

Longitudinal Assessment of Body Mass Index Changes During the First Six Months of Treatment in Pediatric Acute Lymphocytic Leukemia

Masomeh Sabonchi¹, Mehran Noroozi^{1*}

¹Department of Pediatrics, Faculty of Medicine, Urmia University of Medical Sciences, Urmia, Iran

ARTICLE INFO

Article History:

Received: November 10, 2025

Revised: December 14, 2025

Accepted: December 23, 2025

ePublished: December 31, 2025

*Correspondence to

Mehran Noroozi,
Email: mehr_noroozi@yahoo.com

Abstract

Introduction: Acute Lymphocytic Leukemia (ALL) therapy, particularly the intensive use of glucocorticoids, is strongly associated with adverse nutritional outcomes. This study aimed to investigate the changes in nutritional status, assessed by body mass index (BMI) and weight-for-height (WFH), during the first six months of treatment in children with ALL and to identify associated patient characteristics.

Methods: A retrospective cohort study enrolled 325 pediatric ALL patients. Nutritional status was determined at diagnosis and after six months of standard therapy, using WFH for patients <2 years (n=83) and BMI for those ≥2 years (n=242). Baseline demographics, initial white blood cell (WBC) count, CNS involvement, and corticosteroid type were analyzed using the Chi-square test for association with post-treatment nutritional status.

Results: The cohort included 54.5% female patients. A rapid shift toward overnutrition was observed across all age groups. In the ≥2-year-old cohort, the combined prevalence of overweight and obesity increased from 7.5% at diagnosis to 18.6 % at six months, while the underweight prevalence simultaneously decreased from 64.5% to 45.9%. Bivariate analysis revealed two significant risk factors for developing overweight/obesity: female gender ($P=0.002$) and a high baseline WBC count $\geq 10/uL$; ($P=0.006$). No significant association was found between nutritional status and CNS involvement ($P=0.486$) or the specific corticosteroid agent used ($P=0.632$).

Conclusion: The initial 6-month period of ALL treatment is a critical window for the rapid development of overnutrition, driven primarily by the class of glucocorticoids used. Female gender and a high baseline WBC count are crucial, easily identifiable metabolic risk markers that necessitate the implementation of early, targeted nutritional intervention strategies beginning at diagnosis.

Keywords: Body mass index, Children, Acute lymphocytic leukemia, Chemotherapy

Please cite this article as follows: Sabonchi M, Noroozi M. Longitudinal assessment of body mass index changes during the first six months of treatment in pediatric acute lymphocytic leukemia. Int J Basic Sci Med. 2025;10(4):181-188. doi:10.34172/ijbsm.50737.

Introduction

Acute lymphoblastic leukemia (ALL) is the most common pediatric malignancy, accounting for approximately 25% of all childhood cancers worldwide. ¹ Advances in chemotherapy protocols have significantly improved survival rates, with current cure rates exceeding 85% in high-income countries. ² While treatment efficacy has dramatically improved, the intensive, multi-phase chemotherapy regimens are associated with significant long-term and late effects that substantially impact the quality of life and overall health of survivors. ³ Among the most concerning of these effects is the development of adverse changes in body composition, primarily manifested as excessive weight gain and subsequent risk of developing obesity. ⁴ While advancements in treatment

have significantly improved survival rates, they have also led to a greater recognition of associated long-term complications, particularly alterations in nutritional status and body composition. ⁵

Body mass index (BMI) is a widely used indicator of nutritional status and has emerged as a critical parameter in pediatric oncology. ⁶ Several studies have documented substantial changes in BMI during ALL treatment, particularly in the early phases of therapy. ⁷ Chemotherapy agents, combined with corticosteroids such as dexamethasone and prednisone, can induce hyperphagia, fluid retention, and reduced physical activity, leading to rapid weight gain. ⁸ Evidence suggests that the prevalence of overweight and obesity among children with ALL increases markedly during induction and



© 2025 The Author(s); Published by Zabol University of Medical Science. This is an open-access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

consolidation phases, with some cohorts reporting a rise from 7–13% at diagnosis to over 20–40% within the first six months of treatment.^{9, 10} These changes are clinically significant because excess weight during treatment has been associated with adverse outcomes, including increased risk of relapse, treatment-related toxicity, and poorer overall survival.¹¹ Moreover, early weight gain may persist into survivorship, contributing to long-term metabolic complications such as insulin resistance, dyslipidemia, and cardiovascular disease.¹² Despite these concerns, data on BMI trajectories during the initial six months of ALL therapy remain limited, particularly in diverse populations.¹³

Understanding BMI changes during this critical treatment window is essential for developing timely interventions to mitigate obesity-related risks.¹⁴ Therefore, this study aims to investigate the pattern and magnitude of BMI changes in children with ALL during the first six months of chemotherapy, providing insights into potential targets for nutritional and lifestyle interventions.

