

Iñigo Melchor, Jorge Burgos, Ana del Campo, Amaia Aiartzaguena, Julieta Gutiérrez and Juan Carlos Melchor*

Effect of maternal obesity on pregnancy outcomes in women delivering singleton babies: a historical cohort study

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Abstract

Background: Obesity in pregnancy is increasing worldwide, reaching epidemic proportions in many countries and frequently creating challenges for obstetricians. We conducted this study to assess the effects of maternal obesity on maternal and perinatal outcomes.

Methods: A historical cohort study was performed on 16,609 women who delivered singleton babies in a 5-year period (2013–2017). Data were retrieved from the Cruces Perinatal Database (CPD) and only women whose prepregnancy body mass index (BMI) was known were included. Women were categorized according to the World Health Organization (WHO) classification: normal weight (BMI 20–24.9 kg/m²) and obesity (BMI \geq 30 kg/m²). Obstetric, perinatal and neonatal outcomes were compared, and adjusted odds ratios (aORs) and 95% confidence intervals (95% CIs) were calculated using the normal-weight group as the reference.

Results: Compared to women of normal weight (n = 9778), obese women (n = 2207) had a higher risk of preeclampsia (aOR 2.199, 95% CI: 1.46–3.29), rectovaginal group B streptococcus colonization (aOR 1.299, 95% CI: 1.14–1.47), induction of labor (aOR 1.593, 95% CI: 1.44–1.75), cesarean section (aOR 2.755, 95% CI: 2.46–3.08), cesarean section in women with a history of cesarean delivery (aOR 1.409, 95% CI: 1.03–1.92), fetal weight \geq 4000 g (aOR 2.090, 95% CI: 1.803–2.422) and admission to the neonatal intensive care unit (NICU) (aOR 1.341, 95% CI: 1.12–1.59).

*Corresponding author: Juan Carlos Melchor, Obstetrics and Gynecology Department, Biocruces Health Research Institute, Cruces University Hospital (UPV/EHU), Vizcaya, Spain; and Obstetrics and Gynecology Department, Cruces University Hospital (UPV/EHU), Plaza de Cruces s/n, 48903, Barakaldo, Vizcaya, Spain, Tel.: +34-946006000, E-mail: jmelchorm@segovia.es

Iñigo Melchor, Jorge Burgos, Ana del Campo, Amaia Aiartzaguena and Julieta Gutiérrez: Obstetrics and Gynecology Department, Biocruces Health Research Institute, Cruces University Hospital (UPV/EHU), Vizcaya, Spain

No association was found with preterm birth (aOR 0.936, 95% CI: 0.77–1.13), stillbirth (aOR 0.921, 95% CI: 0.41–2.02) or neonatal mortality (aOR 2.205, 95% CI: 0.86–5.62).

Conclusion: Maternal obesity is associated with a higher risk of adverse pregnancy and perinatal outcomes. Pregnancy in this population of women should be considered and managed as high risk.

Keywords: neonatal mortality; obesity; perinatal mortality; pregnancy; pregnancy complications; pregnancy outcome; prenatal care; stillbirth.

Introduction

The increasing prevalence of obesity is a major public health concern especially among women of reproductive age. In Spain, 30.0% of women are overweight and 16.7% are obese [1]. This trend has a major impact on pregnancy outcomes, as has been widely reported.

Specifically, during pregnancy and childbirth, obese women are at greater risk of maternal-fetal complications than women with a normal body mass index (BMI). Obese women are known to be at risk of antenatal, intrapartum, postpartum and neonatal complications such as hypertensive disorders of pregnancy, gestational diabetes mellitus, venous thromboembolism, cesarean section, preterm delivery, fetal macrosomia and unexplained stillbirths [2–9]. Additionally, children born to obese mothers are at increased risk of obesity and metabolic disease [10] and of developing neuropsychiatric and cognitive disorders [11].

The purpose of this study was to examine the association between prepregnancy BMI and obstetric and neonatal outcomes in an unselected population of obese women in a large tertiary referral university hospital in Spain.

Materials and methods

We performed a historical cohort study by reviewing the Cruces Perinatal Database (CPD) for the 5-year period (2013–2017). We

included 16,609 women who delivered singleton babies at ≥ 23 weeks of gestation and for whom prepregnancy BMI data were available.

