HYPEREMESIS GRAVIDARUM

DEFINITION: **It is a severe type of vomiting of pregnancy which has got deleterious effect on the health of the mother and/or incapacitates her in day-to-day activities**

Effects of H G:

dehydration, metabolic acidosis (from starvation) or alkalosis (from loss of hydrochloric acid), electrolyte imbalance (hypokalemia) and weight loss.

INCIDENCE:

There has been marked fall in the incidence during the last 30 years. It is now a rarity in hospital practice (less than 1 in 1000 pregnancies).

The reasons are — (a) Better application of family planning knowledge which reduces the number of unplanned pregnancies, (b) early visit to the antenatal clinic and (c) potent antihistaminic, antiemetic drugs.

ETIOLOGY:

The etiology is obscure but the following are the known facts:

(1) It is mostly limited to the first trimester

(2) It is more common in first pregnancy, with a tendency to recur again in subsequent pregnancies

(3) It has got a familial history — mother and sisters also suffer from the same manifestation

(4) It is more prevalent in hydatidiform mole and multiple pregnancy

(5) It is more common in unplanned pregnancies .

THEORIES:

Hormonal:

(a)Excess of chorionic gonadotropin or higher biological activity of hCG is associated. This is proved by the frequency of vomiting at the peak level of hCG and also the increased association with hydatidiform mole or multiple pregnancy when the hCG titer is very much raised

(b) High serum level of estrogen

(c) Progesterone excess leading to relaxation of the cardiac sphincter and simultaneous retention of gastric fluids due to impaired gastric motility.

Other hormones involved are: thyroxin, prolactin, leptin and adrenocortical hormones

(2) *Psychogenic*: It probably aggravates the nausea once it begins. But neurogenic element sometimes plays a role, as evidenced by its subsidence after shifting the patient from the home surroundings. Conversion disorder, somatization, excess perception of sensations by the mother are the other theories.

(3) *Dietetic deficiency*: Probably due to low carbohydrate reserve, as it happens after a night without food. Deficiency of vitamin B6, Vit B1 and proteins may be the effects rather than the cause.

(4) *Allergic* or *immunological basis.*

(5) *Decreased* gastric motility is found to cause nausea.

Whatever may be the cause of initiation of vomiting, it is probably aggravated by the neurogenic element.

Unless it is not quickly rectified, features of dehydration and carbohydrate starvation supervene and a vicious cycle of vomiting appears — vomiting → carbohydrate starvation → ketoacidosis → vomiting.

PATHOLOGY: There is no specific morbid anatomical findings. The changes in the various organs as described by Sheehan are the generalized manifestations of starvation and severe malnutrition.

Liver: There is centrilobular fatty infiltration without necrosis.

Kidneys: Usually normal with occasional findings of fatty change in the cells of first convoluted tubule, which may be related to acidosis.

Heart: A small heart is a constant finding. There may be subendocardial hemorrhage.

Brain: Small hemorrhages in the hypothalamic region giving the manifestation of Wernicke's encephalopathy. The lesion may be related to vitamin B1 deficiency

METABOLIC, BIOCHEMICAL AND CIRCULATORY CHANGES: The changes are due to the combined effect of dehydration and starvation consequent upon vomiting.

Metabolic: Inadequate intake of food results in glycogen depletion. For the energy supply, the fat reserve is broken down. Due to low carbohydrate, there is incomplete oxidation of fat and accumulation of ketone bodies in the blood. The acetone is ultimately excreted through the kidneys and in the breath. There is also increase in endogenous tissue protein metabolism resulting in excessive excretion of non-protein nitrogen in the urine. Water and electrolyte metabolizm are seriously affected leading to biochemical and circulatory changes.

Biochemical: Loss of water and salts in the vomitus results in fall in plasma sodium, potassium and chlorides.
The urinary chloride may be well below the normal 5 g/liter or may even be absent. Hepatic dysfunction results in acidosis and ketosis with rise in blood urea and uric acid; hypoglycemia; hypoproteinemia; hypovitaminosis and rarely hyperbilirubinemia.

Circulatory: There is hemoconcentration leading to rise in hemoglobin percentage, RBC count and hematocrit values. There is slight increase in the white cell count with increase in eosinophils. There is concomitant reduction of extracellular fluid.

Symptoms: Vomiting is increased in frequency with retching. Urine quantity is diminished even to the
stage of oliguria. Epigastric pain, constipation may occur. Complications may appear if not treated.

Signs: **Features of dehydration and ketoacidosis:** Dry coated tongue, sunken eyes, acetone smell in breath, tachycardia, hypotension, rise in temperature may be noted, jaundice is a late feature. Such late cases are rarely seen these days.

