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Serum Selenium Levels in Patients with Abdominal Aortic Aneurysm: A Case-Control Study and Implications for Pathophysiology

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SÚHRN

Cieľ: Abdominálna aortálna aneuryzma (AAA) je chronické cievne ochorenie spojené so zápalom a oxidačným stresom. Selén je esenciálny stopový prvok zapojený do antioxidačnej obrany, avšak jeho vzťah k AAA u ľudí zostáva nejasný. Cieľom tejto štúdie bolo zhodnotiť asociáciu medzi sérovými koncentráciami selénu a prítomnosťou abdominálnej aortálnej aneuryzmy.
Metódy: Bola realizovaná prípadovo-kontrolná štúdia zahŕňajúca pacientov s AAA a vekovo a pohlavne párované kontrolné osoby bez aneuryzmatického ochorenia. Sérové koncentrácie selénu boli merané štandardizovanými laboratórnymi metódami. Boli zaznamenané klinické charakteristiky a kardiovaskulárne rizikové faktory. Na posúdenie asociácie medzi sérovou hladinou selénu a prítomnosťou AAA bola použitá multivariačná logistická regresná analýza.
Výsledky: Pacienti s AAA mali signifikantne vyššie sérové koncentrácie selénu v porovnaní s kontrolnou skupinou (p = 0,0009). Po korekcii na vybrané kardiovaskulárne rizikové faktory zostali zvýšené hladiny selénu nezávisle asociované s prítomnosťou AAA (pomer šancí 1,42; p = 0,04). Nebola pozorovaná signifikantná asociácia medzi sérovými koncentráciami selénu a priemerom aneuryzmy.
Záver: V tejto kohorte boli vyššie sérové koncentrácie selénu asociované s prítomnosťou abdominálnej aortálnej aneuryzmy. Klinický význam tohto nálezu zostáva neistý a môže odrážať zmenený metabolizmus stopových prvkov alebo kompenzačnú odpoveď na vaskulárny oxidačný stres. Na objasnenie úlohy selénu v patofyziológii AAA sú potrebné ďalšie prospektívne štúdie.

Keywords:
Abdominal aortic aneurysm
Biomarkers
Selenium
Trace elements
Vascular disease

ABSTRACT

Objective: Abdominal aortic aneurysm (AAA) is a chronic vascular disease associated with inflammation and oxidative stress. Selenium is an essential trace element involved in antioxidant defense, but its relationship with AAA in humans remains unclear. The aim of this study was to evaluate the association between serum selenium concentrations and the presence of abdominal aortic aneurysm.
Methods: A case-control study was performed including patients with abdominal aortic aneurysm and age- and sex-matched control subjects without aneurysmal disease. Serum selenium concentrations were measured using standardized laboratory methods. Clinical characteristics and cardiovascular risk factors were recorded. Multivariable logistic regression analysis was used to assess the association between serum selenium levels and the presence of AAA.
Results: Patients with abdominal aortic aneurysm had significantly higher serum selenium concentrations compared with control subjects (p = 0.0009). After adjustment for selected cardiovascular risk factors, elevated serum selenium levels remained independently associated with the presence of AAA (odds ratio 1.42, p = 0.04). No significant association between serum selenium concentrations and aneurysm diameter was observed.
Conclusion: In this cohort, higher serum selenium concentrations were associated with the presence of abdominal aortic aneurysm. The clinical relevance of this finding remains uncertain and may reflect altered trace element metabolism or a compensatory response to vascular oxidative stress. Further prospective studies are needed to clarify the role of selenium in AAA pathophysiology.

