401096 - Out of Hospital Medical Care 2

Week 2 – Gastrointestinal Revision:

Gastrointestinal Tract:

- The continuous muscular digestive tube that winds through the body digesting and absorbing food.
- Functions include mastication, ingestion, digestion, absorption and excretion
- Has four layers:
 - Mucosa lines the lumen
 - Submucosa connective tissue
 - Smooth muscle segmentation and peristalsis
 - Serosa covers the organ in the abdominal region

Peritoneum:

- Serous epithelial membrane that covers the internal walls of the abdominal cavity
 - Parietal covers internal wall
 - Visceral covers the outer surface

Divisions of the GI Tract:

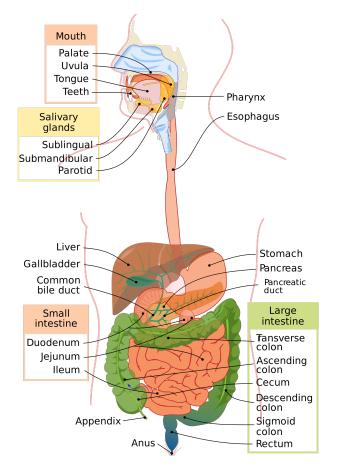
- Foregut: celiac trunk = mouth → first half of duodenum
- Midgut: superior mesenteric artery body = second half of duodenum → proximal half of the large intestine
- Hindgut: inferior mesenteric artery = distal half of the large intestine → rectum

Mouth (Oral Cavity):

- First part of the GIT
- Lined with stratified squamous epithelium
- Includes the salivary glands (parotid, submandibular and sublingual) = contain enzyme salivary amylase to break down starch
- Digestions is mechanical and chemical (via enzymes)

Oesophagus:

- Connects the pharynx and stomach
- Located in the mediastinum anterior to vertebral column and posterior to trachea
- Passes through the diaphragm at an opening called the oesophageal hiatus
- Transports food to stomach via peristalsis
- Gastroesophageal sphincter must open to enter the stomach



Stomach:

- Located under the diaphragm in the left abdominal area
- Stomach layers:
 - Mucosa simple columnar epithelium and has rugae
 - Submucosa contains submucosal plexus controls secretion
 - Smooth muscle contains myenteric plexus coordinates muscles
 - Serosa covering
- Mechanical digestion = food is turned into chyme
- Chemical digestion = HCl and pepsin breaks down proteins

Small Intestine:

- 2 4 metres long
- Nutrient absorption
- Duodenum: secretes alkaline mucus to neutralise acidic chyme
- Jejunum: 2.5m long
- Ileum: 3.5m long and joins the large intestine at the ileocaecal valve
- Mechanical digestion = bile, peristalsis and segmentation
- Chemical digestion = enzymes

Liver Stomach Gallbladder Pylorus **Pancreas** Duodenum Left colic Right colic flexure flexure Transverse Duodenocolon ieiunal junction Ascending Jeiunum Ileocecal Descending junction colon Ileum Cecum Siamoid colon Appendix Rectum Anal canal

Large Intestine:

- Water, electrolytes and vitamins are absorbed
- Caecum → ascending colon → transverse colon → descending colon → sigmoid colon → rectum
- Mechanical digestion = peristaltic waves
- Chemical digestion = bacterial fermentation

Liver:

- Located below the diaphragm in the upper right abdomen
- Is composed of four lobes = right and left, and caudate and quadrate
- Only digestive function is bile production
- Maintains blood glucose levels
- Liver receives blood from the hepatic portal vein (nutrient rich) and the hepatic artery (oxygen-rich)

Gall Bladder:

- Pear shaped sac located underneath the right lobule of the liver
- Stores and concentrates bile (emulsification of large lipid lobules)

Pancreas:

- Mostly retroperitoneal, deep to the greater curvature of the stomach
- Produces pancreatic juice alkaline and neutralises acidic gastric juice
- Alpha cells = glucagon and beta cells = insulin

Week 2 – Gastrointestinal 1 Lecture:

The Gastrointestinal System:

- Overall functions:
 - Ingestion
 - Propulsion
 - Mechanical digestion
 - Absorption
 - Defecation

History Taking in the Patient with GI Complaint:

- Chief complaint
- History of presenting chief complaint
- Specific systems review
 - Abdominal pain
 - Patterns of pain
 - Weight changes and appetite
 - Nausea and vomiting
 - Heart burn and acid regurgitation
 - Dysphagia
 - Diarrhoea
 - Constipation
 - Bleeding
 - Jaundice
- Past medic history
- Social history
- Mental health history

Key Patient History Question:

- How old are you?
- Which came first, pain or vomiting?
 - Pain worse before vomiting suggests surgical disease
- How long have you had the pain?
 - Pain < 48 hours is more likely to be caused by surgical disease
- Have you ever had abdominal surgery?
 - Increases risk of adhesions or obstruction
 - Anything in that region, e.g. C-section
- Is the pain constant or intermittent?
 - Constant more likely to be caused by surgical disease
- Have you ever had this before?
- History of cancer, pancreatitis, kidney failure or inflammatory bowel disease?
 - Suggestive of more serious pathology
- Are you immunocompromised?
 - Consider occult infection of drug-related pancreatitis
 - 7 days after chemotherapy 85% chance of infection
- How much alcohol do you drink?