Women were classified according to their BMI, which was calculated from self-reported prepregnancy weight and height. The information was obtained during their first antenatal visit between gestational weeks 6 and 10. BMI categories were based on the World Health Organization (WHO) standards and were defined as follows [12]: underweight (<18.5 kg/m 2), normal weight (18.5–24.9 kg/m 2), overweight (25.0–29.9 kg/m 2) and obese (class I: 30–34.9 kg/m 2 ; class II: 35–39.9 kg/m 2 ; class III: ≥ 40 kg/m 2). Finally, we compared women with normal weight and women with obesity (including all women with a BMI of ≥ 30 kg/m 2).

Maternal outcomes considered were preeclampsia, chronic hypertension, pregestational diabetes mellitus, gestational diabetes, rectovaginal group B streptococcus colonization, antepartum hemorrhage (including placenta previa, abruptio placentae and third-trimester bleeding), induction of labor, instrumental vaginal delivery, cesarean section and shoulder dystocia (diagnosed clinically by the delivering attending physician).

Perinatal/neonatal outcomes considered were fetal presentation, birth weight (fetal macrosomia ≥ 4000 g, fetal macrosomia ≥ 4500 g, low birth weight <2500 g), preterm birth <37 weeks, meconium-stained amniotic fluid, 5-min Apgar score <7 , fetal acidosis (umbilical cord arterial pH <7.10), admission to the neonatal intensive care unit (NICU), stillbirth at ≥ 23 weeks and neonatal mortality (0–28 days).

Ethical approval

All data were extracted from the CPD by authorized investigators. Ethical approval was granted by the Clinical Research Ethics Committee of our hospital (CEIC E10/12). The database used for the research contains data that have been anonymized.

Statistical analyses

Data were analyzed using SPSS Statistics for Windows, Version 24.0 (IBM, Armonk, NY, USA). Obese and non-obese women were compared with descriptive statistics using unpaired Student *t*- or Mann-Whitney *U*-tests for continuous variables and chi-square (χ^2) or

Fisher's exact tests for categorical variables as appropriate. The Kolmogorov-Smirnov test was used to check whether continuous data were normally distributed. Univariate analyses and multivariate logistic regression analyses (controlling for maternal age, parity, gestational age and chronic hypertension) were performed, and adjusted odds ratios (aORs) and 95% confidence intervals (95% CI) were estimated. Potentially confounding factors were identified earlier in the stratified analyses, and variables for which there were differences between groups were included as covariates in the adjusted analyses. Only factors contributing significantly to the explanatory model were included in the final model.

The group of women with a normal BMI was used as the reference group for all analyses. $P < 0.05$ was considered statistically significant.

Results

Of the 16,609 women identified from the database with complete prepregnancy BMI data, 168 (2.7%) were underweight, 9778 (58.9%) had a normal BMI, 4166 (25.0%) were overweight and 2207 (13.3%) were obese at their first antenatal visit. Of the obese women, 1494 were obese class I (BMI 30–34.9 kg/m 2), 530 obese class II (BMI 35–39.9 kg/m 2) and 183 obese class III (BMI ≥ 40 kg/m 2).

General characteristics of the study population are summarized in Table 1. Statistically significant differences were noted between BMI groups in maternal age (mean: 33.82 ± 4.86 and 34.05 ± 4.94 years in normal-weight and obese women, respectively; $P = 0.045$) and in parity, with a higher rate of primiparity among women with normal weight (58.91% vs. 48.02%; $P < 0.001$). The mean prepregnant BMI was 21.93 ± 1.69 kg/m 2 in normal-weight women and 34.21 ± 4.06 kg/m 2 in obese women (32.03 ± 1.41 , 36.95 ± 1.41 and 44.16 ± 4.14 kg/m 2 in obese classes I, II and III, respectively). While the rates of chronic hypertension were higher in the women with normal weight (4.84% vs. 0.76%; $P < 0.001$), we did not find differences between the groups in the rate of pregestational diabetes.

Table 1: General characteristics of the study population.^a

Parameters	Normal weight (n=9778)	Obesity (n=2207)	P-value
Maternal age, years	33.82 ± 4.86	34.05 ± 4.94	0.045
Nulliparous, n (%)	5761 (58.91%)	1060 (48.02%)	<0.001
Gestational age, weeks	39.06 ± 1.88	39.11 ± 1.91	0.260
Prepregnant body mass index, kg/m 2	21.93 ± 1.69	34.21 ± 4.06	<0.001
Maternal weight, kg	58.62 ± 6.20	90.12 ± 12.54	<0.001
Maternal height, cm	163.36 ± 6.25	162.12 ± 7.16	<0.001
Smoking, n (%)	674/5137 (13.12%)	156/1175 (13.27%)	0.850
Chronic hypertension, n (%)	75 (0.76%)	107 (4.84%)	<0.001
Pregestational diabetes mellitus, n (%)	72 (0.73%)	11 (0.49%)	0.217

^aValues are given as mean \pm standard deviation (SD) unless indicated otherwise.