Material and Methods

Study Design

This study employed a descriptive-analytical, retrospective design, utilizing data collected from patient files over a 13-year period. The study protocol was reviewed and granted ethical approval by the Independent Ethics Committee of Urmia University of Medical Sciences (IR.UMSU.REC.1403.334). The research was conducted in full compliance with the ethical principles of the Declaration of Helsinki, ensuring all patient data were handled confidentially and anonymized prior to analysis.

Study Population

The target population for this research was children diagnosed with ALL. The study cohort consisted of all children under 18 years of age who were newly diagnosed with ALL and referred to the Pediatric Oncology Ward at Shahid Motahari Hospital in Urmia, Iran, from the beginning of the Persian calendar year 1390 (March 2011) through the end of 1402 (March 2024). A census approach (complete enumeration) was utilized, meaning all eligible patients diagnosed during this period were included.

Inclusion Criteria

- Children under 18 years of age diagnosed with ALL
- Confirmed diagnosis of ALL

Exclusion Criteria

- Patients who died within the first six months of initiating chemotherapy treatment, as insufficient data points would preclude longitudinal analysis

Treatment Protocol

All patients included in the study were treated according

to the [modified Berlin-Frankfurt-Münster (BFM)] ALL protocol. This standard regimen consisted of induction and consolidation phases. The primary focus of the BMI analysis was on the high-dose corticosteroid components administered during these initial phases. Specifically, in accordance with the BFM guidelines, all patients received oral prednisolone (60 mg/m²/day) and oral dexamethasone (10 mg/m²/day) as part of their initial therapy.

Data Collection

Patient clinical and demographic data were retrieved retrospectively from medical records and recorded onto a structured, pre-designed data collection form.

- Age at diagnosis (in years)
- Gender
- White blood cell (WBC) count at diagnosis
- Central nervous system (CNS) involvement status
- Body Mass Index (BMI) at diagnosis and after six months of treatment
- Treatment regimen details

Baseline Prognostic Variables

- Age at Diagnosis: Continuous variable, measured in years.
- Sex: Categorical variable (Male/Female).
- Initial White Blood Cell (WBC) Count: Categorical variable, classified into four ordinal groups based on established prognostic risk: <4,000/uL, 4,000-10,000/uL, 10,000-50,000/uL, and > 50,000/uL.
- Central Nervous System (CNS) Involvement: Categorical variable (Present/Absent) at the time of diagnosis.

Outcome Variables

- BMI at Diagnosis (Baseline)
- BMI at Six Months Post-Treatment

BMI Calculation

For children under two years of age, weight-for-height ratio was used instead of BMI. BMI categories were defined according to CDC guidelines for children aged 2–19 years:

- Underweight: BMI < 5th percentile
- Normal weight: 5th ≤ BMI < 85th percentile
- Overweight: 85th ≤ BMI < 95th percentile
- Obese: BMI ≥ 95th percentile

Statistical Analysis

All collected data were coded and entered into the Statistical Package for the Social Sciences (SPSS) software, version 26. Descriptive statistics (e.g., means, standard deviations, frequencies) were used to characterize the study population and the distribution of BMI. The association between categorical variables was evaluated

using either the Chi-squared test or, where appropriate, Fisher’s exact test. Differences in means were assessed using the independent samples t-test. Pearson’s correlation coefficient was also calculated to examine linear relationships between continuous variables as needed. Statistical significance for all analyses was established at a two-sided *P*-value of less than 0.05.

Results

Study Population Demographics

A total of 325 patients diagnosed with ALL met the inclusion criteria and were enrolled in the study. The cohort was slightly predominantly female, comprising 177 patients (54.5%), compared to 148 male patients (45.5%). The age distribution at diagnosis was heavily skewed towards the school-age category (2 to 10 years), which accounted for the majority of the population with 211 patients (64.9%). Patients under 2 years of age represented a substantial minority with 83 individuals (25.5%), while patients over 10 years were the smallest group, comprising 31 individuals (9.5%) (Table 1).

Changes in Nutritional Status Following Six Months of Treatment

Nutritional status was assessed at the time of diagnosis and after 6 months of specific treatment. Different indices were used based on age: Weight-for-Height (WFH) status for patients under 2 years (n=83) and BMI for patients aged 2 years and older (n=242) (Table 2). Nutritional status was assessed at diagnosis and after six months of ALL treatment, utilizing WFH for patients under 2 years of age (n=83) and BMI status for patients aged 2 years and older (n=242) (Table 2).

