

THE ACUTE ABDOMEN

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This lecture was presented by Dr. Bushr Murad.

This file contains his slides and notes. And also **some** Davidson extra notes that the male students told me that they were helpful in the exam. **Black= slides, green= doctor notes, gray= Davidson**

Here is Davidson book link Here is the prof. Hamad book link

Done by: Laila Mathkour

OUTLINE

- Basic Definition and Principles
- Clinical Diagnosis / DDx
 - · Characterizing the pain
 - Other history to elicit
 - Broad differential
 - History & Physical / Labs / Imaging
 - Non-surgical causes of acute abdomen
- Clinical Management
- Decision to Operate
- Atypical presentations
- Take home message



BASIC DEFINITION AND PRINCIPLES

Acute abdomen is a:

- Signs and symptoms of intra-abdominal disease that is *usually* best treated by surgery
- Despite improvements in labs and imaging, <u>history and physical examination</u> remain the <u>mainstays</u> of determining the correct diagnosis !
- Proper evaluation and management requires one to recognize:
 - I. Does this patient need surgery ? Is yes, ask the second question
 - 2. Is it emergent, urgent, or can wait ? If the patient need a surgery, we want to determine that does this patient need to be taken immediately to the OR (emergent), should I optimize him then take him to the OR (urgent), or I'm not sure about the diagnosis maybe surgery is not the best obtion now so let's wait and workup him a little bit more.
- Remember medical "non-surgical" causes of abdominal painthat could mimic surgical abdomen.

Davidson Section: The acute abdomen is a term used to include a spectrum of **surgical, medical and gynecological conditions**, ranging from trivial to life-threatening, which require hospital admission, investigation and treatment. The primary symptom of the condition is abdominal pain. in another word acute abdominal pain = abdominal pain of less than 1 week's duration requiring admission to hospital, which has not been previously investigated or treated. Acute abdominal pain following trauma is usually considered separately. The highest mortality rates are associated with laparotomy for unresectable cancer, ruptured abdominal aortic aneurysm and perforated bowel, especially the colon.

Medical Causes of Acute Abdomen

3 broad categories

Endocrine and Metabolic Causes Uremia Diabetic crisis Addisonian crisis Acute intermittent porphyria الحمى المالطية Hereditary Mediterranean fever

Hematologic Causes Sickle cell crisis Acute leukemia Other blood dyscrasias

Toxins and Drugs Lead poisoning Other heavy metal poisoning Narcotic withdrawal Black widow spider poisoning

This is not an inclusive list, it's classified to three main categories (endocrine and metabolic, hematological, toxin and drugs). Doctor red them all.

Surgical Causes of Acute Abdomen

Doctor read the highlighted ones.

Hemorrhage

Solid organ trauma Leaking or ruptured arterial aneurysm Ruptured ectopic pregnancy Bleeding gastrointestinal diverticulum Arteriovenous malformation of gastrointestinal tract Intestinal ulceration Aortoduodenal fistula after aortic vascular graft Hemorrhagic pancreatitis Mallory-Weiss syndrome Spontaneous rupture of spleen

Infection Appendicitis Cholecystitis Meckel's diverticulitis Hepatic abscess Diverticular abscess Psoas abscess

Perforation

Boerhaave syndrome

Perforated diverticulum

Any part of the GI can perforate, from the esophagus all way to the rectum Perforated gastrointestinal ulcer Perforated gastrointestinal cancer

In other word: obstruction Blockage Adhesion induction small or large bowel obstruction Sigmoid volvulus Cecal volvulus Incarcerated hernias Inflammatory bowel disease Gastrointestinal malignant neoplasm Intussusception

Ischemia

Buerger disease Mesenteric thrombosis or embolism Ovarian torsion Ischemic colitis Testicular torsion Strangulated hernias

CLINICAL DIAGNOSIS

- Characterizing the pain is the key By history taking
 - Onset, duration, location, character and so on...

One way to differentiate the abdominal pain weather it's visceral or parietal pain is by Hx and exam:

- Visceral pain → dull & poorly localized
 - i.e. distension, inflammation or ischemia
- Parietal pain → sharper, better localized
 - Inflammation of parietal peritoneum

Sensory innervation of the viscera

The reason why we are unable to localize the pain either visceral or parietal is gone back to nerve innervation. Think about the segmental innervation to be visceral and the brain will not be able to localize the pain from this pathway. Only when the parietal peritoneum become inflamed, that's when the somatic nerve get stimulated and that's why we can localize the pain



Somatic pain: The parietal peritoneum covers the anterior and posterior abdominal walls, the undersurface of the diaphragm and the pelvic cavity. It develops from the somato-pleural layer of the lateral plate mesoderm, and its nerve supply is therefore derived from somatic nerves supplying the abdominal wall musculature and the skin (T5–L2). The exception to this is the diaphragmatic portion, which is supplied centrally by afferent nerves in the phrenic nerve (C3–C5), and peripherally in the lower six intercostal and subcostal nerves.