The aORs for pregnancy outcomes are shown in Table 2. Rates of preeclampsia (obese women aOR 2.199) and maternal rectovaginal colonization with group B streptococci (obese women aOR 1.299) were significantly higher in the obese group. No significant differences were observed between normal-weight and obese women in rates of pregestational diabetes mellitus, gestational diabetes (5.35% vs. 5.02%, respectively), antepartum hemorrhage, preterm birth (6.16% vs. 6.66%, respectively), breech presentation or stillbirth (0.47% vs. 0.41%, respectively).

Intrapartum outcomes are presented in Table 3. A higher BMI was associated with a greater risk of induction of labor (obese women aOR 1.593) and cesarean section (obese women aOR 2.755). Further, obese women who had previously had a cesarean section were at an increased risk of a repeat cesarean section in their current pregnancy (34.15% vs. 57.25% in normal-weight and obese women, respectively). No significant differences were found in rates of instrumental vaginal delivery or shoulder dystocia. Obesity was associated with an increased

rate of meconium-stained amniotic fluid (obese women aOR 1.352).

Neonatal outcomes are shown in Table 4. A higher BMI was associated with a greater rate of macrosomia ≥ 4000 g (obese women aOR 2.090) and ≥ 4500 g (obese women aOR 3.087). In addition, the infants of obese women were more likely to be admitted to the NICU and have an umbilical cord arterial pH < 7.10 . Nonetheless, rates of low birth weight (< 2500 g) and neonatal mortality were similar in both groups.

Discussion

The results of this historical cohort study among singleton deliveries showed that prepregnancy obese women (including classes I, II and III obesity) have a higher risk of antenatal, intrapartum, postpartum and neonatal complications such as hypertensive disorders of pregnancy, maternal rectovaginal colonization with group B streptococci, induction of labor, cesarean section, repeat

Table 2: Association between adverse pregnancy outcomes and maternal BMI.

Parameters	Normal weight (n = 9778) ^a	Obesity (n = 2207)	aOR (95% CI) ^b	P-value
Preeclampsia	53 (0.54%)	35 (1.58%)	2.199 (1.46–3.29)	0.000
Pregestational diabetes mellitus	72 (0.73%)	11 (0.49%)	0.733 (0.38–1.38)	0.733
Gestational diabetes	524 (5.35%)	111 (5.02%)	0.951 (0.77–1.17)	0.637
Antepartum hemorrhage ^c	603 (0.56%)	147 (0.63%)	1.206 (0.67–2.15)	0.529
Maternal rectovaginal group B streptococcus positive culture	1197 (12.24%)	350 (15.86%)	1.299 (1.14–1.47)	0.000
Preterm delivery < 37 weeks	55 (6.16%)	14 (6.66%)	0.936 (0.77–1.13)	0.497
Breech presentation	310 (3.17%)	64 (2.89%)	0.981 (0.74–1.28)	0.887
Stillbirth	46 (0.47%)	7 (0.31%)	0.921 (0.41–2.02)	0.838

Values are given as number and %. ^aWomen with normal weight were used as the reference group. ^bValues are adjusted odds ratio (aOR) with 95% CI in parentheses. Adjusted for maternal age, parity, gestational age and chronic hypertension. ^cPlacenta previa, abruptio placentae, third trimester gestational bleeding.

Table 3: Association between adverse intrapartum outcomes and maternal BMI.

