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Serial serum procalcitonin changes in the prognosis of acute stroke

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Serial serum procalcitonin changes in the prognosis of acute stroke

Abstract

Inflammatory response is a principal early component in the pathophysiology of stroke [1]. Serum procalcitonin (PCT)-a marker of septicemia and infection severity [2]-has also been proposed as an indicator of systemic inflammatory response in noninfectious situations [3,4]. As no data exist thus far on PCT in stroke, this study aimed to evaluate serum PCT changes in the acute stroke setting, and to correlate them with clinical and laboratory parameters and patient's outcome.

Keywords

procalcitonin, serum, acute, serial, stroke, changes, prognosis

Disciplines

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Letter to the Editor

Serial serum procalcitonin changes in the prognosis of acute stroke

To the Editor:

Inflammatory response is a principal early component in the pathophysiology of stroke [1]. Serum procalcitonin (PCT)—a marker of septicemia and infection severity [2]—has also been proposed as an indicator of systemic inflammatory response in non-infectious situations [3,4]. As no data exist thus far on PCT in stroke, this study aimed to evaluate serum PCT changes in the acute stroke setting, and to correlate them with clinical and laboratory parameters and patient's outcome.

All consecutive patients admitted with first-ever stroke during a 3-month period were included. Exclusion criteria included resolution of symptoms within 24 h (TIA), coexistence of a chronic illness and presence of $T \geq 37.8$ °C or evidence of coexisting infection upon presentation.

For all patients, demographic data and risk factors for stroke were recorded. Thirty patients (14 males, 16 females) were eligible for the study, mean age 77.8 years old. At presentation and at day 7, the Glasgow Coma Scale (GCS), the National Institute of Health Stroke Scale (NIHSS), and the Acute Physiology And Chronic Health Evaluation (APACHE) II score (only on admission) were performed, and full blood count, fibrinogen, C-reactive protein (CRP), glucose, urea, creatinine, electrolytes, albumin, total protein, lipids, creatine kinase (CK), lactate dehydrogenase (LDH) and PCT were measured. PCT was also measured on days 2, 3 and 4, since serum PCT levels reach their peak between 48 and 72 h [3,4]. An axillary temperature measurement was taken every 3 h in all patients.

PCT levels were measured by the use of an immunoluminometric assay (Brahms Diagnostica,

Berlin, Germany). The lower limit of detection of the assay was 0.08 ng/mL. With this assay, serum levels of PCT in healthy adults were less than 0.1 ng/mL [2,4]. Laboratory values were reported as the median values on each measurement day. A two-sided p value <0.05 was considered as statistically significant. The study protocol was approved by our Institutional Ethics Committee.

The main characteristics of serum PCT levels are shown in Fig. 1. The highest median PCT level were recorded on days 2 and 3 (0.17 and 0.18 ng/ml, respectively). No statistically significant differences were observed for the comparison of PCT values between individual days. Nine patients (30%) exhibited serum PCT levels not exceeding the lower detection limit of the method on any measurement day. In seven patients (23%) PCT levels reached their peak at day 7. A statistically significant association was observed between cases exhibiting peak PCT levels at day 7 and the presence of fever (P -value 0.043, Mann–Whitney test). Analyzing the 13 patients with fever separately, median PCT levels at day 7 were higher for eight patients with documented infection, but the difference could not reach statistical significance. Three patients suffered a brain hemorrhage, whereas 27 had a diagnosis of ischemic stroke. Nine patients (30%) succumbed due to stroke or to related complications during the 30-day period following the attack. Among the 21 survivors, 8 (38%) were functionally independent on discharge (Modified Rankin Scale ≤ 3). No correlations were recorded between PCT levels and death, neurologic outcome on discharge or stroke type.

In contrast with PCT, APACHE II score ($p=0.002$), NIHSS score ($p=0.001$), GCS score ($p=0.001$) and serum CK ($p=0.028$) on admission and at day 7 (data not shown), serum LDH at day 7 ($p=0.042$), stroke subtype ($p=0.004$) and the presence of fever ($p=0.020$) were all significantly associated with stroke mortality.

