RESEARCH PAPER

Components of Metabolic Syndrome at 22 years of Age – Findings From Pune Low Birth Weight Study

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Objective: To study the early markers of Metabolic syndrome in a cohort of low birth weight (LBW) children followed up since birth, at the age of 22 years.

Design: Prospective cohort study.

Setting: Tertiary-care hospital

Participants: Neonates weighing less than 2000 g discharged from a neonatal special care unit were followed up prospectively; 153 cases and 77 controls were assessed at 22 years of age.

Methods: Fasting, 30 minute and 120 minute glucose and insulin after a bolus of 75g of glucose was determined. Insulin resistance was calculated. A lipid profile was also done. Anthropometric measurements were taken and abdominal fat was determined by magnetic resonance imaging.

Main outcome: Prevalence of the five components of Metabolic Syndrome as described by the International Diabetic Federation (IDF).

Results: 65.1% of the cohort was born small for gestational age. All three components of Metabolic syndrome were present in only three cases and none of the controls. However, two components were present in 25 (16.4%) cases and 5 (6%) controls (*P*=0.039). Cases in the lowest quartile of birthweight who became big at 22 years had significantly higher fasting insulin (*P*=0.001), Homeostatic Model Assessment – Insulin Resistance (Homa-IR) (*P*=0.001) and higher systolic blood pressure. Sum of skinfold thickness at 4 sites correlated significantly with fasting insulin and HOMA-IR, and was a stronger correlate compared to BMI, waist circumference and MRI fat. There was no difference in the biochemical parameters between appropriate for gestational age and small for gestational age infants.

Conclusion: Prevelence of three or more components of Metabolic syndrome was low in LBW children at 22 years, but of two components was high. Those 'Small at birth and big at 22 years' had high insulin resistance.

Keywords: *Hypertension, Insulin resistance, Outcome, Triglycerides.*

India is experiencing an epidemic of type II diabetes and coronary heart disease (CHD) in young adults and middle-aged population [1]. The cluster of risk factors of CHD, type II diabetes, hypertension and central obesity is known as the Metabolic syndrome. Nutritional deprivation of the fetus during critical periods of development leads to adaptive survival strategies [2], leading to increased susceptibility to adult-onset coronary heart disease and type II diabetes [3,4]. We have been following a cohort of low birth weight (<2000 g) along with normal birth weight controls since their birth for the past 22 years - "Pune low birth weight study – birth to adulthood" [5,6]. In this study, we report the occurrence of various components of metabolic syndrome in this cohort.

Methods

The cohort consisted of infants weighing <2000 g, discharged from a neonatal special care unit between October 1987 to April 1989, and followed up prospectively till the age of 18 years [5]. The LBW infants were classified into appropriate for gestational age (AGA) or small for gestational age (SGA) using the criteria of Singh, *et al*. [7]. Full term neonates born in the same hospital during the same period with birth weight ≥2500g with a normal antenatal, natal and postnatal course, matched for socio-economic status were enrolled as controls. The parents were offered a free check-up as an added incentive. Ethical permission was obtained from the hospital's Ethics Committee. Written informed consent of both the parents and subjects was obtained at the time of enrolment in the 22 year study.

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Infants and parents were examined by the medical officer and questioned regarding any major illness in the past and recent medical problems. Blood pressure was measured with a standard sphygmomanometer by the medical officer. The mean of three readings of systolic and diastolic pressure was recorded. Hypertension was

defined as systolic pressure above 130 mm Hg, and diastolic pressure above 85 mmHg. After an overnight fast, oral Glucose tolerance test was done. A fasting blood sample was collected, and 30 and 120 minute samples were collected after glucose administration. Lipid profile, consisting of total cholesterol, LDL, VLDL, HDL and triglycerides, was measured (enzymatic method). Insulin was measured by Delfia technique and ELISA. Homeostatic Model Assessment – Insulin Resistance (Homa-IR) was calculated using the online Oxford model (*http://www.dtu.ox.ac.uk*).

Weight was measured by an electronic scale with an accuracy of ±10g. Standing and sitting height was measured to the nearest 0.5 cm by a wall-mounted stadiometer using the standard technique, described by Tanner [8]. BMI was calculated and categorized. Waist circumference was measured by a non-stretchable tape to the nearest 0.1 cm midway between the lower costal margin and superior iliac crest in expiration. Hip circumference was measured at the point of maximum protuberance. Skin fold thickness was measured at 4 sites – biceps, triceps, subscapular and suprailiac, using Harpender's caliper. Dietary intake was assessed by 24-hour diet recall (weekday and weekend) and validated food frequency questionnaire [9]. Physical activity was assessed by using a questionnaire and BMR; physical activity level (PAL) was calculated using physical activity ratio (PAR) values of each activity in 24 hours [10].

