ORIGINAL ARTICLE

An audit on ventilator associated pneumonia in the Intensive Care Unit at Teaching Hospital Karapitiya, Galle, Sri Lanka

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ABSTRACT

Background: Critical care is one of the most expensive services provided by a hospital. The aim of this audit was to ascer tain the incidence of v entilator associated pneumonia in the intensi ve care unit.

Type of study: A descriptive study

Place of study: Teaching Hospital Karapitiya, Galle (Sri Lanka)

Duration of study: 1st June 2010 to 30th August 2010

Methodology: All patients, who were admitted to ICU and who stayed there for more than 48hrs during a period of three months were studied. Infections were identified on clinical parameters such as fever and on laboratory investigations such as full count, CRP and cultures.

Results: Out of 82 patients, 48(58.5%) were subsequently discharged to the ward and 30(36.6%) succumbed to their illness. 68(82.9%) were ventilated and 26 of them had an underlying pathology related to an infection. A total of 20(29.4%) patients of this ventilated group subsequently developed a lower respiratory tract infection. The main nosocomial infection w as ventilator associated pneumonia and had an incidence of 21.9%. The most prevalent organisms were mixed gram negative bacilli and Acinetobacter spp.

Conclusion: Nosocomial infections are a cause of increased mor tality and morbidity in the intensi ve care unit. Awareness of the risk factors tog ether with simple prev entive measures and sur veillance will help to reduce its occurrence.

Key words: Nosocomial infection; ventilator associated pneumonia

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INTRODUCTION

Ventilator associated pneumonia (V AP) is a for m of nosocomial infection that occurs in patients recei ving mechanical ventilation for longer than 48hrs[1]. The incidence of VAP is around 22.8%[2], and patients receiving ventilatory support account for 86% of the cases of nosocomial pneumonia[3]. The mor tality rate attributable to V AP is around 27%[4], and the costs of treatment of these infections contribute to a significant por tion of the expenditure incurred in maintaining an intensi ve care unit. Teaching Hospital Karapitiya, Galle, Sri Lanka is one of the main tertiary care centers in the country. All the general medical and surgical patients requiring intensive care are admitted to the main intensi ve care unit where the audit w as conducted. The main objective of this study was to ascertain the incidence of ventilator associated pneumonia in the intensive care unit.

METHODOLOGY

Permission to conduct this prospective audit was obtained from the director Teaching Hospital Karapitiya. The audit was conducted for three months from 1st June 2010 to 30th August 2010. We studied all the patients admitted to the main ICU and who stayed there for more than 48hrs, regardless of whether they were ventilated or not during this period. Reports of cultures from tracheal secretions were obtained from the department of microbiology. However, we not able to perform bronchoscopically collected samples for quantitative cultures due to lack of facilities.

RESULTS

Total number of patients admitted to main ICU for more than 48hrs was 82; out of which 48(58.5%) were subsequently discharged to the ward and 30(36.6%) succumbed to their illness. Of the 82 patients studied 68(82.9%) were ventilated and only 26 of them had an underlying pathology related to an infection (Table 2). A total of 20(29.4%) patients of this ventilated group subsequently developed a lower respiratory tract infection (Table 2). The majority of them [14(70%)] were in categories that had no infection in any part of the body at the time of admission to the ICU or at the time of commencing ventilation (Table 2). We used the following criteria to diagnose lower respiratory tract infection (LRTI).

1. New shadow developing in the chest X- ray
2. Temperature > 99º F
3. Course crepitations on chest auscultation
4. White cell count > 11,000

We used the following criteria to diagnose VAP.

1. New shadow developing in the chest X-ray
2. Temperature < 96.8 or > 990 F
3. Ventilated for more than 48 hrs
4. White cell count > 11,000 or < 4,000
5. Cultures positive from endotracheal secretions

All 20 patients, who developed an LRTI, had their tracheal secretions cultured and 2 of them failed to grow any organisms in the endotracheal secretions. However, if we had the facilities to perform bronchoscopic sampling for quantitative cultures, all of them may have been positive for bacterial invasion. The organisms cultured are shown in Table1.

Table 1: The organisms cultured

Organism	Frequency	%
Acinetobacter spp	5	25
Pseudomonas spp	3	66.6
Coliform spp	2	10
Mixed gram negative bacilli	6	33.3
Staphylococcus aureus	2	10
No growth	2	10
Total	20	100

VAP was diagnosed in 18 (21.9%) patients on these criteria.

Table 2: Patient categories developing LRTI following ventilation

Patient category	No.	Ventilated	LRTI
Head Injury	14	14	4
Trauma other than head injury	6	6	1
Poisoning	5	5	3
Post-op elective Ventilation	9	9	4
Medial reasons without infection	11	8	2
Infections from Medical wards	7	4	0
Infections from other wards	30	22	6
Total	82	68	20

DISCUSSION

The patients needing intensive care usually have low host defense immunity. Immunosupression primarily due to the release of interleukins and other anti inflammatory agents creates a state sometimes termed as immunoparalysis[5]. Endotracheal intubation compromises local defense mechanisms such as coughing, sneezing and mucocilliary clearance. Upper airway and oral secretions can pool above the cuff of an endotracheal tube and line the tube, forming a biofilm, starting as early as 12 hrs after intubation. The biofilm contains large amounts of bacteria that can be disseminated into the lungs by the ventilator induced inspiration[6-8]. The presence of the endotracheal tube also provides a direct route for colonized bacteria to enter the lower respiratory tract. Exogenous colonization arises from cross transmission from the hands of health care workers or visitors[9].

