Category :**Brain: head trauma**

A136 - ***Il1b and tnfa association with functional outcome in adults with traumatic brain injury admitted to icu: a prospective cohort***

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Introduction:

A prospective cohort was conducted to establish the association between inflammatory cytokines and outcomes in patients with Traumatic Brain Injury (TBI) admitted to the Intensive Care Unit (ICU). The inflammatory response after TBI might play a major role in healing and recovery. However, a dysregulated inflammatory response could be deleterious. A better understanding of these dynamics could improve TBI outcomes.

Methods:

From December 2019 to August 2023, we conducted a prospective cohort in a single Colombian center. Patients older than 17 yo, admitted to the ICU for moderate and severe TBI, and an mRS < 3 were enrolled. Data was collected in RedCap, including demographics, the severity of the injury, lab tests, and hospital variables. Blood samples were drawn at 72 h post-injury. IL-1B, IL-6, and TNF-a levels were measured. Outcomes at 6 months were evaluated using the Glasgow Outcome Scale-Extended (GOSE). The unfavorable outcome was defined as GOSE < 4. Univariate and multivariate analysis assessed the association between inflammatory cytokines and GOSE. RStudio was used for the analysis.

Results:

During the study, 96 patients were included. The median (IQR) age was 35.5 (24.2-53.7), and the most frequent cause was road traffic accidents (58%). 83/96 patients (86%) had severe TBI (AIS head $\geq$ 3). At 6-month follow-up, 41/96 (42%) had GOSE < 4. Univariate analysis revealed significant associations between unfavorable outcomes and age, head AIS, APACHE II, TNF-a, IL-1B, and IL-6 at 72 hours. Multivariate logistic regression, adjusted by age, head trauma severity, and systemic compromise, yielded three models for each cytokine. Following adjustment, IL-1B and TNF-a showed significant association. Table 1.

Conclusion:

In this cohort, higher levels of IL-1B and TNF-a measured at 72 h post-injury were positively correlated to fatality and disability. Further research is needed to understand the role of inflammation after TBI and its use as a potential therapeutic target.

Table:

Variables	Univariate Analysis (OR [95% CI]; p-value)	IL - 1B Multivariate Analysis (OR [95% CI]; p-valu	TNF-a Multivariate Analysis (OR [95% CI]; p-value)
Age	1,04 [1,01 - 1,07]; 0.01	1,06 [1,02 - 1,11]; < 0.01	1,04 [1,01 - 1,09]; 0.03
Admission GCS	0,77 [0,68 - 0,88]; <0.01		
AIS head	3,10 [1,67 - 5,76]; <0.01	2,63 [1,08 - 6,36]; 0.03	2,77 [1,07 - 7,16]; 0.03
APACHE II	1,21 [1,10 - 1,33]; <0.01	1,21 [1,05 - 1,38]; 0.01	1,21 [1,05 - 1,40]; < 0.01
Interleukin 1 Beta at 72 hours of admission	1,35 [0,93 - 1,95]; 0.11	1,48 [1,01 - 2,18]; 0.04	
Interleukin 6 at 72 hours of admission	1,01 [1,00 - 1,02]; 0.04		
Tumor necrosis factor αat	1,14 [1,05 - 1,23]; <0.01		1,13 [1,03 - 1,25]; 0.01

72 hours of admission

Table 1. Univariate and multivariate analysis of clinical variables and cytokines with a 6-month unfavorable outcome in patients with TBI admitted to ICU.