Assessment Of Neonatal Morbidity And Maternal Risk Factors İn Term And Small For Gestational Age (SGA) Babies

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Introduction and Objective:
Small for gestational age (SGA) births with multiple aetiologies may lead to short and long term morbidities in babies. In current study we aimed to assess the rate of SGA births, morbidity rates, postnatal complications and maternal risk factors.

Methods and Materials:
A group of 110 babies born in between 38th and 42nd gestational weeks with a birth weight below 10th percentile of their gestational age and other 110 babies, as control group, having similar gestational ages with a birth weight between 10th and 90th percentile were included in our study retrospectively. Forms to find out potential maternal and fetal risk factors for SGA births were filled up during face to face interviews. All of the babies were assessed in compliance with Lubchenko's maturity and intrauterine growth curves. Accordingly, babies with a birth weight below 10th percentile of their gestational age were classified as SGA whereas those between 10th and 90th percentile were regarded as appropriate for gestational age (AGA)

Results:
SGA prevalance in the study population was 6% and the ratio of female/male was found to be 2.05. SGA babies had 2,57 times higher risk of having SGA siblings compared to control group. The most common cause of SGA births was oligohydramnious with a rate of 50%, which was followed by preechlampsia (25,5%) and fetal causes (7,2%), respectively. In addition, the rate of hypoglycemia and polycytemia (14,5% and 14,5%) in SGA group was significantly higher than that of control (0,9% and 1,8%) group (p values; 0,0001 and 0,001, respectively). The risk of developing hypoglycemia in SGA babies was increased by 18,55 times and polycytemia 9,19 times.

Discussion and Conclusion:
SGA births were significantly related to morbidity and mortality. Therefore, pregnant women should be meticulously screened in terms of serious risk factors such as preterm labour and intrauterine growth retardation (SGA in particular) and prenatal surveillance should be performed carefully to avoid adverse events of birth.

Key Words: *small for gestational age, morbidity*

Introduction

Annually, almost 20 million babies are born small for their gestational age (SGA) worldwide (1). While low birth weight occuring frequently in relation with preterm birth and intrauterine growth retardation (IUGR) escalates into a severe public health issue, every year almost 29.7 million SGA babies reaching the full-term (>37 weeks) are born, additionally (2). SGA was defined by World Health Organisation and American Association of Obstetrics and Gynecology

as the birth weight being below the 10th percentile of population and gender specific intrauterine growth curves for gestational age (3,4). In SGA and preterm birth with low birth weight, coexistence of low birth weight and SGA makes the presentation more serious(2). Prevalance of low birth weight in communities is an important predictor to survey neonatal health associated with socioeconomical state. Thus, prevalance of SGA varies widely based on populations chosen in epidemiologic studies and international studies predict it in between 8.6% and 9.6%. Moreover, this rate rises in underdeveloped or developing countries and goes beyond 50% in some countries(2). Although many factors such as fetal (chromosomal anomalies), maternal (socioeconomical state, nutrition, smoking, alcohol, preeclampsia, multiple pregnancies, placental insufficiency) and environmental (infections, intoxications) effects are thought to have a role in multifactorial occurence patterns, its etiology is still ambiguous (5,6). In addition, increased mortality and morbidity is noted in neonatal and postnatal period of SGA babies. Being SGA has also some lifelong consequences (2,5). In this context, being SGA was reported to be accompanied by a lifelong broad spectrum of clinical presentations, namely cardiovascular diseases, neurodevelopmental and growth failure, insulin resistance, hypertension, metabolic syndrome and obesity as well as neonatal infections and perinatal respiratory disease (7,8). In our study we aimed to evaluate SGA birth rates, morbidities, postnatal complications and maternal risk factors of full-term SGA babies.

