Chronic/Recurrent Abdominal Pain

or, be careful with those acronyms please

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Definition:

- Pain lasting more than two weeks, so does NOT include:
  - Volvulus
  - Appendicitis
  - Intussusception
  - Typhlitis
  - Pancreatitis
  - Pending or already present perforation
  - Or any other Surgical emergency

Acute Abdomen

- Typically of less than 1 (or two) day's duration
- Typically (but not always) accompanied by bilious vomiting
- May or may not be accompanied by obstipation or distention (a late finding in obstruction)
- May or may not be accompanied by bleeding -also a (too) late finding

Call the Surgeon

Physiology:

- Pain occurs because we have sensors to monitor for aberrant function.
- In the GI tract they are primarily:
  - Acid sensors in the esophagus, stomach and duodenum
  - Stretch receptors everywhere
  - Serosal pain sensors everywhere as well
- They help prevent injury:

Pain is Mother Nature's way of saying "what you're doing isn't working; try something else."

Think about it:

- There are no pain receptors in brain tissue:
  - Once the sabertooth has sunk its fangs into your brain, there's no hope and no selective advantage in having you suffer any more

Proof:

- There are no acid receptors downstream of the duodenum:
  - Beyond that point, the acid is neutralized by pancreatic secretions so none are usually needed
  - That means the inflammation caused by Meckel's diverticula doesn't hurt until it's ready to perforate.

Acid receptors:

- Located in the esophagus, stomach and duodenum
- Purpose:
  - Reduce overeating, but only 'after the fact'
  - Get you to change position to reduce acid exposure
  - Get you to seek ways to dilute or neutralize acid
- Usually includes a sensation of nausea

Stretch receptors:

- Located in ALL hollow tubular viscera
  - Including non enteric "tubes" such as ureters, fallopian tubes, and even large blood vessels
- Purpose:
  - To get you to reduce luminal pressure to prevent rupture
  - Often exacerbated by peristaltic activity
- Is proportional to degree of distention, leading to a "colicky" description
- If the distention and pain are bad enough, this may trigger nausea and vomiting.

Serosal receptors:

- Located on the peritoneal side of the enteric viscera
- Purpose:
  - To halt behavior contributing to impending perforation
  - To trigger help-seeking behavior (e.g. going to the ED)
- Characterized by far more specific localizaton as it also often involves the inside of the abdominal wall (as in the movement of the pain of appendicitis from the periumbilical area to McBurney's point)

Visceral sensors:

- In several (but not all) solid tissue organs:
  - Pancreas (mostly)
  - Liver (less commonly)
  - Kidney (least likely)
- Keyed to tissue damage, but often these sensors are overshadowed by sensors from adjacent organs, or stretch receptors built into the attached tubular drainage systems.

e.g. Pancreatic pain:

- Only partially due to ductal dilatation
- Mostly due to visceral sensors (Tyrosine Kinase Receptors –TrkA) reacting to release of degradaton products (Nerve Growth Factor-NGF) in damaged cells, with many more candidate stimuli and receptors.
- Again the purpose would be to prevent eating and worsening the inflammation
- The pain is predominantly persistent, without much peristaltic variation, and is more identifiable by location (epigastrium radiating to back and shoulder) than characterization.

Other pain:

- Abdominal wall/musculature pain:
  - Brought on or changes with movement
  - Worsens on voluntary guarding using the affected muscle
    (guarding otherwise protects abdominal contents)
- Referred pain:
  - From other sites, mediated by dermatome ennervation
    - e.g. pneumonia can lead to abdominal pain felt anywhere
    - and pancreatitis and perihepatic abscess can lead to shoulder pain by irritation of the phrenic pain receptors.

Abdominal nociception:

- Skin nociceptors have a specific address… abdominal ones have more of a zip code:
  - Epigastric: esophagus. stomach, duodenum and pancreas
  - Periumbilical: small bowel
  - Hypogastric: colon
- Exceptions: biliary tract, pancreas, urinary tract
- Proof: the crampy periumbilical pain of early appendicitis [SB] that converts to a more persistent one as it moves to McBurney's point [and adheres to the abdominal wall]. Then it perforates and the pain disappears – for a few hours [until it is replaced by generalized pain due to peritonitis].

Evaluation:

- Characterization: type often enables guesses as to cause
- Localization: both location where perceived and of the associated tenderness
- Timing: associated exacerbating and alleviating factors
- Physical findings:
  - Masses
  - Tenderness – not always coincident with perception
  - Other (esp. blood)

So to cover the exeptions first:

Biliary tract pain:

- Described most often as colicky as a cycling tubular distention driven by peristalsis against an obstruction
- Usually follows (fatty) meals and is not always persistent (that would imply some degree of cholecystitis)
- Often accompanied by icterus (scleral vs general) and acholic stools, preceded by onset of bilirubinuria

Get an ultrasound exam to look for stones

Pancreatitis:

- Typically persistent due to tissue destruction, but early on can be spasmodic if dominated by ductal distention and obstruction (by an impacted gallstone).
- Radiates to shoulder and back as the inflammation escapes the organ and begins digestion of the surrounding structures
- Localizing the tenderness helps with assessing the areas inflamed (tail, body, head) and possible options.
- Perception is complicated by the accompanying gastric and duodenal stasis, so nausea is not uncommon.

