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ASSOCIATION OF PREGESTATIONAL OBESITY WITH ANEMIA DURING PREGNANCY: A LITERATURE REVIEW

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Abstract

Relevance: Obese pregnant women have a mild inflammatory response associated with hepcidin overexpression. Inflammation affects iron metabolism by increasing hepcidin levels. Obesity and anemia are serious problems worldwide; expectant mothers often suffer from these conditions. Gravidas with obesity tend to gain more weight during pregnancy than gravidas with normal body mass index. Obesity before pregnancy is associated with a high risk in pregnancy, including hypoferremia and anemia due to iron deficiency, which are already common between expectant mother and their neonates.

This review considers obesity-associated inflammatory mediator activation as a potential primary cause of iron deficiency (ID) or iron deficiency anemia (IDA) in obese pregnant women.

The review aimed to summarize the current studies reporting the measurement of molecular markers of iron metabolism and inflammatory mediators in pregnant women with pregestational obesity.

Methods: This review included all types of publications in English and Russian on anemia and obesity in pregnancy, published from March 2012 to March 2022. The literature search was performed in Medline, Scopus, Web of Science, Google Scholar, PubMed, and CyberLeninka databases by the following keywords: “pregnancy,” “iron deficiency,” “anemia,” “overweight,” “obesity,” “inflammation,” and “body mass index.”

Results: This review assesses whether obesity-induced inflammation may contribute to the increased incidence of ID/IDA in pregnant women. Despite numerous studies, the effect of maternal weight on the risk of ID/IDA during pregnancy remains unclear. Iron status metabolism markers against inflammation are considered. PR-pregnancy obesity carries a greater risk of developing ID/IDA during pregnancy and the postnatal period for the mother and the baby.

Conclusions: Thus, a more careful study of iron levels by trimester is required. The introduction of clearly defined procedures for trimester valuation of iron and inflammatory status in antenatal and postpartum consultations is necessary.

Keywords: *pregnancy, iron deficiency (ID), iron deficiency anemia (IDA), overweight, obesity, inflammation, body mass index (BMI).*

Introduction: Anemia is an ailment caused by an increased or decreased number of erythrocytes leading to insufficient oxygen tolerance to fulfill physiological needs [1]. The most common reason for anemia is insufficient iron, refiring to hemoglobin as the main component of blood protein. Other abnormalities associated with anemia include vitamin A & B12 deficiency, parasitic infections, chronic inflammation, and hereditary diseases [2]. Pregnant women and children are among the most vulnerable groups, and pregnancy is most related to anemia.

Anemia affects about 1.62 billion people, 24.8% of the global population. It mostly prevails among preschool children [3]. WHO (2011) estimates that 32 million pregnant women, or more than 40%, suffer from anemia worldwide. Anemia represents increased perinatal risks for the mother and newborns. Anemia causes 20% of maternal mortality in Africa and Asia. It was also reported that these women were already anemic at faction, with estimates of 43% and 12% among non-pregnant women in developing and advanced countries, respectively. In this population, the frequency of anemia reaches 80-90%, given the WHO-recommended hemoglobin level of 120 g/L [3].

Another interesting observation is that iron deficiency is related to sedentary lifestyles contributing to obesity. Moreover, obesity and iron deficiency are common in groups with low socioeconomic status who consume inexpensive, fast foods that are lower in nutrients and rich in sugars and preservatives [4]. This trend is alarming and requires the attention of governments and international organizations to implement effective nutrition policies and practices to halt disadvantages. The importance of public health programs was highlighted at the Global International Forum on Micronutrients (2014).

This review considers obesity-associated inflammatory mediator activation as a potential primary cause of iron deficiency (ID) or iron deficiency anemia (IDA) in obese pregnant women.

The review aimed to summarize the current studies reporting the measurement of molecular markers of iron metabolism and inflammatory mediators in pregnant women with pregestational obesity.

Materials and methods: This review included all types of publications in English and Russian on anemia and obesity in pregnancy, published from March 2012 to March 2022. Duplicates and articles not related to pregnancy, anemia, and obesity were excluded from the search results; a total of 31 studies were included in the review. Literature searches were done in Medline, Scopus, Web of Science, Google Scholar, and PubMed databases. All were searched using the keywords: “pregnancy,” “iron deficiency,” “anemia,” “overweight,” “obesity,” “inflammation,” and “body mass index.”

