Selenium supplementation may improve COVID-19 survival in sickle cell disease

Abstract
Sickle cell disease is associated with lower selenium levels, and the serum selenium level is inversely associated with haemolysis in SCD. The SCD population is more vulnerable to adverse COVID-19 outcomes. SARS-CoV-2 infection lowers the serum selenium level and this is associated with severity of COVID-19. Selenium supplementation is proposed to improve COVID-19 outcomes in the sickle cell disease population.

Further to Ulfberg & Stehlik’s letter of September 29th, further evidence supports the role of Se in COVID-19 virulence(1). In their pre-print analysis by machine learning of Medicare patients, Dun et al. found that the leading comorbidity associated with COVID-19 mortality, adjusted for age and race, was sickle cell disease (aOR, 1.73; (95% CI 1.21, 2.47)), followed by chronic kidney disease (aOR, 1.32; (95% CI 1.29, 1.36))(2).

Both SCD and kidney disease can lower Se levels by decreasing tubular Se resorption and are associated with deficient Se status.(3,4).

Se status or intake has been correlated with COVID-19 outcomes, including mortality and recovery rates, in four patient groups in China, Germany, South Korea and southern India(5–8). SARS-CoV-2, like other RNA viruses, sequesters Se causing Se levels to drop during infection.(6,9). SARS-CoV-2 may infect cells in bone marrow, suppressing red blood cell formation(10). Se status is inversely associated with haemolysis in SCD and may both inhibit haemolysis and enhance erythropoiesis in SCD.(3,11).

It should be noted that vitamin C and Mg are also commonly deficient nutrients and are required for the activation of vitamin D3 by hydroxylase(12,13). Se supplementation has been associated with COVID-19 and COVID-19 outcomes in hospital populations(14).

Se, supplemented if necessary with its cofactors in vitamin D metabolism, is proposed to be an important protective factor in the general population, but has the potential to reduce mortality from SARS-CoV-2 infection in the sickle cell disease population to an even greater extent.

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References


