Pathology – Research and Practice xxx (2016) xxx–xxx

Contents lists available at ScienceDirect

Pathology – Research and Practice

journal homepage: www.elsevier.com/locate/prp

Original article

Placental findings associated with maternal obesity at early pregnancy

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ARTICLE INFO

Article history:
Received 11 September 2015
Received in revised form
18 December 2015
Accepted 22 January 2016

Keywords:
Obesity
Placenta
Large for gestational age
Marginal cord insertion
Inflammation
Intervillous thrombus

ABSTRACT

Introduction: Obesity during pregnancy is associated with a wide spectrum of maternal, fetal and neonatal complications. This study compared placental pathology in women with obesity and normal weight gravidas.
Materials and methods: This is a retrospective case-control study. The sample was randomly selected from a total of 1000 deliveries of largely Caucasian population in a single institution, recruited for the study of sleep disordered breathing, where the placenta is submitted for pathological examination for clinical indications based on national guidelines. Cases (Body mass index – BMI $\geq$ 30 kg/m^2; n = 47) and controls (BMI < 25 kg/m^2, n = 45) were selected based on BMI obtained from the first prenatal visit. Placental pathology, clinical parameters and limited outcomes were extracted from medical records. Placental weight range was defined as small for gestational age (SGA) if <10th percentile, large for gestational age (LGA) if >90th percentile.
Results: Mean BMI was 36.2 $\pm$ 5.5 in the group with obesity and 21.7 $\pm$ 1.9 in the control group (p < 0.01). There was a significantly higher prevalence of diabetes in cases compared to controls (14/47 vs. 3/45, p = 0.006) while preterm birth was significantly higher in the control group (9/47 vs. 19/46, p = 0.02). There were more LGA placentas in cases versus controls (12/47 vs. 2/46, p = 0.007; even after adjusting for diabetes). More histological features of inflammation, marginal insertion of the umbilical cord and intervillous thrombi in the parenchyma were also noted in the case group.
Conclusions: Results from the current study suggest that maternal obesity measured at early pregnancy may have effects on both placental implantation and growth, and further exacerbate the hypercoagulable state in placenta.

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1. Introduction

Obesity has become an epidemic in many countries in the developed world. In the United States, increasing rates of obesity are reported in the general population and include women of reproductive age [1]. According to data from the 2009–2010 National Health and Nutrition Examination Survey, nearly 32% of women of reproductive age (20–39 years of age) have obesity (BMI $\geq$ 30 kg/m^2) [2]. This prevalence was highest in non-Hispanic blacks (56.2%). Despite recent efforts at reducing the prevalence of obesity in pregnant women in the United States to 15%, rates of obesity rose by 0.5% per year from 2003 to 2009 [3]. Goals of healthcare administration in the United States are to reduce the prevalence of obesity among adults in the United States to 30.5% by 2020 [4].

Maternal obesity is associated with adverse reproductive, pregnancy, fetal, neonatal and childhood outcomes. Obesity is associated with lower rates of fertility and a 2–3 fold increase in the risk of gestational hypertensive disorders (GHD) [5]. A significant increase in the risk of gestational diabetes mellitus (GDM) has also been reported with obesity [6]. Fetal and neonatal adverse outcomes such as preterm and post-term births [6], higher prevalence of miscarriages [7] and congenital anomalies such as neural tube defects [8] have all been described. Maternal obesity may have long term effects on the health of the offspring, through mechanisms such as fetal programming. Fetal programming is a concept of a gene-environment model of pathogenesis that proposes that in-utero experiences and exposures may place the offspring at risk for certain behaviors and disorders. Through this hypothetical model, maternal obesity has been suggested as a predisposing factor to insulin resistance and obesity in the offspring [9,10].

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http://dx.doi.org/10.1016/j.prp.2016.01.006
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Please cite this article in press as: M. He, et al., Placental findings associated with maternal obesity at early pregnancy, Pathol. – Res. Pract (2016), http://dx.doi.org/10.1016/j.prp.2016.01.006

Adverse pregnancy outcomes associated with obesity, such as preeclampsia and gestational diabetes, are known to be either mediated, or influenced, by the placenta. Despite this, there is a paucity of data evaluating the effect of obesity on this key organ [11–13].

Obesity is known to induce a metabolic inflammatory state represented by low-grade chronic inflammation, increased levels of an array of inflammatory cytokines and infiltration of inflammatory cells in adipose tissue [14]. Thus, the feto-placental unit in gravidas with obesity may be exposed to an inflammatory environment. The goal of this study is to compare gross and microscopic placental pathology from pregnant women with and without obesity. We hypothesized that women with obesity are at an increased risk of having complications relating to inflammation and thrombosis as well as large for gestational age placentas.

2. Materials and methods

2.1. Participants

This is a cross-sectional study that screened for sleep disordered breathing using the multivariable apnea prediction index and evaluated potential adverse pregnancy and fetal outcomes associated with the disorder. The study systematically recruited a cohort of 1000 English-speaking, largely Caucasian, women at the time of delivery [15], irrespective of the mode of delivery. Patients' medical records were reviewed for medical history and outcomes.