Results: R. Kawata et al. showed a significant association between malnutrition and anemia and overweight/obesity in pregnant Nepalese women, indicating an urgent need for improved nutrition. In this regard, nutrition programs shall focus on the reproductive generation and families with low

health literacy [5].

The United States Preventive Services Task Force has concluded that recommending standard iron increases during pregnancy to improve maternal or neonatal outcomes is not yet well established. It is unclear whether iron supplementation affects perinatal outcomes in well-nourished women with pregnancy who do not have iron deficiency anemia (IDA) because of insufficient evidence. At the same time, the Centers for Disease Control and Prevention and the American College of Obstetricians and Gynecologists recommend that all pregnant patients take low-dose (27mg) iron from the beginning of pregnancy to improve maternal hematological outcomes [6-8]. Thus, finding the most effective approach to predicting and managing the risks of identifying IDA throughout pregnancy is an important topic for future research.

Anemia during pregnancy leads to adverse pregnancy outcomes for both mother and fetus, including reduced physical capacity, susceptibility to infections, susceptibility to depression, premature rupture of membranes, intrauterine delayed fetal growth, fetal hypoxia, premature birth, low birth weight, antenatal fetal death, poor quality interaction with children in the postnatal period [9].

In developing countries, 12% of low birth weight, 19% of preterm births, and 18% of perinatal deaths are related to anemia during pregnancy. One of the risk factors for iron deficiency/iron deficiency anemia (ID/IDA) in pregnant women is nutritional status before pregnancy. Body mass index (BMI) before pregnancy represents the metabolic and nutritional status of the mother during pregnancy. Women who had a normal BMI before pregnancy may have more iron stores; conversely, if pre-pregnancy BMI is lower than normal, they may experience ID during pregnancy. On the other hand, being overweight, especially obesity is associated with systemic inflammation, which causes an increase in hepcidin and ferritin. Increased hepcidin and ferritin diminished iron assimilation and impaired hemoglobin synthesis [10]. Obesity increases adipose tissue deposition, which enhances proinflammatory cell manifestation [11]. IL-6 induces a transducer and activator of transcription (STAT3) binding signal to the hepcidin promoter, ultimately increasing hepcidin expression.

Hepcidin is a hormone that regulates systemic iron. This hepcidin boost causes hypoferremia, defined by a rise in solvable transferrin receptor (sTfr) and a reduction in serum iron (Fe), while ferritin is also increased [12]. Hepcidin is mainly produced in the liver and controls the mobilization of iron storms in the body through post-translational regulation of Fpn-1. However, hepcidin is also produced in the heart, adipose tissue, placenta, and kidneys. However, it remains unclear whether the hepcidin produced by these peripheral tissues has a local and/or systemic effect.

Several studies have examined hepcidin concentrations during pregnancy. During physiologic pregnancy, hepcidin decreases significantly from an almost undetectable level before the end of the third trimester [13, 14]. Endogenous estrogen, which increases dramatically during pregnancy, probably plays a significant role in suppressing hepcidin levels [15]. Suppression of maternal hepcidin ensures active expression of Fpn-1 at major sites of iron flow, including maternal intestinal enterocytes, liver stores, and the placenta. The fetus also produces hepcidin, but the role of fetal hepcidin in regulating iron flow through the placenta remains unknown [16]. There is evidence that maternal hepcidin is overexpressed in the 3rd trimester, maternal iron is utilized with food, and iron transfer through the placenta is reduced [17].

Low iron intake is one of the main causes of IDA, and most preventive measures, such as taking supplements and food fortification with iron, are aimed at changing it. However, other factors affecting this public health problem miss the attention, such as obesity, though it affects 20% of the broad population and 48% of women.