The parietal peritoneum is sensitive to mechanical, thermal or chemical stimulation, and cannot be handled, cut or cauterized painlessly. As a result of its innervation, when the parietal peritoneum is irritated, there is reflex contraction of the corresponding segmental area of muscle, causing rigidity of the abdominal wall (guarding) and sometimes hyperesthesia of the overlying skin. When the diaphragmatic portion of the parietal peritoneum is irritated peripherally, there will be pain, tenderness and rigidity in the distribution of the lower spinal nerves, but when it is irritated centrally, pain is referred to the cutaneous distribution of C3, 4 and 5. **Somatic pain is classically described as sharp or knifelike in nature, and is usually well localized to the affected area.**

Visceral pain: The visceral peritoneum forms a partial or complete investment of the intraabdominal viscera. It is derived from the splanchnopleurl layer of the lateral plate mesoderm, and shares its nerve supply with the viscera (i.e.the autonomic nerves). Visceral pain is mediated through the sympathetic branches of the autonomic nervous system, with afferent nerves joining the presacral and splanchnic nerves, which eventually join thoracic (T6–T12) and lumbar (L1–L2) segments of the spinal cord. The visceral peritoneum and the viscera are **insensitive** to mechanical, thermal or chemical stimulation, and can therefore be handled, cut or cauterized painlessly. However, they **are sensitive to tension**, whether due to overdistension or traction on mesenteries, visceral muscle spasm and ischaemia.

Visceral pain is typically described as dull and deep seated. It is usually localized vaguely to the area occupied by the viscus during development, and is referred to the overlying skin of the abdominal wall according to the dermatome level with the sympathetic supply, as mentioned earlier. Therefore, pain arising from the intestine and its outgrowths (the liver, biliary system and pancreas) is usually felt in the midline. Irritation of foregut structures (the lower esophagus to the second part of the duodenum) is usually felt in the epigastric area. Pain from midgut structures (the second part of the duodenum) is dudenum to the splenic flexure) is felt around the umbilicus. Pain from hindgut structures (the splenic flexure to the rectum) is felt in the hypogastrium/ suprapubic area.

Although the division of abdominal pain into visceral and somatic pain is useful, it is important to realise that some pathological conditions will result in a mixed picture. For example, acute appendicitis classically presents with acute abdominal pain that is initially felt in the umbilical area (referred pain) resulting from appendicular obstruction, which gradually localises to the right iliac fossa and becomes sharper in nature as the overlying parietal peritoneum becomes inflamed. These diagrams show further differentiation way. The pain could be diffuse and you can't localize it. **If you are able to localize the pain,** it could be either **epigastric**, **umbilical** or **hypogastric** region. Then, we can have the differentials beside on what are the organs in that region.

UMBILICAL REGION

Early Appendicitis

Mesenteric Adenitis Mesenteric Thrombosis Intestinal Obstruction

Inflammatory Bowel Disease

HYPOGASTRIC REGION

Ovarian Cyst/Torsion

Ectopic Pregnancy

Intestinal Obstruction

Inflammatory Bowel Disease Abdominal Wall Hematoma

Gastroenteritis

Pancreatitis

Aneurysm

Cystitis

Diverticulitia

Appendicitis

Endometriosis

Nephrolithiasis

Prostatism

Salpingitis

Hernia

Hernia

Peptic Ulcer Gastritis Pancreatitis Duodenitis Gastroenteritis Early Appendicitis Mesenteric Adenitis Mesenteric Thrombosis Intestinal Obstruction Inflammatory Bowel Disease

EPIGASTRIC REGION

DIFFUSE Peritonitis Early Appendicitis Pancreatitis Leukemia Sickle Cell Crisis Gastroenteritis Mesenteric Adenitis Mesenteric Thrombosis Intestinal Obstruction Inflammatory Bowel Disease Aneurysm Metabolic Causes Toxic Causes Here we divide the abdomen into 4 quadrant, depending on which quadrant and depending on the organs that found in each quadrant we can come up with the list of differentia's

- Parietal pain → sharper, better localized
- Inflammation of parietal
 peritoneum

RIGHT UPPER QUADRANT Cholecystitis Choledocholithiasis Hepatitis Hepatic Abscess Hepatomegaly from Congestive Heart Failure Peptic Ulcer Pancreatitis **Retrocecal Appendicitis** Pyelonephritis Nephrolithiasis Herpes Zoster Myocardial Ischemia Pericarditis Pneumonia Empyema Gastritis Duodenitis Intestinal Obstruction Inflammatory Bowel Disease

