Abstract


Selenium deficiency occurs in some patients with moderate-to-severe cirrhosis and can be corrected by administration of selenate but not selenomethionine: a randomized controlled trial.

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BACKGROUND: Selenomethionine, which is the principal dietary form of selenium, is metabolized by the liver to selenide, which is the form of the element required for the synthesis of selenoproteins. The liver synthesizes selenium-rich selenoprotein P (SEPP1) and secretes it into the plasma to supply extrahepatic tissues with selenium.

OBJECTIVES: We conducted a randomized controlled trial to determine whether cirrhosis is associated with functional selenium deficiency (the lack of selenium for the process of selenoprotein synthesis even though selenium intake is not limited) and, if it is, whether the deficiency is associated with impairment of selenomethionine metabolism.

DESIGN: Patients with Child-Pugh (C-P) classes A, B, and C (mild, moderate, and severe, respectively) cirrhosis were supplemented with a placebo or supranutritional amounts of selenium as selenate (200 or 400 μg/d) or as selenomethionine (200 μg/d) for 4 wk. Plasma SEPP1 concentration and glutathione peroxidase (GPX) activity, the latter due largely to the selenoprotein GPX3 secreted by the kidneys, were measured before and after supplementation.

RESULTS: GPX activity was increased more by both doses of selenate than by the placebo in C-P class B patients. The activity was not increased more by selenomethionine supplementation than by the placebo in C-P class B patients. Plasma selenium was increased more by 400 μg Se as selenate than by the placebo in C-P class C patients. Within the groups who responded to selenate, there was a considerable variation in responses.

CONCLUSION: These results indicate that severe cirrhosis causes mild functional selenium deficiency in some patients that is associated with impaired metabolism of selenomethionine. This trial was registered at clinicaltrials.gov as NCT00271245.

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