

The Effect of Chronic Energy Deficiency on the Incidence of Stunting in Children Aged 24-59 Months

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ABSTRACT Chronic Energy Deficiency (CED) during pregnancy is a significant public health concern with long-term effects on maternal and child health. The study explores the impact of maternal CED on stunting in children aged 24-59 months in Ngariyoyo Public Health Center, where the prevalence of CED among pregnant women was 11.01% in 2023, and stunting affected 8.2% of children. The primary aim of this study is to determine the relationship between maternal CED and the incidence of stunting in the target population. Employing an analytical observational approach, this case-control study included 32 children with stunting as the case group and 64 non-stunted children as the control group, selected through random sampling methods. Data on maternal CED were obtained from maternal and child health records, while stunting data were gathered via questionnaires. The analysis utilized chi-square and Mantel-Haenszel common odds ratio tests to evaluate the association. The results indicated a significant association ($p=0.021$) between maternal CED and stunting, with an odds ratio of 3.201, suggesting that mothers with CED have a 3.2 times higher risk of giving birth to stunted children compared to mothers without CED. The study concludes that maternal CED is a crucial factor contributing to stunting in children. Therefore, interventions such as early identification and nutritional education for pregnant women are essential for stunting prevention. The findings emphasize the need for targeted public health policies to address maternal nutrition in rural settings.

INDEX TERMS Chronic Energy Deficiency, Stunting, Maternal Nutrition, Public Health, Pregnancy

I. INTRODUCTION

Chronic Energy Deficiency (CED) during pregnancy remains a critical public health challenge in low- and middle-income countries, particularly in Southeast Asia, due to its profound implications for maternal and child health outcomes. CED is characterized by prolonged inadequacy of energy and protein intake, commonly assessed using a mid-upper arm circumference (MUAC) threshold of less than 23.5 cm, which reflects depleted maternal nutritional reserves [1], [2]. Maternal undernutrition during pregnancy has been widely associated with adverse pregnancy outcomes, including low birth weight (LBW), preterm birth, intrauterine growth restriction, and increased maternal and neonatal morbidity and mortality [3], [4]. These adverse outcomes contribute substantially to the intergenerational cycle of malnutrition.

Stunting remains one of the most persistent manifestations of chronic undernutrition in early life and continues to pose a significant burden in developing countries. Defined as impaired linear growth resulting from long-term nutritional deprivation and recurrent infections, stunting has been linked to increased risks of poor cognitive development, reduced educational attainment, and decreased productivity in adulthood [5], [6]. Growing

evidence indicates that maternal nutritional status during pregnancy plays a pivotal role in determining fetal growth trajectories and subsequent child growth outcomes [7], [8].

Recent state-of-the-art studies emphasize the life-course and intergenerational framework of stunting, particularly highlighting the first 1,000 days of life as a critical window for effective intervention [9]. Maternal CED has been shown to disrupt placental nutrient transport, alter fetal metabolic programming, and increase susceptibility to LBW, which is a well-established predictor of childhood stunting [10]–[12]. Advances in antenatal care delivery, nutritional surveillance, and community-based maternal nutrition programs have improved early detection of nutritional risk; however, their effectiveness varies considerably across geographical and socioeconomic contexts [13], [14].

Despite the growing body of evidence linking maternal CED and stunting, most studies rely on national or large-scale datasets, which may mask important contextual variations at the local health system level. Several studies have demonstrated a strong association between maternal undernutrition and stunting [15], [16], while others report that adequate postnatal nutrition, breastfeeding practices,

and health service utilization may attenuate these effects [17], [18]. These inconsistencies underscore a critical research gap regarding how maternal CED influences stunting within specific local contexts, particularly in rural settings with integrated maternal and child health interventions.

In Indonesia, stunting prevention strategies increasingly emphasize multisectoral and community-based approaches, including the integration of nutrition services within primary healthcare systems [19], [20]. Programs involving collaboration among midwives, nutritionists, and community health workers aim to strengthen early detection and management of maternal undernutrition. However, empirical evidence evaluating the effectiveness of these approaches at the sub-district or health-center level remains limited, especially in rural areas where socioeconomic vulnerabilities persist [21], [22].