- Consider pancreatitis, hepatitis or cirrhosis
- Pregnant?
 - Consider ectopic pregnancy
 - Any abdominal pain treat as an ectopic unless it can be ruled out
- Are you taking antibiotics or steroids?
 - May mask infection
 - They may not be working
- Did the pain start centrally and migrate to the right lower quadrant?
 - High specificity for appendicitis
- Do you have a history of vascular or heart disease, HTN or AF?
 - Consider mesenteric ischemia or abdominal aneurysm

<u>Assessing the Patient with Abdominal Pain – The Challenge:</u>

- Atypical presentation
- Degree of pain ≠ severity of disease
- Short patient contact time
- Non-specific physical findings are common
- Absence of abnormal VS ≠ absence of serious disease
- Large number of potential differential diagnosis

Main Aims of Pre-Hospital Care Are:

- Analgesia
- Anti-emetics when required
- Recognition of a deteriorating patient
- Transport to an appropriate hospital
- It is more important to recognise an acute abdomen issue than to identify the exact cause of the pain

Abdominal Pain:

- Onset:
 - Sudden maximal pain at or near onset: hints to vascular event or rupture of hollow organ (e.g. perforated peptic ulcer, ruptured AAA, ruptured ectopic pregnancy, mesenteric infarction)
 - Severe pain in early stages: hints to uretic or biliary colic (other possible conditions are acute pancreatitis, strangulated hernia, volvulus or intussusception)
 - Gradual onset: commonly due to inflammatory processes, such as appendicitis, inflammatory bowel disease, chronic pancreatitis, cystitis etc.
- Provocation (and relieving factors):
 - Peritonitis: Increase pain with movement, deep breathing and coughing
 - Peptic ulcers: Decrease pain after food intake, especially antacids or milk, increase pain with hunger/fasting
 - **Biliary colic:** Increase pain after ingestion of fatty meals
- Quality:
 - Colicky pain: Obstruction of hollow organ
 - Non-colicky pain: Inflammatory or vascular pathology

• Radiation:

May suggest specific conditions

Strength / Severity:

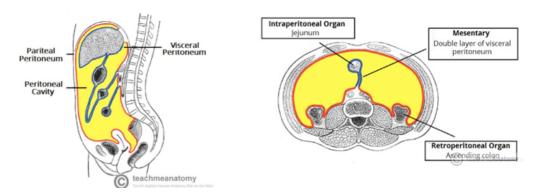
- Degree of pain ≠ severity of disease
- Especially in geriatric population

• Timing:

 Recurrent episodes suggest chronic conditions such as peptic ulcers, biliary colic, renal colic, diverticulitis

<u>Patterns of Abdominal Pain – Visceral Pain:</u>

- Caused by stimulation of nociceptors investing the visceral peritoneum
- Transmitted by visceral afferent nerve fibres in response to stretching or excessive contraction
- Poorly localised
- May be described as "dull, colicky, intermittent and recurrent"
- Visceral pain localises to the abdomen region that correlates with the embryonic segments of the viscera:
 - Foregut structures (stomach, duodenum, liver, biliary tract, pancreas)
 localise to upper abdomen / epigastrium
 - Midgut structures (small bowel, proximal colon, appendix) localise to the periumbilical region
 - Hindgut structures (distal colon, genitourinary tract) localise to the lower abdomen / suprapubic



<u>Patterns of Abdominal Pain – Somatic Pain:</u>

- Caused by local irritation of the parietal peritoneum (innervated by the somatic nerve)
- Pain exacerbated by movement
- Mostly well localised to an area of pathology, but not always (e.g. appendicitis)
- "Sharp, constant and intense"

Patterns of Abdominal Pain - Referred Pain:

- Felt at a distance from the site of origin
- Mechanism not well understood
- Both visceral and somatic pain may manifest as referred pain
- Examples:

Week 5 – BMJ Nephrolithiasis:

Pathophysiology:

