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52.1 Introduction

Although conditions such as acute appendicitis and intussusception are relatively common causes of acute abdominal pain, neither is as common as the “*nonspecific abdominal pain*” (*NSAP*),¹ which is seen in children of all ages and all places for which no diagnostic test or specific remedy exists. Clinical acumen and experience are the usual diagnostic tools, with reassurance and perhaps temporary relief from food as bedside remedies.

The term, *Functional Abdominal Pain Disorders (FAPDs)*, is a recent umbrella term that can be applied to most forms of chronic abdominal pain. This often overlaps with other terms including functional dyspepsia, abdominal migraine, Irritable Bowel Syndrome (IBS), and functional constipation and are largely diagnoses of exclusion.

Of course, there are still important causes of chronic abdominal pain that have a more classical surgical pathology such as uro-sepsis, uro-calculi, inflammatory bowel disease, and rarely chronic intestinal volvulus. Additionally, girls may suffer from pelvic inflammatory disease, torped ovaries, and mittelschmerz.²

Some children may have a more classical metabolic pathology (e.g., diabetic ketoacidosis and hypercalcemia) and hematological disorder (e.g., sickle cell anemia). In all these, abdominal pain will feature and, particularly with de novo cases, confusion can occur as to what is going on.

Apart from the first subject, this chapter is therefore devoted largely to minutiae.

¹Alternatively “No sweat abdominal pain!”

²Mittelschmerz (German) for “middle pain,” i.e., pain felt in middle of period due to ovulation.

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52.2 Functional Abdominal Pain Disorders

Functional Abdominal Pain Disorders (FAPDs) can also be called pain-predominant *Functional Gastro-Intestinal Disorders (FGIDs)*. This is probably the most common cause of chronic abdominal pain in children and adolescents.

- Diagnosed when the pain persists for more than two months with no alarming finding and normal clinical examination.
- Pathological (organic) causes should be ruled out and the focus of diagnosis should also include dyspepsia, constipation, diarrhea, dietary trigger factors (like lactose, gluten, FODMAPs³), and anxiety.
- Management plan may also include probiotics, increasing fiber in diet, antispasmodics, and treatment of any associated depression.
- Children with persistent ongoing pain may be referred to a mental health specialist where psychological interventions such as relaxation, distraction, guided imaginary exercises, and cognitive behavioral therapy may help in coping and decreasing anxiety-related functional pain.

52.2.1 Functional Dyspepsia

- Pain or discomfort in the epigastric region, characterized by fullness, bloating, nausea, vomiting, or retching, which may be increased by eating. In these children, an acid-based peptic disease should be ruled out.
- The pathophysiology is not clearly known. Possible causes could be delayed gastric emptying, reduced gastric capacity, gastroduodenal dysmotility, and psychosocial causes.

52.2.2 Abdominal Migraine

This is characterized by recurrent abdominal pain for more than 6 months associated with at least two of the following features: nausea, vomiting, anorexia, headache, photophobia, and pallor.

- Affects 5–15% of children with chronic abdominal pain.
- Age of presentation is usually 2–10 years.
- Is a clinical diagnosis.
- In about 60% family history of migraines may be found.
- Organic causes should be ruled out in these cases.
- Management is empirical.

³FODMAPs standing for “fermentable oligo-, di-, mono-saccharides and polyols.”

- Helpful factors in management are: avoidance of foods high in amines or xanthenes, avoidance of stress, good sleep habits, and good hydration. Non-analgesic migraine medicines may be helpful in prophylaxis.
- Acute cases may be responsive to the treatment of migraine (e.g., triptans—serotonin receptor agonists, sodium valproate).
- Long-term prognosis is good as most children stop having attacks of intermittent abdominal pain by early adolescence.

52.2.3 Irritable Bowel Syndrome

This is characterized by chronic abdominal pain and altered bowel habit, i.e., constipation or diarrhea without any alarming findings. Such children have a lowered rectal pain threshold and disturbed rectal contractile response to meals, whereas in Functional Dyspepsia the pain is mainly felt in the epigastrium. These children also tend to have associated higher anxiety and depression scores.

52.3 Helicobacter Pylori

Helicobacter Pylori

Is a spiral-shaped Gram –ve bacterium with six or so tails (flagella) first identified living in the stomach in the early 1980s by Australian doctors, Barry Marshall, and Robin Warren.

It escapes the acid by burrowing into the mucus layer of the stomach lining and into the interstices of the cellular (epithelial) layer secreting enzymes which breakdown molecules such as urea into the alkaline ammonia which shields itself, but also induces inflammation in the epithelial layer.

- Usually acquired in childhood (~5% overall), becoming increasingly prevalent with age.
- Associated with large families with poor socioeconomic backgrounds.
- ↑↑ prevalence in North Africans.
- May be ↓ in prevalence (at least in the West and Japan).

52.3.1 Clinical Features

- Mostly asymptomatic
- Gastritis, duodenal, and gastric ulceration
- May be associated with malignant change in adults.
 - Gastric (Mucosa Associated Lymphoid Tissue—MALT) lymphoma
 - Gastric carcinoma

52.3.2 Investigation

- Serology (anti-*H. pylori* antibodies)
 - Can be nonspecific and not recommended
- Stool Helicobacter Antigen Test - >90% sensitive
- ¹⁴C Urea breath test (e.g., PYtest™)
 - Most reliable noninvasive test
- Mucosal biopsy and “CLOtest®”—rapid urease test for “Campylobacter-like organism”

52.3.3 Eradication

- Triple therapy
 - Amoxicillin, clarithromycin (or metronidazole), omeprazole for 1 week
- Bismuth preparations
 - More effective but less palatable
 - Unlicensed in children and may be a cause of Reye’s⁴ syndrome

52.4 Yersinia⁵ Infections

Yersinia enterocolitica, *Y. pseudotuberculosis* (N.B. *Y. pestis* was the cause of plague)

- Gram –ve cocco-bacillus
- Prevalent in Scandinavia, and may be related to ingestion of undercooked pork, contaminated milk, etc.
- Tends to affect young (<5 years) children.
- Maybe a specific cause of mesenteric adenitis, acute ileitis, appendicitis, and even enterocolitis.
- Sensitive to third-generation cephalosporins; gentamicin; doxycycline; Septrin®(sulfamethoxazole/trimethoprim). Resistant to co-amoxycylav.