Patients Under Two Years of Age

In the cohort of 83 patients under 2 years of age, a significant increase was observed in the prevalence of overweight and obesity (based on WFH status) following 6 months of treatment. The frequency of overweight status doubled from 6.0% (n=5) at diagnosis to 12.0% (n=10) post-treatment. Similarly, the prevalence of obesity demonstrated a substantial rise, increasing nearly eight-fold from 1.2% (n=1) at diagnosis to 9.6% (n=8)

after 6 months. Concurrently, the proportion of patients classified as underweight decreased markedly, from 50.6% to 32.6% (Table 2).

Patients Over Two Years of Age

Among the 242 patients aged 2 years and older, a comparable trend in BMI status was evident. The frequency of overweight patients showed a threefold increase, rising from 5.0% (n=12) at diagnosis to 14.9% (n=36) after 6 months. The prevalence of obesity also increased, though more modestly, from 2.5% (n=6) to 3.7% (n=9). Consistent with the younger cohort, the proportion of underweight patients decreased significantly, from 64.5% to 45.9% (Table 2).

Factors Associated with Nutritional Status after Six Months of Treatment

Bivariate analysis using the Chi-square test was conducted on the entire cohort (N=325) to determine associations between baseline characteristics and nutritional status (BMI/WFH) after six months of treatment (Table 3).

Gender

A statistically significant association was found between patient gender and nutritional status at the 6-month follow-up (*P*=0.002). The combined frequency of overweight or obese statuses was significantly higher in female patients (41.8%; overweight: 29.9%, obese: 11.9%) compared to male patients (22.9%; overweight: 18.2%, obese: 4.7%) (Table 3).

WBC Count at Diagnosis

The initial WBC count at diagnosis was also found to be significantly associated with the 6-month nutritional status (*P*=0.006). Patients who presented with higher WBC counts ($> 10 \times 10^3$ uL) showed a greater prevalence of overweight and obesity compared to those with lower counts. Specifically, the groups with WBC counts of 10,000 to 50,000 uL and $> 50,000$ uL demonstrated the highest combined proportions of overweight and obese patients (43.9% and 47.5%, respectively). For instance, in the 10,000 to 50,000 uL group, 32.7% were overweight and 11.2% were obese (Table 3).

Table 1. Baseline Demographic Characteristics of the Study Population

Characteristics	Frequency (n)	Percentage (%)
Gender		
Female	177	54.5
Male	148	45.5
Age Distribution (Years)		
<2	83	25.5
2 to 10	211	64.9
>10	31	9.5
Total Patients	325	100.0

Table 2. Change in Nutritional Status at Diagnosis and After Six Months of Treatment (Patients <2 Years and ≥2 Years)

Nutritional Status Category	Patients <2 Years (WFH Status)	Patients ≥2 Years (BMI Status)
At Diagnosis (n, %)	After 6 Months (n, %)	At Diagnosis (n, %)
Underweight	42 (50.6)	27 (32.6)
Normal Weight	35 (42.2)	38 (45.8)
Overweight	5 (6.0)	10 (12.0)
Obesity	1 (1.2)	8 (9.6)
Total Patients	83 (100.0)	83 (100.0)

Table 3. Associations between baseline characteristics and nutritional status (BMI/WFH) after six months of treatment

Factor	Underweight (n, %)	Normal Weight (n, %)	Overweight (n, %)	Obese (n, %)	P-value
Gender					
Male (n=148)	68 (45.9)	46 (31.1)	27 (18.2)	7 (4.7)	0.002
Female (n=177)	53 (29.9)	50 (28.2)	53 (29.9)	21 (11.9)	
WBC Count (× 10 ³ / uL) at Diagnosis					
<4 (n=95)	46 (48.4)	29 (30.5)	16 (16.8)	4 (4.2)	0.006
4-10 (n=73)	31 (42.5)	25 (34.2)	12 (16.4)	5 (6.8)	
10-50 (n=98)	26 (26.5)	29 (29.6)	32 (32.7)	11 (11.2)	
>50 (n=59)	18 (30.5)	13 (22.0)	20 (33.9)	8 (13.6)	
CNS Involvement					
No (n=304)	113 (37.2)	92 (30.3)	74 (24.3)	25 (8.2)	0.486
Yes (n=21)	8 (38.1)	4 (19.0)	6 (28.6)	3 (14.3)	
Treatment Type					
Dexamethasone (n=41)	17 (41.5)	12 (29.3)	6 (14.6)	6 (14.6)	0.632
Prednisolone (n=36)	12 (33.3)	11 (30.6)	10 (27.8)	3 (8.3)	
Dexamethasone+ Prednisolone (n=248)	92 (37.1)	73 (29.4)	64 (25.8)	19 (7.7)	
Total (N=325)	121 (37.2)	96 (29.5)	80 (24.6)	28 (8.6)	

CNS Involvement and Treatment Type

No statistically significant association was observed between the 6-month nutritional status and either the presence of Central Nervous System (CNS) involvement ($P=0.486$) or the specific type of corticosteroid therapy (Dexamethasone, Prednisolone, or combined regimen) administered ($P=0.632$) (Table 3).

