Sarcouncil Journal of Medical Sciences

ISSN(Online): 2945-3526

Volume- 05| Issue- 06| 2026

Research Article **Received:** 01-05-2026 | **Accepted:** 25-05-2026 | **Published:** 12-06-2026

Serum Calcium Levels as a Potential Biomarker in Pediatric Leukemia

Islam H. Tarrad

Medical Laboratory Techniques Department, Health And Medical Technique College, University of Hilla, Babylon, Iraq.

Abstract: This research evaluates serum calcium as a cost-effective, accessible biomarker for pediatric acute lymphoblastic leukemia (ALL). A study of 80 participants in Sudan revealed that ALL patients have significantly lower serum calcium levels than healthy controls. This hypocalcemia appears to be a consistent metabolic feature of the disease, independent of the patient's age or gender. Furthermore, calcium levels did not vary significantly across different FAB subtypes or chemotherapy cycles. Interestingly, serum calcium showed a strong correlation with disease duration, with lower levels found in the early stages of the illness. These early deficits may stem from increased tumor activity, systemic inflammation, and chemotherapy-induced metabolic disruptions. The findings emphasize that routine calcium monitoring is vital to prevent complications like decreased bone metabolism and cardiac issues. Ultimately, the study advocates for integrated metabolic monitoring and supportive care to improve clinical management in pediatric leukemia.

Keywords: Serum Calcium Levels, Acute leukemia, Pediatric Leukemia.

INTRODUCTION

Acute leukemia represents the most common malignancy in childhood, accounting for approximately 30% of all pediatric cancers, with acute lymphoblastic leukemia (ALL) comprising 70–80% of cases [Jahan, F. *et al*., 2025; Kim, N. T. T. *et al*., 2022]. Despite significant advances in risk stratification and treatment protocols that have improved 5-year survival rates to over 80% in developed countries [Campbell, M. *et al*., 2023; Gottschalk, H. *et al*., 2025], the identification of novel biomarkers remains critical for early diagnosis, risk assessment, and monitoring of treatment response. Current prognostic tools rely heavily on minimal residual disease (MRD) assessment, cytogenetic abnormalities, and immunophenotyping [Campbell, M. *et al*., 2023; Saghir, S. A. M. *et al*., 2023], yet these approaches require invasive bone marrow sampling and specialized laboratory infrastructure. The search for accessible, cost-effective biomarkers that can complement existing diagnostic modalities continues to drive pediatric oncology research.

The pathophysiology of pediatric leukemia involves complex interactions between malignant lymphoblasts and the bone marrow microenvironment, leading to disruption of normal hematopoiesis and metabolic homeostasis [Khalid, A. *et al*., 2023; Katsurayama, F. K. *et al*., 2025]. Leukemic infiltration of bone marrow triggers dysregulation of bone remodeling pathways, particularly the receptor activator of nuclear factor kappa-B ligand (RANKL)/osteoprotegerin (OPG) axis, resulting in uncoupled bone formation and resorption [Muggeo, P. *et al*., 2023; Atilano-Miguel, S. *et al*., 2024]. This pathological bone turnover can manifest as skeletal complications, including osteopenia, pathological fractures, and osteolytic lesions, which are increasingly recognized as significant sources of morbidity in pediatric leukemia patients [Muggeo, P. *et al*., 2023; Utriainen, P. *et al*., 2026]. Furthermore, certain genetic subtypes, such as TCF3::HLF fusion-positive ALL, demonstrate a particularly aggressive phenotype characterized by extensive bone destruction and metabolic derangements [Lin, W. *et al*., 2025]. These observations suggest that biochemical markers reflecting bone metabolism may provide valuable insights into disease biology and clinical outcomes.