RIGHT LOWER QUADRANT Appendicitis Intestinal Obstruction Inflammatory Bowel Disease Mesenteric Adenitis Diverticulitis Cholecystitis Perforated Ulcer Leaking Aneurysm Abdominal Wall Hematoma Ectopic Pregnancy Ovarian Cyst/Torsion Salpingitis Mittelschmerz Endometriosis Ureteral Calculi Pyelonephritis Nephrolithiasis Seminal Vesiculitis Psoas Abscess Hemia

LEFT UPPER QUADRANT Gastritis Pancreatitis Splenic Enlargement Splenic Rupture Splenic Infarction Splenic Aneurysm Pyelonephritis Nephrolithiasis Herpes Zoster Myocardial Ischemia Pneumonia Empyema Diverticulitis Intestinal Obstruction Inflammatory Bowel Disease LEFT LOWER QUADRANT

Diverticulitis Intestinal Obstruction Inflammatory Bowel Disease Appendicitis Leaking Aneurysm Abdominal Wall Hematoma Ectopic Pregnancy Mittelschmerz Ovarian Cyst/Torsion Salpingitis Endometriosis Ureteral Calculi Pyelonephritis Nephrolithiasis Seminal Vesiculitis Psoas Abscess Hernia



• 83 yo F presented to the ED

The main complain is:

Progressive weakness & functional decline over past 5 days otherwise she is doing well

She presented initially with:

- Initially vague abdominal complaints
- Past Medical History: Arthritis
- P/E generalized tenderness maximum over RUQ

So, how would you work up this patient? And what is the significant of having a past medical history with arthritis? After Hx and Exam, we asses the patient by taking laps and do x-ray looking for obstruction, in general when you examine your patient consider complete Hx, examination, investigations. The fact that she have a history of arthritis tells you that she is taking a lot of NSAIDS



For the investigations, we did an x-ray for this patient and as you can see there is an air under the diaphragm. You can see in the right side where the liver is, you can see radiolucency where usually it should be dark in the x-ray. In the left side it could be mistaken with gas bubble of the stomach, usually the stomach has an air within it and it may show air, but in the right side you can see a significant amount of free air with the diaphragm and it higher up the liver.

So we decided to take this patient to the OR because of the free air under the diaphragm that indicate **perforation**

Upon surgery they found perforated duodenal ulcer .

So in this example we have an elderly lady but she is very sick, from the history we know that she have arthritis and she is using a lot of NSAID for a several months because of her arthritis. Then she presented with generalized acute abdomen, we asked for lap before any imaging then we did an x-ray that confirm the perforation.

She has a 5 days history, since perforation is very acute, so most probably she started to perforate only last day while the rest od days she is probably suffering with acute inflammation and pain but didn't perforate yet.



Clinical Diagnosis

Referred pain:

Radiation is the process whereby pain extends directly from one place to another, while usually remaining present at the site of onset. It is not the same as (referred) pain, which means movement of the pain. Acute appendicitis is a classic example of pain that moves, starting as a vague central (referred) pain and then moving to the right iliac fossa as the adjacent parietal peritoneum becomes inflamed.

We ask the patient do you feel the pain anywhere else \rightarrow this is radiation We ask the patient was the pain in one place and then moved to another place \rightarrow this is referred

- Biliary disease \rightarrow radiate to the right shoulder or back.
- Sub-left daiphram abcess \rightarrow radiate to the left shoulder.
- Above daiphram (lungs) → radiate to the neck/ shoulders. Patient had bad flu & fever and she was complaining of shoulder pain. After x-ray we found that pneumonia and that's why she was having shoulder pain and she required a course of antibiotic.
- Acute onset & unrelenting pain (pain that is very) = bad

Pain which resolve usually is not acutely surgical



OTHER HISTORY

To differentiate between the causes that we mentioned earlier

- Gl symptoms
 - Nausea, emesis (? bilious or bloody)
 - Constipation, obstipation (last BM or flatus) The difference between both, constipation: delay in bowel movement and passing of stool
 - Diarrhea (? bloody)

while obstipation mean <u>non</u> passing of anything, solid stool, liquid or gases at all. BM= Bowl movement

- Change in symptoms with eating? Loss of appetite? Is it better with eating or worse?
- NSAID use (perforated Duodenal Ulcer) Or polypharmacy
- Jaundice, acholic stools, dark urine If present, think of obstructive jaundice