Parameters	Normal weight (n = 9778) ^a	Obesity (n = 2207)	aOR (95% CI) ^b	P-value
Induction of labor	2732 (27.94%)	854 (38.69%)	1.593 (1.44–1.75)	0.000
Induction of labor in late term pregnancies ^c	445/2308 (19.28%)	156/737 (21.16%)	1.151 (0.83–1.57)	0.385
Cesarean section	984 (10.06%)	560 (25.37%)	2.755 (2.46–3.08)	0.000
Cesarean section in previous cesarean ^d	153/448 (34.15%)	150/262 (57.25%)	1.409 (1.03–1.92)	0.030
Instrumental delivery ^e	2052/8645 (23.73%)	404/1633 (24.73%)	0.938 (0.83–1.05)	0.296
Meconium-stained amniotic fluid	1345 (13.75%)	396 (17.94%)	1.352 (1.19–1.52)	0.000
Shoulder dystocia	11 (0.11%)	6 (0.27%)	1.617 (0.65–3.99)	0.297

Values are given as number and %. ^aWomen with normal weight were used as the reference group. ^bValues are adjusted odds ratio (aOR) with 95% CI in parentheses. Adjusted for maternal age, parity, gestational age and chronic hypertension. ^cIn our hospital, induction for a late-term pregnancy was routinely performed at 41+5 gestational weeks. ^dIncluded only cesarean sections. ^eIncluded only vaginal deliveries (excluded breech deliveries and cesarean sections).

Table 4: Association between adverse neonatal outcomes and maternal BMI.

Parameters	Normal weight (n=9778) ^a	Obesity (n=2207)	aOR (95% CI) ^b	P-value
Fetal macrosomia (≥ 4000 g)	511 (5.22%)	280 (12.68%)	2.090 (1.80–2.42)	0.000
Fetal macrosomia (≥ 4500 g)	55 (0.56%)	51 (2.31%)	3.087 (2.18–4.37)	0.000
Low birth weight (< 2500 g)	710 (7.26%)	139 (6.29%)	0.794 (0.65–0.96)	0.021
Admission to neonatal intensive care unit	535 (5.47%)	172 (7.79%)	1.341 (1.12–1.59)	0.001
Umbilical cord arterial pH $< 7.10^c$	656/9455 (6.93%)	191/2145 (8.90%)	1.330 (1.12–1.56)	0.001
Neonatal mortality (0–28 days) ^d	12/9716 (0.12%)	6/2188 (0.27%)	2.205 (0.86–5.62)	0.098

Values are given as number and %. ^aWomen with normal weight were used as the reference group. ^bValues are adjusted odds ratio (aOR) with 95% CI in parentheses. Adjusted for maternal age, parity, gestational age and chronic hypertension. ^cIncluding only cases with umbilical cord arterial pH. ^dExcluding intrauterine fetal deaths.

cesarean section in women with a history of cesarean delivery, fetal macrosomia, meconium-stained amniotic fluid and infant admission to the NICU. These findings are consistent with previous reports [2–9, 13–15]. In a recent study, Kim et al. [16] found that the risk of any pregnancy complication was 18–47% higher among obese women.

Conversely, we found no association between maternal obesity and risk of gestational diabetes, preterm delivery < 37 weeks, low birth weight < 2500 g, stillbirth, instrumental vaginal delivery, infant shoulder dystocia or neonatal mortality. This was not totally unexpected, as the association between maternal obesity and some of these effects are less evident in the literature [15].

The prevalence of obesity has significantly increased in many developed countries, particularly over the past two decades in adults, and the WHO considers it to be a significant health threat, particularly because of its association with cardiovascular disease [17]. The latest European Perinatal Health Report showed that the levels of obesity ($\text{BMI} \geq 30 \text{ kg/m}^2$) in pregnant women were lowest in Poland (7.1%), Slovenia (9.0%) and France (9.9%). The majority of other European countries had rates of 12–14%, and in Scotland, 20.7% of all pregnant women had weights in the obese range [18]. The prevalence of maternal obesity found in this study (13.3%) is similar to that reported in one other study of Spanish women [7].

The cause of adverse outcomes in pre pregnancy obese women is uncertain. Causes of maternal obesity are complex and multifactorial. The increased risk may be due to obesity itself or the presence of comorbidities such as diabetes mellitus and hypertensive disorders of pregnancy [16]. In a retrospective study of 112,309 singleton deliveries among mothers without chronic diseases in the Consortium on Safe Labor, the authors found that women who were obese but did not have any pre pregnancy chronic diseases were at significantly greater risk of a wide range of adverse pregnancy and neonatal complications than women with a normal BMI [16]. It seems likely

that the main cause of the complications that appear in this group of pregnant women is obesity itself. Another study found that in women who go into labor, maternal obesity is an independent risk factor for significant neonatal morbidity, even in the absence of hypertensive disorders or diabetes [19].

