration, and purpose of catheterization. Host factors that increase the risk of CLABSI are chronic illnesses (hemodialysis, malignancy, gastrointestinal tract disorders, pulmonary hypertension), immune compromised states (bone marrow transplant, end-stage renal disease, diabetes mellitus), malnutrition, total parenteral nutrition (TPN), extremes of age, loss of skin integrity (burns), prolonged hospitalization before line insertion, catheter type, catheter location (femoral line has the highest, followed by internal jugular, then subclavian), conditions of insertion (emergent versus elective, use of full barrier precautions versus limited), catheter site care, and skill of the catheter inserter. Local risk factors like poor personal hygiene, occlusive transparent dressing, and moisture around the exit site. Other risk factors include contamination, inadequate water treatment, dialyzer re-use, older age, higher total intravenous iron dose, increased recombinant human erythropoietin dose, lower hemoglobin level.[17]

Diagnosis of Clabsi

CLABSI was identified based on various clinical manifestation in central venous catheterized patients. Clinical manifestations vary depending on the severity of illness. Fever and chills are the most common manifestations. Exit site examination to look for signs of inflammation of tunneled catheters with inspection and palpation of the subcutaneous track is important. Patients may report pain, swelling, or discharge from the exit site and redness surrounding the exit site or along the subcutaneous track when exit site or tunnel infections are present. For long-term catheters, difficulty in drawing blood or poor flow are considered risk factors and manifestations of CLABSI. In addition to the clinical exam, lab investigations are important for diagnosis and management. Blood culture is the most important step towards diagnosis in addition to complete blood count, serum electrolytes, and renal and liver function tests, which are necessary to assess for severity and/or co-morbidities. In suspected cases, paired blood cultures (one each from the central line and peripheral vein) must be drawn and labeled accordingly before sending to the lab. The following help in arriving at diagnosis.

Exit site infection: Signs of inflammation confined to an area (typically < 2 cm) surrounding the catheter exit site and the presence of exudates that proves to be culture positive.

Tunnel infection: Inflammation extending beyond 2 cm from exit site (along with the track or cephalad towards the vein entry site or extending beyond the cuff), typically associated with pain and tenderness along the subcutaneous track and culture-positive exudate at the exit site that may not be seen unless expressed by palpation.

Microbiology of Clabsi

The common microorganisms isolated with CLABSI were Staphylococci (S. aureus, CoNS), Enterobacteriaceae (K. pneumonie, E. coli) and Candida. Enterobacter is an important cause of severe CLABSI infections. These Enterobacters are of concern due to high rise of carbapenem, ESBL and multi drug resistance.

Outcomes of Clabsi

CLABSI have caused the highest morbidity and mortality in patients. It is a very common problem in the intensive care unit. Only through best practices, protocols, checklists, and establishing a culture of patient safety in healthcare institutions can one reduce CLABSI to zero. The major problem with central lines is that there are many types of lines, some which are directly inserted, others which are tunneled, and others which are inserted into the forearm and passed into the large veins of the heart. In many hospitals, central lines are inserted by many specialists including anesthesiologists, surgeons, emergency room physicians, radiologists, and critical care physicians. In addition, the use of a central line is not limited to any one nurse but all nurses. This heterogeneity has resulted in varied outcomes, but in almost every study, infections continue to be a common problem.

Prevention of Clabsi

Preventing CLABSIs in the ICU usually requires multiple strategies. Insertion strategies including education and training of those who insert catheters, use of chlorhexidine for skin antisepsis, and use of maximal sterile barrier precautions have a long record of preventing CLABSI. Use of novel technologies such as antibiotic or antiseptic impregnated catheters, suture less securement devices, and disinfection caps should be added to the armamentarium of tools to further reduce CLABSI rates.

CONCLUSION

Health care–associated infections (HAIs) are infections that patients acquire while they are receiving treatment for another condition in a health care setting. HAIs may

be caused by any infectious agent, including bacteria, fungi, and viruses, as well as other less common types of pathogens. They are among the leading causes of preventable deaths and are associated with a substantial increase in health care costs each year. HAIs lead to extended hospital stays, contribute to increased medical costs, and are a significant cause of morbidity and mortality.Numerous factors lead to HAIs, including the use and maintenance of medical devices, such as catheters and ventilators; complications after surgical procedures; transmission between patients and health care workers; contaminated air conditioning systems; a disproportionate nurse-to-patient ratio; and the physical layout of the health care facility. Proper education and training of health care workers increases compliance with and adoption of best practices to prevent HAIs. Despite the development of many hi-tech methods, hand washing with soap and water or alcohol rub is still the most important means of maintaining personal hygiene and preventing HCAIs. However, due to the rise of antibiotic-resistant bacteria and a reluctance of some HCWs to implement best practice infection control, HCAIs remain one of the biggest causes of death in most countries. Therefore, it is essential that strategic, policy, and education initiatives continue to focus on managing and controlling such (predominantly needless) infections.