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Impact of maternal overweight and obesity on neonates delivered at Al-Emamain Al-khadamian medical city

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Abstract

The obesity is a problem of global importance with a significant impact on maternal-fetal health. Maternal overweight and obesity cause pregnancy complications such as gestational diabetes, hypertension and preeclampsia and affects fetal growth. The aim of study is to evaluate neonatal complications at birth associated with maternal obesity, expressed as body mass index (BMI) in a sample unselected geographical population. This is a cross-sectional study; it was done at the departments of NICU and Gynecology in Al-Emamain Alkhadamian Medical City hospital in Baghdad from the period 1st of February 2017 to the 31th of December 2017. A total of 514 pregnant women who gave birth in that period were included in this study. We divided them into three groups according to the (BMI) BMI (20-25 kg/m^2) Normal =257 pregnant women, BMI (25.1-30 kg/m^2). Overweight =183 pregnant women & BMI >30 kg/m^2 Obese = 74 pregnant women. Obese women had a significant increase of incidence than average-weight women to have an infant with congenital heart diseases (14.78%), neural tube defect (9.33%), orofacial clefts (13.61%), multiple congenital anomalies (3.3%), RDS (18.67%), hypoglycemia (10.5%), low Apgar score (53.69%), low birth weights (3.5%), macrosomia (21.4%) and preterm and postdate deliveries (95.71%). In conclusions, a significant association between mothers' BMI > 25 and fetal outcomes, the most common congenital anomalies associated with maternal BMI >25 were the congenital heart diseases and neural tube defects. There was an increase in the frequencies of other neonatal complications like: (RDS, low birth weights, macrosomia, hypoglycemia, orofacial clefts, low Apgar score, and others).

Key words: Body Mass Index, Neonatal complication, Obesity

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Introduction

The most commonly used measurement for defining obesity is BMI (Body Mass Index), which refers to an individual's weight in kilograms divided by the square of his or her height

in meters. Individuals are deemed overweight when they have a BMI between 25 and 30 kg/m^2; obesity is defined as a BMI greater than or equal to 30 kg/m^2, and extreme obesity is defined as a BMI greater or equal to 40 kg/m^2 [1]. It has reached a point at which 50% of women of childbearing age are either overweight (BMI 25-29.9) or obese (BMI>30) [2]. Furthermore 15-20% of women starting pregnancy are obese [3, 4] and 20-40% of pregnant women suffer from excessive weight gain during pregnancy. Maternal obesity is an obstetric risk factor because of its potential consequences for mother and offspring. As a result of increased complication rate, more than 50% of mortality cases during pregnancy, childbirth or the puerperium are of women who are either obese or overweight [5]. Obesity is also associated with a marked increase in fetal/neonatal complications including stillbirths [6], neonatal deaths, neonatal intensive care unit admission, preterm births, and congenital abnormalities [7]. Offspring of obese mothers are also subject to disrupted growth patterns (both IUGR and overgrowth). Increased fetal weight and adiposity at birth increases macrosomia and difficulties associated with delivery of large-for-gestational-age infants. It has also long-term effects on neonatal, childhood and adult health [8].

Data from the Pregnancy Risk Assessment Monitoring System (PRAMS) has shown that the prevalence of pre pregnancy obesity increased by 69% over a 10- year period, from 13% in 1993-1994 to 22% in 2002-2003.In this report, maternal obesity increased across all categories of age; race; education; smoking status; Special Supplemental Nutrition Program for Women, Infants, and Children enrollment; and parity [6]. The major concern in obese pregnant women is fetal macrosomia (defined as an estimated fetal weight of greater than or equal to 4000 g), which appears to be increased 2- to 4 3-fold in obese mothers [9]. Moreover, there appears to be a dose dependent relationship between maternal obesity and fetal macrosomia [1]. In a recent meta-analysis, the prevalence rates of fetal macrosomia were 13.3% and 14.6% for obese and morbidly obese women, respectively, compared with 8.3% for the normal weight control group [10]. In the United States, the mean birth weight between 1985 and 1998 increased from 3423 to 3431g among whites and from 3217 to 3244g among blacks. Maternal obesity is associated also with an increased risk of neural tube defect (NTD) in the offspring, even after controlling for ethnicity, maternal age, education, and socioeconomic status [11-12]. Watkins and coworkers [24] concluded that a 1 kg/m2 increase in BMI is associated with a 7% increased risk of having an infant with NTD among overweight, obese, and morbidly obese women, compared with normal weight women. The mechanism underlying the increased risk of NTD in pregnancies complicated by maternal obesity is unknown. However, a number of theories have been proposed, including a reduction in the amount of folic acid reaching the developing embryo due to insufficient absorption and greater maternal metabolic demands, chronic hypoxia, and

