Plasma Selenium Levels in Renal Failure

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Dear Sir,

I would like to express my congratulations to Dr. Kallistratos et al. [1] for what I assume was a carefully conducted experiment on selenium levels in renal failure. However, I will also lend a word of caution to the unbridled speculation that accompanied this work. First, despite the epidemiological work relating selenium levels in the environment to cancer and cardiomyopathy, there is no proven selenium deficiency in man which is responsive to therapy. However, in animals, where selenium-deficient diseases have been documented (e.g. white muscle disease, liver necrosis), there is a consistent finding that levels must drop by a factor of 10 (0.01 – 0.05 ppm) from normal (0.0 – 0.5 ppm) before the disease becomes apparent [2]. In normal healthy human beings, blood levels can drop from an average of 25.6 µg/dl in Rapid City, S. Dak., USA, to 15.7 µg/dl in Lima, Ohio, USA [3]. This 38% decrease is much more dramatic and statistically significant than the 12% and 17% differences which you report, yet it too is clinically insignificant because it is unaccompanied by any documented pathology. On the other hand, selenium toxicity in humans is well documented [4–9]. Before physicians consider any supplementation or breath the word deficiency, they should give pause to the thought that selenium is toxic at dietary levels comparable to the nutritional requirement for some trace elements [2].

One should not be surprised by the statistically lower levels of selenium in renal failure. There are several reasons to expect such data. Selenium levels have been shown to decrease with decreasing hematocrits [10]. In the anemia of chronic renal failure or the anemia of chronic disease with malignancies, one should expect

low selenium levels. In animals, inorganic selenium needs to be converted to organoselenium compounds to be active biologically. These compounds include: Se-methyl-selenocysteine ($\text{CH}_3\text{-Se-CH}_2\text{-CH}_2\text{-COOH}$); Se-methyl-selenomethionine ($(\text{CH}_3)_2\text{Se-CH}_2\text{-CH}_2\text{-CH}_2\text{-COOH}$); selenocystathionine ($\text{HOOC-CHNH}_2\text{-CH}_2\text{-Se-CH}_2\text{-CH}_2\text{-CHNH}_2\text{-COOH}$); selenohomocystine ($\text{HOOC-CHNH}_2\text{-CH}_2\text{-CH}_2\text{-Se-Se-CH}_2\text{-CH}_2\text{-CHNH}_2\text{-COOH}$); selenocystine ($\text{HOOC-CHNH}_2\text{-CH}_2\text{-CH}_2\text{-Se-Se-CH}_2\text{-CHNH}_2\text{-COOH}$), and selenomethionine ($(\text{CH}_3\text{-Se-CH}_2\text{-CH}_2\text{-CHNH}_2\text{-COOH})$).

Since all of these compounds depend upon essential amino acids, it is not surprising that protein-restricted malnourished patients with renal failure and cancer would have reduced circulating levels. Indeed, protein malnutrition lowered selenium levels from 14 to 8 µg/dl in one study [11], again more dramatic than the finding presented in the work of Kallistratos et al. [1].

Finally, although Dr. Kallistratos and his coworkers found no age-related differences in their selenium levels, such differences have been reported [10]. Selenium levels have been shown to decline with increasing age [10]. The control group presented in this work with a mean age of

27.2 years makes any comparison of data somewhat dubious when they are compared to the renal failure groups with mean ages of 51.1 and 50.8 years for the nondialysis and dialysis patients, respectively.

Since there is no evidence at present to consider the data presented by Dr. Kallistratos clinically significant, and there is a great potential hazard to any supplementation, I trust physicians will heed my word of caution before declaring any deficiency of selenium in dialysis patients based on the present data.

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