Magnetic resonance imaging (MRI) of abdomen was performed on a 1.5 Tesla unit (GE 16 Channel HD, T, USA). Axial T_1, weighted spoiled gradient echo sequence without fat suppression was used to scan the patients from xiphisternum to pubic symphysis (Repetition time 100 msec, Echo time – 1.2 msec, slice thickness – 10 mm, interval 1 mms). Two markings were done at each axial, one on the outer edge of abdominal wall muscles and second at the outermost edge of subcutaneous fat. Values of inner marking were subtracted from that of the outer marking to derive the subcutaneous fat content in the abdominal wall. All these values were added and divided by the number of sections from xiphisternum to pubic symphysis [11].

Female participants were questioned regarding menstruation history and present status, and were examined for hirsutism. Pelvic ultrasonography was done (Aloka SSB 3500 machine) with a 2–5 megahertz convex sector on the 3rd to 5th day of menstruation. Polycystic ovarian disease was diagnosed if there were bilateral enlarged ovaries with increased stromal echogenicity with multiple peripheral follicles. Polycysitc ovary syndrome was diagnosed if there was evidence of hyper-androgenism and ovarian dysfunction, menstural cycle >35 days and/or polycystic ovaries [12].

The Internataional Diabetic Federation [13] lists five components of the metabolic syndrome for Asian population. A diagnosis of metabolic syndrome can be made if three components out of the five are present: (*i*) Waist circumference ≥90 cm in men and ≥80 cm in women; (*ii*) Serum triglycerides ≥150 mg/dL (1.7 mmol/L); (*iii*) Fasting glucose ≥110mg/dL (6.1 mmol/L) (*iv*) Blood pressure ≥130/80 mmHg; and (*v*) High density lipoprotein <40 mg/dL (1.03 mmol/L) in men and <50 mg/dL (1.29 mmol/L) in women.

Statistical analysis: For the variables not normally distributed, appropriate transformations for the underlying normality assumption are used. The linear association between the normally distributed variables was assessed by Pearson's correlation coefficients; otherwise Spearman's correlation coefficients were used. The partial correlation analysis was also used to test the independent associations between several variables of interest. The factors such as sex, socio-economic status and current BMI were used as confounders for most of the correlation analysis.

The LBW cases and the control groups were first compared by using independent sample 't' test for quantitative variables. Simple Chi-square test or Fisher's exact test for independence of attributes was used to explore the differences between the groups in the prevalence of specific components of metabolic syndrome.

For finding the independent predictors of a few quantitative variables such as 120-min glucose and systolic BP, the multivariate analysis was carried out by multiple linear regression technique. The adjusted R^2 was used to determine the predictive power of the model fitted. The statistical significance for the entire analyses was set at $P<0.05$ level. All statistical analyses was performed using Statistical Package for Social Science (SPSS) for Windows (version 11.5).

RESULTS

We have previously described the growth and cognitive development of 161 LBW infants at 18 years [5,6]. There was a dropout of 8 LBW subjects at 22 years, and hence 153 LBW and 77 normal controls were studied. The birth weight of the study group ranged from 866 g to 1999 g with a mean (SD) of 1545.5 (243.9) g and the mean (SD) birth weight of the control group was 2835.0 (305.8) g. There were 93 (60.8%) males and 60 (39.2%) females in the LBW group and 45 (58.4%) males and 32 (41.1%) females in the control group. There were 60 (39.2%)

preterm SGA, 38 (24.8%) full term SGA and 55 (35.9%) preterm AGA babies in the LBW cohort. The comparison of the anthropometric measurements and biochemical parameters and MRI fat between cases and controls is shown in ***Table* I**. There was significant difference between the blood pressure of cases and controls.

