OBSTETRICS

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HYPER EMESIS

GRAVIDARUM
Define as: persistent vomiting in pregnancy not due to other causes, an indicator of acute starvation (large ketonuria), loss of 5% of pre pregnancy weight.

Nausea and vomiting in pregnancy are extremely common; 70–80 per cent of women experience these symptoms early in their pregnancy and approximately 35 per cent of all pregnant patients are absent from work on at least one occasion through nausea and vomiting.
- **INCIDENCE**
- 0.3 - 2% higher incidence in younger than in older women
AETIOLOGY

- Related to a product of placental metabolism it occurs commonly with advanced molar gestation and multiple gestation.
- There is correlation with, HCG, estradiol level, pyridoxin deficiency, psychological factors, biochemical hyperthyroidism.
Worse nusea and vomiting
Younger age, hx of motion sickness, hx of contraception sickness, hx of migaine, earlier in the day, female gender of patient

Associations unique to n.v.of pregnancy
Family hx, female gender of fetus, hx of migraine, multiple gestation, down syndrome, molar gestation
Maternal metabolic disorder
Genetics of HG
In monozygotic twins
Siblings and mother of patients
Ethnic groups

PSYCHOPATHOLOGY
As a result of the mother's inability to respond to life stress
SIGN AND SYMPTOMS

N, V, excess salivation (ptylism) 60% of patients, dehydration (dry and coated tongue, skin turgor decrease, postural changes in blood pressure), significant weight loss, jaundice, various palsy, metabolic acidosis
1-Hyper thyroidism transient and dose not require specific treatment TSH suppressed
2-Liver enzymes increase transiently, bilirubin concentration increase, liver dysfunction resolve with termination of pregnancy
3-s. amylase increase
4-electrolyte abn, decrease Na, k, cl
5-complete blood picture, increase PCV
6- GUE, for keton, albumin, and specific gravity
7-Renal function test increase blood urea and creatinine
8- metabolic alkalosis
9-US to confirmation of pregnancy and rule out molar pregnancy
WERNICKE'S ENCEPHALOPATHY

CNS dysfunction is due to a deficiency in thiamine vitamin B1 present with apathy, confusion, ataxia, nystagmus, blindness, majority either died or end with permanent residual dysfunction.

Prevented 3mg of B1 daily if vomiting sufficient to require IV hydration. 100mg thiamine parenterally daily for 3 days if patient in hospital for HG.

Cardiac dysfunction

Pneumothorax, mallory weiss tears of esophagus, rupture, splenic avulsion, acute tubular necrosis, central pontine myelolysis, and acute peripheral neuropathy.

If HG associated with weight loss more than 5% associated with poor fetal outcome, low birth weight fetus, fetal death.
DIFFERENTIAL DIAGNOSIS

- GIT
- Peptic ulcer
- Pancreatitis
- Gastroenteritis
- GUT
- Pyelonephritis
- Uremia
- Degenerative uterine leiomyoma
- Renal stone
- METABO
- DKA
- Hyperthyroidism
- NEUROLOGICAL
- Tumors of CNS
- MISCELLANEOUS
- Drug intolerance
- Psychological
- PREGNANCYRELATED CONDITION
- Acute fatty liver of pregnancy
MANAGEMENT

- **Prevention** there is evidence that women who taking multivitamins at the time of conception and in early pregnancy are less likely to require intervention for HG later in pregnancy

- **Diet and support**
- To at small portion of whatever seems palatable when ever symptoms allow ,less nausea associated with protein meals compared to CHO and fat and liquid meals are better tolerated than solid

- **Redesign the home environment** to avoid sensory stimuli that provoke symptoms,
**Indication for admission:**

1. Severe dehydration and inability to tolerate oral fluids
2. Severe electrolyte abnormality
3. Acidosis
4. Infection
5. Malnutrition
6. Weight loss