Therefore, this study aims to analyze the effect of Chronic Energy Deficiency during pregnancy on the incidence of stunting among children aged 24–59 months in the working area of Ngariboyo Public Health Center, Magetan Regency. By employing a case-control design and utilizing routine maternal and child health records, this study seeks to generate localized evidence that complements national findings and supports more targeted and context-sensitive stunting prevention strategies.

This study contributes to the existing literature in three key ways. First, it provides empirical evidence on the association between maternal CED and childhood stunting within a rural primary healthcare context. Second, it reinforces the importance of early antenatal nutritional screening as an upstream intervention to prevent stunting. Third, it offers policy-relevant insights to optimize community-based maternal nutrition programs aimed at reducing stunting prevalence [23]–[25].

The remainder of this article is structured as follows. Section II describes the research methodology, including study design, population, data collection procedures, and statistical analysis. Section III presents the study results, followed by a comprehensive discussion in Section IV. Finally, Section V concludes the article and outlines implications for future research and public health policy.

II. METHODS

This study employed an analytical observational design with a case-control approach to examine the effect of maternal Chronic Energy Deficiency (CED) during pregnancy on the incidence of stunting among children aged 24–59 months. A case-control design was selected due to its efficiency in identifying associations between prior maternal exposure and current child health outcomes, particularly for conditions with relatively low prevalence and long latency periods such as stunting [26], [27]. The study was retrospective in nature, as exposure and outcome data were obtained from existing maternal and child health records rather than through prospective follow-up. No experimental intervention was applied, and the study population was not randomized, as participants were selected based on predefined eligibility criteria.

A. STUDY SETTING AND POPULATION

The study was conducted in the working area of Ngariboyo Public Health Center, Magetan Regency, East Java, Indonesia. This area was selected due to its increasing prevalence of maternal CED and ongoing implementation of community-based stunting prevention programs. The study population consisted of all children aged 24–59 months who were registered in the e-PPBGM (Electronic Community-Based Nutrition Recording and Reporting) system and resided permanently in the study area during the data collection period. The target population also included mothers who possessed complete Maternal and Child Health (KIA) handbooks documenting antenatal care and nutritional status during pregnancy.

B. SAMPLE SIZE AND SAMPLING TECHNIQUE

The sample size was determined using a 1:2 case-control ratio to enhance statistical power while maintaining feasibility [28]. A total of 96 children were included, comprising 32 stunted children as the case group and 64 non-stunted children as the control group. Cases were defined as children aged 24–59 months with a height-for-age Z-score (HAZ) below -2 standard deviations according to World Health Organization (WHO) growth standards. Controls were children within the same age range with normal linear growth ($\text{HAZ} \geq -2 \text{ SD}$). The case group was selected using simple random sampling from the list of stunted children recorded in the e-PPBGM database, while the control group was selected using systematic random sampling from non-stunted children to minimize selection bias [29].

C. INCLUSION AND EXCLUSION CRITERIA

Inclusion criteria were children aged 24–59 months residing in the study area, whose mothers had complete-antenatal records in the KIA handbook. Exclusion criteria included children with congenital anomalies, genetic disorders, or physical disabilities that could independently affect linear growth, as well as children with incomplete anthropometric or maternal nutritional data. These criteria were applied to reduce confounding and ensure data reliability [30].

D. STUDY VARIABLES AND OPERATIONAL DEFINITIONS

The independent variable was maternal Chronic Energy Deficiency during pregnancy, operationalized using Mid-Upper Arm Circumference (MUAC) measurements recorded during the first trimester of pregnancy. Mothers with $\text{MUAC} < 23.5 \text{ cm}$ were classified as experiencing CED, in accordance with WHO and international maternal nutrition guidelines [31]. The dependent variable was childhood stunting, defined based on height-for-age Z-scores calculated using WHO growth standards. Covariates descriptively assessed included maternal age, parity, pregnancy interval, education level, family income, birth weight, breastfeeding practices, history of infectious diseases, and nutritional intake.

E. DATA COLLECTION PROCEDURES

Data collection was conducted between February and March 2025. Secondary data on maternal CED status were extracted

from KIA handbooks, while child growth and stunting status were obtained from the e-PPBGM system and verified through structured questionnaires administered to mothers. Anthropometric measurements recorded in the system were collected by trained health workers using standardized equipment and procedures. All data were cross-checked to ensure consistency and completeness prior to analysis [32].

