Ventilators - A Source of Infection in ICU

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Abstract:

Introduction - Ventilator associated pneumonia is the most serious and most commonly acquired infection in ICUs. It is believed that a biofilm that forms in the oropharynx or tracheal tube and pushed into the lungs is responsible for VAP. Since majority of VAP cases are caused by 4–5 multi drug resistant bacteria only, we thought that ventilator itself may be a source of infection. <u>Method</u> - Blood agar plates were exposed to oxygen coming from ventilator tubes for 10 minutes each. Plates were also exposed to oxygen coming directly and to air in ICU also and the colonies that developed were counted and identified. <u>Results</u> - All the plates exposed to oxygen coming through ventilators showed growth of bacteria ranging from <10 to >300 colonies per plate. Majority of the ventilators showed growth of drug resistant. Acinetobacter, Pseudomonas, Klebsiella and staphylococci. The number of colonies developed was proportional to be the duration of use of ventilators. <u>Conclusions</u> - It appears that ventilator itself may be a source of infection in ICUs.

Keywords: Ventilator Associated pneumonia, Mechanical Ventilation, Bio film, Drug Resistant bacteria, ETT cuff

Introduction

Ventilator associated pneumonia (VAP) is defined as infection of the lung parenchyma occurring 48 hours after initiation of mechanical ventilation by endotracheal tube or tracheostomy.^[1] It is the most frequent ICU acquired infection and VAP occurrence generally has been reported to vary between 8 and 28%.^[2-5] The risk of developing pneumonia increases with the duration of Mechanical Ventilation^[6] and Fagon et al,1989^[2] have reported this incremental risk to be 1% per day.

In a normal healthy human being multiple protective mechanisms protect the respiratory tract from infection - the anatomical barriers like glottis and larynx, cough reflex, surface lining and mucociliary action, local IgA and phagocytic cells comprising alveolar macrophages and neutrophils. When these defenses are weak or overcome by a high inoculum of organisms of unusual virulence, pneumonia results.

The main factor in the development of Ventilator associated pneumonia is believed to be biofilm formation within the tracheal tube. The oropharynx becomes rapidly colonized with aerobic Gram negative bacteria and the contaminated secretions pool above the TT cuff. A bacterial biofilm forms on the inner and outer surface of the tracheal tube and is pushed into the lung parenchyma by ventilator cycling.^[7] Sometimes organisms from the stomach or sinuses and dental plaque can also contaminate the subglottic secretions and a small number or cases may result from direct blood streams infections.^[8] Contaminated respiratory care

equipments may sometime be the source of infection.^[9-11] Sometimes contaminated secretions condensate in ventilator tube and drain into the patient's airways.^[12]

Since majority of the Ventilator associated pneumonia patients are infected with a few drug resistant bacteria only, we thought that the source of infection may be ventilator itself rather than oropharynx of the patients. The study was, therefore, undertaken to examine the quality of the oxygen delivered by ventilators.

Material and Methods

The study was conducted during January 2011 to December, 2011 on 18 ventilators available in ICU of a tertiary care hospital. The ventilators were grouped as follows

Less than 1 month in use	
1 month to 1 year of use	- 4
1 year to 2 years of use	- 4
More than 2 years of use	- 8

The blood agar plates 100 mm were exposed to oxygen coming from ventilator tubes for 10 minutes each. They were then incubated at 37^oC overnight. The number of colonies developed on each plate was counted and the different types of colonies were identified following standard procedures.^[13] Blood agar plates were also exposed for 10 min to atmospheric air in the ICU and to oxygen coming directly and not through ventilators. The above procedure was repeated again after two weeks.

Results

The no. of colonies that developed ranged from <10 to more than 300 (uncountable) (Table 1 & Fig 1 to 4) Most of the plates showed mixed growth comprising of Acinetobacter, Pseudomonas, Klebsiella, Staphylococcus and Bacillus in different proportions with Acinetobacter being the dominant bacterial species.

It was observed that ventilators that were recently put to use showed minimum growth that too of aerobic spore bearer (Fig 1).



FIG 1: Growth from Ventilators less than one month in use



FIG 2: Growth from Ventilators 1 month to 1 year in use



FIG 3: Growth from Ventilators 1 year to 2 years in use

On the other hand ventilator that were in use for more that 2 years yielded heavy grow of >300 colonies comprising mainly Acinetobacter and Pseudomonas (Fig 2).



FIG 4: Growth from Ventilators more than 2 years in use

It was also seen that the no. of colonies developed increased with the duration of use of ventilators.

