Comparative study of the effect of Rantidine and Pantoprazole on Incidence of Ventilator-Associated Pneumonia

Thesis

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List of Abbreviations

ARDS : Acute respiratory distress syndrome

ATS : American Thoracic Society

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BAL : Bronchoalveolar lavage

CNS : Coagulase-negative staphylococcus

CPIS : Clinical pulmonary infection score

ESBL : Extended-spectrum beta-lactamase producing bacteria

ETT : Endotracheal tube

GERD : Gastroesophageal Reflux Disease

GI : Gastrointestinal

H2 : Histamine type 2

H2RAs : Histamine2 receptors antagonists

HAIs : Hospital acquired infections

HAP : Hospital-acquired pneumonia

ICU : Intensive care units

IDSA : Infectious Diseases Society of America

IVAC : Infection- related ventilator-associated complications

MDR : Multidrug resistant

MSSA : Methicillin-sensitive Staphylococcus aureus

OGD : Oesophagogastroduodenoscopy

PPIs : Proton pump inhibitors

PSB : Protected specimen brush

RRT : Renal replacement therapy

SRMD : Stress-related mucosal damage'

SUP : Stress ulcer prophylaxis

VAP : Ventilator-associated pneumonia

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Introduction

Nosocomial pneumonia is the leading infectious cause of death in critically ill patients **(Porzecanski et al., 2006).**

The crude mortality rate is estimated to be 20 to 50%, with an average increase in hospital stay of 7 to 9 days **(Tablan et al., 2003).**

Ventilator-associated pneumonia (VAP) is defined as a group of pneumonias that occur 48 h after the patient is ventilated if the patient did not have primary signs of the infection at the time of arriving ICU. VAP is one of the most prevalent nosocomial infections and pneumonia is the cause of 27% of infections in ICU **(Zandyeh et al., 2005).**

In healthy individuals multiple mechanisms work together to fight off the development of pneumonia; the presence of an ETT as well as the typical clinical circumstances of ICU patients (i.e. sedation, supine positioning colonization of the oropharynx with pathogenic microorganisms) interfere with these native defense

mechanisms and predispose intubated patients to the development of VAP **(Keyt et al., 2014).**

Symptoms and signs of pneumonia are fever, leukocytosis and purulent discharge. Laboratory testing is the best diagnostic method. Administration of antibiotics and cooperation with laboratory are important subjects after diagnosis. Furthermore, as the duration of hospitalization increases, the risk of VAP and incidence of more dangerous organisms in ventilated patients increase **(Zandyeh et al., 2005).**

Some studies have reported that the incidence of nosocomial pneumonia increases by 30% following pharmacological stress ulcer prophylaxis. Acid-suppressive medications such as proton pump inhibitors and histamine type 2 (H2) receptor antagonists are used to prevent stress ulcers. Theoretically, the inhibition of gastric acid secretion can be associated with increased gastric colonization as well as retrograde colonization of the pharynx leading to VAP with potential micro-aspiration **(Herzig et al., 2009).**

Pneumonia has also been associated with patient exposure to PPI therapy; Salivary gland secretions, gastric acid and well synchronized gastric motility are innate

mechanisms to restrict bacterial colonization in the gastric lumen. A gastric pH of less than 2 effectively limits bacterial colonization from ingested microbes. Since PPIs effectively increase the gastric pH above 4 for the majority of a 24-h period, this defense mechanism against the colonization of ingested bacteria is compromised. Consequently, due to the reduced gastric acidity, the bacterial load increases in the stomach **(Savarino et al., 2009).**

Single or repeated administration of PPIs may in fact lead to delayed gastric emptying, increased gastric contents, increased bacterial load and increased pressure on the lower esophageal sphincter that lead to retrograde movement of gastric contents up the esophagus. This reflux may then increase the risk of subsequent aspiration of both the gastric contents and the bacteria **(Savarino et al., 2009).**

In randomized Double-Blind Clinical Trial comparing the effect of ranitidine and pantoprazole and reporting the incidence of VAP to be three times higher in patients receiving pantoprazole **(Rahimi et al., 2013).**

Thus, our study designed to determine whether prophylaxis with the PPI pantoprazole increases the risk of nosocomial pneumonia compared with prophylaxis with the H2RA ranitidine in mechanically ventilated patients.

Aim of the Work

The aim of this work is to compare the effects of ranitidine and pantoprazole on incidence of Ventilator Associated Pneumonia (VAP).

Review of Literature

Hospital-acquired (or nosocomial) pneumonia (HAP) is pneumonia that occurs 48 hours or more after admission and did not appear to be incubating at the time of admission **(Kalil et al., 2016).**

Ventilator associated pneumonia (VAP), a subset of HAP, is defined as pneumonia that occurs 48 - 72 hours following endotracheal intubation. It is characterized by the presence of new or progressive pulmonary infiltrates, signs of systemic infection (as fever or altered white blood cell count), changes in sputum characteristics, and detection of a causative agent **(Kalil et al., 2016).**

Ventilator associated pneumonia is the second most common nosocomial infection in the intensive care unit and the most common in mechanically ventilated patients **(Hunter, 2012).**

Together, HAP and VAP are among the most common hospital acquired infections (HAIs), accounting for 22% of all hospital acquired infections (HAIs) in a multistate point-prevalence survey **(Magill et al., 2014).**