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COMPLICATIONS OF FRACTURES OF THE PELVIS AND LOWER EXTREMITIES

Abstract: *this article provides information many important anatomical structures pass through the pelvic cavity, which are often damaged. Vascular injuries (for example, the iliac vein) are possible, which can cause severe bleeding, especially with injuries to the posterior pelvis. The hemorrhage may be external (indicating an open fracture) or only internal; in both cases, hemorrhagic shock may develop.*

Keywords: *fractures of the bones and pelvis include fractures of the ring bones, acetabulum, and sacroiliac joint.*

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ОСЛОЖНЕНИЯ ПРИ ПЕРЕЛОМАХ КОСТЕЙ ТАЗА И НИЖНИХ КОНЕЧНОСТЕЙ

Аннотация: *в данной статье через полость таза проходит множество важных анатомических структур, которые часто повреждаются. Возможны сосудистые повреждения (например, подвздошной вены), которые могут быть причиной тяжелого кровотечения, особенно при травмах заднего отдела таза. Кровоизлияние может быть наружным (свидетельствуя об открытом переломе) или только внутренним; в обоих случаях может развиться геморрагический шок.*

Ключевые слова: *переломам костей, таза относятся переломы, безымянных костей, вертлужной впадины, крестцово-подвздошного сочленения.*

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ТОС СУЯГИ ВА ПАСТКИ ЕКСТРЕМИТАЛАРНИНГ СИНИШИ АСОРАТЛАРИ

Аннотация: *ушбу мақолада кўплаб муҳим анатомик тузилмалар тос бўшлиғидан ўтади, улар кўпинча шикастланади. Қон томирлари шикастланиши (масалан, ёнбош венаси) мумкин, бу оғир қон кетишига олиб келиши мумкин, айниқса тос суяги шикастланиши билан. Қон кетиш ташқи (очиқ синишни кўрсатадиган) ёки фақат ички бўлиши мумкин; иккала ҳолатда ҳам геморрагик шок ривожланиши мумкин.*

Калит сўзлар: *тос суяклари синишига: ёнбош суяги, бўкса, думғоза-ёнбош бирикмалари сохаси синишлари.*

Severe pelvic fractures and long-bone fractures of the lower extremities in polytrauma are frequently complicated by systemic inflammatory response syndrome (SIRS) that can progress to sepsis and multiple organ failure. Trauma-related sepsis occurs in approximately 20–30% of critically injured patients and contributes substantially to mortality [1.2.4]. The immunopa thogenesis involves an initial hyperinflammatory phase followed by compensatory immunosuppression, predisposing patients to infection. Early identification of patients at high risk for sepsis using immune markers could improve outcomes.

Objective: To determine the prognostic value of key immunological and inflammatory markers – IgA, IgG, C-reactive protein (CRP), vitamin D, and osteocalcin – in predicting septic complications in patients with pelvic and lower limb fractures in the intensive care setting.

Materials and Methods. A prospective study was conducted at the Bukhara branch of the Emergency Medicine Center (Uzbekistan) from 2019 to 2024, including 68 patients with severe pelvic and/or lower extremity long-bone fractures requiring intensive care. Mean age was 34.5 ± 9.8 years; 70% were male. Inclusion criteria: high-energy trauma with unstable pelvic fractures or femur/tibia fractures; admission to ICU.

Exclusion: pre-existing severe immunodeficiency or sepsis on admission.

Patients were stratified into groups:

Main group: 21 patients who developed septic or severe infectious complications during hospitalization (e.g., pneumonia, wound infection, sepsis);

Comparison group: 27 patients of similar injury severity who did not develop major infections;

Control group: 20 healthy blood donors for baseline reference. Blood samples were taken at admission and daily for the first 7 days to measure IgA, IgG, high-sensitivity CRP, 25(OH)vitamin D, and osteocalcin. Standard clinical and laboratory parameters (white cell count, procalcitonin, SOFA score) were also recorded. Groups were compared using

Student's t-test or Mann–Whitney U-test; prognostic performance was evaluated via ROC curve analysis.