Waist circumference ≥90 cm in men and ≥80 cm in women was seen in 13 cases (8.5%) and 5 controls (6.5%). Serum triglycerides ≥150 mg/dL (1.7mmol/L) were seen in 13 (8.5%) cases and 4 (5.3%) controls. Fasting glucose ≥110 mg/dL (6.1mmol/L) was seen in 3 (2%) cases and no controls. Blood pressure ≥130/85 mmHg was seen in 12 (7.9%) cases and no controls. High density lipoprotein ≤40 mg/dL (1.03 mmol/L) in men and <50mg/dL (1.29 mmol/L) in women was seen in 109 (71.7%) cases and 55 (72.4%) controls. The difference between cases and controls in each of these parameters was not significant. The presence of three components of metabolic syndrome was present in three cases and no controls (*P*=0.553). However, two components of metabolic syndrome were present in 25 (16.4%) cases and 5 (6%) controls (*P*=0.039).

Higher BMI at 1, 2, 6, 12, 18 and 22 years correlated significantly with higher systolic pressure. Higher BMI at 12 years correlated significantly with higher fasting insulin and HOMA-IR at 22 years. Physical activity and dietary intake was correlated with the above mentioned parameters, after adjusting for sex and current BMI. The 120-minute insulin level showed inverse correlation with physical activity. Abdominal obesity determined by MRI estimation was not significantly different between cases and controls (***Table* I**).

Table* II** presents the results of multiple linear regression analysis with 120-minute glucose as the dependent variable. Sum of four skinfold thickness at 22 years was the significant and independent determinant of 120-minute glucose. A similar regression analysis for independent determinants of Homa-IR at 22 years also showed that sum of four skinfold thickness was a significant determinant (Table* II**). Similar results were seen for triglycerides. The 22-year BMI was a significant independent determinant for blood pressure.

The birthweight and 22-year weight was divided into four quartiles. Those who were born small and became big at 22 years were compared with those who were born small and remained small (***Table* III**) at 22 years. Fasting, 30 min, 120 min insulin and HOMA-R were significantly higher in those born small and became big at 22 years. Serum triglycerides and cholesterol were also higher in this group. They had higher systolic blood pressure and

TABLE I COMPARISON OF ANTHROPOMETRY, BIOCHEMICAL, CLINICAL AND IMAGING PARAMETERS BETWEEN CASES AND CONTROLS (*N*=230)

Parameters	*Cases (n=153)*	*Controls (n=77)*	*P-value*
Birthweight (g)	1545.5 (243.9)	2835 (305.8)	0.214
Height (cm)	162.1 (10.4)	165.9 (10.2)	0.009
BMI (kg/m^2)	22.3 (4.5)	21.6 (3.9)	0.242
Waist to hip ratio	0.83 (0.07)	0.83 (0.06)	0.821
Sum of 4 skinfolds (mm)	69.4 (25.1)	66.9 (29.8)	0.504
Fasting glucose (mg%)	85.9 (10.0)	91.0 (7.2)	0.298
30 min glucose (mg%)	141.9 (27.9)	145.6 (25.9)	0.341
120 min glucose (mg%)	108.3 (27.8)	103.3 (26.2)	0.195
Fasting insulin (mU/L)	8.0 (2.64-51.91)	7.4 (0.66-32.20)	0.117
*30 min insulin (mU/L)	79.8 (5.04-496.20)	83.4 (21.20-322.74)	0.715
*120 min insulin (mU/L)	44.1 (5.41-415.39)	48.2 (11.11-523.86)	0.984
*HOMA-IR	1.68(0.52-12.16)	1.67 (0.14-7.39)	0.349
Total cholesterol (mg%)	143.4 (28.6)	144.3 (28.8)	0.828
Triglycerides (mg%)	85.1 (40.3)	82.6 (40.2)	0.662
HDL cholesterol (mg%)	40.4 (8.1)	39.9 (7.3)	0.658
Systolic BP (mm HG)	113.8 (9.6)	110.4 (11.7)	0.024
Diastolic BP (mm Hg)	76.0 (5.2)	64.3 (7.5)	0.001
*MRI Fat-Mean (mm^2)	112.9 (21.6-372.4)	124.7 (38.0-329.5)	0.588

*HOMA-IR: Homeostatic model assessment-Insulin resistance; BMI:Body mass index; Values in mean (SD) or *median (IQR).*

