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Maternal first and second trimester lipid levels in patients with different glucose tolerance

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Abstract

Objective: Fetal growth is a diverse process affected by genetic, demographic and metabolic factors. In diabetic patients strict glycemic control does not always prevent macrosomia. This suggests that fuels other than glucose such as lipids and aminoacids also contribute to fetal weight gain. The purpose of this study was to determine maternal lipid levels in patients with different glycemic status.

Methods: This prospective study was conducted at Adana Research and Application Hospital of Başkent University between December 2009 and July 2011. Blood samples were drawn to measure fasting blood glucose, serum triglycerides, cholesterol, VLDL, LDL, HDL and hemoglobin at the first and second trimesters. When 50 g oral glucose intake was 135 mg/dl or above, the patients had a 100 g oral glucose tolerance test. The patients were divided into three groups as normal glucose tolerance (NGT, n=333), impaired glucose tolerance (IGT, n=115), and gestational diabetes mellitus (GDM, n=156).

Results: In the first trimester, triglycerides (NGT, 104.5±54.2; IGT, 104.6±43.5; GDM, 128±63), cholesterol (NGT, 170.2±37.9; IGT, 171.6±32.4; GDM, 180.2±37.2) and VLDL (NGT, 21.4±12.8; IGT, 20.8±8.7; GDM, 25.8±12.7) were higher in GDM group when compared to NGT and IGT groups ($p<0.05$).

Conclusion: This study revealed that lipid levels can be different depending on differences in glucose tolerance.

Key words: Diabetes, glucose tolerance, lipid, pregnancy.

Glukoz toleransı farklı olan gebelerde ilk ve ikinci trimester lipit düzeyleri

Amaç: Fetal büyüme genetik, demografik ve metabolik etkenlerden etkilenen değişken bir olaydır. Diyabetik hastalarda katı glisemik kontrol her zaman makrozomiyi engellemez. Bu durum, lipitler ve aminoasitler gibi glukoz harici başka yakıtların da fetal kilo alımında etkili olduğunu gösterir. Bu çalışmanın amacı farklı glukoz toleransına sahip gebelerdeki ilk ve ikinci trimester kan lipit düzeylerini belirlemektir.

Yöntem: Bu prospektif çalışma Başkent Üniversitesi Adana Araştırma ve Uygulama Hastanesi'nde Aralık 2009 ve Temmuz 2011 tarihleri arasında yürütülmüştür. İlk ve ikinci trimesterlerde açlık kan şekeri, serum trigliseritleri, kolesterol, VLDL, LDL, HDL ve hemoglobin için kan alındı. Eğer 50 g oral glukoz yükleme testi sonrası 1. saat kan 135 mg/dl ve üzerinde ise hastalara 100 g oral glukoz tolerans testi yapıldı. Hastalar, normal glukoz toleransı (NGT, n=333), bozulmuş glukoz toleransı (BGT, n=115) ve gestasyonel diabetes mellitus (GDM, n=156) olmak üzere üç gruba ayrıldı.

Bulgular: İlk trimesterde trigliseritler (NGT, 104.5±54.2; BGT, 104.6±43.5; GDM, 128±63), kolesterol (NGT, 170.2±37.9; BGT, 171.6±32.4; GDM, 180.2±37.2) ve VLDL (NGT, 21.4±12.8; BGT, 20.8±8.7; GDM, 25.8±12.7) gestasyonel diabetes mellitus grubunda diğer iki gruba kıyasla daha yüksekti ($p<0.05$).

Sonuç: Bu çalışma farklı glukoz toleransı olan gruplarda lipit düzeylerinin farklı olduğunu göstermiştir.

Anahtar sözcükler: Lipit, gebelik, diyabet, glukoz toleransı.