increased circulating levels of triglycerides, uric acid, estrogen, and insulin (due, in part, to increased insulin resistance) [11-12]. The exact mechanism in which obesity mediates poor health outcomes for both mother and fetus, are far from clear [1]. Pieces of the puzzle are placed together 5 incorporating knowledge gathered from epidemiological studies, experiments on animals and placenta models. The embryo inherits a given set of genes from both parents. It draws upon this genetic milieu for its continued development and growth. Genome-wide association studies have identified several common genetic variants associated with high adiposity and obesity, each with weak effects [7]. Some of the candidate genes mentioned are those of insulin, insulin-like growth factor and their receptors because these hormones are known to influence fetal growth and body composition [8]. The genetic influence of maternal obesity on the developing child cannot be explained by Mendelian-model inheritance of a given set of genes alone. Children of obese mothers are more at risk of overweight or obesity than those of obese fathers [13]. This illustrates the pivotal role of intrauterine conditions on susceptibility to obesity, type 2 diabetes, and cardiovascular disease in the offspring. Epigenetic processes (is the study of heritable changes in gene function that do not involve changes in the DNA sequence) have a crucial role in determining fetal, neonatal and adult health. The developing fetus depends upon a complex feto-maternal interaction. This intricate interaction relies on several components: maternal nutritional intake, placental transfer mechanisms and uterine blood supply, which depend on maternal metabolic and cardiovascular condition. Glucose was considered the main metabolite that transmits the effect of obesity to the developing fetus. The obese population suffers from elevated blood glucose levels, insulin resistance and high rates of overt diabetes. Obese women are characterized by a significantly higher postprandial glucose peak value, increased 1- and 2-hour postprandial glucose levels, increased time interval for glucose peak, and significantly lower mean blood glucose during the night [14]. Maternal hyperglycemia during pregnancy was thought to be one of the most important predictive factors of pregnancy complications in the obese population. It is now recognized that other maternal parameters associated with obesity and/or over nutrition during pregnancy are also involved including hyperglycemia and hypertriglyceridemia. The altered endocrine milieu associated with obesity (increased levels of insulin, androgens, and leptin) [15] is also associated with several maternal metabolic disturbances such as insulin resistance, diabetes and increased blood pressure, all of which influence fetal well-being. Obesity is associated with a chronic state of inflammation [16] and alternation in the homeostasis of cytokines and adipokines. Adipose tissue is an active endocrine organ and a source of proinflammatory cytokines (adipokines) such as adiponectin. Adiponectin is deemed important in promoting insulin sensitivity and stimulating glucose uptake in skeletal

muscle. In pregnancy, it influences placental transfer of nutrients and placental insulin-stimulated amino acid uptake. Pregnant women suffering from obesity demonstrate low circulating levels of adiponectin. Low levels of adiponectin are associated with increased fetal growth [17]. Adiponectin also binds to the adiponectin receptor-2 on the trophoblast cell and activates p38 6 mitogen-activated protein kinases and Peroxisome proliferator-activated receptor alpha, which inhibits the insulin/Insulin-like growth factor -1 signaling pathway. Therefore, it is not surprising that maternal serum adiponectin is inversely correlated to fetal growth throughout the full range of birth weights. Individuals inflicted by obesity demonstrate high plasma levels of glucose and free fatty acids. The 'accelerated starvation' of pregnancy (meaning the increase in ketosis observed in pregnant women) is responsible for the decrease in insulin's ability to suppress lipolysis in pregnant women. This causes a further two- to threefold increase in the blood levels of cholesterol and free fatty acids with advancing gestation [17]. Maternal high levels of triglycerides (which are not readily transferred across the placenta) alternate placental lipases activity and increase placental transfer of metabolites which are stored as fetal fat deposits, oxidized lipids can be cytotoxic and influence gene expression by acting as ligands for nuclear receptors [18]. They may also affect antenatal organ development and impact postnatal response to environmental stimuli. Furthermore, intrauterine exposure to high lipid levels, which act as both transcriptional activators and signaling molecules, may cause epigenetic changes in lipid sensing and metabolism genes [19]. This may mediate epigenetic changes in the offspring of obese mothers that induce obesity in the offspring. Obesity has associated with tissue-specific changes in mitochondrial function and elevated production of reactive oxygen species leading to increased oxidative stress [20]. The combination of excess fatty acids and oxidative stress leads to the production of oxidized lipids. Oxidized lipids can inhibit trophoblast invasion and influence placental development, lipid metabolism and lipid transport [21], which may explain some of the adverse pregnancy outcomes related to obesity such as preeclampsia. Other changes associated with maternal obesity may also lead to alterations in placental development or function. For example, maternal obesity is associated with a reduction in placental villous proliferation and apoptosis which may increase susceptibility to adverse pregnancy outcomes [22]. Epidemiologic studies also demonstrated correlation between maternal metabolic syndrome and placental dysfunction [23]. Serum concentrations of leptin are positively associated with body fat stores [24]. Maternal obesity is accompanied by maternal hyperleptinemia and placental leptin resistance contributing to alternations in placental function and amino acid transfer [25]. Furthermore, some congenital anomalies are more prevalent in the offspring of obese mothers. The increase in the absolute rate of specific congenital anomalies is increased with