Contemporary biomarker strategies in pediatric ALL encompass multiple domains, including genetic profiling, circulating molecular markers, and proteomic signatures [Saghir, S. A. M. *et al*., 2023; Kourti, M. *et al*., 2023; Álvarez-Zúñiga, C. D. *et al*., 2023]. MRD quantification by flow cytometry has become the gold standard for risk stratification, enabling treatment intensification or de-escalation based on early response [Campbell, M. *et al*., 2023]. Genetic aberrations such as ETV6-RUNX1, BCR-ABL1, and IKZF1 deletions inform prognosis and guide targeted therapy selection [Kim, N. T. T. *et al*., 2022; Lee, S. H. *et al*., 2024]. Emerging circulating biomarkers, including long non-coding RNAs such as LINC00958, show promise for non-invasive disease monitoring and relapse prediction [Altieri, F. *et al*., 2024]. Proteomic approaches have identified candidate biomarkers in peripheral blood and cerebrospinal fluid that may detect central nervous system involvement earlier than

***Corresponding Author:** Islam H. Tarrad

DOI- https://doi.org/10.5281/zenodo.20663520

conventional methods [Álvarez-Zúñiga, C. D. *et al*., 2023; 16]. Despite these advances, most novel biomarkers remain in the research phase, and there is a pressing need for simple, routinely measurable parameters that can be integrated into standard clinical practice.

A notable gap exists in the systematic evaluation of serum calcium as a potential biomarker in pediatric leukemia. While severe hypercalcemia has been documented as a rare but dramatic presenting feature of childhood ALL, often associated with life-threatening complications such as altered consciousness, cardiac arrhythmias, and renal dysfunction [Chen, M., Ni, J., & Lu, X. 2022; Bonilla Gonzalez, C. *et al*., 2022; Arenas Camacho, L. D. *et al*., 2024], the broader spectrum of calcium dysregulation in pediatric leukemia remains poorly characterized. Case reports have described hypercalcemia as the sole initial manifestation of ALL in children with normal peripheral blood counts and initially unremarkable bone marrow examinations, leading to diagnostic delays [Chen, M., Ni, J., & Lu, X. 2022; Bonilla Gonzalez, C. *et al*., 2022]. Conversely, recent cohort data suggest that hypocalcemia may also occur in pediatric ALL patients, with serum calcium levels inversely correlating with liver enzymes and lactate dehydrogenase [Jahan, F. *et al*., 2025]. These conflicting observations raise important questions about the prevalence, clinical significance, and mechanistic basis of calcium abnormalities across the spectrum of pediatric leukemia.

The potential role of serum calcium as a biomarker is supported by several lines of evidence. First, the pathophysiological link between leukemic bone marrow infiltration and dysregulated bone remodeling provides a mechanistic rationale for calcium dysregulation [Muggeo, P. *et al*., 2023; Atilano-Miguel, S. *et al*., 2024]. Studies have demonstrated elevated bone resorption markers, including C-terminal telopeptide of type I collagen (CTX) and tartrate-resistant acid phosphatase 5b (TRACP5b), in children with ALL following intensive chemotherapy, indicating ongoing calcium mobilization from bone [Muggeo, P. *et al*., 2023]. Second, the RANKL/OPG pathway, which is central to osteoclast activation and bone resorption, shows significant alterations from diagnosis through remission in pediatric leukemia patients [Atilano-Miguel, S. *et al*., 2024]. Third, vitamin D deficiency is highly prevalent in newly diagnosed ALL children (up to 91% in some cohorts), and the vitamin D-parathyroid hormone (PTH)-calcium axis demonstrates dynamic changes during treatment [Barbosa-Cortés, L. *et al*., 2025]. These findings suggest that serum calcium and related metabolic parameters may reflect underlying disease activity and treatment effects.