Methods

In the study, 110 SGA babies born from May 1st, 2009 to May 1st, 2010 in between 38th and 42nd gestational weeks with a birth weight below 10th percentile of their gestational age were included. For the control group, 110 AGA neonates born on the same days in similar gestational weeks with a birth weight between 10th and 90th percentile were included. Written and verbal informed consents were obtained from the mothers of both study and control groups before the research enrolment. Forms created in compliance with the study goals, which contained various parameters to find out potential prenatal risk factors for SGA births were filled up during face to face interviews with mothers. Maternal obstetric history (gravidity, parity, abortus etc.), smoking during the pregnancy, presence of chronical illness such as diabetes mellitus(DM) type I, DM type II, gestational DM (diabetes of pregnancy), hypertension, hyperthyroidism and hypothyroidism, drugs taken during pregnancy and former delivery of SGA infants were questioned and recorded along with regular demographic data including age, height and weight. From the neonatal risk factors; gender, birth weight, birth length, head circumference at birth, mode of birth (caesarean/ vaginal delivery) , other risk factors leading to SGA births, any hospitalization to the neonatal intensive care unit (NICU), diagnosis and follow-up length in case of hospitalization to a NICU and presence of issues commonly encountered in SGA babies such as hypoglycemia, polycytemia, hypothermia, hypocalcemia, jaundice, difficulty of nutrition were also questioned and put on file. Babies were examined after the delivery and their anthropometric assessments(weight, length, head circumference) were carried out. The gestational age of each neonate was determined by using Dubowitz Scoring Method. Birth weights and their gestational age were evaluated by using Lubchenko's maturity and intrauterine growth curves. Babies with a birth weight below 10th percentile of their gestational age were regarded as SGA whereas those between 10th and 90th percentile were classified as appropriate for gestational age (AGA) and those above 90th percentile as large for gestational age (LGA). Stillbirths and LGA babies were excluded from the study and control groups. The whole set of data collected throughout the study was recorded, filed and compared between groups.

Statistical Analysis

In our study, the version 21.0 of SPSS (Statistical Package ort he Social Sciences, IBM, Armonk, NY, USA) software was used. Definitive statistics was expressed as mean±standard deviation or median (minimum-maximum) for discrete and continuous numerical variables and as number of cases with percentage for categorical variables. Cross table statistics were used in comparison of categorical variables (chi-square, Fisher). Normally distributed parametric data were analyzed with Student t-test and ANOVA, whereas abnormally distributed non-parametric data were compared by using Mann Whitney U and Kruskal Wallis tests. Comparisons between multiple groups were performed with Post Hoc Tukey analysis. Taking the distribution of variables into account, correlation between measurements was evaluated with Spearman's Rho and Pearson tests. Statistical significance was defined as $p<0.05$ in results.

Results

During the study period, a total of 2582 live births of 38 to 42 gestational weeks occured in our hospital. The prevalence of single and live born SGA babies was found to be 6% (n=154). 110 of those with parents who gave consent were recruited to the study group. From the SGA born babies, 74 were female (67,3%) and 36 male (32,7%), where the ratio of female/male was 2,05. The control group was composed of 57 female (51,8%) and 53 male (46,4%) neonates. The rate of female neonates in the study group 67,3%) was noted to be significantly higher than that of control group (52,7%) (p=0,028). Overall, birth weights in 3,6% (n=4) of our sample group were below 2000gr. In the study group, mean birth weight was 2272,45±132,35 gr, mean birth length 48,33±0,84 cm and mean head circumference 33,84±0,85 cm, while they were 3110,55±349,58 gr, 49,29±0,81 cm and 34,56±0,43 cm in the control group, respectively. Unsurprisingly, mean values of birth weight, birth length and head circumference measured in SGA group were found to be significantly lower than that of control group.(p=0,0001, 0,0001 and 0,0001, respectively)(Table 1.). Although no statistically significant difference was detected between groups in terms of SGA siblings, the probability of having a SGA sibling was measured to be 2,57 times higher in SGA study group compared to AGA babies. In addition, 25 of SGA babies (22,7%) were born by cesarean section and 85 (77,3%)by normal spontaneous vaginal delivery. No significant difference was observed between SGA-born study group and AGA-born control group upon mode of delivery.(p=0,221)(Table 2).

