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ORIGINAL PAPER

Serum levels of salusin β in acute lymphoblastic leukaemia and Wilms' tumour survivors – preliminary report

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ABSTRACT

Introduction: Cardiovascular diseases (CVD) are one of the long-term side effects of childhood cancer treatment. Salusin β is an indicator of atherosclerosis development.
The aim of the study was to evaluate serum salusin β levels in long-term acute lymphoblastic leukaemia survivors (ALLs) and Wilms' tumour survivors (WTs) and compare these levels to established CVD risk factors.
Material and methods: Thirty-seven ALLs and eleven WTs underwent physical examination and laboratory tests after an overnight fast at least 5 years after the end of oncological treatment. The laboratory tests included analysis of lipid profiles, serum glucose levels, renal parameters and salusin β levels.
Results: The groups did not vary in age, time from the end of the treatment, number of obese persons, blood pressure, lipid profile, serum creatinine and glucose levels. The ALLs had greater weights and greater waist circumferences. The serum cystatin level was higher and the cystatin-based estimated glomerular filtration rate was lower in the WTs. Salusin β was higher in the WT group, but the result was not statistically significant.
Conclusions: Acute lymphoblastic leukaemia survivors and WTs differ in terms of types of long-term side effects. Acute lymphoblastic leukaemia survivors more often develop obesity and metabolic problems, whereas WTs tend to develop renal disorders.
Salusin β levels are associated with the level of LDL in ALLs and can indicate lipid disorders in patients with higher risk of obesity, suggesting that it could be a predictor of atherosclerosis. Further investigations are necessary to confirm this result. It is necessary to continue follow-up among adults who have been treated for childhood cancers to reveal long-term side effects such as cardiovascular disorders.

KEY WORDS:
childhood cancer survivors, long-term side effects, salusin β, metabolic disorders, hypertension.

INTRODUCTION

In recent years, the number of childhood cancer survivors (CCS) has been constantly and significantly increasing. However, this increased survival has not been achieved without a cost. Oncological treatment is associated with long-term morbidity and mortality. The cardiovascular sequelae of cancer treatment are some of the most serious complications. Cardiovascular diseases (CVD) develop earlier and lead to more severe outcomes in CCS than in the rest of the population. These effects are largely caused by the direct toxic effects

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of radio- and chemotherapy. In addition, the prevalence of other risk factors for CVDs, such as obesity, hypertension and hypercholesterolaemia, are also increased in CCS. These facts have been confirmed by numerous studies in older populations of survivors [1–4].

In parallel, CVD are of great concern to researchers because they are one of the main causes of death worldwide. Scientists are searching for biomarkers that could diagnose CVD early in development. Salusin β is considered a potential biomarker of atherosclerosis. Salusin β is a peptide that contributes to endothelial injury [5, 6]. The concentration of salusin β positively correlates with blood pressure (BP) and triglyceride levels and is elevated in conditions that lead to cardiovascular complications, such as diabetes mellitus or polycystic ovary syndrome [7–9].

The aim of this study was to evaluate the serum salusin β levels in long-term acute lymphoblastic leukaemia survivors (ALLs) and Wilms' tumour survivors (WTs) and compare these levels to established CVD risk factors that are classified as indicators of metabolic syndrome.

MATERIAL AND METHODS

PATIENTS

This study enrolled 37 ALL and 11 WT survivors who were treated between 2000 and 2013 at the Department of Paediatrics, Haematology and Oncology of the Medical University of Gdansk. The patients, ranging 7–18 years of age, were examined at least 5 years after the end of oncological treatment.

All of the patients with ALL underwent standard chemotherapy, and 9 of them also received cranial radiotherapy. Two children were assigned to the standard risk group, 11 to the intermediate risk group and 4 to the high risk group.

Nine patients with WT underwent total nephrectomy, and 3 of them also received abdominal radiotherapy. Two patients with WT who suffered from bilateral tumours underwent partial nephrectomy. In addition to surgery, all the WT patients were treated with chemotherapy. Two patients were assigned to stage I, 4 to stage II, 3 to III and 2 to V.