The clinical utility of serum calcium measurement lies in its simplicity, low cost, and widespread availability in routine laboratory panels. Unlike specialized genetic or proteomic assays, serum calcium can be measured in any clinical setting, making it an attractive candidate for resource-limited environments where pediatric leukemia burden is high but diagnostic infrastructure is limited [Jahan, F. *et al*., 2025; Kim, N. T. T. *et al*., 2022]. Furthermore, if validated as a prognostic marker, serum calcium could potentially identify patients at risk for skeletal complications who might benefit from early interventions such as bisphosphonate therapy or vitamin D supplementation [Chen, M., Ni, J., & Lu, X. 2022; Bonilla Gonzalez, C. *et al*., 2022]. However, the inconsistency of calcium abnormalities across reported cohorts and the predominance of single-case reports rather than systematic studies limit current understanding of its biomarker potential.

Given the paucity of comprehensive data on serum calcium patterns in pediatric leukemia and the need for accessible biomarkers in this population, this study aims to systematically evaluate serum calcium levels at diagnosis in children with acute leukemia, examine their correlation with established prognostic factors and disease characteristics, and assess their potential utility as a diagnostic and prognostic biomarker. By elucidating the prevalence and clinical associations of calcium dysregulation in a well-characterized pediatric leukemia cohort, this research seeks to determine whether serum calcium measurement can contribute to improved risk stratification and clinical decision-making in the management of childhood leukemia.

MATERIALS AND METHODS

This cross-sectional analytical study was conducted in Gezira State, Sudan, from January to June 2016. A total of 80 participants were included, comprising 50 pediatric patients diagnosed with acute lymphoblastic leukemia (ALL), aged 3–14 years and undergoing chemotherapy, and 30 age-matched healthy children as controls. Data were collected using a structured questionnaire administered at the time of sample collection. Approximately 2.5 mL of

Publisher: SARC Publisher

venous blood was collected aseptically from each participant into lithium heparin containers, centrifuged to obtain plasma, and stored at −20°C until analysis. Serum levels of calcium were measured using a Cobas e 411 analyzer. Data were analyzed using the Statistical Package for Social Sciences (IBM SPSS) version 20, applying independent t-tests, cross-tabulation, and correlation analysis. The dependent variables included calcium levels, while age, sex, disease duration, and treatment duration were considered independent variables. Pediatric ALL patients aged 1–14 years were included, while those with liver disease, renal disease, thyroid dysfunction, or mental retardation were excluded. Ethical approval was obtained, and informed consent was secured from participants or their guardians prior to data and sample collection.

