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SPOT URINARY ZINC LEVELS AND NUTRITIONAL STATUS IN CHILDREN WITH ACYANOTIC CONGENITAL HEART DISEASE: A CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Acyanotic congenital heart disease (CHD) causes increased metabolic demands and nutrient malabsorption, potentially triggering zinc deficiency. Urinary zinc measurement offers a non-invasive method to assess zinc status, especially zinc excretion status. This study evaluates the association between urinary zinc levels and nutritional status in children with acyanotic CHD.

Methods: An analytical cross-sectional study involving 50 children aged 2–60 months with echocardiography-confirmed acyanotic CHD was conducted at the Pediatric Cardiology Outpatient Clinic, DR. M. Djamil General Hospital, Padang, from October 2024 to August 2025. Nutritional status was assessed by anthropometry using WHO growth standards (weight-for-height z-scores). Spot urinary zinc levels were measured using a colorimetric zinc assay kit (Elabscience, E-BC-K137-M) with absorbance read at 560 nm. One-way ANOVA and Fisher's Least Significant Difference (LSD) post hoc test were used for group comparisons.

Results: Malnutrition prevalence reached 62% (undernutrition 32%, severe malnutrition 30%). Hypozincuria (urinary zinc <8 µmol/L) was found in 38% of subjects. Urinary zinc levels varied between nutritional status groups ($p<0.001$). Urinary zinc was highest in the well-nourished group (9.6±1.2 µmol/L), followed by undernutrition (8.4±1.8 µmol/L), and lowest in severe malnutrition (6.1±1.5 µmol/L). Post-hoc analysis confirmed significant differences between well-nourished and severely malnourished ($p<0.001$), and between undernutrition and severe malnutrition ($p<0.001$).

Conclusion: Ventricular septal defect (VSD) was the most common defect identified, followed by atrial septal defect (ASD) and patent ductus arteriosus (PDA). Most subjects were malnourished, while only about one-third had normal nutritional status. Although the majority of subjects had normal urinary zinc levels, approximately one-third showed low levels. A significant association was observed between urinary zinc levels and nutritional status in children with acyanotic congenital heart disease, particularly among those who were malnourished.

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INTRODUCTION

Congenital heart disease (CHD) represents the most common category of congenital malformation in children. Globally, the birth prevalence of CHD is approximately 8 per 1,000 live births, ranging from 6 to 12 per 1,000 across different populations and referral centres [1]. The Global Burden of Disease analysis indicates that Asia carries the highest regional prevalence, reaching 9.3 per 1,000 live births [2]. In Indonesia, hospital-based data consistently report CHD incidence of 8–10 per 1,000 live births, with ventricular septal defect (VSD), atrial septal defect (ASD), and patent ductus arteriosus (PDA) constituting the three most common lesions [3,4].

Acyanotic CHD with left-to-right shunting produces chronic pulmonary overcirculation, increased ventricular volume load, and pulmonary congestion. These haemodynamic changes elevate resting energy expenditure by 30–50% above normal, generating heightened metabolic demands [5,6]. Simultaneously, pulmonary oedema and splanchnic congestion cause intestinal mucosal oedema and reduced splanchnic perfusion, directly impairing absorption of key micronutrients—including zinc—at the duodenum and proximal jejunum [7]. Malnutrition consequently occurs in 50–90% of children with CHD across multiple studies [8,9].

Zinc is an essential trace element functioning as a cofactor for more than 300 enzymes, including antioxidant enzymes such as superoxide dismutase, and plays critical roles in protein and DNA synthesis, somatic growth, and immune regulation [10,11]. Zinc deficiency has been linked to growth faltering (stunting), impaired cellular and humoral immunity, increased infection susceptibility, and delayed wound healing [10,12]. Children with CHD face compounded risk of zinc deficiency through the combination of increased metabolic requirements, malabsorption from intestinal oedema, and elevated urinary zinc losses driven by oxidative stress, neurohumoral activation (renin-angiotensin-aldosterone system), and long-term use of diuretics and ACE inhibitors [13,14].