Results.

Septic complications (meeting Sepsis-3 criteria) occurred in 15 patients (22% of the cohort) in the main group, with a sepsis-attributable mortality of 33%. The main group showed distinct immunologic deviations compared to patients without complications. Patients who went on to develop sepsis had significantly elevated CRP on admission (mean 96±15 mg/L vs 45±10 mg/L in non-septic patients, $p<0.01$), along with paradoxically lower immunoglobulin levels: IgA averaged 1.8±0.4 g/L versus 2.5±0.5 g/L in patients without complications ($p<0.05$), and a similar relative decrease was seen in IgG (~20% lower, $p<0.05$). On post-trauma days 3–5, the main group exhibited a second CRP "spike" >100 mg/L, whereas CRP in uncomplicated patients declined below 50 mg/L by that time.Vitamin D deficiency (<20 ng/mL) was prevalent in both groups but more pronounced in those with complications (mean 15±4 ng/mL vs 21±5 ng/mL, $p<0.05$).

Osteocalcin levels were broadly suppressed in the acute phase for all patients (less than half of normal), reflecting stress-induced bone formation inhibition; however, by 3 weeks post-injury, osteocalcin rose significantly in patients without sepsis (indicating active fracture healing), whereas it remained low in septic patients, correlating with delayed bone union.

Analysis of prognostic performance showed that an admission CRP > 80 mg/L was an early predictor of sepsis (sensitivity 88%, specificity 70%). Low IgA on admission (< 2.0 g/L) was also associated with higher risk of sepsis (OR ~4.7, $p=0.03$). The combination of CRP>80 and IgA<2.0 yielded an overall predictive accuracy of ~85% (AUC 0.87). Clinically, the main group required longer ICU stays (12 vs 7 days median, $p<0.05$) and had a higher incidence of organ dysfunction (28% vs 4%).

Discussion.

Our findings underscore the pivotal role of immune dysfunction in post-traumatic sepsis. A high early CRP level signals a robust inflammatory response that, if unchecked, can transition to infection [1.3]. This aligns with prior studies showing that persistently elevated CRP by days 3–4 post-surgery is associated with the development of deep infections. The observed decrease in IgA/IgG in severely injured patients suggests trauma-induced immunosuppression – an "immune paralysis" phenomenon described in critical illness [5.6]. Indeed, literature reports that simultaneous low IgG, IgA, IgM levels after severe trauma predict poor outcomes. Conversely, we also noted that patients who did mount high IgA/IgG responses at sepsis onset had worse outcomes, mirroring results from the ALBIOS trial (2021) where excessive IgA/IgG at sepsis onset correlated with higher mortality [2], possibly reflecting a dysregulated or autoaggressive immune response.

Vitamin D status emerged as another key factor: deficiency was ubiquitous and more severe in those with complications. Vitamin D is known to modulate immunity, and deficiency (present in ~70% of ICU patients) is linked to higher infection rates and mortality [6]. Our data support this trend, suggesting that screening for and correcting hypovitaminosis D in trauma patients could be beneficial.

Universally low osteocalcin in acute trauma indicates suppressed bone formation; however, its prolonged suppression in septic patients correlated with their delayed fracture healing, highlighting that systemic inflammation adversely affects bone repair.

Conclusion.

An exaggerated systemic inflammatory response and subsequent immune dysfunction are central to the development of sepsis in patients with major fractures. Elevated CRP in the first 24 hours and depressed immunoglobulin levels (IgA, IgG) are valuable early warning markers of impending sepsis. Routine monitoring of these markers, alongside vitamin D status, should be considered in the ICU management protocol for severe trauma. Identifying high-risk patients by their immune profile enables timely prophylactic interventions – such as intensified monitoring, early broad-spectrum antibiotics, or adjunctive immunotherapy (e.g., IV immunoglobulins which in turn may reduce sepsis incidence and improve survival.

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