

UC Irvine

UC Irvine Previously Published Works

Title

Pre-pregnancy Obesity and the Risk of Peripartum Cardiomyopathy

Permalink

<https://escholarship.org/uc/item/6xb3s86q>

Journal

American Journal of Perinatology, 38(12)

ISSN

0735-1631

Authors

Cho, Seo-Ho
Leonard, Stephanie A
Lyndon, Audrey
et al.

Publication Date

2021-10-01

DOI

10.1055/s-0040-1712451

Peer reviewed



Published in final edited form as:

Am J Perinatol. 2021 October ; 38(12): 1289–1296. doi:10.1055/s-0040-1712451.

Pre-pregnancy Obesity and the Risk of Peripartum Cardiomyopathy

Seo-Ho Cho, MS¹, Stephanie A. Leonard, PhD², Audrey Lyndon, PhD³, Elliott K. Main, MD⁴, Barbara Abrams, PhD⁵, Afshan B. Hameed, MD^{6,7}, Suzan L. Carmichael, PhD^{8,9}

¹Division of Neonatal and Developmental Medicine, Department of Pediatrics, Stanford Prevention Research Center, Stanford University School of Medicine, Stanford, California

²Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, Stanford University School of Medicine, Stanford, California

³Rory Meyers College of Nursing, New York University, New York, New York

⁴California Maternal Quality Care Collaborative, Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, Stanford University School of Medicine, Stanford, California

⁵Division of Epidemiology, University of California Berkeley School of Public Health, Berkeley, California

⁶Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, University of California Irvine School of Medicine, Irvine, California

⁷Division of Cardiology, Department of Medicine, University of California Irvine School of Medicine, Irvine, California

⁸Division of Neonatal and Developmental Medicine, Department of Pediatrics, Stanford University School of Medicine, Stanford, California

⁹Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, Stanford University School of Medicine, Stanford, California

Abstract

Objective—The aim of this study is to evaluate the contribution of pre-pregnancy obesity and overweight to peripartum cardiomyopathy.

Study Design—This population-based study used linked birth record and maternal hospital discharge data from live births in California during 2007 to 2012 ($n = 2,548,380$). All women who had a diagnosis of peripartum cardiomyopathy during the childbirth hospitalization or who were diagnosed with peripartum cardiomyopathy during a postpartum hospital readmission within 5 months of birth were identified as cases. Pre-pregnancy body mass index (BMI, kg/m^2) was classified as normal weight (18.5–24.9), overweight (25.0–29.9), obesity class 1 (30.0–34.9),

Address for correspondence Suzan L. Carmichael, PhD, Department of Pediatrics and Department of Obstetrics and Gynecology, Stanford University School of Medicine, 1265 Welch Road., X111, Stanford, CA 94305 (scarmichael@stanford.edu).

Conflict of Interest
None declared.

obesity class 2 (35.0–39.9), and obesity class 3 (40). Because of small numbers, we excluded women with underweight BMI, and in some analyses, we combined obesity classes into one group. Logistic regression was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs) expressing associations between BMI and peripartum cardiomyopathy, adjusted for maternal age, race/ethnicity, education, health care payer, parity, plurality, and comorbidities.

Results—The overall prevalence of peripartum cardiomyopathy during hospital admissions was 1.3 per 10,000 live births ($n = 320$). Unadjusted ORs were 1.32 (95% CI: 1.01–1.74) for women with overweight BMI and 2.03 (95% CI: 1.57–2.62) for women with obesity, compared with women with normal pre-pregnancy BMI. Adjusted ORs were 1.26 (95% CI: 0.95–1.66) for overweight women and 1.38 (95% CI: 1.04–1.84) for women with obesity. The ORs suggested a dose–response relationship with increasing levels of obesity, but the 95% CIs for the specific classes of obesity included 1.00.

Conclusion—Pre-pregnancy obesity was associated with an increased risk of peripartum cardiomyopathy. These findings underscore the importance of BMI during pregnancy. There is a need to recognize the increased risk of peripartum cardiomyopathy in women with high BMI, especially in the late postpartum period.

Keywords

body mass index; heart failure; hypertensive disorder; maternal health; pregnancy complication

Peripartum cardiomyopathy is an idiopathic dilated cardiomyopathy that typically presents toward the last month of pregnancy or within 5 months following childbirth. Peripartum cardiomyopathy is a serious condition with the outcome ranging from a relatively benign course with full recovery to major organ system dysfunction including congestive heart failure, residual left ventricular systolic dysfunction, heart transplantation, cardiac arrest, acute kidney injury, and even death.¹⁻³ In recent decades, the prevalence of peripartum cardiomyopathy has increased in the United States.^{4,5} The reported prevalence in the United States varies from two to nine cases per 10,000 live births, with a case fatality rate of 3 to 6%.^{2,4-10} Based on national data from 2011 to 2015, cardiomyopathy was the leading cause of maternal mortality in the late postpartum period (42–365 days), accounting for 45% of all such deaths.¹¹

