Commentary

Does ventilator-associated tracheobronchitis need antibiotic treatment?

Antonio Torres[1] and Mauricio Valencia[2]

[1]Chairman, Department of Pulmonology and Critical Care, Hospital Clinic, IDIBAPS, Facultat de Medicina, Universitat de Barcelona, Red Gira and Red Respira, Barcelona, Spain
[2]Critical Care Researcher, Department of Pulmonology and Critical Care, Hospital Clinic, IDIBAPS, Facultat de Medicina, Universitat de Barcelona, Red Gira and Red Respira, Barcelona, Spain

Corresponding author: Antonio Torres, atorres@clinic.ub.es

Published online: 3 May 2005
This article is online at http://ccforum.com/content/9/3/255

Critical Care 2005, **9:**255-256 (DOI 10.1186/cc3535)

See related research by Nseir *et al.* in this issue [http://ccforum.com/content/9/3/R238]

Abstract

It is difficult to define ventilator-associated tracheobronchitis (VAT). The most accepted definition includes fever (temperature >38°C), new or increased sputum production, a microbiologically positive respiratory sample with counts above the accepted thresholds and absence of pulmonary infiltrates on chest X-ray. Although we have no doubt that this pathologic process exists, the main controversy lies on whether this entity has any impact on the outcome and, thus, a specific therapeutic approach is suitable. We will discuss the strengths and drawbacks of the article on this topic published in this issue by Nseir *et al*.

Ventilator-associated tracheobronchitis (VAT) is a difficult entity to define. Indeed, the recently published American Thoracic Society/Infectious Diseases Society of America guidelines [1] do not address this issue. VAT is defined as the presence of fever (temperature >38°C), new or increased sputum production, a microbiologically positive respiratory sample (with counts above accepted thresholds), and absence of pulmonary infiltrates on chest radiography. The apparent crude incidence of VAT ranges from 3% to 10% [2], but it is very difficult to determine the exact incidence and importance of VAT for several reasons. First, the definition of VAT has not been validated. There are no studies with acceptable 'gold standard' techniques evaluating the accuracy of the criteria mentioned above or other such criteria. Second, the term 'new or increased sputum production' is rather imprecise and depends on subjective impression. Finally, to confirm the absence of infiltrates on a chest radiograph, a computed tomography (CT) scan is required. In fact, it is not possible with portable chest X-ray machines to view some small infiltrates or opacities that may be detected by CT scans.

However, we have no doubt that this entity exists from the clinical point of view. This belief results from the findings of post-mortem studies [3], in which it is not infrequent to find high bacterial counts in lung samples without histological pneumonia. In addition, some years ago Rouby and coworkers [4] described the existence of bronchiolitis without histological pneumonia in lung samples taken from mechanically ventilated patients shortly after death. Establishing comparisons with severe community-acquired infections you can also observe patients with pneumonia and patients with infectious bronchitis without pulmonary infiltrates on chest radiography as is the case of chronic obstructive pulmonary disease (COPD) or bronchiectasis.

The reason why some patients develop VAT and not ventilator-associated pneumonia is unknown but is probably due to a counterbalance in the local inflammatory response. This hypothesis requires confirmation in prospective studies.

In the study presented in this issue of *Critical Care*, Nseir and colleagues [5] report the results of a retrospective case–control study conducted in patients with VAT. They were able to include and match 55 patients. The criteria defining VAT were those mentioned above. Those investigators excluded patients with chronic respiratory failure, those with tracheostomy, trauma patients and immunosuppressed patients. Importantly, the matching criteria were very strict, employing six different matching variables. However, cases more frequently received prior antibiotic treatment than did controls (72% versus 27%). Although the mortality rates were similar in the two populations and the rate in patients with VAT was not related to the adequacy of antibiotic

COPD = chronic obstructive pulmonary disease; CT = computed tomography; PPM = potentially pathogenic micro-organism; VAT = ventilator-associated tracheobronchitis.

treatment, the duration of mechanical ventilation and the length of intensive care unit stay were almost double in cases compared with controls. In a logistic regression analysis, VAT was found to be independently associated with increased duration of mechanical ventilation (odds ratio 3.5). Importantly, all micro-organisms isolated in VAT patients were potentially pathogenic micro-organisms (PPMs), and *Pseudomonas aeruginosa* accounted for one-third of these isolates.