RESULTS

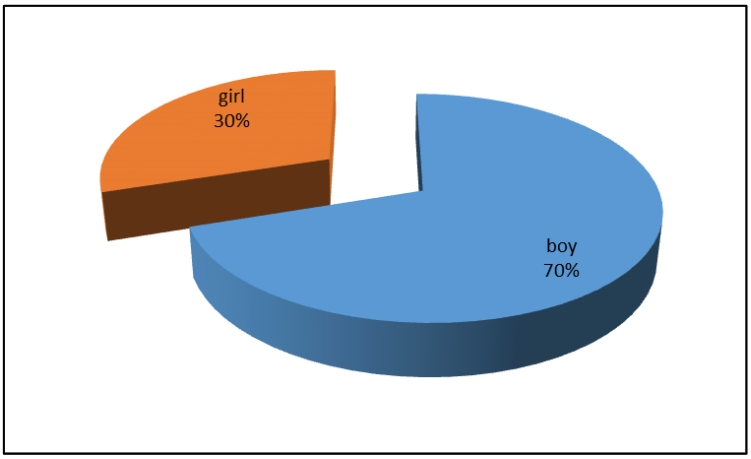

Figure 1: Distribution of study population according to sex

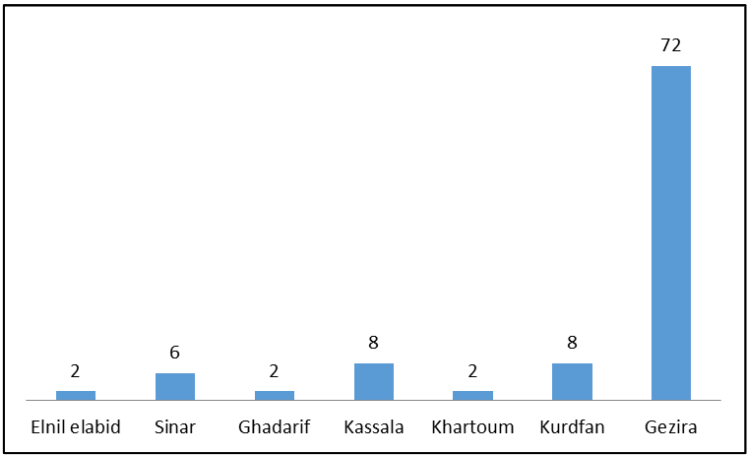

Figure 2: Distribution of study population according to State

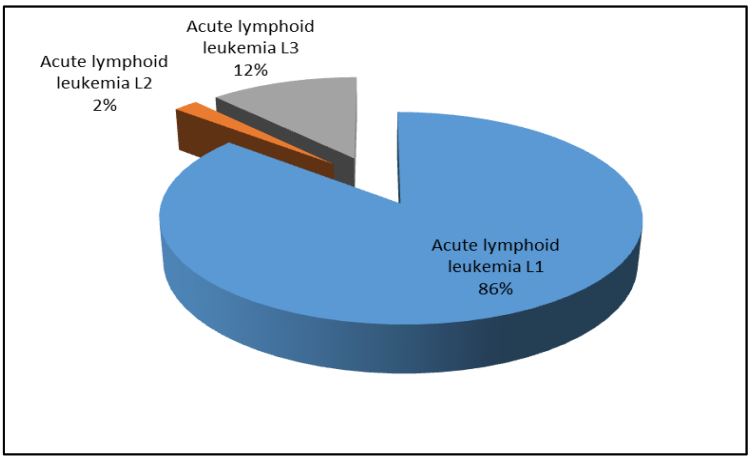

Figure 3: Frequency distribution of ALL types among study population

Publisher: SARC Publisher

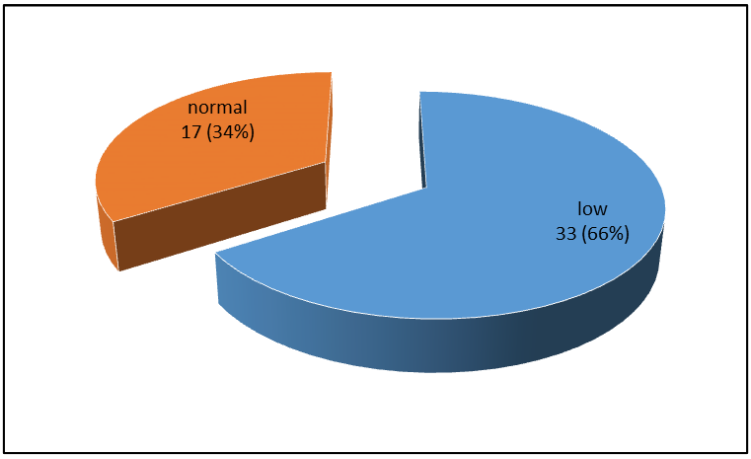

Figure 4: Distribution of serum calcium among cases

Table 1: The mean of serum calcium in cases and controls

Serum Calcium	Cases N=50	Control N= 30	P. Value
	Means± SD	Means± SD	
	8.5 ± 0.6	9.4 ± 0.6	0.000

Table 2: The mean of serum calcium in cases according to gender

Serum Calcium	Cases N=50	Control N= 30	P. Value
	Means± SD	Means± SD	
	8.5 ± 0.6	9.4 ± 0.6	0.000

Table 3: The mean of serum calcium in patients according to age groups

Calcium	Age	N	Mean	Std. Deviation	P. Value
	1-8 Year	25	8.492000	.5559676	.743
	9-16 Year	25	8.440000	.5590170	
	Total	50	8.466000	.5524011	

Table 4: The mean of serum calcium in cases according to the type of acute leukemia.

