

Peritoneal IL-8 as a Candidate Biomarker for Traumatic Brain Injury in Damage-Control Laparotomy Patients

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Introduction: In response to intraabdominal trauma, the peritoneum generates a pro-inflammatory-mediator rich fluid. Previous studies show that cytokine levels in cerebrospinal fluid (CSF) have been identified to be strong biomarkers for traumatic brain injury, particularly cytokines IL-1 β and IL-6 being upregulated in response to blood-brain barrier disruption. However, the relationship between peritoneal fluid inflammatory profiles and traumatic brain injury (TBI) is ill-defined. This retrospective study aims to identify if the elevation of certain cytokines in peritoneal fluid samples taken from trauma patients following damage control laparotomy is associated with diagnosis of traumatic brain injury.

Methods: Peritoneal fluid was collected from adult trauma patients who underwent damage control laparotomy (N=40). Data collected from samples and included demographic information, type of trauma, abbreviated injury scale (AIS) scores, and cytokine profiles were determined using a Multiplex assay. Adult trauma patients will be stratified by presence or absence of TBI. TBI severity was determined based on the Glasgow Coma Scale (GCS) as Mild (13–15), Moderate (9–12), and Severe (3–8). Two-sided Mann–Whitney U + Benjamini-Hochberg FDR correction was used as the primary cytokine screen.

Results: Of the 40 patients, 8 had TBI (20%). Log-transformed cytokine concentrations were compared between patients with and without TBI using Mann-Whitney U test. Patients with TBI demonstrated significantly lower peritoneal concentrations of IL-8 (median 848.0 vs. 8,761.2 pg/mL; Mann–Whitney $p = 0.0018$), IL-1 β (11.7 vs. 113.9 pg/mL; $p = 0.0167$), and IL-6 (2,165.1 vs. 5,187.7 pg/mL; $p = 0.0367$) compared with patients without TBI. After Benjamini–Hochberg correction for multiple comparisons, only IL-8 remained significant ($q = 0.0178$). IL-1 β ($p = 0.00167$, $q = 0.0835$) and IL-6 ($p = 0.0367$, $q = 0.1223$) showed non-significant trends toward lower concentrations in TBI patients. No significant differences were observed for IFN- γ , IL-4, IL-10, IL-17A, MCP-1, FGF-2, or VEGF.

Conclusion: IL-8 is associated with neutrophil recruitment, suggesting that TBI may impair neutrophil recruitment to the site of injury. With this information, IL-8 presents itself as a potential biomarker for TBI in peritoneal fluid collected from damage-control laparotomy patients. Future studies with a larger sample size are necessary to establish the robustness of this association.