A systematic review of the literature calculated that for each 1 kg/m^2 increase in BMI the prevalence of gestational diabetes mellitus increased by 0.92% [3]. Another review found that the risk of preeclampsia doubled with each $5–7 \text{ kg/m}^2$ increase in prepregnancy BMI [20]. In our study, we have found that obesity is associated with higher rates of hypertensive disorders (chronic hypertension and preeclampsia), though not with a higher rate of diabetes (pregestational or gestational). Specific effect of obesity vs. gestational diabetes remains imprecise and unclear. All obese women are considered as being at high risk of gestational diabetes whereas the majority do not develop such disorder [21]. In our population of obese pregnant women, the rate of gestational diabetes is much lower than that described in other similar studies conducted in North America and Western Europe [16, 22], and this may be one of the reasons explaining the absence of differences in the rate of gestational diabetes between obese and normal-weight pregnant women. Neither the analysis by subgroups of obese pregnant women showed significant differences in the rates of gestational diabetes. Only when both hyperglycemic disorders (pregestational diabetes and gestational diabetes) of obese pregnant women are included together, the differences are significant (OR: 3.62). Therefore, it is likely that obesity is not only an independent factor, and that the results we have found may partly be due to the comorbidities that accompany obesity.

Similarly, increasing BMI correlates linearly with cesarean delivery rates. We have found that cesarean sections are almost 3 times more common among obese pregnant women (aOR 2.755) and that the rate of cesarean section in women with a history of cesarean delivery

is also higher (aOR 1.409). Previous reports found similar results [23]. Several factors explain these findings. In a conceptual framework for the impact of obesity on risk of cesarean delivery, Rogers et al. [24] pointed out that the comorbidities, obstetric complications, slower progression of labor and prolongation of pregnancy are the main causes of this increase among nulliparous. Among parous women, the negative effect of the previous cesarean section should also be included. On the other hand, fetal macrosomia is also a factor that would increase cesarean rates. We have found that the rate of fetal macrosomia is 2- to 3-fold higher among obese pregnant women than among normal-weight pregnant women.

In this study, we found a higher rate of maternal rectovaginal colonization with group B streptococcus in obese pregnant women than women with normal weight. This finding has also been described by other authors [7, 25]. It is plausible that the cause of this difference lies in the difficulty of maintaining good genital hygiene due to obesity, which would facilitate vaginal contamination. Interestingly, these results have been described both in clinical settings with a very low colonization rate (4.9%) [25] and with rates similar to ours (13.2%) [7].

Previous studies found that maternal obesity is associated with higher neonatal mortality [26], especially in infants of extremely obese women. In our study, although there is a higher incidence of problems during pregnancy and delivery, these events are not reflected in neonatal mortality, probably due to the reduced number of neonatal deaths (three in obesity type I and three in obesity type II). There were no cases in pregnant women with extreme obesity.

Strength and limitations

Limitations of our study include its historic and observational nature as well as the limitations inherent to a registry-based analysis. On the other hand, perinatal registers offer possibilities for quality improvement in pregnancy and childbirth and research [27]. Not all data that might affect the outcome were reliably available in the registry, but the number of obese women is high and therefore our results may be applied in similar settings.

The data on BMI used in this study were based on self-reported information about weight and height obtained at the first antenatal visit which may not be accurate. Nevertheless, we considered this information to be non-biased as there is no reason to believe that obese women have more difficulties recalling these data than women with normal weight. Therefore, we think that BMI is a

consistent indicator of obesity and has been validated in previous publications [28]. Further, the database lacked information on some confounding factors, for example, social class and maternal race, which might have influenced the relation between obesity and pregnancy outcomes. Despite these limitations, we believe that clinically important conclusions can be drawn.

The strengths of our study include the large number of obese women studied, in particular, those with class II–III obesity, who are often under-represented in research. Furthermore, we have studied only obese ($BMI \geq 30 \text{ kg/m}^2$) and normal-weight ($BMI 18.5\text{--}24.9 \text{ kg/m}^2$) pregnant women, excluding women who were overweight, while some studies have included overweight categories that overlapped with normal or obese definitions.

Conclusion

Obesity in pregnancy is increasing worldwide, reaching epidemic proportions in many countries and frequently creating challenges for the obstetrician. In this study, our results indicate that maternal obesity is associated with an increased risk of adverse maternal and fetal/neonatal complications. Pregnancy in this population of women should, therefore, be considered and managed as high risk.

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