Discussion

The results of this study demonstrated a significant and rapid change in the nutritional profile of children undergoing treatment for ALL during the critical first six months. This period, characterized by intensive chemotherapy and mandatory glucocorticoid use, led to a pronounced shift from a high prevalence of underweight status at diagnosis to a considerable increase in overweight and obesity by the six-month follow-up.

The observed nutritional transition in this study, where a high baseline prevalence of underweight (50.6% for patients <2 years and 64.5% for patients ≥2 years) rapidly shifted to increased overnutrition within the first six months, strongly mirrors outcomes reported internationally, underscoring a well-recognized adverse metabolic effect of intensive ALL therapy.¹⁵ This transition is evidenced by the surge in the combined prevalence of overweight and obesity to 18.6% post-treatment for patients ≥2 years, a trend consistent with studies like Egnell et al.¹⁶ and Zhang et al.,¹⁷ which documented similar substantial increases in overnutrition during treatment and follow-up. This metabolic perturbation is overwhelmingly attributed to the required administration of glucocorticoids (prednisolone and dexamethasone) during the induction and consolidation phases.¹⁸ Glucocorticoids stimulate appetite (hyperphagia) and

promote visceral adiposity, while the cytotoxic effects of chemotherapy and associated fatigue often lead to reduced physical activity, creating a sustained positive energy balance.¹⁹

This study identified two important patient characteristics, gender and initial WBC count, as significant independent factors associated with adverse nutritional status (overweight/obesity) at the six-month follow-up. The finding that female patients exhibited a significantly higher combined prevalence of overweight/obesity post-treatment strongly confirms a potential gender-specific vulnerability to the obesogenic environment of ALL therapy.²⁰ While some investigations, such as a study by Touyz et al.,²¹ reported no significant association between post-treatment BMI and gender, a growing body of evidence supports this differential response. For instance, a study by Galati et al.²² identified gender as a major predictor of post-treatment BMI. The heightened risk in girls, as corroborated by our findings and the majority of the literature, is hypothesized to be linked to several factors: increased sensitivity to steroids and resulting hyperphagia,²³ a higher predisposition to developing insulin resistance,²⁴ greater accumulation of abdominal obesity, and a subsequently elevated risk of persistent obesity.²⁰ This differential response underscores the necessity for a tailored approach to nutritional counseling, prioritizing early, intensive intervention for female patients.²⁵

The observed significant association between a high baseline WBC count (≥ 10 uL) and a significantly higher prevalence of overweight/obesity at the six-month follow-up is a clinically critical finding, establishing this factor, historically a marker of increased tumor burden and disease aggressiveness, as an early metabolic risk marker.

This is corroborated by the literature, where studies have acknowledged the relevance of WBC counts above 10,000 uL in assessing post-treatment BMI changes,²⁶ and where an association between higher WBC counts at diagnosis and higher post-treatment BMI has been reported.²⁷ The mechanism is posited to stem from the need for intensified therapeutic demands in high-risk patients, as those with higher WBC counts often receive more potent chemotherapy, leading to greater metabolic disruption than in standard-risk patients.²⁸ This intensified treatment exposure is hypothesized to exaggerate the known obesogenic effects of glucocorticoids, driving greater increases in appetite and insulin resistance, enhanced fat accumulation, and more prolonged inactivity.²⁹ Consequently, the finding necessitates the implementation of stringent early metabolic monitoring and proactive intervention specifically for ALL patients classified as high-risk based on their initial WBC count.³⁰

The absence of a statistical association between the adverse nutritional outcome at six months and either CNS involvement or corticosteroid type is noteworthy. The lack of association with CNS status in our study may reflect the efficacy of modern ALL treatment protocols, which have largely moved away from routine cranial radiotherapy (CRT) in favor of intensive intrathecal chemotherapy for prophylaxis.³¹ This is crucial because CRT, historically used for CNS-directed therapy, is known to cause damage to the hypothalamus and pituitary gland.³² Such damage leads to hormonal disorders, including hypothyroidism and growth hormone deficiency, which are established secondary causes of severe obesity and weight gain.³³ While studies have shown variable results—for instance, Touyz et al.²¹ associated a higher post-treatment BMI with CNS involvement, whereas Arpe et al.³⁴ found no significant association, our finding suggests that if CNS involvement did not necessitate CRT, the primary mechanism for hypothalamic-mediated obesity was avoided. However, it must be acknowledged that children with CNS involvement are often classified as high-risk and may require more intensive systemic chemotherapy, which itself contributes to weight gain through non-hypothalamic pathways like increased appetite and insulin resistance.³⁵ The radiotherapy intended for these patients can cause damage to the hypothalamus and pituitary gland, and this damage causes more hormonal disorders (hypothyroidism, growth hormone deficiency, increased cortisol levels), all of which are associated with obesity and weight gain.³⁶

