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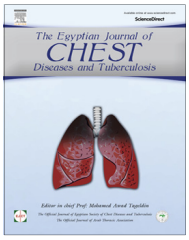

ORIGINAL ARTICLE

Diagnostic and prognostic role of procalcitonin in CAP

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KEYWORDS

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Abstract Despite advances in antimicrobial therapy, CAP remains the seventh leading cause of death in USA. Procalcitonin (PCT) is the pre-hormone of calcitonin, which is normally secreted by the C cells of the thyroid in response to hypercalcemia, its concentration was significantly increased in CAP. In lower respiratory tract infections, measuring serum PCT may aid physicians in differentiating between typical bacterial and non-bacterial causes of inflammation, using a cut-off value of 0.5 ng/mL and serum PCT guidance can reduce total antibiotic use. Furthermore, serum PCT is useful in predicting bacteraemia and in assessing disease severity in CAP patients.

Aim of the work: To determine the usefulness of procalcitonin as a predictor of etiology and prognosis in patients with CAP.

Patients and methods: This study was conducted at Tanta University Hospital over 50 patients with clinical and radiological findings compatible with CAP, 25 mild and moderate CAP and 25 severe pneumonia, thorough history taking, full Clinical examination, plain Chest X-ray, arterial blood gases, sputum samples for Gram stain and culture, blood samples for procalcitonin level measurement by monoclonal immunoluminometric assay was done.

Results: There was a statistically significant rise of PCT in severe CAP as its mean levels were 4.7 ± 0.5 and 11.9 ± 27 ng/ml in mild and severe CAP groups respectively, with a positive correlation between the level of PCT and the severity of CAP. There was a statistically significant rise of PCT in typical pneumonia with a mean level of 9.9 ± 2.24 ng/ml in comparison to atypical pneumonia with a mean level of 3.2 ± 1.96.

Conclusion: PCT measurement may provide an important indicator of severity for patients with CAP, also it can assess treatment response in these patients.

Introduction

Despite advances in antimicrobial therapy, rates of mortality due to pneumonia have not decreased significantly. Patients with certain coexisting illnesses as COPD, diabetes mellitus, congestive heart failure (CHF), coronary artery disease, active

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malignancy, immunosuppression, chronic liver and renal disease, have an increased incidence and mortality of CAP.

CAP is defined as "an acute infection of the pulmonary parenchyma that is associated with at least some symptoms of acute infection, accompanied by the presence of an acute infiltrate on a chest radiograph or auscultatory findings consistent with pneumonia (such as altered breath sounds and/or localized rales), in a patient not hospitalized or residing in a long-term care for more than 14 days before the onset of symptoms. Most patients have nonspecific symptoms such as fatigue, headache, myalgia, anorexia and symptoms of pneumonia may include fever or hypothermia, sweating, rigors, dyspnea, chest discomfort, new cough with or without expectoration, or a change in the color of sputum in patients with chronic cough [1,2].

First data regarding the increasing procalcitonin (PCT) concentration in the blood during inflammation were obtained by a group of French military doctors (Dr. Carsin, etc.), who studied markers of acute lung injury in patients with extensive burns; its concentration was significantly increased in many cases.

Retrospective analysis revealed that patients with the highest levels of procalcitonin in blood had infectious complications, including sepsis and septic shock [3].

PCT is the pre-hormone of calcitonin, which is normally secreted by the C cells of the thyroid in response to hypercalcemia; under normal conditions, negligible serum PCT concentrations are detected [4]. The mechanism proposed for PCT production after inflammation and its role are still not completely known. It is believed that PCT is produced by the liver [5] and peripheral blood mononuclear cells [6], modulated by lipopolysaccharides and sepsis-related cytokines.