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Introduction

The term *aneurysm* derives from the Greek word meaning dilation. Abdominal aortic aneurysm (AAA) is defined as a progressive dilatation of the abdominal aorta, most commonly affecting the infrarenal segment, and is associated with substantial morbidity and mortality due to the risk of rupture. Multiple definitions exist depending on arterial or venous location; however, within the infrarenal aorta, a diameter of 30 mm is generally accepted in Europe as the diagnostic threshold for both men and women.[1,2] The major established risk factors for the development of AAA include smoking, age over 65 years, male sex, family history and genetic predisposition, cardiovascular comorbidities, and body metrics.[3–5] The global prevalence of abdominal aortic aneurysm increases markedly with age. Over the past two decades, both the prevalence and incidence of AAA have declined. These trends indicate that although the burden of AAA remains concentrated in older populations, overall global prevalence has shown a modest decrease in recent years.[6] According to epidemiological modelling data, in 1990 the prevalence of AAA ranged from 8.43 per 100,000 individuals aged 40–44 years to 2,422.53 per 100,000 among those aged 75–79 years. By 2010, a slight decline was observed across all age groups, with prevalence ranging from 7.88 per 100,000 in individuals aged 40–44 years to 2,274.82 per 100,000 in those aged 75–79 years.[7] Factors contributing to this decline include decreasing smoking rates, improved management of arterial hypertension, widespread use of antiplatelet therapy, broader statin uptake, and improved access to diagnostic imaging.[8] The most serious complication of AAA is rupture, resulting in massive internal bleeding into the retroperitoneal or peritoneal cavity, sudden hypotension, and hemorrhagic shock. Ruptured AAA carries an extremely high mortality rate, with an estimated global number of 150,000–200,000 deaths annually.[9] Screening programs are recommended primarily for high-risk populations.[10] Surveillance of diagnosed aneurysms is typically performed using ultrasonography, with recommended follow-up intervals of every 5 years for diameters of 25–29 mm, every 3 years for 30–39 mm, annually for 40–49 mm, and every 6 months for aneurysms measuring ≥50 mm.[10] Despite well-established risk factors such as smoking, advanced age, and hypertension, the underlying pathophysiology of AAA remains incompletely understood. Increasing evidence suggests that oxidative stress, chronic inflammation, and extracellular matrix degradation play central roles in aneurysm formation and progression.[11–13] To date, no serum biomarker is recommended for routine screening or surveillance. In this context, attention has recently focused on the role of micronutrients, particularly selenium, due to its essential function in antioxidant defense, redox homeostasis, and vascular integrity.[14,15] Experimental studies in animal models suggest that selenium may represent a potential diagnostic, therapeutic, and follow-up marker in patients with AAA.[16] The present study investigates whether serum selenium concentrations are associated with AAA diameter, with the aim of evaluating its potential role as a biomarker in clinical practice.

Materials and methods

Study design

A case-control study was conducted. A total of 40 participants were included.

The case group consisted of 20 patients with abdominal aortic aneurysm (AAA), while the control group comprised 20 age-matched individuals without AAA.

Inclusion and exclusion criteria

Participants were eligible for inclusion if they were aged 50 years or older, regardless of sex. Patients in the case group were required to have an infrarenal abdominal aortic aneurysm confirmed by computed tomography angiography (CTA) and documented by a board-certified radiologist. Only aneurysms with a minimum outer-to-outer diameter of ≥35 mm, measured on 1-cm axial slices, were included. The control group consisted of individuals with no history or imaging evidence of aneurysmal disease, including abdominal, thoracic, popliteal, or visceral aneurysms. Participants in both groups were excluded if they had a history of arteritis or systemic vasculitis, connective tissue disorders such as Marfan syndrome or Ehlers–Danlos syndrome, or if they followed specific dietary regimens that could influence trace element status. Individuals receiving either long-term or short-term supplementation with selected micronutrients, including selenium or zinc, were also excluded. To minimize potential genetic bias, individuals with a positive family history of aneurysmal disease in first-degree relatives were not included in the control group. Patients presenting with acute aneurysm rupture were not enrolled in the study.

Recruitment of participants

Recruitment and blood sample collection for both groups were carried out at the University Hospital Bratislava, Slovakia, between January 2025 and August 2025. All participants were managed and followed in the outpatient clinics of the University Hospital Bratislava.

