Obesity and Systolic Blood Pressure in Young Adult Men Born Small for Gestational Age

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A B S T R A C T

Individuals born small for gestational age (SGA) are supposed to be at higher risk to develop cardiovascular disorders, and recent report showed that concurrent obesity influences blood pressure (BP) in SGA children. Our aim was to investigate the impact of obesity and birth weight on blood pressure values in young adult men born SGA and controls born after normal pregnancy, Normotensive, non-treated adult men were enrolled (N=185; mean age 21.29 ± 0.9 years). Birth parameters were obtained from medical records and SGA was defined as birth weight (BW) under 10th percentile for gestational age and obesity as BMI >25 kg/m^2. According to the presence or absence of obesity and BW the subjects were divided into four groups: (1) non-obese with normal BW (N=50), (2) non-obese SGA (N=67), (3) obese with normal BW (N=40), (4) obese SGA (N=28). BP was measured using Omron M6 and Spacelab 90207 device following the ESH/ESC guidelines. Systolic BP, 24-hour BP variability and pulse pressure were significantly higher in SGA subjects than in those with normal BW ($p<0.05$). The highest 24-hour and daytime systolic BP values as well as 24-hour pulse pressure were found in the subgroup of obese SGA subjects ($p<0.001$). Significant differences for the above parameters were observed between obese SGA group and non-obese SGA group ($p<0.05$). Obese SGA subjects had higher 24-hour and daytime systolic BP values compared to obese normal BW group. No difference was found in BP between non-obese SGA and non-obese group with normal BW ($p>0.05$). In addition to BW and shorter pregnancy duration, obesity concurrently and significantly determines systolic BP in young normotensive men and point to a need for more agressive implementation of healthy lifestyle as early as possible.

Key words: *birth weight, small for gestational age, hypertension, obesity, young adult men*

Introduction

Cardiovascular diseases (CV) are the leading cause of mortality worlwide with arterial hypertension (AH) being the main independent risk factor[1,2]. The importance of identifying CV risk factors represents major issue in health care. Traditional major risk factors could not completely explain overall CV risk, thus the interest for so called non-traditional risk factors is increasing. Twenty years ago Barker hypothesised that altered fetal development which results in lower birth weight contributes to increased risk of CV diseases in later life particularly in small for gestational age (SGA) newborns[3–6]. Epidemiological studies have shown that intrauterine growth retardation resulting in small for gestational age individuals is a risk factor for development of AH[7–9]. Recent report on impact of obesity on blood pressure (BP) in children born after intrauterine growth retardation suggests that obesity might play crucial and detrimental role in that continiuum[10].

Whether intrauterine growth retardation itself or only in conjuction with predisposing factor results in higher blood pressure in adolescent age remains unsolved. The aim of this study was to investigate the role of concurrent obesity on BP in young, adult men born small for gestational age.

Materials and Methods

Study was performed in Outpatient clinic of Department of Nephrology, Arterial Hypertension, Dialysis and

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TABLE 1
BASIC CHARACTERISTICS OF THE STUDY GROUPS DIVIDED ACCORDING OBESITY STATUS AND BIRTH PARAMETERS

Variables	Nonobese Normal BW N=50	Nonobese SGA N=67	Obese Normal BW N=40	Obese SGA N=28	p
Age (years)	22 (20–23)	21 (20–23) *‡	21 (20–23)	21 (20–22) *‡	0.001
Weight (kg)	78 (57–96)	71 (47–90) *‡	89 (75–118) *	89 (68–118) *‡	< 0.001
Height (cm)	183.5 (160–200)	176 (160–190) *‡	181 (171–198)	180 (156–190) *	< 0.001
BMI (kg/m^2)	22.9 (17.6–25.5)	22.8 (16.7–25.1)	26.6 (25.7–33.5) *†	27.8 (25.7–35.2) *†	< 0.001
Waist circumference (cm)	80 (70–92)	79 (66–90)	86 (81–105) *†	92 (78–108) *†	< 0.001
Birth weight (g)	3656 (2800–4750)	2330 (1300–2850) *‡	3650 (2630–4700)	2150 (1400–2800) *‡	< 0.001
Birth lenght (cm)	52 (49–56)	46 (40–51) *‡	52 (48–56)	45 (41–49) *‡	< 0.001
Gestation duration (week)	39 (37–42)	37.5 (28–40) *‡	40 (37–42)	36 (30–40) *‡	< 0.001