Cook and colleagues noted that the administration of paralytic agents was an independent predictor of nosocomial pneumonia in their study of 1,014 mechanically ventilated patients[10]. Sedative drugs and stress ulcer prophylaxis have all been implicated as risk factors.

In a report from the National Nosocomial Infection Surveillance (NNIS) system involving data from 498,998 patients, 83% of episodes of nosocomial pneumonia was associated with mechanical ventilation[11]. Langer and colleagues showed an increased incidence from 5% in patients who received 1 day of mechanical ventilation to 69% in those who received 30 days of venilation[12]. One of the largest databases related to nosocomial infection in intensive care is the EPIC study [13]. In this 1-day point prevalence study, information was obtained on all patients who occupied a bed in an intensive care unit over 24hrs. 10,038 patients were recruited from 1,407 western European intensive care units. 2,064 of them (21%) had an intensive care unit acquired infection. The incidence of nosocomial pneumonia in the EPIC study was 47% and 31% in the NNIS study.

For many years VAP has been diagnosed by the clinical criteria published by Johanson et el in 1972, which include the appearance of a new pulmonary infiltrate, fever, leukocytosis and purulent tracheobroncheal secretions[14]. However these criteria are non specific . VAP is most accurately diagnosed by quantitative cultures and microscopic examination of lower respiratory tract secretions which are best obtained by bronchoscopically directed techniques, such as the protected specimen brush and bronchoalveolar lavage. These techniques have acceptable repeatability and interpretation of results is unaffected by antibiotics administered concurrently for infections in extra pulmonary sites as long as the antimicrobial therapy has not been changed for <72 hrs before bronchoscopy[15,16]. The accuracy of quantitative cultures and microscopic examination of lower respiratory tract secretions for the diagnosis of VAP was validated by Chastre et al[17,18]. However, other published studies have concluded that bronchoscopically directed techniques were not more accurate for diagnosis of VAP than clinical X-ray criteria combined with cultures of tracheal aspirates[19-22].

Prevention has a key part to play in limiting VAP. Several studies have reported high rates of contamination with potentially pathogenic organisms by the hands of healthcare workers[23,24]. Hand disinfection with the use of alcohol based antiseptic hand rub solutions have been shown to be effective in reducing hand contamination[25]. Chlorhexidine oral rinse twice daily has been used by some workers to reduce oral colonization[26]. However, no evidence based protocols for oral care have been tested to decrease the incidence of VAP in patients receiving mechanical ventilation. According to the studies done so far , stress ulcer prophylaxis does not play a significant role in the development of VAP. Saline lavage of endotracheal tubes before suctioning can dislodge bacteria from the endotracheal tubes in to the lower airways increasing the risk of VAP[27]. Positioning the patient in a semi recumbent position prevents reflux and aspiration. Maintaining an adequate cuff pressure decreases the likelihood of secretions leaking around the cuff . Use of tubes with ports for continuous subglottic suctioning can decrease the incidence of VAP by 50%[28,29] and therefore the oropharynx should be thoroughly suctioned. Daily interruption of continuous sedation and paralysis can shorten the duration of mechanical ventilation. Infection surveillance can reduce nosocomial infection rates when incorporated with infection prevention prgrammes[30].

CONCLUSION

VAP, although often preventable, is a cause of increased mortality and morbidity; it is also responsible for excessive resource expenditure in the intensive care unit. Awareness of the risk factors and attention to simple preventive measures such as hand hygiene can reduce the incidence of these infections . Surveillance together with a multidisciplinary approach of prevention is the other important aspect of its limitation.

REFERENCES

1. Kollef MH. The prevention of ventilator associated pneumonia. N Engl J Med. 1999;340(8):627-34.

2. Safdar N, Dezfulian C, Collard HR, Saint S. Clinical and economic consequences of ventilator associated pneumonia: a systemic review. Crit Care Med. 2005;33(10):2184-93.

3. Richards MJ, Edwards JR, Culver DH, Gaynes RP. Nosocomial infections in medical intensive care units in the United States . Crit Care Med. 1999;27:887-92.

4. Craven DE. Epidemiology of ventilator associated pneumonia. Chest. 2000;117(4):1865-75.

5. Docke WD, Randow F, Syrbe HP et al. Monocyte deactivation in septic patients:restoration by IFN treatment. Nat Med. 1997;3:678-81.

6. Kunis KA, Puntillo KA. Ventilator associated pneumonia in the ICU:its pathophysiology, risk factors and prevention. Am J Nurs. 2003;133(8):64A-64G.

