SYSTEMIC CYTOKINE AND ACUTE-PHASE RESPONSE IN THE EARLY PERIOD OF MECHANICAL SKIN INJURIES

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Introduction. Local infection or delayed healing develops in 15–35% of patients with mechanical skin injuries, even after timely primary debridement. The course of the wound is shaped by the early systemic immune response, yet integrated profiles of cytokines, acute-phase proteins and complement in mechanical wounds remain poorly described. The aim was to characterise the systemic immune profile of patients at hospital admission.

Materials and methods. The study was performed in 2025–2026 at the Bukhara State Medical Institute and the Bukhara branch of the Republican Research Centre of Emergency Medicine. Thirty patients with mechanical skin injuries (10 incised, 7 stab, 6 contused, 7 lacerated wounds; mean age 41.2 ± 12.1 years) were examined on admission before treatment. Thirty healthy volunteers (39.8 ± 11.4 years) formed the control group. Serum IL-1β, IL-6, TNF-α, IL-10, VEGF-A, C-reactive protein (CRP) and complement C3 were measured by ELISA. Groups were compared with Student's t-test ($p < 0.05$).

Results. All seven markers differed significantly from controls ($p < 0.001$). The largest shifts were in CRP, 35.39 ± 9.68 vs 3.84 ± 1.62 mg/L (9.2-fold), and IL-6, 47.42 ± 7.61 vs 6.18 ± 1.54 pg/mL (7.7-fold). IL-1β (22.31 ± 3.46 vs 8.14 ± 1.32 pg/mL) and TNF-α (11.25 ± 2.05 vs 4.18 ± 0.74 pg/mL) rose about 2.7-fold, and C3 by 32% (1.74 ± 0.15 vs 1.32 ± 0.11 g/L). At the same time, IL-10 increased 1.6-fold (22.58 ± 3.35 pg/mL) and VEGF-A by 13% (266.84 ± 10.37 pg/mL).

Discussion. Mechanical skin injury triggers a marked systemic inflammatory response dominated by IL-6 and CRP, with activation of the complement system.

Rising IL-10 and VEGF-A show that anti-inflammatory regulation and angiogenesis are switched on already at admission. IL-6 and CRP are the most sensitive baseline markers and can serve as the reference point for monitoring treatment.

Keywords: mechanical skin injury, cytokines, IL-6, C-reactive protein, complement C3.