Infantile Hypertrophic Pyloric Stenosis

Professor Alaa El-Suity

"Success is never final and failure is never fatal"

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Epidemiology

- 4 per 1,000 live births and increasing
- More common in Caucasians
- Male:female 4:1 to 6:1
- Increased incidence in B and O blood types
- More common in first born

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Etiology

- Abnormal muscle innervation
- Nitric oxide synthetase defect
- Neonatal hypergastrinemia
- Gastric hyperacidity
- Erythromycin

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Pathology

Hypertrophy of the circular muscle

- *Stops abruptly at the duodenum*

"Duednal fornices like cervix uteri"

empty intestine → collapsed

- *Tapers towards the body*

dilated stomach → gastritis

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Complications

1) Dehydration.
2) Electrolyte imbalance:

Hyponatraemia, hypochloraemia, hypocalcaemia, metabolic alkalosis

3) Death:

- Intercurrent Infection
- Bronchopneumonia
- Gastroenteritis

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Presentation

- 3-6 wks of life
- Increase in frequency, volume and force of nonbilious vomiting
- Hungry vomiter
- Lethargy
- Weight loss
- Symptoms continue despite changes in formula

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Objective Findings

- Physical exam
  - Peristaltic waves
  - RUQ mass
  - Weight loss
- Labs
  - Hypochloremia
  - Hypokalemia
  - Metabolic alkalosis

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Imaging

- Ultrasound
  - Criteria for diagnosis
    - Pyloric muscle thickness $\geq$ 4 mm
    - Diameter $\geq$ 14 mm
    - Channel length $\geq$ 16 mm
- Upper GI series
  - Shoulder sign
  - Double track or string sign
- Upper endoscopy

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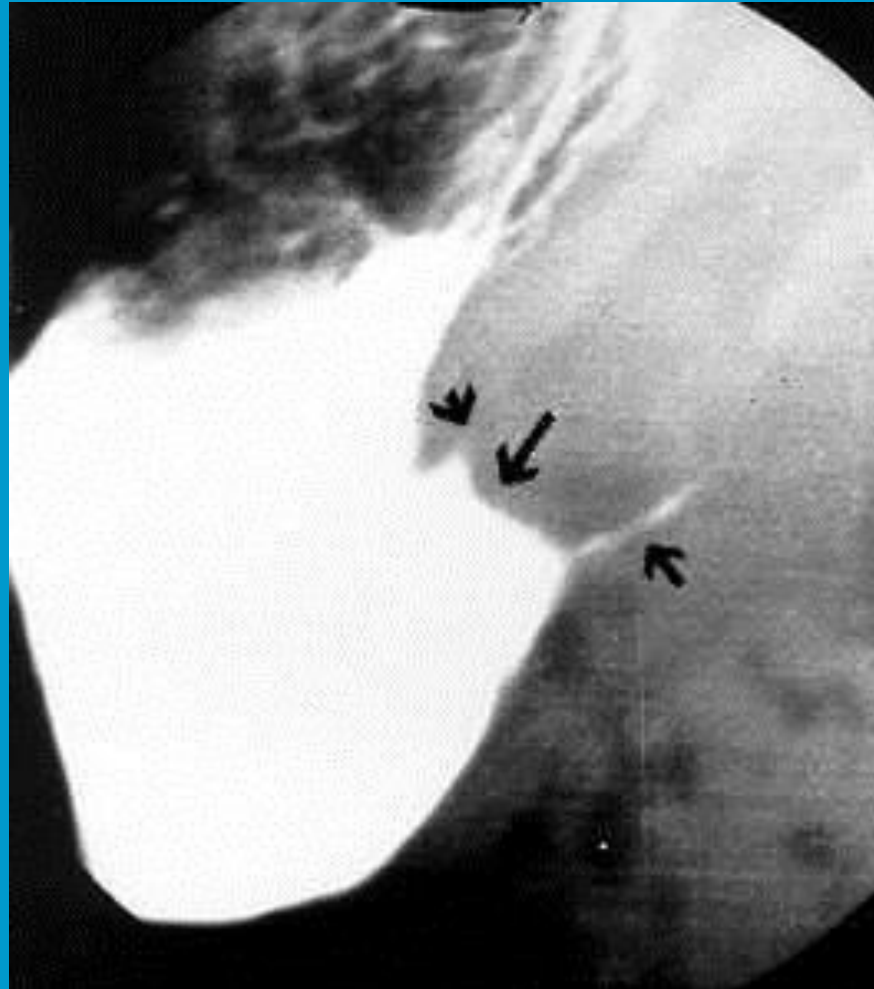
String Sign



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Differential diagnosis

1) Birth trauma.
2) Duodenal atresia.
3) Volvulous neonatorum.
4) Gastroenteritis.

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Treatment

- Conservative management with atropine
- Surgical intervention:

Ramstedt pyloromyotomy

- Pre-op correction of volume depletion and electrolyte

imbalance

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Operative Technique

- Open approach
  - Transverse RUQ incision
  - Supraumbilical curvilinear incision with midline fascial opening
- Laparoscopic
  - Decreased post-op emesis
  - Less post-op pain and analgesic use

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Morbidity and Mortality

- Morbidity 1-5%
  - Complications
    - Incomplete myotomy
    - Wound dehiscence
  - Recurrence 1-3%
- Mortality <0.5%

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Take Home Points

- Typically presents within first 6 wks of life with progressive vomiting, palpable RUQ mass, wt loss, and electrolyte abnormalities.
- Ultrasound is preferred for visualization of hypertrophied pylorus.
- Surgical intervention is standard of care after volume depletion and electrolytes have been corrected.
- Feedings should be restarted 6-12 hrs post-op.

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Gastritis

"Our senses do not deceive us, our judgment does"

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Type A gastritis

- Autoimmune gastritis

"Spared antrum gastritis

- Antiparietal cell antibodies → destruction of parietal cells → achlorohydria & vit B12 deficiency

- G-cell hyperplasia

- precancerous

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Type B gastritis

- Helicobacter pylori infection
- Pangastritis (antrum is affected)
- Precancerous

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Reflux gastritis

- Usually after Bilroth II reconstruction

“ Afferent loop syndrome”

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Erosive gastritis

- NSAIDs → inhibition of cycloxegenase type I receptor enzyme → destruction of gastric mucosa due to ↓ prostaglandin

- COX 2 mediated NSAIDs do not cause erosive gastritis.

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Thank you