Introduction

Fetal growth is a diverse process affected by genetic, demographic and metabolic factors.[1] Impaired glucose metabolism has been proven to affect fetal growth in a linear fashion.[2,3] Even minor degrees of impaired glucose tolerance, as demonstrated by single elevated level of glucose in 100 g glucose tolerance test, are associated with macrosomia.[4] Although poor glycemic

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control is commonly associated with accelerated fetal growth,[5,6] strict glycemic control does not always prevent macrosomia, particularly in overweight women.[7] This suggests that fuels other than glucose such as lipids and aminoacids also contribute to weigh gain.[8]

Pre-pregnancy body mass index (BMI), weight gain during pregnancy, glucose tolerance and triglycerides are reported to be associated with infant birth weight.[9] These variables are also interrelated. They affect each other.

The purpose of this study was to determine maternal lipid levels during first and second trimesters in patients with different glycemic status.

Methods

This prospective study was conducted at Başkent University Adana Research and Application Hospital between December 2009 and July 2011. Women with singleton pregnancies were approached at the first antenatal visit in first trimester. Smoking women, women with pre-gestational diabetes, thyroid and hypertensive disorders, lupus and anti-phospholipid syndrome, multiple pregnancies, and pregnancies with structural fetal abnormalities, or aneuploidy were excluded in order to eliminate the influence of these conditions or diseases on lipid metabolism and infant birth weight. There were no women using anti-lipidemic drugs. Written informed consent was obtained from patients prior to the enrollment. The study was approved by the local ethics committee.

At the first admission, after an overnight fast, blood samples were drawn to measure fasting blood glucose, serum triglycerides, cholesterol, very low-density lipoprotein (VLDL), low-density lipoprotein (LDL), high density lipoprotein (HDL) and hemoglobin. During 24th-28th weeks of gestation, a fasting blood was drawn for the same parameters and the patients had 50 g oral glucose challenge test. When blood samples taken one hour after 50 g oral glucose intake showed glucose of 135 mg/dl or above, the patients had a 100- g oral glucose tolerance test (OGTT). Carpenter- Coustan criteria were used to diagnose gestational diabetes mellitus (GDM).[10]

When two or more of the plasma glucose measurements met or exceeded the following thresholds, the diagnosis of GDM was confirmed: fasting plasma glucose=95 mg/dl, one hour plasma glucose=180 mg/dl, two hour plasma glucose=155 mg/dl and three hour plasma glucose=140 mg/dl. When the patients had 50 g OGTT <135 mg/dl, they were included in normal glucose tolerance (NGT) group. When the patients had 50 g OGTT ≥135mg/dl, but the 100 g OGTT glucose levels were normal, or when they had a single elevated glucose level at 100 g OGTT, they were included in impaired glucose tolerance group (IGT). Blood glucose, triglycerides, cholesterol and HDL were determined by enzymatic calorimetric assay (oxidase). VLDL and LDL cholesterol were calculated by Friedewald formula: LDL-C= (total-C)-(HDL-C)-(VLDL-C) VLDL-C (mg/dl) = triglyceride (mg/dl)/5. All tests were performed by Hitachi 912 analyzer (Roche Diagnostics, Mannheim, Germany).

Gestational age was calculated on the basis of the last menstrual period and was confirmed by the measurement of crown rump length (CRL) in the first trimester. Obstetricians performed sonographic evaluations using Voluson 730 (GE healthcare, Milwaukee, WI, USA) equipped with abdominal transducers of 5-9 MHz. CRL was measured during 11th-14th gestational weeks. The calibers were placed starting from the most cephalic to the most caudal pole. This was converted to the gestational weeks by Hadlock et al.'s formula.[11] The difference between measured and expected fetal ages was expressed in gestational weeks as delta CRL (Δ CRL). BMI was calculated based on the self-reported pre-pregnancy weight and height. Weight gain was calculated by subtracting weight before pregnancy from weight before delivery. All patients were followed up until delivery and delivered at our institution. Data regarding the birth weights were derived from the hospital records.