Calcium	Age	N	Mean	Std. Deviation	P. Value
	1-8 Year	25	8.492000	.5559676	.743
	9-16 Year	25	8.440000	.5590170	
	Total	50	8.466000	.5524011	

Table 5: The mean of serum calcium in cases according to the number of chemotherapy cycles

Calcium	Age	N	Mean	Std. Deviation	P. Value
	1-8 Year	25	8.492000	.5559676	.743
	9-16 Year	25	8.440000	.5590170	
	Total	50	8.466000	.5524011	

Table 6: The mean of serum calcium in cases according disease duration

Calcium	Duration	N	Mean	Std. Deviation	P. Value
	From 1 month to 1 year	15	8.173333	.5324695	.001
	From 2-4 year	28	8.475000	.5074446	
	From 5-7 year	7	9.057143	.2225395	
	Total	50	8.466000	.5524011	

The current study showed that boys were more likely than girls to have pediatric acute lymphoblastic leukemia (ALL), with the majority of cases coming from Gezira State. This finding probably reflects patterns of healthcare access and case discovery. The L1 subtype predominated in the FAB classification, which is consistent with normal pediatric appearances. One important discovery was that patients' serum calcium levels were significantly lower than those of controls, suggesting that hypocalcemia is a prevalent biochemical anomaly in ALL. Since there were no

Publisher: SARC Publisher

discernible differences between the two groups, this decline seemed to be unrelated to age or gender, indicating that calcium imbalance is more likely to be caused by illness than by demographics. Furthermore, there was no significant difference in blood calcium levels between the various ALL subtypes or based on the number of chemotherapy cycles, suggesting that neither treatment intensity nor illness classification had a significant effect on calcium levels. Patients in the early stages of the disease showed lower calcium levels than those in the later stages, which may indicate more severe metabolic abnormalities at the beginning of the disease with gradual stabilization over time. Overall, our results underline the significance of keeping an eye on calcium levels throughout the course of the illness and show that hypocalcemia is a constant feature in pediatric ALL.

DISCUSSION

This study examined serum calcium levels in pediatric patients in order to highlight significant biochemical alterations associated with acute lymphoblastic leukemia (ALL). Serum calcium levels were statistically substantially lower in cases than in controls, according to the results, suggesting that hypocalcemia is a significant metabolic anomaly in kids with ALL.

Males have higher incidence rates of ALL than females, according to established epidemiological trends, which are consistent with the observed male predominance (70%). This variance has been found to be caused by genetic, hormonal, and environmental factors that influence leukemogenesis [Siegel, D.A. *et al*., 2020; Terwilliger, T., & Abdul-Hay, M. J. B. C. J. 2017]. The majority of cases from Gezira State may indicate geographical variations in healthcare access, diagnostic facilities, or referral systems rather than true variability in illness frequency [Inaba, H. *et al*., 2023].

The FAB L1 subtype, which is the most common morphological variant in pediatric ALL and is usually associated with a favorable prognosis, was assigned to the majority of the children in this study. This is in line with global statistics that indicate the majority of childhood ALL cases are L1 [Hunger, S. P., & Mullighan, C. G. 2015; Pui, C. H. *et al*., 2019]. The significant drop in serum calcium levels among ALL patients is one of the study's key conclusions. This physiologically realistic result can be explained by a variety of ways. Malignancy-related metabolic changes, such as elevated cytokine activity and tumor load, may disturb calcium homeostasis. Furthermore, calcium absorption may be hampered by chemotherapy-induced gastrointestinal damage and altered vitamin D metabolism, and hypocalcemia may be made worse by nutritional deficits frequently seen in pediatric cancer patients [Belizário, J. E. *et al*., 2022; Howard, S. C. *et al*., 2021]. Together, these mechanisms highlight the complex nature of calcium imbalance in leukemia.