Serum zinc, the most widely used biomarker, is susceptible to acute inflammation and diurnal variation, limiting its reliability as a single-point assessment. Urinary zinc measurement represents a promising non-invasive alternative: spot or 24-hour urinary zinc can be analysed by colorimetric or spectrometric methods, is more readily accepted by young children, and reflects short-term zinc excretion and homeostasis [15,16]. Recent evidence suggests that spot urinary zinc may be a useful population-level indicator of zinc deficiency, although internationally standardised cut-off values remain to be established [16].

Despite the strong pathophysiological rationale, data specifically linking urinary zinc concentrations to nutritional status in children with acyanotic CHD—particularly from Southeast Asian populations—are scarce. Identifying zinc deficiency early in CHD patients has significant clinical implications: if a meaningful association is confirmed, urinary zinc screening could become part of routine nutritional evaluation, enabling earlier and more targeted zinc supplementation, potentially reducing infection burden and improving long-term outcomes [17].

Based on the above background, the researchers wanted to conduct a study assessing the relationship between urinary zinc levels and nutritional status in children with cyanotic CHD, particularly at Dr. M Djamil Hospital in Padang. This study is expected to provide an initial overview of the urinary zinc profile in this population, its contribution to nutritional status, and baseline data for the development of more specific screening and nutritional intervention strategies for children with CHD in Indonesia and other Asian regions with similar epidemiological characteristics and health resources.

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METHOD

Study Design, Participants, and Sample

This analytical observational study employed a cross-sectional design and was conducted at the Pediatric Cardiology Outpatient Clinic of DR. M. Djamil General Hospital, Padang, West Sumatra, Indonesia, from October 2024 to August 2025. Ethical approval was obtained from the Research Ethics Committee of DR. M. Djamil General Hospital, with number DP.040/D.XVI.XI/468/2024. Informed consent was secured from each child's parent or legal guardian prior to enrollment. 50 Children with aged 2 to 60 months echocardiography-confirmed acyanotic congenital heart disease (CHD) — specifically atrial septal defect (ASD), ventricular septal defect (VSD), patent ductus arteriosus (PDA), or combinations thereof — attending the outpatient clinic during the study period were eligible for inclusion. Children were excluded if they had complex CHD involving lesions beyond those specified, immunodeficiency conditions (including malignancy, cytotoxic therapy, or long-term corticosteroid use), current diuretic therapy, or an acute illness within two weeks prior to assessment. Consecutive sampling was employed for participant recruitment. The minimum sample size was determined using the formula for a descriptive categorical proportion: $n = Z\alpha^2 \times P \times Q / d^2$, with $Z\alpha = 1.96$ (two-tailed, $\alpha = 0.05$), $P = 0.14$ (based on the reported prevalence of zinc deficiency in children with malnutrition from prior literature), $Q = 0.86$, and $d = 0.10$, yielding a minimum of 46 subjects. A 10% dropout allowance was added, resulting in a final target of 50 children.

Nutritional Assessment

Body weight and length/height were measured using calibrated digital scales and a stadiometer. Nutritional status was classified according to WHO 2006 growth standards using the weight-for-height (or weight-for-length) z-score: well-nourished (z-score ≥ −2 SD), undernutrition (z-score −2 to −3 SD), and severe malnutrition (z-score < −3 SD). Stunting was defined as height-for-age z-score < −2 SD.

Urinary Zinc Measurement

A single morning spot urine specimen (minimum 1 mL) was collected using a urine collector provided to the parents. Zinc concentration was determined using a commercially available colorimetric zinc assay kit (Elabscience, E-BC-K137-M). In this assay, zinc ions react with 5-Br-PADAP to form a coloured complex; absorbance was measured at 560 nm using a spectrophotometer. Urinary zinc concentration was expressed in µmol/L. In this study spot zinc was not corrected with urinary creatinin and this limitation for this study. A value below 8 µmol/L was defined as hypozincuria, consistent with reference ranges zinc excretion for children reported in the literature (normal 8–11 µmol/L) [15].