The clinical study was performed during routine follow-ups. The study consisted of a patient history and a physical examination, including anthropometric measurements, triple BP measurements, and blood and urine sample collection.

METHODS

The height, weight, and waist circumference of the patients were measured using standard techniques (Mensor WE 150, 2014).

During laboratory testing, we evaluated complete blood count, serum creatinine, cystatin C, glucose, lipid profiles (total cholesterol, LDL, HDL, TG) and salusin β levels after an overnight fast.

The serum creatinine concentration was analysed using an enzymatic method (Alinity c Creatinine Reagent Kit Abbott). The serum cystatin C levels were detected by immunonephelometry (N Latex Cystatin C Siemens). The estimated glomerular filtration rate (eGFR) was calculated based on the serum creatinine and cystatin C levels.

The estimated glomerular filtration rate was measured indirectly using the original Schwartz, creatinine and BUN-based equation and Filler formulas.

The Schwartz formula is defined as follows: GFR in ml/min/1.73 sq m = k × height of child in cm/serum creatinine concentration in mg/dl, where the constant k was defined using the published literature value of k = 0.413 for children [10]. Creatinine and BUN-based eGFR was calculated according to the equation – 40.7 (height/SCr)0.64(30/BUN) 0.202 [11].

Additionally, the serum concentration of cystatin C was evaluated, and GFR was calculated according to the Filler formula: logGFR = 1.962 + [1.123 × log(1/cystatin C)] [12].

The plasma lipid profile was determined by electrophoresis (Hydragel 15 Lipo + Lp(a) Sebia). The concentration of salusin β (pg/ml) was determined by an immunoenzymatic method using an ELISA kit for salusin β (produced by Cloud-Clone Corp, 2018).

The International Diabetes Federation criteria were used to define metabolic syndrome and central obesity [13].

BLOOD PRESSURE

Blood pressure was measured in every child in the study by an oscillometric method using a standard clinical sphygmomanometer (professional blood pressure monitor HBP-1100-E, OMRON HEALTHCARE Co., Ltd. Kyoto, Japan, 2014) according to guidelines and recommendations of the Polish Pediatric Nephrology Society [14, 15]. Blood pressure was measured three times in each patient. The mean values of the systolic and diastolic pressure were determined. The results were then compared to the reference values matched according to sex, age and height.

STATISTICAL METHODS

For each parameter, the mean, median, standard deviation, range (minimum-maximum), and lower and upper quartiles (25Q and 75Q) were calculated. Statistical significance between means for different groups was calculated by one-way analysis of variance (ANOVA), or using the nonparametric Mann-Whitney *U* test or Kruskal-Wallis test when the variances in groups were not homogeneous (the homogeneity of variance was determined by Bartlett's test).

Statistical significance of differences between frequencies was calculated by χ^2_{df} with Yate's correction with corresponding degrees of freedom df (df = (m–1) × (*n*–1),

where m indicates the number of rows, and *n* indicates the number of columns.

The relationship between two parameters was assessed using correlation analysis, and Spearman correlation coefficients were calculated.

The standard deviation score (SDS) was evaluated using the following formula: SDS = (observed value – mean value in referenced population)/SD value in reference population.

For the reference population, we used the results of the OLAF study, which was performed in children from the Polish population aged 7–18 years [16, 17].

A *p*-value of less than 0.05 was required to reject the null hypothesis. Statistical analysis was performed using EPIINFO Ver. 7.1.1.14 software package.

ETHICS COMMITTEE

This study was approved by the Independent Bioethical Committee of Scientific Researchers at the Medical NKBBN/359/NKBBN/359/2015 from September 15, 2015, NKBBN/359-58/2018 from February12, 2018, NKBBN/359-9/2019 from January10, 2019, NKBBN/359-721/2021 from September 21, 2021). Written informed consent was obtained from the legal guardians of the children. The procedures followed were in accordance with the Declaration of Helsinki of 1975, as revised in 2000.

RESULTS

The detailed characteristics of the studied groups are shown in Table 1. The two groups of patients did not differ significantly in terms of the time from the end of their treatment (ALLs vs. Wilms' tumour survivors (8 (25Q–75Q: 6–9) vs. 10 years (25Q–75Q: 6–13), $p = 0.188$).