increasing maternal weight. Stothard et al., [26] reviewed 39 observational studies and 18 meta-analyses. They calculated pooled ORs for specific anomalies in the obese population. Compared with mothers of normal BMI, obese mothers were at increased odds of pregnancies affected by neural tube defects (NTD), spina bifida, cardiovascular anomalies, septal anomalies, cleft palate, cleft lip, anorectal atresia, hydrocephaly, and limb reduction anomalies. The risk of gastroschisis among obese mothers was significantly reduced [27]. This risk of NTDs increases with increasing pre-pregnancy weight and may not be ameliorated by increase in folate intake [28]. Maternal obesity adversely affects trans-abdominal ultrasound imaging of fetal anatomy during the second trimester. There is little to be done to technically improve the image obtained. Delaying, repeating, or increasing the duration of the examination may only partially, if at all, mitigate the technical limitations that obesity imposes on visualization [29]. In the obese population early fetal anatomic assessment by trans-vaginal ultrasound may provide an alternative and more satisfactory window for better evaluating fetal anatomy [30]. Later in life, there is an inverse relationship between birth weight and health at both ends of the birth weight curve. Excessive maternal body weight or weight gain during pregnancy disrupts intrauterine environment and brings about permanent changes in the hypothalamus, pancreatic islet cells, adipose tissue and other body weight-regulating mechanisms of the offspring. Maternal pre pregnancy overweight has been found to be an independent risk factor for infant and adolescence overweight and abdominal obesity [31]. Several studies have found that infants born at the highest end of the distribution curve for weight were at a higher risk of being obese in childhood, adolescence and adulthood when compared with normal-sized infants [32, 33]. Some studies also suggest that children exposed to pregnancies complicated by metabolic conditions, such as diabetes, hypertension, and obesity, showed an increased risk of neurodevelopmental disorders, autism spectrum disorders and developmental delay [34], although interpretation of data is limited by lack of information on potential confounders. Maternal over nutrition and/or obesity during pregnancy afflict upon the developing child a fate of later life overweight, thus creating a vicious cycle of epidemic scale. However, this mechanism is not a ‘perpetuum mobile’. Obese women and health care providers have it within their power to reverse the tide on the obesity epidemic. Dietary restriction and weight loss prior to pregnancy are proven strategies to improve infant health outcome. Pregnancies after bariatric surgery are less likely to be complicated by gestational diabetes mellitus, hypertension, preeclampsia, and macrosomia than are pregnancies of obese women who have not undergone such surgery [35-36]. Bariatric surgery prior to a planned pregnancy is the best strategy for reducing obesity-related complication for mother and child. In unplanned pregnancies, controlled or minimal weight gain during pregnancy may also

mitigate the impact of obesity and produce a dramatic positive impact on pregnancy outcome. Pregnancy also provides a 'window of opportunity' in which obese women are more susceptible for lifestyle interventions such as diet and exercise counseling, thus allowing health care givers to intervene [1].

The aims of study are to evaluate the effect of overweight and obesity on the neonates delivered In AlEmamain Al-khadimian Medical City.

