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Eosinopenia: Is it a good marker of sepsis in comparison to procalcitonin and C-reactive protein levels for patients admitted to a critical care unit in an urban hospital?

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Abstract

Introduction: The role of eosinopenia as a marker of sepsis has recently been evaluated. The aim of our study was to test the value of eosinopenia as a diagnostic marker of sepsis in comparison to procalcitonin and C-reactive protein levels.

Methods: A prospective study of critically ill adult patients admitted to the medical intensive care unit at an urban hospital. Procalcitonin, C-reactive protein (CRP) levels and eosinophil counts were measured on admission. Patients were classified as non-infected or infected by the medical residents, fellows, and attendings.

Results: A total of 68 patients were enrolled into the study. At a cut-off value of 70 mg/L, the CRP level yielded a sensitivity of 94%, a specificity of 84%, a positive predicted value (PPV) of 83% and a negative predicted value (NPV) of 94%. At a cutoff value of 1.5 μg/L, the sensitivity of the procalcitonin test was 84%, specificity of 92%, PPV 90%, and NPV of 87%. The eosinophil cell count (cutoff of 50 cells/mm^3) produced a sensitivity of 81%, specificity of 65%, a PPV of 66%, and a NPV of 80%.

The comparison of the eosinophil cell count (<50 cells/mm^3) and procalcitonin levels among the non-infected and infected groups showed a significant statistical difference (Fisher exact test, $P = .0239$). There was no statistical difference observed when comparisons were made between CRP levels and eosinophil count (Fisher exact test, $P = .12$). There was also a lack of significant statistical difference when CRP levels were compared to procalcitonin levels (Fisher exact test, $P = .49$).

Conclusion: Eosinopenia is a very sensitive yet not specific serological marker of sepsis in the intensive care unit and can be utilized to guide physicians in the diagnosis of sepsis.
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1. Introduction

The early diagnosis of sepsis plays an integral role in the morbidity and mortality of patients admitted to the intensive care unit (ICU) because it ensures the early administration of

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antibiotics therapy. The clinical parameters that make up the sepsis syndrome are not specific and frequently overlap with the clinical presentation of a systemic inflammatory response syndrome (SIRS) secondary to other noninfectious causes [1-3].

There is no ideal marker of infection that is highly specific, highly sensitive, easy to measure, rapid, inexpensive, and correlated with the severity and prognosis of infection. Some studies have evaluated the role of measuring eosinophil count to assist in the early diagnosis of infection in patients admitted to the critical care unit [4]. More recent studies have looked at the role of procalcitonin levels and triggering receptor expressed on myeloid cells–1 and their ability to differentiate infectious from noninfectious causes of SIRS [5-15].

These tests are usually expensive and this coupled with the fact that it takes a long time for the results to be attained, does not make them ideal for the early diagnosis of sepsis. Eosinopenia is a common inflammatory response to acute infection [16-18], and it was first reported by Zappert et al in 1893 [17]. It has been assumed that the eosinopenia seen in acute infection is related to the production of stress-related chemo-tactic factors [19]. Gil et al studied the role of eosinopenia in inflammatory syndromes and they concluded that sepsis was strongly associated with hyperleucocytosis above 10 000 cells/mm^3 and eosinophils counts under 40 cells/mm^3 [20]. Abidi et al conducted the first study to highlight the diagnostic value of eosinopenia in distinguishing infection from non-infection in patients admitted to the ICU by comparing it to CRP [4]. However, in the Abidi et al study, eosinopenia was a moderate marker in discriminating between SIRS and infection in newly admitted critically ill patients.

The aim of this study was to compare the diagnostic value of eosinopenia, to differentiate infectious and non-infectious causes of SIRS, with procalcitonin and CRP levels in newly admitted ICU patients in an inner city hospital.

2. Materials and methods

2.1. Study design

A prospective study was conducted of adult patients admitted to a medical ICU (MICU) of St Michael's Medical Center, an inner city teaching hospital in Newark, NJ, between August 2008 and March 2009.

Patients who died or were discharged within 24 hours after admission were excluded from the study. Surgery patients were not included in the study because they are admitted to a different unit in our institution. The study protocol was approved by the institutional review board of St Michael's Medical Center. Informed consent was not demanded because this observational study did not require any deviation from routine medical practice.

2.2. Data collection and definitions

The patients were classified by admitting internal medicine residents, under the close supervision of critical care attendings (who were blinded to the eosinophils, procalcitonin and C reactive protein levels), as one of the 5 groups: negative (no systemic inflammatory response syndrome [SIRS]), SIRS, sepsis, severe sepsis, or septic shock.

According to the Criteria of the American College of Chest Physicians/Society of Critical Care Medicine [2], patients were classified as having systemic inflammatory response syndrome (SIRS), sepsis, severe sepsis, or septic shock at the time of admission. SIRS is defined by two or more of the following criteria: body temperature >38°C or <36°C, heart rate >90 beats/min, respiratory rate >20/min or $PaCO_2$ <32 Torr, and white blood cell count >12 000 cells/mm^3, <4000 cells/mm^3, or >10% immature forms. Sepsis is a SIRS with the presence of an infectious process. Infection was diagnosed by standard criteria [1-4]. Severe sepsis is a sepsis associated with organ dysfunction, hypoperfusion, or hypotension (systolic blood pressure <90 mm Hg or a reduction ≥40 mm Hg from baseline). Septic shock is a subset of severe sepsis and is defined as a persisting sepsis-induced hypotension despite adequate fluid resuscitation that typically requires vasopressor support. The medical residents and fellows were educated about these standard criteria. Importantly, all medical records pertaining to each patient were retrospectively reviewed and independently classified the diagnosis as SIRS, sepsis, severe sepsis, or septic shock at the time of admission on the basis of the review of the complete patient charts, results of microbiologic cultures, and radiographs. The fellow, who was the major investigator of this study, was blinded to the eosinophil cell count, CRP, and procalcitonin levels. The fellow also determined the clinical classification of SIRS, sepsis, severe sepsis, or septic shock, prior to attaining the laboratory results.

For each patient admitted to the ICU, we evaluated their age, sex, principal diagnosis, and vital signs. The white blood cell count, the eosinophil cell count, the CRP (CRP) level and the procalcitonin level was systematically recorded on admission to the ICU and not daily during the entire ICU stay. We also documented any evidence of multi-organ dysfunction syndrome which was defined as the presence of altered organ function in 2 or more organ systems in acutely ill patients that makes it difficult to maintain homeostasis without medical intervention [21,22].

Blood samples were obtained by venipuncture on admission. The clinical practice in the unit follows the recommendations of the task force of the American College of Critical Care Medicine of the Society of Critical Care Medicine [2], blood cultures were taken if a patient's body temperature exceeded 38.3°C (101°F), if a patient has clinical signs of severe sepsis, or if there is a need for vasopressor therapy for suspected septic shock.

Other cultures including urine, cerebrospinal fluid, and respiratory secretions (including a sputum specimen for

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