There were 12 patients with central obesity in the ALL group and 2 in the WT group, which was not a significant difference. However, the weight and waist circumference

TABLE 1. Characteristics of acute lymphoblastic leukaemia survivors and Wilms' tumour survivors – results in years, median (25Q–75Q)

Parameters	ALLs ($n = 37$)	WTs ($n = 11$)	p
Sex (F/M)	14/23	6/5	0.324
Age at diagnosis	3 (2.6–4.78)	2 (1.43–2.77)	0.081
Age at time of study	14 (12–15)	14 (10–16)	0.753
Time from the end of treatment	8 (6–9)	10 (6–13)	0.188

ALLs – acute lymphoblastic leukaemia survivors, WTs – Wilms' tumour survivors

TABLE 2. Comparison of established risk factors for cardiovascular diseases in acute lymphoblastic leukaemia survivors and Wilms' tumour survivors (median, 25Q–75Q)

Parameters	ALLs ($n = 37$)	WTs ($n = 11$)	p
Central obesity	12	2	0.361
Weight [kg]	64.6 (52.5–69.5)	48.2 (40.7–62.7)	0.024*
SDS of the weight	0.464 (0.088–1.286)	0.149 (–0.479–1.168)	0.263
Height [cm]	166 (154–177)	160 (154–169)	0.243
SDS of the height	0.626 (–0.287–1.420)	0.711 (0.153–1.738)	0.847
BMI [kg/m^2]	21.7 (18.7–24.0)	19.0 (17.0–21.8)	0.065
SDS of the BMI	0.607 (–0.206–1.298)	–0.173 (–0.672–1.350)	0.123
Waist circumference [cm]	74.0 (67.0–81.0)	67.0 (64.0–71.5)	0.019*

ALLs – acute lymphoblastic leukaemia survivors, BMI – body mass index, SDS – standard deviation score, WTs – Wilms' tumour survivors
** statistically significant differences*

TABLE 3. Comparison of blood pressure in acute lymphoblastic leukaemia survivors and Wilms' tumour survivors (median, 25Q–75Q)

Parameters	ALLs ($n = 37$)	WTs ($n = 11$)	p
Systolic BP [mm Hg]	117 (113–122)	111 (111–124)	0.263
Pc of the systolic BP	71 (41–81)	56 (43–88)	0.981
Diastolic BP [mm Hg]	75 (72–78)	75 (74–81)	0.594
Pc of the diastolic BP	94 (85–97)	96 (89–99)	0.285

ALLs – acute lymphoblastic leukaemia survivors, BP – blood pressure, WTs – Wilms' tumour survivors

TABLE 4. Comparison of biochemical parameters in acute lymphoblastic leukaemia survivors and Wilms' tumour survivors (median, 25Q–75Q)

Parameters	ALLs ($n = 37$)	WTs ($n = 11$)	p
Salusin β [pg/ml]	82.1 (48.5–198)	114.4 (70.2–224.2)	0.576
Total cholesterol [mg/dl]	154 (137–177)	151 (132–167)	0.722
LDL [mg/dl]	90 (74–108)	83 (64–97)	0.426
HDL [mg/dl]	50 (44–58)	56 (51–58)	0.373
TG [mg/dl]	58 (48–85)	67 (60–83)	0.320
Creatinine-based eGFR [ml/min/1.73 m^2][1]	111 (98–129)	106 (94–114)	0.164
Creatinine and BUN-based eGFR [ml/min/1.73 m^2][2]	92 (85–100)	89 (85–89)	0.114
Cystatin C [mg/dl]	0.77 (0.7–0.81)	0.85 (0.73–1.11)	0.042*
Cystatin-based eGFR [ml/min/1.73 m^2][3]	123 (116–137)	110 (82–131)	0.042*

ALLs – acute lymphoblastic leukaemia survivors, WTs – Wilms' tumour survivors
[1]eGFR based on the revised Schwartz equation
[2]eGFR based on the creatinine and BUN equation
[3]eGFR based on the Filler equation
**statistically significant differences*

of the ALLs were significantly greater than those of the WTs (Table 2). Central obesity was observed in 3 out of 9 patients with ALL and treated with cranial radiotherapy. No patients met the criteria for metabolic syndrome.