The etiology of peripartum cardiomyopathy remains unknown. Early evidence suggested nutritional deficiencies play a role as increased risk of peripartum cardiomyopathy was reported among women who were malnourished.^{12,13} Recent studies do not support this relationship; rather, evidence suggests peripartum cardiomyopathy to be a type of myocarditis arising from an infectious, autoimmune, or idiopathic process.^{9,12,13} Other studies suggest that peripartum cardiomyopathy and hypertensive disorders may share pathophysiology.¹⁴⁻¹⁶ A meta-analysis of 22 studies ($n = 979$ cases) showed that pregnancy-related hypertensive disorders are associated with peripartum cardiomyopathy, with the prevalence of preeclampsia being four times higher among women with peripartum cardiomyopathy.¹⁴ In addition, the prevalence of peripartum cardiomyopathy is higher among women with non-Hispanic Black race/ethnicity, age > 35 years old, and prolonged use of tocolytics.^{4-6,8,9,17-21}

One potential explanation for the increasing prevalence of peripartum cardiomyopathy is the increasing prevalence of obesity.²² In the United States, the prevalence of pre-pregnancy obesity increased from 13% in 1994 to 26% in 2015.²² Both pregnancy and obesity place a burden on the heart and increase susceptibility to potential problems, such as cardiomyopathy.²³ Obesity cardiomyopathy has been recognized as a myocardial disease in nonpregnant obese individuals that cannot be explained by other etiologies.²⁴ Prior studies are suggestive of an association of maternal obesity with the development of peripartum cardiomyopathy but have had important limitations.^{2,8,16,25-27} In particular, some have used diagnostic billing codes to identify obesity, which is known to have poor sensitivity^{2,25-27} or lacked access to postpartum records, although a large proportion of cases occur after childbirth hospitalization.^{2,25} Some studies have also included women with other known heart conditions, which may misclassify women with peripartum cardiomyopathy,²⁵ had small sample sizes¹⁶ or were restricted to maternal deaths related to peripartum cardiomyopathy although the vast majority of women survive.⁸

The objective of this study was to determine the association of pre-pregnancy BMI with peripartum cardiomyopathy, using data from more than 2.5 million women who gave birth in California from 2007 to 2012.

Materials and Methods

Information on 3,039,568 live births that occurred in California hospitals between January 1, 2007 and December 31, 2012 was obtained from birth certificates and maternal hospital discharge data, which were linked by the Office of Statewide Health Planning and Development. For each year of births, the data files contained linked information on maternal hospitalizations during pregnancy through 9 months postpartum. Maternal height and weight were added to the California birth certificates in 2007, and linked data after 2012 were not available at the time of analysis; thus, we focused our analyses on birth cohorts from 2007 to 2012. Each birth cohort file contains International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) codes for up to 25 diagnoses and 21 procedures during each hospitalization that are recorded as well as data recorded on birth certificates. Over 98% of the births had successful linkage of maternal discharge and birth certificate data.

The primary outcome of peripartum cardiomyopathy was identified using ICD-9-CM diagnosis code 674.5. All women who had a diagnosis of peripartum cardiomyopathy during the childbirth hospitalization or during a postpartum hospital readmission within 5 months of birth were identified as cases. Because peripartum cardiomyopathy is a diagnosis of exclusion, we followed the approach of previous studies^{2,26,28} that used administrative data and excluded women with diagnosis codes for the following other cardiovascular conditions: heart valve disease, congenital heart disease, conduction disorders and cardiac dysrhythmias, pulmonary circulation disease/other and ill-defined heart disease/coronary atherosclerosis, hypertensive congestive heart failure, unspecified cardiovascular diseases, or nonperipartum cardiomyopathy (►Fig. 1; ICD-9-CM diagnosis codes provided in ►Supplementary Table S1 [available in the online version]). Before these diagnostic exclusions, there were 1,180 women with an ICD-9-CM diagnosis code for peripartum cardiomyopathy; 68% ($n =$

805) of them were excluded because they had other heart disease diagnosis codes. In a sensitivity analysis, we included women with these diagnoses when identifying peripartum cardiomyopathy cases to be able to compare our results to those of prior studies.²⁵

We also excluded women with underweight BMI because the number of potential cases with underweight BMI was small ($n = 28$). After these exclusions, there were 2,880,866 births, including 372 cases, eligible for analyses. Among these births, 2,548,380 (320 cases) were not missing data on pre-pregnancy BMI or covariates and were included in analyses. The State of California Committee for the Protection of Human Subjects and the Stanford University Research Compliance Office approved the study protocol.

Pre-pregnancy overweight and obesity were the exposures of interest. They were assessed using pre-pregnancy BMI calculated as weight in kilograms divided by height in meters squared. Pre-pregnancy weight and height are largely self-reported at childbirth hospitalization. Pre-pregnancy weight <75 lbs or >450 lbs and height <48 in or >78 in were considered implausible. Pre-pregnancy BMI was classified as normal weight (18.5–24.9), overweight (25.0–29.9), obesity class 1 (30.0–34.9), obesity class 2 (35.0–39.9), and obesity class 3 (40). Obesity (30) was also assessed as one group due to the small number of cases when separating obesity classes.

