CODEN [USA]: IAJPBB ISSN : 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES
SJIF Impact Factor: 7.187
http://doi.org/10.5281/zenodo.4394610

Available online at: http://www.iajps.com *Research Article*

"THE IMPACT OF MATERNAL OBESITY ON MATERNAL AND FETAL HEALTH"

[1]Dr Atif Ikram,[2]Dr Tahira Batool, [3]Dr Saman Zahoor

[1]MBBS, University College of Medicine and Dentistry, University of Lahore, Lahore.
[2]MBBS, University Medical and Dental College, Fasialabad.
[3]MBBS, Islamic International Medical College, Rawalpandi.

Article Received: July 2020 **Accepted:** August 2020 **Published:** September 2020

Abstract:

The rise in obesity among mothers is a major concern in obstetrics. Maternal obesity can cause both women and fetuses to lose out. During breastfeeding, maternal complications include pre-eclampsia and gestational diabetes. Fetal disorder and congenital defects are at risk. Pregnancy obesity can also affect mother and infant health later in life. The risks for women include heart failure and high blood pressure. The risk for potential obesity and heart disease rests with children. The risk for diabetes is rising for women and their offspring. Gynecologists are well equipped to stop and manage this crisis.

Corresponding author:

Dr. Atif Ikram,

MBBS, University College of Medicine and Dentistry, University of Lahore, Lahore Rural

QR code

Please cite this article in press Atif Ikram et al, ***The Impact Of Maternal Obesity On Maternal And Fetal Health.,*** *Indo Am. J. P. Sci, 2020; 07(09).*

INTRODUCTION:

Over the past few decades, the global prevalence of obesity has dramatically increased. Changes in the environment, technology and lifestyles produced plenty of cheap, high-calorie food and decreased physical activity (1). We eat more, and walk less. There is a variety of potential environmental causes, including pollutants from modern factories, correlated with metabolic dissegulation among obese individuals (2). The risk of several complications in pregnancy, including preeclampsia, gestational diabetes mellitus (GDM), and caesarean delivery, is increased due to maternal obesity. Excessive weight gain in infancy and continuation of infancy are major factors in women's subsequent obesity. Maternal health can have a major impact on the utero environment and, therefore, on fetal development and child health later in life (3).

According to the Barker hypothesis in utero fetal programming, birth size is related to the risk of later life disease development. Although the Barker theory was initially based on low birth weight, there is some evidence that later in life high birth weight may have its own set of complications. A correlation between first-quarter maternal obesity and childhood obesity was shown (4).

One factor that is believed to underlie these interactions is dietary stimulation in utero fetal programming. Fetuses must adapt to the supply of nutrients that enter the placenta, whether a shortage or an overabundance, and these changes can alter their physiology and metabolism permanently (5). These programmed improvements can serve as the cause of a number of later-life diseases, including heart disease, asthma, and non-insulin - dependent diabetes. In addition, obesity may become a self-perpetuating epidemic due to foetal programming (6).

Definitions of obesity

The term most widely used to describe obesity is BMI, which refers to the weight of a person in kilograms divided by the square of his or her height in meters (7). Persons with BMIs of 25 to 30 kg / m2, are considered overweight; obesity is classified as BMIs of 30 kg / m2 or higher and extreme obesity as BMIs of 40 kg / m2 or more. But it's important to remember that BMI can be confusing. Weight lifters, for example, and elite athletes appear to have elevated BMI because they have an elevated body mass and not extra fat (8). The health consequences of obesity arise from excess adipose tissue, not the height of the body, and are not at risk of the metabolic health. Despite this constraint, BMI remains the best instrument available from a wide perspective on health policy since it is simple to quantify (9).

Biology of adipose tissue

Fat is an important tissue which performs a wide range of roles, including dietary, hormonal and even structural support. The body's largest depots of fat are adipose. Adipocytes are cells primarily engineered for the storing of fat that act as a potential supply of energy and help reduce the harmful effects of the toxic deposition of cell lipids in organs like the stomach, liver and heart. Adipose, however, is not an organ that is inert. It controls metabolism effectively through different, but overlapping, paths (10).

Adipose tissue also includes a diverse variety of nonfat cells, including mast cells, macrophages and leukocytes, and immune cells. These causes are known to affect local and systemic physiology and are a synthesis and secretion of various peptide, steroid hormones, cytokines, and physiologic chemokine. Thus adipose tissue acts as an endocrine organ, causing much of the pathology of obesity through the metabolism of adipose tissue (11).

Tissue adipose functions in many ways as an endocrine cell. This extracts and activates steroid hormones preformed and transforms precursors to biologically active hormones. Adipocytes are also used to express a variety of enzymes important to the biosynthesis and metabolism of steroid hormones. Estrone in peripheral adipose tissue, for example, is transformed to estradiol. Indeed, in postmenopausal women, much if not all of the circulating estradiol comes from adipose tissue (12).

