The obesity and pregnancy: A review

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ABSTRACT: One of particular interest has been the impact of overweight and obesity on maternal and newborn health. Biomedical research has equipped us with a breadth of knowledge about the physical health implications of overweight and obesity in pregnancy for both mothers and babies, such as infertility, hypertension, gestational diabetes, caesarean delivery, hemorrhage, congenital abnormalities and late fetal death. This literature review based on the previous researches on PubMed database, focuses on the implications of maternal overweight and obesity, the experiences of pregnant women who are overweight or obese, and the provision of maternity care for overweight or obese women.

KEYWORDS: Obesity, Pregnancy, Women

1. INTRODUCTION

Obesity and overweight are not only problem of developed nations but also an increasing problem in developing countries with health consequences. Studies have highlighted the link between obesity, infertility and adverse reproductive health outcome. Obesity contributes majorly to two most common medical risks in pregnancy: diabetes and hypertension. It is associated with increased risk of large-for-gestational age and hypertensive disorders, including pre-eclampsia, which may be associated with low birth weight. Combination of factors via individual lifestyle, environmental and genetic play role in obesity. Weight loss has demonstrated to improve fertility in obese women through recovery of spontaneous ovulation, and improved response to ovarian stimulation in infertility treatment. Therefore, weight management interventions, should be considered for overweight and obese infertile women [1]. So, the aim of the literature review with a PubMed search was to determine new findings on effects of maternal overweight and obesity on pregnancy.

2. DEFINITION OF OBESITY
Overweight and obesity are increasing dramatically among the adults. There was an increase in the prevalence of obesity among adults 20-74 years of age [2]. The adverse effects of obesity may be more severe for women than men. Furthermore, women are more likely to be obese compared to the men. In addition, women are twice as likely as men to experience a major weight gain over a 10-year period [3].

3. DETERMINATION OF THE OBESITY

A common and objective way to define under-, normal- and overweight and obesity is through calculating Body Mass Index (BMI) as body weight in kilograms divided by length squared. Obesity is defined as BMI >30 kg/m². Obesity is subdivided into three classes: class I BMI 30.0–34.9 kg/m², class II BMI 35.0–39.9 kg/m² and class III BMI >40.0 kg/m². For comparison, normal weight is set at BMI 18.5 to <25 kg/m² and the intermediate weight range from BMI 25 to <30 kg/m² for overweight [10].

4. IMPACT OF OBESITY ON MATERNAL HEALTH

Obesity during pregnancy is related to higher overall health care expenditures, measured by length of stay after delivery and use of other services. The majority of this difference is caused by higher cesarean section rates and higher rates of high-risk obstetric conditions such as diabetes and hypertension [9]. Pre-pregnancy obesity contributes to the development of many pregnancy complications including pregnancy-induced hypertension, preeclampsia, gestational diabetes and neonatal death [10]. Weight loss via bariatric surgery decrease many pregnancy complications. A retrospective cohort study that included 585 women who had undergone bariatric surgery found that women who had delivered children after surgery (as compared with women who delivered before surgery) had decreased rates of hypertension during pregnancy [11].

5. OBESITY AND INFERTILITY

The impact of obesity on reproduction starts at a young age. Obese girls frequently experience the onset of puberty at a younger age than their normal-weight peers [4]. Obesity negatively affects contraception. Studies have shown hormonal contraception
methods are less effective in obese women. For example, a retrospective cohort analysis of 2822 person-years of oral contraceptive use suggested that women in the highest quartile of body weight (>70.5 kg) had a 60% higher risk of failure than women of lower weight [5]. A retrospective cohort study of 22840 women demonstrated that obesity was associated with reduced fecundity for all weight-adjusted groups of women and persisted for women with regular cycles. In addition, obesity may alter the quality of oocytes and embryos [8]. Some studies demonstrate increased female sexual dysfunction in obese patients, whether caused by the physical or psychological impacts of obesity on female sexuality. Cross-sectional studies indicate that 30-47% of overweight and obese women have irregular menses [6]. Weight loss can improve the fertility of obese women by the return of spontaneous ovulation, thus leading to the recommendation of implementing weight-loss interventions such as diet, exercise and medication treatment as initial management of infertile overweight and obese women [7].

A systematic review of IVF outcomes among overweight and obese women demonstrated that the dose of gonadotrophins required was higher in women with BMI of>25 kg/m² in comparison with those with BMI of <25 kg/m². Gonadotrophin requirements were higher in obese women when compared to non-obese women [32]. A large cohort study has shown that in comparison with women of normal weight, overweight women have significantly fewer oocytes retrieved. Also, oocyte fertilization rates have been shown to be lower in morbidly obese women [33].

