Acta Paediatrica

WILEY

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NURTURING THE CHILD

ORIGINAL ARTICLE OPEN ACCESS

Age- and Sex-Adjusted Body Mass Index Increases in Childhood Acute Lymphoblastic Leukaemia Patients From Diagnosis to Five-Year Follow-Up

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Received: 2 July 2025 | **Revised:** 27 November 2025 | **Accepted:** 2 December 2025

Keywords: acute lymphoblastic leukaemia | childhood cancer survivor | late effect of childhood cancer | overweight | weight gain

ABSTRACT

Aim: Children with acute lymphoblastic leukaemia (ALL) are at risk of metabolic and cardiovascular complications. We evaluated the development of overweight and obesity for 5 years after diagnosis in children and adolescents treated for ALL.

Methods: The medical records of children diagnosed with ALL at one centre during 2000–2018 were assessed. Weight and height measurements were retrieved from medical records and were used to calculate age- and sex-adjusted International Obesity Task Force-Body Mass Index (ISO-BMI). ISO-BMI was determined at selected time points during treatment and up to 5 years after diagnosis, and the change in mean ISO-BMI was assessed.

Results: We studied 115 patients diagnosed with ALL, 54 (47%) of whom were male. Mean age at diagnosis was 6.6 ± 4.6 (range 0–17.99) years. ISO-BMI increased significantly during treatment ($p < 0.0001$) and remained elevated at 5 years after diagnosis ($p < 0.0001$). The number of overweight and obese patients increased from 17% and 4% at diagnosis to 26% and 16% at the five-year follow-up.

Conclusions: Patients treated for ALL are at significant risk of weight gain and obesity, with the prevalence of overweight and obesity doubling from diagnosis to 5 years post-treatment. ISO-BMI remained persistently elevated across all treatment risk groups.

1 | Introduction

Acute lymphoblastic leukaemia (ALL) is the most common type of cancer in children. Advances in treatment and collectively defined treatment protocols have made it possible for up to 90% of paediatric patients diagnosed with ALL to be cured [1, 2]. In Finland and other Nordic countries, the Nordic Society of Pediatric Haematology and Oncology (NOPHO) protocols have been used to treat ALL [1, 3]. A variety of metabolic and cardiovascular complications, as well as signs of accelerated aging, commonly occur in survivors of childhood ALL and other cancers [4–9]. Metabolic complications are due to several factors, such as hormonal imbalance induced by various treatments [10].

Studies with older treatment protocols have shown that obesity is more common in ALL survivors than in healthy cohorts [11].

Abbreviations: ALL, acute lymphoblastic leukaemia; BMI, body mass index; EsPhALL, European intergroup study of post-induction treatment of Philadelphia-chromosome-positive acute lymphoblastic leukaemia; INTERFANT, International Collaborative Treatment Protocol for Infants Under One Year with Acute Lymphoblastic Leukaemia or Biphenotypic Leukaemia; ISO-BMI, International Obesity Task Force-Body Mass Index; NOPHO, Nordic Society of Pediatric Haematology and Oncology.

Summary

- Children and adolescents with acute lymphoblastic leukaemia (ALL) are at risk of metabolic and cardiovascular late effects.
- The number of overweight and obese subjects doubled from ALL diagnosis to five-year follow-up, with over 40% of children and adolescents being overweight or obese at 5 years after diagnosis.
- Excess weight gain during ALL treatment affected all the treatment risk groups, so effective ways to intervene are needed.

International Obesity Task Force-Body Mass Index (ISO-BMI) is conducted from the population [12, 13]. It is applicable for children aged 2–18 years. Besides height and weight, the ISO-BMI model also takes the child's age and sex into account. As the body composition changes through childhood and adolescence, ISO-BMI has been suggested to be the best available tool for monitoring weight gain and the development of obesity in children and adolescents [12, 13]. Traditionally, weight in Finnish children has been assessed as a percentage of the median weight-for-height. New growth curves with data for ISO-BMI were published in 2011, making it possible to use national reference values for ISO-BMI [13]. Considering the differences in prevalence of overweight and obesity among different populations, using national reference values should be preferred. Furthermore, studies on weight change in Nordic childhood ALL survivors have not included long-term follow-up after cessation of treatment.

