Common Pediatric Surgical Problems

Dr. Abbas Az. Alhasani

MBChB FIBMS FACS FRCS Pediatric surgeon

Congenital Abdominal Wall Defects

* From the embryological point of the view, there are 4 folds forming the anterior abdominal wall, cephalic, caudal, and 2 lateral folds, forming a ring around the umbilical cord.
* Failure of the specific fold to develop results in a characteristic abdominal wall defect as follows:
* Failure of the lateral folds causes Omphalocele or umbilical cord hernia.
* Failure of the cephalic fold results in the Pentalogy of Cantrell.
* Failure of the caudal fold results in ectopia vesica.

**Classification**

There are 2 main forms of congenital abdominal wall defects, which are:

1. Omphalocele (synonym: exomphalos)
2. Gastroschisis

**Omphalocele**

* Central abdominal; wall defect at the umbilicus.
* >4cm in diameter.
* Always covered by a thin membrane (of 2 layers, outer amnion and inner peritoneum).
* In addition to the midgut, it may contain liver, spleen, gonads or other structures.

**Umbilical cord hernia**

* Central abdominal; wall defect at the umbilicus.
* <4cm in diameter.
* Always covered by a thin membrane (of 2 layers, outer amnion and inner peritoneum).
* Contains only the part or all of the midgut.
* May contain a single loop of ileum that may be clamped with the umbilical clamp causing intestinal obstruction or enero-cutaneous fistula.

**Gastroschisis**

* A small eccentric defect of <4 cm diameter.
* Just to the right of the normal umbilicus.
* No sac.
* Usually only the midgut is herniated and edematous.

**Management**

* Routine neonatal care, to prevent hypothermia and hypoglycemia in particular and to exclude any associated malformations.

***Omphalocele***

* Non-operative management for those with:
1. Premature neonates with gigantic intact sac.
2. Associated anomalies
* Skin flap closure making a ventral hernia, which needs a later repair.
* Staged repair.
* Primary closure.

***Gastroschisis***

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Infantile Hypertrophic Pyloric Stenosis (I.H.P.S)

* It is the most common cause of surgical vomiting in infancy.
* White > black infants.
* Males > females by 4 times, i.e. the ratio is (4:1).
* 1st born baby has increased incidence.
* Hereditary condition (mother with IHPS has 15% of her offspring affected)
* Less common in Africans and Asians.

# Theories:

1. Congenital mucosal redundancy.
2. Abnormalities of pyloric innervations.
3. Exposure to Erythromycin (in utero or up to 2 weeks post-natal).

# Clinical presentation:

 The age of presentation is (2-8 weeks) in full term infants, but 10% of patients are preterm, in whom the presentation is delayed (42-50 weeks post-conception).

* Projectile non-bilious vomiting.
* The infant is eager to feed after vomiting.
* Dehydration and failure to thrive.
* Sometimes mild jaundice may develop.

On examination:

* Mild to moderate upper abdominal distention, but the abdomen becomes scaphoid after vomiting.
* Visible peristalsis in the upper abdomen (from the left to the right).
* Palpation of the hypertrophied pylorus. (Also called pyloric mass or pyloric olive) is possible in 90% of cases just above and to the right of the umbilicus, this is added by emptying of the stomach by nasogastric tube and examination of the abdomen while breastfeeding (feeding test). This is an important sign because the typical nature of vomiting in a typical age with palpable pyloric mass is enough for the diagnosis so that no other confirmatory investigations are needed.

# Differential diagnosis:

1. Medical conditions causing repeated vomiting, e.g., viral enteritis, meningitis, urinary tract infection …etc.
2. Pylorospasm.
3. GERD (gastroesophageal reflux disease).

# Investigations:

1. Abdominal ultrasound scan (U.S.) is sensitive in 97%, it is going to show the length of the hypertrophied pyloric canal (if > 15mm is diagnostic) and pyloric diameter (if > 4mm is diagnostic).
2. Contrast radiology (Barium meal) is useful if the U/S scan is not diagnostic, it will show:
* String sign, which means elongation and narrowing of the pyloric canal.
* Shouldering sign, this is caused by the impression of the pyloric mass on the stomach.
1. Analysis of serum electrolytes is mandatory in those patients, e.g. Na+, K+ and Cl- …etc.