Adipose tissue expresses 11 adjacent hydroxysteroid dehydrogenase type 1 (11 adjacent to HSD1), which converts cortisone to cortisol, as well as 5 adjacent reductase, which converts cortisol to 5 adjacent tetrahydrocortisol, respectively. Thus adipose tissue controls the local glucocorticoid production and contributes to its metabolic clearance. Adipose tissue ultimately secretes a vast number of bioactive peptides and cytokines, commonly known as adipokines (13). Fat is good in our diet and on our bodies, as long as it remains in balance. Too much fat becomes maladaptive, and forced past optimal control, natural physiology becomes disease, a phenomenon known as allostatic surcharge. Pathology in the sense of obesity progresses outside the tolerable therapeutic spectrum leading to an accumulation in adipose tissue. The metabolic effect of obesity of hyperplasia in the endocrine organ is thus similar to endocrine dysfunctions. Consider the physiological and cardiovascular implications of multiplying the size of the liver, thyroid or adrenal gland (14).

Source of Data on Obesity

The main source of longitudinal statistics on obesity and overweight is the Annual Health and Diet Review Test, which involves a detailed take-home assessment and a physical test in a mobile testing center. A main advantage of NHANES is that the calculation of height and weight is uniform and thus the precise estimation of BMI is possible. The current community based tracking system that analyses pregnancy obesity rates through maternal demographic and behavioral features and is a source of obesity data. Pregnancy Risk Assessment management system PRAMS gathers autonomous evidence from maternal pregnancy-related behavior. PRAMS gathers evidence on pregnancy-associated attitudes from maternal questionnaires. The NVSS includes birth records as recorded on certificates of birth which makes it a convenient way to gather sufficient details. Unfortunately, there are drawbacks to all these data sets. Similarly, national data gathered by the NVSS on birth certificates include mother weight, but not height.

Patterns of Maternal Obesity

PRAMS data found that pregnancy obesity prevalence rose by 69 percent over a 10-year period, from 13 percent in 1996-1999 to 22 percent in 2018-2019. Maternal obesity expanded in this study across all age categories; race; education. The National Institutes of Health's Institute of Medicine (IOM) and National Heart, Lung, and Blood Institute have set standards for safe levels of weight gain during pregnancy (15). PRAMS results found that only 1 in 3 women had weight gain that was consistent with IOM guidelines. Race and racial influences have a strong effect on weight gain during pregnancy.

According to Brawarsky and colleagues, African American women are more likely to be overweight before pregnancy and were more likely to gain weight above the IOM recommendations, white women were more likely to achieve target weight gain, Hispanic women were less likely to achieve target gains and Asian women were more likely to gain less than the required weight gain (16).

The postpartum cycle may be a crucial time for long-term weight gain and for maternal obesity to evolve. Excess weight gain and chronic weight loss 1 year postpartum after breastfeeding are good predictors of overweight a decade or more later. According to the National Maternal and Child Health Survey, more than 30 per cent of women maintained 14 lb. or more according to their pregnancy weight recall, with African American women showing a greater weight gain after pregnancy and less postpartum weight loss. A more recent research found that at least 11 lb. 1 year postpartum was maintained by 12 per cent of women. These women were more likely to have gained excessive weight during pregnancy, and to be younger, heavier before pregnancy, nonwhite, unmarried, primiparous, and of lower socioeconomic status. Weight preservation from prior pregnancies and the level of health care obtained between pregnancies tend to be significant determinants of the resulting weight of pregnancy for multiparous females (17). Some researchers have indicated that more rigorous postpartum treatment in women who are overweight or obese (such as graded exercise and weight loss programmers) may have a major effect on subsequent outcome of pregnancy, although that remains to be proved unequivocally. Importantly, in a large epidemiologic study in Sweden, an increase in interpregnancy BMI (by at least 3 kg/m2) was associated with a higher risk of adverse pregnancy outcomes.

Effect of Obesity on Maternal Complications in Pregnancy

Maternal obesity increases the risk of a number of pregnancy complications and, as such, requires adjustment to routine prenatal care. Maternal obesity is a risk factor for spontaneous abortion (for both spontaneous conceptions and conceptions achieved through assisted reproductive technology), as well as for unexplained stillbirth (intrauterine fetal demise). A recent meta-analysis of 9 studies revealed that obese pregnant women have an estimated risk of stillbirth that is twice that of normal weight pregnant women (18). Several mechanisms have been proposed for this relationship, including the increased risks of hypertensive disorders and gestational diabetes that are associated with maternal obesity during pregnancy.