In our study, mean age of mothers from the case group was 27,98±5,81 years and that from the control group 28,1±5,26 years (p=0,874). Additionally, no significant difference was noted between groups regarding mean parity and duration of hospitalization (p values; 0,304 and 0,595 respectively)

Oligohydranmnios was determined as the most common prenatal cause of SGA with a frequency of 50%, which was followed by preeclampsia with a rate of 25,5% and fetal causes in 7,2% (chromosomal abnormalities in 0.9% of patients, TORCH infections in 1,8% and fetal malnutrition in 4,5%).

When comparing causes of hospitalization between groups, no significant difference (p>0,05) was found regarding presence of respiratory distress syndrome (RDS), hyperbilirubinemia and transient tachypnea of the newborn (TTN), whereas RDS frequency of SGA group was found to be 2,52 times higher than that of controls.

In addition, hypoglycemia (8,2%) and polycytemia (7,3%) rates in SGA group were shown to be significantly higher than those in AGA group (1,8% and 1,8%)(p values; 0,027 and 0,049 respectively)

No significant difference (p>0,05) was detected between SGA and AGA groups regarding presence of infections, hypothermia, respiratory problems and mode of treatment for jaundice. In contrary, the rate of hypoglycemia in SGA group (14,5%) was found to be significantly higher than that in AGA group (p=0,0001). In SGA-born babies, the risk of developing

hypoglycemia was determined as 18,55 times higher. Similarly, polycytemia rate of SGA group (14,5%) was significantly higher than that of AGA group (1,8%)(p=0,001). Risk of developing polycytemia in SGA-born babies was increased by 9,19 times. While no significant difference was observed between groups regarding difficulties of nutrition and occurence of convulsion, risk of developing nutritional problems and occurence of convulsion were calculated as being 4,23 and 3,2 times higher in SGA babies. Additionally, no significant difference was observed between groups regarding presence of hypocalcemia and maternal smoking, whereas risk of developing hypocalcemia in SGA neonates is increased by 7,19 times and the risk of developing SGA in babies of smoking mothers by 1,5 times.(Table 3).

Discussion

Being born SGA carries increased lifelong morbidity and mortality risk covering perinatal, chilhood and adulthood period. In addition to many diseases such as nutritional, cardiovascular, metabolic and neurodevelopmental issues defined so far, these risks may also rise to serious levels in case of failed catch-up growth. High insidence rates and adverse results documented for SGA births obligate analysis of risk factors as correctly and precisely as possible together with efficacious screening programs and treatment procedures (9). Neonatal morbidity risk is directly associated with gestational age and birth rate (10). Preterm labour, preterm delivery and SGA are intersecting definitions due to their etiological, pathophysiological and adverse outcomes and presence of low birth weight leads to further complications. More than half of the low birth weight deliveries are caused by preterm birth (11). Thus, premature neonates born before 38th week were excluded from our trial.

The prevalence of SGA babies is closely related to the socioeconomic status of the countries. In a research from United States, the frequency of SGA births in the population was 2,3%.(12). In asian countries, however, it ranged between 5,3% and 41,5% in different studies.(2).

Moreover, birth weights of female babies are reported to be 118-121 gr less in avarage than those of males. Also, female gender is associated with an 20% increased risk of SGA births and reported as having 2,5 times increased risk of IUGR (13). When considering all single and live births during our research, the prevalence of SGA births was determined as 6%.

On the other hand, the ratio of female babies in SGA group was noted to be significantly higher than that of controls and female to male ratio was calculated as being 2.05 in the study population. Variations in mitochondrial genome may play a major role influencing neonatal birth weight, as some recent studies could strongly relate birth weight of the baby as an inherited feature to the maternal birth weight (9). In addition, there are reports suggesting an elevated risk of SGA birth for women with a sister who gave a SGA birth (14).

Although no statistically significant difference was detected in our study between groups in terms of SGA siblings, the probability of having a SGA sibling was found to be 2,57 times higher in SGA study group compared to AGA babies,, supporting formerly published data.