Potentially confounding factors were determined *a priori* based on previous evidence.^{2,4,6,7,10,16,25-27,29} Covariates obtained from the birth certificate included mother's race/ethnicity (foreign-born Hispanic/Latina, U.S.-born Hispanic/Latina, non-Hispanic white, Asian/Pacific Islander, non-Hispanic Black, other), level of education (less than high school degree, high school degree equivalent, higher than high school degree), health care payer (nonprivate insurance or private insurance), age, parity (primiparous or multiparous), and plurality (singleton or multiple birth). Maternal comorbidities were identified if present in the hospitalization record or birth certificate; they included pre-pregnancy diabetes, pre-pregnancy hypertension, and pregnancy-related hypertensive disorders (gestational hypertension, preeclampsia, or eclampsia; ICD-9-CM diagnosis codes provided in ►Supplementary Table S1 [available in the online version]).

We compared characteristics of women with and without peripartum cardiomyopathy using Chi-square tests, *t*-tests, and Fisher's exact test. We then used logistic regression to estimate odds ratios (OR) and 95% confidence intervals (CI) for the associations between pre-pregnancy BMI and peripartum cardiomyopathy. Given the rarity of peripartum cardiomyopathy, we consider the OR a close approximation of the relative risk. We conducted unadjusted models and then adjusted for race/ethnicity, education, health care payer, age, parity, plurality, pre-pregnancy diabetes, and pre-pregnancy hypertension. As a sensitivity analysis, we also conducted the regression models among women without pregnancy-related hypertensive disorders to determine whether associations persisted among these women and because simply adjusting for them would be inappropriate due to temporality issues (i.e., they emerge subsequent to the main exposure, pre-pregnancy BMI). We additionally repeated analyses using a broader case ascertainment of peripartum cardiomyopathy that does not exclude cases with diagnoses of nonperipartum cardiomyopathy or other heart conditions. This was done to facilitate comparison of our

findings with other studies.²⁵ A p -value <0.05 was considered statistically significant. SAS 9.4 was used for statistical analysis.

Results

Peripartum cardiomyopathy occurred in 320 of the 2,548,380 live births included in analyses, yielding a prevalence of 1.3 per 10,000 births. Relative to women who were included in the analyses, women who were excluded due to missing or implausible data were slightly more likely to have peripartum cardiomyopathy (1.6 per 10,000 live births) and to have normal BMI, foreign-born Hispanic ethnicity, less than high school education, and non-private insurance; they were similar with respect to comorbidities (►Supplementary Table S2 [available in the online version]).

Among the cases, 134 (42%) were diagnosed with peripartum cardiomyopathy during childbirth hospitalization and 186 (58%) were first diagnosed during a postpartum readmission within 5 months of giving birth. Relative to women without peripartum cardiomyopathy, women with peripartum cardiomyopathy were more likely to have non-Hispanic Black race/ethnicity, greater than high school education, older age, nulliparity, early gestational age at birth, nonsingleton birth, pre-pregnancy diabetes or hypertension, and pregnancy-related hypertensive disorders (►Table 1).

The prevalence of peripartum cardiomyopathy increased monotonically across the BMI categories, from 1.0 per 10,000 live births for women with normal BMI, to 3.4 for women with class 3 obesity (►Table 2). Increasing pre-pregnancy BMI category was associated with increasing odds of peripartum cardiomyopathy in unadjusted regression models (►Table 2). After adjusting for confounders, odds ratios were attenuated—especially for obesity. Confidence intervals included the null for all BMI categories except for obesity class 3 (OR: 1.57, 95% CI: 1.00–2.47) and all obesity classes combined (OR: 1.38, 95% CI: 1.04–1.84).

When we excluded women with pregnancy-related hypertensive disorders, adjusted odds ratios were 1.47 (95% CI: 1.04–2.07) for women with overweight BMI (vs. 1.26 before the exclusion) and 1.32 (95% CI: 0.91–1.92) for women with obese BMI (vs. 1.38 before the exclusion), compared with women with normal BMI (►Supplementary Table S3 [available in the online version]). When we used the broader case definition of peripartum cardiomyopathy that included women with diagnoses of nonperipartum cardiomyopathy or other heart disease, the prevalence of peripartum cardiomyopathy was 3.8 per 10,000 live births (►Supplementary Table S4 [available in the online version]). The adjusted odds ratios were 1.10 (95% CI: 0.93–1.29) and 1.22 (95% CI: 1.04–1.44) for women with overweight and obese BMI, respectively.

Discussion

In this population-based dataset of more than 2.5 million women, the prevalence of peripartum cardiomyopathy increased with severity of overweight and obesity. After adjusting for eight covariates including maternal age and race/ethnicity, obesity was associated with a 38% increase in risk of peripartum cardiomyopathy relative to women

with normal BMI. The adjusted risk was slightly attenuated to 32% after excluding women with pregnancy-related hypertensive disorders and further reduced to 22% when we used a broader definition that included cases with other reported heart disease. Overall, our results suggest that maternal obesity is associated with peripartum cardiomyopathy. The risk is relatively modest but important given the high prevalence of obesity and the potential severity of peripartum cardiomyopathy.

