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DOI: [10.31965/infokes.Vol21Iss1.879](https://doi.org/10.31965/infokes.Vol21Iss1.879)Journal homepage: <http://jurnal.poltekkeskupang.ac.id/index.php/infokes>**RESEARCH****Open Access****Maternal Parity, History of Obesity and History of Maternal GDM Risk a Macrosomia Baby****Listyaning Eko Martanti^{1a*}, Dhita Aulia Octaviani^{1b}, Rizky Amelia^{1c}, Suparmi^{1d}, Khobibah^{1e}**¹ Department of Midwifery, Politeknik Kesehatan Kementerian Kesehatan Semarang, Semarang, Central Java, Indonesia^a Email address: listy@poltekkes-smg.ac.id^b Email address: dhitaaulia@poltekkes-smg.ac.id^c Email address: rizkyamelia81@yahoo.com^d Email address: parmiadi@ymail.com^e Email address: khobibah@poltekkes-smg.ac.id

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Abstract

Gestational diabetes mellitus (GDM) is a carbide tolerance disorder that occurs or is first recognized during pregnancy (usually at 24 weeks gestation). For some patients, this complication returns to normal after delivery. The incidence of macrosomic infants or infants weighing >4000 grams is approximately 5% of all births. Maternal GDM is a significant risk factor in the development of fetal macrosomia. This study aimed to determine the risk factors for macrosomia in newborns. The design of this research is cross-sectional design. The population of this study was macrosomic babies born at Dr. Kariadi Semarang from 2015 until 2021. The formula for estimating the sample size using a hypothesis on the mean of two independent populations obtained a total sample of 60 respondents. The sampling technique is convenience sampling. The type of data used is secondary data. This study was analyzed using the Chi-Square test. The results indicated a relationship between parity and a history of obesity and macrosomia incidence in infants with a p-value < 0.05. In contrast, there was no relationship between maternal age, gestational age, and a history of diabetes in the mother and the incidence of macrosomia in infants with a p-value > 0.05. Therefore, it is essential to educate the mother about the risk factors that can cause complications for both the mother and the fetus, including macrosomia.

Keywords: History of Obesity, Macrosomia, Maternal GDM, Maternal Parity, Newborns.***Corresponding Author:**

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1. INTRODUCTION

The incidence of macrosomia babies in Indonesia is around 3.7% of national births (Kementerian Kesehatan Republik Indonesia, 2019). *Macrosomia* is defined as a fetus or infant bigger than standard, sometimes known as a giant infant. Typically, a birth weight of greater than 4000 grams is used to define macrosomia. All newborns weighing four thousand grams or over, regardless of gestational age, are considered to have macrosomia (Rahayu & Rodiani, 2016). Compared to the incidence of low birth weight (LBW), which reaches 6.2% nationally, the prevalence of macrosomia is low (Kementerian Kesehatan Republik Indonesia, 2019). But so far, macrosomia is linked to pre-pregnancy women's diet and health, making it difficult for developing countries, including Indonesia, to reduce it.

The risk factors for fetal macrosomia include obesity, gestational diabetes, type 2 diabetes, obese parents, post-term pregnancy, and multiparity. Diabetes mellitus (DM) is a chronic disease characterized by elevated glucose levels in the blood. Lack of the hormone insulin, which is generated by the pancreas to reduce blood sugar levels, is the cause of diabetes (P2PTM Kemenkes RI, 2020). Gestational diabetes mellitus (DMG) is a normal pregnancy characterized by heightened insulin resistance (pregnant women fail to maintain euglycemia). Typically, it happens between the second and third trimesters. Gestational diabetes mellitus (GDM) is a significant risk factor for the development of fetal macrosomia in the mother (Adli, 2021).