Statistical Analysis

Data were analysed using SPSS version 30. Univariate analysis described the characteristics of participants and the distribution of urinary zinc levels and nutritional status using frequencies, percentages, means, and standard deviations. Normality test before bivariate analysis with saphiro wilk with p-value >0,05. Bivariate analysis employed one-way ANOVA to compare mean urinary zinc levels across the three nutritional status groups (well-nourished, undernutrition, severe malnutrition). Post-hoc (Fisher's Least Significant Difference) pairwise comparisons were performed when the omnibus test was significant. A significance level of $\alpha = 0.05$ was adopted throughout.

RESULTS AND DISCUSSION

Participant Characteristics

Fifty children with acyanotic CHD met all inclusion criteria and were enrolled. The gender distribution was 48% and 52% for male and female, consistent with global CHD epidemiology which reports no significant sex difference in incidence [1, 2]. The mean age was 20.4 ± 16.3 months (range 2–

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60 months), reflecting a population predominantly of infants and toddlers—a critical window for somatic growth and nutritional development.

VSD was the most commonly identified lesion (42%), followed by ASD (24%), PDA (22%), and combined defects (12%). This distribution aligns with the recognised epidemiology of acyanotic CHD in both Indonesia and globally, where VSD, ASD, and PDA together account for more than 80% of cases [3, 4]. The majority of defects (58%) were classified as small on echocardiography, while 42% were large; mean defect size was 6.9 ± 3.8 mm. Haemodynamically, larger defects generate greater left-to-right shunt volumes, higher pulmonary overcirculation, and consequently greater metabolic burden, which is directly relevant to the nutritional and zinc-related outcomes described below [5,6].

The nutritional status of respondents showed considerable variation, reflecting heterogeneous nutritional health conditions within the study population. The prevalence of combined malnutrition (malnutrition + severe malnutrition) reached 62% of the total sample, a figure consistent with epidemiological reports of malnutrition in children with CHD at various referral centers [5,6]. A systematic review and meta-analysis from Diao et al, reported that malnutrition is highly prevalent among children with congenital heart disease, with pooled prevalences of 27.4% for underweight, 24.4% for stunting, and 24.8% for wasting [8].

Table 1. Characteristics of study participants

Variable	n	%
Gender		
Male	24	48.0
Female	26	52.0
Age (months)		
Mean ± SD	20.4 ± 16.3	—
Cardiac Lesion		
VSD	21	42.0
ASD	12	24.0
PDA	11	22.0
Combined defects	6	12.0
Defect size		
Small	29	58.0
Large	21	42.0
Mean ± SD (mm)	6.9 ± 3.8	—
Nutritional status		
Well-nourished	19	38.0
Undernutrition	16	32.0
Severe malnutrition	15	30.0
Stunting (short stature)		
Yes	18	36.0
No	32	64.0
Spot urinary zinc (µmol/L)		
Mean ± SD	8.1 ± 2.1	—
Normal (≥8)	31	62.0
Hypozincuria (<8)	19	38.0

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Urinary Zinc Distribution

The overall mean spot urinary zinc concentration was 8.1 ± 2.1 µmol/L. Sixty-two percent of children had urinary zinc within the normal reference range (≥8 µmol/L), while 38% exhibited hypozincuria (<8 µmol/L). This substantial proportion of hypozincuria is consistent with the pathophysiological mechanisms expected in acyanotic CHD: oxidative stress from pulmonary overcirculation and malnutrition [13,14]. Prior studies from Trasobares et al, have documented that ACE inhibitor and furosemide therapy are independently associated with lower serum zinc and higher urinary zinc excretion in heart failure patients [14], reinforcing the clinical relevance of urinary zinc monitoring in this population.

Unlike serum zinc, which is acutely affected by inflammation and circadian variation, spot urinary zinc more directly reflects renal zinc excretion and may provide a dynamic figure of short-term zinc homeostasis [15, 16]. Study by Likoswe et al. shows that spot urine zinc levels have the potential to be a good biomarker for determining zinc deficiency at the population level [17]. The finding that a meaningful minority of children in this cross sectional study showed hypozincuria supports the use of urinary zinc as a potentially useful screening biomarker, although the absence of an internationally standardised cut-off value remains a limitation for clinical application.