F. STATISTICAL ANALYSIS

Data were processed and analyzed using statistical software. Univariate analysis was performed to describe the distribution of maternal and child characteristics. Bivariate analysis was conducted using the Chi-square test to assess the association between maternal CED and stunting incidence. The magnitude of risk was estimated using the Mantel-Haenszel common odds ratio (OR) with a 95% confidence interval. A p-value of less than 0.05 was considered statistically significant. This analytical approach is widely recommended for categorical data analysis in case-control studies [33], [34].

G. ETHICAL CONSIDERATIONS

Ethical approval for this study was obtained from the institutional ethics committee prior to data collection. Written informed consent was obtained from all participating mothers. Confidentiality and anonymity of respondents were strictly maintained, and all data were used exclusively for research purposes in accordance with international ethical standards for observational health research [35].

III. RESULTS

A. Maternal Characteristic During Pregnancy

TABLE 1
Frequency Distribution of Maternal Characteristics in the Working Area of Ngariboyo Public Health Center, 2025

| Variable | Group | | | | | |
|---------------------------|--------------|-------|----------|-------|-----|------|
| | Not stunting | | Stunting | | Sum | |
| | f | % | f | % | f | % |
| Age | | | | | | |
| Not risk | 55 | 70,5% | 23 | 29,5% | 78 | 100% |
| High Risk | 9 | 50% | 9 | 50% | 18 | 100% |
| Pregnancy Interval | | | | | | |
| > 2 tahun | 58 | 69% | 26 | 31% | 84 | 100% |
| < 2 tahun | 6 | 50% | 6 | 50% | 12 | 100% |
| Parity | | | | | | |
| Primipara | 29 | 76,3% | 9 | 23,7% | 38 | 100% |
| Multipara | 35 | 60,3% | 23 | 39,7% | 58 | 100% |
| Grandmultipara | 0 | 0% | 0 | 0% | 0 | 0% |
| Education | | | | | | |
| Basic | 2 | 100% | 0 | 0% | 2 | 100% |
| Intermediate | 53 | 65,4% | 28 | 34,6% | 81 | 100% |
| Higher | 9 | 69,2% | 4 | 30,8% | 13 | 100% |
| Income | | | | | | |
| Low | 12 | 46,1% | 14 | 53,9% | 26 | 100% |
| Middle | 45 | 76,3% | 14 | 23,7% | 59 | 100% |
| High | 7 | 63,6% | 4 | 36,4% | 11 | 100% |

Based on TABLE 1, the characteristics of mothers at Ngariboyo Public Health Center show that among non-stunted children, half of the mothers had pregnancies at a high-risk age, half had a birth interval of less than two years, the majority were multiparous, and nearly half had low family income. Meanwhile, among stunted children, half of the mothers also had pregnancies at a high-risk age, half had

a birth interval of less than two years, nearly half were multiparous, nearly half had a secondary level of education, and the majority had low family income.

B. Risk Factor Of Stunting

TABLE 2
Frequency Distribution of Stunting Risk Factors in the Ngariboyo Health Center Area in 2025

| Variable | Group | | | | | |
|-----------------------------------|--------------|-------|----------|-------|-----|------|
| | Not stunting | | Stunting | | Sum | |
| | f | % | f | % | f | % |
| Low Birth Weight (LBW) | | | | | | |
| Not LBW | 60 | 70,6% | 25 | 29,4% | 85 | 100% |
| LBW | 4 | 36,4% | 7 | 63,6% | 11 | 100% |
| Exclusive Breastfeeding | | | | | | |
| Exclusive breastfeeding | 54 | 72% | 21 | 38% | 75 | 100% |
| Not Exclusive breastfeeding | 10 | 47,6% | 11 | 52,3% | 21 | 100% |
| Complementary Feeding | | | | | | |
| Given Complementary feeding | 64 | 66,7% | 32 | 33,3% | 96 | 100% |
| Not given complementary feeding | 0 | 0% | 0 | 0% | 0 | 0% |
| Infection | | | | | | |
| No Infection | 62 | 70,5% | 26 | 29,5% | 88 | 100% |
| Infection | 2 | 25% | 6 | 75% | 8 | 100% |
| Health Services | | | | | | |
| Good | 64 | 66,7% | 32 | 33,3% | 96 | 100% |
| Poor | 0 | 0% | 0 | 0% | 0 | 0% |
| Sanitation and clean water | | | | | | |
| Good | 64 | 66,7% | 32 | 33,3% | 96 | 100% |
| Poor | 0 | 0% | 0 | 0% | 0 | 0% |
| Nutritional intake | | | | | | |
| Good | 56 | 98,2% | 1 | 0,6% | 57 | 100% |
| Adequate | 8 | 24,2% | 25 | 75,8% | 33 | 100% |
| Poor | 0 | 0% | 6 | 100% | 6 | 100% |

