HYPEREMESIS GRAVIDARUM

Presented by : Jood Allan
Yaqut madadha

Supervised by : Dr. Abdulfatahsalem

Nausa and vomiting in pregnancy

- Nausea and vomiting in pregnancy (NVP) affects up to 80% of pregnant women and is one of the most common indications for hospital admission among pregnant women, with typical stays of between 3 and 4 days
  - NVP is defined as the symptom of nausea and/or vomiting during early pregnancy where there are no other causes
  - It typically starts between the 4-7th weeks of pregnancy, peaks around 9th week, and resolves by the 20th week in 90% of women. Rule out other causes if first presentation is after 10 completed weeks of pregnancy.

HYPEREMSIS GRAVIDARUM

- hyperemesis gravidarum can be defined as

intractable vomiting associated with loss of more than 5% of pre pregnancy weight, dehydration,

electrolyte disturbances, or need for hospital admission.

ATEIOLGY

- The aetiologicaltheories for NVP and HG range

from the fetoprotectiveand genetic to the biochemical, immunological and biosocial. They are primarily thought to be associated with rising levels of beta human chorionic gonadotrophin (hCG) hormone, progesterone, estrogen.

Risk factors and associations

- First pregnancy
- Multiple pregnancy
- History of severe nausea and vomiting in previous pregnancies, motion
- sickness, or nausea with oral contraceptive use
- Gestational Trophoblastic disease (GTD), including molar pregnancy
- History of migraines
- History of first degree relative with NVP
- Obesity
- Stress
- Being seropositive for Helicobacter pylor

- * The condition spontaneously resolves in the vast majority of patients and complications are rare.

Complications

1. Electrolyte imbalance: NVP and HG are associated with hyponatraemia, hypokalaemia, low serum urea, raised haematocritand ketonuria with a metabolic hypochloraemicalkalosis. If severe, a metabolic acidaemiamay develop
2. Abnormal LFTs: Liver function tests are abnormal in up to 40% of women with HG, with the most likely abnormality being a rise in transaminases. Bilirubin levels can be slightly raised but without jaundice, and amylase levels can be mildly raised too. These abnormalities improve as the HG resolves . The LFT abnormalities are mild and inconsequential with regard to the outcome. No specific intervention is required; however, atypical patterns may necessitate other investigations to rule out another underlying disease

.

3. Abnormal thyroid function test : In two-thirds of patients with HG, there may be abnormal thyroid function tests (based on a structural similarity between thyroid-stimulating hormone [TSH] and hCG) with a biochemical thyrotoxicosisand raised free thyroxine levels with or without a suppressed thyroid stimulating hormone level. These patients rarely have thyroid antibodies and are euthyroid clinically. The biochemical thyrotoxicosis resolves as the HG improves22 and treatment with antithyroid drugs is inappropriate

4.Acute: kidney injury (AKI):Volume depletion—Volume depletion, typically due to severe vomiting (ie, hyperemesis gravidarum), is a common cause of prerenal AKI early in pregnancy (<20 weeks).