Prior evidence suggests an association between obesity and peripartum cardiomyopathy but is limited. Two prior studies that examined U.S. hospital births and antenatal data reported that obesity was more common among women with peripartum cardiomyopathy; the first reported a 1.4-fold higher risk of peripartum cardiomyopathy with obesity,²⁵ and the second reported an approximate twofold higher risk after adjustment for several covariates.² An additional study of insurance claims data reported an unadjusted 1.5-fold increased risk of peripartum cardiomyopathy among obese women.²⁶ The first study did not exclude women with other heart disease when identifying their study population and all three studies used ICD-9-CM diagnosis codes to identify pre-pregnancy obesity, although it is well recognized that obesity is highly under-reported by these codes in administrative data.^{30,31} A study of 60 women with peripartum cardiomyopathy in Denmark did use high-quality BMI and peripartum cardiomyopathy data and reported that women with obesity were at approximately twofold increased risk of cardiomyopathy.¹⁶ Maternal mortality studies provide additional evidence of increased peripartum cardiomyopathy risk among women with obesity. Hameed et al⁸ reported that out of 42 pregnancy-related cardiomyopathy deaths among women who gave birth in California from 2002 to 2006, 26% ($n = 11$) occurred among overweight women and 43% ($n = 18$) among obese women. Another study, of a large sample of U.S. births, reported that the case-fatality rate for peripartum cardiomyopathy was twofold higher for women who were obese, but the estimate was imprecise (adjusted OR: 1.95, 95% CI: 0.23–16.3).²⁷ Compared with previous studies, we observed similar relative measures of association; our study observed an unadjusted twofold increased risk and an adjusted 1.4-fold increased risk of peripartum cardiomyopathy among women with obese BMI. Our study additionally observed an adjusted 1.3-fold increased risk of peripartum cardiomyopathy among women with obese BMI when we excluded women with pregnancy-related hypertensive disorders, and an adjusted 1.2-fold increased risk when we used a broader case definition of peripartum cardiomyopathy. These estimates suggested that pre-pregnancy obesity was associated with an increased risk of peripartum cardiomyopathy.

The prevalence of peripartum cardiomyopathy in this study was 1.3 per 10,000 births, which is lower than some previous studies; these studies have reported prevalences of two to four cases per 10,000 births and used varied approaches to identify cases.^{6,7,9,25} We used a conservative case ascertainment approach to minimize misclassification of other heart conditions as peripartum cardiomyopathy is defined by exclusions. Our approach may explain why our calculated prevalence is low compared with other studies. However, some other studies have reported prevalences similar to our study.^{16,25,28} When we ran the sensitivity analysis including women with diagnoses for nonperipartum cardiomyopathy or other heart disease, the prevalence was 3.8 per 10,000 births— which is more in alignment with previous studies. Associations between obesity and peripartum cardiomyopathy

remained but were some-what attenuated when using this broader case definition. Obesity is likely associated with other cardiac outcomes in the peripartum period,⁸ which could have contributed to this finding; examining other specific cardiac conditions merits future investigation.

An association of obesity with peripartum cardiomyopathy has biologic plausibility. Obesity, with its related hemodynamic changes, is known to cause structural remodeling of the heart.^{32,33} Changes such as increased left ventricular wall thickness and mass along with left ventricular dilation are thought to be directly related to the excessive adipose tissue,³³ which increases blood volume and stroke volume and may lead to ventricular dilation.^{33,34} Ventricular dilation diminishes myofibril shortening, which may result in reduced systolic myocardial contraction and increased wall stress.³³⁻³⁵ These changes over time may cause ventricular hypertrophy and/or dilation with resultant diastolic and/or systolic dysfunction which may manifest as heart failure. Pregnancy also causes physiologic changes in the cardiovascular system including decreased systemic vascular resistance with resultant tachycardia and increased cardiac output due to the increased stroke volume. The cardiovascular changes related to obesity coupled with the hemodynamic stress of pregnancy may result in cardiac decompensation in the form of peripartum cardiomyopathy. In particular, it is important to recognize that this susceptibility extends to the late postpartum period.

This study has several limitations common to retrospective studies utilizing large administrative hospital databases. Peripartum cardiomyopathy diagnoses and some covariates using administrative data and ICD-9-CM diagnosis codes are prone to underreporting for rare maternal conditions.³⁶ In particular, the ICD-9-CM code for peripartum cardiomyopathy was introduced in 2003 and has not been systematically validated.²⁵ Compared with earlier years, the ICD-9-CM code offers more specific peripartum cardiomyopathy diagnosis,^{2,5,25} but we did not have access to patient-level medical records to verify its accuracy (e.g., left ventricular systolic function, other biomarkers, and no prior occurrence of cardiomyopathy). Thus, we indirectly confirmed the peripartum cardiomyopathy diagnosis by excluding women who had other heart disease in their medical record, following the approach of prior studies.^{2,26,28} Pre-pregnancy weight used in assessing maternal BMI was either self-reported by the mother or measured and reported by health professionals. Self-reported pre-pregnancy weight may incur bias due to measurement error. However, a systematic review on the accuracy of self-reported pregnancy-related weight concluded that although individuals with overweight or obesity underreport pre-pregnancy weight, the magnitude of error tends to be small (0.3–3 kg) and largely does not bias associations with birth outcomes.³⁷ Underreporting of pre-pregnancy weight may imply that more women with overweight or obesity are misclassified, thus attenuating the true possible link between obesity and peripartum cardiomyopathy. We recognize that pre-pregnancy BMI is only one aspect of women's weight and further studies are needed to assess the relationship between gestational weight gain and risk of peripartum cardiomyopathy.³⁸⁻⁴¹ Our study used California data, which may limit the generalizability of the results. However, one in eight U.S. births occur in California,⁴² and California is a state with relatively diverse demography and geography.⁴³ Finally, this study did not include peripartum cardiomyopathy cases that were only diagnosed at autopsy.