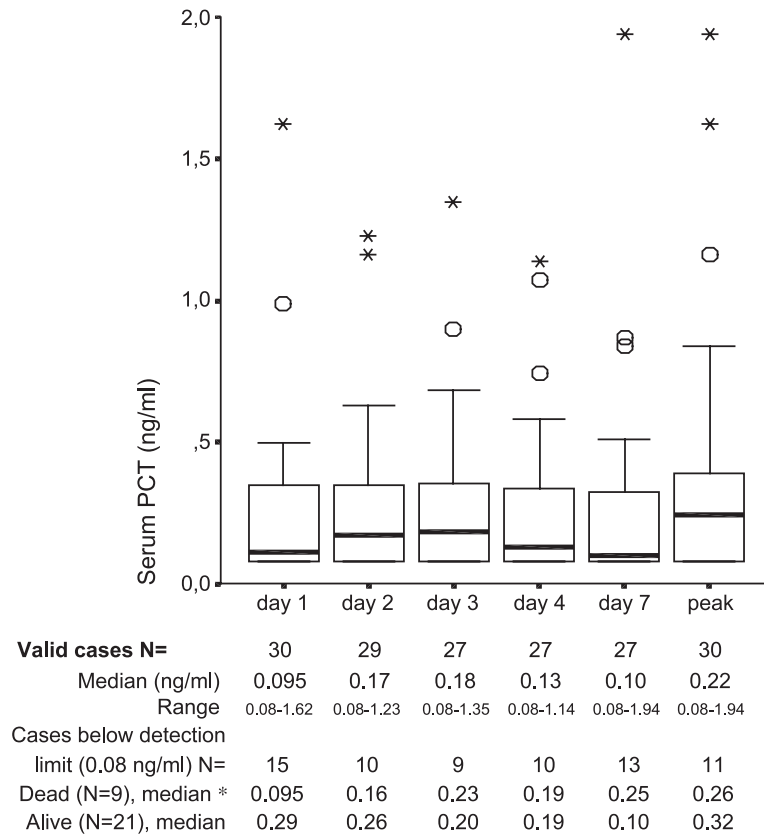


Fig. 1. Evolution of serum Procalcitonin (PCT) levels, as well as peak serum PCT levels for the patients of the study. Results are depicted as median (**bold horizontal line**), 25th–75th percentiles (**box**) and 90th percentiles (**plot lines**). Cases that are outliers (with values between 1.5 and 3 box lengths from the upper edge of the box) are represented with circles (O), whereas extremes (cases with values more than 3 box lengths from the upper edge of the box) are illustrated as asterisks (*). For each case series, are also given the number of valid cases, the median, the minimum and maximum values, as well as the number of cases with values below the PCT detection limit. *Not statistically significant differences between the median of dead and alive patients.

Neurologic outcome correlated with APACHE II score and GCS score on admission, NIHSS score on admission and at day 7, as well as with CK at day 7 (data not shown).

This was a pilot study on the prognostic value of PCT in acute stroke. Serial serum PCT levels did not correlate with stroke mortality or neurologic outcome at discharge. Studies have correlated laboratory parameters with prognosis in acute stroke, sometimes with conflicting results [5–7]. Prognostic utility of laboratory parameters in acute stroke is limited by the need to perform serial measurements, together with the interrelation of at least some of the inflammatory markers. Hence, determination of stroke severity by clinical criteria and appropriate imaging techniques

remains the main prognostic tool in acute stroke settings [8], and the optimal biochemical marker (if any) is still unknown.

The present study clearly shows that serum PCT is not candidate for such a marker. Small sample size and analytical restrictions of the assay constitute the main limitations of the present study. However, the immunoluminometric method for PCT measurement is widely accepted and was used to establish the role of PCT in sepsis [2]. Moreover, other parameters and clinical scoring systems examined in this study did correlate with outcome, regardless of the small number of cases.

Increased serum PCT levels imply for an infectious origin of fever complicating an underlying non-

infectious febrile disease [9,10]. The present study was not designed to examine PCT as a marker of intercurrent infections in stroke. Nevertheless, correlation of fever with cases exhibiting peak PCT levels at day 7 might imply for a role of PCT in identifying a subset of stroke patients, which later develop fever due to infection. In this study such a hypothesis could not be sustained due to insufficient sample size. A future larger study, examining serial PCT changes after fever onset, might answer whether PCT can serve as marker of bacterial infections complicating stroke.

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