# Serum selenium concentrations in cirrhotic children

## Sirozlu çocuklarda serum selenyum konsantrasyonu

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***Background/aims:*** *Selenium is an essential trace element for humans. Plasma selenium concentration is decreased in adults with cirrhosis. We aimed to investigate the serum selenium concentration in cirrhotic children.* ***Methods:*** *The serum selenium concentration of 38 patients was determined by spectrofluorometric method. The results of the patients were compared with those of 41 age- and gender-matched healthy children. Correlations between the liver function tests, Child classes and serum selenium concentrations in cirrhotic children were also investigated.* ***Results:*** *The mean serum selenium concentration in cirrhotic children was significantly lower than that of controls (42.4 ± 8.2 µg/L vs 64.4 ± 16.9 µg/L, p<0.05). There was no significant difference between the serum selenium concentrations of cirrhotic children who were in Child-Pugh class A versus B+C (p>0.05). Except for serum aspartate aminotransferase level (Pearson coefficient = -0.34), there was no correlation between serum selenium concentration and liver function tests in cirrhotic children.* ***Conclusions:*** *Serum selenium concentration in cirrhotic children was found to be low; supportive selenium administration may be beneficial in cirrhotic children in appropriate cases.*

**Key words**: Cirrhosis, selenium, children

***Amaç:*** *Selenyum insanlar için esansiyel bir eser elementtir. Erişkinlerde sirozda plazma selenyum konsantrasyonunda azalma gösterilmiştir. Bu çalışmada sirozlu çocuklarda selenyum konsantrasyonu araştırılması planlanmıştır.* ***Yöntem:*** *Otuzsekiz sirozlu çocuğun serum selenyum konsantrasyonu spektroflometrik yöntemle ölçülmüş ve sonuçlar yaş ve cinsiyet uyumlu 41 sağlıklı çocukla karşılaştırılmıştır. Serum selenyum konsantrasyonları ile hastaların karaciğer fonksiyon testleri ve Child siroz sınıflamaları da karşılaştırılmıştır.* ***Sonuçlar:*** *Sirozlu çocukların serum selenyum konsantrasyonu sağlıklı kontrol grubundan belirgin olarak düşük bulunmuştur (sırasıyla 42.4 ± 8.2 µg/L ve 64.4 ± 16.9 µg/L, p<0.05). Child-Pugh sınıflamasına göre A ve B+C olan gruplar arasında selenyum konsantrasyonları arasında fark bulunmamıştır. Serum aspartat aminotransferaz dışında (pearson katsayısı:-0,34) diğer karaciğer fonksiyon testleriyle serum selenyum düzeyleri arasında korelasyon saptanmamıştır. Sirozlu çocuklarda serum selenyum seviyesi düşük bulunmuştur. Sirotik çocukların destekleyici selenyum tedavisi verilmesi açısından değerlendirilmeleri uygun olabilir.*

**Anahtar kelimeler**: Siroz, selenyum, çocuk

## INTRODUCTION

Selenium (Se) is an essential trace element for humans. It is the essential component of several major metabolic pathways, being the constituent of human selenoproteins (1). The most important and known action is its antioxidant effect because it forms selenocysteine, part of the active center of the glutathione peroxidase (GSH-Px) enzyme (2). Se is also essential for optimal endocrine functions; regulates thyroid hormone synthesis and metabolism; plays a role in proper liver function; is essential in endothelial development and in cardiac muscle function; is required for fertility; moderates mood and cognitive functions; has antiviral properties; and is a potent chemopreventive agent at supranutritional levels. It is obtained from dietary sources including cereals, grains and vegetables. The Se content in food principally depends on the concentration and physicochemical forms existing in the soil (2-4). The measurement of Se in plasma (or serum) is a frequently used method for assessment of Se nutritional status (1).

Serum Se levels were found significantly lower in both alcoholic and non-alcoholic liver diseases compared to healthy controls (5). Further, mean serum Se concentrations were found significantly lower in cirrhotic adults when compared with

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patients with chronic hepatitis (6). In another study, it was found that although plasma Se is decreased, the increase in plasma GSH-Px activity, which occurs in patients with cirrhosis, suggests that they do not have Se deficiency (7).

The aim of this study was to investigate the serum Se concentrations in cirrhotic children.

## MATERIALS AND METHODS

A total of 38 cirrhotic children were enrolled. The congenital metabolic diseases such as Wilson disease and biliary atresia were the most common causes of cirrhosis. Of all patients, 11 were diagnosed as Wilson's disease, 5 as extrahepatic biliary atresia, 3 as autoimmune hepatitis, 3 as progressive familial intrahepatic cholestasis, 2 as cystic fibrosis, 1 each as tyrosinemia and Budd-Chiari syndrome, and 12 as cryptogenic cirrhosis.

The serum Se concentrations of 38 cirrhotic children (26 male; 54.2%) with a mean age of 10.6 ± 4.3 years (3 to 17 years) and 41 age- and sex-matched controls were determined using a spectrofluorometric method (normal >50 µg/L).