5. Wernicke's encephalopathy

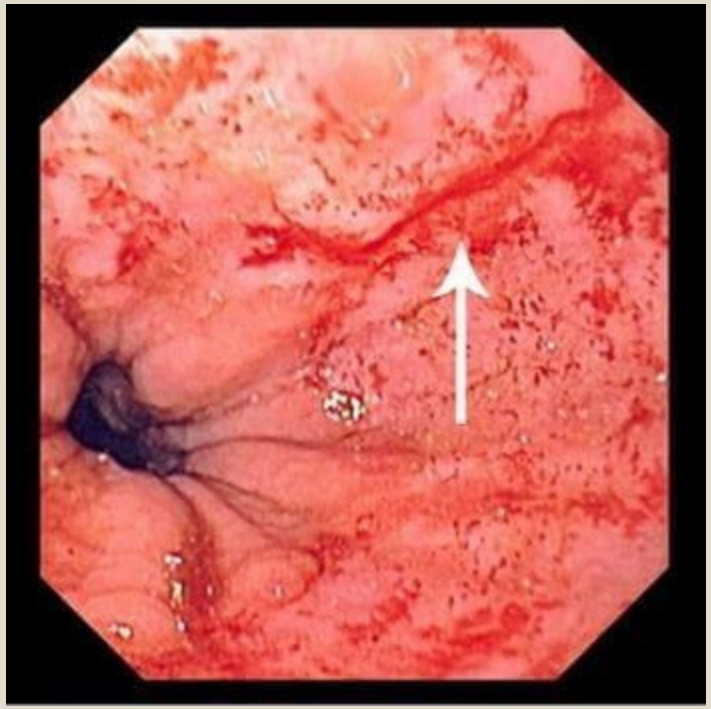
Wernicke's encephalopathy due to vitamin B1

(thiamine) deficiency classically presents with

blurred vision, unsteadiness and confusion/memory areflexia, gait and/or finger–nose ataxia. problems/drowsiness and on examination there is usually nystagmus, ophthalmoplegia, hyporeflexia or

- Depression and poor psychological health were found to be associated with NVP and HG in numerous studies, but resulted from the disease and were not the cause of HG or NVP. A prospective case–control study of 32 women compared with 41 matched controls found that, compared with controls, women with HG had significantly higher levels of somatisation, depression, anxiety and overall psychological distress even when HG had resolved to mild NVP.

6. Mechanical complications including Mallory-Weiss tears, pneumothorax, pneumomediastinum, diaphragmatic rupture, and gastroesophageal rupture, which is Boerhaavesyndrome



DIAGNOSIS

Signs and symptoms

- Loss of 5% or more of pre-pregnancy body weight
- Dehydration, causing ketosis,[16] and constipation
- Nutritional disorders, such as vitamin B1 (thiamine) deficiency, vitamin B6 (pyridoxine) deficiency or vitamin B12 (cobalamin) deficiency
- Metabolic imbalances such as metabolic ketoacidosis or thyrotoxicosis
- Physical and emotional stress
- Difficulty with activities of daily living

How is NVP diagnosed?

NVP should only be diagnosed when onset is in the first trimester of pregnancy and other causes of nausea and vomiting have been excluded.Onset of NVP is in the first trimester and if the initial onset is after 10+6 weeks of gestation, other causes need to be considered. It typically starts between the fourth and seventh weeks of gestation, peaks in approximately the ninth week and resolves by the 20th week in 90% of women.

How is HG diagnosed?

HG can be diagnosed when there is protracted NVP with the triad of more than 5% prepregnancy weight loss, dehydration and electrolyte imbalance..

history of NVP/HG

nausea, vomiting, hypersalivation, spitting, loss of weight,inability to tolerate food and fluids, effect on quality of life ● History to exclude other causes:– abdominal pain– urinary symptoms– infection– drug history– chronic Helicobacter pylori infection

physical exam:

● Pulse● Blood pressure● Oxygen saturations● Respiratory rate● Abdominal examination● Weight● Signs of dehydration● Signs of muscle wasting● Other examination as guided by history

STAGING:

How can the severity of NVP be classified?

PUQE (Pregnancy-Unique Quantification of Emesis)

- to assess the **severity of emesis** (nausea and vomiting) in pregnancy
- This questionnaire contains **three** questions:

regarding the time-span of **nausea**, **vomiting**, and **retching**, respectively,
as well as one question assessing the **global psychological and physical quality of life** (QOL)

- Initially, the questionnaire evaluated symptoms during the last 12 hours, but it has been modified to encompass **24 hours** and the whole of the first trimester of pregnancy
- If your PUQE-24 score is:

- between **4 – 6**, you have **mild** NVP
- between **7 – 12**, you have **moderate** NVP
- ⩾**13**, you have **severe** NVP (also **known as HG**).

