Chapter 29 - Nausea and Vomiting

Episode Overview:

1) Describe the mechanism of development of a Hypochloremic Metabolic Alkalosis in vomiting
2) List commonly used anti-emetics including their dose and their receptor site of action.
3) List causes of vomiting specific to age groups: infant, newborn, child, and teen.

Wisecracks:

1) What are the three main areas providing afferent inputs to the vomiting centre?
2) Name 6 critical causes and 6 complications of vomiting

Rosen's in Perspective

Three phases of vomiting: - see fig 29-1

1. **Nausea**
    a. May occur in isolation
    b. "Vague, extremely unpleasant feeling that precedes vomiting"
        i. Pathways that cause nausea are not fully known
        ii. During nausea the muscle tone in the duodenum & jejunum increase and gastric tone decreases
            Also: hypersalivation, tachycardia repetitive swallowing
2. **Retching**
    a. "Rhythmic, synchronous contractions of the diaphragm, abdominal muscles, intercostal muscles against a closed glottis"
        i. Leads to increased abdominal pressure, and decreased intrathoracic pressure → gastric contents then move into stomach -> esophagus and out
3. **Vomiting**
    a. "Forceful expulsion of gastric contents through the mouth"
        i. The abdominal and thoracic muscles work together to simultaneously relax and contract certain muscles to expel the contents out.
        ii. The glottis is usually closed to prevent aspiration

Pivotal findings:

History

- Acute vomiting - more likely serious etiologies
- Chronic - more likely partial obstructions, motility disorders, neurologic conditions
- Timing
    - Delayed - 1 - 12 hours post eating:
        Gastric outlet obstruction or gastroparesis (diabetes)
- Content of vomit:
    - Bile - means duodenum and stomach are connected (no outlet obstruction)
    - Undigested food - achalasia, esophageal stricture, Zenker's diverticulum,
    - Feculent material - distal bowel obstruction
- Associated symptoms and signs:
    - Chronic nausea/vomiting with headaches - CNS pathology
    - Vomiting without nausea - CNS pathology
- Social/Medical history:
    - ETOH use
    - GI disease / surgeries
    - Medication list

Physical exam

- **Red flags:**
    - Adult vs. Pediatric exam varies - need to do a full physical exam
        - Bulging fontanelle
        - Projectile vomiting
        - Unusual odors
        - Visible bowel loops
        - Dental enamel loss with large parotid glands
    - Look for
        - CNS causes / labyrinthitis / stroke
        - GI infections or surgical problem
        - Cardiac / pulmonary disease / Liver disease

Ancillary studies

- Guided based on history and physical
    - CBC - of little use!
    - Electrolytes - rarely abnormal (unless >3 days of symptoms - hypoCl, hypoK)
    - Drug levels (EtOH, Tylenol, ASA)
    - Urine Beta-hCG and microanalysis (ketones, sugar, infection)
    - CT scan (abdominal and/or head)
    - **X-rays of little use**
    - ECG - to screen for ACS

1) Describe the mechanism of development of a Hypochloremic Metabolic Alkalosis in vomiting

Metabolic alkalosis

a. Due to loss of hydrogen ions in the vomit
b. Alkalosis further promoted by:
    i. Volume contractions
    ii. Hypokalemia
    iii. Chloride depletion → shift of extracellular hydrogen ions into cells
    iv. Increased aldosterone

Hypokalemia

c. Due to loss of potassium in the urine (and the alkalosis leads to large amounts of HCO3- in the distal tubule), AND there is secondary hyperaldosteronism

2) List commonly used antiemetics including their dose and their receptor site of action.

