

S-100 protein

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S-100 protein, described initially by Moore, constitutes a large family of at least 20 [proteins](#) with calcium-binding ability. It is found as homo- or hetero-dimers of two different subunits (A and B). Types S-100AB and S-100BB are described as S-100B proteins and are shown to be highly specific for nervous tissue. It is present in the [cytosol](#) of glial and Schwann cells, and also in adipocytes and chondrocytes, although in very low concentrations in the latter two. The role of protein S-100B is not yet fully understood. It is suggested that it has intracellular and extracellular neurotropic as well as neurotoxic functions. At nanomolar levels, S-100B stimulates neurite outgrowth and enhances the survival of neurons. However, at micromolar levels, it stimulates the expression of inflammatory cytokines and induces apoptosis. Recently, serum S-100B protein has been proven to be an attractive surrogate marker of primary severe brain injury and secondary insults. It can be measured in the arterial and venous serum; it is not affected by hemolysis and remains stable for several hours without the need for immediate analysis. Its short half-life makes measurements crucial in emergency and intensive care settings ¹⁾.

Brain tumors

Data suggest the possibility of using S-100 as an additional biochemical criterion of cerebral lesions in oncology patients ²⁾.

Creutzfeldt Jacob Disease

Serum assays for S-100 protein are so insensitive and nonspecific that it can only be used as a diagnostic adjunct

Measurement of serum concentrations of S100 is a valuable tool that can be used more easily than tests on the cerebrospinal fluid in the differential diagnosis of Creutzfeldt-Jakob disease. More studies are needed to determine whether serial testing of serum S100 improves diagnostic accuracy ³⁾.

S100B in traumatic brain injury

[S100B in traumatic brain injury](#)

S100 in meningioma

[S100 in meningioma](#)

S100B in Neurovascular compression syndrome

[S100B in Neurovascular compression syndrome](#)

Case series

In 102 patients (45 SAH and 57 TBI) under intensive care unit (ICU) treated between January 2011 and December 2012 with [external ventricular drain](#) (EVD) S100B measurements were performed simultaneously in serum and CSF during the first 5 days and before and after EVD exchange. [Glasgow coma scale](#) (GCS) was assessed on admission and [Glasgow outcome scale](#) (GOS) 6 months later.

Peak S100B levels in CSF and serum were measured on the first day after admission and concentrations decreased during the ensuing days post injury gradually. CSF and serum S100B concentrations in TBI patients were significantly higher than in SAH ($p<0.005$). Both in TBI and SAH patients S100B concentrations in CSF and serum were significantly higher in patients with an unfavorable outcome (GOS 1-3) in comparison to patients with a good outcome (GOS 4-5). Correlation of S100B concentrations in serum and GOS score at 6 months was significant both in TBI and SAH ($p<0.05$). Serum S100B concentrations $>0.7\mu\text{g/l}$ correlated with 100% mortality. Correlation between S100B in CSF and GOS was significant in SAH ($p<0.05$), whereas it was not significant in TBI. After EVD exchange ($n=53$) we found a significant increase of S100B concentration in CSF ($p<0.005$).

Initial S100B levels have a limited prognostic value in neurotrauma with CSF concentrations being highly sensitive to smallest influences like EVD placement. However, high initial S100B levels of $>0.7\mu\text{g/dl}$ in serum are associated with 100% mortality, which might help to guide therapy strategies in severe neurotrauma ⁴⁾

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