OTHER HISTORY

- Drinking history (pancreas) If yes think of pancreatitis
- Prior surgeries (adhesions → SBO, ?still have gallbladder & appendix) adhesions usually cause small bowel obstruction and it's the most common cause of it
- History of hernias We ask do you have **bulge** some where? If yes and he is presenting now with abdominal distention and obstipation so that maybe the hernia become obstructed
- Urine output (dehydrated) It's important because you want to know is the patient dehydrated or not?
- Constituational Symptoms
 - Fevers/chills Why to have chills in neoblastic setting? Once there is release of pyrogens, this mean the tumor is being significant and causing release of these modulators which cause a chills.
- Sexual/mesnstrual history

THINK BROAD CATEGORIES

When you see a case of acute abdomen think of these serious categories:

- Inflammation
- Obstruction
- Ischemia

All of the above can end up with perforation

- Perforation (any of above can end here)
 - Offended organ becomes distended
 - Lymphatic/venous obstruction due to **1** pressure
 - Arterial pressure exceeded → ischemia
 - Prolonged ischemia → perforation

This is the classic pathway: the organ distended \rightarrow this lead to lymphatic obstruction \rightarrow which lead to increase in pressure \rightarrow if arterial pressure is exceeded \rightarrow there will be an ischemia. Prolonged ischemia \rightarrow will lead perforation. The classic example is strangulation like **strangulated hernia**

Inflammation: No matter what the trigger of the inflammation, the subsequent pathological process is the same. There is reactive hyperemia of the injured tissue as a result of capillary and arteriolar dilatation I exudation of fluid into the tissues as a result of an increase in the permeability of the vascular endothelium; and an increase in filtration pressure. Finally, there is migration of leucocytes from the vessels into the inflamed tissues.

The clinical consequences of the inflammatory process depend in many factors. The underlying condition is the most important: its severity and duration, the organ involved, the patient's age and comorbidity. In general, the patient will complain of abdominal pain and tenderness, which occurs as a result of tissue stretching and distortion, and is due to the release of inflammatory mediators.

On general examination the patient will have pyrexia and have a tachycardia. Examination of the abdomen will reveal tenderness in the affected area, with guarding, rebound and rigidity if the parietal peritoneum is involved. Investigations may reveal a raised

white cell count.

Table 12.4 Injurious agents causing inflammation	
Infective	
 Bacterial Viral Fungal Parasitic 	
Noninfective	
 Chemical Ischaemic Physical trauma Heat Cold Radiation Immune mechanisms 	

Peritonitis: Inflammation of the peritoneum may be classified according to extent (either localized or generalized) and an etiology. In a surgical setting, the most common cause of generalised peritonitis is perforation of an intraabdominal viscus.

Inflammation of the peritoneum results in an increase in its blood supply and local edema formation. There is transudation of fluid into the peritoneal cavity, followed by accumulation of a protein-rich fibrinous exudate. In the normal state, the greater omentum constantly alters its position within the abdominal cavity as a result of intestinal peristalsis and abdominal muscle contraction, but in the presence of inflammation it will adhere to, and surround, the abnormal organ. The fibrinous exudate effectively glues the omentum to the inflamed viscus, walling it off and preventing further spread of the inflammatory process. In addition, the exudate inhibits intestinal peristalsis, resulting in a paralytic ileus, which limits the spread of the inflammation and infection. As a result of the ileus, fluid accumulates within the lumen of the intestine and, along with the formation of intraperitoneal transudate and exudate, this leads to a decrease in the intravascular volume, producing

the clinical features of hypovolaemia.

Clinical features: The clinical features of peritonitis vary considerably but the most common symptom is abdominal pain,

which is constant and often described as sharp. The pain is usually well localized if it is secondary to inflammation of an

intraabdominal viscus and involves the parietal peritoneum, but may spread to involve the whole peritoneal cavity. Primary

peritonitis can present rather more subtly, and as many as 30% of affected individuals may be asymptomatic.

The term 'peritonitis' or 'peritonism detected on clinical examination' is used to describe the collection of signs associated

with inflammation of the parietal peritoneum, and includes tenderness, 'guarding' (voluntary), rigidity (involuntary) and

'rebound' tenderness.

	Table 12.5 Classification of peritonitis
	Generalised peritonitis
	 Primary: infection of the peritoneal fluid without intraabdominal disease Haematogenous spread Lymphatic spread Direct spread: usually associated with continuous ambulatory peritoneal dialysis catheters Ascending infection: from the female genital tract
У	Secondary: inflammation of the peritoneum arising from an intraabdominal source Infectious Noninfectious Blood Ischaemia Bile Chemical Foreign body Perforation
	 Localised peritonitis Usually due to spreading inflammation across the wall of an intraabdominal viscus

Obstruction: impedance of the normal flow of material through a hollow viscus. It may be caused by the presence of a lesion within the lumen of the viscus, an abnormality in its wall, or a lesion outside the viscus causing extrinsic compression.