The influence of maternal age on SGA birth is one of the most debated issues investigated. In fact, women from 20 to 29 years old are believed to constitute the group with the lowest maternal and perinatal mortality and morbidity. Though a series of research reported an elevated risk of SGA birth in women of 35 years and above, there are also other authors defending that advanced age is not among risk factors of SGA birth.(15,16). While low birth weight(LBW), very low birth weight(VLBW), early preterm labour, anemia and IUGR are more frequent in adolescent pregnancies, advanced age (>35 years) pregnancies lead to more common LBW babies, birth weights above 4000gr, stillbirths and increased perinatal mortality.(17)

In their meta analysis covering 14 published studies on SGA birth risk associated with maternal age, Kozuki et al.reported that the highest risk of SGA birth was in mothers of 18 years and below along with an increased risk also in mothers above 35 years.(18). In our study, no statistically significant difference was found between mothers of SGA group and controls in

terms of age. However, in SGA group 7 mothers were below 20 years and 13 were above 35 without any case below 15 years. There was only one mother above 35 years in control group.
Currently, lots of maternal risk factors leading to SGA births have been identified. Some major factors among them are smoking, alcohol, nutirional issues, past preterm or SGA births, multiple pregnancy and maternal chronic illnesses.(19). No doubt, smoking is one of the most important SGA birth risk factors being dose-dependent and putting back the fetal growth (20). Smoking is blamed for 15% of preterm births, 20-30% of LBW babies and for a 50% increase in perinatal mortality (21). Wang et al. concluded that smoking during pregnancy is associated with a drop of 377gr in birth weight(22). Maternal nutrition has also well-known effects on birth weight. Limitation of calorie intake and inadequate maternal body weight is also associated with LBW (9).
Mitchell et al. revealed that mothers of AGA babies are nourished with a rich diet which contains significantly more fruits, vitamin supplementations and higher amounts of carbohydrates, meat and fish compared to those of SGA babies (23). Additionally, past obstetric history is also closely related to adverse birth outcomes. Kleijer et al. reported that past history of SGA birth increases the SGA risk by 4 times(15).
Multiple pregnancies are regarded as a risk factor for both LBW and preterm birth. Many studies reported that mean birth weight drops progressively with multiple deliveries (9). In the research of Blondel et al., twin births in particular were found to increase the risk of preterm birth and LBW more than triple births (24). Similarly, the findings of our study analyzed together with whole data showed a 1,5 fold increase in SGA birth risk while smoking during pregnancy and 2,57 fold increase in SGA birth for mothers with past SGA birth.
Maternal chronic diseases and hypertension, in particular, were found to be associated with increased perinatal mortality in SGA and preterm births. In pregnant women with chronic hypertension, the rate of giving SGA births is increased compared to normal population, whereas the rates of preterm birth and perinatal mortality did not differ significantly.(25)
In babies of hypertensive mothers, the frequency of IUGR was reported as 45.4% in literature (26). As hypertension is one of the major risk factors of SGA births, the risk is increased by 2.9 fold in case of hypertension and by 18.7 fold in preeclamptic pregnancies with an attributed risk of 28,4%. Grisaru et al declared that there was no significant difference between groups in terms of hypertension in their study, however mothers of SGA births with hypertension had significantly more past history of SGA birth and that was a result of negatively affected fetal growth factors due to hypertension.(27)
In compliance with the published data, our study showed that rate of hypertension is increased by 6,4 fold in mothers of SGA babies.
In the study of Hadders et al. comparing full term and preterm SGA neonates to AGA neonates, 19% of full term SGA births were associated with preeclampsia whereas it was 59% in preterm SGA births(28). Ley et al. found the rate of preeclampsia as 8% in mothers of AGA babies and 44,4% in mothers of SGA babies, which bears a significant difference in SGA group (29).
In another study, preeclampsia being among the risk factors of SGA births was found to increase the development of SGA by 4 fold(30). Also in compliance with these data, our study revealed that presence of preeclamsia is a statistically significant risk factor of SGA birth and 25,5% of the study group had preeclampsia. There was no case of preeclamsia in control group.
Oligohydroamnios was also significantly associated with IUGR and SGA(31). Thus, in a meta analysis of Chauhan et al. evaluating 18 studies , pregnant women with an antepartum or intrapartum diagnosis of oligohydramnios were shown to have an elevated risk of SGA birth due to fetal distess (32). In the trial of Casey et al. carried on 6423 pregnant women above 34.gestational week, the rate of oligohydramnios was 2.3% and there revealed a significant relationship between oligohydramnios and stillbirth along with impairment on fetal monitors, neonatal mortality and SGA birth(33).

