Pre-pregnancy BMI associated with placental dysfunction in early but not late preterm birth

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Abstract

Background: Maternal obesity is a risk factor for preterm birth (PTB), particularly before 32 weeks’ gestation. Obesity may increase the risk of PTB through placental damage, but heterogeneity in PTB etiology hinders efforts to find key placental pathways.

Objective: We applied latent class analysis to identify placental pathology phenotypes in early (<32wks) and late PTBs (32 to <37wks) associated with pre-pregnancy BMI using placental pathology data.

Methods: Women with a singleton PTB at Magee-Womens Hospital (Pittsburgh, PA) in 2008-2012 and a placental evaluation (89% of PTBs) were stratified into early (n=900, 61% spontaneous) and late PTBs (n=3362, 57% spontaneous). Pre-pregnancy BMI was self-reported at first prenatal visit and 15 abstracted placental features were included. Placental features were clustered in early and late PTBs separately by latent class analysis. The optimal number of clusters was selected by comparing model fit statistics. The probability of cluster membership across BMIs was estimated in early PTBs and in late PTBs by pseudo-class regression adjusting for race, smoking, education and parity.

Results: Early PTBs clustered into 4 groups: “acute inflammation” (39% of cases), “maternal vascular malperfusion with inflammation” (29%), “maternal vascular malperfusion” (25%), and “fetal vascular malperfusion” (8%). As BMI increased from 20 to 50kg/m², the predicted probabilities of maternal vascular malperfusion and fetal vascular malperfusion steadily increased while the probability of maternal vascular malperfusion with inflammation decreased. (Figure 1) There was minimal change in the probability of acute inflammation with increasing BMI. Late PTBs also clustered into 4 groups: “maternal vascular malperfusion” (22%), “acute inflammation” (12%), “fetal vascular malperfusion” (9%) and “low risk pathology” (58%). Unlike early PTBs, the predicted probabilities for all 4 clusters were relatively unchanged with increasing BMI in late PTBs.

Conclusions: Obesity may predispose women to PTB through placental dysfunction but mainly in early PTBs.
Figure 1: Predicted probabilities of placental pathology clusters across pre-pregnancy BMI (kg/m$^2$) estimated by pseudo-class regression in early PTBs (left) and late PTBs (right). Regression models adjusted for race, education, smoking, and parity. Abbreviations: MVM, maternal vascular malperfusion; FVM, fetal vascular malperfusion.