The main reservations we have regarding this study and its results are inherent to the definition of VAT and whether it really exists, and are addressed above. However, in the critical care arena we do see patients like those described by Nseir and colleagues [5], although not very often.

The main issue raised by this study is the potential influence of VAT on length of stay and duration of mechanically ventilation. In relation to this, the first question that clinicians would ask is whether VAT must be treated with antibiotics and for how long. Although that study cannot specifically address this issue, there is indirect evidence that support administration of antibiotics to patients with VAT. In a study conducted by Nouira and colleagues [6], COPD patients requiring mechanical ventilation without pneumonia were randomly assigned to receive ofloxacin or placebo. Mortality and other outcome measures were much worse in patients receiving placebo. Most of these patients probably were admitted with infectious bronchitis. In contrast, in an observational study conducted some years ago, Fagon and coworkers [7] could not find any difference in outcome in patients suffering an exacerbation of COPD that required mechanical ventilation whether they were treated with antibiotics or not. In our opinion, PPMs in high counts in patients with symptoms of infection must be treated with antibiotics. We demonstrated some years ago [8] that in stable COPD patients the presence of PPMs at low concentrations (≥100 colony-forming units/ml) is associated with a significant local inflammatory response. On the other hand, it is not infrequent in clinical practice to observe weaning failure in patients who have purulent secretions and an endotracheal aspirate with growth of PPMs. Usually, when these patients receive antibiotics for few days weaning failure is reversed and they can be successfully extubated. What we do not know is for how long we must treat these patients. Probably, short courses of antibiotics should be sufficient.

In summary, VAT is a nosocomial infection with no validated definition. Consequently, we do not know what proportion of these patients have real ventilator-associated pneumonia. Studies involving CT scans are needed to establish this. It seems that VAT caused by PPMs at high concentrations is associated with an increased period of mechanical ventilation and length of stay. Our recommendation is to treat these patients with antibiotics, particularly when they present with persistent weaning failure.

Competing interests

The author(s) declare that they have no competing interests.

References

1. American Thoracic Society; Infectious Diseases Society of America: **Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia.** *Am J Respir Crit Care Med* 2005, **171:**388-416.
2. Nseir S, Di Pompeo C, Pronnier P, Beague S, Onimus T, Saulnier F, Grandbastien B, Mathieu D, Delvallez-Roussel M, Durocher A: **Nosocomial tracheobronchitis in mechanically ventilated patients: incidence, aetiology and outcome.** *Eur Respir J* 2002, **20:**1483-1489.
3. Fabregas N, Torres A, El-Ebiary M, Ramirez J, Hernandez C, Gonzalez J, de la Bellacasa JP, de Anta J, Rodriguez-Roisin R: **Histopathologic and microbiologic aspects of ventilator-associated pneumonia.** *Anesthesiology* 1996, **84:**760-771.
4. Rouby JJ, Martin De Lassale E, Poete P, Nicolas MH, Bodin L, Jarlier V, Le Charpentier Y, Grosset J, Viars P: **Nosocomial bronchopneumonia in the critically ill. Histologic and bacteriologic aspects.** *Am Rev Respir Dis* 1992, **146:**1059-1066.
5. Nseir S, Di Pompeo C, Soubrier S, Lenci H, Delour P, Onimus T, Saulnier F, Mathieu D, Durocher A: **Effect of Ventilator-associated tracheobronchitis on outcome in patients without chronic respiratory failure: a case–control study.** *Crit Care* 2005, **9:**R238-R245.
6. Nouira S, Marghli S, Belghith M, Besbes L, Elatrous S, Abroug F: **Once daily oral ofloxacin in chronic obstructive pulmonary disease exacerbation requiring mechanical ventilation: a randomised placebo-controlled trial.** *Lancet* 2001, **358:**2020-2025.
7. Fagon JY, Chastre J, Trouillet JL, Domart Y, Dombret MC, Bornet M, Gibert C: **Characterization of distal bronchial microflora during acute exacerbation of chronic bronchitis. Use of the protected specimen brush technique in 54 mechanically ventilated patients.** *Am Rev Respir Dis* 1990, **142:**1004-1008.
8. Soler N, Ewig S, Torres A, Filella X, Gonzalez J, Zaubet A: **Airway inflammation and bronchial microbial patterns in patients with stable chronic obstructive pulmonary disease.** *Eur Respir J* 1999, **14:**1015-1022.