Association Between Urinary Zinc and Nutritional Status

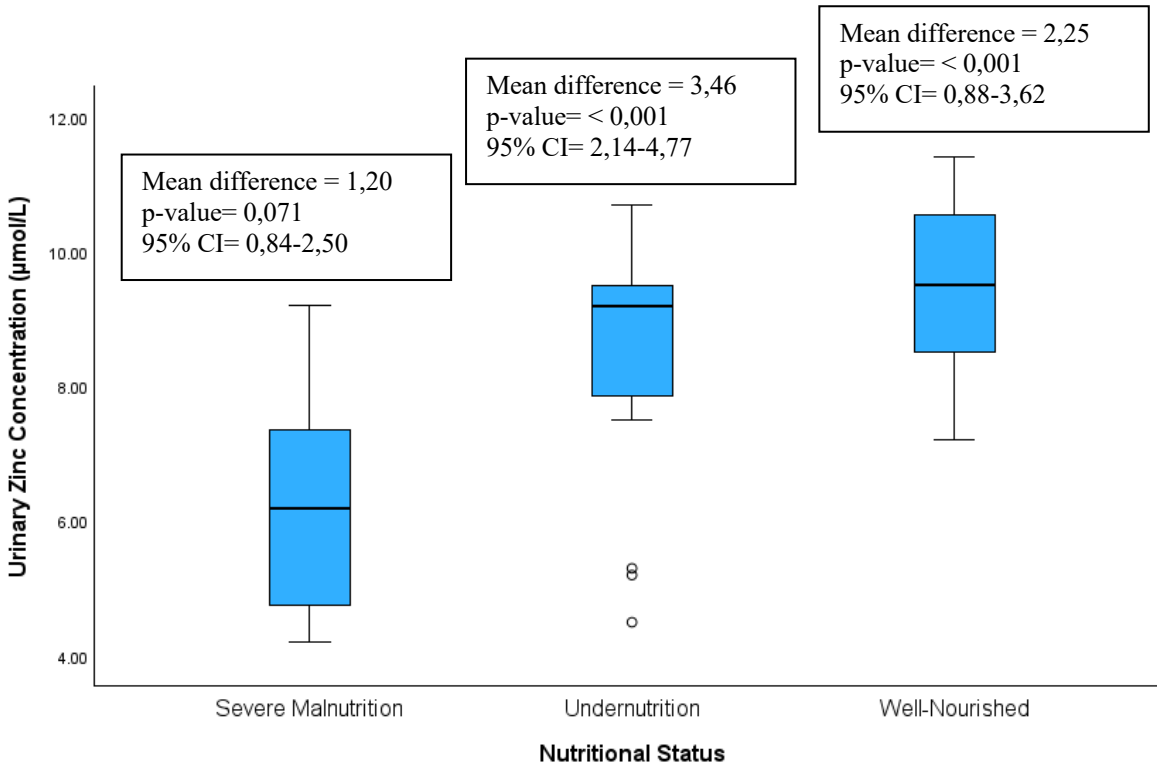

Figure 1. Box-and-whisker plots illustrating the distribution of urinary zinc concentrations (µmol/L) across three nutritional status categories: severe malnutrition, undernutrition, and well-nourished status. Each box represents the interquartile range (IQR; 25th–75th percentile), the central line denotes the median, and whiskers extend to the minimum and maximum values within 1.5× IQR. Circles (○) represent statistical outliers.

The distribution of urine zinc concentrations (µmol/L) among three groups categorized by nutritional status—severe malnutrition, undernutrition, and well-nourished status—is compared using box-and-whisker plots in Figure 1. Urinary zinc levels and nutritional status appear to be positively correlated, as seen by a continuous stepwise climbing pattern in zinc concentrations throughout nutritional status groups from left to right.

The median urine zinc content in the severe malnutrition group was the lowest of all three groups, at around 6.2 µmol/L. Significant variation in zinc levels within this subgroup was demonstrated by this

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group, which had the largest interquartile range, ranging from around 4.0 to 9.0 µmol/L. Such variability can be the result of uneven metabolic control, which is frequently linked to diseased conditions and severe dietary deficiencies.

