Reviewer's report

Title: Pseudomonas aeruginosa in a neonatal intensive care unit: molecular epidemiology and infection control measures

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The article by Crivaro at al., “Pseudomonas aeruginosa in a neonatal intensive care unit: molecular epidemiology and infection control measures” is addressing molecular epidemiology of P. aeruginosa in the NICU and infection control measures adopted to stop the spread of the organism. However, manuscript fails to both describe molecular epidemiology in the unit, as well as prove that the infection control measures were responsible for the decrease in the infection rate.

Major deficiencies and questions:

Methods:
1. Definition of infections used for this study is not clear (only severe infection). How about other potential infections caused by P. aeruginosa that were “not severe”?
2. Were all of the infants inborn, or some were transferred from the other institutions?
3. Were respiratory secretion cultures always considered to be infectious samples, never colonization?

Results:
4. Did the infection control measures affect the rate of infection by other organisms?
5. Did PFGE pattern of the surveillance cultures of 11 infected patients matched PFGE pattern of clinical samples in the same patients?
6. Why was PFGE pattern not identified in all of the stains? Why would the authors not determine PFGE type in 33.4% of the stains, if they were determining molecular epidemiology of the P. aeruginosa in the NICU?
7. It is hard to see how would collection of samples alone explain decrease in infection rates. How was the decision made to collect strains retrospectively and how would this affect the infection rate?
8. Why would the rate of colonization increase and infection rate decrease with surveillance and contact isolation?
9. Where were samples taken from for environmental surveillance? The only sites mentioned were sinks and hands of a nurse. In looking for environmental reservoirs, the authors could look in tap water, sink drains, liquid medications,
respiratory-therapy equipment, hand soaps, hand creams, and water baths used to warm formula, for example. How many health care workers were cultured, how often and what was the method used? The description of surveillance is extremely superficial.

Discussion:

10. In the discussion, authors are stating that the mortality of 2 of the patients is “probably affected” by multiresistant P. aeruginosa strains, however, there are no details of timing and choice of antibiotics in these infants. They are mentioning that this is “one of the first accounts on two carbapenem-resistant P. aeruginosa genetically unrelated strains which caused two sepsis in a NICU”. However, we don’t have any information on the surveillance cultures in this patients, time between their presentations and possibility of multiclonal infection.

11. As the sinks are well known reservoirs for P. aeruginosa, it is unclear how did the authors speculate that sink sample is a result not the origin of the pathogen’s circulation in the ward. How many sinks were cultured? What are the “multiple and distinct P. aeruginosa reservoirs, both environmental and human” that authors are mentioning?

12. Finally, strict adherence to hand hygiene is a well-established infection control measure. This study did nothing new to confirm that, and failed to appropriately describe (in descriptive study, as per authors’ own statement) how did a ½ hour a week hand washing education affect P. aeruginosa infections, except for association with possible transient hands colonization.

Minor comments:

14. In the first paragraph of the Methods section there is a line missing between line 5 and 6.

14. Why using three terms (nosocomial infections, hospital-acquired infections and healthcare-associated infections) in the same manuscript?

Level of interest: An article of limited interest

Quality of written English: Acceptable

Statistical review: No, the manuscript does not need to be seen by a statistician.

Declaration of competing interests:

I declare that I have no competing interests