TABLE II INDEPENDENT DETERMINANTS OF 120-MINUTES GLUCOSE AND HOMA-R AT 22 YEARS

Variable in the model	*Unstandardized Coefficients*		*Standardized Coefficient*	*P-value*
	B	*Std. Error*	*Beta*	
120 minutes glucose (Adjusted R^2=12.9%)				
(Constant)	125.051	38.369	—	0.001
Birthweight	-0.011	0.008	-0.285	0.176
Gestational age	0.236	0.980	0.029	0.810
Sex	-3.642	4.375	-0.067	0.406
Sum of skinfolds 22years	0.341	0.074	0.343	0.001
Physical activity level (PAL)	-7.790	10.571	-0.055	0.462
Total calories (Kcal)	-0.007	0.006	-0.114	0.266
SGA / AGA Status	6.316	10.534	0.116	0.550
HOMA-IR (Adjusted R^2 = 18.0%)				
(Constant)	0.159	0.887	—	0.858
Birthweight	0.000	0.000	-0.143	0.413
Gestational age	-0.004	0.023	-0.015	0.876
Sex	-0.124	0.110	-0.081	0.260
Sum of skinfolds 22years	0.012	0.002	0.448	0.001
Physical activity level (PAL)	0.100	0.244	0.027	0.681
Total calories (Kcal)	0.000	0.000	0.115	0.177
SGA / AGA Status	0.215	0.260	0.132	0.409

TABLE III PARAMETERS OF 'SMALL AT BIRTH AND BIG AT 22 YEARS' AND 'SMALL AT BIRTH AND SMALL AT 22 YEARS' SUBJECTS

Parameters	*Small at birth and Big at 22 years (n= 29)*	*Small at birth and small at 22 years (n=35)*	*P value*
Fasting glucose (mg%)	87.0 (8.7)	84.1 (7.2)	0.234
30 min glucose (mg%)	142.4 (30.1)	136.3 (25.9)	0.476
120 min glucose (mg%)	110.4 (30.9)	100.9 (22.6)	0.232
Fasting insulin (mU/L)	10.9 (4.1-51.91)	6.2 (2.6-10.59)	0.001
*30 min insulin (mU/L)	154.5 (35.7-396.1)	60.32 (21.8-251.4)	0.001
*120 min insulin (mU/L)	53.5 (13.6-415.4)	37.61 (5.4-144.5)	0.017
*HOMA-IR	2.33 (0.9-12.2)	1.19 (0.5-2.5)	0.001
Total cholesterol (mg%)	154.6 (38.2)	132.8-21.8	0.015
Triglycerides (mg%)	99.3 (43.5)	67.7 (21.1)	0.001
HDL cholesterol (mg%)	38.4 (6.6)	41.1 (8.0)	0.271
Systolic BP (mmHg)	115.7 (8.9)	107.4 (9.1)	0.006
Diastolic BP (mmHg)	77.6 (3.6)	729 (5.9)	0.108
*MRI Fat-Mean (mm^2)	218.7 (111.9-372.4)	96.9 (21.6-201.6)	0.001

*Values in mean (SD) or *median (IQR).*

had more abdominal obesity on MRI (***Table* III**).

There were only six cases of polycystic ovarian syndrome. None of the females had hirsutism. The preterm AGA and SGA cases were compared for all biochemical parameters and blood pressure and MRI fat (***Web Table* I**). There was no difference in any of the parameters, except for systolic blood pressure.

The BMI of both mother and father was compared separately with that of the cases and controls. The BMI of parents correlated with BMI of the study group. When the

WHAT IS ALREADY KNOWN?

- Low birth weight children develop metabolic syndrome in adulthood, especially small for gestational age infants.

WHAT THIS STUDY ADDS?

- Components of metabolic syndrome at 22 years of age are more common among those born low birth weight.
- Those small at birth and big at 22 years had higher frequency of insulin resistance.

incidence of the five components of Met-S between those having family history of diabetes and cardiovascular disease and those not having a family history was compared, there was no significant difference. During our study, six parents were found to be prediabetic, two were frank diabetic and five parents had hypertension, and were not aware of it.

DISCUSSION

This study showed that the prevalence of three components of metabolic syndrome was low at 22 years in LBW children. However, when only two components were concerned, there was a significant difference between their occurrence in cases and controls, hypertension being the first component to appear. The study also showed that 'small at birth and big at 22 years' had many adverse effects like hypertension, increased cholesterol and triglycerides, increased insulin levels and insulin resistance, and increased abdominal obesity. Sum of skinfold thickness at 22 years was an independent determinant of 120-minute glucose, insulin resistance and triglycerides.

This is the last phase of a long prospective study spanning 22 years. In order to enroll the 22-year-old subjects, we offered a free check-up for parents as an added incentive. The dietician could assess the diet of the family and give advice regarding appropriate diet. The main limitation of this study was the less number of controls compared to the cases.