 Patients with IHPS are liable for electrolyte disturbances that are the major cause of morbidity and mortality. The patients are liable for:

* Hypochloraemia
* Hyponatraemia
* Hypokalaemia
* Metabolic alkalosis
* In severe cases paradoxical aciduria.

## The etiology of the electrolyte disturbances is as follows:

Repeated vomiting >> loss of gastric fluids >> Cl- loss and volume depletion >> renal compensation to conserve fluid and Na+ by exchange of Na+ with K+ through Aldosterone hormone mechanism >> progressive K+ loss >> K+ depletion >> absorption of Na+ in exchange with H+ >> augment the metabolic alkalosis and lastly paradoxical aciduria.

# Management:

* Nasogastric tube (controversial).
* Fluid and electrolytes replacement:

» Mild fluid and electrolytes disturbances >> (0.45% NaCl in 5% dextrose solution).

» Severe fluid and electrolytes disturbances >> 0.9% NaCl (normal saline) bolus 10-20ml/kg body weight followed by (0.9% NaCl in 5% dextrose solution).

KCl added to the intravenous fluid when the patient passes urine.

The patients need 25-50% above the maintenance.

## Operative intervention:

 Ramstedt's operation is the operation of choice in which incision of the serosa and muscular layer of the hypertrophied pyloric canal sparing the mucosa (pyloric seromyotomy).

The operation is not an emergency one, but is best to be done after correction of the fluid and electrolytes, generally 24-48 hours after the diagnosis.

## Postoperative management:

 Naso-gastric tube is not necessary unless mucosal injury had been occurred, in this condition naso-gastric section is used for 24 hours.

 Postoperative feeding can be started as early as 6 hours after the operation with 5% dextrose water solution orally gradually increased as tolerated and conversion to full feeding 12-24 hours after the operation.

# Postoperative complications:

1. Wound infection (1%).
2. Unrecognized perforation of the gastric mucosa.
3. Persistent vomiting (> 48 hours) occurs in about 3% mostly caused by gastric atony.
4. Incomplete seromyotomy.

Pediatric Intussusception

# Introduction

The invagination of one portion of intestine into an adjacent segment.

* It can affect any age and sex.
* It can occur in any part of small and large intestine.
* It typically causes a strangulating bowel obstruction, which can progress to gangrene and perforation.

# Classification

According to the part of GIT affected, it can be:

1. Ileo-colic intussusception (80% of cases)
2. Ileo-ileo-colic
3. Ilio-ileal
4. Colo-colic
5. Others

According to the leading point:

1. Primary intussusception (90%)
* Commonest below 2 years of age.
* 80% ileo-colic form.
* No pathological leading point can be identified.
* In the majority, it’s caused by hyperplasia of the gut lymphoid tissue, which may in turn be caused by viral infection.
1. Secondary intussusception (10%)
* More likely affects children over the age of 2 years.
* There is a pathological leading point can be identified e.g. Meckel’s diverticulum, appendix, polyp, neoplasms (like non-Hodgkin’s lymphoma and carcinoid), and others.

# Presentation

Classically, a previously healthy infant present with

* Colicky abdominal pain.
* Repeated vomiting (milk then bile).

Between episodes, the child initially appears well, later they may pass a

* ‘Redcurrant jelly’ stool (blood and mucus).

On examination, we may see:

* Dehydration
* Abdominal distention
* Palpable sausage-shaped mass in the right upper abdomen.
* PR examination may reveal blood, or the examiner finger can rarely feel the mass.

# Investigations

1. Plain abdominal X ray
* Air-Fluid levels
* Soft tissue opacity
1. Abdominal ultrasound
* Best way to confirm the diagnosis.
1. Contrast (Barium) enema.

# Treatment

* General measures:
* Intravenous fluid resuscitation.
* Broad-spectrum antibiotics.
* Nasogastric tube.
* Reduction of intussusception:
1. Non-operative techniques (>70%):
* Hydrostatic reduction
* Peumostatic reduction

Non-operative reduction is contraindicated if there are signs of peritonitis.

1. Operative reduction techniques:
* Open surgery
* Laparoscopic surgery