

Letter**Is eosinopenia a reliable marker of sepsis?**Alex Smithson¹, Rafael Perelló² and Josep-Maria Nicolas³¹Infectious Diseases Unit, Hospital Clínic, IDIBAPS, University of Barcelona, 08036 Barcelona, Spain²Emergency Department, Hospital Clínic, IDIBAPS, University of Barcelona, 08036 Barcelona, Spain³Medical Intensive Care Unit, Hospital Clínic, IDIBAPS, University of Barcelona, 08036 Barcelona, SpainCorresponding author: Alex Smithson, asa30412@hotmail.com

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See related research by Abidi *et al.*, <http://ccforum.com/content/12/2/R59>

We have read with interest the article by Abidi and colleagues [1] in which the authors point out that eosinopenia could be useful to differentiate between noninfection and infection in patients recently admitted to an intensive care unit (ICU). The association of eosinopenia with infections is not new and has been described previously [2].

To test this hypothesis, we reviewed 191 patients (age >18 years, with a minimum ICU stay of 24 hours) admitted to

the medical ICU of our hospital. We excluded HIV-infected patients and those with hematological malignancies. Total leukocyte and eosinophil count (EC) were measured at ICU admission. The results are shown in Table 1. Although the EC was lower and the proportion of patients with eosinopenia (<40 cells/ml) was higher in the noninfectious systemic inflammatory response syndrome (SIRS) group compared with the infectious SIRS group, these differences were not statistically significant. Therefore, the EC was not useful to

Table 1**Baseline characteristics of the ICU patients included in the study**

	Infectious SIRS (n = 142)	Noninfectious SIRS (n = 49)	P-value*
Age (years)	62.7 ± 15.3	66.8 ± 14.3	0.1
APACHE II score	16.6 ± 6.5	17.8 ± 6	0.27
SAPS II score	36.6 ± 12.1	36.4 ± 11.4	0.95
SOFA score	8.8 ± 3.2	7.7 ± 2.7	0.034
Length of ICU stay (days)	11.4 ± 11.3	7.5 ± 7.8	0.03
Sites of infection			
Community-acquired pneumonia	57 (40.1%)	NA	
Hospital-acquired pneumonia	9 (6.3%)	NA	
Urinary tract infection	11 (7.7%)	NA	
Bacterial meningitis	4 (2.8%)	NA	
Peritonitis	23 (16.2%)	NA	
Other infections	38 (26.7%)	NA	
ICU mortality	39 (27.4%)	13 (26.5%)	0.89
Total leukocyte count (cells/ml)	13,497 ± 7,254	10,345 ± 5,569	0.006
Total eosinophil count (cells/ml)	105 ± 220	114 ± 186	0.8
Eosinopenia (<40 cells/ml)	70 (49.3%)	18 (36.7%)	0.12

Data are presented as the mean ± standard deviation or n (%). *Calculated by means of the Student *t*-test (quantitative variables) and chi-square test (qualitative variables). APACHE, Acute Physiology and Chronic Health Evaluation; ICU, intensive care unit; NA, not applicable; SAPS, Simplified Acute Physiology Score; SIRS, systemic inflammatory response syndrome; SOFA, Sequential Organ Failure Assessment.

EC = eosinophil count; ICU = intensive care unit; SIRS = systemic inflammatory response syndrome.

distinguish between infection and noninfection. Although one limitation of our study was the absence of a non-SIRS group, the EC of our noninfectious SIRS group was similar to the EC found in the non-SIRS group in the study by Abidi and colleagues [1]. Another study failed to observe an association between eosinopenia and bacteremia [3].

In conclusion, eosinopenia was not a reliable marker of infection. Other analytical parameters, such as C-reactive protein, have demonstrated to be helpful not only for the diagnosis of infection but also as a marker of severity of organ dysfunction in sepsis [4].

Authors' response

Khalid Abidi, Ibtissam Khoudri, Jihane Belayachi, Naoufel Madani, Amine Ali Zeggwagh and Redouane Abouqal

Smithson and colleagues, in their letter on our report recently published in *Critical Care* [1], suggest that eosinopenia is not a reliable marker of infection in critically ill patients. We have demonstrated for the first time that eosinopenia is a good diagnostic marker of infection on ICU admission with good sensitivity and specificity [1].

The study performed by Smithson and colleagues has several limitations that should be considered. First, the retrospective nature of their study could cause methodological limitations,

at the least because some data were not available for all patients. Second, to evaluate the usefulness of EC to distinguish between noninfectious and infectious SIRS patients, Smithson and colleagues do not describe how the infection was defined and confirmed. Third, no non-SIRS group was included, although the authors report that the EC in the noninfectious SIRS group was similar to that found in our non-SIRS group. However, ECs for non-SIRS groups from both studies should really be determined for a completely valid comparison.

Competing interest

The authors declare that they have no competing interests.

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