7. Olson ME, Har mon BG, K ollef MH. Silv er coated endotracheal tubes associated with reduced bacterial burden in the lungs of mec hanically v entilated dogs. Chest. 2002;121:863-70.

8. Morehead RS, Pinto SJ. Ventilator associated pneumonia. Arch Intern Med. 2002;160(13):1926-30.

9. Wang JT, Chang SC, Ko WJ, et al. A hospital acquired outbreak of methicillin resistant Staph ylococcus aureus infection initiated b y a surg eon car rier. J Hosp Infect. 2001;47:104- 09.

10. Cook DJ, Walter SD, Cook RJ, et al. Incidence of and risk factors for ventilator associated pneumonia in critically ill patients. Ann Inter n Med. 1998;129:433-40.

11. Richards MJ, Edw ards JR, Culv er DH, Ga ynes RP. Nosocomial infection in combined medical surgical intensive care units in the United States . Infect Control Hosp Epidermiol 2000;21:510-15.

12. Langer M, Mosconi P, Cigada M, Mandelli M. Long ter m respiratory support and risk of pneumonia in critically ill patients. Am R ev R espir Dis 1989;140:302-05.

13. Vincent JL, Bihari D, Suter PM, et al. T he prevalence of nosocomial infection in intensive care units in Europe: the results of the EPIC study . J AMA 1995;274:639-44.

14. Johanson W G, Pierce AK, Sanford JP , T homas GD. Nosocomial respiratory infections with gram negative bacilli. The significance of colonization of the respirator y tract. Ann Intern Med 1972;77:701-6.

15. Timsit JF, Misset B, Renaud B, Goldstein FW, Carlet J. Effect of previous antimicrobial therapy on the accuracy of the main procedures used to diagnose nosocomial pneumonia in patients who are using ventilation. Chest 1995:108:1036-40.

16. Souweine B, Verber B, Bedos JP, Gachot B, et al. Diagnostic accuracy of protected specimen brush and bronchoalveolar lavage in nosocomial pneumonia: impact of previous antimicrobial treatment. Crit Care Med 1998;26:236-44.

17. Chastre J, Fagon JY, Bor net-Lecso M, Calv at S, et al. Evaluation of bronchoscopic techniques for the diagnosis of nosocomial pneumonia. Am J R esp Crit Care Med 1995;152:231-40.

18. Chastre J, Viau F, Brun P, Pierre J, et al. Prospective evaluation of the protected specimen br ush for the diagnosis of pulmonary infections in ventilated patients. Am Rev Respi Dis 1984;130:924-9.

19. Torres A, El-Ebiary M, Padro L, Gonzalez J, et al. Validation of different tec hniques for the diagnosis of v entilator associated pneumonia. Comparison with immediate postmortem pulmonary biopsy. Am J Respi Crit Care Med 1994;149:324-31.

20. Marquette CH, Copin MC , Wallet F, Neviere R, et al. Diagnostic tests for pneumonia in v entilated patients: Prospective evaluation of diagnostic accuracy using histology as a diagnostic g old standard. Am J R espi Crit Care Med 1995;151:1878-88.

21. Kirtland SH, Corely DE, Winterbauer RH, Springmeyer SC, et al. The diagnosis of ventilator associated pneumonia. A comparison of histological, microbiological and clinical criteria. Chest 1997;112:445-7.

22. Fabregas N, Ewig S, Torres A, El-Ebiary M. et al. Clinical diagnosis of ventilator associated pneumonia revisited: comparative validation using immediate post mortem lung biopsies. Thorax 1999;54:867-73.

23. Rangel-Frausto MS, Wiblin T, Blumberg HM, et al. National epidemiology of mycoses sur vey (NEMIS): v ariation in rates of blood stream infection due to Candida species in seven surgical intensive care units and six neonatal intensive care units. Clin Infect Dis 1999;29:253-58.

24. Pittet D, Dharan S, Touveneau S, Sauvan V, Perneger TV. Bacterial contamination of the hands of hospital staff during routine patient care. Arch Intern Med 1999;159:821-26.

25. Pittet D, Hugonnet S, Harbarth S, et al. Effectiveness of a hospital wide programme to improve compliance with hand hygiene. Lancet 2000;356:1307-12.

26. DeRiso AJ, Ladowski JS, Dillon T A, Justice JW, Peerson AC. Chlorhexidine gluconate 0.12% oral rinse reduces the incidence of total nosocomial respirator y infection and systemic antibiotic use in patients undergoing heart surgery. Chest 1996;109(6):1556-61.

27. Moore T. Suctioning tec hniques for the remo val of respiratory secretions. Nurs Stand 2003;18(9):47-55.

28. Craven DE. Preventing ventilator associated pneumonia in adults: sowing seeds of change. Chest 2006;130(1):251-60.

29. Shorr AF, O'Malley PG. Continuous subglottic suctioning for the prev ention of ventilator associated pneumonia: potential economic implications. Chest 2001;119(1):228-35.

30. Haley RW, Culver DH, W hite JW, et al. T he efficacy of infection surveillance and control programmes in preventing nosocomial infections in US hospitals. Am J Epider miol 1985;121:182-205.