Maternal hyperlipidemia was defined as a value higher than the 75th percentile of each lipid concentration. Women with gestational diabetes were followed up with endocrinology clinic. Patients were put on a diet and exercise program was recommended. Patients were called 3 weeks later and insulin was commenced according to the fasting and postprandial blood sugar level. All patients were regularly followed up until delivery and insulin doses were adjusted according to the blood sugar.

Numerical variables were evaluated for normality of data by using Kolmogorov-Smirnov test. One way analysis of variance (ANOVA) or Welch ANOVA was performed to compare the differences between the groups. Welch ANOVA was used when homogenity of variance was not satisfied. Chi-square test was used for

the analysis of categorical variables. Power analyses were made to detect differences in lipid levels between the glucose tolerance groups by using NCSS-PASS 2007 program for this study. Power of the study was found to be 75%. Data were expressed as means ± standard deviation (SD) or percentages where appropriate. p<0.05 was considered statistically significant. SPSS for Windows (version 17.0; SPSS, Inc., Chicago, IL, USA) was used for statistical analyses.

Results

During the study period 2383 women delivered at our institution between December 2009 and July 2011. Among these patients the incidence of GDM was %6.5. Because our hospital is a reference hospital seven hundred and ninety-three women admitted to our clinic at the first trimester had all the data thus were included in the study. Seventy-eight women had abortion [spontaneous abortions (53), fetal anomaly (8), intrauterine exitus (17)] and 56 women were lost to follow up. Fifty-five women were excluded because of hypertension [preeclampsia (23), pregnancy induced hypertension (32)]. Thus a total of 604 women were studied. Data about 333 patients with NGT, 115 patients with IGT, and 156 patients with GDM were analyzed.

Maternal and fetal characteristics and data regarding the first trimester scan and maternal serum markers are given in **Table 1.** Mean maternal age, pre-pregnancy BMI and weight gain during pregnancy were similar in NGT and IGT groups. However, the patients were significantly older (p<0.05), pre-pregnancy BMI was significantly higher (p<0.05) and weight gain was lower in GDM group (p<0.05). The distribution of nulliparity was similar in all groups. The mean CRL, NT, free β-hCG and PAPP-A MoMs were similar in all groups. Gestational age at delivery was similar in IGT and GDM groups and was significantly lower than the NGT group (p<0.05).

Fasting blood glucose and serum lipid levels in patients with different glucose tolerance are given in **Table 2.** In the first trimester fasting blood glucose were similar in NGT and IGT groups and significantly higher in GDM group (p<0.05). Fasting blood glucose was similar in all groups in the second trimester. In the first trimester, triglycerides were higher in GDM group when compared to NGT and IGT groups (p<0.05). In the second trimester, triglycerides were similar in IGT and GDM groups and significantly increased when compared to NGT group (p<0.05). In the first trimester, cholesterol levels were higher in GDM group (p<0.05) when compared to NGT and IGT groups. In the second trimester, cholesterol levels were similar in all groups. In the first trimester, VLDL levels were higher in GDM group when compared to NGT and IGT groups (p<0.05). In the second

Table 1. Maternal and fetal characteristics and data regarding the first trimester scan and maternal serum markers.

	NGT (n=333)	IGT (n=115)	GDM (n=156)
Maternal characteristics			
Age (years)	28.4±5.7	29.2±4.5	31.1±5.3*†
Nulliparity (%)	49.3	52.6	48.3
Pre-pregnancy BMI (kg/m²)	23.8±4.15	24.5±4.2	25.8±4.8*†
Weight gain (kg)	13.7±5.6	13.7±8.2	11.2±6.1*†
First trimester scan parameters			
CRL (mm)	59.6±9.3	59.3±8.6	61.5±8.4
NT (mm)	1.3±0.4	1.3±0.4	1.3±0.4
PAPP-A (MoM)	1.05±0.58	1.05±0.63	1.01±0.5
f β-hCG (MoM)	1.18±1.23	1.17±1.10	1.08±0.79
Fetal and neonatal parameters			
Gestational age at delivery (weeks)	38.5±1.4	37.9±1.8‡	37.8±2.3*
Birth weight (g)	3280±500	3196±600	3280±689

IGT: Impaired glucose tolerance group; **GDM:** Gestational diabetes mellitus group; **NGT:** Normal glucose tolerance group; **BMI:** Body mass index; **LGA:** Large for gestational age; **SGA:** Small for gestational age; *p<0.05 GDM vs. NGT; †p<0.05 GDM vs. IGT ‡p<0.05 IGT vs. NGT.