Materials and Methods

This is a case-control study, it was done at the departments of NICU and Gynecology in Al-Emamain Al-khadamian Medical City in Baghdad from the period 1st of February 2017 to the 31th of December 2017. A total of 514 pregnant women who gave a births in that period were included in this study. We divided them into three groups according to the (BMI) Which were calculated as weight in (kg) divided by height (m) squared. The pregnant women of normal BMI (control) were their numbers=257, those who are overweight their numbers=183 and those who are obese mothers their numbers=74, this classification was done according to their BMI [1].

Normal: BMI 20-25

Overweight: BMI 25-30

Obese: BMI >30

The various outcomes of pregnancy included in this study in the maternal BMI groups were calculated and multiple logistic regression models were then constructed to assess the magnitude and significance of the independent effect of BMI. The study group (group1) include first group (257 cases) BMI>25 and the other (257 cases) was the control group BMI 20-25, who were healthy pregnant women attend in the hospital for delivery The information's (from both groups) were gathered after taking permission from the families of the neonates and the pregnant mothers with the help of the gynecologist surgeon and pediatricians responsible for the cases. Information's were obtained via prepared questionnaire form mothers of the neonates including: name, age, mode of delivery, time of delivery (preterm, term or postdate), gravidity, the use of multivitamin, any medical illnesses and education level which were subdivided into three level (high => 12 years' education, intermediate = between 6-12-year education, low =years education or illiterate) [37].

On examinations: of the pregnant women for her weight, height were recorded and complete examinations of the neonates for his/her weight , gestational age and systemic examinations for any gross congenital anomalies, blood sugar, Apgar score at 5 minute due to it seems more predictive value than 1 minute and admission to the NICU [38] were recorded from case sheet. Also, the need to use the ultrasonography and/or echocardiography, blood tests

and other facilities to diagnosis the neonatal illnesses and to search for any hidden anomalies were suspected.

Inclusion criteria: Singleton, healthy multigravida pregnant women, which their age between (18 -35 years).

Exclusion criteria: Primagravida pregnant women, those who are 35 years or smokers or with Diabetes mellitus, chronic hypertension, gestational Diabetes mellitus, gestational hypertension or any medical conditions, those without intake of folic acid during pregnancy and those with BMI <20 were considered underweight and families who refuse to be included in this study.

Statistical analysis

Statistical analysis was done using SPSS version 20 software programs, Chi square and independent t-test sample were used and a P value < 0.05 was considered significant.

Result

Information on maternal BMI was available for 514 completed singleton pregnancies including 183 (35.6%) overweight and 74 (14.4%) obese and normal weight 257 (50%) as a control case. The distribution of maternal and neonates' characteristics are similar among cases and controls with the proportion (%) of each risk factor or outcome being presented for each of the three BMI groups. A total 53 patients with congenital heart disease were distributed as the following matter: 16(6.22) in Control group and 20(10.9),18(24.32) in overweight and obese mothers respectively. CHD were more frequent in overweight and obese mother; the P value was significant as shown in table 2.

Table 2.

The distributions of neonates with CHD according to the mothers BMI

	Total No=514	Control No=257 (%)	Overweight No=183 (%)	Obese No=74 (%)	P value
VSD	29 (5.64)	8(3.11)	14(7.65)	7(9.45)	0.039
ASD	10(1.94)	4(1094)	3(1.63)	3(4.04)	0.364
Multiple congenital heart diseases	15(2.91)	4(1.55)	3(1.63)	8(10.81)	<0.001

A total 30 patients were diagnosed with *Neural Tube Defect* were distributed in the following matter: 5(1.94) Control and 15(8.19),9(12.16) in Overweight and Obese mothers respectively. NTD were more frequent in overweight and obese mother; the P value was significant as shown in table 3.

Table 3.

The distributions of neonates with NTD according to the BMI

	Total No=514	Control No=257 (%)	Overweight No=183 (%)	Obese No=74 (%)	P value
Hydrocephaly	13(2.52)	2(0.77)	6(3.27)	5(6.75)	0.011
Spina bifida	16(3.11)	3(1.16)	9(4.91)	4(5.4)	0.038

A total 47 patients were diagnosed with *Orofacial clefts* the were distributed in the following matter: 12(4.66) in Control, 23(12.56), 12(16.21) in Overweight and Obese mothers respectively. Orofacial clefts were more frequent in overweight and obese mother, the P value was significant as shown in table 4.

