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ABSTRACT N. PO21

EXPLORING ENDOTHELIAL DAMAGE: THE INTERPLAY BETWEEN COAGULOPATHY, CAPILLARY LEAK AND VASOPLEGIA IN SEPSIS.

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Background: Sepsis-Induced Coagulopathy (SIC) is a severe complication characterized by
alterations in hemostasis, associated with increased mortality. Endothelial damage, triggered by
cytokine storm, plays a crucial role in SIC. Endothelial damage is also known to cause increased
capillary permeability and vasoplegia. The interconnection between these phenomena remains
clinically unexplored.

Aim: To evaluate the clinical relationship between tissue permeability alteration, coagulopathy, and
vasoplegia caused by sepsis-induced endothelial damage.

Methods: A prospective single-center study conducted on 75 patients with community-acquired
sepsis admitted to the Intermediate Care Unit. The following were analyzed: SIC score (according
to ISTH criteria), serum albumin (a surrogate for capillary leak), Total Peripheral Resistance Index
(TPRI) (a surrogate for vasoplegia), obtained through NICaS hemodynamic monitoring. Structural
Equation Modeling (SEM) was performed to explore the relationship between variables,
hypothesizing a common latent factor (endothelial damage). Principal Component Analysis (PCA)
was used to assess the presence of a common component among variables explaining their variance.

Results: The mean SIC score was 3.4 (SD 1.3), with 44% of patients affected by SIC. TPRI and
albumin showed mean values of 1954 (SD 738) and 2.58 (SD 0.59), respectively, both negatively
correlated with SIC: TPRI -0.263 ($p=0.023$) and albumin -0.454 ($p<0.001$). SEM revealed that SIC,
albumin, and TPRI are associated with a latent factor (endothelial damage), which explains 68% of
the variance. The model showed excellent fit (CFI=1.000, RMSEA=0.000) with SIC as the

reference variable, albumin inversely correlated with the latent factor ($p=0.004$) and TPRI
significantly associated ($p=0.003$). PCA identified three principal components explaining the
variance: albumin (58.95% of variance, loading = 0.606), representing metabolic state; SIC
(23.24% of variance, loading = -0.590), representing coagulation state; TPRI (17.81% of variance,
loading = 0.534), representing hemodynamic state.

Conclusions: SEM and PCA highlight interconnections among permeability, coagulation, and
hemodynamics, hypothesizing endothelial damage as a common factor.

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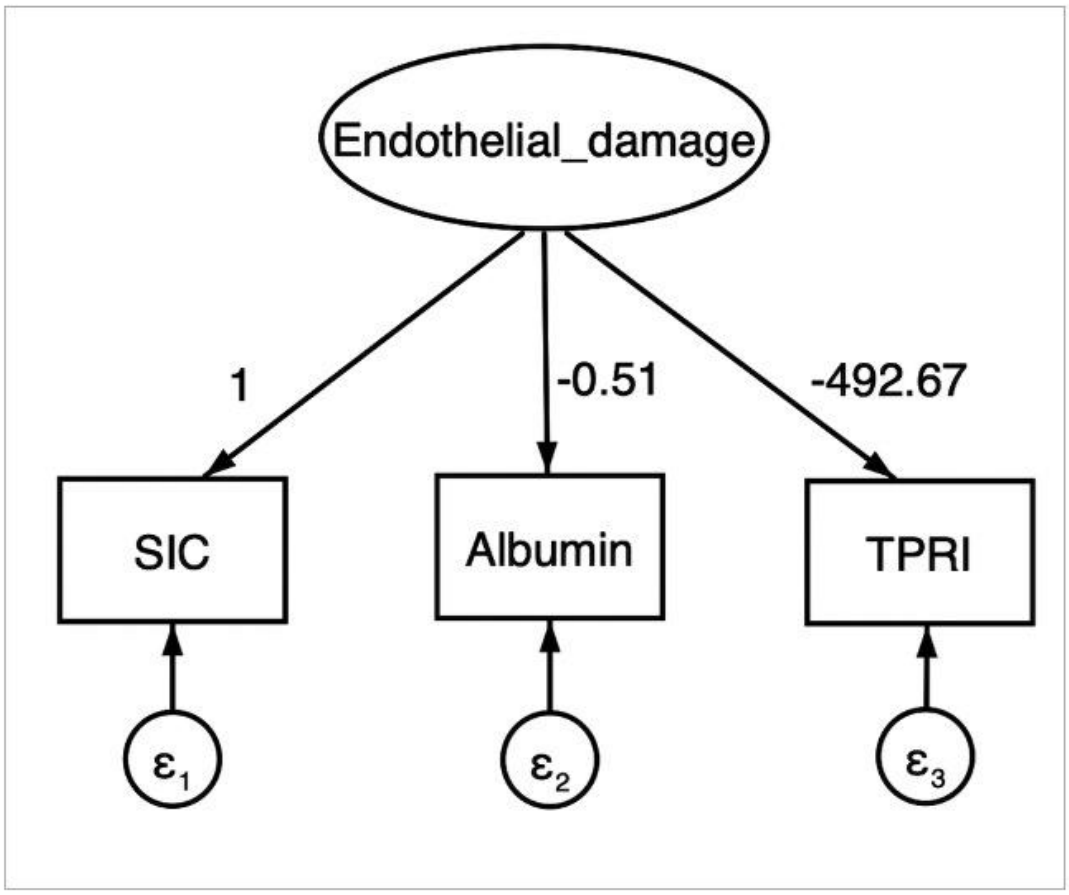

Structural Equation Modeling (SEM) showing the hypothesized latent factor of endothelial damage and its associations with SIC score, albumin, and TPRI. SIC is used as the reference variable (coefficient 1), while albumin shows an inverse relationship with the latent factor (coefficient -0.51, $p<0.01$), and TPRI is significantly negatively associated (coefficient -492.67, $p<0.01$). The model explains 68% of the total variance and exhibits excellent fit indices (CFI=1.000, RMSEA=0.000).