Vaginal examination and/or ultrasonography is done to confirm the diagnosis of pregnancy.

DIAGNOSIS: **The pregnancy is to be confirmed first**.
Thereafter, all the associated causes of vomiting
are to be excluded.

Ultrasonography is useful not only to confirm the pregnancy but also to exclude other, obstetric (hydatidiform mole, multiple pregnancy), gynecological, surgical or medical causes of vomiting

Investigations:

- **Urinalysis:** (1) Quantity—small (2) Dark color (3) High specific gravity with acid reaction
(4) Presence of acetone, occasional presence of protein and rarely bile pigments (5) Diminished or even absence of chloride.
- **Biochemical and circulatory changes:** The changes are mentioned previously. Routine and periodic estimation of the serum electrolytes (sodium, potassium and chloride) is helpful in the management of the case.
- **Ophthalmoscopic examination** is required if the patient is seriously ill. Retinal hemorrhage and detachment of the retina are the most unfavorable signs.
- **ECG** when there is abnormal serum potassium level.

Differential diagnosis:

When vomiting is persistent in spite of usual treatment other causes of severe vomiting (medical or surgical) should be considered

COMPLICATIONS:
The majority of the clinical manifestations are due to the effects of dehydration and
starvation with resulting ketoacidosis. Leaving aside those symptomatology, the following complications may
occur which are fortunately rare now-a-days. (1) Neurologic complications — (a) Wernicke's encephalopathy
due to thiamine deficiency (b) Pontine myelinolysis (c) Peripheral neuritis (d) Korsakoff's psychosis.
(2) Stress ulcer in stomach (3) Esophageal tear (Mallory-Weiss syndrome) (4) Jaundice (5) Convulsions and (6)
Coma and (7) Renal failure.

PREVENTION: The only prevention is to impart effective management to correct simple vomiting of pregnancy

MANAGEMENT

The principles in the management are:

- To control vomiting
- To correct the fluids and electrolytes imbalance
- To correct metabolic disturbances (acidosis or alkalosis)
- To prevent the serious complications of severe vomiting

Hospitalization:

Whenever a patient is diagnosed as a case of hyperemesis gravidarum, she is admitted.

Surprisingly, with the same diet and drugs used at home, the patient improves rapidly.

Fluids: Oral feeding is withheld for at least 24 hours after the cessation of vomiting. During this period,
fluid is given through intravenous drip method.

The amount of fluid to be infused in 24 hours is calculated as follows: The total amount of fluid approximates 3 liters, of which half is 5% dextrose and half is Ringer's
solution. Extra amount of 5% dextrose equal to the amount of vomitus and urine in 24 hours, is to be added.
With this regime — dehydration, ketoacidosis, water and electrolyte imbalance are likely to be rectified.

Serum electrolyte should be estimated and corrected if there is any abnormality. Enteral nutrition through nasogastric tube may also be given.

Drugs:

(a) **Antiemetic drugs promethazine** (Phenergan) 25 mg or **prochlorperazine** (Stemetil) 5 mg or **trifluopromazine** (Siquil) 10 mg may be administered twice or thrice daily intramuscularly. Trifluoperazine (Espazine) 1 mg twice daily intramuscularly is a potent antiemetic therapy. Vitamin B6 and doxylamine are also safe and effective.

Metoclopramide stimulates gastric and intestinal motility without stimulating the secretions. It is found useful.

(b) **Hydrocortisone** 100 mg IV in the drip is given in a case with hypotension or in intractable vomiting.
Oral method prednisolone is also used in severe cases.

(c) **Nutritional support —** with vitamin B1, Vit B6, Vit C and Vit B12 are given.

Nursing care: **Sympathetic but firm handling** of the patient is essential. Social and psychological support should be extended

Hyperemesis progress chart is helpful to assess the progress of patient while in hospital.

Daily record of pulse, temperature, blood pressure at least twice daily, intake-output, **urine** for acetone, protein, bile, blood biochemistry and ECG (when serum potassium is abnormal) are important.

Clinical features of improvement are evidenced by — (a) subsidence of vomiting (b) feeling of hunger (c) better look (d) disappearance of acetone from the breath and urine (e) normal pulse and blood pressure and (f) normal urine output.

Diet: Before the intravenous fluid is omitted, the foods are given orally. At first, dry carbohydrate foods like biscuits, bread and toast are given. Small but frequent feeds are recommended. Gradually full diet is restored.

Termination of pregnancy is rarely indicated. Intractable hyperemesis gravidarum inspite of therapy is rare these days.