The plates exposed to oxygen coming directly (not through ventilator) and to ICU air hardly showed any growth (Fig 5 & 6)



FIG 5: Growth from oxygen coming directly not through Ventilators



FIG 6: Growth from ICU air

Table 1: No of colonies vs duration of use of ventilators

Duration of use	No. of	No. of colonies
of ventilators	ventilators	grown
< 1 month	2	5-6
1 month to 1 year	4	40-100
1 year to 2 years	4	100-200
> 2 years	8	>300

Discussion

VAP is a specific hospital acquired pneumonia that develops 48 hours after initiation of mechanical ventilation.^[14,15] and is one of the most important preventable cause of morbidity and mortality in ICU.^[16] It is believed that oropharynx is rapidly colonized by aerobic Gram negative bacteria after hospital admission. A biofilm forms within the tracheal tube and is pushed into the distal airways by ventilator cycling.^[7,17] In the present study we found that all the ventilators yielded same types of drug resistance bacterial pathogens in different proportions. It has already been proposed that the contaminated equipment may sometimes be the source of infection.^[9-11] Our findings strengthen our hypothesis that the source of infection may lie in the ventilator itself. It is possible that the oropharynx is colonized with bacteria coming from ventilators. This fact is further substantiated by the fact that in majority of the patients Ventilator associated pneumonia is caused by same 4 or 5 multidrug resistant bacteria only. This predominance of drug resistant bacteria has also been reported by others.^{[18-}

References

- [1] Tablan, O.C., Anderson L.J., Besser, R, et al. Guidelines for preventing health acare associated pneumonia : Recommendations of CDC and the Healthcare Infection Control Practice Advisory Committee 2003. Morb Mort Weekly R Ventilator associated pneumonia2004; 53 (RR-3): 1-36.
- [2] Fagon JY. Chastre J, Domart Y, Trouillet JL, Pierre J, Darne C,m Gibert C. Nosocomial pneumonia in patients receiving continuous mechanical ventilation. Propective analysis of 52 episodes with use of protected specimen brush and quantitative culture techniques. Am Rev Resp Dioas 1989, 139 877-884.
- [3] Torres A. Aznar R, Gatell JM, Jimenez P, Gonzalez J, Ferrer A, Celis R., Rodrignez-Roisin R. Incidence, rid and prognosis factors of nosocomial pneumonia in mechanically ventilated patients. Am Rev Respir Dis 1990, 142:523-528.
- [4] Cook DJ, Walter Sd, Cook RJ, Griffith LE, Gyuatt GH, Lease D, Jaeschke RZ, Brun-Buisson C. Incidence of and risk factors for ventilatorassociated pneumonia in critically ill patient. Ann Intrn Med 1998, 129:433-440.
- [5] Joshi N, Localio AR, Hamory BH. A predicative risk index for nosocomial pneumona in the intensive care unit. Am J Med 1992, 93: 135-142.
- [6] Hunter JD. Ventilator associated pneumonia. BMJ 2012; 344: 40-44
- [7] Inglis T, Millar J, Jones J. Jones J, Robinson D. Tracheal tube biofilm as a source of bacterial

colonization of the lung. J Clin Microbial, 1989; 27: 2014-2018.

- [8] Estes RJ, Meduri GU. The pathogenesis of ventilator-associated pneumonia: I. Mechanisms of bacterial transcolonization and airway inoculation. Intensive care Med 199521365-383.
- [9] Rogers D. The changing patterns of life threatening microbial disease. N Engl J Med 1959; 261:677-683.
- [10] Kneeland Y, Price K. Antibiotics and terminal pneumonia a postmortem microbiologic study. Am J Med. 1960; 29:967-979.
- [11] Craven DE, Lichtenberg DA, Goularte TA. Et al. Contaminated medication nebulizers in mechanical ventilator circuits. Cource of bacterial aerosols. Am J Med 198477834 – 838.
- [12] Craven DE Steger KA. Hospital acquired pneumonia perspectives for the healthcare epidemiologist. Infect Control Hosp Epidemiol 1997; 18: 783-795.
- [13] Collie JG, Fraser AG, Marmion BP, Simmons A.(ed). Mackie & Mc Cartney's Practical Medical Microbiology. 14th Edition. Elsevier, 2006
- [14] ATS Board of Directors and IDSA Guideline Committee. Guidelines for the management of adults with hospital acquired, ventilator associated and health care associated pneumonia. Am J Respir Crit Care Med. 2005; 171:416.
- [15] Mayhall, GC. Ventilator associated pneumonia or not ? Contemporary diagnosis. Emer Infect Dis, 2001; 7(2): 200-204.
- [16] Delaney A, Gray H, Laupland BL, Zuege JD. Kinetic bed therapy to prevent nosocomial pneumonia in mechanically ventilated patients: A systematic review and meta analysuis. Cricical Care 2006; 10: R70.
- [17] Gunasekera P, Gratrix A. Ventilator-assocated pneumonia. BJA Education.2016; 16(6): 198-202.
- [18] Medell M, Medell M, Martinez A, Valdes R. Characterirization and sensitivity to antibiotic of bacteria isolated from lower respiratory tract of ventilated patients hospitalized in intensive care units. Braz J Infect Dis.2012;16:45-51
- [19] Souza-Oliveira AC, Cunha TM, da Sila Pasos LB, Lopes GC, Gomes FA, de Brito Roder. Ventilator associated pneumonia: the influence of bacterial resistance, prescription errors and de-esalation of antimicrobial therapy on mortality rates. Braz J Infect Dis.2016;20:437-444

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