Maternal obesity is associated with elevated risk of pregnancy hypertensive disorders like preeclampsia (gestational proteinuric hypertension), with an odds ratio (OR) ranging from 2 to 3. The risk increases linearly with BMI increasing. A significant 2-fold rise in risk of contracting preeclampsia occurs with each rise in BMI of 5 to 7 kg / m2.

Around the time of labor and childbirth obese women are at greater risk of complications. The rate of successful vaginal delivery is steadily decreasing as maternal BMI rises. The caesarean delivery average for women weighing less than 200 lb. was 18 percent compared to 39.6 percent for women who were listed as highly obese, according to Ehrenberg and colleagues (19). This 2 to 3-fold rise in the delivery rate of caesareans is true for both prim gravid and multigravid females. It's not clear if this is due to increased fetal size or other maternal characteristics.

The success rate of attempted vaginal birth after caesarean (VBAC) is also affected by maternal obesity. Carroll and colleagues found that women weighing less than 200 lb. had an 81.8 percent VBAC performance rate compared to 57.1 percent for women weighing 200-300 lb. and 13.3 percent for women weighing more than 300 lb. In a subsequent analysis using BMI rather than total maternal weight, a similar association was found with VBAC performance rates ranging from 84.7 percent in women with BMI below 19.8 kg / m2 to 54.6 percent in those with BMI over 30 kg / m (20). Besides an elevated rate of surgery, intraoperative complications, including higher infectious morbidity and thromboembolic cases, are also growing. The risk of anesthetic problems such as missed intubation during general endotracheal anesthesia is also increased. Several clear guidelines to eliminate intra-operative risks in obese pregnant women have been proposed.

Effect of Maternal Obesity on Perinatal Outcome

Maternal obesity correlates with an irregular development of the fetus. Women that are more serious are less likely to have a pregnancy complicated by a small for gestational age baby or restriction of intrauterine development, but this preventive effect tends to dissipate as the maternal BMI exceeds the level of obesity (almost 30 kg / m2). Fetal macrosomal (defined as an average fetal weight greater than or equal to 4500 g) is the main concern in obese pregnant women, which tends to be increased 2- to 3-fold in obese parturient (21).

Moreover, the relationship between maternal obesity and fetal macrosomia tends to be dosage dependent. In a new meta-analysis, fetal macrosomia prevalence rates were 13.3 percent and 14.6 percent, respectively, for obese and morbidly obese individuals, compared to 8.3 percent for the average weight management group. The mean birth weight in the United States between 1985 and 1998 increased from 3423 to 3431 g among whites, and from 3217 to 3244 g among blacks (22). The mean birth weight in Canada increased from 3391 to 3427 over the same period. In Denmark, the mean birth weight rose from 3474 g to 3519 g (an rise of 45 g) between 1990 and 1999 and macrosomia rates rose from 16.7% to 20% (23).

Fetal macrosomy in obese women is not only associated with a development of the fetal's total size but also with a shift in body shape. The average fat mass of babies from mothers with a typical BMI (25 kg / m2) was found by Sewell and colleagues in 334 g, giving a body fat composition of 9.7 percent. In the case of women with a BMI = 25 kgs / m2, the total fat mass was 416 g or 11.6 per cent. It should be remembered that the bulk of the impact is due to weight gain during pregnancy. In particular, the pregnancy BMI only tends to account for 6.6% of the difference found in baby fat and just 7.2% of body fat.

Maternal obesity, and even after race, maternal age, gender, socioeconomic status, is associated with an increased risk of neural tube defect (NTD). BMI growth of 1 kg / m2 was associated with an increased 7 percent chance of a child being infected with NTD in Watkins and co-workers. Latest meta-analysis carried out by Rasmussen as well as other colleagues reported for the birth of a child with NTD of overweight, obese and unhealthy obese women 1.22 (95%, 0.991.49), 1.70% (95 % CI, 1.34%-2.15), and 3.11 (95% CI, 1.75%-5.46), respectively (24).

It is not clear what cause is causing an elevated risk of NTD in maternal obesity pregnancies. There are a few reports, however, that the amount of folic acid that enters the developing embryo is decreased because of inadequate synthesis and higher maternal metabolic demands, and chronic hypoxia and elevated triglyceride secretion, uric acid, estrogen and insulin production (due in part to elevated insulin resistance).

CONCLUSIONS:

There is to be an unprecedented rise in the prevalence of maternal obesity and its related comorbid conditions (diabetes, cardiovascular disease). The effects are important to public health. Maternal obesity affects not only mothers, but also the health of children, contributing to greater obesity and diabetes in youth. While we are more aware of this endocrinopathy, certain challenges appear to occur with respect to these women's health treatment. Obstetric gynecologists are at the heart of the disease prevention and recovery.