TABLE 3
Proportion of Stunting Cases from Exposure to Maternal Chronic Energy Deficiency

| Variable | Stunting | |
|--------------|----------|-------|
| | F | % |
| CED | 11 | 34,4% |
| Not CED | 21 | 65,6% |
| Total | 32 | 100% |

Based on TABLE 2, the risk factors for stunting in the working area of Ngariboyo Health Center show that among non-stunted toddlers, nearly half were born with low birth weight (LBW), nearly half did not receive exclusive breastfeeding, most were given complementary feeding (MPASI), a small portion experienced infections, most received good health services, most had access to adequate sanitation and clean water, and a small portion had adequate nutritional intake. In contrast, among stunted toddlers, the majority were born with LBW, the majority did not receive exclusive breastfeeding, nearly half were given complementary feeding, the majority experienced infections, nearly half received good health services, nearly half had

access to adequate sanitation and clean water, and most had adequate nutritional intake.

C. Proportion of Stunting Cases from Exposure to Maternal Chronic Energy Deficiency (CED) During Pregnancy

TABLE 3 indicates that almost half of the stunted children were born to mothers who experienced chronic energy deficiency during pregnancy, whereas a larger proportion of stunted children were observed among those whose mothers did not have a history of chronic energy deficiency.

D. Proportion of Non-Stunting Cases from Exposure to Maternal Chronic Energy Deficiency (CED) During Pregnancy

TABLE 1

Proportion of Non-Stunting Cases from Exposure to Maternal Chronic Energy Deficiency

| Variable | Not Stunting | |
|----------|--------------|-------|
| | F | % |
| CED | 9 | 14,1% |
| Not CED | 55 | 85,9% |
| Total | 64 | 100% |

TABLE 4 demonstrates that only a small proportion of non-stunted children were born to mothers who experienced chronic energy deficiency during pregnancy, whereas the vast majority of non-stunted children were born to mothers without a history of chronic energy deficiency.

E. Analysis of the Effect of Chronic Energy Deficiency (CED) on the Incidence of Stunting

TABLE 2

Analysis of the Effect of Chronic Energy Deficiency (CED) on the Incidence of Stunting

| Variable | Group | | | | Sum | | p-value | OR |
|----------|----------|-------|--------------|-------|-----|------|---------|-------|
| | Stunting | | Not Stunting | | | | | |
| | f | % | f | % | f | % | | |
| CED | 11 | 55% | 9 | 45% | 20 | 100% | 0,021 | 3,201 |
| Not CED | 21 | 27,6% | 55 | 72,4% | 76 | 100% | | |

From the TABLE 5, the statistical test results on the effect of chronic energy deficiency (CED) on the incidence of stunting showed a p-value of 0.021 ($p < 0.05$). It can be concluded that there is a significant effect between CED and the occurrence of stunting. The odds ratio (OR) was 3.201, which means that mothers with CED have a 3.2 times higher chance of giving birth to stunted children compared to mothers without CED.

IV. DISCUSSION

A. Maternal of Maternal Characteristics and Stunting Risk Factors

The findings of this study indicate that several maternal and child characteristics are closely associated with the occurrence of stunting, reinforcing the multifactorial nature of growth faltering in early childhood. Maternal age, pregnancy interval, parity, educational attainment, and

household income emerged as important contextual variables influencing both maternal nutritional status and child growth outcomes. Mothers who experienced pregnancy at high-risk ages (<19 years or >35 years) demonstrated a greater likelihood of having stunted children, which may be attributed to increased nutritional demands or declining physiological capacity to support optimal fetal growth. This finding supports the biological plausibility that maternal age influences nutritional reserves and placental efficiency, thereby affecting fetal development.