The smooth muscle in the wall of the obstructed viscus will contract in an effort to overcome the impedance. This reflex contraction produces 'colicky abdominal pain', such as seen in 'ureteric colic'. The exception to this rule is 'biliary colic'. The gallbladder and biliary system have little smooth muscle in their wall and attempts at contraction tend to be more continuous than 'colicky'. Similarly 'renal colic' is a misnomer and it should be referred to as 'renal pain'.

If the obstruction is not overcome, there will be an increase in intraluminal pressure and proximal dilatation. The end result depends on the anatomical location of the obstruction, whether it is partial or complete, and whether the blood supply to the organ is compromised. For example, a ureteric calculus in a single functioning kidney can cause urinary outflow obstruction resulting in a dilatation of the proximal ureter and renal pelvis, and subsequent 'postrenal' renal failure. An obstructed inguinal hernia, on the other hand, will not only produce proximal dilatation of the intestine (usually associated with vomiting) but may also result in ischaemia of the bowel wall, leading to infarction and perforation.

Clinical assessment

The ability to make an accurate assessment by taking a good history and performing an appropriate examination is a vital skill in the

management of the patient with acute abdominal pain. Although an exact diagnosis is often impossible to make after a detailed history and initial assessment, and often relies on further investigations, it is the formulation of an appropriate, safe and effective management plan that is the most important issue. In most cases, it is possible to take a full history and perform a thorough examination, but this is not always so, and occasionally a rapid evaluation followed by immediate resuscitation is required.

Ischemia: An infarct is an area of ischemic necrosis caused either by an occlusion of the arterial supply or the venous drainage in a particular tissue, or by a generalized hypoperfusion in the context of shock. The typical histological feature of infarction is ischemic coagulative necrosis. An inflammatory response begins to develop along the margins of an infarct within a few hours, stimulated by the presence of the necrotic tissue.

The consequences of decreased perfusion of a tissue depend on several factors: the availability of an alternative vascular supply, the rate of development of the hypoperfusion, the vulnerability of the tissue to hypoxia, and the blood oxygen content. In the context of acute abdominal pain, intestinal infarction is the most common cause. Other organs that may infarct include the ovaries, kidneys, testes, liver, spleen and pancreas.

Clinical assessment

In general, the patient will complain of severe abdominal pain and the onset will depend on the nature

of the underlying process. Embolization will result in sudden onset of pain, whereas the onset in

thrombosis is likely to be more gradual. Infarction and ischemia.

Table 12.6 Aetiology of infarction

nature	Occlusive
	Arterial Embolism Thrombosis Extrinsic compression
	Venous Thrombosis Extrinsic compression
	Nonocclusive
	 Shock Hypovolaemia Cardiogenic Sepsis
	Vasoconstrictor drugs

Perforation: Spontaneous perforation of an intraabdominal viscus may be the result of a range of pathological processes. Weakening of the wall of the viscus, which might be associated with a locally advanced malignancy of the bowel, as well as degeneration, inflammation, infection or ischemia, will all predispose to perforation. When the obstructed bowel cannot decompress itself proximally (obstructing colonic tumors with an intact ileo-caecal valve or a volvulus of either the small or large bowel), will predispose to perforation, as will peptic ulceration, acute appendicitis and acute diverticulitis. Other less common causes are inflammatory bowel disease and acute cholecystitis.

Perforation can also be iatrogenic, and may occur during the insertion of a Verres needle at laparoscopy, because of a careless cut or suture placement during surgery, and during the course of an endoscopic procedure.

Clinical assessment

Spontaneous perforation of a viscus normally results in the sudden onset of severe abdominal pain, which is usually well localized to the affected area. The resultant clinical picture depends on the nature of the perforated viscus and the relative sterility and toxicity of the material within the abdominal cavity, in addition to the speed with which the perforation is surrounded and sealed (if at all) by the adjacent structures and omentum. The inevitable peritoneal contamination will lead to either localised or generalised peritonitis, and the associated symptoms and signs, as already discussed. Intestinal content, blood and bile are all irritant to the peritoneum.