Table 4.

The distributions of neonates with Orofacial clefts according to the mothers BMI

	Total No=514	Control No=257 (%)	Overweight No=183 (%)	Obese No=74 (%)	P value
Cleft lip + palate	16(3.11)	3(1.16)	9(4.91)	4(5.4)	0.039
Cleft lip	22(4.28)	5(1.94)	11(6.01)	6(8.1)	0.026
Cleft palate	9(1.75)	4(1.55)	3(1.63)	2(2.7)	0.795

A total 22 patients were diagnosed with *Multiple Congenital Anomalies* the were distributed in the following matter: 5(1.94) Control, 10(5.46), 7(9.45) in Overweight and Obese mothers respectively. The multiple congenital anomalies were more frequent in overweight & obese mother & the P value was significant as shown in table 5.

Table 5.

Comparison of others congenital anomalies according to mothers BMI

	Total No=514	Control No=257 (%)	Overweight No=183 (%)	Obese No=74 (%)	P value
Ambiguous genitalia	6(1.16)	2 (0.77)	3 (1.63)	1 (1.35)	0.860
Gastroschisis	1(0.19)	0	1(0.54)	0	0.625
Multiple congenital anomalies	22(4.28)	5(1.94)	10(5.46)	7(9.45)	0.012
Renal dysplastic disease	3(0.5)	0	3(1.63)	0	0.271

The distributions of neonates with congenital anomalies according to the BMI in cases of *RDS* the total was 77 distributed in the following matter: 29(11.28) control and 31(16.93), 17(22.97) in overweight and obese mothers respectively. Regarding the *Hypoglycemia* the total cases was 40 distributed in the following matter: 13(5.05) Control and 17(9.28), 10(13.51) in Overweight and Obese mothers respectively. And the distributions of cases of *Low Apgar score* were in the following matter: total cases were 199, 61(23.73) Control and 103(56.38), 35(47.29) in Overweight and Obese mothers respectively Which the RDS ,hypoglycemia & low Apgar score were more frequent in overweight and obese mother & the P value were significant as shown in table (6).

Table 6.

Comparison of others neonatal complications according to BMI of mothers

	Total No=514	Control No=257 (%)	Overweight No=183 (%)	Obese No=74 (%)	P value
RDS	77(14.9)	29(11.28)	31(16.93)	17(22.97)	0.044
Hypoglycemia	40(7.78)	13(5.05)	17(9.28)	10(13.51)	0.036
Low Apgar score at 5 minutes	199(38.7)	61(23.73)	103(56.38)	35(47.29)	<0.001

And the distributions of neonates with *macrosomia* were in the following matter: total cases were 13, 4(1.55) Control and 2(1.09), 7(9.45) in overweight and obese mothers respectively and the distributions of cases of *Low birth weight* were in the following matter: total cases were 89, 34(13.22)Control and 46(25.13), 9(14.06) in overweight and obese mothers respectively. Both macrosomia and LBW were more frequent in overweight and obese mother and the P value were significant as shown in table (7).

Table 7.

The distribution of the neonates according to Birth Weight

	Total No=514	Control No=257 (%)	Overweight No=183 (%)	Obese No=74 (%)	P value
BW >90th percentile(macrosomia)	13(2.5)	4(1.55)	2(1.09)	7(9.45)	0.001
BW<5thpercentile (low birth weight)	89(17.31)	34(13.22)	46(25.13)	9(14.06)	0.004

Overweight and obese women were having a significant increase in incidence to have preterm and postdate delivery than control group in which the P value was significant as shown in table (8).

Table 8.

the distribution of neonates according to Gestational age at delivery

	Total N=514	Control N=257 N (%)	Overweight N=183 N (%)	Obese N=74 N (%)	P value
Preterm deliveries	119(23.15)	41(15.95)	75(40.98)	3(4.05)	<0.001
Term deliveries	63(12.25)	52(20.23)	8(4.37)	3(4.05)	0.000
Postdate deliveries	332(64.59)	164(67.31)	100(49.72)	68(91.89)	<0.001

Discussion

In this study there is a significant relationship between overweight and obesity and the Congenital Heart Diseases (CHD this result agrees with other studies [37, 39, 40, 41, 42, 43, 44]. These results consistent with the pathophysiology of cardiac malformations, which is multifactorial, with interactions between genetic and environmental factors.