Median (min–max)
*significant difference ($p<0.05$) with the nonobese normal bw; † significant difference ($p<0.05$) with the nonobese sga; ‡ significant difference ($p<0.05$) with the obese normal bw;] significant difference ($p<0.05$) with the obese sga

Transplantation at University Hospital Center Zagreb, School of Medicine, University of Zagreb. Power analysis was performed showing that minimal number of subjects included should be 115 based on following parameters: z test – logistic regression, tail(s)=Two; odds ratio=1.8; Pr(Y=1|X=1) H0=0.35; α error prob=0.05; power (1-β error prob)=0.80; critical z=1.9599640; actual power= 0.8009351. Analyses were conducted using G*Power for Windows ver 3.1.2. In this study we enrolled 185 young, healthy, normotensive non-treated adult men (mean age 21.29 ± 0.9 years). Participants were selected from medical records of all male newborns born in the city of Zagreb beetwen 1987 and 1990. We found 453 males born SGA. We also randomly selected a 500 men born after normal, on term pregnancies. A letter of invitation was sent for participation in the study to last known address of mothers. After exclusion of non responders due to wrong address, refusal of participation or health conditions, a 95 men (mean age 21.0 ± 0.89 years) were enrolled in SGA group (defined as being born under 10th percentile of birth weight for gestational age according to reference tables of birth weight and gestational duration for Croatian population (taking into account parity of the mother)[11] and 90 men (mean age 21.5+1.02 years) were enrolled in the control goup appropriate for gestational age born participants (AGA). Inclusion criteria for the control group were: birth weight (BW) > 2500 g and pregnancy duration of 37–42 weeks. Obesity was defined as body mass index (BMI) higher than 25 kg/m^2. Non-responders did not significantly differ from responders in terms of birth weight and gestational age. All subjects without history of hypertension, kidney disease or diabetes mellitus who were willing to participate were enrolled in the study and writen informed consent was obtained. All subjects were neighter taking any medication nor had any clinically manifested illness. According to the presence or absence of obesity and according to the BW subjects were divided into four groups: (1) non-obese with normal BW (N=50), (2) obese with normal BW (N=40), (3) non-obese SGA (N=67), (4) obese SGA (N=28). Office BP was measured using Omron M6 and ambulatory blood pressure monitoring (ABPM) was done with Spacelab 90207 devices following the ESH/ESC guidelines[12], and the average 24-hour, daytime and nighttime systolic and diastolic BP values, blood pressure load, BP variability (estimated as standard deviation), heart rate (HR) and pulse pressure values were analysed.

Data distribution of numerical variables were analysed by Kolmogorov-Smirnov test and data were presented with means and standard deviations if distribution of data was normal. In the case of distribution other than normal, data were presented as medians with minimum and maximum range. Nominal variables were presented as absolute number and percentage.

Due to the number of participants in each study group, nonparametric statistical tests were used. Kruskal-Wallis test was used to explore the differences between more than 2 independent groups with post-hoc analysis by Mann-Whitney U test. Correlations were assesed by Kendall tau correlation coefficient, while independent in-

fluence on BP parameters of statisticaly significant correlations were tested by multiple linear regression analysis. Statistical significance was assessed assuming a type I error of 5%. All statistical analyses were conducted using the STATISTICA package version 10.

Results

The anthropometric characteristics of participants are shown in Table 1. According to clasification of the groups, difference was observed in gestational parameters and status of obesity. Small for gestational age subjects that became obese during their life had smallest body proportions at birth. However, there was no difference in body proportions between non-obese and obese subjects born small for gestational age (p=0.080). No differences were observed between groups in smoking status, family history of hypertension, physical activity or income (Table 2.).

Systolic office BP, 24-hour BP variability and pulse pressure were significantly higher in subjects who were born small for gestational age than in those with normal birth weight (p<0.05).

BP parameters divided by groups are presented in Table 3. Obese subjects born small for gestational age had higher office diastolic, 24-hour and daytime systolic BP values as well as 24-hour and daytime pulse pressure compared to non-obese subjects born small for gestational age (all p<0.001). Interestingly, non-obese subjects born small for gestational age had lowest values of office and ambulatory parameters of BP. Analysis between obese subjects born small for gestational age and obese subjects born appropriate for gestational age revealed higher 24-hour and daytime systolic BP values in obese ones born small for gestational age (all p<0.001). Small for gestational age subjects had higher heart rate than those with normal BW, and difference was statistically significant between non-obese groups (p=0.007). There were no difference in office, 24-hour daytime and nighttime systolic BP values between non-obese SGA and non-obese AGA (all p >0.05).