Short pregnancy intervals were also frequently observed among mothers of stunted children. Insufficient spacing between pregnancies limits maternal nutritional recovery and increases the risk of chronic energy deficiency during subsequent pregnancies. From a biological perspective, inadequate replenishment of maternal energy stores compromises fetal nutrient supply, which may predispose children to impaired linear growth. Similarly, higher parity was associated with stunting, likely reflecting cumulative nutritional depletion and reduced maternal attention to nutritional intake during pregnancy. These results underscore the importance of family planning and adequate birth spacing as indirect strategies for stunting prevention.

Socioeconomic factors, particularly maternal education and household income, played a substantial role in shaping stunting risk. Mothers with lower educational attainment may have limited access to nutrition-related information and reduced capacity to adopt appropriate health-seeking behaviors. Low household income further constrains access to diverse and nutrient-dense foods, exacerbating the risk of both maternal undernutrition and childhood stunting. Collectively, these findings highlight that stunting cannot be attributed solely to biological determinants but is strongly embedded within broader social and economic contexts.

Regarding child-related risk factors, this study identified low birth weight (LBW), non-exclusive breastfeeding, history of infection, and suboptimal nutritional intake as important contributors to stunting. LBW emerged as a dominant predictor, reflecting the long-term consequences of intrauterine growth restriction. Children born with LBW often experience impaired catch-up growth, especially in environments characterized by inadequate nutrition and recurrent infections. The lack of exclusive breastfeeding further increases vulnerability to infections and nutrient deficiencies during the critical first six months of life, thereby amplifying growth failure.

B. Comparison with Previous Studies and Explanation of Similarities and Differences

The association between maternal Chronic Energy Deficiency and childhood stunting observed in this study is consistent with findings from recent international literature. Several studies have reported that maternal undernutrition during pregnancy significantly increases the likelihood of stunting in children, with odds ratios ranging from two to four times higher compared to children born to well-nourished mothers [36], [37]. These findings align with the current study's odds ratio of 3.201, suggesting that maternal

CED is a strong upstream determinant of impaired linear growth.

The biological mechanisms underlying this relationship have been well documented. Maternal CED disrupts placental nutrient transfer, reduces fetal energy availability, and alters metabolic programming, all of which contribute to restricted fetal growth and increased risk of LBW [38]. Subsequent exposure to suboptimal postnatal environments further compounds growth deficits, leading to stunting. Similar conclusions were reported by longitudinal and case-control studies conducted in low- and middle-income countries, emphasizing the intergenerational transmission of malnutrition [39], [40].

However, some studies have reported weaker or non-significant associations between maternal CED and stunting, particularly in settings with strong postnatal nutrition programs and high coverage of maternal health services [41]. These discrepancies may be explained by differences in study design, timing of nutritional assessment, and contextual factors such as dietary diversity, breastfeeding practices, and access to healthcare. In the present study, a small proportion of non-stunted children were born to mothers with CED, suggesting that adequate postnatal nutrition and healthcare utilization may partially mitigate the adverse effects of maternal undernutrition.

Comparisons with studies focusing on breastfeeding and infection further support the present findings. Research consistently demonstrates that exclusive breastfeeding and effective infection control play protective roles against stunting [42]. Infections increase metabolic demands and impair nutrient absorption, thereby exacerbating growth faltering in already vulnerable children. The high proportion of infections among stunted children in this study reinforces the synergistic relationship between undernutrition and infectious disease.

Differences in sanitation and health service utilization across studies may also explain variations in stunting prevalence. While some studies identify sanitation as a key determinant of stunting, others report no significant association when access to clean water and health services is uniformly high [43]. In the present study, relatively good access to sanitation and healthcare may have reduced their influence as dominant risk factors, shifting the emphasis toward maternal nutrition and early-life feeding practices.

C. Limitations, Strengths, and Public Health Implications

Several limitations of this study should be acknowledged. First, the retrospective case-control design limits causal inference, as exposure and outcome data were collected from existing records rather than through prospective follow-up. Second, reliance on secondary data from maternal and child health handbooks and the e-PPBGM system may introduce measurement bias due to incomplete or inaccurately recorded information. Third, the absence of multivariate analysis restricts the ability to control for potential confounding variables such as maternal education, household income, and environmental conditions.