Renal colic:

- Spasmodic if due to stone passage due to intermittent obstruction
- Typically localized to flanks, and unilateral, rotating around the side as the stone passes lower and lower in the urinary tract

Confirmation with blood in urine and imaging
(again, ultrasound is best)

Reproductive tract pain:

Multiple causes lead to multiple manifestations:

- Can be spasmodic if related to tubular structures
  - Fallopian tube obstruction
  - Ectopic pregnancy
  - Uterine irritation
- Can be persistent if due to serosal pain receptors
  - Pelvic inflammatory disease
  - Ovarian cysts

Largely beyond the scope of this lecture

Now for the truly persistent ones:

Dyspepsia:

- With nausea and epigastric localization:
  - While asking how long after meals it arises can help differentiate esophagitis and gastritis and ulcer, the overlap makes this too nonspecific.

Dyspepsia 2:

- I find positional questions more helpful as acid related pain clears as soon as the receptors clear.
- As the stomach is a closed bottle, it can never be completely clear of acid, while the esophagus can clear with sitting up, so short lived pain vs pain lasting an hour or more hints as to the site.

Dyspepsia 3:

- Nocturnal symptoms are VERY helpful to point toward esophageal irritation:
  - Restless sleep (to find a position of comfort)
  - Sleeping prone (to lift the lower esophagus out of the acid)
  - Drooling at night (reflex hypersalivation)

  (and, even more useful)
  - Waking in the middle of the night that requires sitting up to clear the discomfort, or
  - Sleeping through the night only to wake hoarse in the morning (reflux laryngitis)

Dyspepsia 4:

- Tenderness helps as well:
  - Esophageal irritation usually is accompanied by tenderness immediately inferior to the xiphoid process, overlying the base of the esophagus – pressure here often produces some reflux and resultant retrosternal discomfort
  - Gastric irritation is more commonly localized midway between the xiphoid and umbilicus, in the midline, overlying the antrum, as that is the primary acid producing mucosa.
  - Tenderness to the right of the midline, particularly if associated with rebound tenderness is worrisome for duodenal irritation and ulceration.

Dyspepsia 5:

- THE best test however is a whopping big dose of antacid:
  - Complete neutralization of the gastric contents shuts down acid receptors in seconds
  - H2 blockers and PPIs do not stop pain, they only stop acid SECRETION and that not very well. Pain can only be cleared by removing the acid
- A quick two week trial of antacid at all pain episodes and an additional bedtime dose coupled to an early suppertime with no after supper snacking maximizes healing overnight and can clear most dyspepsia completely within 1-2 weeks.

It can however recur.

Pet Peeve: Helicobacter 1

- An organism that not only survives gastric acid but actually USES that chemical gradient as an energy source
- An UNCOMMON cause of dyspepsia in pediatrics
- BUT while it is quite sensitive to use of H2 blockers and PPIs, becoming unrecoverable within a couple of doses,
- It is a survivor and will recur when those medications are discontinued, with a very variable timeline as it grows slowly
- It also has a widely varying antibiotic sensitivity pattern

Pet peeve: Helicobacter 2

- Serologic tests are LESS than worthless with a built-in false positive rate higher than the TRUE positive rate.
- Fecal antigen tests are more reliable, but the manufacturer does not advocate their use as a primary investigative method.
- Biopsy remains the best means of identification but is now MORE vital as we now have the ability to culture the organism and establish its antibiogram

Pet peeve: Helicobacter 3

Why work so hard to make a diagnosis, establish effective antibiotic regimen, and prove a cure?

- Because Helicobacter is causally associated with gastric adenocarcinoma, commonly striking in the 5th decade of life in the face of chronic carcinogen exposure and as early as the 7th decade even with a healthy lifestyle.

It struck close to home: we lost a senior anesthesiologist to the disease from an infection he likely acquired as a child in Japan.

Pet peeve: Helicobacter 4

- The manufacturer of the fecal antigen test recommends a 2 week hiatus in PPI use, and a 4 WEEK wait after H2 blockers. Either is a LONG time in a symptomatic patient.
- Given my 'druthers' I'd rather you started the 2 week trial of antacid and dietary measures and call to set up the referral. I will commit to getting the patients an appointment to be evaluated during that time interval

Spasmodic bowel pain:

- Can arise from infection, non-infectious inflammation, and non-inflammatory motility issues.
- This leads to the case definition of 2 weeks for chronicity: most infections clear in two weeks as it takes 10-14 days for a healthy immune system to produce specific antibodies to clear an infection. The associated inflammation takes only another week to clear on a regular diet that offers enough nutrients for repair.