We found no significant differences in either systolic or diastolic BP or in the serum levels of total cholesterol, LDL cholesterol, HDL cholesterol and triglycerides between the two groups (Tables 3, 4).

We assessed BP in both groups of survivors. We detected BP ≥ 95 pc in 4 (36%) WTs and in 14 (37.8%) ALLs. We did not observe a difference in the prevalence of BP ≥ 95 pc between the groups ($p = 0.192$).

The median serum salusin β concentration (pg/ml) was higher in the WT group than in the ALL group, but the difference was not statistically significant. The serum level of cystatin C was significantly higher, and the cystatin-based eGFR was significantly lower, in the WT group than in the ALL group (Table 4). This type of difference was not observed for creatinine.

The correlations between salusin β and BP, weight, SDS of weight, body mass index (BMI), SDS of BMI, waist circumference, glucose, lipids, cystatin C and eGFR were assessed in all the survivors together and in the ALL group. A correlation between salusin β and LDL was found in the ALLs ($r = 0.35$, $p = 0.044$) (Table 5). The correlation in WTs was not assessed because of the small size of the group.

DISCUSSION

Recent studies of Polish paediatric and adolescent cancer survivors revealed a high incidence of cardiovascular problems. The assessment of the health status of Polish children and adolescents after cancer treatment has shown that cardiovascular problems were observed in 31.7% of the whole group, and obesity or short stature was present in 21.4% of all survivors. A higher frequency of circulatory system problems was observed in males than in females ($p = 0.029$), in children diagnosed within the ages of 1–4 years,5–9 years, 10–14 years, and > 15 years than in children diagnosed in infancy ($p < 0.0001$), and in groups of patients 5–10 and 11–15 years after treatment completion than in groups of patients with < 2 years of follow-up ($p < 0.0001$). Thirty-eight percent of patients who underwent treatment for ALL presented symptoms or complaints that suggested circulatory system problems in contrast to 26.6% of patients who underwent treatment for WT. Symptoms such as short stature and obesity were present in 23.7% and 13% of ALL and WT survivors, respectively [18].

The study by Ociepa *et al.* reported a prevalence of hypertension among ALL survivors of 37% [19].

These facts have been confirmed by numerous other national studies among older populations of survivors [1, 2]. The results of these studies justify the search for new indicators of cardiovascular diseases.

Salusins have recently been identified as endogenous bioactive peptides that have hypotensive and bradycardiac impacts. Salusins are synthesized and present in many tissues of the human body. Salusin α seems to suppress the formation of macrophage foam cells and the development of atherosclerosis. The concentration of salusin α is decreased in patients with conditions leading to atherosclerosis compared to that in healthy individuals. Salusin β influences BP and heart rate through parasympathetic stimulation and negative inotropism. The central action of salusin β is the regulation of fluid balance. The peripheral effect is potentially atherosclerotic. Elevated concentrations of salusin β probably mediate acute renal dysfunction induced by cisplatin and sepsis [20]. Research performed by Kołakowska *et al.* revealed a higher level of serum salusin β among patients with hypertension than in the reference group (subjects diagnosed with white-coat hypertension). This finding may confirm the important role of salusin β in the pathogenesis of hy-