Crucially, there was no discernible variation in serum calcium levels based on age or gender, indicating that calcium dysregulation is mostly caused by disease rather than being impacted by demographic variables. This result is in line with earlier research showing that treatment effects and disease pathology, rather than patient characteristics, are the primary causes of electrolyte abnormalities in leukemia [Coiffier, B. *et al*., 2020]. Similarly, there were no statistically significant variations in calcium levels between the various FAB subtypes of ALL, indicating that calcium imbalance is not subtype-specific but rather a basic metabolic characteristic of leukemia. But in other subgroups, especially L2, the small sample size limits conclusive findings and calls for careful interpretation.

Additionally, there was no discernible correlation between the number of treatment cycles and serum calcium levels. This study implies that baseline illness features and supportive care parameters may be more important than treatment intensity alone, even though chemotherapy is known to affect metabolic and electrolyte balance [Kılıç, S.C. *et al*., 2020; Bhatia, S. *et al*., 2021].

On the other hand, a strong correlation between serum calcium levels and the length of the illness was found. Compared to patients in earlier stages, individuals with prolonged disease durations had comparatively greater calcium levels. This could be the result of better metabolic stability over time brought about by continued therapy, dietary assistance, and physiological adaptation. On the other hand, more severe hypocalcemia may result from increased tumor activity and systemic inflammation in the early stages of the illness [Ward, E. *et al*., 2020; Allemani, C. *et al*., 2023]. These results highlight the significance of routinely monitoring serum calcium levels in pediatric ALL patients from a clinical standpoint. Significant side effects, including as decreased bone metabolism, heart problems, and neuromuscular excitability, can result from

Publisher: SARC Publisher

hypocalcemia. Comprehensive leukemia care must include early detection and adequate treatments, including nutritional support and treatment of underlying causes.

CONCLUSION

This study concludes that, regardless of age, gender, or leukemia subtype, hypocalcemia is a substantial biochemical aberration in pediatric ALL patients and is linked to the length of the disease. These results emphasize the necessity of supportive care techniques and integrated metabolic monitoring in the treatment of pediatric leukemia.