We failed to find correlation between low BW and BMI (r=0.107; p=0.157), neither between gestational duration and BMI (r=0.050; p=0.543).

In the whole group BMI positively correlated with office systolic BP (r= 0.30; p<0.001), 24-hour and daytime systolic BP (r=0.288; p<0.001; r=0.345; p<0.001 respectively), as well as 24-hour pulse pressure (r=0.310; p<0.001), whereas gestational duration negatively correlated with daytime systolic BP (r= –0.277; p=0.009).

Accordingly, these parameters that significantly correlated with BP parameters were tested by multiple linear regression analysis. In multiple linear reggresion analysis in the whole group BMI independently and positively influences office, 24-hour, daytime and nighttime systolic BP, and both BMI and gestational duration were independently associated with daytime systolic BP (Table 4.). BMI positively and gestational duration negatively influences daytime systolic BP. In small for gestational age group, BMI independently and positively influences office systolic and daytime systolic BP (R^2=0.182, B=1.252, p=0.002; R^2=0.090, B=0.728, p=0.042, respectively).

Discussion

Intrauterine growth retardation in the small for gestational age newborns leads to lower nephron number and altered vascular structure, thus predisposing future cardiovascular disorders[13–15]. As atherosclerosis begins in childhood[16], all effors should be done to modificate risk factors. Although intrauterine growth retardation predispose development of AH later in life[8,9,17–19], it seems that obesity accelerates this contributing to higher BP values in young adulthood.

In our group of young adult men we found that obesity plays an important contributing role in manifesting propensity for increased blood pressure values. Small for gestational age subjects who are obese in young adulthood have the highest risk for higher BP later in life.

Our results indicate that BMI have an important effect on daytime systolic BP in young, adult men born small for gestational age.

TABLE 2
SOCIAL CHARACTERISTICS AND HABITS OF THE STUDY GROUPS DIVIDED ACCORDING OBESITY STATUS AND BIRTH PARAMETERS

Variables	Nonobese Normal BW N=50	Nonobese SGA N=67	Obese Normal BW N=40	Obese SGA N=28	p
Smoking status (yes)*	3 (7.5)	13 (20.6)	2 (6.3)	6 (26.1)	0.060
Family history of hypertension (yes)*	12 (31.6)	29 (46.8)	13 (40.6)	12 (54.5)	0.301
Income *					
Low	0 (0)	1 (1.6)	0 (0)	0 (0)	0.936
Average	33 (91.7)	57 (91.9)	29 (93.5)	17 (89.5)	
High	3 (8.3)	4 (6.5)	2 (6.5)	2 (10.5)	
Physical activity (yes)	29 (82.9)	42 (70.0)	27 (79.4)	15 (65.2)	0.336

*N (%)

TABLE 3

OFFICE AND AMBULATORY BP PARAMETERS IN SUBJECTS DIVIDED ACCORDING TO OBESITY STATUS AND BIRTH PARAMETERS

Variables	Nonobese Normal BW N=50	Nonobese SGA N=67	Obese Normal BW N=40	Obese SGA N=28	p
Office BP					
Systolic BP, mmHg	125.5 (110–146)	123 (103–149)	130 (112–144)	130 (108–170)	0.104
Diastolic BP, mmHg	80 (70–90)	80 (63–96)	80 (70–90) †	80 (70–116) †	0.029
PP, mmHg	45.5 (32–60)	47 (30–67)	48 (34–60)	46 (30–74)	0.543
HR, bpm	62 (42–100)	70 (44–116) *	68 (47–89)	71 (51–92) *	0.008
24-hour ambulatory BP					
Systolic BP, mmHg	121 (106–139)	119.5 (107–146)	122.5 (112–137)	127.5 (109–181) *†‡	0.004
Diastolic BP, mmHg	71 (60–80)	67 (52–89)	68.5 (57–82)	70.5 (59–97)	0.072
PP, mmHg	51 (38–67)	53 (39–77)	55 (40–68) *	56 (46–84) *†	0.005
HR, bpm	69 (48–74)	71 (49–95)	70.5 (56–86)	71.5 (50–103)	0.627
Daytime amulatory BP					
Systolic BP, mmHg	126 (108–143)	122.5 (109–147)	126 (114–141)	133 (105–153) *†‡	0.001
Diastolic BP, mmHg	73 (61–90)	71.5 (52–92)	71.5 (59–88)	73 (59–87)	0.060
PP, mmHg	50.5 (30–67)	52 (38–83)	55 (41–66) *†	56.5 (46–75) *†	0.002
HR, bpm	73 (51–111)	76 (51–104)	74.5 (59–94)	75.5 (55–105)	0.377
Nighttime ambulatory BP					
Systolic BP, mmHg	117 (101–139)	114 (103–143)	116 (104–140)	121 (109–138)	0.056
Diastolic BP, mmHg	66 (55–78)	61 (47–85) *	63 (52–90)	64 (49–86)	0.048
PP, mmHg	52 (36–65)	54 (39–68)	54 (38–71)	56.5 (39–73)	0.067
HR, bpm	63 (45–80)	63 (44–87)	62 (49–88)	64 (42–96)	0.974