Among several markers of inflammation and sepsis, PCT is studied to investigate its role and accuracy for the diagnosis of bacterial infections. PCT is a 116 amino acid peptide with no known hormonal activity [7]. In lower respiratory tract infections, measuring serum PCT may aid physicians in differentiating between typical bacterial and non-bacterial causes of inflammation, using a cut-off value of 0.5·ng/mL [8] and serum PCT guidance can reduce total antibiotic use [9]. Furthermore, serum PCT is useful in predicting bacteraemia and in assessing disease severity in patients with community-acquired pneumonia [10].

Most children with CAP are treated with antibiotics without determination of the causative agent leading to a considerable over-use of antibiotics that increases the risk of bacterial resistance, the incidence of drug-related adverse events, and therapeutic costs [11]. A number of trials have been made to differentiate viral and bacterial infections, and rationalize antibiotic use by means of easily determined biomarkers [12].

Procalcitonin stands out as one of the most accurate sepsis markers, with a superior diagnostic utility in sepsis compared with C-reactive protein, interleukin-6, and lactate [3,13]. PCT level was more sensitive (88% vs. 75%) and more specific (81% vs. 67%) than CRP level for differentiating bacterial from non-infective causes of inflammation [3].

High procalcitonin concentrations are both sensitive and specific for the diagnosis of sepsis. P. Hausfater et al. evaluated the sensitivity, specificity, and predictive value of the PCT for identifying cases of systemic infection in patients attending an emergency department. PCT concentrations assay were measured in serum samples by use of an immunoluminometric assay (LUMI test PCT; Brahms Diagnostica). The detection limit of the assay was 0.08 ng/mL, and the functional sensitivity (inter assay variation coefficient, 20%) was 0.33 ng/mL. The upper limit of normal was 0.5 ng/mL. All samples were tested in duplicate. They reported that the procalcitonin level has excellent specificity (0.99) but a sensitivity of only 0.35 (with use of a cutoff point of 0.5 g/mL) for the diagnosis of systemic infection. However, lowering the procalcitonin cutoff point to 0.2 ng/mL led to improved sensitivity (0.62) with persistent good specificity (0.88) [14].

Identifying the etiology of community-acquired pneumonia (CAP) is a clinical difficulty because single clinical, radiologic, or laboratory parameters have limited value to predict the infectious organism [3], and no rapid test has been standardized for the diagnosis of "atypical" or viral pathogens, so empirical broad-spectrum antibiotic therapy is usually chosen [4,5]. Serum PCT levels might help clinicians to choose proper antibiotic by differentiating between classic bacterial and atypical or viral etiology [15].

Aim of the work

The aim of the work was to determine the usefulness of procalcitonin as a predictor of etiology and prognosis in patients with CAP.

Patients and methods

This study was conducted at Tanta university Hospital over a 12 month period from December 2012 to December 2013 over 50 patients with clinical and radiological findings compatible with CAP, 25 mild and moderate CAP (12 males and 13 females) with a mean age of 47 ± 3.43 years and 25 severe pneumonia (11 males and 14 females) with a mean age of 45 ± 3.21 years. Four were on mechanical ventilation with no mortality.

Inclusion criteria

CAP was defined as an acute illness associated with at least one of the following symptoms as fever, new cough with or without sputum production, pleuritic chest pain, dyspnea, or change in the color of sputum in patients with chronic cough or signs as altered breath sound, rales, plus chest X ray showing an opacity compatible with acute pneumonia [1,2].

Criteria for severe CAP

Minor criteria

(1) Confusion/disorientation.
(2) Respiratory rate >30 breaths/min.
(3) Heart rate >120 beat/min.
(4) Hypotension requiring aggressive fluid resuscitation.
(5) Hypothermia (core temperature, <36 °C).
(6) Multilobar infiltrates.
(7) Leucopenia (WBC count <4000 cells/mm^3).
(8) Uremia (BUN level, >20 mg/dL).
(9) PaO_2/FiO_2 ratio <250.
(10) Thrombocytopenia (platelet count, $<100{,}000$ cells/mm^3).