LBW in term and preterm births are suggested to be in close relation to neonatal complications such as polycytemia and hypoglycemia (34). Onyiriuka et al detected significantly higher rates of polycytemia in SGA born babies(8,2%) compared to controls(2,2%) (35). Bhat et al, on the other hand, reported a hypoglycemia rate of 25,2% in SGA births (36).In compliance with the published data, our study showed that oligohydramnios is the most common prenatally diagnosed cause of SGA birth with a rate of 50%. In addition, the rate of hypoglycemia and polycytemia were significantly elevated in SGA neonates and the risk of developing hypoglycemia in SGA born babies was increased by 18,55 fold along with an elevated risk of polycytemia by 9,19 fold.

Conclusion

SGA birth was significantly associated with higher morbidity and mortality, supported also by the findings of our study. Therefore, pregnant women should be screened meticulously for important risk factors including preterm labour, IUGR and history of SGA birth and prenatal surveillance should be performed delicately to prevent adverse birth events. Additionally, both health professionals and families should be informed regularly about prenatal and postnatal diagnosis, follow-up and treatment.

References

1- *Black RE. Global prevalence of small for gestational age births. InLow-Birthweight Baby: Born Too Soon or Too Small 2015;81:*

2- *Lee AC, Katz J, Blencowe H, Cousens S, Kozuki N, Vogel JP, Adair L, Baqui AH, Bhutta ZA,Caulfield LE, Christian P. National and regional estimates of term and preterm babies born small for gestational age in 138 low-income and middle-income countries in 2010. The Lancet Global Health. 2013;1(1):e26-36.*

3- *WHO Expert Committee on Physical Status. Physical status: the use of and interpretation ofanthropometry, report of a WHO expert committee. Geneva: World Health Organization, 1995*

4- *American College of Obstetricians and Gynecologists. ACOG Practice bulletin no. 134: fetal growth restriction. Obstetrics and gynecology. 2013;121(5):1122.*

5- *Nam HK, Lee KH. Small for gestational age and obesity: epidemiology and general risks. Annals of pediatric endocrinology & metabolism. 2018;23(1):9.*

6- *Mericq V, Martinez-Aguayo A, Uauy R, Iñiguez G, Van der Steen M, Hokken-Koelega A. Long term metabolic risk among children born premature or small for gestational age. Nature Reviews Endocrinology. 2017;13(1):50.*

7- *Ludvigsson JF, Lu D, Hammarström L, Cnattingius S, Fang F. Small for gestational age and risk of childhood mortality: A Swedish population study. PLoS medicine. 2018;15(12):e1002717.*

8- *Lee AC, Kozuki N, Cousens S, Stevens GA, Blencowe H, Silveira MF, Sania A, Rosen HE,Schmiegelow C, Adair LS, Baqui AH. Estimates of burden and consequences of infants born small for gestational age in low and middle income countries with INTERGROWTH-21st standard: analysis of CHERG datasets. bmj. 2017;358:j3677.*

9- *Saenger P, Czernichow P, Hughes I, Reiter EO. Small for gestational age: short stature and beyond. Endocrine reviews. 2007;28(2):219-51.*

10- *Goldenberg RL. The management of preterm labor. Obstetrics & Gynecology. 2002;100(5):.1020-37.*

11- *Raine T, Powell S, Krohn MA. The risk of repeating low birth weight and the role of prenatal care. Obstet Gynecol 1994;84.: 485–9.*

12- *Talge NM, Mudd LM, Sikorskii A, Basso O. United States birth weight reference corrected for implausible gestational age estimates. Pediatrics. 2014;133(5):844-53.*

13- *Kramer MS. Determinants of low birth weight: Methodological assesment and metoanalysis. Bull WHO 1987; 65: 663- 757.*