REFERENCES:

1- Carroll CS Sr, Magann EF, Chauhan SP, et al. Vaginal birth after cesarean section versus elective repeat cesarean delivery: weight-based outcomes. Am J Obstet Gynecol. 2003;188: 1516-1520.
2. Durnwald C, Ehrenberg H, Mercer B. The impact of maternal obesity and weight gain on VBAC success. Am J Obstet Gynecol. 2003;189:S205.
3. Soens MA, Birnbach DJ, Ranasinghe JS, van Zundert A. Obstetric anesthesia for the obese and morbidly obese patient: an ounce of prevention is worth more than a pound of treatment. Acta Anaesthesiol Scand. 2008;52:6-19.
4. Vahratian A, Zhang J, Troendle JF, et al. Maternal pre-pregnancy overweight and obesity and the pattern of labor progression in

term nulliparous women. Obstet Gynecol. 2004;104:943-951.
5. Nuthalapaty FS, Rouse DJ, Owen J. The association of maternal weight with cesarean risk, labor duration, and cervical dilation rate during labor induction. Obstet Gynecol. 2004;103: 452-456.
6. Denison FC, Price J, Graham C, et al. Maternal obesity, length of gestation, risk of postdates pregnancy and spontaneous onset of labour at term. BJOG. 2008;115:720-725.
7. Ehrenberg H, Mercer B, Catalano P. The influence of obesity and diabetes on the prevalence of
macrosomia. Am J Obstet Gynecol. 2004;191: 964-968.
8. Anath CV, Wen SW. Trends in fetal growth among singleton gestations in the United States and Canada, 1985 through 1998. Semin Perinatol. 2002;26:260-267.
9. Ørskou J, Kesmodel U, Henriksen TB, Secher NJ. An increasing proportion of infants weigh more than 4000 grams at birth. Acta Obstet Gynaecol Scand. 2001;80:931-936.
10. Surkan PJ, Hsieh CC, Johansson AL, et al. Reasons for increasing trends in large for gestational age births. Obstet Gynecol. 2004;104: 720-726.
11. Sewell MF, Huston-Presley L, Super DM, Catalano P. Increased neonatal fat mass, not lean body mass, is associated with maternal obesity. Am J Obstet Gynecol. 2006;195: 1100-1103.
12. Hull HR, Dinger MK, Knehans AW, et al. Impact of maternal body mass index on neonate birthweight and body composition. Am J Obstet Gynecol. 2008;198:416.e1-416.e6.
13. Catalano PM, Ehrenberg HM. The short- and long-term implications of maternal obesity on the mother and her offspring. BJOG. 2006;113: 1126-1133.
14. Waller DK, Mills JL, Simpson JP, et al. Are obese women at higher risk for producing malformed offspring? Am J Obstet Gynecol. 1994;170: 541-548.
15. Watkins ML, Rasmussen SA, Honein MA, et al. Maternal obesity and risk for birth defects. Pediatrics. 2003;111:1152-1158.
16. Rasmussen SA, Chu SY, Kim SY, et al. Maternal obesity and risk of neural tube defects: a metaanalysis. Am J Obstet Gynecol. 2008;198:611-619.
17. Hedderson MM, Williams MA, Holt VL, et al. Body mass index and weight gain prior to pregnancy and risk of gestational diabetes mellitus. Am J Obstet Gynecol. 2008;198:409.e1-409.e7.
18. Chu SY, Callaghan WM, Kim SY, et al. Maternal obesity and risk of gestational diabetes mellitus. Diabetes Care. 2007;30:2070-2076.
19. Radaelli T, Varastehpour A, Catalano P, Haugueldе Mouzon S. Gestational diabetes induces placental genes for chronic stress and inflammatory pathways. Diabetes. 2003;52:2951-2958.
20. Yogev Y, Langer O. Pregnancy outcome in obese and morbidly obese gestational diabetic women. Eur J Obstet Gynaecol Reprod Biol. 2008;137: 21-26.
21. Catalano PM, Thomas A, Huston-Presley L, Amini SB. Increased fetal adiposity: a very sensitive marker of abnormal in utero development. Am J Obstet Gynecol. 2003;189:1698-1704.
22. Gillman MW, Rifas-Shiman S, Berkey CS, et al. Maternal gestational diabetes, birth weight, and adolescent obesity. Pediatrics. 2003;111:e221-e226.
23. Honda K. Factors underlying variation in receipt of physician advice on diet and exercise: applications of the behavioral model of health care utilization. Am J Health Promot. 2004;18: 370-377.
24. Power ML, Cogswell ME, Schulkin J. Obesity prevention and treatment practices of U.S. obstetrician-gynecologists. Obstet Gynecol. 2006; 108:961-968.