Additionally, the relatively small sample size may limit the generalizability of the findings beyond the study area. Recall bias related to breastfeeding practices and infection history may also affect data accuracy. Despite these limitations, the study has several notable strengths. The use of standardized WHO growth references enhances measurement validity, while the integration of multiple data sources improves data reliability. Furthermore, focusing on a localized primary healthcare setting provides context-specific insights that are often overlooked in large-scale studies.

The findings of this study have important public health implications. Maternal CED emerged as a critical intervention point for stunting prevention, underscoring the need for routine nutritional screening during antenatal care. Early identification of CED using simple tools such as MUAC measurement can facilitate timely nutritional interventions, including supplementary feeding and targeted counseling. Strengthening maternal nutrition programs within the first 1,000 days framework may yield substantial reductions in stunting prevalence.

At the policy level, integrating maternal nutrition surveillance into existing community-based programs, such as family assistance and primary healthcare services, offers a cost-effective strategy for addressing stunting. Health education initiatives should emphasize the importance of adequate maternal nutrition, optimal birth spacing, exclusive breastfeeding, and infection prevention. Future research should employ prospective cohort designs and multivariate analyses to clarify causal pathways and evaluate the long-term effectiveness of maternal nutrition interventions. Overall, this study reinforces the central role of maternal nutritional status in shaping child growth outcomes and highlights the necessity of addressing maternal undernutrition as a core component of stunting prevention strategies.

V. CONCLUSION

This study was conducted to analyze the effect of maternal Chronic Energy Deficiency (CED) during pregnancy on the incidence of stunting among children aged 24–59 months in the working area of Ngariboyo Public Health Center, Magetan Regency. The findings demonstrate a statistically significant association between maternal CED and childhood stunting, with a p-value of 0.021 and an odds ratio of 3.201, indicating that children born to mothers with CED had more than three times higher risk of being stunted compared to those born to mothers without CED. In addition to maternal nutritional status, several contextual factors—including low birth weight, non-exclusive breastfeeding, history of infection, low maternal education, short pregnancy intervals, and low household income—were observed to coexist with stunting cases, underscoring the multifactorial nature of growth faltering in early childhood. The results affirm that maternal undernutrition represents a critical upstream determinant of stunting, operating through impaired fetal growth and increased vulnerability during the first 1,000 days of life. These findings emphasize the importance of early detection and management of CED during antenatal care as a strategic entry point for stunting prevention. Routine screening using mid-upper arm circumference

measurements, combined with targeted nutritional counseling and supplementation for at-risk pregnant women, may substantially reduce the likelihood of stunting. Nevertheless, this study is limited by its retrospective case-control design, relatively small sample size, and reliance on secondary data, which restrict causal inference and generalizability. Future research should employ prospective cohort designs with larger populations and multivariate analyses to control for potential confounding factors and to better elucidate causal pathways linking maternal nutrition and child growth outcomes. Further studies are also warranted to evaluate the effectiveness of integrated maternal nutrition interventions and postnatal feeding practices in mitigating the long-term impact of maternal CED. Overall, the evidence generated by this study supports the prioritization of maternal nutritional status within stunting prevention policies and highlights the need for comprehensive, context-specific strategies that address both prenatal and postnatal determinants of child growth.

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DATA AVAILABILITY

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

AUTHOR CONTRIBUTION

All authors contributed substantially to this study. N.K.Q. conceptualized the research design, conducted data collection, and prepared the initial manuscript draft. A.S. supervised the study, provided methodological guidance, and critically revised the manuscript for intellectual content. N.S. contributed to data analysis, interpretation of results, and manuscript refinement. S. provided overall academic supervision, reviewed the final manuscript, and approved the version to be published. All authors have read and agreed to the final manuscript.

DECLARATIONS

ETHICAL APPROVAL

Ethical approval for this study was obtained from the institutional ethics committee prior to data collection. The study protocol was reviewed to ensure compliance with ethical principles involving human participants, including respect for autonomy, confidentiality, and data protection. All procedures were conducted in accordance with the Declaration of

Helsinki and relevant national and institutional guidelines governing health research.

CONSENT FOR PUBLICATION PARTICIPANTS.

Informed consent for publication was obtained from all participants involved in this study.

COMPETING INTERESTS

The authors declare that they have no competing interests.

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