IJNRD.ORG ISSN : 2456-4184
INTERNATIONAL JOURNAL OF NOVEL RESEARCH AND DEVELOPMENT (IJNRD) | IJNRD.ORG
An International Open Access, Peer-reviewed, Refereed Journal

HYPEREMESIS GRAVIDARUM : A LITERATURE REVIEW

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ABSTRACT :

Hyperemesis gravidarum is a complex condition with a multifactorial etiology characterized by severe intractable nausea and vomiting. Despite a high prevalence, studies exploring underlying etiology and treatments are limited. We performed a literature review, focusing on articles published over the last 10 years, to examine current perspectives and recent developments in hyperemesis gravidarum.

KEYWORDS :

Hyperemesis gravidarum

Pregnancy

INTRODUCTION :

Up to 80% of all pregnant women experience some form of nausea and vomiting during their pregnancy. The International Statistical Classification of Disease and Related Health Problems, Tenth Revision, defines hyperemesis gravidarum (HG) as persistent and excessive vomiting starting before the end of the 22nd week of gestation and further subdivides the condition into mild and severe, with severe being associated with metabolic disturbances such as carbohydrate depletion, dehydration, or electrolyte imbalance. HG is a diagnosis of exclusion, characterized by prolonged and severe nausea and vomiting, dehydration, large ketonuria, and more than 5% body weight loss. Affecting approximately 0.3%–2.0% of pregnancies, HG is the commonest indication for admission to hospital in the first half of pregnancy and is second only to preterm labor as a cause of hospitalization during pregnancy.

According to the Hyperemesis Education and Research Foundation, conservative estimates indicate that HG can cost a minimum of $200 million annually in in-house hospitalizations in the United States. Taking into account other factors such as emergency department treatments, potential complications of severe HG, and

the fact that up to 35% of women with paid employment will lose time from work through nausea, the actual cost of HG to the economy is significantly higher. In a related economic analysis, Piwko et al projected that the United States spends nearly $2 billion in costs attributed to pregnancy-related nausea and vomiting; 60% of this expenditure is a result of direct costs (eg, drugs, hospital admission), and 40% is a result of indirect costs (eg, time lost from work).

To date, studies investigating the association between HG and adverse pregnancy outcomes and maternal morbidities have provided conflicting results. In all aspects of research involving HG, the interpretation of results and associations must be with caution, as the majority of the studies have been limited by retrospective study design, small numbers, bias, lack of control for potential confounders, and variable definitions of HG.

Thus, to examine current clinical perspectives of HG, we performed a review of MEDLINE (1994–January 2014), EMBASE (1994–January 2014), and the Cochrane Library. Articles related to "hyperemesis gravidarum" and/or "nausea and vomiting of pregnancy" were considered for inclusion in our review. Reference lists of selected articles were reviewed to identify additional articles. Although the review focused on articles published in the last 10 years, a second search with unrestricted time limits was performed to identify key papers related to HG that were also considered in the review.

CAUSES :

Almost all pregnant people experience some degree of morning sickness. Morning sickness is nausea and vomiting during pregnancy. Despite the name, morning sickness isn't confined to the morning. It can occur at any time. Morning sickness and HG seem to have a connection to human chorionic gonadotropin (hCG). This is a hormone created during pregnancy by the placenta. Your body produces a large amount of this hormone at a rapid rate early in pregnancy. These levels typically peak about 10 to 12 weeks into your pregnancy and then begin to decline.

EPIDEMIOLOGY :

Up to ninety percent of women experience nausea during pregnancy. Studies showed that approximately 27 to 30 percent of women experience only nausea, while vomiting may be seen in 28 to 52 percent of all pregnancies. The incidence of hyperemesis gravidarum ranges from 0.3 to 3 percent, depending on the literature source. Geographically, hyperemesis appears to be more common in western counties.

PATHOPHYSIOLOGY :

The exact cause of hyperemesis gravidarum remains unclear. However, there are several theories for what may contribute to the development of this disease process.

Hormone Changes

- Levels of human chorionic gonadotropin (hCG) have been implicated. hCG levels peak during the first trimester, corresponding to the typical onset of hyperemesis symptoms. Some studies show a correlation between higher hCG concentrations and hyperemesis. However, this data has not been consistent.
- Estrogen is also thought to contribute to nausea and vomiting in pregnancy. Estradiol levels increase early in pregnancy and decrease later, mirroring the typical course of nausea and vomiting in pregnancy. Additionally, nausea and vomiting are the known side effects of estrogen-containing medications. As the level of estrogen increases, so does the incidence of vomiting.

