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Editorial Open Access

High Serum Procalcitonin: Interpret with Caution

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Editorial

Serum procalcitonin (PCT), which is usually produced in thyroid C-cells, is a precursor of calcitonin. In general, PCT is not released into the blood and, therefore, cannot be detected in healthy individuals [1]. However, serum procalcitonin increased significantly in response to stimulation by bacterial infections showing a favorable pharmacokinetic profile as clinical markers for bacterial infection. Consequently PCT has been used increasingly to identify systemic bacterial infections since mid of 1990 [2]. Moreover, a growing body of evidence has suggested that high serum PCT is a useful biochemical marker for discriminating between sepsis and some non-infectious causes of systemic inflammatory response syndrome and is a valuable prognostic marker [3]. A rise or no change in procalcitonin level in second week was a good predictor of outcome suggesting intensification of antibiotic therapy [2]. Recent randomized clinical trials showed that serum PCT level can be used to guide antibiotic therapy in patients with severe sepsis; it resulted in a significant reduction of antibiotic therapy and similar medical outcomes. In addition, the length of intensive care treatment in the PCT-guided group was significantly shorter than that in the control group [4,5]. In febrile neutropenia, PCT was found to be useful in diagnosing bacterial infection in these patients [6]. Noteworthy, most of the above mentioned clinical implications of PCT were derived from observational studies.

Despite these growing evidences, elevation of serum PCT continues to challenge the diagnostic acumen of physicians as more recent studies have produced conflicting results [7]. Additionally, usefulness of PCT measurement in sepsis confirmation has some limitations such as false-positive and false-negative results; some patients with sepsis may not have increased PCT levels while others with high serum PCT may not have sepsis. According to available evidences, elevation in serum PCT levels in the absence of a bacterial infection can be seen in situations of massive stress, for example after severe trauma and surgery, in patients after cardiac shock or some autoimmune diseases such as Kawasaki disease and adult-onset Still's disease [2]. Procalcitonin also may be elevated in medullary thyroid cancer, small cell lung cancer, postoperative complications, cirrhosis, pancreatitis, ischemic bowel and paraneoplastic syndrome [8,9]. Conversely, high PCT levels may not be found in patients with mycoplasma community acquired pneumonia and in patients who received antibiotic pre-treatment [5]. Second limitation is that there is no single cut-off range of PCT levels for defining sepsis. While for some types of infections and clinical settings optimal PCT cut-offs have been established, optimal cut-off levels for other infections have not been established yet. Thus, the clinical benefit and safety of using serum PCT level in different clinical settings remains undefined. Third limitation is related to the efficacy of PCT level monitoring as guidance for antibiotic therapy. This property has been tested for certain infections such as respiratory tract infections and is not applicable to other infections.

In view of available evidences it is too early to come up with final conclusions on the efficacy of PCT in diagnosing sepsis or other related situations. Therefore, high serum PCT level should be interpreted with caution; physicians should incorporate this biomarker with medical context of the disease and other tests related to infection when diagnosing sepsis.

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