Regarding the Neural Tube Defect (NTD), we found in this study great increase incidence of NTD, consistence with other studies [1, 45, 46, 47, 37]. It is knowing that maternal obesity is associated also with an increased risk of neural tube defect (NTD) in the offspring, even after controlling for ethnicity, maternal age, education, folic acid intake and socioeconomic status [11,12]. Watkins and coworkers [12] concluded that a 1 kg/m^2 increase in BMI is associated with a 7% increased risk of having an infant with NTD. The mechanism underlying the increased risk of NTD in pregnancies complicated by maternal obesity is unknown. However, several theories have been proposed, including a reduction in the amount of folic acid reaching the developing embryo due to insufficient absorption and greater maternal metabolic demands, chronic hypoxia, and increased circulating levels of triglycerides, uric acid, estrogen. Gary M. Shaw 2008 [48] in USA said that his data do not fully support earlier findings with respect to the relationships of obesity with anencephaly and spina-bifida.

Regarding the Orofacial clefts this study shows 2-3 folds' increase incidence of Orofacial clefts, it is agreeing with other studies [49, 50, 51, 52] these studies review that maternal obesity has been associated not only with orofacial clefts, but also with other major structural birth defects, such as those of the neural tube and heart. However, the obesity-teratogenic

mechanism has not been fully elucidated. Regarding the Gastroschisis. Although our data were limited, we found no significant association between maternal obesity and increase the risk of Gastroschisis which goes with Katherine J. Stothard 2009 in UK [53] although the mechanism were not clear yet Regarding the Multiple Congenital Anomalies .we found in this study group that the percentage of all multiple congenital anomalies in neonates of obese women were (6.61%) while in the control group were (1.94%) which show great increase incidence of multiple congenital anomalies , which is significant & the P value was significant and it is agree with other studies [37, 54] that suggested that the pathophysiology of malformations is multifactorial, with interactions between genetic and environmental factors. Other environmental factors associated with increased risks of malformations include maternal smoking, alcohol use [55-56], In the present study, we excluded malformations with a known cause other than maternal overweight or obesity. The possible teratogenic role of other metabolic derangements associated with obesity, such as insulin resistance, hyperlipidemia, and inflammation, is unclear. The adipose tissue is an active metabolic and endocrine organ [57] distributed subcutaneously and in the visceral compartment. Visceral obesity in pregnancy is associated with a state of inflammation, vascular dysfunction, and abnormal placental metabolism [58] which may adversely influence organogenesis and fetal development. But others [59, 60] said no relations between multiple congenital anomalies and maternal obesity. Regarding the Renal dysplastic diseases, other study [61] suggested that maternal obesity is of a negative impact renal development and increase risk of chronic kidney diseases in later life. My result was not significant due to the limited numbers of cases. Regarding the Ambiguous genitalia, my data were limited that's why the results were not significant Regarding Respiratory Distress Syndrome (RDS) we found in this study group that the percentage of RDS in neonates of obese women were (18.67%) while in the control group were (11.28) which is significant and the P value was significant and it is agree with other study [62] in Palestine. This study suggested that increase incidence of preterm deliveries in obese pregnant women especially before 32 weeks gestational age which indeed increase the incidence of Respiratory Distress Syndrome while Cededgen MI 2004 [63] show no relationship between maternal body weight and increase the incidence of RDS [64] said that maternal BMI>40 will reduce the incidence of RDS Regarding the Low Apgar score at 5 minute of the delivered neonates it was found that the percentage of low Apgar score in obese women (53.6%) while in controls were (23%) which show increase the risk of Low Apgar score which is significant and the P value was significant and it is agree with other studies [74, 65]. Use and abuse of the Apgar score [66, 67] where show the exact etiology were unknown. While

other studies show no associations between maternal obesity and Low Apgar score T.S. Usha Kiran 2005 in Wales [68].

Regarding the Hypoglycemia of the delivered neonates we found in this study group that the percentage of neonates of obese women with hypoglycemia were (10.5%) while in the control group were (5.05%) which show increase incidence of hypoglycemia. which is significant and the P value being significant and it is agree with other studies [69, 70] these studies suggested that During the latter part of pregnancy, increased insulin resistance favors the transfer of nutrients to the fetus [71-72]. A large amount of weight gain during pregnancy may increase the flux of maternal amino acids, glucose, free fatty acids, and triglycerides from maternal to fetal compartments and may affect fetal growth and development of neonatal hypoglycemia.

