

Sébastien Gibot

Soluble triggering receptor expressed on myeloid cells-1 and diagnosis of ventilator-associated pneumonia

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Comment on "Soluble Triggering Receptor Expressed on Myeloid cells-1 in bronchoalveolar lavage fluid is not predictive for ventilator-associated pneumonia."
Published in Intensive Care Medicine (doi:10.1007/s00134-009-1463-y).

An author's reply to this comment is available at: doi:10.1007/s00134-009-1548-7.

Dear Sir: The quest for a reliable diagnostic marker of ventilator-associated pneumonia (VAP) is still ongoing and whether determination of the alveolar concentration of the soluble form of the triggering receptor expressed on myeloid cells (TREM)-1 could be a valuable candidate is debated. Unfortunately, the study reported in this issue of Intensive Care Medicine will just generate further perplexity [1].

First, the presented work is the result of an ancillary study originating from the series reported last year in this same journal [2], which the authors should have clearly acknowledged.

This fact raises concerns regarding the quality of the samples' conservation (since 2001) especially if they have been subject to freeze/thaw cycles that rapidly degrade sTREM-1. Because alveolar sampling was initially not dedicated to sTREM-1 measurement, bronchoalveolar lavage (BAL) fluid was centrifuged at 250g for 10 min, a procedure largely insufficient to eliminate cell

particulates that express membrane-bound TREM-1 (alveolar macrophages or neutrophils): this may have led to artificially increased sTREM-1 concentrations.

Second, the effects of previous antibiotherapy of the diagnosis of VAP are not discussed. VAP was diagnosed by the presence of $\geq 2\%$ cells containing intracellular organisms (ICO) or quantitative culture of $\geq 10^4$ cfu/ml. Although previous antibiotics do not seem to interfere with ICO percentage [2], this is obviously not the case for quantitative cultures. Unfortunately, no data are presented on the number of VAP diagnosed by ICO percentage or quantitative culture: a subset of patients may have been classified as no VAP just because $< 2\%$ ICO and $< 10^4$ cfu/ml were observed while antibiotics were given. By the way, we have no idea on the final diagnosis that was retained in the no-VAP patients.

Despite the fact that all these limitations by themselves preclude the drawing of any conclusion, another problematic issue must be pointed out. The authors used a commercially available assay, namely the human TREM-1 Quantikine assay from RnD Systems (Minneapolis, USA), to measure sTREM-1 concentrations. It happens that this assay was recalled by RnD systems during the early summer of 2008 because of its inability to properly measure soluble TREM-1 concentration. I want to believe that the authors were not aware of that.

Of note, the three studies [3–5] reporting good performances of BAL sTREM-1 measurement in diagnosing VAP used an ELISA technique (Duoset, RnD Systems) different from the one employed in the two studies [1, 6] showing limited sTREM-1 usefulness (Quantikine).

It may well be that sTREM-1 measurement will finally prove to be disappointing in diagnosing VAP, but

the current study is just not interpretable.

Conflict of interest statement No conflict of interest.

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S. Gibot (✉)
Service de Réanimation Médicale, Hôpital Central, 54000 Nancy, France
e-mail: s.gibot@chu-nancy.fr
Fax: +33-3-83858511