Within this population, women who had placental pathology examination ordered for clinical indications were subsequently identified and divided into 3 groups: the group with obesity (pre-pregnancy BMI $\geq 30\,kg/m^2$) and the lean group (pre-pregnancy BMI $< 25\,kg/m^2$); women with BMI > 25 but $< 30\,kg/m^2$ were excluded from this study. The obese group will be referred to throughout the manuscript as cases or case group and the lean group will be referred to as controls or control group. Once the group with obesity and the control group with available placentas were identified, a random selection of cases and controls was then performed, yielding the final sample of 47 women with obesity and 45 lean women.

2.2. Clinical data

Clinical information collected by chart review included maternal age, BMI and medical history. BMI was assessed at the time of the first prenatal visit. Obstetric information included gestational age (GA) at delivery and adverse outcomes. Adverse outcomes included preterm birth (defined as birth before 37 weeks), and intrauterine growth restriction (IUGR, defined as estimated fetal weight below 10th percentile). Covariates included preeclampsia (defined as the presence of two elevated blood pressures with proteinuria according to guidelines at the time of the study) and gestational diabetes (defined based on Carpenter and Coustan criteria [16]).

2.3. Placental studies

Placentas were submitted for examination based on clinical indications following national guidelines [17]. All placental examinations were performed in the Division of Perinatal Pathology, Department of Pathology and Laboratory Medicine at the Women and Infants' Hospital of Rhode Island following a standard protocol [18]. Placental pathology reports were generated by one of five board certified pediatric pathologists. Ten percent of all placental slides were audited and reviewed weekly. If there is disagreement between the two pathologists, additional pathologists would review the slides to help reach a consensus. This audit ensures that the reports generated vary minimally among individual pathologists.

For the current study, gross placental parameters and histopathology were extracted from the pathology report. Placental weight is the trimmed weight of the placenta after removing the membranes. Small for gestational age (SGA) is defined as weight <10th percentile of age range, appropriate for gestational age (AGA) defined as weight from 10th to 90th percentile, and large for gestational age (LGA) defined as weight >90th percentile. Fetal to placental (F/p) weight ratio is often used as a marker for placental efficiency. F/p ratio less than 10th percentile is usually suggestive of placental insufficiency. Pathology seen in multi-gestational pregnancy is counted once per side (or disk).

Histopathological diagnosis was classified into four groups based on the presence of markers of maternal circulatory disorders, fetal vascular obstruction, infection/inflammation and others.

Table 1
Patient information of obese gravida and controls.

Patient information	Obese gravida ($n=47$)	Control ($n=45$)	p-Value
Age (years)	29.6 ± 5.6 (19–40)	28.8 ± 6.2 (18–40)	
BMI (kg/m^2)	36.2 ± 5.5	21.7 ± 1.9	
Maternal complications			
Diabetes mellitus	14 (29.8%)	3 (6.7%)	0.006
Pregnancy-induced hypertension	11 (23.4%)	8 (17.8%)	0.6
Preeclampsia	6 (12.8%)	7 (15.6%)	0.8
Obstetric and fetal outcomes			
Preterm birth, *N*	9 (19.2%)	19 (42.2%)	0.02
Congenital anomalies	0	4 (8.9)	0.054
IUGR, clinical	2 (4.3%)	10 (22.7%)	0.01
Macrosomia	4 (8.5)	1 (2.2)	0.4

2.4. Statistical analysis

Descriptive analyses were performed using Microsoft Excel®. Demographic variables are described in mean, median and standard deviation. The prevalence of specific pathological findings was reported dichotomously (present or absent) in the two groups and compared using the Fisher's exact test, with $p < 0.05$ considered significant. Adjustment for potential confounders was performed by the Mantel–Haenszel method.

3. Results

3.1. Patient characteristics

A total of 47 pregnant patients with obesity (cases) and 45 lean women (controls) were identified. Subject characteristics, obstetric complications and fetal outcomes are summarized in Table 1. Mean age was 29.6 ± 5.6 years in cases (range 19–40) and 28.8 ± 6.2 years (range 18–40) in controls, $p = 0.5$. Mean BMI was $36.2 \pm 5.5\,kg/m^2$ in the case group and $21.7 \pm 1.9\,kg/m^2$ in the control group.

3.2. Clinical outcomes

There were significantly more cases of diabetes (gestational, type 1 and type 2) in the case group compared to the control group (14/47 vs. 3/45, $p = 0.006$). Incidence of pregnancy induced hypertension (PIH, 11/47 vs. 8/45, $p = 0.6$) and preeclampsia (6/47 vs. 7/45, $p = 0.8$) was similar in both groups.

Mean gestational age at birth was significantly higher in the case group compared to the control group (38.2 ± 3.2 weeks vs. 36.0 ± 4.5 weeks $p = 0.008$). Prevalence of preterm delivery in our cohort sample was elevated in both groups but was significantly higher in the control group (19.1% in the case group and 41.3% in

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