Median (min–max)

*significant difference ($p<0.05$) with the nonobese normal bw; † significant difference ($p<0.05$) with the nonobese sga; ‡ significant difference ($p<0.05$) with the obese normal bw;] significant difference ($p<0.05$) with the obese sga

These results are in concordance with Lurbe et al. who demonstrated that both, low BW and obesity, when adjused by age and sex, have independent effect on BP phenotype in children[10].

In our group those who became obese in young adulthood had smallest body proportions at birth. It could be speculated whether rapid postnatal catch up growth is responsible for development of obesity later in life as reported by other authors[20,21]. In children born appropriate for gestational age, those in upper percentile of weight gain have higher risk for development of obesity and higher BP later in life[22]. As shown by Vohr et al.[23] this effect is further amplified in adolescents born before 32nd week of gestation whose main predictors of increased BP were early and rapid weight gain and male gender. Moreover, in adolescent age those born before 32nd week of gestation are more likely to be obese than in terms controls[23].

Numerous epidemiological studies analyzed relationship between low birth weight and AH[7–9,18,] and as low BW can result either from IUGR or preterm birth or from both, many efforts were undertaken to elicit which determinant plays important role for future cardiovascular risk[19,24].

Leon et al. showed that systolic BP is independently and inversely associated with lower birth weight and ges-

TABLE 4
REGRESSION MODEL TO ASSES FACTORS INDEPENDENTLY ASSOCIATED WITH SYSTOLIC BP IN THE WHOLE GROUP

Variables	B	SE (B)	p	R^2
Office systolic BP				0.113
BMI	1.146	0,287	<0.001	
Birth weight	0.002	0.002	0.300	
Gestation duration	–0.711	0.516	0.170	
24-hour systolic BP				0.124
BMI	0.914	0.242	<0.001	
Birth weight	0.001	0.001	0.668	
Gestation duration	–0.707	0.419	0.094	
Daytime systolic BP				0.208
BMI	1.053	0.218	<0.001	
Birth weight	0.001	0.001	0.369	
Gestation duration	–1.080	0.378	0.005	
Nighttime systolic BP				0.075
BMI	0.619	0.225	0.007	
Birth weight	0.001	0.001	0.609	
Gestation duration	–0.553	0.390	0.158	

tational duration[24]. They reported that extension of gestation for 1 week leads to decrease of systolic BP of – 0,25 mmHg[24] what is in line with our results. Also, average increase of 1 kilogram of BW leads to decrease of systolic BP for approximately 2 mmHg[8,9]. Likewise, Law et al. demonstrated that intrauterine growth retardation is associated with higher systolic BP in young adults[21]. Nevertheless, Dalziel et al.[24] found that preterm birth, rather than intrauterine growth retardation, influences BP values in inverse order.

We found that BMI is important predictor of the BP values. Keijzer-Veen et al.[25] found higher prevalence of AH in intrauterine growth retardation and preterm subjects. They also found that current BMI was independently associated with BP values[25]. Huxley et al. in their meta-analysis debated that non-adjustment for potential confounders could result in inverse correlation of intrauterine growth retardation and BP values[26]. Nevertheless, Gamborg and co-workers in meta-analysis of 20 Nordic cohorts found that inverse association between BW and systolic BP existed despite the adjustment for concurrent body mass index[27].

The role of obesity in development of AH is well established [28,29] and it seems that in this particularly vulnerable population has detrimental, accelerating effect on BP levels.

