The impact of gestational weight gain on subclinical atherosclerosis, placental circulation and neonatal complications

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Gestational weight gain (GWG) has been related to altering future weight-gain curve and increased risks of obesity later in life. Obesity may contribute to vascular atherosclerotic changes as well as excess of cardiovascular morbidity and mortality observed in these patients. Noninvasive arterial testing such as ultrasonographic measurement of carotid IMT is considered a surrogate for systemic atherosclerotic disease burden and is predictive of cardiovascular events in asymptomatic individuals as well as recurrent events in patients with known cardiovascular disease. Currently, there is no consistent evidence regarding vascular impact of excessive GWG.

The present study was designed to investigate the impact of GWG on early atherosclerotic changes during late pregnancy, using intima media thickness, as well as placental vascular circulation and inflammatory lesions and pregnancy outcome.

The study group consisted of 59 pregnant women who gave birth and underwent a placental histopathological examination at the Department of Obstetrics and Gynecology, Edith Wolfson Medical Center, Israel during 2019.

According to the IOM guidelines the study group has been divided into two groups: Group 1 included 32 women with pregnancy weight gain within recommended range; Group 2 included 27 women with excessive weight gain during pregnancy.

The IMT was measured from non-diseased intimal and medial wall layers of the carotid artery on both sides, visualized by high-resolution 7.5 MHz ultrasound (Apogee CX Color, ATL).

Placental histology subdivided placental findings to lesions consistent with maternal vascular and fetal vascular malperfusion according to the criteria of the Society for Pediatric Pathology, subdividing placental findings to lesions consistent with maternal vascular and fetal vascular malperfusion, as well as inflammatory response of maternal and fetal origin.

Results: IMT levels differed between groups and was significantly higher in group 1 compared to Group 2 (0.7+/-0.1 vs 0.6+/-0/1, p=0.028).

Multiple linear regression analysis of IMT included variables based on their associations in univariate analyses with a backward approach. Included in the model were pre-gestational BMI, HDL cholesterol and fasting

glucose. The model was significant (p=0.001), and correctly classified 64.7% of study patients. In this model, pre-pregnancy BMI remained a significant independent predictor of subclinical atherosclerosis assessed by IMT (OR 4.314, 95% CI 0.0599-0.674, p=0.044). Among placental lesions related to fetal vascular malperfusion, villous changes consistent with fetal thrombo-occlusive disease (FTOD) were significantly higher in Group 1 than in group 2, p=0.034).

In the present study, excessive weight gain during pregnancy was associated with an increased IMT in late pregnancy and emerged as a significant predictor of subclinical atherosclerosis. Among neonatal complications, rate of macrosomia was significantly higher in women with excessive weight gain during pregnancy compared with pregnant women with weight gain within recommended range. Weight gain during pregnancy was associated with higher rate of placental villous changes consistent with fetal thrombo-occlusive disease (FTOD).

In conclusion, the present study demonstrated that excessive weight gain during pregnancy is associated with an adverse effect on early stages of subclinical atherosclerosis, placental vascular circulation and neonatal complications. The precise mechanism for these vascular changes, as well as the overall clinical impact of weight control during pregnancy on IMT, placental vascular circulation as well as pregnancy outcomes, deserves further investigation.

Biography

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