In our study, BMI after 6 months of treatment of patients was not significantly associated with their type of treatment. In the study of Belle et al., cranial radiotherapy was one of the risk factors associated with obesity. Steroids were not associated with overweight or obesity.³⁷ In the study of Galati et al., the type of chemotherapy regimen was a significant predictor of BMI after treatment.²² In

the study of Egnell et al., children who had undergone steroid treatment were most prone to overweight.¹⁶ Given that corticosteroids increase appetite and accumulation of abdominal fat and insulin resistance, and the radiation therapy intended for patients can cause damage to the hypothalamus and pituitary and further hormonal disorders (hypothyroidism, growth hormone deficiency, increased cortisol levels), all of which are associated with obesity and weight gain.³⁸ Therefore, the type of treatment regimen for children with ALL is associated with an increase in BMI after their treatment.³⁹

The lack of a statistically significant association between nutritional status at six months and the specific corticosteroid type used (Dexamethasone vs. Prednisolone) in our cohort is highly informative. This finding confirms that the class effect of glucocorticoids is the dominant metabolic contributor to early weight gain, overshadowing any subtle differences in potency, half-life, or dosing schedules between the two agents within the context of the complex ALL regimen.⁴⁰ This perspective aligns with clinical consensus, which views total glucocorticoid exposure as the primary metabolic challenge.⁴¹ Studies often show mixed results regarding the specific agent: while some research indicates greater toxicity (including potentially greater weight gain) with Dexamethasone due to its higher potency,⁴² our results emphasize that the underlying mechanism, steroid-induced hyperphagia, insulin resistance, and visceral fat accumulation, is fundamental to both drugs.

Despite our finding on steroid type, the overall treatment regimen is undeniably associated with post-treatment BMI, reflecting the combined impact of all therapeutic components.⁴³ In contrast to our study's non-association with specific steroid type, the study by Egnell et al.¹⁶ specifically found that children who had undergone steroid treatment were most prone to becoming overweight. Furthermore, Galati et al.²² identified the type of chemotherapy regimen as a significant predictor of post-treatment BMI, highlighting that the entire drug cocktail matters. It is essential to recognize that other treatment components, particularly cranial radiotherapy (CRT), are strongly linked to obesity risk, as shown by Belle et al.³⁷ CRT can damage the hypothalamus and pituitary gland, leading to hormonal disorders (e.g., hypothyroidism, growth hormone deficiency, elevated cortisol levels) that directly promote severe, persistent obesity and greater difficulty with weight management than steroid exposure alone.⁴⁴ Therefore, while we did not find a difference between the two most common steroids, the type of treatment regimen, particularly the use of CRT or more intensive systemic chemotherapy, remains crucial in determining the ultimate risk of BMI increase.⁴⁵

Limitations of study

This study provides key insights into early nutritional

changes during ALL treatment but is subject to several limitations. The primary constraint is the reliance on BMI and WFH, which are surrogate measures that fail to distinguish between fat mass and lean muscle mass, thus potentially underestimating the true cardiometabolic risk associated with treatment-induced fat gain (specifically visceral adiposity). Secondly, the lack of granular, longitudinal data on caloric intake, specific dietary composition, and physical activity levels prevents establishing precise causality between treatment side effects (like hyperphagia) and weight accumulation. Furthermore, being a single-center study, the generalizability of the findings may be restricted by ethnic, socioeconomic, and institutional variances. The short, six-month follow-up captures the peak period of weight gain but offers no information on the long-term persistence of the acquired weight status or the subsequent risk of metabolic syndrome in survivorship. Finally, the analysis did not account for the cumulative glucocorticoid dose received by each patient, which may be a more accurate predictor of metabolic disruption than the specific corticosteroid agent utilized.

Conclusion

In conclusion, this study validates the urgent need for comprehensive metabolic risk management beginning at the time of ALL diagnosis. The rapid progression to overweight and obesity within the first six months is a critical window for intervention. The identified risk factors, female gender and high WBC count, should be integrated into clinical algorithms to stratify patients for high-intensity nutritional and physical activity support. Future research must focus on prospective, randomized controlled trials of early dietary and exercise interventions aimed at mitigating glucocorticoid-induced weight gain and improving long-term cardiovascular health in these vulnerable patients.

Acknowledgments

The authors would like to express their gratitude to the clinical research development unit of Imam Khomeini Hospital, Urmia University of Medical Sciences, for English editing.

Authors' Contribution

Conceptualization: Mehran Noroozi
Data curation: Masomeh Sabonchi
Formal analysis: Masomeh Sabonchi
Funding acquisition: Mehran Noroozi
Investigation: Masomeh Sabonchi
Methodology: Mehran Noroozi
Project administration:
Resources: Masomeh Sabonchi
Software: Masomeh Sabonchi
Supervision: Mehran Noroozi
Validation: Mehran Noroozi
Visualization: Mehran Noroozi
Writing—original draft: Masomeh Sabonchi
Writing—review & editing: Masomeh Sabonchi

Competing Interests

The authors have no competing interests to declare that are relevant to the content of this article.

