Enolase 2

Gamma-enolase, also known as enolase 2 (ENO2) or neuron-specific enolase (NSE), is an enzyme that in humans is encoded by the ENO2 gene.

Gamma-enolase is a phosphopyruvate hydratase.

Gamma-enolase is one of the three enolase isoenzymes found in mammals. This isoenzyme, a homodimer, is found in mature neurons and cells of neuronal origin. A switch from alpha enolase to gamma enolase occurs in neural tissue during development in rats and primates.

The value of neuron-specific enolase (NSE) in predicting clinical outcomes has been investigated in a variety of neurological disorders.

Results suggest the hypothesis that trigeminal neuralgia would be related to the neural damage instead of the systemic inflammatory status and indicate NSE as a possible biomarker of response in patients submitted to MVD [1)].

Talk et al. retrospectively reviewed the records of patients with SAH from June 2008 to June 2012. The severity of SAH bleeding at admission was measured radiographically with the Fisher scale and clinically with the Glasgow Coma Scale, Hunt and Hess grade, and World Federation of Neurosurgical Societies grading. Outcomes were assessed with the modified Rankin Scale at discharge.

They identified 309 patients with nontraumatic SAH, and 71 had NSE testing. Median age was 54 years (range, 23-87 years), and 44% were male. In multivariable analysis, increased NSE was associated with a poorer Hunt and Hess grade (P = .003), World Federation of Neurologic Surgeons scale score (P < .001), and Glasgow Coma Scale score (P = .003) and worse outcomes (modified Rankin Scale at discharge; P = .001). There was no significant association between NSE level and Fisher grade (P = .81) in multivariable analysis.

They found a significant association between higher NSE levels and poorer clinical presentations and worse outcomes. Although it is still early for any relevant clinical conclusions, the results suggest that NSE holds promise as a tool for screening patients at increased risk of poor outcomes after SAH [2)].

Assessment of initial disease severity after subarachnoid haemorrhage (SAH) remains difficult. The objective of a study is to identify biochemical markers of brain damage in peripheral blood after SAH. Hospital admission S100beta, glial fibrillary acidic protein (GFAP) and neuron-specific enolase (NSE) serum levels were analysed in 67 patients with SAH. Disease severity was determined by using the World Federation of Neurological Surgeons (WFNS) scale and the Fisher CT (computerized tomography) grading scale. Mean astroglial serum concentrations taken at hospital admission were increased (S100beta 2.8-fold and GFAP 1.8-fold) compared with the upper limit of normal laboratory reference values (P95). The mean NSE concentration was within normal limits. S100beta (P < 0.001) and GFAP (P =0.011) but not NSE levels were higher in patients who were in coma at the time of hospital admission compared with patients who were not. Similarly S100beta and GFAP but not NSE serum levels increased with higher WFNS scores, raised intracranial pressure and higher CT Fisher

grade scores. Concerning the location of the aneurysm, S100beta and GFAP serum levels were within normal limits after a perimesencephalic type of haemorrhage and significantly increased after aneurysmal type SAH. Increased glial (S100beta and GFAP) but not neuronal (NSE) protein serum concentrations are found after SAH, associated to the clinical severity of the initial injury [3].

1)
Rapisarda A, Baroni S, Gentili V, Moretti G, Burattini B, Sarlo F, Olivi A, Urbani A, Montano N. The role of biomarkers in drug-resistant trigeminal neuralgia: a prospective study in patients submitted to surgical treatment. Neurol Sci. 2022 Feb 28. doi: 10.1007/s10072-022-05971-7. Epub ahead of print. PMID: 35226213.
2)
Tawk RG, Grewal SS, Heckman MG, Rawal B, Miller DA, Edmonston D, Ferguson JL, Navarro R, Ng L, Brown BL, Meschia JF, Freeman WD. The Relationship Between Serum Neuron-Specific Enolase Levels and Severity of Bleeding and Functional Outcomes in Patients With Nontraumatic Subarachnoid Hemorrhage. Neurosurgery. 2016 Apr;78(4):487-91. doi: 10.1227/NEU.0000000000001140. PubMed PMID: 26606669.
3)
Vos PE, van Gils M, Beems T, Zimmerman C, Verbeek MM. Increased GFAP and S100beta but not NSE serum levels after subarachnoid haemorrhage are associated with clinical severity. Eur J Neurol. 2006 Jun;13(6):632-8. PubMed PMID: 16796588.