Table 2. Blood glucose and serum lipid levels in patients with different glucose tolerance.

	First trimester			Second trimester		
	NGT (n=333)	IGT (n=115)	GDM (n=156)	NGT (n=333)	IGT (n=115)	GDM (n=156)
Triglyceride (mg/dl)	104.5±54.2	104.6±43.5	128±63	177.5±78	185.9±62.2‡	218.4±89*
Cholesterol (mg/dl)	170.2±37.9	171.6±32.4	180.2±37.2*†	222.2±42.9	229.4±45	222.6±46.4
VLDL (mg/dl)	21.4±12.8	20.8±8.7	25.8±12.7*†	37.1±27	37.5±12.7	45.2±23
LDL (mg/dl)	96.5±31.7	99±24.8‡	103.9±28.9*	124.1±37.6	125±39.8	119.7±45.1
HDL (mg/dl)	52.6±13.4	52.9±12	50.7±13.9	65.9±23.3	65.7±15.8	58.8±13.9*†
Fasting glucose (mg/dl)	81±8.5	83.3±8.7	87.3±15*†	78.1±6.8	81±8.3	86.4±19.5
50 g OGTT (mg/dl)				111.6±11.2	150.4±13.4‡	162±21.2*†

GDM: Gestational diabetes mellitus group; **HDL:** High-density lipoprotein; **IGT:** Impaired glucose tolerance group; **LDL:** Low-density lipoprotein; **NGT:** Normal glucose tolerance group; **OGTT:** Oral glucose tolerance test; **VLDL:** Very low-density lipoprotein; *p<0.05 GDM vs. NGT; †p<0.05 GDM vs. IGT; #p<0.05 IGT vs. NGT.

Table 3. Frequency of hyperlipidemia among different glucose tolerance groups.

	First trimester			Second trimester		
	NGT (n=333)	IGT (n=115)	GDM (n=156)	NGT (n=333)	IGT (n=115)	GDM (n=156)
$Cholesterol_{75p}$ (%)	104.5±54.2	104.6±43.5	128±63	177.5±78	185.9±62.2‡	218.4±89*
$Triglyceride_{75p}$ (%) (mg/dl)	20.7	23.5‡	35.9*†	19.2	25.4‡	37.1*
$VLDL_{75p}$ (%)	22.2	23.5	37.5*	17.4	23.7	37.9
LDL_{75p} (%)	20.7	25.7	31 *	23.4	27	22.4
HDL_{75p} (%)	29.5	29.6	21.8	26.2	28.6	17.5

GDM: Gestational diabetes mellitus group; **HDL:** High-density lipoprotein; **IGT:** Impaired glucose tolerance group; **LDL:** Low-density lipoprotein; **NGT:** normal glucose tolerance group; **VLDL:** Very Low-density lipoprotein; *p<0.05 GDM vs. NGT; †p<0.05 GDM vs. IGT; ‡p<0.05 IGT vs. NGT.

