

Cysteine, sulfite, and glutamate toxicity: a cause of ALS?

[Woolsey PB.](#)

pbwoolsey@NutritionConsultant.net

BACKGROUND: Amyotrophic lateral sclerosis (ALS) of nonmutant superoxide dismutase (SOD) type may be caused by toxicity of the reduced glutathione (GSH) precursors glutamate and cysteine, and sulfite (a metabolite of cysteine), which accumulate when one or more of the enzymes needed for GSH synthesis are defective. **OBJECTIVES:** A case is examined where the patient exhibited elevated sulfur on a hair mineral analysis, elevated blood cysteine, positive urine sulfite, elevated urine glutamate, and low whole blood GSH. During the time when strict dietary and supplement measures normalized the patient's whole blood GSH, blood cysteine, and urine sulfite, the patient did not experience additional physical decline. The possible causes of abnormalities of the patient's laboratory test results, as well as the nutrition measures used to normalize them, are discussed in relationship to the functions and importance of cysteine, sulfite, and glutamate in glutathione metabolism in ALS. **CONCLUSIONS:** Since elevated plasma cysteine has been reported in other ALS patients, sulfite and cysteine toxicity may be involved in other cases of ALS. Patients with ALS with nonmutant-SOD should be tested for sulfite toxicity, cysteine, glutamate and GSH levels, and whether they have low levels of GSH metabolism enzymes. Since glutamate metabolism appears to be inhibited by sulfite, research on the effect of sulfite on glutamate levels in patients with ALS should be pursued. Life might be prolonged in those patients with ALS with sulfite toxicity by closely monitoring the blood cysteine and urine sulfite levels and minimizing their dietary intake, as well as increasing GSH by using sublingual GSH. A long-term solution might be found through research to determine methods to increase GSH synthesis without using sulfur-containing supplements that may add to the cysteine and sulfite toxicity.

PMID: 18973429 [PubMed - indexed for MEDLINE]

Related articles

Decreased glutamate transport by the brain and spinal cord in amyotrophic lateral sclerosis [N Engl J Med. 1992]

Amyotrophic lateral sclerosis: amino acid levels in plasma and cerebrospinal fluid [Ann Neurol. 1990]

Increased [35S]glutathione binding sites in spinal cords from patients with amyotrophic lateral sclerosis [Neurosci Lett. 1993]

Review Serotonergic mechanisms in amyotrophic lateral sclerosis [Int J Neurosci. 2006]

Review Isolated sulfite oxidase deficiency: a case report with a novel mutation [Pediatrics. 2005]

» See reviews... | » See all...

Recent Activity

[Turn Off](#) [Clear](#)

Cysteine, sulfite, and glutamate toxicity: a cause of ALS?