	Inflammation vers	sus Obstruction		
Organ	Lesion	Location	Lesion	
Stomach	Gastric Ulcer Duodenal Ulcer		Adhesions Bulges In othe	r word hernias
Biliary Tract	Acute chol'y +/- choledocholithiasis	Small Bowel Obstruction	C rohn's disease Gallstone ileus Intussusception	y in elderly patien
Pancreas	Acute, recurrent, or chronic pancreatitis		Volvulus	
Small Crohn's disease Intestine Meckel's diverticulum		Large Bowel Obstruction	Malignancy Volvulus: cecal or sigmoid	
Large Intestine	Appendicitis Diverticulitis		Diverticulitis	

ISCHEMIA / PERFORATION

Acute mesenteric ischemia

SMA= superior mesenteric artery

- Usually acute occlusion of the SMA from thrombus or embolism → Consider it with patient with atrial fib, the
- Chronic mesenteric ischemia

- → Consider it with patient with atrial fib, the thrombus could go to the brain causing stroke or go down and occlude the SMA which is blood supply to most of GI tract causing acute mesenteric ischemia
- Typically smoker, vasculopathy with severe atherosclerotic vessel disease
- Ischemic colitis Can be caused either by acute or chronic mesenteric ischemia, usually present with bloody diarrhea
- Any inflammation, obstructive, or ischemic process can progress to perforation
- Ruptured abdominal aortic aneurysm or in other word raptured of triple A. this can lead to a biggest perforation. If the patient diagnosed with aneurysm in the past and suddenly it get raptured they will present with back pain and suddenly he is hypotensive. In most cases it is not recognized quickly and we lost the patient.

Gynecological Etiologies Of acute abdomen

Organ	Lesion
Ovary Ovaries can mimic acute abdomen, especially if it's the right ovary. Sometimes we admit a few patients with RLQ pain that we though that she have appendicitis but after the work up and CT scan and US we only find a small amount of free fluid in the pelvis and prominent ovary. This is a normal physiological changes and can be sever enough to cause a sever pain that bring the patient to the emergency.	 Raptured Graafian follicle Torsion of the ovaries. Means ovary twists around the ligaments that hold it in place. This twisting can cut off blood flow to the ovary and fallopian tube. It's requires rapid evaluation and management in order to salvage the ovary. Tubo-ovarian abscess (TOA). It's one of the inflammatory causes
Fallopian Tube	 Ectobic pregnancy Acute salpingitis. Inflammation of the fallopian tube Pyosalpinx
Uterus	 Uterine rapture Especially in pregnancy Endometritis

These are classic signs they have been named after the person that discovered them you need to know the name of the signs (left column), the clinical presentation (middle table) and the diagnosis (last column) There are pictures of the signs net slide

Doctor read the highlighted ones

Aaron sign	Pain or pressure in epigastrium or anterior chest with persistent firm pressure	Acute appendicitis
Rassler sign	Sharp pain created by compressing appendix between abdominal wall and iliacus	Chronic appendicitis
Blumberg sign	Transient abdominal wall rebound tenderness	Peritoneal inflammation
Carnett sign	Loss of abdominal tenderness when abdominal wall muscles are contracted	Intra-abdominal source of abdominal pain
Chandelier sign	Extreme lower abdominal and pelvic pain with movement of cervix	Pelvic inflammatory disease
Charcot sign	Intermittent right upper abdominal pain, jaundice, and fever	Choledocholithiasis
Claybrook sign	Accentuation of breath and cardiac sounds through abdominal wall	Ruptured abdominal viscus
Courvoisier sign	Palpable gallbladder in presence of jaundice	Periampullary tumor
Cruveilhier sign	Varicose veins at umbilicus (caput medusae)	Portal hypertension
Cullen sign	Periumbilical bruising	Hemoperitoneum
Danforth sign	Shoulder pain on inspiration	Hemoperitoneum
Fothergill sign	Abdominal wall mass that does not cross midline and remains palpable when	Rectus muscle hematomas
	rectus is contracted	
Grey Turner sign	Local areas of discoloration around umbilicus and flanks	Acute hemorrhagic pancreatitis
lliopsoas sign	Elevation and extension of leg against resistance create pain	Appendicitis with retrocecal abscess
Kehr sign	Left shoulder pain when supine and pressure placed on left upper abdomen	Hemoperitoneum (especially from splenic origin)
Mannkopf sign	Increased pulse when painful abdomen is palpated	Absent if malingering
Murphy sign	Pain caused by inspiration while applying pressure to right upper abdomen	Acute cholecystitis
Obturator sign	Flexion with external rotation of right thigh while supine creates hypogastric pain	Pelvic abscess or inflammatory mass in pelvis
Ransohoff sign	Yellow discoloration of umbilical region	Ruptured common bile duct
Rovsing sign	Pain at McBurney point when compressing the left lower abdomen	Acute appendicitis
ten Horn sign	Pain caused by gentle traction of right testicle	Acute appendicitis

Extra Slides that shows you the previous signs pictures





Bassler sign





Extra Slides that shows you the previous signs pictures



Cruveilhier sign



Chandelier sign



Cullen sign



Kehr sign



Gray turner sign



Murphy sign

Extra Slides that shows you the previous signs pictures



Ransohoff sign

Rovsing's Sign

Pain elicited in RLQ Suggestive of acute appendicitis

Rovsing sign



Blumberg sign

Labs

After you finished your history and exams here is the tool that will help you reach your diagnosis, we start with laps then imaging.