The aim of this study was to investigate changes in ISO-BMI during ALL treatment and up to 5 years after diagnosis, in a comprehensive, non-selected cohort treated with contemporary protocols. We hypothesised that ISO-BMI will increase in children diagnosed with ALL regardless of original weight status, and children treated for ALL present an elevated ISO-BMI 5 years after diagnosis compared to the time of diagnosis.

2 | Patients and Methods

2.1 | Study Subjects

We conducted a single-centre, retrospective cohort study. The study population was under 18-year-old patients at Turku University Hospital, Turku, Finland, diagnosed with acute lymphoblastic leukaemia from 1 January 2000 to 31 December 2018. All patients treated with the NOPHO ALL2000, NOPHO ALL2008, European intergroup study of post-induction treatment of Philadelphia-chromosome-positive ALL (EsPhALL) or International Collaborative Treatment Protocol for Infants Under One Year with Acute Lymphoblastic Leukaemia or Biphenotypic Leukaemia (Interfant)—protocols were included in the study. We obtained all data retrospectively by reviewing the patients' files. The characteristics of the study population are shown in Table 1. The exclusion criteria were cancer disease not classified as ALL, death before the end of treatment, or missing height and weight measurements at the time of diagnosis. In addition, due to low numbers of patients treated with the Interfant or EsPhALL protocols, these patients were excluded from the analysis done by treatment protocol or risk group. The follow-up lasted for 5 years, or until the patient's death, whichever occurred first. Seven of the patients (6%) had a relapse during treatment, and an additional 10 (9%) patients had a relapse during the follow-up period. A total of 8 (7%) patients died during the follow-up period (Table 1).

2.2 | Weight and Height Measurements

The subjects' height and weight charts were assessed, and the corresponding ISO-BMI at each time point was calculated [13]. The 30-month treatment period included assessments at diagnosis and at three, six, 12 and 30 months after treatment initiation. Following treatment, growth curves were assessed at 1 year post-treatment (42 months since diagnosis) and at 5 years (60 months) post-diagnosis.

The growth reference curves for Finnish children have been updated in 2011 [13]. BMI-for-age curves described for children aged 2–18 years were used, and the weight status of the patients was divided into four classes according to the ISO- BMI [13]. The ISO-BMI cut-offs for grade 2 thinness, overweight and obesity are defined as BMI-for-age percentile curves that pass through adult values of 17, 25 and 30 kg/m^2, making the ISO-BMI values interpretable like the adult BMI values [12, 13]. The weight classification by ISO-BMI in this study was carried out accordingly: underweight (under 17 kg/m^2), normal weight (17–24.9 kg/m^2), overweight (25–29.9 kg/m^2) and obese ($\geq$ 30.0 kg/m^2).

For subjects aged under 2 years, ISO-BMI was not applicable, so weight-for-height percentage was used to determine the weight status and divide the subjects into weight groups underweight, normal weight, overweight and obese according to the Finnish growth references. Although weight-for-height % indicates percentual difference from the mean weight of children with the same height and gender, a weight-for-height % between −10% and +10% in children aged under 2 years indicates normal weight. Weight-for-height percentages under −10% were considered underweight, over +10% were considered overweight and over +20% were considered obese.

2.3 | Statistical Analysis

Linear mixed models for repeated effects were used to analyse change in ISO-BMI between the study time points. Log transformation was used to achieve normal distribution. Follow-up measurements were compared to the baseline measurement using Dunnett's correction.

We categorised the subjects into two groups by treatment protocol: NOPHO ALL2000 and NOPHO ALL2008. In addition, the same analyses were performed by categorising the subjects further by gender, weight classification at diagnosis (underweight, normal weight, overweight and obese or severely obese), age group at diagnosis ($<$ 5, 5–9.99 and $\geq$ 10 years) and treatment risk group (standard, intermediate and high risk). Due to the low number of patients treated with the Interfant or EsPhALL

TABLE 1 | Patient characteristics.