Regarding the Birth Weights (BW) of the delivered neonates we found in this study group that the percentage of the neonates who had the body weight >90th were 13(2.5), 4(1.55) in control and 9(3.5) in overweight and obese mothers which show significant increase the risk of macrosomia, which the P value was significant also. Moreover, there appears to be a dose dependent relationship between maternal obesity and fetal macrosomia Fetal macrosomia is more common in the obese non-diabetic mother compared to the lean mother with gestational diabetes. Obesity is associated with maternal insulin resistance and fetal hyper-insulinaemia even in the absence of maternal diabetes [73]. Insulin resistant individuals have higher fasting plasma triglyceride levels and greater leucine turnover [74] Amino acids are insulin secret gouges and an increased fluxon amino acids could stimulate fetal hyper-insulinaemia. Triglycerides are energy rich and placental lipases can cleave triglyceride and transfer free fatty acids to the fetus [75].

Also we found that the percentage of the neonates who had the BW <5th percentile (49.5%) which is also show significant increase the risk of LBW and the P value was significant where there is 2- 3 fold increase the risk of low birth weight in which the controls were (13.22%) as in many studies [76, 77] in which maternal obesity may contribute to multiple etiologic pathways leading to placental dysfunction and ultimately fetal growth restriction and stillbirth. Obesity has been associated with elevated baseline inflammation. There has been growing interest in the role of maternal inflammation on risks of preterm labor, placental dysfunction and fetal epigenetics While in Meaghan A. Leddy 2008 in USA [1] said that Maternal obesity is associated with abnormal fetal growth. Women who are heavier are less likely to have a pregnancy complicated by a small for-gestational age infant or intrauterine growth restriction.

Regarding the incidence of preterm and postdate delivery we found in this study group that the percentage of all incidence of preterm and postdate delivery in neonates of obese

women were (95.71%) while in the control group were (79.7%) which is significant & the P value was significant and it is agree with other studies [78, 79, 80] the factors that control length of gestation and onset of parturition are not well understood. obesity is associated with activation of the hypothalamic–pituitary–adrenal axis, cortisol clearance is also increased, and plasma cortisol levels are often low or normal [80-82]. Obese women may therefore have lower circulating cortisol levels during pregnancy than those of normal weight. This could reduce placental corticotrophin-releasing hormone production and consequently influence timing of delivery [83]. Alternatively, in obese women, the concentration of estrogen in adipose tissue may result in a reduction in levels of circulating estrogen and an alteration in the estrogen: progesterone ratio in maternal plasma, which increases prior to normal delivery [84] other studies [85] show that the increase risk of preterm delivers to be not fully understood but may be due to this mechanism: In spontaneous preterm deliveries, there are increased levels of inflammatory proteins (cytokines) such as interleukin6, interleukin 1, and tumor necrosis factor (TNF) ,these cytokines are associated with cervical ripening and may also cause both weakening of the membranes and preterm myometrial contractions, probably through stimulation of prostaglandin production and matrix-degrading enzymes [86-87]. Mechanisms by which maternal stress, smoking, vaginal bleeding، and infections influence risk of preterm delivery involve maternal systemic or intrauterine inflammation [88]. Maternal obesity is associated with inflammatory upregulation through increased production of adipokines by adipose tissue and enhanced systemic secretion of pro-inflammatory cytokines [89]. In pregnancy, visceral fat mass is increased, particularly in obese women and adipokines from visceral fat are known to increase systemic inflammation, visceral fatness is also accompanied by decreased insulin sensitivity and elevated levels of glucose [90]. The Hyperglycemia adverse pregnancy outcome study clearly demonstrated increasing risks of preterm delivery with increasing maternal glucose levels in women without diabetes.

In conclusions; there is significant associations between mothers BMI>25 and fetal outcomes; the most common congenital anomalies associated with maternal BMI >25 were the congenital heart diseases followed by orof-ascial clefts and neural tube defects; there were increase in the frequencies of others neonatal complications like; (RDS, post maturity, macrosomia, hypoglycemia, low Apgar score and others).

Ethical Approval

The study was approved by the Ethical Committee.

Conflicts of Interest

The authors declare that they have no competing interests.

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