Changes in the Gastrointestinal System

- It is well-known that the lower esophageal sphincter relaxes during pregnancy due to the elevations in estrogen and progesterone. This leads to an increased incidence of gastroesophageal reflux disease (GERD) symptoms in pregnancy, and one symptom of GERD is nausea. Studies examining the relationship between GERD and emesis in pregnancy report conflicting results.

Genetics

- An increased risk of hyperemesis gravidarum has been demonstrated among women with family members who also experienced hyperemesis gravidarum.
- Two genes, GDF15 and IGFBP7, have been potentially linked to the development of hyperemesis gravidarum.

TREATMENT AND MANAGEMENT :

Treatment should be guided by the American College of Obstetrics and Gynecology (ACOG) Nausea and Vomiting in Pregnancy guidelines. Initial treatment should begin with non-pharmacologic interventions such as switching the patient's prenatal vitamins to folic acid supplementation only, using ginger supplementation (250 mg orally 4 times daily) as needed and applying acupressure wristbands. If the patient continues to experience significant symptoms, the first-line pharmacologic therapy should include a combination of vitamin B6 (pyridoxine) and doxylamine. Three dosing regiments are endorsed by ACOG, including pyridoxine 10 to 25 mg orally with 12.5 mg of doxylamine three or four times per day, 10 mg of pyridoxine and 10 mg of doxylamine up to 4 times per day, or 20 mg of pyridoxine and 20 mg of doxylamine up to 2 times per day. These are all FDA pregnancy category A medications.

Second-line medications include antihistamines and dopamine antagonists such as dimenhydrinate 25 to 50 mg every 4 to 6 hours orally, diphenhydramine 25 to 50 mg every 4 to 6 hours orally, prochlorperazine 25 mg every 12 hours rectally, or promethazine 12.5 to 25 mg every 4 to 6 hours orally or rectally. If the patient continues to experience significant symptoms without exhibiting signs of dehydration, metoclopramide, ondansetron, or promethazine may be given orally. In the case of dehydration, intravenous fluid boluses or continuous infusions of normal saline should be given in addition to intravenous metoclopramide, ondansetron, or promethazine. Electrolytes should be replaced as needed. Severe refractory cases of hyperemesis gravidarum may respond to intravenous or intramuscular chlorpromazine 25 to 50 mg or methylprednisolone 16 mg every 8 hours, orally or intravenously.

COMPLICATIONS :

As hyperemesis gravidarum involves at least two patients, both the mother and the fetus(s) must be considered when discussing complications.

Maternal Complications

In severe cases of hyperemesis, complications include vitamin deficiency, dehydration, and malnutrition, if not treated appropriately. Wernicke encephalopathy, caused by vitamin-B1 deficiency, can lead to death and permanent disability if left untreated. Additionally, there have been case reports of injuries secondary to forceful and frequent vomiting, including esophageal rupture and pneumothorax. Electrolyte abnormalities such as hypokalemia can also cause significant morbidity and mortality. Additionally, patients with hyperemesis may have higher rates of depression and anxiety during pregnancy.

Fetal Complications

Studies report conflicting information regarding the incidence of low birth weight and premature infants in the setting of nausea and vomiting in pregnancy. However, studies have not shown an association between hyperemesis and perinatal or neonatal mortality. The frequency of congenital anomalies does not appear to increase in patients with hyperemesis.

CONCLUSION :

Despite the prevalence and considerable morbidity associated with HG, good-quality research investigating the underlying etiology and interventions to treat and prevent HG remains scarce. Exploring new pharmacological interventions in pregnant women for the prevention and treatment of HG remains elusive, and this may be a result of avoiding inducing unnecessary risk for the developing fetus. Controversies such as that involving the administration of thalidomide to women with morning sickness, which subsequently resulted in significant congenital malformations, has likely discouraged researchers from investigating other interventions for HG. As a result, the current mainstay of treatment remains regular hydration and antiemetics. Nonetheless, because of the prevalence and morbidity associated with this condition, safe, well-conducted, good-quality research is needed to investigate and clarify the etiology, prevention, and treatment of this condition.

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