Pregnancy and obesity have opposite effects on hepcidin and thus on iron homeostasis and nutritional status. On the one hand, the maternal body needs more iron for the fetal and placental formation and growth. On the other hand, elevated iron levels in the body cause suppression of hepcidin activity, which in turn promotes the absorption of dietary iron by enterocytes of the duodenum. Changes in pregnancy-related iron homeostasis are reflected in the concentration of a wide range of dietary iron biomarkers. Serum iron and serum transferrin receptor (sTfr) either remain unchanged or are regulated to decrease or increase, respectively, reflecting available iron; ferritin decreases over time as iron stores are mobilized. Meanwhile, functional iron, reflected by hemoglobin, decreases in the and of the first trimester due to increased plasma volume and gradually increases after that [18].

Obesity alters iron homeostasis due to excess adipose tissue, which causes mild chronic inflammation involving cytokines: interleukin 6 (IL-6) and leptin. This mechanism increases hepcidin production, leading to obesity-related hypoferremia, manifested by increased sTfr and decreased serum iron; ferritin values increase or remain unchanged [19].

However, the relationship between obesity and maternal iron condition within pregnancy remains unclear. The few studies comparing women with and without obesity report conflicting results [20-25]. An important limitation of the comparisons made between the studies of their findings was the failure to account for important variables such as diet and supplemental iron intake or iron condition in early pregnancy. C. Cao et al. failed to demonstrate an effect of obesity on hepcidin and iron biomarkers in pregnant women. This negative result could be explained by the fact that the participants in this study were adolescent girls at known risk for ID regardless of obesity [22].

M.D. Koenig et al. could find no differences in iron levels in the ending trimester of pregnancy in pregestational women with and without obesity. Despite a higher incidence of anemia, women with pregestational obesity had less depleted iron stores, demonstrating some degree of iron sequestration [26].

Surveys on the impact of obesity on iron homeostasis during pregnancy often show conflicting results. The reason could be that important nutritional status variables, such as pre-pregnancy iron status, supplements, or their simultaneous effects, are not always considered [20-22]. M.E. Flores-Quijano et al. considered those modifying factors of available or circulating iron, iron stores, and available iron, as well

as other markers in the form of higher sTfr and reduced serum iron levels in the pregnant group. sTfr concentration is known to increase in a greater need for iron due to ID or increased erythropoiesis, as in pregnancy. During pregnancy, the concentration of this soluble receptor remains constant or increases as iron stores are depleted [18, 25, 27].

Several studies have found lower levels of available iron in pregestational obese women, which has not been confirmed in the results of other authors [20-23]. A study involving adolescent women showed that serum iron levels were lower in the obese group in the second trimester but did not differ during delivery, while sTfr levels did not vary between those periods and remained stable at any specific time. Another study involving adult women showed that as the pregnancy progressed, women in the obese group had lower increases in sTfr concentrations, suggesting that obesity protects against developing iron deficiency. However, both studies included a significant proportion of women with anemia and iron deficiency. The authors argued that, in this context, the effect of obesity on iron homeostasis is null [22]. Also, some studies have reported no difference in ferritin content between obese and non-obese women [21, 24, 25], except when comparing the highest categories of obesity (pre-pregnancy BMI ≥35). This fact suggests that a high degree of obesity may cause changes in iron homeostasis reflected in ferritin concentrations [22]. In contrast, the longitudinal analysis showed that a decrease in ferritin during pregnancy occurred in groups of normal-weight and obese women, but during delivery, ferritin was restored only in women with a normal BMI [15]. The authors suggested a relationship between this phenomenon and higher hepcidin levels in the obese group.

Discussion: L. Tussing-Humphreys et al. reported that hepcidin increases moderately in obesity compared with inflammatory diseases. This “moderate” increase in hepcidin does not completely suppress iron absorption or mobilization of iron stores. Available iron is sufficient to maintain adequate erythropoiesis without affecting hemoglobin concentration but may not be sufficient to maintain iron stores [19]. The most important stimuli associated with changes in hepcidin concentration during pregnancy are increased maternal erythropoiesis and the demands of the growing fetus and placenta. As pregnancy progresses, this increased need for available iron is reflected in a decrease in hepcidin levels, reaching its lowest level in the third trimester when the fetal need for iron is greatest. This has been observed in longitudinal studies [25, 28, 29].