Important strengths of our study are that we had access to data on a large, diverse population that enabled examination of peripartum cardiomyopathy through 5 months postpartum; reported rather than code-based data on maternal body mass index; and the ability to examine levels of obesity. To sufficiently study obesity and peripartum cardiomyopathy, one needs to have data on pre-pregnancy weight and height in addition to diagnosis codes from childbirth through 5 months postpartum in a very large population because of the rarity of this very severe disease. The linked data used for this study had these qualities. Most epidemiologic studies examining peripartum cardiomyopathy have none to very limited access to postpartum data.^{2,25,29} These studies fail to capture the full 5-month postpartum follow-up data, which accounted for more than half of the cases in our study. Using pre-pregnancy height and weight data from the birth certificate to calculate BMI is preferable to reporting from ICD-9-CM codes, and we were able to examine the less common extremes of BMI (obesity class 3). We also examined whether the association still held after excluding women with pregnancy-related hypertensive disorders, which further contributes to our understanding of the independent contribution of obesity to peripartum cardiomyopathy. This is particularly important, given that 38% of women with peripartum cardiomyopathy had pregnancy-related hypertensive disorders, versus only 6% of women who did not have this outcome. Future studies, with similar features regarding data quality, are needed to replicate our findings. We adjusted for a variety of potential confounders, which we chose *a priori*. Other factors in the dataset could also be potential confounders but tend to be relatively uncommon and/or under-reported (e.g., 0.22% of women had ICD-9 codes for autoimmune disorders, 1.14% had codes for substance use disorders) (►Supplementary Table S5 [available in the online version]).

Pre-pregnancy obesity was associated with an increased risk of peripartum cardiomyopathy. These findings underscore the importance of reducing pre-pregnancy obesity to improve health outcomes. Additionally, there is a need to recognize and manage the increased risk of peripartum cardiomyopathy in women with high BMI, especially in late postpartum period.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding

This project was supported by grant R01NR017020 from the National Institute of Nursing Research (awarded to S.L. C.), postdoctoral fellowship F32HD091945 from the Eunice Kennedy Shriver National Institute of Child Health and Development (awarded to S.A.L.), and Stanford Maternal and Child Health Research Institute. The funding sources had no involvement in the conduct of research or preparation of this article.

References

1. Elkayam U, Akhter MW, Singh H, et al. Pregnancy-associated cardiomyopathy: clinical characteristics and a comparison between early and late presentation. *Circulation* 2005;111(16): 2050–2055 [PubMed: 15851613]
2. Masoomi R, Shah Z, Arany Z, Gupta K. Peripartum cardiomyopathy: an epidemiologic study of early and late presentations. *Pregnancy Hypertens* 2018;13:273–278 [PubMed: 30177065]