6. OBESITY AND PREGNANCY

Once obese women are pregnant, their risk of pregnancy complications is significantly greater than their lean counterparts. As reviewed by Catalano [12] in this issue, many of the adverse outcomes of obesity (including gestational diabetes and pre-eclampsia) occur as a result of increased insulin resistance. Denison et al. [13] review the contribution of adipose tissue and the placenta to the biology of obese pregnant women. There is a clear link between adipose tissue invasion of T cells and macrophages, increased circulating cytokines of adipose tissue origin and insulin resistance in non-pregnant individuals but less information on whether this also occurs in pregnancy [12].

The increased insulin resistance in obese pregnant women leads not only to pregnancy complications for the mother, but also greater growth and disproportionately greater fat mass for the baby. Thus, high pre-pregnancy and early pregnancy BMI predisposes to the birth of an overweight baby [15]. Fetal macrosomia in obese women is associated not only
with an increase in the absolute size of the fetus, but also in a change in body composition. Sewell and coworkers [14] found that the average fat mass of infants born to mothers with a normal BMI (<25 kg/m2) was 334 g, giving a body fat composition of 9.7%. The offspring of women with a BMI > 25 kg/m2, on the other hand, had a mean fat mass of 416 g, or a body fat composition of 11.6%. Of note, the majority of this effect appears to be a result of weight gain during pregnancy [16].

7. INTERACTION OF ADIPOSE WITH FERTILITY AND PREGNANCY

Adipose tissue functions as an endocrine organ in a number of ways. It stores and releases preformed steroid hormones, converts precursors to biologically active hormones and converts active hormones to inactive metabolites. To this end, adipocytes express a number of enzymes critical to steroid hormone biosynthesis and metabolism [17]. For example, estrone is converted to estradiol in peripheral adipose tissue. Indeed, most if not all circulating estradiol in postmenopausal women comes directly from adipose tissue. Adipose tissue expresses 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1), which converts cortisone to cortisol, as well as 5α-reductase, which converts cortisol to 5α-tetrahydrocortisol. Thus, adipose tissue regulates the local concentration of glucocorticoids and contributes to their metabolic clearance. Finally, adipose tissue secretes a large number of bioactive peptides and cytokines, collectively known as adipokines [18].

Fat in our diet and on our bodies is beneficial as long as it exists in moderation. Too much fat becomes maladaptive, and normal physiology pushed beyond adaptive function becomes pathology, a concept referred to as allostatic overload. In the setting of obesity, pathology develops because of an increase in adipose tissue beyond the tolerable functional range. In this way, the metabolic consequences of obesity are analogous to the endocrine dysfunction seen in hyperplasia of any endocrine organ. Consider for a moment the metabolic and health consequences if a person’s liver, thyroid, or adrenal gland doubled in size [19].

Body fat distribution has been shown to substantially affect SHBG concentrations. Fat accumulation in the abdominal viscera (visceral fat) has been described as a possible cause of insulin resistance and the resulting metabolic syndrome. Women with central obesity and with higher proportion of visceral fat usually have high insulin resistance leading to lower SHBG concentrations in comparison with those with peripheral obesity (Ramón et al. 2008).

In insulin resistance syndrome, excess insulin is capable of stimulating steroidogenesis, excessive androgen production from the theca cells and excessive estrogen production...
from the granulosa cells of the ovaries. In addition, by directly inhibiting SHBG synthesis, excess insulin may further increase the delivery of free androgens to target tissues. The excess in local ovarian steroidogenesis induced by excess circulating of insulin may cause prema-ure follicular atresia and then favor anovulation [28].

8. EFFECTS OF UNSATURATED FATTY ACIDS

Two PUFAs, arachidonic acid (AA) and docosahexaenoic acid (DHA) are critical to fetal and infant central nervous system growth and development. Most fatty acids can be synthesised in the body, but humans lack the enzymes required to produce two fatty acids. These are called the essential fatty acids and must be acquired from the diet. In humans, the essential fatty acids are the n-3 polyunsaturated fatty acid α-linolenic acid and the n-6 polyunsaturated fatty acid linoleic acid [20]. Although humans can elongate dietary α-linolenic acid to the long chain n-3 polyunsaturated fatty acids eicosapentaenoic acid and docosahexaenoic acid, the rate of synthesis may not be sufficient to meet requirements and it is therefore recommended that good sources of these fatty acids, namely oil-rich fish, are also included in the diet. Unsaturated fatty acids are now a nutritional hot topic and their presence in foods has attracted both public and industrial interest [21].