Sample collection and processing

Venous blood samples were collected from all participants in a fasting state using Vacuette® serum tubes (Greiner Bio-One, Austria), coated with micronized silica particles to activate coagulation. Samples were transported under standard laboratory conditions to the central laboratory of Medirex a.s. (Bratislava, Slovakia) within 12 hours of collection, where pre-analytical processing was performed. Following centrifugation, serum samples were stored and subsequently transported to the Spadia laboratory (Czech Republic) for trace element analysis. Serum selenium concentrations were quantified using inductively coupled plasma mass spectrometry (ICP-MS), with a minimum required sample volume of 120 µL. Analytical results were provided to the investigators under anonymized identification codes and expressed in µmol/L. The reference interval for serum selenium concentrations established by the Spadia laboratory was 0.75–1.86 µmol/L.

Clinical data collection

For both cases and controls, data on age, sex, body mass index (BMI), presence of peripheral arterial disease, arterial hypertension, and heart failure were collected.

Statistical analysis

Continuous variables were assessed for normality using the Shapiro-Wilk test. As serum selenium concentrations and aneurysm diameters were not normally distributed, data are presented as median values with interquartile ranges (IQR). Differences between the AAA and control groups were analyzed using the Mann–Whitney U test for continuous variables. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Correlations between serum selenium levels and continuous variables, including aneurysm diameter and age, were evaluated using Pearson's correlation coefficient. *between serum selenium concentration and the presence of AAA, adjusting for potential confounders including age, sex, and BMI. **All statistical analyses were conducted using SPSS software (version 29.0; IBM Corp., Armonk, NY, USA)**. A two-sided p value < 0.05 was considered statistically significant.

Ethical considerations

All participants provided written informed consent prior to enrolment, and signed consent forms are securely archived at the University Hospital Bratislava. The study protocol was approved by the Ethics Committee of St. Cyril and Methodius Hospital, University Hospital Bratislava, and conducted in accordance with the principles of the Declaration of Helsinki.

The study was performed in accordance with the approved study protocol (ClinicalTrials.gov identifier: NCT07236281).

Results

A total of 40 participants were included, comprising 20 patients with abdominal aortic aneurysm (AAA) and 20 age-matched control subjects without AAA. Baseline demographic and clinical characteristics of the study population are summarized in **Table 1**. The median age was 74.0 years (interquartile range [IQR] 71.0–77.5) in the AAA group and 71.0 years (IQR 66.0–75.0) in the control group. Men accounted for 70% of patients with AAA and 40% of control subjects. Median body mass index (BMI) was 29.0 kg/m² (IQR 27.5–30.0) in the AAA group and 24.5 kg/m² (IQR 23.0–28.0) in controls. The prevalence of diabetes mellitus was 20% in the AAA group and 70% in controls, peripheral arterial disease was present in 25% and 90%, arterial hypertension in 90% and 100%, and heart failure in 25% and 35% of patients with AAA and controls, respectively.

Serum selenium concentrations were higher in AAA compared with controls (median [IQR]: 1.00 [0.90–1.10] µmol/L vs. 0.70 [0.60–0.80] µmol/L; p = 0.0009, Mann–Whitney U) (**Fig. 1**). Values below the laboratory reference range (0.75–1.86 µmol/L) were observed in 1/20 (5%) of AAA patients versus 12/20 (60%) of controls.

Within the AAA cohort, the median aneurysm diameter was 42 mm (IQR 39–51). A weak, non-significant inverse correlation was observed between aneurysm diameter and serum selenium concentration (Pearson r = –0.24; p = 0.317) (**Fig. 2**).