Ethics Approval

The research was conducted in accordance with the tents of the Declaration of Helsinki. The Ethics Committee of Urmia University approved this study (IR.UMSU.REC.1403.334).

Funding

The authors did not receive support from any organization for the submitted work.

References

1. Bhojwani D, Yang JJ, Pui CH. Biology of childhood acute lymphoblastic leukemia. *Pediatr Clin North Am.* 2015;62(1):47–60. doi:10.1016/j.pcl.2014.09.004
2. Wells JC, Sharma S, Del Paggio JC, Hopman WM, Gyawali B, Mukherji D, et al. An Analysis of Contemporary Oncology Randomized Clinical Trials From Low/Middle-Income vs High-Income Countries. *JAMA Oncol.* 2021;7(3):379–85. doi:10.1001/jamaoncol.2020.7478
3. Lee EM, Jiménez-Fonseca P, Galán-Moral R, Coca-Membrives S, Fernández-Montes A, Sorribes E, et al. Toxicities and Quality of Life during Cancer Treatment in Advanced Solid Tumors. *Curr Oncol.* 2023;30(10):9205–16. doi:10.3390/curroncol30100665
4. Fruh SM. Obesity: Risk factors, complications, and strategies for sustainable long-term weight management. *J Am Assoc Nurse Pract.* 2017;29(S1):S3–s14. doi:10.1002/2327-6924.12510
5. Olfa B, Salma M, Othman Rym B, Mansour Hajer B, Houyem K, Henda J, et al. Impact of Nutritional Status on Chemotherapy Related Digestive Toxicity in Women With Breast Cancer. *Nutr Metab Insights.* 2024;17:11786388241297142. doi:10.1177/11786388241297142
6. Braidotti S, Curci D, Zanon D, Maestro A, Longo A, De Vita N, et al. Impact of Body Mass Index on Outcomes in Pediatric Allogeneic Hematopoietic Stem Cell Transplantation Recipients: A Single-Center Retrospective Study. *Nutrients.* 2024;16(21):3638. doi:10.3390/nu16213638
7. Browne EK, Zhou Y, Chemaitilly W, Panetta JC, Ness KK, Kaste SC, et al. Changes in body mass index, height, and weight in children during and after therapy for acute lymphoblastic leukemia. *Cancer.* 2018;124(21):4248–59. doi:10.1002/cncr.31736
8. Poggioli R, Ueta CB, Drigo RA, Castillo M, Fonseca TL, Bianco AC. Dexamethasone reduces energy expenditure and increases susceptibility to diet-induced obesity in mice. *Obesity (Silver Spring).* 2013;21(9):E415–20. doi:10.1002/oby.20338
9. Chow EJ, Pihoker C, Hunt K, Wilkinson K, Friedman DL. Obesity and hypertension among children after treatment for acute lymphoblastic leukemia. *Cancer* 2007;110(10):2313–20. doi:10.1002/cncr.23050
10. Withycombe JS, Smith LM, Meza JL, Merkle C, Faulkner MS, Ritter L, et al. Weight change during childhood acute lymphoblastic leukemia induction therapy predicts obesity: a report from the Children's Oncology Group. *Pediatr Blood Cancer.* 2015;62(3):434–9. doi:10.1002/pbc.25316
11. Orgel E, Sposto R, Malvar J, Seibel NL, Ladas E, Gaynon PS, et al. Impact on survival and toxicity by duration of weight extremes during treatment for pediatric acute lymphoblastic leukemia: A report from the Children's Oncology Group. *J Clin Oncol.* 2014;32(13):1331–7. doi:10.1200/jco.2013.52.6962
12. Cebeci D, Kaya Z, Bideci A, Koçak Ü, Yılmaz C, Gürsel T. The

Prevalence, Risk Factors and Cardiometabolic Complications of Obesity in Childhood Survivors of Acute Lymphoblastic Leukemia. *Indian J Hematol Blood Transfus.* 2024;40(3):415–22. doi:10.1007/s12288-024-01734-w

13. Gallagher C, Moschonis G, Lambert K, Kanellakis S, Karaglan E, Mourouti N, et al. Infant BMI trajectories as early risk markers of poor psychosocial health in preadolescence. *BMC Public Health.* 2024;24(1):2890. doi:10.1186/s12889-024-19872-1

14. Jørgensen RM, Støvring H, Østergaard JN, Hede S, Svendsen K, Vestergaard ET, et al. Long-Term Change in BMI for Children with Obesity Treated in Family-Centered Lifestyle Interventions. *Obes Facts.* 2024;17(6):570–81. doi:10.1159/000540389