3. Sliwa K, Petrie MC, Hilfiker-Kleiner D, et al. Long-term prognosis, subsequent pregnancy, contraception and overall management of peripartum cardiomyopathy: practical guidance paper from the Heart Failure Association of the European Society of Cardiology Study Group on Peripartum Cardiomyopathy. *Eur J Heart Fail* 2018;20(06):951–962 [PubMed: 29578284]
4. Nelson-Piercy C, Head C. Management of cardiomyopathies in pregnancy. In: Gatzoulis MA, Steer PJ, eds. *Heart Disease and Pregnancy*. 2 ed. Cambridge: Cambridge University Press; 2016: 160–173
5. Kolte D, Khera S, Aronow WS, et al. Temporal trends in incidence and outcomes of peripartum cardiomyopathy in the United States: a nationwide population-based study. *J Am Heart Assoc* 2014;3(03):e001056 [PubMed: 24901108]
6. Harper MA, Meyer RE, Berg CJ. Peripartum cardiomyopathy: population-based birth prevalence and 7-year mortality. *Obstet Gynecol* 2012;120(05):1013–1019 [PubMed: 23090517]
7. Gunderson EP, Croen LA, Chiang V, Yoshida CK, Walton D, Go AS. Epidemiology of peripartum cardiomyopathy: incidence, predictors, and outcomes. *Obstet Gynecol* 2011;118(03):583–591 [PubMed: 21860287]
8. Hameed AB, Lawton ES, McCain CL, et al. Pregnancy-related cardiovascular deaths in California: beyond peripartum cardiomyopathy. *Am J Obstet Gynecol* 2015;213(03):379.e1–379.e10 [PubMed: 25979616]
9. Sliwa K, Hilfiker-Kleiner D, Petrie MC, et al.; Heart Failure Association of the European Society of Cardiology Working Group on Peripartum Cardiomyopathy. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. *Eur J Heart Fail* 2010;12(08): 767–778 [PubMed: 20675664]
10. Elkayam U. Clinical characteristics of peripartum cardiomyopathy in the United States: diagnosis, prognosis, and management. *J Am Coll Cardiol* 2011;58(07):659–670 [PubMed: 21816300]
11. Petersen EE, Davis NL, Goodman D, et al. Vital signs: pregnancy-related deaths, United States, 2011–2015, and strategies for prevention, 13 states, 2013–2017. *MMWR Morb Mortal Wkly Rep* 2019;68(18):423–429 [PubMed: 31071074]
12. Brown CS, Bertolet BD. Peripartum cardiomyopathy: a comprehensive review. *Am J Obstet Gynecol* 1998;178(02):409–414 [PubMed: 9500508]
13. Sliwa K, Fett J, Elkayam U. Peripartum cardiomyopathy. *Lancet* 2006;368(9536):687–693 [PubMed: 16920474]
14. Bello N, Rendon ISH, Arany Z. The relationship between preeclampsia and peripartum cardiomyopathy: a systematic review and meta-analysis. *J Am Coll Cardiol* 2013;62(18):1715–1723 [PubMed: 24013055]
15. Parikh P, Blauwet L. Peripartum cardiomyopathy and preeclampsia: overlapping diseases of pregnancy. *Curr Hypertens Rep* 2018; 20(08):69 [PubMed: 29971645]
16. Behrens I, Basit S, Lykke JA, et al. Hypertensive disorders of pregnancy and peripartum cardiomyopathy: a nationwide cohort study. *PLoS One* 2019;14(02):e0211857 [PubMed: 30785920]
17. Louis JM, Mogos MF, Salemi JL, Redline S, Salihu HM. Obstructive sleep apnea and severe maternal-infant morbidity/mortality in the United States, 1998–2009. *Sleep (Basel)* 2014;37(05):843–849
18. Shani H, Kuperstein R, Berlin A, Arad M, Goldenberg I, Simchen MJ. Peripartum cardiomyopathy - risk factors, characteristics and long-term follow-up. *J Perinat Med* 2015;43(01):95–101 [PubMed: 24887948]
19. Witlin AG, Mabie WC, Sibai BM. Peripartum cardiomyopathy: an ominous diagnosis. *Am J Obstet Gynecol* 1997;176(1 Pt 1):182–188 [PubMed: 9024111]
20. Loftin R, Van Hook J. Peripartum Cardiomyopathy and Pulmonary Edema. *Management of Acute Obstetric Emergencies*: Elsevier; 2011:85–92
21. Peripartum Cardiomyopathy (PPCM). 2015 [cited 2018 Aug 28]; Available at: <https://www.heart.org/en/health-topics/cardiomyopathy/what-is-cardiomyopathy-in-adults/peripartum-cardiomyopathy-ppcm>. Accessed August 28, 2018