To meet the criteria for gestational diabetes, poor glucose tolerance during pregnancy must return to normal within six weeks following birth. Diabetes mellitus (not gestational) is suspected if poor glucose tolerance persists after birth (Rahayu & Rodiani, 2016). GDM is often diagnosed after 20 weeks of pregnancy when the placental hormone that has the opposite effect of insulin on glucose metabolism rises significantly. This situation is transitory, and blood glucose levels will return to following normal delivery (Adli, 2021). With subsequent pregnancies, women with gestational diabetes have a significant chance of acquiring diabetes mellitus. In addition, gestational diabetes is a risk factor for type II diabetes if the mother's blood sugar levels stay elevated after delivery. Infants born to moms with gestational diabetes are more likely to develop macrosomia. GDM can occur in pregnant women over 30, obese women (BMI > 30), women with a family history of DM, or women who had babies with birth weights > 4000 grams and glucosuria in previous pregnancies (Rahayu & Rodiani, 2016).

Diabetes in pregnancy brings several challenges. Many hormones increase in quantity during pregnancy, resulting in insulin resistance and elevated glucose levels in pregnant women. In addition, insulin kinetics and insulin resistance are altered, such that glucose levels in the mother's plasma increase and blood sugar levels are up while insulin levels stay elevated. Assisted diffusion across the placental barrier causes aberrant glucose levels in fetal circulation (Rahayu & Rodiani, 2016).

The increased serum levels of metabolites in diabetic mothers (e.g., glucose, free fatty acids, ketone compounds in the body, triglycerides, and amino acids) will increase nutrient transfer to the fetus, resulting in hyperglycemia in the uterine environment and altering fetal growth and body composition. During the second trimester of pregnancy, the pancreas of fetuses with maternal gestational diabetes mellitus responds to hyperglycemia in the uterine environment by boosting insulin synthesis, resulting in hyperinsulinemia in the fetus. Hypoglycemia, polycythemia, hyperbilirubinemia, respiratory distress syndrome, and poor fetal growth or macrosomia result from the completion of metabolic processes in the uterus (Rahayu & Rodiani, 2016).

Various factors, including the uterine environment of pregnant women, the functioning of the placenta, and the availability of nutritional intake for the mother and fetus, influence the growth and development of a macrosomic fetus. During early pregnancy, insulin and insulin

developmental factors are significant determinants of fetal organ growth and development. The production of insulin in the fetus, which begins between 8-10 weeks of gestation, is primarily determined by the mother's glucose level, which $\pm 80\%$ is distributed to the fetus through the placental membrane. Mothers with gestational diabetes mellitus offspring who have poor glycemic control are continuously exposed to high levels of glucose and insulin in the uterus, which can accelerate fetal growth. The growth of macrosomic fetuses in utero tends to accelerate (after 38 weeks), whereas non-macrosomic fetal growth is more linear during pregnancy (Rahayu & Rodiani, 2016).

Macrosomia can result in problems for both the mother and the child. Postpartum hemorrhage, vaginal lacerations, ripped perineum, and cervical lacerations are the mother's problems. Shoulder dystocia in neonates can result in brachial plexus damage, humeral fractures, and clavicle fractures. Postpartum hemorrhage and shoulder dystocia cause maternal fatalities connected with macrosomia births, whereas shoulder dystocia, poor Apgar scores, and suffocation cause macrosomia-related newborn deaths (Dungga & Husain, 2019).

According to the above description, the authors are interested in investigating the risk factors for macrosomia in infants. Once the risk factors for macrosomia are recognized, preventive measures can be taken to reduce macrosomia's incidence and mitigate its adverse effects.

2. RESEARCH METHOD

The design of this research is cross-sectional. This design studies exposure status, disease, or other outcomes simultaneously in individuals from a community at a time enabling epidemiologists to determine the prevalence, distribution, or relationship between disease risk factors and their manifestations (Vionalita, 2020). Given that this is a retrospective study with more than two variables, a cross-sectional design is appropriate so that the research may be conducted reasonably rapidly. This approach is helpful for macrosomia-related public health planning, monitoring, and evaluation. This study included macrosomic babies delivered at Dr. Kariadi Semarang in 2015 and 2021. Using a hypothesis test on the mean of two separate populations, the technique for determining the sample size yielded a total sample size of 60 responders. The sampling approach is convenient (non-probability); the sample is gathered without a specified procedure, and secondary data are utilized. This study's data collecting instrument is a data collection format (master table) in the form of an observation sheet to record the research outcomes.