15. Sepúlveda-Lizcano L, Arenas-Villamizar VV, Jaimes-Duarte EB, García-Pacheco H, Paredes CS, Bermúdez V, et al. Metabolic Adverse Effects of Psychotropic Drug Therapy: A Systematic Review. *Eur J Investig Health Psychol Educ.* 2023;13(8):1505–20. doi:10.3390/ejihpe13080110

16. Egnell C, Närhinen H, Merker A, Jonsson Ó G, Lepik K, Niinimäki R, et al. Changes in body mass index during treatment of childhood acute lymphoblastic leukemia with the Nordic ALL2008 protocol. *Eur J Haematol.* 2022;109(6):656–63. doi:10.1111/ejh.13848

17. Zhang FF, Rodday AM, Kelly MJ, Must A, MacPherson C, Roberts SB, et al. Predictors of being overweight or obese in survivors of pediatric acute lymphoblastic leukemia (ALL). *Pediatr Blood Cancer.* 2014;61(7):1263–9. doi:10.1002/pbc.24960

18. Pourhassan H, Murphy L, Aldoss I. Glucocorticoid Therapy in Acute Lymphoblastic Leukemia: Navigating Short-Term and Long-Term Effects and Optimal Regimen Selection. *Curr Hematol Malig Rep.* 2024;19(4):175–85. doi:10.1007/s11899-024-00735-w

19. Kuckuck S, van der Valk ES, Scheurink AJW, van der Voorn B, Iyer AM, Visser JA, et al. Glucocorticoids, stress and eating: The mediating role of appetite-regulating hormones. *Obes Rev.* 2023;24(3):e13539. doi:10.1111/obr.13539

20. Shapira N. Women’s higher health risks in the obesogenic environment: a gender nutrition approach to metabolic dimorphism with predictive, preventive, and personalised medicine. *Epm j.* 2013;4(1):1. doi:10.1186/1878-5085-4-1

21. Touyz LM, Cohen J, Neville KA, Wakefield CE, Garnett SP, Mallitt KA, et al. Changes in body mass index in long-term survivors of childhood acute lymphoblastic leukemia treated without cranial radiation and with reduced glucocorticoid therapy. *Pediatr Blood Cancer.* 2017;64(4):e26344. doi:10.1002/pbc.26344

22. Galati PC, Rocha PRS, Gruezo ND, Amato AA. Body mass trajectory from diagnosis to the end of treatment in a pediatric acute lymphoblastic leukemia cohort. *Sci Rep.* 2023;13(1):13590. doi:10.1038/s41598-023-39287-z

23. Heymsfield SB, Avena NM, Baier L, Brantley P, Bray GA, Burnett LC, et al. Hyperphagia: current concepts and future directions proceedings of the 2nd international conference on hyperphagia. *Obesity (Silver Spring).* 2014;22 Suppl 1(0 1):S1–s17. doi:10.1002/oby.20646

24. Ding H, Zhang J, Zhang F, Zhang S, Chen X, Liang W, et al. Resistance to the Insulin and Elevated Level of Androgen: A Major Cause of Polycystic Ovary Syndrome. *Front Endocrinol (Lausanne).* 2021;12:741764. doi:10.3389/fendo.2021.741764

25. Rothpletz-Puglia P, Smith J, Pavuk C, Leotta J, Pike K, Presley CJ, et al. How a Medically Tailored Meal Intervention with Intensive Nutrition Counseling Created Active Coping with Behavior Change for Vulnerable Patients with Lung Cancer. *Res Sq.* 2024;rs.3.rs-3915333. doi:10.21203/rs.3.rs-3915333/v1

26. Chung GE, Yim JY, Kim D, Kwak MS, Yang JI, Chung SJ, et al. Associations between White Blood Cell Count and the Development of Incidental Nonalcoholic Fatty Liver Disease. *Gastroenterol Res Pract.* 2016;2016:7653689. doi:10.1155/2016/7653689

27. Li Z, Yao Z, Liu Q. The association between white blood cell counts and metabolic health obesity among US adults. *Front Nutr.* 2025;12:1458764. doi:10.3389/fnut.2025.1458764

28. Anand U, Dey A, Chandel AKS, Sanyal R, Mishra A, Pandey DK, et al. Cancer chemotherapy and beyond: Current status, drug candidates, associated risks and progress in targeted therapeutics. *Genes Dis.* 2023;10(4):1367–401. doi:10.1016/j.gendis.2022.02.007

29. Geer EB, Islam J, Buettner C. Mechanisms of glucocorticoid-induced insulin resistance: focus on adipose tissue function and lipid metabolism. *Endocrinol Metab Clin North Am.* 2014;43(1):75–102. doi:10.1016/j.ecl.2013.10.005