Test	Reason
CBC w diff If the WBC is very high this may indicate sever sepsis. Also if it's low may indicate that the infection is very sever that caused reduction in WBC production. If you look at WBC and it's normal but you have a left shift (which mean the neutrophils count is higher than the WBC) this indicate acute infection.	- Left shift can be very telling
Basic metabolic panel (BMP) Any situation that cause loss of fluid we need to check the urea and electrolytes to check if there is any metabolic abnormalities	N/V Electrolytes Acidosis Dehydration
Amylase In past we thought that it's related only to the pancreas but we understood that it can be produce from the bile it self. So high amylase can come with ischemic bowl, perforated duodenal ulcer as well as pancreatitis. Lipase enzyme is more specific to pancreas	 Pancreatitis Duodenal ulcer perforation Bowel ischemia

Cont..

Test	Reason
LFT LFT is a wrong terminology but it's been used for a long time. The really liver function is related to bilirubin, ammonia, glucose and so on But routinely we use terminology LFT to every thing related to liver. If you have elevated bilirubin and patient has dark urine and bale stool → obstructed jaundice and what is the cause of obstruction? Is it stone? Is the stone cause inflamed gall bladder (cholecystitis) and compressing the bile duct? Or there is no stone but there is a lesion that cause obstruction and jaundice, so we need to know the cause When do we think about hepatitis? When AST, ALP, ALT Is elevated. In hepatitis you need to do the serology, and ask in the hx about traveling, contact with sick patient. But In emergency we usually think about more seruise infections	- Jaundice - Hepatitis
UA Urea electrolytes are imp, because it's indicate dehydration which is a result of stones	GU-UTIStonesHeamaturia
Beta-hCG Pregnancy test should be performed in all women of childbearing age (14-50 years old) coming to ER before any imaging. Not only is this important if x-rays are to be taken, but it will also raise the possibility of an ectopic pregnancy if positive.	- Ectopic

Imaging

Test	Reason
 KUB Flat & Upright. There is also three views x-ray which is flat, upright and lateral decubitus You want to look for free air and signs of bowl obstruction. Air fluid level is the most common sign of bowl obstruction The high the number = high obstruction The upright chest x-ray (CXR) is the most appropriate first investigation for the detection of free intraperitoneal gas and should be carried out in any patient who might have a perforation. If the condition of the patient prevents an upright being taken, then a left lateral abdominal decubitus film might be helpful. 	 Small bowl obstruction (SBO) Free air Stones
US US: one of the main modality in assessing the genitourinary system, biliary system. US is most commonly used to assess acute abdominal pain. As a general investigation it might reveal small amounts of intraperitoneal fluid in conditions such as perforation and infection, whereas in specific conditions such as acute cholecystitis, biliary obstruction, aortic aneurysms and ovarian cysts it can be diagnostic. Although some studies have reported high levels of sensitivity and specificity in the diagnosis of acute appendicitis, ultrasonography is highly operator dependent and a negative result cannot be relied upon, particularly if the clinical picture suggests otherwise.	 Chol'y Jaundice Gynecological pathology

Imaging

Test		Reason
CT scan (diagnostic accuracy) CT is most modality using in ER. Mostly all patient will get an CT, it could be replaced by MRI in some situation but CT is 95% accurate. When you are not sure but you have a an Initial diagnosis and you can't localized the problem exactly, the CT scan can shows you where is the problem exactly (anatomical localization), but you need to have an initial diagnosis by hx and exam and that your diagnosis is not relies on CT.	-	Anatomical dx Case not straight forward
Diagnostic Laparoscopy It's a microscopic assisting of intra abdomen. Flexible sigmoidoscopy is performed on patients who present with an acute abdomen associated with rectal bleeding and in those patients with large bowel obstruction to evaluate the anorectum. Additional information can be obtained from a colonoscopy. Both flexible sigmoidoscopy and colonoscopy can be therapeutic in the management of sigmoid volvulus and pseudoobstruction. Upper endoscopy is used to investigate patients with acute upper abdominal pain in whom a perforated peptic ulcer has been excluded.		Anatomical dx Case not straight forward

I like this slide ;)