trimester, VLDL levels were similar in all groups. In the first trimester, LDL levels were similar in IGT and GDM groups and were significantly higher in NGT group than in the other groups (p<0.05). In the second trimester, LDL levels were similar in all groups. In the first trimester, HDL levels were similar in all groups. In the second trimester, HDL levels were significantly lower in GDM group than in NGT and IGT groups (p<0.05). The distribution of hyperlipidemia according to glucose tolerance is given in **Table 3.** The frequency of having hypercholesterolemia was similar for all groups in first and second trimester. In the first trimester, hypertriglyceridemia was significantly more common in GDM group when compared to IGT (p<0.05). IGT group also had significantly higher levels of triglycerides than NGT (p<0.05). In the second trimester, GDM and IGT groups had significantly more frequent hypertriglyceridemia when compared to NGT group (p<0.05). In the first trimester, VLDL and LDL levels in the upper quartile were significantly more common in GDM group than in NGT (p<0.05). The frequencies of VLDL and LDL hyperlipidemia in the upper quartile were similar in all the groups in the second trimester. The frequency of HDL hyperlipidemia in the upper quartile was similar in first and second trimesters in all groups.

Discussion

This study revealed that lipid levels can be different depending on differences in glucose tolerance. In all groups, triglycerides, cholesterol, and other lipoproteins increased from first to second trimester, consistent with the other studies.[12] This happens as a result of estrogen enhanced hepatic production of VLDL triglycerides,[13] increased intestinal absorption of

dietary lipid, reduced clearance of triglycerides and decreased extrahepatic lipoprotein lipase activity[14] and increased insulin resistance in pregnancy.[15]

There are contradictory information in the literature about the lipid levels of pregnant patients. We found a significantly higher triglyceride concentration in GDM group compared to the other groups in first trimester. In the second trimester, triglycerides increased in IGT group more than in NGT, and were significantly higher in GDM and IGT groups compared to NGT. In GDM group compared to NGT group triglycerides are reported to be unchanged[17-21] or increased[9] in first trimester and increased in second trimester.[16,22,23] Toescu et al. reported that triglycerides start to increase at an earlier gestational age in GDM.[21] We demonstrated that in patients with GDM triglycerides start to increase as early as first trimester. Increased triglycerides in GDM group in our study may result from insulin resistance which is present even at first trimester. In diabetic pregnancies triglycerides are increased with worsening glycemic control.[24] In IGT group probably insulin resistance becomes more prominent in second trimester, causing a triglyceride level close to GDM at this gestational age. Previous studies reported cholesterol to be elevated[22] or unchanged[17-19] in first trimester or unchanged,[16,17] higher[22] or lower[23] in second trimester. In contrast, we found significantly elevated cholesterol in GDM group compared to the other two groups in first trimester. Cholesterol level was similar in the second trimester in all groups, as reported by Tarim et al. before.[16]

Very low-density lipoprotein is higher in diabetic patients with poor metabolic control compared to those with good metabolic control[21] in GDM patients when compared to NGT patients and when compared to patients with good glycemic control.[16] We demonstrated that in patients with GDM VLDL levels are increased even in the first trimester. A significantly higher LDL score is reported in diabetic women compared to the non-diabetic group.[21] Other studies reported LDL to be lower.[22,23] or unchanged[17,19] in patients with GDM. In our study, LDL was higher in IGT and GDM groups compared to NGT group in first trimester. The excess triglyceride, cholesterol, VLDL and, LDL in GDM group may result from overproduction induced by pregnancy and decreased catabolism related to deficient LPL activity because of insulin resistance.

Fasting blood glucose is significantly higher in the first trimester in the patients who are destined to develop GDM. However, it does not discriminate patients who will have NGT or IGT in the second trimester. In second trimester, fasting blood glucose of all groups was similar which may result from good glycemic control in women with GDM. There are studies which reported similar[16] or higher blood glucose[6] during second trimester in GDM compared to normal or impaired glucose tolerant women. Maternal age, prepregnancy BMI, and weight gain during pregnancy have potential to influence lipid levels. GDM group had a significantly greater mean maternal age. This may be due to worsening of glucose intolerance with age.[25] At the beginning of the pregnancy, BMI was higher in patients with GDM concordant with the other studies.[7,16,21] GDM group gained significantly less weight probably due to the low calorie diet they consumed.