Characteristics	Total ($n=115$) *n* (%)
Gender	
Female	61 (53)
Male	54 (47)
Age at diagnosis	
0–4.99	61 (53)
5–9.99	27 (23)
10–17.99	27 (23)
Treatment protocol	
NOPHO ALL2000	48 (42)
NOPHO ALL2008	61 (53)
Other	6 (5)
Risk group	($n=109$)
Standard risk	45 (41)
Intermediate risk	44 (40)
High risk	20 (18)
Irradiation	
Cranial irradiation	2 (2)
Total body irradiation	12 (10)
Total body irradiation with central nervous system boost	2 (2)
No irradiation	99 (86)
Haematopoietic stem cell transplant	
Yes	18 (16)
No	96 (83)
CAR-T	1 (1)
Relapse	
Yes, during treatment	7 (6)
Yes, during 5-year follow up	10 (9)
No	98 (85)
Death during 5-year follow-up	
Yes	8 (7)
No	107 (93)
Lost to follow-up before 5-year time point (not including death)	5 (4)
Weight class at diagnosis	($n=114$)
Underweight	8 (7)
Normal	82 (72)
Overweight	19 (17)
Obese	5 (4)
Weight class after treatment period (30 months)	($n=109$)
Underweight	6 (6)
Normal	58 (53)
Overweight	30 (28)
Obese	15 (14)
Weight class after 5-year follow-up	($n=102$)
Underweight	6 (6)
Normal	54 (53)
Overweight	26 (25)
Obese	16 (16)

Abbreviation: CAR-T, Chimeric antigen receptor T cell therapy.

protocols, these children were excluded from the analyses done by the treatment protocol or risk group.

Furthermore, we performed frequency analyses with the weight classes. We compared the frequencies in each weight class at diagnosis, the 30-month time point, and at the end of follow-up using cross tabulation.

Data are given as means and standard deviations. *p*-values <0.05 were considered statistically significant. Statistical analyses were performed using SAS JMP Pro 16.1.0 (SAS Institute Inc., North Carolina, USA).

3 | Results

A total of 118 paediatric patients were diagnosed with ALL within the Hospital District of South-West Finland and treated at Turku University Hospital during 2000–2018. Three of these patients were excluded from the study, one due to death before initiation of treatment, one due to death during induction treatment and one cancer type ending up not being classified as ALL. Therefore, a total of 115 patients were included in the study. Furthermore, 109 patients were treated with the NOPHO ALL2000 and NOPHO ALL2008 protocols and included in the comparisons between the different protocols and risk groups.

Of the patients included in this study, 54 (47%) were male, and the mean age at diagnosis was 6.6 ± 4.6 (range 0–17.99) years. At diagnosis, 7% of these subjects were underweight, 72% were normal weight, 17% were overweight and 4% were obese (Table 1). There were no statistically significant differences in the mean ISO-BMI at diagnosis between the NOPHO ALL2000 and 2008 treatment protocols ($p=0.71$), risk groups ($p=0.94$), genders ($p=0.50$) or age groups ($p=0.33$).

The mean ISO-BMI for the whole study group increased during treatment ($p < 0.0001$). ISO-BMI was significantly higher after the five-year follow-up period than at the time of diagnosis ($p < 0.0001$) (Table 2). As ISO-BMI is applicable for children aged 2–18 years [13], subjects who were under 2 years old at the time of diagnosis were included in the comparisons after their age exceeded 2 years.

The change in ISO-BMI during the follow-up did not differ between the different treatment protocols (timepoint × treatment protocol interaction, $p = 0.27$) or weight classes at diagnosis (timepoint × weight classification at diagnosis interaction, $p = 0.47$). No significant differences were found between genders (timepoint × gender interaction, $p = 0.30$) or age groups (timepoint × age group at diagnosis interaction, $p = 0.25$). However, the change in ISO-BMI was significantly different between the treatment risk groups (timepoint × risk group interaction, $p = 0.038$), so the change in ISO-BMI was analysed separately also for each risk group (Figure 1).

