



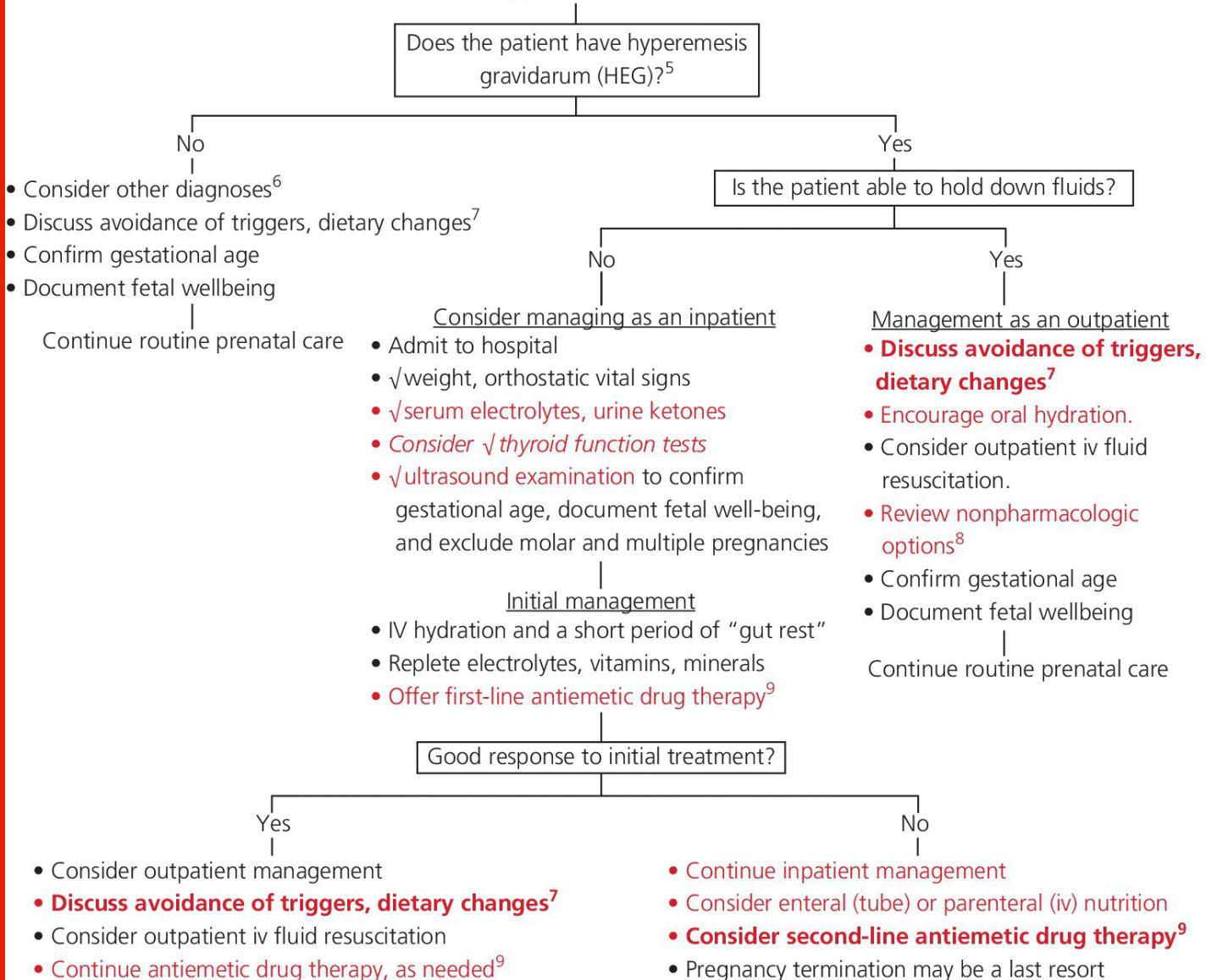
Learn simply

Hyperemesis Gravidarum

Passion profession same

Nausea and vomiting in pregnancy (NVP)

- Understand the pathophysiology of NVP¹
- Exclude underlying pathologic conditions²
- Be aware of risk factors for NVP³
- Discuss strategies for prevention⁴