14- *Svensson AC, Pawitan Y, Cnattingius S, Reilly M, Lichtenstein P. Familial aggregation of small-for-gestational-age births: the importance of fetal genetic effects. American journal of obstetrics and gynecology. 2006;194(2):475-9.*

15- *Kleijer ME, Dekker GA, Heard AR. Risk factors for intrauterine growth restriction in a socio-economically disadvantaged region. The Journal of Maternal-Fetal & Neonatal Medicine. 2005;18(1):23-30.*

16- *Spinillo A, Capuzzo E, Piazzi G, Nicola S, Colonna L, Iasci A. Maternal high-risk factors and severity of growth deficit in small for gestational age infants. Early human development.1994;38(1):35-43.*

17- *Verrier M, Spears W, Ying J, Kerr GR. Patterns of birth weight in relation to gestational age, maternal age, parity, and prenatal care in Texas' triethnic population, 1984 through 1986. Tex Med.3351993; 89(12):51- 6.*

18- *Fawzi W, Humphrey J. The associations of parity and maternal age with small-for-gestational-age, preterm, and neonatal and infant mortality: a meta-analysis. BMC public health. 2013;13(3):S2.*

19- *McCowan L, Horgan RP. Risk factors for small for gestational age infants. Best practice &research clinical obstetrics & gynaecology. 2009;23(6):779-93.*

20- *Cnattingius S. The epidemiology of smoking during pregnancy: smoking prevalence, maternal characteristics, and pregnancy outcomes. Nicotine Tob Res 2004; 6(2): 125–S140.*

21- *Gomez C, Berlin I, Marquis P, Delcroix M: Expired air carbon monoxide concentration in mothers and their spouses above 5 ppm is associated with decreased fetal growth. Preventive Medicine 2004;40:10- 15.*

22- *Wang X, Tager IB, van Vunakis H, Speizer FE, Hanrahan JP. Maternal smoking during pregnancy, urine cotinine concentrations, and birth outcomes. A prospective cohort study. Int j Epidemiol 1997; 26:978–987*

23- *Mitchell EA, Robinson E, Clark PM, Becroft DM, Glavish N, Pattison NS, Pryor JE, Thompson JM, Wild CJ. Maternal nutritional risk factors for small for gestational age babies in a developed country: a case-control study. Archives of Disease in Childhood-Fetal and Neonatal Edition. 2004;89(5):F431-5.*

22- *Blondel B, Kogan MD, Alexander GR, Dattani N, Kramer MS, Macfarlane A, Wen SW. The impact of the increasing number of multiple births on the rates of preterm birth and low Birth weight: an international study. American journal of public health. 2002;92(8):1323-30.*

22- *Tuntiseranee P. Geater A., Chongsuvivatwong V., Koranantakul O. The effect of heavy maternal workload on fetal growth retardation and preterm delivery. A study among southern Thai women. J Occup Environ Med 1998; 40 (11): 1013- 21.*

23- *McCowan LM, Buist RG, North RA, Gamble G. Perinatal morbidity in chronic hypertension. Br J Obstet Gynaecol. 1996; 103(2):123- 9 64.*

24- *GrisaruGranovsky S, Halevy T, Eidelman A, Elstein D, Samueloff A. Hypertensive disorders of pregnancy and the small for gestational age neonate: not a simple relationship. American journal of obstetrics and gynecology. 2007;196(4):335-e1.*

25- *Hadders-Algra M, Huisjes HJ, Touwen BC. Preterm or small-for-gestational-age infants. European Journal of Pediatrics. 1988;147(5):460-7.*

26- *Ley D, Wide Swensson D, Lindroth M, Svenningsen N, Marsal K. Respiratory distress syndrome in infants with impaired intrauterine growth. Acta Pædiatrica. 1997;86(10):1090-6.*

27- *Blumenstein M, McCowan LM, Wu S, Cooper GJ, North RA. Plasma clusterin increased prior to small for gestational age (SGA) associated with preeclampsia and decreased prior to SGA in normotensive pregnancies. Reproductive Sciences. 2012;19(6):650-7.*