Appendix II: Pregnancy-Unique Quantification of Emesis (PUQE) Index[20]

Total score is sum of replies to each of the three questions. PUQE-24 score: Mild ≤ 6; Moderate = 7–12; Severe = 13–15.

Motherisk PUQE-24 scoring system					
In the last 24 hours, for how long have you felt nauseated or sick to your stomach?	Not at all (1)	1 hour or less (2)	2–3 hours (3)	4–6 hours (4)	More than 6 hours (5)
In the last 24 hours have you vomited or thrown up?	7 or more times (5)	5–6 times (4)	3–4 times (3)	1–2 times (2)	I did not throw up (1)
In the last 24 hours how many times have you had retching or dry heaves without bringing anything up?	No time (1)	1–2 times (2)	3–4 times (3)	5–6 times (4)	7 or more times (5)

PUQE-24 score: Mild ≤ 6; Moderate = 7–12; Severe = 13–15.

How many hours have you slept out of 24 hours? _____ Why? _____

On a scale of 0 to 10, how would you rate your wellbeing? _____

0 (worst possible) → 10 (the best you felt before pregnancy)

Can you tell me what causes you to feel that way?

INVESTIGATION

The evaluation should include:

- urinalysis (to check for ketonuria and specific gravity)
- complete blood count (CBC) and electrolyte evaluation.

(An elevation in hemoglobin or hematocrit may be due to hemoconcentration in the setting of dehydration)

(Potassium, calcium, magnesium, sodium, and bicarbonate may be affected by prolonged bouts of vomiting and reduced oral intake of fluids)

- KFTs : Significant dehydration may result in acute kidney injury as evidenced by elevated serum creatinine, blood urea nitrogen, and reduced glomerular filtration.
- Thyroid tests, lipase, and liver function testing may also be completed to evaluate for alternate diagnoses.
- Radiographic studies: may be appropriate to rule out alternate diagnoses.

1- Obstetrical ultrasounds may be considered to rule out multiple gestations, ectopic pregnancy, and gestational trophoblastic disease, depending on the patient's history and prior obstetrical evaluations.

2- Magnetic resonance imaging (MRI) may be used to assess alternative diagnoses, such as appendicitis.

MANAGEMENT

- Hospitalization / NPO
- IV fluids & correct electrolytes
  - - IV or PR Antiemetics
  - (as metoclopramide , meclozine , ondansetrone)
    - Rarely termination (in severe cases of encephalopathy)

- All patients with HG require **emotional support** with frequent reassurance and encouragement from nursing and medical staff.
- Drugs that may cause nausea and vomiting should be temporarily **discontinued**. The commonest example is **iron supplements**.
- Any woman who is **ketotic** and **unable to maintain adequate hydration** requires **i.v.fluids** and **parenteral anti-emetics.**
- For **less severe** cases, outpatient management with administration of **i.v.fluid therapy** and **anti-emetics** as required should be **first line**.
- The natural history of HG is gradual improvement with increasing gestation, although in a minority of women symptoms may persist beyond 20 weeks' gestation. The only definitive cure is termination of the pregnancy

1-Intravenous fluid therapy

- Adequate and appropriate fluid and electrolyte replacement is the most important component of management.
- Infusion of dextrose-containing fluids (dextrose saline, 5% dextrose, 10% dextrose) is mistakenly thought by some to be desirable to provide the patient with calories, but this assumption is erroneous and dangerous.
- Normal saline (sodium chloride 0.9%; 150 mmol/L Na+) and Hartmann's solution (sodium chloride 0.6%; 131 mmol/L Na+) are appropriate solutions.
- Correction of the hypokalaemia is essential and it is usually necessary to use infusion bags containing 40 mmol/L of potassium chloride.
- There is no place for the use of double-strength saline (2n saline), even in cases of severe hyponatraemia, as this results in too rapid a correction of serum sodium with the risk of central pontine myelinolysis.

