

Editorial

Factors influencing inflammatory mediators' secretion after pediatric burn injury

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Because of their extensive wounds, burn patients are exposed to inflammatory mediators, which are necessary components to integrate the complex signaling network that lead to wound closure and barrier restoration. In addition to that, the loss of the primary barrier in burn patients facilitates microorganisms invasion that induce an inflammatory response. If the infection is generalized and systemic, an exacerbated inflammatory response with unbalanced pro- and anti-inflammatory mediators may lead to sepsis, which is accompanied with metabolic disturbances and tissue damages [1]. Therefore, the secretions of the inflammatory mediators in burn patients differ, depending among other factors, on the burn surface area and the gravity of the infection [2].

The nature of the burn injury, i.e. thermal, electric, chemical or radioactive may also produce different inflammatory responses. The patient's age is also a critical factor. Children are particularly vulnerable to burn injuries, accounting in some studies, for almost 50% of all burn patients. However, the mortality rate of elderly patients with sepsis is higher than children patients with sepsis, presumably because the immune function is declined during aging [3].

In the present issue of the *Journal of Pediatric Biochemistry*, Zahran et al. report a study evaluating the serum levels of some key inflammatory mediators in Egyptian children with thermal burn admitted in the burn unit of the hospital. The authors make a comparative study of the serum levels of these inflammatory mediators in patients with- and without- sepsis, as well as patients with more or less than 30% of the total burn surface area (TBSA). In addition to the classic cytokines evaluated in these kind of studies, the authors also evaluated the serum levels of the hormone leptin and the hormone precursor procalcitonin (PCT), which have been referred to recent studies as important modulators in the inflammatory response [4,5].

The authors found an increase of inflammatory mediators, such as IL-6, TNF- α and CRP, in the burn patients at the 2nd day post-injury. These levels were even higher in patients that developed sepsis at 8th day post-injury. Consequently, serum levels of IL-6, TNF- α and CRP can be used to predict the risk of developing sepsis, as it has been reported in some European and American studies [2,6]. The differences in the serum inflammatory mediators were, however, more moderated when the total TBSA and the survival were included as a variable, i.e. higher TBSA and death have minor effects on serum levels of IL-6, TNF- α and CRP.

On the other hand, TGF- α which is involved in re-epithelization and angiogenesis, was found to be the best marker for burn injury independently of the sever-

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ity of the infection (sepsis), TBSA or survival. Similarly to previous studies [7], TGF- α levels were correlated with leptin levels since the latter one seems to induce the expression of TGF- α [7]. In fact, leptin levels were increased in post-burn injury patients but, interestingly, the levels were higher in survivors than in non-survivors. This observation is important because therapeutic strategies focused in the maintenance of certain levels of leptin may be useful in the clinic. Finally, the serum levels of PCT were also found to be a valuable marker for prognosis since their values were directly correlated with the burn injury, sepsis, higher TBSA and non survivors.

In conclusion, the patterns of inflammatory mediators showed in Egyptian children with thermal burn (with or without sepsis) do not differ significantly from other data published elsewhere in American and European population. As a consequence, serum inflammatory mediators profiling can be used as a diagnostic tool to follow the effectivity of the treatment after the burn injury, as well as to identify patients at risk of the development of sepsis. These inflammatory mediators can also be targets for potential therapies or may be used to test the efficacy of new therapies.

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References

- [1] D.G. Greenhalgh, J.R. Saffle, J.H. Holmes et al., American Burn Association consensus conference to define sepsis and infection in burns, *J Burn Care Res* **28** (2007), 776–790.
- [2] C.C. Finnerty, D.N. Herndon, D.L. Chinkes and M.G. Jeschke, Serum cytokine differences in severely burned children with and without sepsis, *Shock* **27** (2007), 4–9.
- [3] G. Escames, D. Acuna-Castroviejo, L.C. Lopez et al., Pharmacological utility of melatonin in the treatment of septic shock: experimental and clinical evidence, *J Pharm Pharmacol* **58** (2006), 1153–1165.
- [4] Z. Yilmaz, Y.O. Ilcol and I.H. Ulus, Endotoxin increases plasma leptin and ghrelin levels in dogs, *Crit Care Med* **36** (2008), 828–833.
- [5] A. Lavrentieva, T. Kontakiotis, L. Lazaridis et al., Inflammatory markers in patients with severe burn injury. What is the best indicator of sepsis? *Burns* **33** (2007), 189–194.
- [6] M.G. Schwacha, B.M. Thobe, T. Daniel and W.J. Hubbard, Impact of thermal injury on wound infiltration and the dermal inflammatory response, *J Surg Res* **158** (2010), 112–120.
- [7] P.C. Konturek, T. Brzozowski, Z. Sulekova et al., Role of leptin in ulcer healing, *Eur J Pharmacol* **414** (2001), 87–97.