Patients were evaluated for the presence of growth retardation. Growth retardation was defined when height measurement was below the 2SD score corresponding to the height for the age- and sex-matched healthy children.

Correlations between the liver function tests (serum aspartate and alanine aminotransferases (AST, ALT), gamma glutamyl transferase (GGT), total bilirubin, albumin, prothrombin time) and serum Se concentrations in cirrhotic children were investigated.

The Child classes of cirrhotic children were determined from clinical and laboratory data according to the Child-Pugh classification using assessments of ascites, encephalopathy, bilirubin, albumin, and prothrombin time (8). The differences in the serum Se concentration of the patients according to the different Child classes were investigated.

Statistical analysis was performed using a SPSS for Windows version 10.0 program. The data were expressed as mean ± SD. Paired and unpaired t-tests, chi-square test, Mann-Whitney test and Pearson correlation coefficients were used for statistical analysis as appropriate.

## RESULTS

The mean serum Se concentration in cirrhotic children was 42.4 ± 8.2 µg/L (range: 26.7-58.9) and of the controls was 64.4 ± 16.9 µg/L (range: 39.0-125.1). There was no correlation between age and gender and Se concentration in cirrhotic children. The mean serum Se concentration in cirrhotic children was significantly lower compared to that of healthy controls ($p<0.05$). Thirty-one (81.6%) of the cirrhotic children and 5 (12.2%) of the controls had low serum Se concentration. Twenty-two (57.9%) of the children were in Child-Pugh class A, and their mean serum Se concentration was 42.6 ± 7.2 µg/L. The other 16 (42.1%) cirrhotic children, who were in Child-Pugh class B+C, had a mean serum Se concentration of 42.1 ± 9.7 µg/L. There was no significant difference between the serum Se concentrations of cirrhotic children who were in Child-Pugh class A versus B+C ($p>0.05$). Except for serum AST level (Pearson coefficient = -0.34), there was no correlation between serum Se concentration and liver function tests of cirrhotic children. There was no significant difference between the serum Se concentration of patients with or without growth retardation. Seventeen of the patients were defined as growth retardation and 13 (76.5%) of them had low serum Se levels, while 18 (85.7%) of 21 patients without growth retardation had low serum Se levels. The relation between the etiology of cirrhosis and Se levels could not be evaluated due to the small numbers of the groups.

## DISCUSSION

There are few studies related with the Se levels of children with cirrhosis. In this study, the serum Se concentration in cirrhotic children was significantly lower when compared with the controls. Burk et al. (7) confirmed that adult patients with cirrhosis have a depressed plasma Se concentration, and they reported that selenoprotein P synthesized by the liver is depressed whereas GSH-Px synthesized by the kidneys is increased in cirrhotics. Their explanation for these changes in cirrhotic patients was that cirrhosis impairs the hepatic synthesis of selenoprotein P. Thuluvath et al. (5) reported that Se levels are low in liver disease irrespective of etiology and that low levels of Se are more likely to be related to the overall nutritional status of patients. It was also shown that progressive enhancement in the hepatic injury produced a major decrease in the serum Se levels (9). However, we did not find any difference between the serum Se concentration of the patients in Child-Pugh class A compared to those in class B+C. This might be due to the low num-

bers of our patients. Se deficiency occurs in some areas where people eat primarily plant-derived foods grown locally in Se-deficient soil, such as in China (1, 3). Se status of Turkish children was found to be lower than that reported in the literature; marginal Se deficiency could be important in the development of some Se deficiency-related diseases (10). Aras et al. (11) reported low Se content of soil and low Se intake in people in Turkey. Although Navarro-Alarcon et al. (6) showed a correlation between the serum Se concentration and serum total cholesterol and GGT levels, we could not show any correlation between the serum Se concentration and liver function tests except for AST levels. Children with protein deficiency diseases, Kwashiorkor or marasmus, tend to be low in Se. Malnourished children appear to have increased needs for Se due to pro-oxidative defects of malnutrition. We could not find any difference between serum Se levels of patients with or without growth retardation, but we believe that children with cirrhosis and growth retardation must be carefully evaluated for trace elements. Se is required for the proper functioning of the immune system, and its deficiency affects the occurrence, virulence or disease progression of some viral infections (2). Although our group was very heterogeneous for liver disease etiology, liver function status and age, we believe that this study will at least show the Se status of cirrhotic children and lead to further investigations. We were not able to study the relation between Se level and oxidative stress markers in the serum or in the liver of children with liver cirrhosis, and this is the weakest point of the study.

As a result, Se supplementation in cirrhotic children should be kept in mind. Further studies are necessary to define the role of Se in hepatic diseases, the most appropriate dose of Se and the timing to initiate the supplementation trial.

In conclusion, cirrhotic children have low levels of Se, and supplementation should be remembered in appropriate cases.

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