The variables measured in this study are the mother's age, the period of healthy reproduction (age range 20-35 years) for women, parity, maternal gestational age, gestational diabetes mellitus, obesity, and macrosomia in newborns. Data were analyzed using the Chi-square test. This study has obtained ethical feasibility from the ethics committee of Dr. RSUP. Kariadi Semarang with No. 907/EC/KEPK-RSDK/2021.

3. RESULTS AND DISCUSSION

Table 1. Distribution of Macrosomia Variable Frequency, Health Reproduction of Mother, Parity, Maternal Gestational Age, History of Diabetes Mellitus, History of Obesity.

| Variable | N (60) | % |
|--------------------------------------|--------|------|
| Macrosomia | | |
| No | 35 | 58.3 |
| Yes | 25 | 41.7 |
| Health reproduction of mother | | |
| Healthy Reproduction (20-35 years) | 49 | 81.7 |

| | | |
|--|----|-------|
| Unhealthy Reproduction (<20 years and >35 years) | 11 | 18.3 |
| Parity | | |
| Multipara | 41 | 68.3 |
| Primipara | 19 | 31.7 |
| Mother's Gestational Age | | |
| Aterm | 59 | 98.3 |
| Serotinus | 1 | 1.7 |
| History of Diabetes Melitus | | |
| No | 59 | 98.3 |
| Yes | 1 | 1.7 |
| Obesitas History | | |
| No | 55 | 91.7 |
| Yes | 5 | 8.3 |
| Quantity | 60 | 100.0 |

Table 1 reveals that of the 60 study samples, 35 (58.3%) did not have macrosomia, came from 49 (81.7%) women of healthy reproductive age (age range 20-35 years), parity of more than one was 41 (68.3%), almost all mothers were at term 59 (98.3%), nearly all mothers did not have a history of Diabetes Mellitus as many as 59 (98.3%). The sample was not overweight/obese, as many as 55 (91.7%).

Table 2. Bivariate Analysis of the Relationship between Maternal Healthy Reproductive Age and Macrosomia.

| Health Reproduction of Mother | Macrosomia | | | | Total | p-value | OR | CI | |
|--|------------|-------|-----|-------|-------|---------|-------|-----|-------------|
| | No | | Yes | | | | | | |
| | N | % | N | % | | | | | |
| Healthy reproduction (20-35 years) | 29 | 82.9 | 20 | 80 | 49 | 81.7 | 1.000 | 1.2 | 0.324-4.507 |
| Unhealthy Reproduction (<20 years and >35 years) | 6 | 17.1 | 5 | 20 | 11 | 18.3 | | | |
| Quantity | 35 | 100.0 | 25 | 100.0 | 60 | 100.0 | | | |

Table 2 shows no relationship between maternal age risk factors and infant macrosomia incidence. However, the maternal age factor is 1.2 times the risk of having a macrosomic baby. This relationship is not significant, as indicated by the p-value 1,000 (> 0.05).

The higher the mother's age, the higher the risk of macrosomia (Merita, 2015). The mother's age is closely related to the baby's birth weight. Pregnancy under 16 years is a high-risk pregnancy, 2-4 times higher than pregnancy in women who are old enough. Developing reproductive organs and physiological functions is not optimal at a young age. In addition, her emotions and psychology are not yet mature enough, so during pregnancy, the mother has not been able to respond to her pregnancy ideally, and complications often occur. In addition, the younger the pregnant woman ages, the more danger of the baby being born prematurely, bleeding, and being born lightly (Sari, Amdadi & Hidayati, 2021). Age-related conditions in pregnant women that must be monitored can impact labor complications; for instance, hemorrhage frequently occurs in women over 35, as does the risk of congenital defects.

