Reviewer's report:

**Title:** Acinetobacter baumannii nosocomial pneumonia: Is the outcome more favorable in non-ventilated than ventilated patients?

**Version:** 2  **Date:** 17 December 2012

**Reviewer:** Nele Brusselaers

I read with interest the article of Yang et al about Acinetobacter infections in both HAP and VAP. The authors concluded that the survival in HAP patients infected with Acinetobacter was worse compared with VAP patients. I actually think they authors didn’t evaluate their results as efficient as possible. Now it just seems that patients not on a ventilator are treated suboptimal, since they have less multidrug resistant strains, are less severely ill, and have less frequently bilateral pneumonia. Or is it due to other confounding factors?

**Major**

- The study is based on a rather small sample size (N=144, 48 HAP, 96 VAP) over a 10 year study period. This is less than half of the patients with A. baumannii bacteraemia. It is not clear however how many of these patients are treated in intensive care units and for which underlying conditions, which is quite essential to estimate the severity of disease. I would expect that all VAP patients were treated in an ICU, since they receive mechanical ventilation. Which other units were involved? The whole hospital except for paediatrics?

- Definitions of HAP and VAP should be reported in more detail. E.g. period on mechanical ventilation before VAP onset, which sampling techniques were used? Were all pneumonia cases confirmed with BAL? It is not clear which proportion of the pneumonia patients was diagnosed with BAL. There is quite a big difference between BAL (golden standard) and sputum cultures (questionable for analysing colonization and infection!). Or did you mean endotracheal aspirates instead of “sputum”. Please discuss in more detail when the different types of cultures (respiratory and blood) were used (before, at onset and after clinical symptoms of pneumonia).

- So HAP patients had the same comorbidity scores as the VAP patients on admission. But is this admission to the hospital or to the ICU? Or when? I suppose all VAP patients were treated in the ICU since they were on a ventilator (thus more severely ill) compared to the HAP. Shouldn’t the HAP patients have been treated in the ICU even before HAP onset? This “suboptimal treatment” could explain the differences in survival...

- It’s quite logic that there are more multidrug resistant strains in the ICU patients (based on current literature). This means that treatment of non-multidrug resistant strains should be more effective – yet antimicrobial therapy was
inappropriate in the majority of both HAP and VAP. It is not clear to me how initial antimicrobial therapy was initiated.

- The authors say that the crude mortality rates were not significantly different, but I suppose they only looked at the p-value since a 30% higher mortality in HAP does seem clinically relevant.

- By looking at table 1 it is quite remarkable that 50% of the HAP patients presented with a malignancy versus only 30% in the VAP patients. The authors appear to have identified the link with malignancy through logistic regression, but didn’t notice this in table 1?

- It considers a rather long time period to evaluate nosocomial infections: a lot can change in the main flora causing the infections, but also in infection control and antibiotic therapy. Did the authors take this into account when evaluating the results?

- Are routine respiratory surveillance cultures performed in the ICU? Please discuss based on current evidence. Or how did you decide on the antibiotic treatment regimen?

- How many patients were missed because of missing data/incomplete medical records?

- How many patients did have different Acinetobacter species in both blood and respiratory tract cultures (e.g. antimicrobial susceptibility)?

- Why did you only analyse chronic kidney injury and not AKI? The definition mentioned seems applicable to both.

- How many HAP patients did receive mechanical ventilation because of the pneumonia?

- Why did you not analyse polymicrobial episodes (separately)? If less-pathogenic strains were found, it could be assumed that the pneumonia is mainly caused by the Acinetobacter spp. And you excluded more than half of the patients from the study, which makes the internal validity and generalisability of the study results questionable.

- Please report data of logistic regression in more detail

- Please discuss strengths and limitations of the study in the discussion.

- Table 1 is presented poorly and contains unnecessary information.

Minor:
- In methods you only mention 14-d mortality

**Level of interest:** An article of limited interest
Quality of written English: Acceptable

Statistical review: Yes, and I have assessed the statistics in my report.

Declaration of competing interests:

I declare that I have no competing interests