Among the high-risk patients, ISO-BMI at timepoints 3, 6 and 12 months after the diagnosis did not differ from baseline ($p = 0.45$, $p = 0.82$ and $p = 0.21$, respectively). Their ISO-BMI at timepoints 30, 42 and 60 months after diagnosis was significantly higher compared to the baseline at diagnosis ($p = 0.001$, $p = 0.018$ and $p = 0.02$, respectively). For the intermediate-risk patients, ISO-BMI at 3 months did not differ significantly from baseline ($p = 1.00$), but at all the other timepoints, their ISO-BMI was significantly higher compared to the baseline ($p < 0.0001$ at 6 months, $p = 0.0007$ at 12 months, $p = 0.002$ at 30 months, $p < 0.0001$ at 42 months and $p = 0.0004$ at 60 months). For the standard-risk patients, ISO-BMI was higher at all time points compared to the baseline ($p = 0.049$ at 3 months, $p = 0.005$ at 6 months, $p = 0.0001$ at 12 months, $p = 0.008$ at 30 months, $p < 0.0001$ at 42 months, $p = 0.003$ at 60 months) (Figure 1).

As ISO-BMI was unsuitable for subjects under 2 years old, analyses were extended to include weight classifications at diagnosis, treatment completion, and five-year follow-up. The number of subjects in different weight classes differed significantly between the different time points ($p = 0.019$). The number of overweight or obese patients increased, and the number of normal weight patients decreased during the five-year follow-up period. The distribution of patients within different weight classes at different time points is shown in Figure 2.

TABLE 2 | Mean ISO-BMI at different time points.

Time point (months)	Estimate ISO-BMI (converted from log)[a]	Lower 95% CI	Upper 95% CI	p[b]
0	21.73	20.87	22.64	
3	22.44	21.53	23.39	0.1657
6	23.59	22.65	24.57	< 0.0001
12	23.75	22.81	24.72	< 0.0001
30	23.77	22.83	24.75	< 0.0001
42	24.39	23.42	25.40	< 0.0001
60	23.83	22.88	24.82	< 0.0001

Abbreviation: ISO-BMI, International Obesity Task Force-Body Mass Index.
[a]Back-transformed estimated marginal mean from the model. Values were log-transformed for statistical analysis and estimated marginal mean was back-transformed from the log scale.
[b]p-value of the comparison of mean ISO-BMI value with time of diagnosis (time point 0); Dunnett's method was used to compare follow-measurements to the baseline measurement.

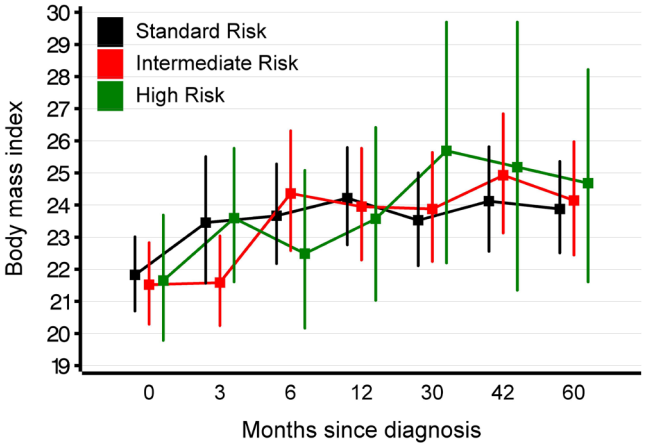

FIGURE 1 | Change in the mean (with 95% CI) ISO-BMI during ALL treatment and the follow-up period in different treatment risk groups.