Table 3. Bivariate Analysis of Maternal Parity with Macrosomia.

| Paritas | Macrosomia | | | | Total | | <i>p-value</i> | OR | CI |
|-----------|------------|-------|-----|-------|-------|-------|----------------|-------|------------|
| | No | | Yes | | N | % | | | |
| | N | % | N | % | | | | | |
| Multipara | 20 | 57.1 | 21 | 84 | 41 | 68.3 | 0.05 | 0.254 | 0.72-0.897 |
| Primipara | 15 | 42.9 | 4 | 16 | 19 | 31.7 | | | |
| Quantity | 35 | 100.0 | 25 | 100.0 | 60 | 100.0 | | | |

Table 3 shows a significant relationship between parity and infant macrosomia with a *p*-value of 0.05. Because the OR value is 0.254 (<1), parity is a protective factor, meaning there is a negative relationship between parity risk factors and the incidence of babies born with macrosomia.

Based on the results of the study showed that most of the mothers who gave birth to macrosomia were multiparous. The uterus that gives birth to more than one baby experiences a change in its elasticity. The more the number of births, the more elastic and larger the uterus will become. Hence, it may be inferred that multigravida women are more likely to have infants weighing more than 4,000 grams (macrosomia) (Osok et al., 2017). There is a tendency that the birth weight of the second child and so on will be greater than that of the first child (Dungga & Husain, 2019). Multiparity moms have a higher risk of diabetes mellitus and a tendency to have a high body mass index, both of which are key predictors of macrosomia. Multiparity is a factor in the prevalence of macrosomia, according to Fajariyana (2020), which supports this view. The results of this study were possible because some of the mothers who gave birth were mothers with 2-4 times parity (multipara) in the healthy reproductive age range. The possibility of maternal health is still the same as the previous pregnancy, so subsequent pregnancies tend to give birth to babies with higher birth weights (Sujianti, 2015). Especially if the mother has had a macrosomic baby before, there is a 5 to 10 times greater risk of giving birth to a macrosomic baby again (Lestari & Sudarmanto, 2022). Consequently, it can be stated that women who give birth several times have the potential to give birth to larger babies, and the more the parity, the greater the danger of the baby developing macrosomia.

Table 4. Bivariate Analysis of Gestational Age with Macrosomia.

| Gestational Age | Macrosomia | | | | Total | | <i>p-value</i> | OR | CI |
|-----------------|------------|-------|-----|-------|-------|-------|----------------|-------|-------------|
| | No | | Yes | | N | % | | | |
| | N | % | N | % | | | | | |
| Aterm | 35 | 100 | 24 | 96 | 59 | 98.3 | 0.417 | 0.407 | 0.299-0.897 |
| Serotinus | 0 | 0 | 1 | 4 | 1 | 1.7 | | | |
| Quantity | 35 | 100.0 | 25 | 100.0 | 60 | 100.0 | | | |

Table 4 demonstrates, with a *p*-value of 0.417 (>0.05), that there is no correlation between the mother's gestational age, both aterm (at term) and serotinus (over months), and the occurrence of macrosomia. OR = 1 (0.407), which indicates that the mother's gestational age is also a protective factor since there is a negative link between the risk factor of maternal gestational age and the occurrence of kids delivered with macrosomia.

Contrary to Yunita's study (2016), findings indicated serotinus pregnancies increased macrosomia risk. Compared to term pregnancies, serotinus pregnancies have a 4.426 risk of producing macrosomia babies. This study disproves the hypothesis that post-term pregnancy increases mortality, morbidity, perinatal, or macrosomia. Because the placenta provides nutrients, O₂, and other functions, post-term pregnancy alters fundamentally. If the placenta functions well, it can maintain the fetus's growth and development so that its weight increases with gestational age, eventually reaching around 4000 grams, making it a macrosomic infant.

According to Zwerding, the proportion of post-term pregnancies with an average fetal weight of more than 3,600 grams was 44.5%, while that of aterm pregnancies was 30.6%. Post-term pregnancies raise the chance of having a baby weighing more than 4000 grams by two to four times compared to aterm pregnancies (Sakinah, 2020). A post-term fetus indicates that the placenta is regularly working and that the fetus can handle the obligations of regular delivery without issue. Yet, the ongoing growth of the fetus might cause an alarming amount of cephalopelvic dysport (Handaria et al., 2016). In predicting the possibility of shoulder dystocia, clavicle fractures, or brachial plexus injuries (Kusumawati et al., 2014), which can lead to cesarean section birth, prenatal estimations of the fetus can be performed (Handaria et al., 2016). Lubis, (2019) found that the prevalence of macrosomia may raise the risk of problems such as fetal mortality. Thus, early recognition of instances and treatment of risk factors are necessary to prevent excessive fetal development.