30. Handelsman Y, Butler J, Bakris GL, DeFronzo RA, Fonarow GC, Green JB, et al. Early intervention and intensive management of patients with diabetes, cardiorenal, and metabolic diseases. *J Diabetes Complications.* 2023;37(2):108389. doi:10.1016/j.jdiacomp.2022.108389

31. Wilejto M, Di Giuseppe G, Hitzler J, Gupta S, Abula O. Treatment of young children with CNS-positive acute lymphoblastic leukemia without cranial radiotherapy. *Pediatr Blood Cancer.* 2015;62(11):1881–5. doi:10.1002/pbc.25620

32. Follin C, Erfurth EM. Long-Term Effect of Cranial Radiotherapy on Pituitary-Hypothalamus Area in Childhood Acute Lymphoblastic Leukemia Survivors. *Curr Treat Options Oncol.* 2016;17(9):50. doi:10.1007/s11864-016-0426-0

33. Park HK, Ahima RS. Endocrine disorders associated with obesity. *Best Pract Res Clin Obstet Gynaecol.* 2023;90:102394. doi:10.1016/j.bpobgyn.2023.102394

34. Arpe ML, Rørvig S, Kok K, Mølgaard C, Frandsen TL. The association between glucocorticoid therapy and BMI z-score changes in children with acute lymphoblastic leukemia. *Support Care Cancer.* 2015;23(12):3573–80. doi:10.1007/s00520-015-2718-5

35. Sachdeva P, Ghosh S, Ghosh S, Han S, Banerjee J, Bhaskar R, et al. Childhood Obesity: A Potential Key Factor in the Development of Glioblastoma Multiforme. *Life (Basel).* 2022;12(10):1673. doi:10.3390/life12101673

36. Contrera KJ, Phan J, Waguespack SG, Aldehaim M, Wang X, Lim TY, et al. Prevalence of pituitary hormone dysfunction following radiotherapy for sinonasal and nasopharyngeal malignancies. *Head Neck.* 2023;45(10):2525–32. doi:10.1002/hed.27476

37. Belle FN, Schindera C, Ansari M, Armstrong GT, Beck-Popovic M, Howell R, et al. Risk factors for overweight and obesity after childhood acute lymphoblastic leukemia in North America and Switzerland: A comparison of two cohort studies. *Cancer Med.* 2023;12(20):20423–36. doi:10.1002/cam4.6588

38. Chiloiro S, Vicari A, Mongelli G, Costanza F, Giampietro A, Mattogno PP, et al. Effects of glucocorticoid replacement therapy in patients with pituitary disease: A new perspective for personalized replacement therapy. *Rev Endocr Metab Disord.* 2024;25(5):855–73. doi:10.1007/s11154-024-09898-6

39. Atkinson HC, Marsh JA, Rath SR, Kotecha RS, Gough H, Taylor M, et al. Increased Body Mass Index during Therapy for Childhood Acute Lymphoblastic Leukemia: A Significant and Underestimated Complication. *Int J Pediatr.* 2015;2015:386413. doi:10.1155/2015/386413

40. Berthon BS, MacDonald-Wicks LK, Wood LG. A systematic

review of the effect of oral glucocorticoids on energy intake, appetite, and body weight in humans. *Nutr Res.* 2014;34(3):179–90. doi:10.1016/j.nutres.2013.12.006

41. Kmiec P, Świątkowska-Stodulska R. Glucocorticoids: The culprit behind metabolic disorders in primary Aldosteronism? A narrative review. *J Clin Transl Endocrinol.* 2025;41:100401. doi:10.1016/j.jcte.2025.100401

42. Beaupere C, Liboz A, Fève B, Blondeau B, Guillemain G. Molecular Mechanisms of Glucocorticoid-Induced Insulin Resistance. *Int J Mol Sci.* 2021;22(2):623. doi:10.3390/ijms22020623

43. Ma SJ, Khan M, Chatterjee U, Santhosh S, Hashmi M, Gill J, et al. Association of Body Mass Index With Outcomes Among Patients With Head and Neck Cancer Treated With Chemoradiotherapy. *JAMA Netw Open.* 2023;6(6):e2320513. doi:10.1001/jamanetworkopen.2023.20513

44. van Santen HM, Müller HL. Management of Acquired Hypothalamic Dysfunction and the Hypothalamic Syndrome; It Is More Than Obesity. *Endocr Rev.* 2025;46(6):891–907. doi:10.1210/endrev/bnaf025

45. Kapala A, Surwiłło-Snarska A, Jodkiewicz M, Kawecki A. Nutritional Care in Patients with Head and Neck Cancer during Chemoradiotherapy (CRT) and Bioradiotherapy (BRT) Provides Better Compliance with the Treatment Plan. *Cancers (Basel).* 2021;13(11):2532. doi:10.3390/cancers13112532