Omega-3 fatty acids are essential and can only be obtained from the diet. The requirements during pregnancy have not been established, but likely exceed that of a nonpregnant state. Omega-3 fatty acids are critical for fetal neurodevelopment and may be important for the timing of gestation and birth weight as well. Most pregnant women likely do not get enough omega-3 fatty acids because the major dietary source, seafood, is restricted to 2 servings a week. For pregnant women to obtain adequate omega-3 fatty acids, a variety of sources should be consumed: vegetable oils, low-mercury fish servings a week and supplements [22].

The omega-3 fatty acid EPA and the omega-6 fatty acid AA are essential structural components of every cell in the body. Both EPA and AA serve as precursors for biologically active compounds called eicosanoids. These fatty acids compete for the enzyme systems cyclooxygenase, which makes prostaglandins and thromboxanes, and lipoxygenase, which makes leukotrienes. Diets that are rich in omega-6 fatty acids produce potent eicosanoids, whereas a diet with a more balanced intake of omega-6 and omega-3 fatty acids makes less inflammatory and less immunosuppressive eicosanoids [23]. Specific to pregnancy, although both DHA and AA appear to be essential to fetal CNS development, the relatively poor intake of EPA coupled with the high intake of linoleic acid (which produces AA), may affect pregnancy outcome by altering the balance

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of the eicosanoids produced [23]. A high ratio of AA to EPA may promote untoward effects such as preterm labor and preeclampsia [26].

A linoleic acid–rich diet produces an abundance of AA, which serves as a precursor of the potent 2-series prostaglandins (PGs) E2 and PGF2α, and the vasoconstrictor thromboxane (TX) A2. Both PGE2 and PGF2α are closely associated with the initiation of labor and preterm labor, whereas thromboxane A2 has been associated with preeclampsia. Whereas the omega-3 fatty acid, DHA, is not usually considered to be involved with eicosanoid formation, EPA is a precursor for the 3-series of PGs and produces PGE3 and PGI3, which promote relaxation of myometrium [25]. Also, EPA and DHA competitively displace AA in the membrane phospholipids and thereby reduce production of 2-series eicosanoids. Thus, a diet that provides a closer balance of omega-3 to omega-6 fatty acids may be as important to pregnant women as the absolute individual plasma levels of these fatty acids [24].

9. OBESITY AND LEPTIN

Fertility processes involve a complex of factors and mechanisms of both ovarian and extra ovarian origin. Obesity may interfere with many neuroendocrine and ovarian functions, thereby reducing both ovulatory and fertility rates in otherwise healthy women. Normal ovarian function resulting in normal puberty and reproductive competence is controlled primarily by the gonadotrophins LH and FSH from the pituitary gland, the secretion of which is regulated by the brain hormone, GnRH. Nutrition is linked to the female reproductive system through the effects of a hormone emanating from fat cells (leptin) and by insulin from the pancreas, which alters the bio-availability of estradiol and testosterone by affecting production of SHBG (sex hormone-binding globulin) from the liver. Insulin can also function directly on the ovary [31].

In overweight women and/or those with polycystic ovary syndrome, an increase in the number of fat cells results in a cascade of changes involving increased leptin and insulin levels and a preferential increase in LH, but not FSH levels. The net effect of these changes is to stimulate the partial development of follicles that secrete supra-normal levels of testosterone, but which rarely ovulate (hence low progesterone). These changes are exacerbated by insulin-induced reduction in SHBG which amplifies ovarian testosterone production and action [29]. Leptin plays a potentially important role in human infertility given the discovery of its regulatory effect on fertility in the mouse. There is a strong correlation between serum leptin concentrations and body fat and BMI in humans [30].
Leptin levels have also been reported to be increased in women with polycystic ovary syndrome although this was not supported by many other studies. Given the well-established effect of leptin on ovarian steroidogenesis and ovulation in rodents and in humans, it can be speculated that the high concentration of leptin might have a role in the pathogenesis of polycystic ovary syndrome and reproductive disorders influenced by obesity [27].

10. CONCLUSION

As rates of overweight and obesity have increased in recent years, more attention has been paid to the role that overweight and obesity play on maternal and newborn health. The current review of literature shows that there is a breadth of knowledge about potential physical health implications of maternal overweight and obesity for both mothers and babies. We also know little about health providers’ experiences of providing care to pregnant women with high BMI. Obesity in women has impacts on fertility and fertility treatment. Increase in BMI reduces the chance of conception in ovulatory women and affects the outcome of ovulation induction treatment. More research is needed to explore the psycho-social aspects of overweight and obesity among pregnant women, including studies that consider the intersections of multiple social determinants in the health and well-being of pregnant women with overweight and obesity.

REFERENCES


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