Table 5. Bivariate Analysis of Relationship History of Diabetes Mellitus with Macrosomia.

| History of DM | Macrosomia | | | | Total | <i>p-value</i> | OR | CI | |
|---------------|------------|-------|-----|-------|-------|----------------|-------|-------|-------------|
| | No | | Yes | | | | | | |
| | N | % | N | % | | | | | |
| No | 35 | 100 | 24 | 96 | 59 | 98.3 | 0.417 | 0.407 | 0.299-0.897 |
| Yes | 0 | 0 | 1 | 4 | 1 | 1.7 | | | |
| Quantity | 35 | 100.0 | 25 | 100.0 | 60 | 100.0 | | | |

Table 5 reveals no correlation between a mother's history of diabetes and the prevalence of macrosomia. The *p-value*, which is more than 0.05, confirms this. The history of diabetes mellitus in the mother is a protective factor since there is a negative association between the history of diabetes mellitus in the mother and the prevalence of babies born with macrosomia if the OR value is 1. In other terms, a maternal history of diabetes mellitus is not a risk factor for macrosomia based on the data shown in the table above. There is no correlation between gestational diabetes mellitus and the delivery of macrosomic babies. Hence it cannot be predicted that the newborns of mothers with gestational diabetes mellitus will have macrosomia (Muhtar, 2018). This is supported by Setiawan et al., (2014)'s finding that there was no correlation between gestational diabetes and macrosomia.

In contrast to the findings of Rachmawati, (2021) and Zain et al., (2020), which indicated a substantial correlation between diabetes in pregnancy and macrosomia, our study found no such correlation. During pregnancy, mothers with diabetes are 6,029 times more likely to give birth to babies with high birth weights. Theoretically, if the maternal glucose level rises, the baby will absorb 80 percent of the glucose via the placental membrane beginning between 8 and 10 weeks of pregnancy (Rahmawati & Bachri, 2019). Moreover, DMG raises the risk of macrosomia in neonates. This risk is influenced by birthweight, gestational age, and early breastfeeding beginning (Biade et al., 2016).

In this study, based on 60 research samples, there were 25 occurrences of infants with macrosomia. However, only one came from a mother with a history of diabetes mellitus, so there was no statistically significant relationship between a history of diabetes mellitus and the incidence of macrosomia. This result may be due to intense efforts to achieve reasonable metabolic control during pregnancy and shorter gestational duration. In addition, it may also be due to the large number of samples that may not check blood sugar levels, both before and during pregnancy. Laboratory tests, especially blood sugar levels, can prevent complications, both for the mother and the fetus, such as bleeding after delivery and the baby's death in the womb. Preferably, blood sugar checks are carried out in the second trimester, in the 24-28 week gestational age range. Normal blood sugar results are blood sugar levels equal to or less than 140 mg/dL (Nilsson et al., 2015). Based on this explanation, pregnant women with DM do not

always have a risk of having a baby with macrosomia, of course, with excellent and regular supervision or pregnancy checks.

Table 6. Bivariate Analysis of the Relationship between Obesity History and Macrosomia.

| History of Obesity | Macrosomia | | | | Total | <i>p-value</i> | OR | CI | |
|--------------------|------------|-------|-----|-------|-------|----------------|------|-------|-------------|
| | No | | Yes | | | | | | |
| | N | % | N | % | | | | | |
| No | 35 | 100 | 20 | 91.7 | 55 | 91.7 | 0.01 | 0.364 | 0.256-0.516 |
| Yes | 0 | 0 | 5 | 8.3 | 5 | 8.3 | | | |
| Quantity | 35 | 100.0 | 25 | 100.0 | 60 | 100.0 | | | |

There was a significant relationship between a history of obesity and infant macrosomia, with a *p*-value of 0.01. There is a negative link between the risk factors of Obesity History and the incidence of macrosomia in newborns. It might be stated that a history of maternal obesity is not a risk factor for macrosomia in infants. On the other hand, the history of obesity is a protective factor for infant macrosomia when viewed from the OR value of less than one, which is 0.364. It indicates that a history of obesity is a protective factor for infant macrosomia.