The median zinc content rose significantly to about 9.2 µmol/L in the undernutrition group. This group displayed the smallest interquartile range (around 8.0–9.5 µmol/L) compared to the severe malnutrition group, indicating a more uniform distribution of urine zinc concentrations. Nonetheless, three data values that fell between 4.5 and 5.5 µmol/L were found to be statistical outliers. These outliers are people who, according to anthropometric standards, are classed as moderately malnourished yet have urine zinc amounts that are similar to those of the severe malnutrition category. Clinically speaking, this pattern can refer to acute vitamin shortage or underlying zinc malabsorption, necessitating further diagnostic testing.

With an interquartile range of up to 11.4 µmol/L, well-nourished status group had the highest median urine zinc content at around 9.5 µmol/L. The total distribution of the good nutritional status group was higher, indicating a larger accumulation of zinc within sufficient nutritional conditions, even though the median values for the unternutrition and well-nourished status groups were closely linked.

This study indicates a gradual relationship between the degree of malnutrition and zinc deficiency, whereby the poorer the nutritional status, the lower the detected urinary zinc levels. The results of this study are in line with a study by Escobedo-Monge, et al., which shows that zinc supplementation can provide significant benefits to the nutritional status of children with chronic diseases, with improvements in anthropometric and biochemical parameters [22]. Previous studies have evaluated serum zinc levels in children with congenital heart disease. Sadoh et al, reported that although the overall serum zinc levels were not significantly different between children with CHD and healthy controls, lower zinc levels were observed in CHD patients with pneumonia, suggesting that comorbid conditions may influence zinc status [17].

Pathophysiological Interpretation

The observed graded association between nutritional status and urinary zinc can be interpreted through a well-established pathophysiological cascade specific to acyanotic CHD. Left-to-right shunting causes pulmonary overcirculation, ventricular volume overload, and splanchnic congestion [5, 6]. Intestinal mucosal oedema and reduced mesenteric perfusion impair zinc absorption at its primary site in the duodenum and proximal jejunum [7]. At the same time, chronic energy deficit of 20–40% from elevated cardiac metabolic demands further worsens the nutritional balance [8]. Zinc deficiency then amplifies immune dysfunction by —impairing neutrophil chemotaxis, NADPH oxidase activity, T-cell differentiation, and Th1/Th2 balance. It is —increasing the risk of recurrent respiratory infections, which themselves worsen catabolism and nutritional deterioration [10,11]. This creates a self-reinforcing cycle of pulmonary overcirculation, malabsorption, zinc deficiency, immune compromise, recurrent infection, and deepening malnutrition [20]. This observation highlights that the anatomical complexity of the underlying CHD meaningfully influences both overall nutritional status and micronutrient metabolism.

Strengths and Limitations

The study benefited from confirmed diagnoses via echocardiography, a validated colorimetric assay for urinary zinc, and WHO-standardised anthropometric assessments. Urinary zinc was chosen deliberately over serum zinc to avoid the confounding effects of acute inflammation—a common scenario in CHD patients. However, several limitations warrant acknowledgement. Urinary zinc test for spot zinc was not corrected with urinary creatinin and this limitation for this study, because spot urinary zinc without creatinine correction is highly dependent on hydration status. Prospective longitudinal follow-up would be required to establish the temporal direction of the zinc–nutrition relationship. Additionally, the relatively small sample size may have reduced statistical power to detect the difference between well-nourished and undernutrition groups. Finally, serum zinc, hair zinc, or dietary zinc intake data were not collected, which limits the ability to fully characterise zinc status and its determinants.

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Conclusion

This cross-sectional study demonstrated a statistically and clinically meaningful association between spot urinary zinc levels and nutritional status in children aged 2–60 months with acyanotic congenital heart disease. VSD was the most common acyanotic congenital heart defect in this study, followed by ASD and PDA, with malnutrition affecting the majority of patients. While urinary zinc levels were generally within the normal range, a substantial proportion of children had low levels. The significant association between urinary zinc levels and nutritional status, particularly among malnourished children, suggests a potential link between zinc status and nutritional impairment in acyanotic congenital heart disease. These findings underscore the importance of routine nutritional evaluation and consideration of micronutrient status in the management of affected children with CHD.

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