Notable is the degree to which hepcidin has decreased among different studied populations. Thus, some studies reported the lowest concentrations of about 5 ng/ml, while others stated indeterminate levels. Such changes can be explained by the counterbalancing or reinforcing effects of hepcidin-regulating factors such as nutritional status, iron before pregnancy, and the benefit of iron supplements. In the study by M.E. Flores-Quijano et al., obesity stimulated the hepcidin concentration [29]. This is consistent with the observations in non-pregnant adults, adolescents, children [30], and pregnant obese women [20, 23]. However, two studies revealed no differences between normal-weight and obese pregnant women, except for obesity [4, 22]. This could be interpreted as a dose-dependent effect the degree of obesity has on hepcidin production. When considering the results of other studies, two important observations can be made: first, hepcidin concentrations may be higher in obese women, even though statistical models control for inflammatory biomarker concentrations; second, inflammatory markers are many times higher in the obese women group in comparative studies but are not directly related to hepcidin.

On the contrary, two other studies reported an association between hepcidin and CRP or IL-6 during labor [22, 23]. However, the hepcidin concentration increase during labor is inflammatory-induced, especially in the case of natural delivery. This could be because hepcidin is an acute-stage protein that responds to a proinflammatory environment usually present during labor and not associated with obesity [28]. However, these observations do not contradict the hypothesis that obesity-associated inflammation may increase hepcidin concentrations and modify iron homeostasis during pregnancy.

Conclusions: Thus, pre-pregnancy obesity and obese pregnancy are evidently associated with an increased risk of ID/IDA for the mother and child. Available studies report high CRP, hepcidin, sTfR, and IL-6, warning that the inflammatory profile is present in obese pregnant women and thus may play a role in the progress of ID/IDA in obese women in pregnancy [23, 30, 31]. Studies have also shown that inflammation plays no role in controlling iron metabolism during pregnancy and that children born to young obese mothers had significantly higher levels of iron and hemoglobin in the body compared with children of lean mothers [22]. Trimester evaluation of iron and inflammatory status in antenatal and postnatal consultations is necessary. Despite numerous reliable studies, the relationship between obesity and IDA is not fully understood, requiring more careful monitoring of iron metabolism markers by trimester. Therefore, further research is required.

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АССОЦИАЦИЯ ПРЕГЕСТАЦИОННОГО ОЖИРЕНИЯ С РАЗВИТИЕМ АНЕМИИ ВО ВРЕМЯ БЕРЕМЕННОСТИ: ОБЗОР ЛИТЕРАТУРЫ

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Аңдатпа

Аннотация

Актуальность: Беременные женщины с ожирением имеют легкую воспалительную реакцию, связанную с высокой экспрессией гепсидина. Воспаление может повлиять на метаболизм железа за счет повышения уровня гепсидина. Ожирение и анемия являются серьезными проблемами во всем мире, и беременные женщины часто страдают от этих состояний. Беременные женщины с ожирением, как правило, прибавляют в весе во время беременности больше, чем женщины с нормальным индексом массы тела. Прегестационное ожирение связано с повышенным риском беременности, включая дефицит железа (ЖД) и железодефицитную анемию (ЖДА), которые уже широко распространены среди беременных женщин и их новорожденных.

В этом обзоре ожирение, связанное с активацией медиатором воспаления, рассматривается как потенциальная первопричина ЖД или ЖДА у беременных женщин с ожирением.

Цель обзора – обобщение результатов современных исследований, в которых сообщается об измерении уровня молекулярных маркеров обмена железа и медиаторов воспаления у беременных женщин с прегестационным ожирением.

Методы: Настоящий обзор включает анализ опубликованных на сегодняшний день данных о беременных с анемией и ожирением. Поиск литературы проводился в таких базах данных, как Medline, Scopus, Web of Science, Google Scholar и PubMed. Поиск проводился по всем типам публикаций на английском и русском языках по ключевым словам, «беременность», «железодефицит», «анемия», «избыточная масса тела», «ожирение», «воспаление» и «ИМТ».

Результаты: В нашем обзоре анализируется вероятность повышения заболеваемости ЖД/ЖДА, вызванной ожирением, у беременных женщин. Несмотря на многочисленные исследования, влияние массы тела матери на риск ЖД/ЖДА во время беременности остается неясным. Рассмотрены уровни маркеров метаболизма железа при наличии воспаления. Ожирение до беременности несет в себе больший риск развития ЖД/ЖДА для матери во время беременности и в послеродовом периоде, а также для ребенка.