We had earlier studied these subjects for central obesity by taking all their anthropometric measurements for adiposity at 18 years, and found no evidence of adiposity [5]. There are several reports that Asians have normal BMI, but have excess deposition of fat around the waist [14]. The Rancho Bernardo study [16] showed that low birth weight coupled with adult obesity is a strong determinant of metabolic syndrome in postmenopausal women. We found that BMI at 12, 18 and 22 years significantly correlated with higher insulin and HOMA-IR. Sachdev, *et al*. [16] reported that serial measurements of childhood BMI give useful prediction regarding risk of developing adultmetabolic syndrome. Higher BMI at 1, 2, 6 and 18 and 22 years correlated with higher systolic blood pressure in our study. A lot of stress has been placed on BMI in many of the studies on the metabolic syndrome, but very little attention has been given to measurements of adiposity. Dalleck, *et al*. [17] found that waist circumference was independently associated with HDL cholesterol. We found sum of four skinfold thicknesses to be relatively a stronger correlate for biochemical parameters as well as blood pressure compared to BMI, waist circumference and MRI estimation of subcutaneous abdominal fat. A simple measurement of adiposity such as sum of four skinfold thickness may be sufficient, compared to a time consuming and expensive measurement like MRI. Lloyd, *et al*. [18] carried out a meta-analysis of all studies that linked childhood obesity with risk of developing metabolic syndrome. They also reported that there is a little evidence to suggest that greater BMI in childhood was an independent factor for dyslipidemia in adulthood.

We found that "small at birth and big at 22 years" had higher insulin levels and high HOMA-IR. A study from Denmark [19] showed that weight gain in first 3 months of life may increase the risk of metabolic syndrome, particularly glucose metabolism in SGA children. A large study from China [20] reported several components of metabolic syndrome presenting in LBW children in late adulthood. Bavdekar, *et al*. [21] reported "small at birth and big at 8 years" to have impaired glucose tolerance test. Although we had very few cases with three components to meet the IDF criteria of Metabolic Syndrome, the LBW cases had a higher number of two components compared to controls at 22 years. Our findings suggestive of early onset of trends of meatbolic syndrome need further study and reassessment again after a few years. However, it definitely points out the need to initiate early interventions of dietary and lifestlye influences.

Contributors: SC: conceived and carried out the study and wrote the manuscript and shall be guarantor for the paper; MO: helped in data collection and counseling of the family; MH: was responsible for getting the patients and collecting blood samples; AP: supervised the project; MS: statistical analysis. All authors contributed to manuscript writing and its approval.