REFERENCES

1. Jahan, F., Chakraborty, S., Parvin, M., Rahman, A. A., Howlader, Z. H., & Hosen, M. I. "Genetic polymorphisms in the chromatin modifier gene ARID5B in modulating the risk of pediatric acute lymphoblastic leukemia in Bangladesh." *Journal of Medicine, Surgery, and Public Health* 5 (2025): 100184.
2. Kim, N. T. T., Phan, V. H., Tran, K. H. "Clinical, laboratory features and some genetic abnormalities in childhood acute leukemia at Hue Central Hospital, Viet Nam." *Tạp chí Y Dược học.* 5.19 (2022).
3. Campbell, M., Kiss, C., Zimmermann, M., Riccheri, C., Kowalczyk, J., Felice, M. S., & Schrappe, M. "Childhood acute lymphoblastic leukemia: results of the randomized acute lymphoblastic leukemia intercontinental-Berlin-Frankfurt-Münster 2009 trial." *Journal of Clinical Oncology* 41.19 (2023): 3499-3511.
4. Gottschalk, H., Möricke, A., Conter, V., Schrappe, M., Starý, J., Cario, G., & Zimmermann, M. "Reducing Daunorubicin in Induction Therapy in Children With B-Lineage ALL With Favorable Prognosis: Results of Phase III Trial AIEOP-BFM ALL 2009." *Journal of Clinical Oncology* 43.35 (2025): 3739-3749.
5. Saghir, S. A. M. "A new insight updates in diagnosis and management of acute lymphoblastic leukemia, cytogenetics, immunophenotyping, and proteomic profile." *Electronic Journal of General Medicine* 20.5 (2023).
6. Khalid, A., Ahmed, M., & Hasnain, S. "Biochemical and Hematologic profiles in B-Cell acute lymphoblastic leukemia children." *Journal of Pediatric Hematology/Oncology* 45.7 (2023): e867-e872.
7. Katsurayama, F. K., de Mello, M. E. C. P., Van Santen, M. L. C., Schmickler, I. E. S. B., da Conceição, C. S., Lima, R. F., & Ribas Filho, S. "Leucemias Pediátricas: Perspectivas Clínicas, Marcadores Prognósticos e o Impacto do Diagnóstico na Criança e em sua Rede Familiar." *Brazilian Journal of Implantology and Health Sciences* 7.10 (2025): 1314-1335.
8. Muggeo, P., Grassi, M., D'Ascanio, V., Brescia, V., Fontana, A., Piacente, L., & Santoro, N. "Bone remodeling markers in children with acute lymphoblastic leukemia after intensive chemotherapy: the screenshot of a biochemical signature." *Cancers* 15.9 (2023): 2554.
9. Atilano-Miguel, S., Barbosa-Cortés, L., Ortiz-Muñiz, R., Maldonado-Hernández, J., Martin-Trejo, J. A., Rodríguez-Cruz, M., & Rodríguez-Palacios, M. E. "Changes in RANKL, OPG, and 25 (OH) D Levels in Children with Leukemia from Diagnosis to Remission." *Cancers* 16.16 (2024): 2811.
10. Utriainen, P., Hartmann, M. A., Blouin, S., Sofia, T., Mäkitie, O., & Fratzl-Zelman, N. "Low Bone Turnover and Decreased Bone Matrix Mineralization at Diagnosis in Children with Acute Lymphoblastic Leukemia." *Calcified Tissue International* 117.1 (2026): 53.
11. Lin, W., Zhang, Y. Y., Yu, J. J., Wu, Y., Qi, P. J., Fan, J., & Zhang, R. D. "Analysis of 8 children with TCF3:: HLF fusion gene positive acute lymphoblastic leukemia." *Zhonghua er ke za zhi= Chinese Journal of Pediatrics* 63.8 (2025): 896-900.
12. Kourti, M., Aivaliotis, M., & Hatzipantelis, E. "Proteomics in childhood acute lymphoblastic leukemia: challenges and opportunities." *Diagnostics* 13.17 (2023): 2748.
13. Álvarez-Zúñiga, C. D., Garza-Veloz, I., Martínez-Rendón, J., Ureño-Segura, M., Delgado-Enciso, I., & Martinez-Fierro, M. L. "Circulating biomarkers associated with the diagnosis and prognosis of B-cell progenitor acute lymphoblastic leukemia." *Cancers* 15.16 (2023): 4186.
14. Lee, S. H., Oh, B., Iyer, P., Chan, Y. H., Chiew, K. H., Kham, S. K. Y., & Yeoh, A. E. J. "Improved Outcomes for Pediatric B-ALL but Inferior Survival for T-ALL Using the Same Treatment Protocol: Results of Consecutive Malaysia-Singapore 2003 and