Heart rate is a surogat marker of increased sympathetic activity[30]. While animal studies showed increased sympathetic activity in inviroment of intrauterine retardation[31], results of human studies on adult subjects born small for gestational age are contradictory[32,33]. In our study, observed difference in heart rate between non-obese subjects born small with those born appropriate for gestational age suggests that in non-obese subjects born small for gestational age increased sympathetic activity could play important pathophysiological role in future development of hypertension.

We failed to find difference in heart rate between obese subject born small for gestational age and obese subjects with normal birth weight. This could indicate that simpathetic activity is generally upregulated in obesity. Higher BP parameters in obese subjects born small for gestational age compared to obese subjects born appropriate for gestational age suggests that hypertension might be mediated by different mechanisms.

Increased sympathetic activity in subjects born after intrauterine growth retardation alters kidney function by increasing renin synthesis and leads to retention of sodium[17]. However, this is probably just one arm of the vicious circle because even slight kidney impairment due to lower nephron number could increase afferent sympathetic activity from kidney to the brain[34]. Several limitations of the study should be bare in mind: the cross-sectional study cannot show causality, relatively small number of participants and potential selection bias of paricipants because obviously more educated and more health-interested individuals consented to paticipate and the others did not. We investigated male subjects and this fact is both limitation and strength because data can be extrapolated on one gender. Longitudinal prospective follow-up of this group of subjects and involvement of female subjects are plans for further investigations.

Conclusion

In addition to birth weight and shorter pregnancy duration, obesity significantly determines systolic BP in young adult men born small for gestational age and may contribute to the early signs of vascular ageing. Our findings are in agreement with the results obtained in children and point out the need for agressive implementation of healthy lifestyle as early as possible in this population subset particularly prone to development of hypertension.

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PRETILOST I ARTERIJSKI TLAK U MLADIH ODRASLIH MUŠKARACA ROĐENIH NAKON INTRAUTERINOG ZASTOJA U RASTU

S A Ž E T A K

Predmnijeva se da su osobe rođene s manjom porodnom težinom u odnosu na gestacijsku dob (SGA) pod većim rizikom za razvoj kardiovaskularnih bolesti (KV), a nedavni izvještaji ukazuju da pretilost u SGA djece utječe na vrijednosti arterijskog tlaka (AT). Ispitat ćemo utjecaj pretilosti i porođajne težine na vrijednosti arterijskog tlaka u zdravih, normotenzivnih, odraslih muškaraca (N=185; srednja dob 21.29 ± 0.9 godina). Podaci o trudnoći i porodu dobiveni su iz registara poroda. SGA je definirana porođajnom težinom (PT) ispod 10. percentile za gestacijsku dob, a pretilost BMI >25 kg/m^2. Prema porođajnoj težini i statusu pretilosti ispitanici su podijeljeni u četiri skupine: (1) ne-pretili normalne PT (N=50), (2) ne pretili SGA (N=67), (3) pretili normalne PT (N=40), (4) pretili SGA (N=28). AT je mjeren pomoću Omron M6 i Spacelab 90207 uređaja prema ESH/ESC smjernicama. Analizirane su srednje vrijednosti 24-satnog, dnevnog, noćnog sistoličkog i dijastoličkog AT, opterećenje AT, varijabilnost AT (procijenjena preko standardne devijacije), srčana frekvencija i tlak pulsa. Sistolički AT, 24-satna varijabilnost AT i tlak pulsa su bili značajno viši u SGA ispitanika nego s normalnom PT ($p<0,05$). Skupina SGA pretilih ispitanika imala je najviše vrijednosti ambulantnog, 24-satnog i dnevnog sistoličkog AT, kao i 24-satnog tlak pulsa. Značajne razlike u gore navedenim parametrima su uočene između pretilih i nepretilih SGA ispitanika ($p<0,05$). Pretili SGA ispitanici su imali više vrijednosti 24-satnog i dnevnog sistoličkog AT u odnosu na pretile ispitanike s normalnom PT ($p<0,05$). Nije bilo razlike u vrijednostima AT između nepretilih ispitanika rođenih SGA i nepretilih s normalnom PT ($p>0.05$). Uz intruterini zastoj u rastu, pretilost dodatno i značajno određuje vrijednost sistoličkog AT u mladih normotenzivnih muškaraca i doprinosi ranom vaskularnom starenju. Stoga je u osoba s anamnezom intrauterinog zastoja u rastu uvođenje zdravih životnih navika i prevencija pretilosti ključna.