Treatment pathophysiology:

- Because the CTZ is stimulated by dopamine & other neurotransmitters, most of the drugs to relieve N/V are:
    - **Dopamine D2 antagonists** (metoclopramide / Maxeran)
    - **Serotonin receptor antagonists (**ondansetron / Zofran)
    - **Cholinergic & histamine receptors antagonists** are in the lateral vestibular nucleus (diphenhydramine, scopolamine, dimenhydrinate)
    - **Cannabinoid receptors** also inhibit reflex

Medications:

- Pharmacologic categories:
    - Histamine antagonists - inhibit vestibular stimulation and vestibular-cerebellar pathways
        - Also have some anticholinergic effects
        - Dimenhydrinate or meclizine - great for motion sickness
        - s/e: drowsiness, blurred vision, dry mouth, hypotension
        - Cetirizine - is less effective as an antiemetic, but is non-sedating
    - Muscarinic antagonists (anticholinergic)
        - Scopolamine (patch) or hyoscine
            - Good for motion sickness only
    - Dopamine antagonists - D2 receptor in the CTZ
        - Prochlorperazine, haloperidol, promethazine
            - s/e: dystonic reactions (4%), sedation, restlessness (16%)
                - Treated: with diphenhydramine or benztropine
        - Metoclopramide (prokinetic agent - increased gastric emptying)
            - Has mild anticholinergic and antiserotonin effects
            - Useful in GERD, gastroparesis

        - s/e: tardive dyskinesia, drowsiness, diarrhea,
    - Serotonin antagonists
        - Ondansetron -
            - s/e headaches and constipation/diarrhea
    - Benzodiazepines:
        - No literature to support their routine use for non-specific N/V in the ED
    - Non-pharm agents with limited efficacy
        - Oxygen
        - Ginger
        - Acupressure on P6 at the wrist

There are no perfect agents studied for use in the ED to treat N/V: try to find the underlying cause and use whatever therapy works best.

3) List causes of vomiting specific to age groups: infant, newborn, child, and adolescent

Table 29-4 Etiology of Nausea and Vomiting in Pediatric Age Groups

ETIOLOGIC CATEGORY	NEWBORN	INFANT	CHILD	ADOLESCENT
Infectious	Sepsis, meningitis, UTI, thrush	Pneumonia, otitis media, thrush	Gastroenteritis	Gastroenteritis, URI
Anatomic	Atresia and webs, malrotation, stenosis, meconium ileus, Hirschsprung's disease	Pyloric stenosis, intussusception, Hirschsprung's disease	Bezoars, chronic granulomatous disease	PUD, superior mesenteric syndrome
Gastrointestinal	Reflux, overfeeding, gastric outlet obstruction, volvulus	Reflux, gastritis, milk intolerance	Appendicitis, pancreatic, hepatitis, other food intolerance	Achalasia, hepatitis
Neurologic	Subdural hematoma, hydrocephalus	Subdural hematoma	Neoplasia, migraine, Reye's syndrome, motion sickness, hypertension	Neoplasia, migraine, motion sickness, hypertension
Metabolic	Organic or amino acidemias, urea cycle defects, galactosemia, hypercalcemia, phenylketonuria, kernicterus	Hereditary fructose intolerance, disorders of fatty acid metabolism, uremia, adrenal hyperplasia, kernicterus	Diabetes, vitamin A excess	Diabetes, pregnancy, acute intermittent porphyria
Other	Idiopathic, cardiac failure	Rumination, cardiac failure	Cyclic vomiting syndrome, toxins, food poisoning, Munchausen syndrome by proxy	Psychogenic, anorexia

Adapted from Li HK, Sunku BK: Vomiting and nausea. In Wyllie R, Hyams JS, eds: Pediatric Gastrointestinal and Liver Disease: Pathophysiology, Diagnosis, Management. Philadelphia: WB Saunders; 2005:127-149.
PUD, peptic ulcer disease; *URI*, upper respiratory infection; *UTI*, urinary tract infection.