Hypertriglyceridemia is one of the major characteristics of insulin resistance syndrome in non-pregnant adults.[26] Our study demonstrated that the frequency of having hyperlipidemia gradually increases with increasing glucose intolerance. Triglycerides, VLDL and LDL over the 75th percentile was more commonly seen in GDM group; however, among these, only hypertriglyceridemia continued to be more common in second trimester in GDM group. Fuels other than glucose such as lipids and aminoacids suggested to effect fetal weight because strict glycemic control does not always prevent macrosomia.[7] In this study, despite strict glycemic control and increased triglycerides, fetal weights were similar.

Impaired glucose tolerance and increased serum lipid levels increase future cardiovascular risk.[27] First trimester fasting blood glucose and lipids are significantly increased in patients who will develop GDM when compared to women who will remain normotolerant. First trimester LDL levels are increased in patients who will develop GDM and IGT. Patients carrying a future cardiovascular risk can be identified at the first trimester. Differences are less prominent in the second trimester. Only triglycerides are increased in patients with GDM. HDL has anti-inflammatory characteristics.[27] While HDL is similar in all groups in the first trimester, it is significantly decreased in the second trimester in patients with gestational diabetes. The decrease in HDL levels may be responsible for the increased cardiovascular risk.

Conclusion

Even minor alterations in blood glucose affect serum lipid levels. However, there are many parameters which can affect serum lipid or birth weight and cannot be controlled at all times. Normalization of blood glucose and serum lipids is the primary goal for healthy pregnancy.

Conflicts of Interest: No conflicts declared.