28- *Johnson JM, Chauhan SP, Ennen CS, Niederhauser A, Magann EF. A comparison of 3 criteria of oligohydramnios in identifying peripartum complications: a secondary analysis. Am J Obstet Gynecol 2007;197(2):207.e1-7.*

29- *Chauhan SP, Sanderson M, Hendrix NW, Magann EF, Devoe LD. Perinatal outcome and amniotic fluid index in the antepartum and intrapartum periods: a meta-analysis. American journal of obstetrics and gynecology. 1999;181(6):1473-8.*

30- *Casey BM, McIntire DD, Bloom SL, Lucas MJ, Santos R, Twickler DM, Ramus RM, Leveno KJ. Pregnancy outcomes after antepartum diagnosis of oligohydramnios at or beyond 34 weeks' gestation. American journal of obstetrics and gynecology. 2000;182(4):909-12.*

31- *Singh G, Chouhan R, Sidhu K. Maternal factors for low birth weight babies. Medical Journal Armed Forces India. 2009;65(1):10-2.*

32- *Onyiriuka AN, Okolo AA. Small-for-gestational age, ponderal index and neonatal polycythaemia: a study of their association with maternal hypertension among Nigerian women. Annals of African Medicine 2005;4(4):154-159*

33- *Bhat MA,Kumar P, Bhansali A, Majumdar S, Narang A. Hypoglycemia in small for gestational age babies. The Indian Journal of Pediatrics. 2000;67(6):423-428*

Tables

Table1. Comparison of anthropometric measurements between study and control groups.

	Control Group (Mean±SD)	**SGA Group (Mean±SD)**	**p- value**
Birth weight (gr)	3110,55±349,58	2272,45±132,35	**0,0001***
Birth length	49,29±0,81	48,33±0,84	**0,0001***
Head circumference	34,56±0,43	33,84±0,85	**0,0001***

SD= Standard deviation

***** = p<0.05 statistically significant

Table 2. Comparison of gender, mode of birth and history of SGA born sibling in SGA neonates and controls.

		Kontrol Grubu n:110		SGA Grubu n:110			OR (%95 GA)
SGA Kardeş	Yok	105	95,50%	98	89,10%	χ^2:3,12	2,57
	Var	5	4,50%	12	10,90%	p=0,077	0,87-7,56
Cinsiyet	Kız	58	52,70%	74	67,30%	**χ^2:4,85**	1,84
	Erkek	52	47,30%	36	32,70%	**p=0,028**	1,06-3,18
Doğum Şekli	NSVD	77	70,00%	85	77,30%	χ^2:1,50	1,45
	C/S	33	30,00%	25	22,70%	p=0,221	0,79-2,66

*= **p<0.05 is statistically significant**

Table 3. Common neonatal morbidities and maternal smoking in study and control groups

		Kontrol Grubu		SGA Grubu			OR (%95 GA)
Beslenme Problemi	**Yok**	108	98,20%	102	92,70%	χ^2:3,77	4,23
	Var	2	1,80%	8	7,30%	p=0,052	0,87-20,4
Hipoglisemi	**Yok**	109	99,10%	94	85,50%	**χ^2:14,34**	18,55
	Var	1	0,90%	16	14,50%	**p=0,0001**	2,41-142
Konvülziyon	**Yok**	110	100,00%	109	99,10%	χ^2:1,00	3,2
	Var	0	0,00%	1	0,90%	p=0,316	0,12-75
Polistemi	**Yok**	108	98,20%	94	85,50%	**χ^2:11,86**	9,19
	Var	2	1,80%	16	14,50%	**p=0,001**	2,05-41,03
Hipokalsemi	**Yok**	110	100,00%	107	97,30%	χ^2:3,04	7,19
	Var	0	0,00%	3	2,70%	p=0,081	0,36-14,1
Sigara	**Yok**	84	76,40%	75	68,20%	χ^2:1,84	1,5
	Var	26	23,60%	35	31,80%	p=0,175	0,83-2,73

*= **p<0.05 is statistically significant**