

REVERSAL OF SELENIUM DEFICIENCY WITH ORAL SELENIUM

To the Editor: Although selenium deficiency causing low erythrocyte glutathione peroxidase activity and cardiomyopathy has been recognized in human beings,^{1,2} proof is lacking that supplements of selenium will reverse the abnormal state. Van Rij et al.¹ reported little change in the selenium level and glutathione peroxidase activity in erythrocytes after 24 days of intravenous selenium supplementation in a patient with selenium deficiency. We report here the effect of oral selenium supplementation on selenium concentration and glutathione peroxidase activity in erythrocytes.

After total parenteral nutrition for 14 months, during which multiple enterocutaneous fistulas from necrotizing pancreatitis were repaired, a 39-year-old man had deficiencies in erythrocyte selenium concentration (0.042 ng per milligram of hemoglobin; normal, 0.7 ± 0.2 ng per milligram of hemoglobin) and in erythrocyte glutathione peroxidase activity (2.86 IU per gram of hemoglobin; normal, 17.5 IU per gram of hemoglobin). (Glutathione peroxidase is a selenium-dependent enzyme.³) His cardiac rhythm was normal. Conventional solutions used for total parenteral nutrition are low in selenium.^{4,5} For the next 12 days, selenium was given orally (100 μ g four times daily for one week, then 25 μ g four times daily). The erythrocyte glutathione peroxidase activity approached 50 per cent of the normal level (9.5 IU per gram of hemoglobin; control, 17.3 IU per gram of hemoglobin). After oral selenium supplementation for five months, both the erythrocyte selenium concentration (0.6 ng per milligram of hemoglobin) and the erythrocyte glutathione peroxidase activity (20.4 IU per gram of hemoglobin) returned to normal.

Oral supplementation with selenium reversed the biochemical evidence of the patient's deficiency state.

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