CLINICAL FINDINGS ASSOCIATED WITH SURGICAL DISEASES IN THE SETTING OF ACUTE ABDOMINAL PAIN

Physical Examination and Laboratory Findings	Radiographic Findings
 Abdominal compartment pressures >30 mm Hg Worsening distention after gastric decompression Involuntary guarding or rebound tenderness Gastrointestinal hemorrhage requiring >4 units of blood without stabilization Unexplained systemic sepsis Signs of hypoperfusion (acidosis, pain out of proportion to examination findings, rising liver function test results) 	 Massive dilation of intestine Progressive dilation of stationary loop of intestine (sentinel loop) This is very rare finding but if recognize them it will help you Pneumoperitoneum → abnormal presence of air or other gas in the peritoneal cavity Extravasation of contrast material from bowel lumen → Leaking of the contrast from the Vascular occlusion on angiography Fat stranding or thickened bowel wall with systemic sepsis

Diagnostic Peritoneal Lavage (1000 mL)

- >250 white blood cells per milliliter of aspirate
- >300,000 red blood cells per milliliter of aspirate
- · Bilirubin level higher than plasma level (bile leak) within aspirate
- Presence of particulate matter (stool)
- Creatinine level higher than plasma level in aspirate (urine leak)

 19 year old man with periumbilical pain that shifted to RLQ

This is a typical appendicitis case, s young man patient with the pain in he center that is shifted to RUQ

 On exam febrile, sick and tender RLQ

CT scan

These days having CT is one of the standard care to confirm the diagnosing. Or at least to have an US

 What is the diagnosis? Appendicitis



There is non compressible lumen in the RLQ (arrow) with fast stranding around it. Fat stranding is a common finding on CT of the abdomen, when present, it directs the radiologist's to the site of pathology. It refers to an abnormal increased attenuation in fat.

The Atypical Presentations

This is an atypical presentations that can make your diagnosing harder

- Pregnancy (physiological changes, management concerns) \rightarrow do US

Hormonal changes, gastric parsing, increase in WBC and blood flow, increased pulse rate all are normal physiological changes

- Pediatric (common is common, congenital causes) treatment uasully is conservative → do US
 You cant communicate with them, so think about common classic signs.
 Appendicitis we treated by antibiotic in pediatric only.
 Your main stay modality is US, you don't want to expose them to a radiation early on there life
- The criticallu ill (ICU setting) ightarrow do CT

If the patient was in the ICU intubated then extubated and now presenting with acute abdomen (distension) it will be very

hard to take hx and your examination is not reliable

- Immunocomprimased (Not only HIV) \rightarrow do CT

They are difficult because they do not show the usual or normal response to the information

- Morbis obesity (atyplical, late) \rightarrow D/L

They present with atypical symptoms and the exam is tough and usually they present late

The Atypical Presentations

You can see here the McBurney's going up from it's location in the RLQ. We see a pregnant in third trimester with pain in RUQ this happens because the gravid uterus is growing over time and compress the other organs inside the abdomen



SPECIAL CIRCUMSTANCES

- Situations making diagnosis difficult
 - Stroke or spinal cord injury
 - Influence of drugs or alcohol In the patient you are examining
- Severity of disease can be masked by:
 - Steroids
 - Immunosuppression (i.e. AIDS)
- Threshold to operate must be even lower !

EMERGENCY OR!!!

- Peritonitis
 - Tenderness w/ rebound, involuntary guarding
- "Unstable" (hemodynamically, or septic)
 - Tachycardic, hypotensive, white count
- Intestinal ischemia, including Very imp
 - Strangulation
 - Closed loop obstruction
- Pneumoperitoneum
- Complete or "high grade" obstruction

Diagram that may help you addressing the patient with acute abdomen



These are big faults:

- Failure to thoroughly examine and document findings
- Failure to perform a rectal or vaginal examination when appropriate
- Failure to evaluate for <u>hernias</u>, including the <u>scrotal</u> region
- Failure to conduct a <u>pregnancy test</u> or to consider pregnancy in the diagnosis
- Failure to reassess the patient frequently while developing a differential diagnosis
- Failure to reconsider an established diagnosis when the clinical situation changes
- Failure to <u>recognize immune compromise</u> and to appreciate its <u>masking</u> effect on the historical and examination findings
- Allowing a normal laboratory value to <u>dissuade</u> a diagnosis when there is cause for clinical concern
- Failure to consult colleagues when appropriate
- Failure to take age- and situation-specific diagnoses into consideration
- Failure to make specific and concrete <u>follow-up</u> arrangements when monitoring a clinical situation on an outpatient basis

• <u>Hesitancy</u> to go to the operating room <u>without a firm diagnosis</u> when the clinical situation suggests surgical disease There is a situation when we have to establish the diagnosing in the OR

TAKE HOME POINTS

- Careful history (pain, other GI symptoms)
- Remember DDx in broad categories
- Narrow DDx based on hx, exam, labs, imaging
- Always perform ABCs, Resuscitate before diagnosis
- Don't forget GYN/medical causes, special situations
- · Common things are common in acute abdomen