Table 1 – Baseline characteristics of the study population

Variable	AAA (n = 20)	Controls (n = 20)
Age, years (median [IQR])	74.0 [71.0–77.5]	71.0 [66.0–75.0]
Male sex, n (%)	14 (70%)	8 (40%)
BMI, kg/m² (median [IQR])	29.0 [27.5–30.0]	24.5 [23.0–28.0]
Diabetes mellitus, n (%)	4 (20%)	14 (70%)
Peripheral arterial disease, n (%)	5 (25%)	18 (90%)
Arterial hypertension, n (%)	18 (90%)	20 (100%)
Heart failure, n (%)	5 (25%)	7 (35%)
Serum selenium, µmol/L (median [IQR])	1.00 [0.90–1.10]	0.70 [0.60–0.80]

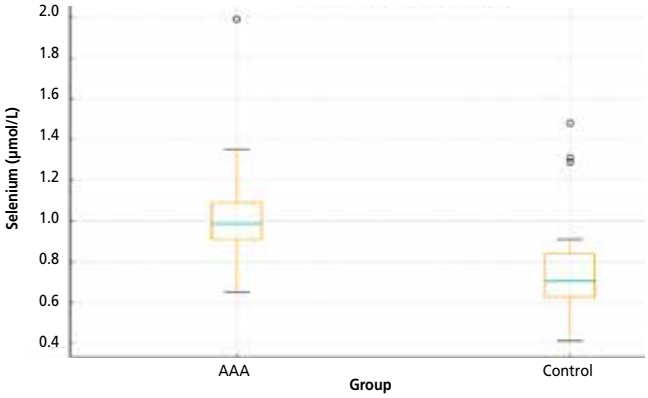

Fig. 1 – Serum selenium concentrations in patients with AAA and age-matched controls. Box-and-whisker plots represent the median, interquartile range, and range of serum selenium levels in each group. Patients with AAA exhibited significantly higher serum selenium concentrations compared with controls (p = 0.003, Mann–Whitney U test).

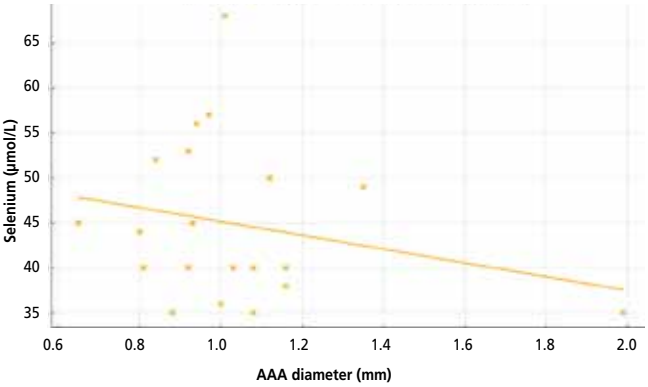

Fig. 2 – Scatter plot showing the correlation between AAA diameter and serum selenium concentration. Each point represents an individual patient with AAA. The dashed red line indicates the linear regression fit. A weak, non-significant inverse correlation was observed (r = –0.24, p = 0.317).

Selenium was also not significantly associated with age (Pearson r = –0.21; p = 0.24). In multivariable logistic regression adjusted for age, sex, and BMI (selenium scaled per 0.1 µmol/L; age per 10 years), each 0.1 µmol/L increase in serum selenium was associated with higher odds of AAA (OR 1.42, 95% CI 1.02–1.98, p = 0.04). Age, sex, and BMI were not independent predictors (p = 0.13, 0.15, and 0.15, respectively).

