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Procalcitonin – a new marker of severe infection and sepsis

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Severe infections and sepsis are accompanied by clinical and laboratory signs of systemic inflammation on the basis of which diagnosis is established. However, similar signs and symptoms may be present in patients suffering from non-infectious inflammation making it difficult to diagnose infection based on clinical findings alone. It would therefore be helpful to identify markers of infection which would allow early treatment, imperative in reducing mortality and morbidity. An ideal method or marker of infection should be cheap, easy to measure, be highly specific and sensitive, should allow early diagnosis of sepsis, correlate with the severity of infection, and help gauge the efficacy of therapeutic measures. A number of statistical tests should be employed to satisfy some of these requirements. Although many parameters have been recently named, studied and proposed as possible markers of the inflammatory response to infection, none fulfill all the requirements of an ideal marker.

In this supplement of *Intensive Care Medicine*, procalcitonin is introduced and reviewed as a new marker of the inflammatory response to infection. Procalcitonin is a propeptide of calcitonin which is normally produced in the C-cells of the thyroid glands. Procalcitonin is normally not released into the circulation so that plasma levels are very low during health. Chance and scientific insight led to the discovery that procalcitonin increases dramatically during severe systemic infections. This finding fueled the interest of investigating this parameter as a marker of severe infection or inflammation. A number of studies and reviews in this issue of *Intensive Care Medicine* focus in which way and in which patients groups can procalcitonin help in the differential diagnosis of infectious and non-infectious inflammation. It must, however, be appreciated that procalcitonin may not or may only slightly increase when an infection remains confined to a tissue or organ without systemic manifestations. It is not infection per se but infection accompanied by severe systemic reactions or poor organ perfusion that increases procalcitonin levels. These properties make procalcitonin less useful for the diagnosis of simple infections but a very promising marker of severe infections especially in the critical care setting. In this setting, therapies effective in controlling sepsis and reducing severity of disease may lead to reductions in procalcitonin levels. We would like to remind the reader that markers such procalcitonin do not replace but complement clinical signs and symptoms and other laboratory parameter in arriving at a diagnosis of sepsis and severe infections.

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