Skin collagen synthesis is depressed in patients with severe sepsis

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Purpose

Collagen synthesis is a central feature of tissue regeneration. In sepsis, coagulation, inflammation and tissue regeneration are activated in order to restore homeostasis. Skin is an essential barrier in maintaining a stable internal environment. It is not known whether the host response in sepsis alters skin collagen synthesis.

Patients and methods

In this prospective observational study, experimental blisters were induced on abdominal skin four times: within the first 48 hours from the first organ failure, on the fifth day after the first set of blisters and at 3 and 6 months thereafter. Fifteen healthy adults were used as controls.

Patients: Forty-four patients with severe sepsis were enrolled. The median age was 63 years (25th–75th percentile 53–71). The median APACHE II score on admission was 26 (22–30). Thirty-day mortality was 25 %.

Measurements and main results

To evaluate skin collagen synthesis, aminoterminal propeptides of collagens III and I (PIIINP, PINP) were measured from blister fluid. PIIINP and PINP levels in septic patients were lower in comparison with controls in the early blister (40.8 μg/L [25th–75th percentile 22.2–77.1] vs. 69.6 μg/L [47.2–104.7], P= 0.028 and 69.9 μg/L [32.4–112.7] vs. 243.2 μg/L [82.3–342.9], P< 0.001, respectively) as well as in the late blister (38.8 μg/L [19.9–68.5] vs. 69.6 μg/L [47.2–104.7], P< 0.001 and 90.0 [35.1–138.8] vs. 243.2 μg/L [182.3–342.9] , P< 0.001, respectively). In long-term survivors, PIIINP and PINP levels were increased at 3 and 6 months compared with their levels in sepsis.

Conclusions

Skin collagen synthesis is depressed during severe sepsis and is followed by a compensatory response 3 and 6 months after the onset of sepsis. ❐