2. Thromboprophylaxis

HG is a **risk factor for venous thrombosis** probably because of dehydration and immobilization .Therefore, all women admitted with hyperemesis should receive appropriate doses of **low-molecular-weight heparin** (LMWH).

3. Thiamine Therapy

Thiamine supplementation **should be given** to anyone suffering from **prolonged vomiting.**

Requirements for thiamine increase during pregnancy to **1.5 mg/day**, and women admitted with a diagnosis of hyperemesis have usually been vomiting for at least 1–2 weeks prior to admission.

- If the woman is able to tolerate tablets, thiamine can be given as **thiamine hydrochloride tablets 25–50 mg thrice daily**. If i.v.treatment is required for those unable to tolerate tablets, this is given as thiamine **100 mg diluted in 100 mL of normal saline** and **infused over 30–60 minutes**.
- Treatment (as opposed to prevention) of **Wernicke's encephalopathy requires much higher doses of thiamine.**

4. PHARMACOLOGICAL THERAPY

Anti-emetics:

- Women presenting to secondary care who **do not respond to i.v.fluids and electrolytes alone** should be offered anti-emetic therapy.
- Extensive data exist to show a **lack of teratogenesis** or other adverse pregnancy outcomes with:

- **Antihistamines** (H1-receptor antagonists, e.g., promethazine, cyclizine, cinnarizine, doxylamine, dimenhydrinate)
- **Phenothiazines** (chlorpromazine, prochlorperazine)
- **Dopamine antagonists** (metoclopramide, domperidone)
- **Serotonin (5HT3) inhibitors** (ondansetron)

If symptoms do not improve, the anti-emetic should be prescribed and given regularly.

Histamine2 (H2)-receptor blockers and proton pump inhibitors (PPIs):

• H2-receptor blockers (e.g., ranitidine) and the PPIs (e.g., omeprazole) are used in cases where **oesophagitis** or **gastritis** accompanies the nausea and vomiting of HG.They are **safe** for use in pregnancy.

Corticosteroids:

Corticosteroids have resulted in dramatic and rapid improvement in case series of women with **severe refractory HG.**

- They **should not be used until** conventional treatment with i.v.fluid replacement and regular parenteral anti-emetics has **failed**
- Suggested doses are **prednisolone** 40–50 mg **orally daily** in divided doses or **hydrocortisone** 100 mg **i.v.twice daily.**
- In cases who do **respond** to steroid therapy, the dose **must be reduced slowly.**
- In cases who **do not respond** to steroid therapy, it should be **discontinued.**

Enteral feeding:

- If women fail to respond to i.v.fluid and anti-emetic treatment and corticosteroid treatment, then nutritional support may be required in the form of enteral or parenteral feeding.
- When the gastrointestinal tract is intact and usable, it is preferable to use enteral rather than parenteral hyperalimentation to treat malnutrition
- Enteral feeding options include nasogastric (NG), nasoduodenal or nasojejunal (NJ) tubes, or percutaneous endoscopic gastrostomy or jejunostomy feeding.

Total parenteral nutrition:

- TPN with peripherally inserted central catheters (PICC line) is often better tolerated than enteral, but it carries more risk.
- TPN has also been shown to have a rapid therapeutic effect in some case
- Metabolic and infectious complications are a risk and strict protocols and careful monitoring are obligatory . The central line site must be inspected regularly for signs of infection.
- Phlebitis and thrombosis are other recognized complications of TPN.
- Parenteral feeding is usually reserved for extremely severe life-threatening cases.

Points to remember:

- - HG is a diagnosis of exclusion.
- - HG may be associated with both abnormal liver and thyroid function tests.
  - Adequate and appropriate (normal saline and potassium chloride) fluid and electrolyte replacement is the most important component of management.
  - Thiamine supplementation to prevent Wernicke's encephalopathy and thromboprophylaxis should be given to all women admitted with hyperemesis.
  - The common anti-emetics are not teratogenic.
  - Corticosteroids may have a role to play in severe resistant cases.

THANK YOU