22. Deputy NP, Dub B, Sharma AJ. Prevalence and trends in prepregnancy normal weight - 48 states, New York City, and District of Columbia, 2011-2015. *MMWR Morb Mortal Wkly Rep* 2018;66 (51-52):1402–1407 [PubMed: 29300720]
23. Van Tintelen JP, Pieper PG, Van Spaendonck-Zwarts KY, Van Den Berg MP. Pregnancy, cardiomyopathies, and genetics. *Cardiovasc Res* 2014;101(04):571–578 [PubMed: 24451172]
24. Wong C, Marwick TH. Obesity cardiomyopathy: diagnosis and therapeutic implications. *Nat Clin Pract Cardiovasc Med* 2007;4 (09):480–490 [PubMed: 17712361]
25. Kao DP, Hsich E, Lindenfeld J. Characteristics, adverse events, and racial differences among delivering mothers with peripartum cardiomyopathy. *JACC Heart Fail* 2013;1(05):409–416 [PubMed: 24163791]
26. Malhamé I, Dayan N, Moura CS, Samuel M, Vinet E, Pilote L. Peripartum cardiomyopathy with co-incident preeclampsia: a cohort study of clinical risk factors and outcomes among commercially insured women. *Pregnancy Hypertens* 2019;17:82–88 [PubMed: 31487662]
27. Krishnamoorthy P, Garg J, Palaniswamy C, et al. Epidemiology and outcomes of peripartum cardiomyopathy in the United States: findings from the Nationwide Inpatient Sample. *J Cardiovasc Med (Hagerstown)* 2016;17(10):756–761 [PubMed: 25943626]
28. Kuklina EV, Callaghan WM. Cardiomyopathy and other myocardial disorders among hospitalizations for pregnancy in the United States: 2004-2006. *Obstet Gynecol* 2010;115(01):93–100 [PubMed: 20027040]
29. Pearson GD, Veille JC, Rahimtoola S, et al. Peripartum cardiomyopathy: national heart, lung, and blood institute and office of rare diseases (national institutes of health) workshop recommendations and review. *JAMA* 2000;283(09):1183–1188 [PubMed: 10703781]
30. Quan H, Li B, Saunders LD, et al.; IMECCHI Investigators. Assessing validity of ICD-9-CM and ICD-10 administrative data in recording clinical conditions in a unique dually coded database. *Health Serv Res* 2008;43(04):1424–1441 [PubMed: 18756617]
31. Martin B-J, Chen G, Graham M, Quan H. Coding of obesity in administrative hospital discharge abstract data: accuracy and impact for future research studies. *BMC Health Serv Res* 2014; 14:70–70 [PubMed: 24524687]
32. Wong C, Marwick TH. Obesity cardiomyopathy: pathogenesis and pathophysiology. *Nat Clin Pract Cardiovasc Med* 2007;4(08): 436–443 [PubMed: 17653116]
33. Timoh T, Bloom ME, Siegel RR, Wagman G, Lanier GM, Vittorio TJ. A perspective on obesity cardiomyopathy. *Obes Res Clin Pract* 2012;6(03):e175–e262
34. Alpert MA. Obesity cardiomyopathy: pathophysiology and evolution of the clinical syndrome. *Am J Med Sci* 2001;321(04): 225–236 [PubMed: 11307864]
35. Dela Cruz CS, Matthay RA. Role of obesity in cardiomyopathy and pulmonary hypertension. *Clin Chest Med* 2009;30(03):509–523, ix [PubMed: 19700049]
36. Lydon-Rochelle MT, Holt VL, Cardenas V, et al. The reporting of pre-existing maternal medical conditions and complications of pregnancy on birth certificates and in hospital discharge data. *Am J Obstet Gynecol* 2005;193(01):125–134 [PubMed: 16021070]
37. Headen I, Cohen AK, Mujahid M, Abrams B. The accuracy of self-reported pregnancy-related weight: a systematic review. *Obes Rev* 2017;18(03):350–369 [PubMed: 28170169]
38. Endo D, Morikawa M, Sakakibara M, Sugita T, Yamada T, Minakami H. Extraordinary weight gain: initial finding in a patient with peripartum cardiomyopathy. *Case Reports Perinatal Medicine* 2014;3(01):x
39. Matsumiya H, Saito N, Minakami H, Kataoka S. Gestational weight gain and peripartum cardiomyopathy in a twin pregnancy. *Case Rep Obstet Gynecol* 2015;2015:317146 [PubMed: 26221550]
40. Sakamoto A, Umazume T, Morikawa M, et al. Gestational weight gain in a woman with peripartum cardiomyopathy. *Crit Care Obst Gyne* 2016;2(04):x
41. Umazume T, Yamada T, Yamada S, Minakami H. Peripartum cardiomyopathy in a woman with preeclampsia with twin pregnancy. *BMJ Case Rep* 2014;2014:2014
42. Statistics CNCfH. State and Territorial Data. CDC/National Center for Health Statistics [web page] 2017 3 31, 2017; Available at: <https://www.cdc.gov/nchs/fastats/state-and-territorial-data.html>. Accessed March 14, 2019

43. Leonard SA, Main EK, Carmichael SL. The contribution of maternal characteristics and cesarean delivery to an increasing trend of severe maternal morbidity. *BMC Pregnancy Childbirth* 2019;19(01):16 [PubMed: 30626349]

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Key Points

- Pre-pregnancy obesity affects maternal health.
- Effects may extend to peripartum cardiomyopathy.
- The risk includes peripartum cardiomyopathy that emerges postpartum.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