- Newborn: In the first week of life vomiting may be associated with:
    - Obstructive disease, inborn errors of metabolism, serious infection
- Infant: After the first week of life
    - Consider pyloric stenosis, consider all the infectious causes from head to toe,
    - Consider hirschsprung's disease, intussusception
- Child: After the first month of life:
    - Infections, metabolic diseases, cow's milk intolerance, FTT, abuse, intussusception, hirschsprung
    - Diabetes, appendicitis, gastroenteritis
- Teen:
    - All the common adult causes
    - PUD, PID, diabetes
    - Porphyria, SMA syndrome, achalasia
- ***Feeding problems are a diagnosis of exclusion***

Wisecracks:

1) What are the three main areas providing afferent inputs to the vomiting centre?

<u>The vomiting centre:</u>

- Thought to mediate and coordinate the process - located in the lateral reticular formation of the medulla

1) Afferent pathways: - receive input from
    a. ***GI tract*** *- vagal and sympathetic impulses*
        i. *Pharynx, small bowel, colon, biliary system, peritoneum, genitalia, heart*
    b. *Vestibular system (inner ear)*
    c. *CTZ - chemoreceptor trigger zone*
        i. Floor of the 4th ventricle
        ii. Part of this area is **OUTSIDE** the blood brain barrier
        iii. Activated by:
            1. <u>Hormones</u>
            2. <u>Peptides</u>
            3. <u>Medications</u>
            4. <u>Toxins (</u>opiates, digitalis, chemotherapy agents, salicylate, dopamine)

<u>Leads to Activation of:</u>

2) Efferent pathways:
    a. *Vagus, phrenic, and spinal nerves* → produce the integrated neuromuscular response of nausea/retching/vomiting

2) Name 6 critical causes and 6 complications of vomiting

Critical diagnoses:

1. GI
    a. Boerhaave's syndrome
        i. Multiple CXR findings
            - SC air
            - Pleural effusion
            - Wide mediastinum
            - Pneumomediastinum
    b. Ischemic bowel
    c. GI bleeding (peptic or duodenal ulcer)
    d. Testicular torsion
2. Neurological etiologies
    a. ICH / Ischemic stroke
    b. Meningitis
    c. Raised ICP due to tumour/mass

3. Endocrine
    a. **DKA**
        i. Often triggered by infection/trauma/MI/surgery
    b. Pregnancy
        i. Peaks at 10-16 weeks. 75% of pregnancies. Benign abdominal exam.
        ii. Absence of ketones; normal lytes; <5% weight loss = rules out **hyperemesis gravidarum**
4. Cardiac
    a. **MI**
        i. Diabetics and elders may only have N/V and epigastric pain
5. Toxin
    a. Sepsis
    b. Carbon monoxide poisoning
    c. **Organophosphate overdose**
    d. **Tylenol / Digoxin / ASA OD**

Complications from N/V:

- Hypovolemia
    - Loss of water and sodium chloride in the vomitus
    - Contraction of the extravascular space leads to activation of the RAAS
- Metabolic alkalosis with hypokalemia
    - Due to loss of hydrogen ions in the vomit.
    - Alkalosis further promoted by:
        - Volume contractions
        - Hypokalemia
        - Chloride depletion (in the vomit)
        - Shift of extracellular hydrogen ions into cells
        - Increased aldosterone
    - Hypokalemia
        - Due to loss of potassium in the urine (and the alkalosis leads to large amounts of HCO3- in the distal tubule), AND there is secondary hyperaldosteronism
- Mallory-Weiss tears
    - Due to forceful retching/vomiting. This is a 1-4 cm tear through the mucosa and submucosa. It may occur in the stomach and at the GE junction
    - Mild, self-limited bleeding - very rarely does it result in severe bleeding
- Boerhaave's syndrome
    - Perforation of ***ALL layers*** of the esophagus due to forceful vomiting. The pleura is also torn leading to a **connection into the mediastinum** and thorax.
    - This is a **surgical emergency**
    - 50% mortality rate if no surgery in 24 hrs.
- Aspiration
    - AMS and pulmonary findings - post vomiting