In regulating energy balance, excessive energy intake causes an increase in adipose tissue with an increase in circulating leptin. The hypothalamic anorexia center leptin reduces neuropeptide Y (NPY) production and reduces appetite. On the other hand, when energy requirements are higher than energy intake, adipose tissue is reduced, the hypothalamic anorexia center is stimulated, and appetite increases. High leptin levels do not cause a decrease in appetite. Leptin resistance occurs in most obese people.

Maternal obesity can cause complications for the fetus and newborn, such as giant babies, shoulder dystocia, high birth weight, and childhood obesity. Women with obesity and gestational diabetes are at risk of giving birth to giant babies at the 90th percentile gestational age (LGA), or as much as 4.5 kg. Maternal obesity is also associated with abnormal fetal growth or the development of fetal macrosomia (Natalia et al., 2020). It occurs via a resistance-raising mechanism (maternal non-diabetic mellitus) that increases fetal glucose and insulin levels. Placental lipase metabolizes triglycerides in the mother's blood and transfers free fatty acids as nutrients for the growing fetus. Increased triglyceride levels in obese mothers are associated with excessive fetal growth through increased free fatty acids (Merita, 2015).

One of the supervision of pregnant women is regulating diet and weight. It is essential because nutritional deficiencies and excesses can cause unwanted abnormalities in pregnancy. If the mother does not get adequate nutrition during pregnancy or her weight gain is less than recommended, their pregnancy is associated with an increase in low birth weight (<2500 grams). Meanwhile, if the mother gains excess weight before pregnancy or during pregnancy, there is an increase of more than 15 kg, which increases the risk of macrosomia (≥ 4000 grams) (Alfianti et al., 2022; Susianti, 2017). Pregnant women should avoid high-fat, incredibly saturated fatty acids as prevention because the fat can increase the appearance of fat masses that stick to the walls of blood vessels. Reducing fat can burn 30% of total calories, and reducing the consumption of excess carbohydrates will help one normal weight gain in pregnant women. Pregnant women need to ensure a balanced diet and physical activity. Physical activity improves weight management by burning calories. A good lifestyle can prevent hypercholesterolemia and high blood pressure (Natalia et al., 2020). In addition, the Royal Australian and New Zealand College of Obstetrics and Gynecology (RANZCOG) states that nutritionists and family specialists must support obesity management. Various dietary supplements are indicated for pre-pregnant individuals, including 5 mg of folic acid per day for women with a BMI of 30 kg/m² or above (Gilmore & Redman, 2015). According to RCOG recommendations, vitamin D supplementation is advised due to the inverse relationship between BMI and vitamin D levels. RANZCOG also recommends 150 g of iodine and vitamin

D per day if the patient is deficient in vitamin D (Pellonperä et al., 2018). Excessive maternal weight before pregnancy (obesity) and the rise in maternal weight gain during pregnancy might result in big infants (Farahdiba & Agusalam, 2018). This makes it necessary for mothers with a history of obesity to calculate their body mass index before pregnancy to control weight gain in each trimester of pregnancy so that they can anticipate the occurrence of macrosomia babies or other complications related to obesity.

4. CONCLUSION

Eighty-one percent (81.7%) of the sixty moms were of healthy reproductive age (20-35 years). Most parity women were multiparous (2-4 births), 41 out of 60 mothers (68.3%). Virtually all gestational ages were at term, 59 out of 60 (98.3%) moms. Among 60 women with a history of diabetes mellitus, nearly all (59) had no history of diabetes mellitus (98.3%). The vast majority (91.7%), or 55 of 60 moms, had no history of obesity.

Parity and obesity history affect macrosomic newborns with a p-value <0.05. However, there was no relationship between maternal age, gestational age, history of diabetes mellitus in the mother, and the incidence of macrosomia in infants with a p-value > 0.05. The recommendation from this study is that further research is needed on other risk factors associated with macrosomia incidence with a larger sample size and BMI monitoring during pregnancy.

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