Publisher: SARC Publisher

2010 Childhood ALL Trials." *Blood* 144 (2024): 4192.
15. Altieri, F., Buono, L., Lanzilli, M., Mirabelli, P., Cianflone, A., Beneduce, G., & Smaldone, G. "LINC00958 as new diagnostic and prognostic biomarker of childhood acute lymphoblastic leukaemia of B cells." *Frontiers in Oncology* 14 (2024): 1388154.
16. Rangel-Cova, L. S., Soto-Álvarez, S., Estudillo, E., & Huerta-Núñez, L. F. "Potential protein biomarkers of central nervous system infiltration in childhood acute lymphoblastic leukemia." *Gaceta médica de México* 160.5 (2024): 489-497.
17. Chen, M., Ni, J., & Lu, X. "Hypercalcemia and osteolytic lesions as presenting symptoms of acute lymphoblastic leukemia in children: case report and literature review." *Frontiers in Pediatrics* 10 (2022): 923297.
18. Bonilla Gonzalez, C., Vargas Muñoz, S. M., Contreras Diaz, M. L., Obando Belalcazar, E., & Uribe, C. "Malignant hypercalcemia as the debut of acute lymphoblastic leukemia in a pediatric patient—a diagnostic and therapeutic approach: Case report." *Frontiers in Pediatrics* 10 (2022): 1027421.
19. Arenas Camacho, L. D., Peña Jiménez, J. C., Delgado Beltrán, A. M., Obregón Martínez, M. D. P., & Estupiñán Perico, D. I. "Debut de leucemia linfoblástica aguda infantil con hipercalcemia maligna, presentación inusual: reporte de caso." (2024).
20. Barbosa-Cortés, L., Atilano-Miguel, S., Manuel Cortés-Beltrán, V., Berenice Morales-Montes, S., Alfonso Martín-Trejo, J., Maldonado-Hernández, J., & Manuel Domínguez-Salgado, J. "Hipovitaminosis D y marcadores bioquímicos del metabolismo óseo en niños con leucemia." *Revista Medica del IMSS* 63 (2025).
21. Siegel, D.A., Henley, S. J., Li, J., Pollack, L. A., Van, Dyne E. A., White. A. "Trends in pediatric acute lymphoblastic leukemia incidence in the United States." *Cancer Epidemiol Biomarkers Prev*. 29.3 (2020): 593–602.
22. Terwilliger, T., & Abdul-Hay, M. J. B. C. J. "Acute lymphoblastic leukemia: a comprehensive review and 2017 update." *Blood cancer journal* 7.6 (2017): e577-e577.
23. Inaba, H., Greaves, M., Mullighan, C.G. "Acute lymphoblastic leukaemia." *Lancet*. 401.10374 (2023): 1371–1387.
24. Hunger, S. P., & Mullighan, C. G. "Acute lymphoblastic leukemia in children." *New England Journal of Medicine* 373.16 (2015): 1541-1552.
25. Pui, C. H., Nichols, K. E., & Yang, J. J. "Somatic and germline genomics in paediatric acute lymphoblastic leukaemia." *Nature reviews Clinical oncology* 16.4 (2019): 227-240.
26. Belizário, J. E., Napolitano, M. "Mechanisms of metabolic alterations in cancer patients." *Rev Assoc Med Bras*. 68.1 (2022): 1–8.
27. Howard SC, Jones DP, Pui CH. Tumor lysis syndrome in pediatric malignancies. N Engl J Med. 2021;384(19):1844–1855.
28. Coiffier, B., Altman, A., Pui, C.H., Younes, A., Cairo, M.S. "Management of metabolic complications in hematologic malignancies." *Lancet Oncol*. 21.4 (2020): e146–e158.
29. Kılıç, S.C., Karabulut, H. G., Yılmaz, D., *et al*. "Electrolyte imbalance in pediatric leukemia patients." *J Pediatr Hematol Oncol*. 42.6 (2020): e456–e461.
30. Bhatia, S., Landier, W. "Long-term outcomes and late effects in pediatric cancer survivors." *Nat Rev Clin Oncol.* 18.11 (2021): 685–698.
31. Ward, E., DeSantis, C., Robbins, A., Kohler, B., Jemal, A. "Childhood and adolescent cancer statistics." *CA Cancer J Clin*. 70.6 (2020): 443–459.
32. Allemani, C., Matsuda, T., Di, C. V., *et al*. "Global trends in cancer survival." *Lancet*. 401.10374 (2023): 102–115.

Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:
Tarrad, I. H. "Serum Calcium Levels as a Potential Biomarker in Pediatric Leukemia." *Sarcouncil journal of Medical sciences* 5.6 (2026): pp 8-14.

Publisher: SARC Publisher