Заключение: Таким образом, предлагается более внимательно изучить уровень железа по триместрам. Необходимо ввести в практику антенатальных и послеродовых консультаций четко определенные процедуры триместровой оценки железа и воспалительного статуса.

Ключевые слова: *беременность, анемия, дефицит железа, избыточная масса тела, ожирение, воспаление, ИМТ.*

ЖҮКТІЛІК КЕЗІНДЕГІ АНЕМИЯНЫҢ ДАМУЫМЕН ПРЕГЕСТАЦИЯЛЫҚ СЕМІЗДІКТІҢ БАЙЛАНЫСЫ: ӘДЕБИ ШОЛУ

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Аңдатпа

Өзектілігі: Семіздігі бар жүкті әйелдердің ағзасында гепсидиннің шамадан тыс экспрессиясымен байланысты жеңіл қабыну реакциясы болады. Қабыну гепсидин деңгейін жоғарылату арқылы темір алмасуына әсер етуі мүмкін. Семіздік пен анемия бүкіл әлемде күрделі мәселелер болып табылады және жүкті әйелдер жиі осындай жағдайлардан зардап шегеді. Семіздігі бар жүкті әйелдер қалыпты дене салмағының индексі бар әйелдерге қарағанда жүктілік кезінде көбірек салмақ қосады. Жүктілікке дейінгі семіздік жүкті әйелдер мен олардың жаңа туған нәрестелерінде жиі кездесетін темір тапшылығы (ТТ) және темір тапшылығы анемиясын (ТТА) қоса алғанда, жүктілік қаупінің жоғарылауымен байланысты.

Бұл шолу семіз жүкті әйелдердегі темір тапшылығының (ID) немесе темір тапшылығы анемиясының (IDA) ықтимал негізгі себебі ретінде қабыну медиаторының белсендірілуімен байланысты семіздікті зерттейді.

Зерттеудің мақсаты: Бұл шолудың мақсаты жүкті әйелдердегі жүкті әйелдердегі темір алмасуының молекулалық маркерлері мен қабыну медиаторларының деңгейін өлшеу туралы есеп беретін ағымдағы зерттеулердің нәтижелерін қорытындылау болып табылады.

Әдістер: Бұл шолуда жарияланған мақалалардың талдауы және анемия және семіздікпен ауыратын жүкті әйелдер туралы қазіргі уақытта қолда бар деректер кіреді. Әдебиеттерді іздеу Medline, Scopus, Web of Science, Google Scholar және PubMed сияқты деректер қорларында жүргізілді. Іздеу ағылшын және орыс тілдерінде жарияланған зерттеулердің барлық түрлері бойынша «жүктілік», «темір тапшылығы», «анемия», «артық салмақ», «семіздік», «қабыну» және «ВМІ» түйінді сөздері бойынша жүргізілді.

Нәтижелері: Бұл шолу семіздіктің салдарынан болған қабынудың жүкті әйелдерде ТТ/ТТА жиілігінің артуына ықпал ете алатынын бағалайды. Көптеген зерттеулерге қарамастан, ана салмағының жүктілік кезіндегі ТТ/ТТА қаупіне әсері әлі толықтай анық емес. Біз бұл шодуда қабыну кезінде темір алмасуының маркерлерінің деңгейін талқылаймыз. Жүктілікке дейінгі семіздік ана үшін жүктілік кезінде және босанғаннан кейінгі кезеңде, сондай-ақ бала үшін ТТ/ТТА даму қаупі жоғары деп болжаймыз.

Қорытынды: Осылайша, біз триместр бойынша темір деңгейін мұқият қарауды ұсынамыз. Антенатальды және босанғаннан кейінгі консультацияларда темір мен қабыну жағдайын триместрлік бағалаудың нақты анықталған процедураларын енгізу қажет.

Кілт сөздер: *жүктілік, анемия, темір тапшылығы, артық салмақ, семіздік, қабыну, ДМИ.*

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