After the 30-month treatment period, applicable weight and height data at this time point were provided for 109 subjects. Of these subjects, 6% were underweight at this time point, 53% were normal weight, 28% were overweight and 14% were obese. After the five-year follow-up, applicable weight and height data were available for 102 subjects. Of these subjects, 6% were underweight, 53% were normal weight, 26% were overweight and 16% were obese (Table 1).

4 | Discussion

This retrospective, single-centre study characterises the longitudinal course of ISO-BMI over 5 years following ALL diagnosis in paediatric patients treated with the two most recent Nordic protocols. Overall, ISO-BMI increased during the five-year follow-up in all risk groups. However, there was a significant difference in the pattern of how the change in ISO-BMI developed between the risk groups. The development of ISO-BMI did not differ significantly between the treatment protocol eras, age groups at diagnosis, weight groups at diagnosis or genders.

Several studies have shown that patients with ALL are at risk of gaining weight during treatment [14–18] and this was consistent with our results. The mean ISO-BMI increased during treatment and remained significantly higher at the end of the five-year follow-up period. Some of the studies on older ALL treatment protocols have suggested a similar pattern, indicating that the weight gain during ALL treatment is not temporary [16, 19, 20]. However, it is not yet completely clear whether the excess in weight gain continues after treatment and into adulthood in all risk groups [21, 22]. Belle et al. combined data from the Swiss Childhood Cancer Survivors Study and the North American Childhood Cancer Survivor Study, indicating that

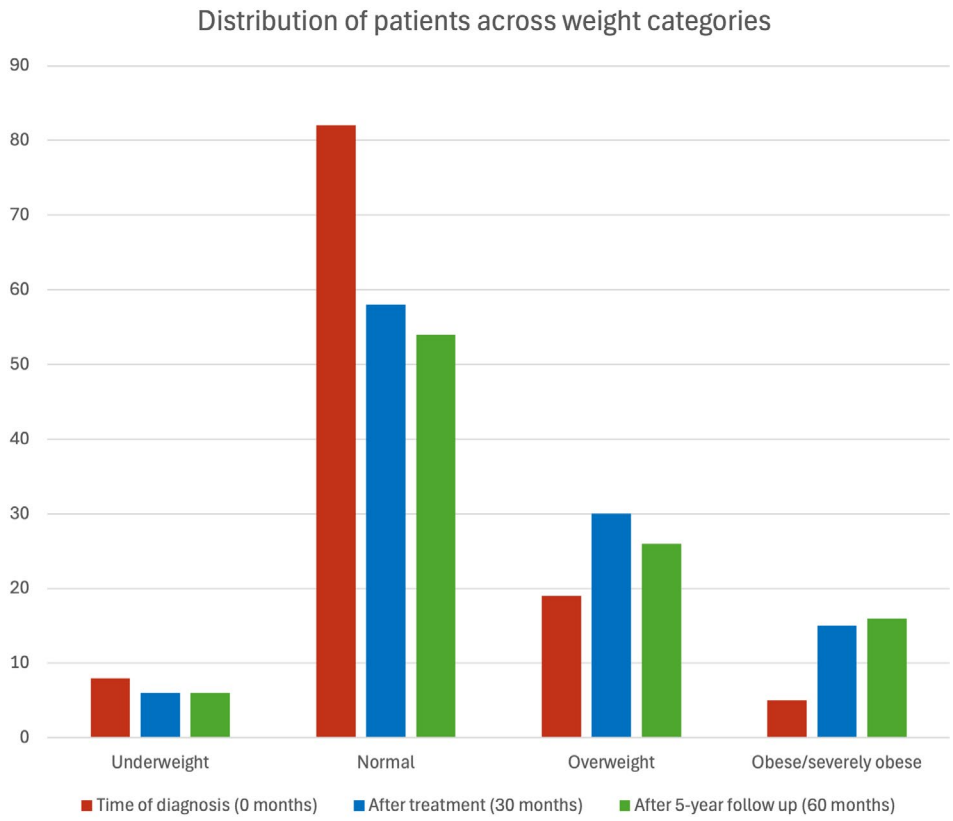

FIGURE 2 | Distribution of patients across the weight categories at different time points.

overweight and obesity were more common among survivors of childhood cancer than their siblings in both cohorts, but the prevalence of overweight and obesity seemed to vary greatly between the cohorts, also among the siblings [23].