Discussion

In this case-control study, we explored the association between serum selenium concentrations and abdominal aortic aneurysm (AAA), as well as aneurysm diameter, with the aim of evaluating selenium as a potential biomarker in clinical research settings. The study was motivated by experimental evidence suggesting that selenium deficiency may contribute to aneurysm formation through impaired selenoprotein synthesis and reduced antioxidant protection. In animal models, selenium depletion has been shown to induce excessive oxidative stress and structural weakening of the aortic wall, ultimately promoting aneurysm development.[15] However, translation of these experimental findings to human disease remains challenging. If confirmed in human populations, selenium-related mechanisms could have potential clinical relevance, as selenium status is relatively easy to assess and potentially modifiable through dietary or pharmacological interventions. Several previous studies have reported lower serum selenium concentrations in patients with aortic aneurysms compared with healthy controls, suggesting a possible association between selenium deficiency and AAA risk.[13,16] In contrast to this hypothesis, our results demonstrated significantly higher serum selenium concentrations in patients with AAA compared with control participants. No association between serum selenium levels and aneurysm diameter was observed. Similar inconsistencies in selenium-related findings have been reported in the literature, indicating that the relationship between selenium status and AAA may be more complex than initially presumed. Several explanations may account for these observations. First, selenium metabolism in humans differs substantially from that in experimental animal models, where controlled diets and induced aneurysms do not fully replicate the multifactorial and heterogeneous nature of human disease. Second, serum selenium concentrations may not accurately reflect intracellular selenium availability or selenoprotein activity, which represent the biologically active forms responsible for antioxidant defense.[16,17] Furthermore, chronic inflammation and oxidative stress associated with AAA could influence selenium redistribution or selenoprotein turnover, potentially resulting in paradoxically elevated serum levels. Dietary habits, medication use, or unrecognized supplementation may also contribute to interindividual variability in selenium concentrations and obscure subtle associations.[13] These proposed mechanisms remain speculative and require further experimental and clinical validation. The present findings should be interpreted within the context of existing human studies investigating selenium and cardiovascular disease. While some investigations have identified lower selenium levels in patients with atherosclerosis and other cardiovascular conditions, others have reported no association or even elevated selenium concentrations in affected individuals. These discrepancies may reflect differences in study populations, regional selenium availability, analytical methodologies, and the timing of sample collection relative to disease progression.[16] Moreover, accumulating evidence suggests that both selenium deficiency and excess may exert adverse cardiovascular effects, supporting the concept of a U-shaped relationship that complicates linear interpretations. Only a limited number of studies have examined selenium specifically in relation to AAA, and the present study contributes additional human data complementary to experimental research. Several methodological limitations warrant consideration. The case-control design precludes causal inference, and the relatively small sample size may limit statistical power to detect modest associations. Selenium concentrations were measured at a single time point and may not reflect long-term exposure or temporal changes. In addition, specific selenoproteins such as glutathione peroxidase or selenoprotein P were not assessed, although they may better represent selenium's functional activity. Nevertheless, the use of standardized laboratory methods and a well-characterized clinical cohort strengthens the internal validity of the findings.

The clinical implications of this study underscore the complexity of trace element involvement in vascular pathology. While our results do not support selenium deficiency as a risk factor for AAA, they suggest that selenium may act as a modulatory factor within oxidative and inflammatory pathways rather than as a direct determinant of disease presence or severity. Future studies should employ longitudinal designs to assess whether selenium status predicts aneurysm development or progression and should incorporate mechanistic analyses of selenoprotein activity, oxidative stress markers, and inflammatory pathways. An integrated approach considering multiple micronutrients may provide a more comprehensive understanding of redox balance in AAA pathophysiology.

Conclusion

In conclusion, this study did not confirm an association between selenium deficiency and abdominal aortic aneurysm. Instead, higher serum selenium concentrations were observed in patients with AAA, underscoring the complexity of selenium metabolism in human vascular disease. No significant relationship between serum selenium levels and aneurysm diameter was identified. Although these findings do not support the immediate clinical use of serum selenium measurement for AAA screening, risk stratification, or surveillance, they do not exclude a potential modulatory role of selenium in aneurysm-related oxidative and inflammatory processes. Further large-scale, longitudinal, and mechanistic investigations are required to clarify whether selenium-related pathways may have diagnostic or therapeutic relevance in the management of abdominal aortic aneurysm.

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Conflict of interest
The authors declare that they have no conflicts of interest.

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Ethical approval
The study protocol was reviewed and approved by the Ethics Committee of St. Cyril and Methodius Hospital, University Hospital Bratislava, and the study was conducted in accordance with the principles of the Declaration of Helsinki.
Importantly, the study was prospectively registered at ClinicalTrials.gov (identifier: NCT07236281).

Informed consent
Written informed consent was obtained from all participants prior to inclusion in the study.

Data availability
The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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