TABLE 5. Spearman correlation

All patients	*N*	*R*	*p*
Salusin β & pc of systolic BP	48	0.06	0.690
Salusin β & pc of diastolic BP	48	0.11	0.461
Salusin β & SDS of weight	48	–0.14	0.326
Salusin β & SDS of BMI	48	–0.04	0.792
Salusin β & pc of waist circumference	48	0.16	0.271
Salusin β & total cholesterol	46	0.07	0.630
Salusin β & LDL	45	0.18	0.235
Salusin β & HDL	46	–0.05	0.765
Salusin β & TG	46	–0.11	0.450
Salusin β & glucose level	46	–0.10	0.489
Salusin β & creatinine	48	–0.11	0.456
Salusin β & creatinine-based eGFR[1]	48	0.06	0.682
Salusin β & creatinine and BUN-based eGFR[2]	46	–0.04	0.804
Salusin β & cystatin C	47	0.15	0.300
Salusin β & cystatin-based eGFR[3]	47	–0.15	0.300
ALLs			
Salusin β & pc of systolic BP	37	0.04	0.812
Salusin β & pc of diastolic BP	37	0.00	0.997
Salusin β & SDS of weight	37	–0.11	0.521
Salusin β & SDS of BMI	37	–0.01	0.953
Salusin β & pc of waist circumference	37	0.16	0.346
Salusin β & total cholesterol	35	0.19	0.273
Salusin β & LDL	34	0.35	0.044*
Salusin β & HDL	35	0.02	0.923
Salusin β & TG	35	–0.07	0.675
Salusin β & glucose level	35	–0.11	0.537
Salusin β & creatinine	37	–0.03	0.861
Salusin β & creatinine-based eGFR[1]	37	0.00	0.978
Salusin β & creatinine and BUN-based eGFR[2]	36	–0.07	0.666
Salusin β & cystatin C	36	0.14	0.409
Salusin β & cystatin-based eGFR[3]	36	–0.14	0.409

ALLs – acute lymphoblastic leukaemia survivors, BP – blood pressure, SDS – standard deviation score
[1]eGFR based on the revised Schwartz equation
[2]eGFR based on the creatinine and BUN equation
[3]eGFR based on the Filler equation,
** statistically significant differences*

pertension [21]. Increases in serum salusin β levels have been observed in patients with coronary artery disease and diseases that lead to cardiovascular disorders [8, 22]. Elevated serum salusin β levels were observed in children with primary hypertension and were positively correlated with serum triglyceride levels, triglyceride/HDL-cholesterol ratio, hs-CRP and ADMA [23, 24]. Thus, salusin β seems to be a useful indicator of developing CVD.

Overexpression of salusin β may also facilitate the development of chronic renal failure, but it has not yet been confirmed.

In our study, the median serum salusin β concentration was higher in the WT group, but the difference was not statistically significant. We found that both groups significantly differ in weight, waist circumference and renal function. Such results were confirmed by previous studies. Poor renal function in WTs was also observed in our previous studies [25, 26].

In ALLs we found a correlation between LDL and salusin β. The group of patients has higher risk of metabolic and lipid disorders; it was not revealed in our study, probably because of the young age of the participants. However salusin β as an indicator of atherosclerosis may be a predictor of CVD in ALL survivors. This information emphasizes the need for follow-up in adulthood.

The lack of a significant difference in levels of salusin β between groups might have been influenced by the low number of enrolled patients, which was not high enough to reach definite conclusions. In particular, the group of WT survivors was markedly small. The young age, relatively short time from the end of treatment and variety of treatments could also play important roles. These factors are some limitations of our study, especially the relatively small study population and lack of a control group. The fact that the patients were not diagnosed with metabolic syndrome may also be relevant. Atherosclerosis develops gradually and is exacerbated in middle age. Obesity, hyperlipidaemia, hypertension and insulin resistance significantly accelerate the development of CVD. Thus, further studies need to be performed to determine whether the concentration of salusin β correlates with the development of endothelial injury and atherosclerosis in survivors of childhood cancers.

CONCLUSIONS

Many types of long-term side effects are observed among survivors of paediatric cancers. Patients treated for ALL and WT differ in terms of the types of side effects that they experience.

Acute lymphoblastic leukaemia survivors more often develop obesity and metabolic problems, whereas WT survivors tend to develop chronic kidney disease.

Salusin β levels are correlated with LDL levels in ALLs ($r = 0.35$, $p = 0.044$); this correlation can indicate that lipid disorders and salusin β levels are related in patients with a higher risk of obesity and suggests that salusin β could be a predictor of atherosclerosis.

However, higher levels of salusin β were found in WT survivors, which might be associated with worse renal function.

Further investigations are necessary to confirm the results.

It is necessary to continue follow-up of adults who were treated for childhood cancers to reveal long-term side effects, such as cardiovascular disorders.

DISCLOSURE

The authors declare no conflict of interest.

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