The geographical differences in the prevalence of overweight and obesity at different ages make the comparisons difficult between different cohorts, making the national growth curves the most reliable controls. The ISO-BMI curves of the Finnish population-based growth curves have been built in a way that, for instance, an ISO-BMI value of 30 kg/m^2 corresponds to an adult BMI value of 30 kg/m^2. When building these curves, the cut-off curve for overweight (BMI 25 kg/m^2) was at the 87.8th percentile for females and 78.2nd percentile for males, while the cut-offs for obesity (BMI 30 kg/m^2) were at the 98.2nd percentile for females and 95.6th percentile for males [13]. Thus, in the present study, the number of overweight or obese survivors (41% in total) at the five-year time point indicates a far higher prevalence of overweight or obesity among survivors of ALL compared to the Finnish child population.

Some studies have shown that weight gain was confined to, or greater among, patients who underwent cranial irradiation [21, 24], but contrasting findings have also been reported [14, 15]. In this study, ISO-BMI trajectories during ALL treatment and follow-up period were similar across gender, age at diagnosis, baseline weight category, and treatment era, yet differed between risk groups. However, patients in all treatment risk groups had their ISO-BMI elevated by the end of treatment and at the later follow-up timepoints compared to the baseline. It is interesting that patients in the standard risk and intermediate risk groups gained weight earlier than the high risk patients. Our hypothesis is that this reflects the fact that the high risk patients treated with very intensive block-based chemotherapy are generally more ill and suffer from treatment-related complications such as severe mucositis and neutropenic fevers or septicaemias more often than the standard and intermediate risk patients. Thus, they may rather be at risk of malnutrition, especially at the 3- and 6-month timepoints, and only start gaining weight during the maintenance treatment or following haematopoietic stem cell transplantation. Also, the timing of the delayed intensification phase with dexamethasone was later in the high risk group, possibly affecting their weight gain at later phases of the treatment [1, 3].

Some previous studies reporting the weight gain patterns in patients diagnosed with ALL during therapy suggest that these patients tend to gain weight, especially during the first month of treatment and during maintenance therapy, but these studies have not reported the results separately for each risk group [15]. The weight gain during induction or the first months of the treatment has generally been attributed to corticosteroid exposure, but the prolonged weight gain, seen for example in our study, suggests that corticosteroids are not the only factor contributing to weight gain and obesity. For example, studies have shown that childhood cancer patients and survivors exhibit a decrease in physical activity and fitness levels compared to healthy cohorts [25, 26]. It has also been suggested that survivors of childhood cancer may have more frequent cravings for fast food [27]. One of the most proposed risk factors for weight gain is hypothalamic–pituitary axis dysfunction and the consequent growth hormone deficiency. Cranial irradiation treatment is thought to be one of the main risk factors for this dysfunction

[10]. However, cranial irradiation treatment was omitted already from the NOPHO ALL2000 protocol, except for a small subset of the high-risk patients. Only two patients in this study population had received prophylactic cranial irradiation treatment, while two had received total body irradiation with a central nervous system boost before haematopoietic stem cell transplantation (Table 1). However, some studies have suggested that even total body irradiation without a central nervous system boost could increase the risk of growth hormone deficiency and hypothalamic–pituitary axis dysfunction [28]. In this study, 10% of patients undergoing treatment received 12 Gy total body irradiation without a central nervous system boost. Thus, it is possible that in these high-risk patients, the total body irradiation could be contributing to their weight gain also via growth hormone deficiency. Also, increased levels of leptin, the hormone produced by adipose tissue, have been reported in ALL survivors, but the mechanisms behind this remain poorly understood [29].