References

1. Langer O. Fetal macrosomia: etiologic factors. Clin Obstet Gynecol 2000;43:283-97.
2. Kjos SL, Buchanan TA. Gestational diabetes mellitus. N Engl J Med 1999;341(23):1749-56.
3. Sermer M, Naylor CD, Gare DJ, Kenshole AB, Ritchie JW, Farine D, et al. Impact of increasing carbohydrate intolerance on maternal-fetal outcomes in 3637 women without gestational diabetes. The Toronto Tri-Hospital Gestational Diabetes Project. Am J Obstet Gynecol 1995;173:146-56.
4. Çok T, Tarim E, Bagis T. Isolated abnormal value during the 3-hour glucose tolerance test: which value is associated with macrosomia? J Matern Fetal Neonatal Med 2011;24: 1039-41.
5. Landon MB, Gabbe SG, Piana R, Mennuti MT, Main EK. Neonatal morbidity in pregnancy complicated by diabetes mellitus: predictive value of maternal glycemic profiles. Am J Obstet Gynecol 1987;156:1089-95.
6. Langer O, Levy J, Brustman L, Anyaegbunam A, Merkatz R, Divon M. Glycemic control in gestational diabetes mellitus -- how tight is tight enough: small for gestational age versus large for gestational age? Am J Obstet Gynecol 1989;161: 646-53.
7. Schaefer-Graf UM, Kjos SL, Kilavuz O, Plagemann A, Brauer M, Dudenhausen JW, et al. Determinants of fetal growth at different periods of pregnancies complicated by gestational diabetes mellitus or impaired glucose tolerance. Diabetes Care 2003;26:193-8.
8. Freinkel N. Banting Lecture 1980. Of pregnancy and progeny. Diabetes 1980;29:1023-35.
9. Di Cianni G, Miccoli R, Volpe L, Lencioni C, Ghio A, Giovannitti MG, et al. Maternal triglyceride levels and newborn weight in pregnant women with normal glucose tolerance. Diabet Med 2005;22:21-5.
10. Carpenter MW, Coustan DR. Criteria for screening tests for gestational diabetes. Am J Obstet Gynecol 1982;144:768-73.
11. Hadlock FP, Shah YP, Kanon DJ, Lindsey JV. Fetal crown-rump length: reevaluation of relation to menstrual age (5-18 weeks) with high-resolution real-time US. Radiology 1992;182:501-5.
12. Herrera E, Munilla MA. Placental function and fetal nutrition. Nestlé Nutrition Workshop Series. Philadelphia: Lippincott-Raven; 1997. p. 169-182.
13. Knopp RH, Bonet B, Lasuncion MA, Montelongo A, Herrera E. Lipoprotein metabolism in pregnancy. In: Herrera E, Knopp RH, editors. Perinatal biochemistry. Boca Raton: CRC Press; 1992. p. 19-51.
14. Pedersen J. Weight and length at birth of infants of diabetic mothers. Acta Endocrinol 1954;16:330-42.
15. Julius U, Fritsch H, Fritsch W, Rehak E, Fücker K, Leonhardt W, et al. Impact of hormone replacement therapy on postprandial lipoproteins and lipoprotein(a) in normolipidemic postmenopausal women. Clin Investig 1994;72:502-7.
16. Tarim E, Yigit F, Kilicdag E, Bagis T, Demircan S, Simsek E, et al. Early onset of subclinical atherosclerosis in women with gestational diabetes mellitus. Ultrasound Obstet Gynecol 2006;27:177-82.
17. Montelongo A, Lasunción MA, Pallardo LF, Herrera E. Longitudinal study of plasma lipoproteins and hormones during pregnancy in normal and diabetic women. Diabetes 1992;41:1651-9.
18. Marseille-Tremblay C, Ethier-Chiasson M, Forest JC, Giguère Y, Masse A, Mounier C, et al. Impact of maternal circulating cholesterol and gestational diabetes mellitus on lipid metabolism in human term placenta. Mol Reprod Dev 2008;75:1054-62.
19. Rizzo M, Berneis K, Altinova AE, Toruner FB, Akturk M, Ayvaz G, et al. Atherogenic lipoprotein phenotype and LDL size and subclasses in women with gestational diabetes. Diabet Med 2008;25:1406-11.
20. Schaefer-Graf UM, Meitzner K, Ortega-Senovilla H, Graf K, Vetter K, Abou-Dakn M, et al. Differences in the implications of maternal lipids on fetal metabolism and growth between gestational diabetes mellitus and control pregnancies. Diabet Med 2011;28:1053-9.
21. Toescu V, Nuttall SL, Martin U, Nightingale P, Kendall MJ, Brydon P, et al. Changes in plasma lipids and markers of oxidative stress in normal pregnancy and pregnancies complicated by diabetes. Clin Sci 2004;106:93-8.
22. Sánchez-Vera I, Bonet B, Viana M, Quintanar A, Martín MD, Blanco P et al. .Changes in plasma lipids and increased low-density lipoprotein susceptibility to oxidation in pregnancies complicated by gestational diabetes: consequences of obesity. Metabolism 2007;56:1527-33.
23. Hollingsworth DR, Grundy SM. Pregnancy-associated hypertriglyceridemia in normal and diabetic women. Differences in insulin-dependent, non-insulin-dependent, and gestational diabetes. Diabetes 1982;31:1092-7.
24. Merzouk H, Madani S, Korso N, Bouchenak M, Prost J, Belleville J. Maternal and fetal serum lipid and lipoprotein concentrations and compositions in type 1 diabetic pregnancy: relationship with maternal glycemic control. J Lab Clin Med 2000;136:441-8.
25. Makgoba M, Savvidou MD, Steer PJ. An analysis of the interrelationship between maternal age, body mass index and racial origin in the development of gestational diabetes mellitus. BJOG 2012;119:276-82.
26. Rydén L, Mellbin L. Glucose perturbations and cardiovascular risk: challenges and opportunities. Diab Vasc Dis Res 2012;9:170-6.
27. Fisher EA, Feig JE, Hewing B, Hazen SL, Smith JD. High-density lipoprotein function, dysfunction, and reverse cholesterol transport. Arterioscler Thromb Vasc Biol 2012;32: 2813-20.