Spasmodic bowel pain 2:

Presuming infection has been ruled out we are left with

- Inflammatory bowel disease and
- Non-inflammatory bowel… irritation

Inflammatory bowel disease:

- Involves longer lasting spasmodic discomfort that strikes at all hours of the day or night:
- The presence of inflammation releases enough cytokines that sooner (rather than later) triggers nocturnal waking for defecation.
- Long before then, adverse effects on growth are seen, both in weight and halting of linear growth.
- These are typically present if the process has been going on as little as 2-3 months

Inflammatory bowel disease 2:

- Bleeding often occurs within that time interval, but may still be occult.
- Longer term inflammatory markers (ESR) may be more helpful than the short term ones (CRP) in differentiating infection during the early presentation (first week) of bloody diarrhea – if the blood is due to IBD, the process has been active for months, and lab and growth data should already be positive.

Irritable bowel syndrome:

By its very nature includes NO inflammation, so

- The LONGER the symptom of pain has been present without progressing to occult blood in the stool, nocturnal symptoms, or decrements in growth, the more reassuring I can be regarding causality.

Irritable bowel syndrome 2:

Is NOT a 'waste-basket' diagnosis. Numerous consensus conferences have differentiated it into at least 3 diagnostic categories:

- Constipation dominant
- Diarrhea dominant, and
- Mixed type

With specific timelines and diagnostic criteria

Irritable bowel syndrome 3:

- All deal with altered motility, with varied theories as to origin in over and under-stimulation of the myenteric plexus affecting smooth peristalsis.
- This has led to several serotonin-based medications that have gotten into widespread therapeutic use, only to be forcibly halted due to severe adverse side effects related to serotonin's effect on other automatic-muscle function (particularly cardiac)

Irritable bowel syndrome 4:

- For all types, I prefer starting with a good intake of dietary fiber – plain fruit or vegetables.
- They need not be fresh, but can be cooked, canned, frozen, dried, pureed, but NOT juiced – we need that fiber, intact.
- I specify 6 servings daily so that I do not need to specify WHICH fruit or veggies, and can let the patient choose, as long as they take a wide mix.

It's only 2 servings at each meal; readily accomplished.

Irritable bowel syndrome 5:

- For constipation type:
  - The fiber and twice daily commode sitting after meals to take advantage of the gastrocolic reflex to promote more regular rectal emptying usually suffices.
  - If not, there is always miralax as a non-stimulant softener.
  - Avoid laxatives – patients tend to get lax and become dependent on the stimulation.
  - If miralax proves necessary, it will need to be weaned off.

Special case: Encopresis

- If impactions have been a routine occurrence, soiling appears by age 4 as the pelvis enlarges to the point where the anus can no longer maintain continence.
- The rectum is usually so dilated (2 inches and more) that it cannot be expected to empty with dietary fiber alone and a softener like miralax (or mineral oil) will be needed to facilitate slow reduction of the rectal diameter and return of function. Expert help is recommended as this typically takes a few months.

Irritable bowel syndrome 6:

- With diarrheal dominant and mixed types, there is often a component of fermentation occurring. The more flatus is a part of the picture, the more likely the patient is ingesting an unrecognized poorly absorbed carbohydrate that is being passed to the colon bacteria intact and capable of triggering osmotic diarrhea.

Irritable bowel syndrome 7:

- With these patients, I have found the concept of FODMAP to be quite helpful. The acronym is ***Fermentable Oligo-Di-Monosaccharides And Polyols*** and the process is well discussed online.
- We actually LET some carbohydrates through the small bowel (lactose, sorbitol, stacchyose, etc.) but jealously scavenge all sucrose, maltose and the other sugars preferred by pathogens to minimize their growth and encourage the beneficial bacteria.

  The concept is PRE-biotics rather than PRO-biotics.

Irritable bowel syndrome 8:

- The goal is not to remove ALL the oligosaccharides at once, but to identify the worst offenders and either reduce or otherwise manage the substrate (as through use of lactase or Beano) to maintain a healthy diet throughout the investigative process.
- The 2 most common offenders are lactose and sorbitol:
  - We NEED dairy for the calcium (800 mg/d)
  - We DON'T need apple juice

Irritable bowel syndrome 9:

- There ARE those whose GI tracts are simply too sensitive to various stimuli and stresses, leading to too rapid or too slow or too erratic motility and for these Behavioral Health involvement can be quite useful to offer alternate management techniques (imaging, self hypnosis, etc.) to address the worst of the spasming but I find the straightforward mechanical approach covers most quite well.
- If Behavioral Health is needed, it IS best to get them involved as soon as possible, and NOT as an afterthought 'when all else has failed'

Any questions?

References – seminal articles

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