52.5 Pediatric Inflammatory Multisystem Syndrome (PIMS or PIMS-TS)

Emanating from Wuhan in China, *infection with a coronavirus (COVID-19)* quickly became a pandemic with major life-threatening respiratory symptoms principally in adults and particularly the elderly. Children were thought to be relatively protected,

⁴Douglas Reye (1912–1977). Australian pathologist who published cases of encephalopathy in 1963.

⁵Alexandre EJ Yersin (1863–1943). Swiss bacteriologist who discovered the cause of bubonic plague (*Y. pestis*) while working in Hong Kong.

not from the virus necessarily but from any pathological consequence. However, a new syndrome was identified, now called PIMS-TS, initially in the United Kingdom where among a list of possible symptoms, abdominal pain featured. At first, this masqueraded with sufficient severity to warrant surgical exploration as possible appendicitis until it was realized that viral infection was the cause. The cause is believed to be an excessive immunological response (“cytokine storm”) to viral infection.

52.5.1 Key Clinical Features

- Acute abdominal pain, diarrhea, vomiting, fever, muscle pain, lymphadenopathy, ↓ blood pressure (common)
- Coronary artery aneurysms (Kawasaki disease—like), cardiogenic shock.
- Acute renal failure
- ↑incidence in Afro-Caribbean ethnicity

WHO Case Definition: PIMS

Children and adolescents 0–19 years of age with fever >3 days.

AND

Two of the following:

- Rash or bilateral non-purulent conjunctivitis or mucocutaneous inflammation signs (oral, hands, or feet)
- Hypotension or shock
- Features of myocardial dysfunction, pericarditis, valvulitis, or coronary abnormalities (including ECHO findings or elevated Troponin/NT-proBNP)
- Evidence of coagulopathy (by PT, PTT, elevated d-Dimers)
- Acute gastrointestinal problems (diarrhea, vomiting, or abdominal pain)

AND

- Elevated markers of inflammation such as ESR, C-reactive protein, or procalcitonin

AND

- No other obvious microbial cause of inflammation, including bacterial sepsis, staphylococcal, or streptococcal shock syndromes

AND

- Evidence of COVID-19 (RT-PCR, antigen test, or serology positive) or likely contact with COVID-19

52.6 Lead Poisoning (Painter's Colic)

Chronic exposure, usually ingestion, may lead to poisoning. Found in paint (toys), petrol, plumbing (both pipes and solder)—particularly that of previous centuries, and some herbal preparations. Sometimes due to ingestion of contaminated soil.

52.6.1 Clinical Features

- Abdominal colic
- Neurocognitive symptoms
 - Lethargy, hyperactivity, seizures
- ↑ Blood pressure

52.6.2 Investigation

- Blood lead level should be <10 µg/L.

52.7 Porphyrrias⁶

Family of metabolic defects within the synthetic pathway of heme. This leads to tissue accumulation of toxic precursors in the skin, liver, nervous system, etc.

52.7.1 Acute Intermittent Porphyrria (AIP): Example

52.7.1.1 Clinical Features

Most present in adolescence, or have +ve family history.

- Recurrent abdominal pain
 - Typical features in AIP, can be triggered by certain drugs (e.g., phenobarbitone), hormones, infection, and fasting. Can be extreme, with minimal abdominal signs, usually lasts for days.
- Discolored urine—red, brown, purple.
- Autonomic neuropathy—↑ heart rate, ↑BP—peripheral neuropathy.
- Anemia.
- Skin sensitivity—sunlight.
- Psychiatric and neurological symptoms (e.g., seizures).

⁶ Porphyrria (Greek—πορφύρα)—purple. Denoting one of the characteristic features—discoloration of urine.

52.7.1.2 Investigations

- ↑↑ porphyrins in urine, feces, and blood.

Treatment is medical and supportive and involves loading with carbohydrate (IV glucose) and hematin supplementation to suppress heme synthesis.

52.8 Familial Mediterranean Fever

One of the autoinflammatory conditions of childhood, is a hereditary autosomal dominant condition (single gene mutation (*MEFV*) on Ch16) afflicting characteristic groups clustered around the Mediterranean (Armenian, Greeks, Turkey, Sephardi Jews, etc.). There is a deficit in the protein *pyrin*, a key part of the inflammatory cascade.

52.8.1 Clinical Features

- Abdominal pain
 - Childhood onset, often prolonged over a period of days. Probably caused by intrinsic peritonitis
- Other inflammatory membranes
 - For example, pleuritis/pericarditis/tunica vaginalis (acute scrotum)
- Joint pain
- Fever (~25%)

52.8.2 Investigation

- Nothing specific acutely but ↑↑ CRP and ESR.
- ↑ haptoglobin (indicating red cell breakdown).
- Mutational analysis is possible.

Treatment is supportive but colchicine may have a role in ↓ attacks.

Further Reading

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