Reviewer's report

Title: Diagnostic value of triggering receptor expressed on myeloid cells-1 and C-reactive protein for patients with lung infiltrates: an observational study

Version: 1 Date: 21 April 2010

Reviewer: eduardo ferat

Reviewer's report:

The authors state that it is important to differentiate pulmonary infiltrate of bacterial origin from that of non infectious ethiology, in order to start the appropriate therapy. TREM-1 is a receptor that amplifies the inflammatory response by producing inflammatory mediators, and although there is no recognized ligand until now, some reports propose PAMPs like LPS or DAMPs like Hsp70, as possible TREM-1 ligands. It was reported that TREM-1 ligand could be found in the serum of septic patients (Clin Exp Inmmunol 2006;145:448)

The authors include references to the works of Koussoulas, Tzivras, Ho, Mohamadzadeh and Radsak, which report increased TREM-1 expression related to non infectious inflammation. Besides them, there are other reports by Adib-Conquy (Shock 2007;28:406) and Isibasi’s group (J Surg Res 2008;150:110, Crit Care 2009;14:R69), both proposing increased TREM-1 expression associated with non infectious diseases. The last group reported increased expression of TREM-1 in surgical patients with Systemic Inflammatory Response Syndrome (SIRS) and in patients with acute pancreatitis, both since the beginning of their illness.

The standard errors seem rather small, even if the number of patients and the homogeneity of the groups are considered. How do the authors explain these small standard errors?

The patients with TB infection did not show increased expression of TREM-1, and this finding is not consistent with a report in which BCG increases TREM-1 expression (Infection and Immunity 2004;72:937). Could the authors briefly mention the clinical characteristics of these patients?

The results shown in Table 1 represent the values in the first blood sample? If this is the case, where are the results from the second and third blood samples? How many days did the non-surviving patients stayed in the ICU?

It is possible that the increased expression of TREM-1 could be related with disease severity (as assessed by increased SOFA and APACHE scores), and not with the presence of an infectious process?

It could be helpful if the authors comment how they performed the stains for flow cytometry.
The text refers to PCR results in Table 2, but in this table there are no PCR results. Do patients included in this table correspond to group A? What happened with nTREM-1 and mTREM-1 in the same group of patients?

In figure 2, the differences in the sTREM-1 panel are with respect to the column of Gram(+)/Haemophilus? It could be helpful to show your results in graphs with box and whiskers.

The patients of group A had higher values of APACHE and SOFA than those in group B. Could it be possible that the increased Inflammatory mediators are related to the severity of the illness rather than to the presence of infection?

In discussion:
You mention that sTREM-1 levels were decreased in patients from group A after the initiation of appropriate antibiotic therapy, the improvement of clinical symptoms and the resolution of CAP. It would be interesting to know how much time does it take to see this reduction.

In those patients that died, how do you explain the decrease of sTREM-1 in the second and third blood samples?
There are a lot of issues with these results that are not sufficiently explained in discussion, and there are some results that should be shown, especially those for day 3 and 7 blood samples.

**Level of interest:** An article of importance in its field

**Quality of written English:** Acceptable

**Statistical review:** Yes, but I do not feel adequately qualified to assess the statistics.

**Declaration of competing interests:**
I declare that I have no competing interest.