- Nephrolithiasis refers to the presence of crystalline stones (calculi) within the urinary system (kidneys and ureter).
- Renal stones are crystalline mineral deposition that form from microscopic crystals in the loop of Henle, distal tubules, or the collecting duct.
- This is usually in response to elevated levels of urinary solutes such as calcium, uric acid, oxalate and sodium, as well as decreased levels of stone inhibitors such as citrate and magnesium.
- Low urinary volume and low or high pH can lead to urine supersaturation with stoneforming salts and subsequent stone formation.
- Once crystals are formed, they either pass out with the urine or become retained in the kidney, where they can grow, and stones can form.
- Pain from urinary calculi can also be due to local inflammatory mediators, oedema, hyperperistalsis, and mucosal irritation

Risk Factors – Strong:

High protein intake	Secondary to increased hyperuricosuria, hypocitraturia and hypercalciuria
High salt intake	Associated with higher urinary sodium and calcium levels and decreased urinary citrate – this promotes calcium salt crystallisation
White ancestry	Highest prevalence
Male sex	3:1
Dehydration	A low urine output can produce a higher concentration of urinary solutes
Obesity	Evidence links obesity with low urine pH and uric acid stones – hypercalciuria
Crystalluria	Increased excretion of calcium oxalate

Risk Factors – Weak:

Occupational exposure to dehydration	Those exposed to high temperatures demonstrate lower urine volumes and pH and higher uric acid levels leading to higher urinary saturation of uric acid and calcium oxalate
Warm climate	Higher incidence in summer
Family history	Twice as likely to form stones
Precipitant medications	Medications containing calcium – lead to higher urinary levels of calcium, uric acid, sodium or oxalate, in turn promoting stone formation

History and Examination Factors:

Acute, severe flank pain	Renal colic pain
Previous episodes	More than 50% will have another episode
Nausea and vomiting	
Urinary frequency / urgency	As stones get lodged can lead to bladder irritation
Haematuria	Present in urinalysis
Testicular pain	As stones pass through the ureter
Fever	Also associated with urinary obstruction
Tachycardia and hypotension	Sepsis
Costovertebral angle tenderness	Acute renal colic

Differential Diagnosis:

Condition	Differentiating Symptoms
Acute appendicitis	Usually presents with RLQ pain and signs of peritonitis
Ectopic pregnancy	Woman of childbearing age present with missed menstrual period, RLQ pain or pelvic pain with some sign of vaginal bleeding.
Ovarian cyst	May present with lower pelvic / abdominal discomfort. Can be a palpable mass on pelvic examination
Diverticular disease	May present with LLQ pain or abdominal pain as opposed to flank pain
Bowel obstruction	Present with abdominal distension, vomiting and constipation
Acute pancreatitis	History of gallstones or alcohol abuse Typically have epigastric pain that radiates to the back as opposed to flank pain.
Peptic ulcer disease	May or may not have history Pain is abrupt, severe in intensity and may be localised to the RLQ and often related to eating meals
Gastroenteritis	Typically, have diffuse abdominal pain and no flank pain Vomiting is prominent and patient may have diarrhoea
AAA	Pain typically presents as sudden onset of abdominal pain, radiating to the back Vital signs will show hypovolemic shock
Pyelonephritis	May present with costovertebral angle tenderness and urinary symptoms of dysuria, frequency. Flank pain may radiate to the back.
Tubo-ovarian cyst	Presents with acute lower abdominal pain and vaginal discharge

Ovarian Torsion	Presents with lower abdominal pain, nausea and vomiting
Mesenteric ischaemia	Present with acute peri-umbilical pain with nausea and vomiting
Constipation	Present with LLQ pain and abdominal distension
Cholecystitis or Biliary colic	Presents with RUQ or epigastric pain, fevers and leukocytosis

Week 5 – Renal and Ureteric Stones Management:

Pain Management:

- 1. Offer a NSAID by any route as first-line treatment for suspected renal colic
 - NSAID's reduce the need for rescue medication
 - Reduce pain and have fewer side effects
- 2. Offer IV paracetamol for suspected renal colic if NSAID's are contraindicated or are not giving sufficient pain relief
- 3. Consider opioids if both NSAID's and IV paracetamol are contraindicated or are not having sufficient pain relief

Week 5 – BMJ UTI's in Men:

Pathophysiology:

- Urinary tract infection is an inflammatory reaction of the urinary tract epithelium in response to pathogenic micro-organisms, most commonly bacteria.
- UTI results from pathogenic organisms gaining access to the urinary tract and not being eliminated effectively. The bacteria ascend from the urethra.
- Complicated UTI's occur as a result of structural or functional abnormality of the urinary tract that impair urine flow, such as:
 - Prostate disorders
 - Calculi within the collecting system or the kidney
 - External drainage devices
 - Urinary diversion surgeries
 - Neurogenic bladder disorders including diabetes
- UTI's in men are often complicated

Presentations:

- Symptoms specifically related to UTI include dysuria, frequency, urgency, suprapubic pain or costovertebral pain.
- In men, fever and urethral discharge may also occur.
- Older patients may present with altered mental status secondary to UTI