Studies have shown that changes in gut microbiota caused by various factors, such as antibiotics, may contribute to weight gain and the development of metabolic syndrome in children. It has been speculated that changes in gut microbiome—caused by cancer and cancer treatments—may contribute to weight gain in childhood cancer. However, this has not yet been extensively studied [30].

Our study suggests that excessive weight gain during ALL treatment is still existent within the most recent NOPHO ALL2000 and NOPHO ALL2008 treatment protocols. This occurs even though the prophylactic cranial irradiation treatment was already omitted for the majority of the patients in the NOPHO ALL2000 protocol [3]. Our study also shows that ISO-BMI is significantly higher at 5 years after diagnosis compared to the start of treatment, which suggests that the weight gain is not temporary. In NOPHO ALL2000 and ALL2008 protocols, the corticosteroid used in the induction phase was prednisolone, except for the T-ALL patients and patients with leucocyte count $\geq 100 \times 10^9$/L in ALL2008 [1, 3]. The current ALLTogether1 -protocol used in many of the European countries differs from this as it uses dexamethasone (https://clinicaltrials.gov/study/NCT03911128). In the future, weight gain should be compared between these previous protocols and ALLTogether1 to see whether using different corticosteroids makes an impact on the weight gain in patients diagnosed with ALL.

The increase in ISO-BMI during and after ALL treatment does not seem temporary. It is likely to be one of the contributing factors in the elevated risk of cardiovascular disease in patients diagnosed with ALL. Studies have shown that childhood cancer survivors have a higher incidence of cardiovascular disease and purchasing cardiovascular medications, as well as a significantly higher risk of cardiovascular morbidity than their healthy siblings [5, 6, 31].

5 | Strengths and Limitations

The follow-up period for up to 5 years after diagnosis is an important strength of this study. Many previous studies present a shorter follow-up, and more studies with longer follow-up periods with contemporary protocols are essential to further analyse and understand weight gain in paediatric patients diagnosed with ALL. Also, our study is comprehensive as to including patients diagnosed within a long period of time (years 2000–2018), and it included 97% of the surviving patients diagnosed with ALL and treated at the Turku University Hospital District. Furthermore, ISO-BMI with population-based reference values is the best available instrument for evaluating weight gain in children [12, 13].

This study had also some important limitations. ISO-BMI is only applicable for children aged 2–18 years. Therefore, subjects who were under 2 years old at the time of diagnosis were included in the comparisons only after their age exceeded 2 years. Also, even though the study material included all the patients diagnosed within the years 2000–2018, the pool of subjects in the different risk groups was relatively small. Thus, because of the small overall number of subjects in the high-risk group, we could not differentiate the subjects who had undergone a stem cell transplant.

6 | Conclusion

Based on these findings, continuous follow-up and preventive interventions are needed in ALL patients. Weight gain during treatment is likely to persist in the long-term follow-up. Early identification of these patients is essential to enable the earliest possible intervention. More studies should be conducted on the reasons for weight gain to more efficiently intervene at early stages.

Author Contributions

Aino Kytömäki, Anu Huurre, Päivi Lähteenmäki and Liisa Järvelä contributed to the study conception and design. Data collection was performed by Aino Kytömäki and Liisa Järvelä. Data analyses were performed by Aino Kytömäki, Liisa Järvelä and Tero Vahlberg. The first draft of the manuscript was written by Aino Kytömäki, and all authors commented on previous versions of the manuscript. All authors have read and approved the final manuscript.

Acknowledgement

Open access publishing facilitated by Turun yliopisto, as part of the Wiley - FinELib agreement.

Funding

The authors have nothing to report.

Ethics Statement

According to the institutional guidelines of the Hospital District of South-West Finland, a permit to use patient records in this register-based study was requested from the Hospital District of South-West Finland (TO8/028/19). According to Finnish legislation, no informed consent is obtained in register-based studies.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The datasets generated and analysed during the current study are available from the corresponding author on reasonable request. The data are not publicly available due to privacy or ethical restrictions.

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