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Introduction:
Sepsis-induced brain dysfunction has been neglected until recently due to the absence of specific clinical or biological markers. There is increasing evidence that sepsis may pose substantial risks for long term cognitive impairment.

Methods:
To find out clinical and inflammatory factors associated with acute sepsis-induced brain dysfunction (SIBD) serum levels of cytokines, complement breakdown products and neurodegeneration markers were measured by ELISA in sera of 86 SIBD patients and 33 healthy controls. Association between these biological markers and cognitive test results was investigated.

Results:
SIBD patients showed significantly increased IL-6, IL-8, IL-10 and C4d levels and decreased TNF-α, IL-12, C5a and iC3b levels than healthy controls. No significant alteration was observed in neuronal loss and neurodegeneration marker (neuron specific enolase (NSE), amyloid β, tau) levels. Increased IL-1β, IL-6, IL-8, IL-10, TNF-α and decreased C4d, C5a and iC3b levels were associated with septic shock, coma and mortality. Transient mild cognitive impairment was observed in 7 of 21 patients who underwent neuropsychological assessment. Cognitive dysfunction and neuronal loss were associated with increased duration of septic shock and delirium but not baseline serum levels of inflammation and neurodegeneration markers.

Conclusions:
Increased cytokine levels, decreased complement activity and increased neuronal loss are indicators of poor prognosis and adverse events in SIBD. Cognitive dysfunction and neuronal destruction in SIBD do not seem to be associated with systemic inflammation factors and Alzheimer disease-type neurodegeneration but rather with increased duration of neuronal dysfunction and enhanced exposure of the brain to sepsis-inducing pathogens.