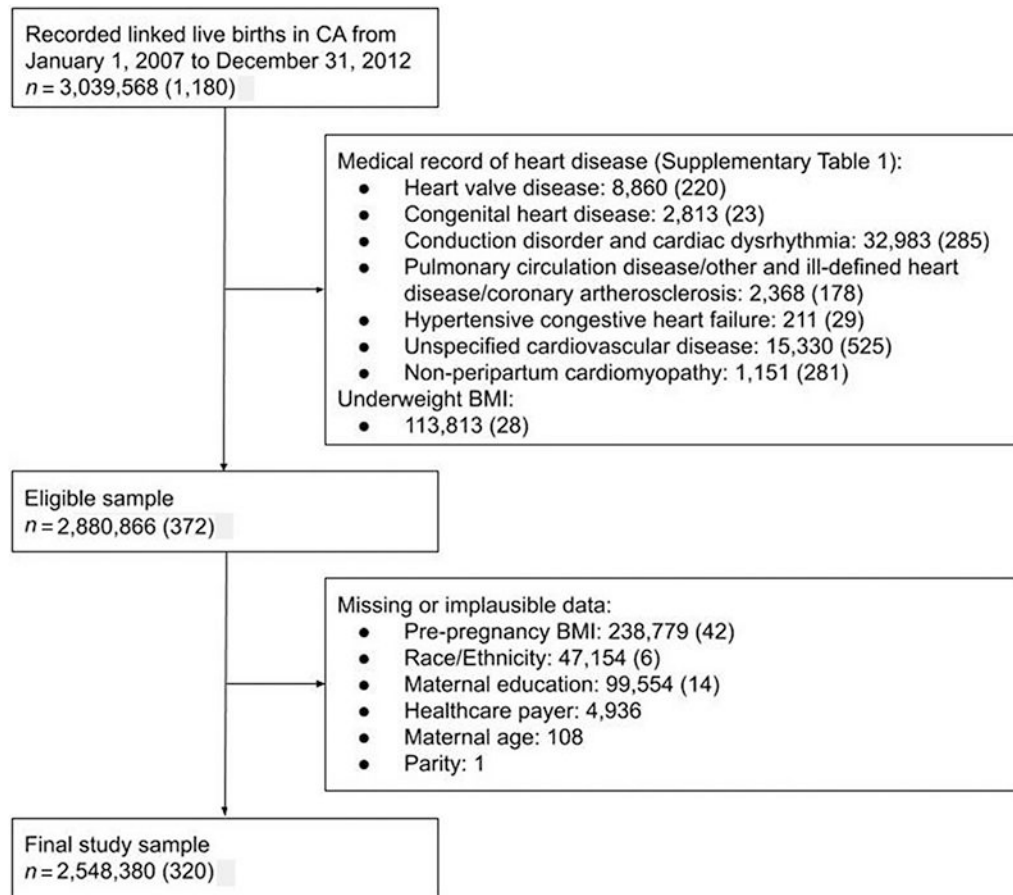


Fig. 1. Selection of study population. Numbers of peripartum cardiomyopathy cases are shown in parentheses.

Table 1 Characteristics of women with and without peripartum cardiomyopathy, California from 2007 to 2012

	Women with PPCM (n = 320)	Women without PPCM (n = 2,548,060)	p-Value
Pre-pregnancy BMI, kg/m ² (%)			<0.001
Normal weight (18.5–24.9)	39.4	51.5	
Overweight (25.0–29.9)	27.2	26.9	
Obese class I (30.0–34.9)	15.9	13.1	
Obese class II (35.0–39.9)	9.1	5.4	
Obese class III (≥ 40)	8.4	3.1	
Race/ethnicity (%)			<0.001
Foreign-born Hispanic	13.1	28.3	
US-born Hispanic	23.8	24.7	
Non-Hispanic White	29.1	28.1	
Asian/Pacific Islander	15.0	12.7	
Non-Hispanic Black	18.8	5.7	
Other	<1	<1	
Education (%)			0.002
< High school	16.6	24.5	
High school equivalent	26.3	26.3	
> High school	57.8	49.2	
Health care payer (%)			0.328
Nonprivate	50.0	52.7	
Private	50.0	47.3	
Age in years (mean ± SD)	30.46 ± 7.01	28.36 ± 6.27	<0.001
Parity (%)			0.044
Primiparous	44.7	39.2	
Multiparous	55.3	60.8	
Gestational age at birth (%)			<0.001
< 32 weeks	5.3	1.4	
32–36 weeks	19.1	6.5	
37 weeks	75.6	92.0	

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

	Women with PPCM (n = 320)	Women without PPCM (n = 2,548,060)	p-Value
Plurality (%)			<0.001
Singleton birth	89.4	98.5	
Multiple births	10.6	1.6	
Pre-pregnancy comorbidities (%)			
Diabetes mellitus	5.6	1.3	<0.001
Hypertension	26.9	2.6	<0.001
Pregnancy-related hypertensive disorders (%)			<0.001
None	61.9	93.7	
Gestational hypertension	4.7	1.6	
Preeclampsia	29.7	4.5	
Eclampsia	3.8	<1	

Abbreviations: BMI, body mass index; PPCM, peripartum cardiomyopathy; SD, standard deviation.

Table 2

Peripartum cardiomyopathy prevalence by pre-pregnancy body mass index and odds ratios for associations with pre-pregnancy body mass index, California from 2007 to 2012 ($n = 2,548,380$)

Pre-pregnancy BMI	Total number of women in each group	Peripartum cardiomyopathy prevalence per 10,000 births (<i>n</i> of cases)	Unadjusted OR (95% CI)	Adjusted ^a OR (95% CI)
Normal weight	1,312,660	0.96 (126)	Reference	Reference
Overweight	685,958	1.27 (87)	1.32 (1.01–1.74)	1.26 (0.95–1.66)
Obesity class 1	333,998	1.53 (51)	1.59 (1.15–2.20)	1.30 (0.93–1.82)
Obesity class 2	136,403	2.13 (29)	2.22 (1.48–3.32)	1.42 (0.93–2.18)
Obesity class 3	79,361	3.40 (27)	3.55 (2.34–5.37)	1.57 (1.00–2.47)
Obese total	549,762	1.95 (107)	2.03 (1.57–2.62)	1.38 (1.04–1.84)

Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio.

^a Adjusted for maternal race/ethnicity, education, health care payer, age, parity, plurality, pre-pregnancy diabetes mellitus, and pre-pregnancy hypertension.