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REFERENCES

1. Ramachandran A, Snehalatha C, Latha E, Vijay V, Viswanathan M. Rising prevalence of NIIDM in an urban population in India. Diabetologia. 1997;40:232-7.
2. Barker DJP. Foetal origins of coronary heart disease. BMJ. 1995;311:171-4.
3. Reddy KS. Cardiovascular diseases in India. World Health Stat. 1993;46:101-7.
4. Sachdev HPS. Nutritional transition in the backdrop of the early life origins of adult disease: a challenge for the future. Indian J Med Res. 2004;119:iii-v.
5. Chaudhari S, Otiv M, Khairnar B, Pandit A, Hoge M, Sayyad M. Pune low birth weight study – Growth from birth to adulthood. Indian Pediatr. 2012;49:727-32.
6. Chaudhari S, Otiv M, Khairnar B, Pandit N, Hoge M, Sayyad M. Pune low birth weight study-Birth to adulthood – cognitive development. Indian Pediatr. 2013;50:853-7.
7. Singh M. Care of the Newborn. 4th ed. New Delhi: Sagar Publications, 1979.
8. Tanner JM. Normal growth and techniques of growth assessment. Cl Endocr and Metab. 1986;3:411-51.
9. Gopalan C, Rama Shashtri BV, Balsbramanian SC. Nutritive Value of Indian Foods. National Institute of Nutrition, Indian Council of Medical Research, 2002.
10. Kurpad AV, Swaminathan S, Bhat S. IAP National Task Force for Childhood Prevention of Adult Diseases: the effect of childhood physical activity on prevention of adult diseases. Indian Pediatr. 2004;41:37-62.
11. Golan R, Shelef I, Rudich A, Gepner Y, Shemesh E, Chassidim Y, *et. al*. Abdominal superficial subcutaneous fat: A putative distinct protective far subdepot in Type 2 diabetes. Diabetes Care. 2012;35:640-7.
12. Rumack CM, Wilson SR, Charboneau JW. Diagnostic Ultrasound. Vol I, 2nd ed. Mosby 1998. *P*. 313.
13. The IDF Consensus Worldwide Definition of the Metabolic Syndrome Available from :*https://www.idf.org/webdata/docs/IDF_Meta_def_Final.pdf*.
14. Mishra A, Khurana L. Obesity and the metabolic syndrome in developing countries. J Clin Endocrinol Metab. 2008;93:509-25.
15. Yarbrough D, Barrott ConnorE, Krietz Silverstein, Wingard DL. Birth weight, adult weight, and girth as predictors of metabolic syndrome in postmenopausal women: the Rancho Bernardo Study. Diabetes Care.1998;21:1652-8.
16. Sachdev HP, Osmond C, Fall CH, Lakshmy R, Ramji S, Dey Biswas SK, *et. al*. Predicting adult metabolic syndrome from childhood body mass index: follow-up of the New Delhi cohort. Arch Med Res. 2008;39:796-802.
17. Dalleck LC, Kiellan EM. The prevalence of metabolic syndrome and metabolic syndrome risk factors in college-aged students. Am J Health Promot. 2012;27:37-42.
18. Lloyd LJ, Langley-Evans, McMuellen S. Childhood obesity a risk of adult metabolic syndrome: a systematic review. Intl J Obesity. 2012;36:1-11.
19. Fabricius-Bjerre S, Jensen RB, Færch K, Larsen T, Mølgaard C, Michaelsen KF, *et al*. Impact of birth weight and early infant weight gain on insulin resistance and associated cardiovascular risk factors in adolescence. PLoS One. 2011;6:e20595.
20. Xiao X, Zhang ZX, Li WH, Feng K, Sun Q, Cohen HJ, *et al*. Low birth weight is associated with components of the metabolic syndrome. Metabolism. 2010;59:1282-6.
21. Bavdekar A, Yajnik CS, Fall CH, Bapat S, Pandit AN, Deshpande V *et. al*. The insulin resistance (IRS) in eight year old Indian children: small at birth big at 8 years or both? Diabetes. 2000;48:2422-8.

WEB TABLE I CLINICAL AND LABORATORY CHARACTERISTICS OF PRETERM SMALL-FOR-GESTATIONAL AGE (PTSGA) AND PRETERM APPROPRIATE-FOR-GESTATIONAL AGE (PTAGA) NEWBORNS AT 22 YEARS OF AGE (*N*=115)

Parameters	*PTSGAs (n=60)*	*PTAGAs (n=55)*	*P value (Significance)*
Height (cm)	160.2 (9.3)	163.9 (9.2)	0.035
Waist to hip ratio	0.83 (0.07)	0.84 (0.08)	0.266
Sum of 4 skinfolds (mm)	67.1 (23.9)	72.1 (27.3)	0.299
Fasting glucose (mg%)	86.3 (7.2)	85.9 (7.5)	0.781
30 min glucose (mg%)	141.0 (26.3)	141.0 (25.6)	0.996
120 min glucose (mg%)	109.3 (24.8)	105.4 (24.5)	0.402
*Fasting insulin (mU/L)	8.3 (2.64-51.91)	8.1 (2.99-29.60)	0.621
*30 min insulin (mU/L)	84.1 (21.40-392.49)	79.6 (5.04-396.13)	0.871
*120 min insulin (mU/L)	39.3 (8.97-326.08)	43.9 (5.41-415.39)	0.470
*HOMA-IR, median (IQR)	1.75 (0.58-12.16)	1.17 (0.58-7.21)	0.618
Total cholesterol (mg%)	142.4 (28.6)	147.7 (25.3)	0.291
Triglycerides (mg%)	93.1 (46.9)	84.1 (39.4)	0.272
HDL cholesterol (mg%)	39.2 (7.4)	41.4 (8.6)	0.151
Systolic BP (mmHg)	116.1 (9.2)	75.7 (5.3)	0.050
Diastolic BP (mmHg)	76.4 (5.3)	75.7 (5.3)	0.484
*MRI Fat-Mean (mm^2)	107.8 (21.6-323.7)	129.5 (36.3-365.1)	0.156

*Values in mean (SD) or *median (IQR).*