1. Some degree of nausea and vomiting in pregnancy (NVP) or “morning sickness” occurs in >80% of all pregnancies. The mean onset of symptoms is 5-6 weeks, peaking at 9 weeks, and usually abating by 16-18 weeks; however, symptoms continue into the third trimester in 15-20% of women and 5% have symptoms that persist to term. The cause of NVP is not known, although it is thought to be related to hCG production.
2. NVP is not a sign of an unhealthy pregnancy; in fact, pregnancies complicated by NVP have a better outcome than those that are not, with lower rates of miscarriage and stillbirth.
3. Conditions that may present with severe NVP include molar pregnancy, higher-order multiple pregnancy, and women with theca lutein cysts. An ultrasound can exclude these underlying conditions.
4. Risk factors for NVP include: (i) severe NVP in a prior pregnancy (recurrence rate is 15-20%); (ii) nausea and vomiting after estrogen exposure (such as combined oral contraceptive pill), with motion sickness, with migraine, or with exposure to certain tastes; (iii) a pre-existing psychiatric disorder; and (iv) diabetes.
5. A number of protective factors have also been identified, including anosmia (inability to smell), advanced maternal age, and smoking.



1. Several strategies have been tried to prevent NVP, but most have been shown to be no better than placebo. The only intervention that may be somewhat effective in preventing NVP is multivitamin supplementation from the time of conception.
2. Hyperemesis gravidarum (HEG) refers to the severe end of the spectrum of NVP, and complicates 0.3-2% of all pregnancies.
3. It is a clinical diagnosis made in the first trimester and characterized by three criteria: (i) persistent vomiting; (ii) weight loss >5% of pre-pregnancy body weight; and (iii) ketonuria.
4. A number of other laboratory abnormalities have been described in the setting of HEG, including electrolyte derangements (hypokalemia, metabolic alkalosis), hemoconcentration (increase in hematocrit), abnormal liver enzyme (elevated ALT and mild hyperbilirubinemia), and hyperthyroidism (mildly elevated free T4 and depressed TSH, although patients are clinically euthyroid and do not need treatment). However, none of these abnormalities is diagnostic of HEG.
5. The differential diagnosis of HEG is extensive, and includes medication-induced nausea and vomiting, infections (gastroenteritis), small bowel obstruction, peptic ulcer disease, inflammatory bowel disease, and (rarely) central nervous system disorders, malignancies, and endocrine/metabolic derangements. After 20 weeks, preeclampsia must always be excluded. NVP predating pregnancy or with abdominal pain, fever or neurologic signs suggests an alternative cause.



1. Patients should be counselled about avoidance of environmental triggers for NVP, including odors (coffee, perfume, food, smoke), noise, and visual or physical motion (flickering lights).
2. Dietary changes may relieve NVP in some women, such as frequent, small, high-carbohydrate/low-fat meals; elimination of spicy foods; eating salty or high-protein meals and cold, carbonated, or sour fluids (ginger ale, lemonade). Powdered ginger (1 g daily) and foods containing ginger (ginger lollipops) have been shown to be effective in mild NVP.
3. Nonpharmacologic interventions (such as acupuncture, acupressure wristbands, hypnosis, and psychotherapy) have been proposed to treat mild NVP, but with variable results. Meta-analyses of randomized trials show no significant benefit over sham therapy.



1. Several drugs have been shown to be more effective than placebo in treating NVP with few side-effects and no increased risk of congenital anomalies.
2. A stepwise approach to antiemetic therapy is recommended.
3. **First-line agents include:**
 - (i) pyridoxine (vitamin B6) (10-25 mg q 8 h po) with or without the antihistamine doxylamine succinate (20 mg);
 - (ii) antihistamines (H1-receptor antagonists) such as promethazine (12.5-25 mg q 4 h po/im/pr).
4. **Second-line agents include:**
 - (i) the selective 5-HT₃ serotonin receptor antagonist ondansetron (8 mg q 12 h po/im);
 - (ii) dopamine antagonists such as metoclopramide (5-10 mg q 8 h po/iv), prochlorperazine (5-10 mg q 3-4 h po/im or 25 mg q 12 h pr), phenothiazines or droperidol; and
 - (iii) a short course of corticosteroids (methylprednisolone 16 mg q 8 h po/iv 3 3-14 days).
5. The efficacy of corticosteroids is unproven and it has been associated with preterm premature rupture of membranes (PPROM) and oral clefts when administered <10 weeks of gestation; as such, they should be used only as a last resort.

