his family 6 months ago. Insidious onset of

HPC

shortness of breath with reduced exercise tolerance. Normally sleeps easy with 2 pillows at night but recently has been feeling particularly unwell. His wife commented that his clothes are a lot sweatier in the mornings. Denies chest pain, palpitations, weight loss, loss of appetite. Had tuberculosis as a teenager when in Pakistan but did not have any further episodes.

PMH Rheumatoid arthritis, HTN, T2DM, PE 10 years ago, Heart failure, MI x 3 with CABG 15y ago. **DH**

Carvedilol, Spironolactone, Ramipril, Ranolazine, GTN Spray, Metformin. Prednisolone, Methotrexate. Infliximab infusions started a few months ago.
FH Nil Significant
SH Lives with wife, son, daughter in law and 2 grandchildren. Independent with good mobility. **ICE** "Is it cancer, doctor?"
Dx Tuberculosis
Ix CXR, Sputum Culture x 3 (or Bronchoalveolar lavage if unable) followed by Staging CT.

Mx
Refer to infectious diseases and start Combination Drugs (Rifampicin, Isoniazid, Ethambutol and Pyrinzinamide)

ABDOMINAL HISTORIES
ABDOMINAL PAIN

"WOCC SOCRATES"

"WIPE" – Introduce yourself, whilst gaining consent
- Wash hands
- Introduce self – "Hello, my name is X and I am a medical student"
- Patient details – "Could I ask your full name and age?"
- Explain –"I have been asked to speak with you about what brings you in, would that be alright?"

Open
- I understand you're suffering from pain in your tummy, can you tell me a little bit more about that?
- Clarification
- Consider pain relief – You seem to be in a lot of pain. Have you been offered pain relief?

Site – Where exactly is the pain?

O nset
1. When did you first notice the pain? Did anything happen then?
2. Did the pain come on suddenly or gradually?
3. Does it come and go or is it always there?
4. Has the pain been getting worse?
5. Have you ever had a pain like this before?

Character – What does the pain feel like?
- E.g. Sharp stab, dull ache, colicky/ cyclical pain
Radiation – Does this pain go anywhere else?

A ssociated
- Symptoms
Meals – Is the pain related to meals?
Fatty food – do fatty foods make the pain worse? (Biliary colic)
Jaundice – Have you noticed any yellowing of your skin or eyes? (Cholecystitis, cholangitis) **Periods** – is the pain related to your periods? (Menstrual pain)
- Systems
Cardiorespiratory – Have you had any chest pain? Breathlessness? Cough? ±sputum/ blood? (Pneumonia or ACS can mimic abdominal pain)
Gastrointestinal – How's your appetite? Swallow? Any vomiting (±blood)? How have your bowels been (diarrhoea/ constipation)? Have you noticed any changes in your stool? (mucus/ blood) **Genitourinary** – How are the waterworks? Is the pain related to passing urine? Have you noticed any blood in your urine? Have you been going more frequently? Urgency in going? Have you been incontinent of urine?
Gynaecological – A more personal question. Is there any chance you could be pregnant? When was last period?

Constitutional – Had any weight loss? Any fevers? Any night sweats?

T iming - done **E**xacerbating/ Relieving
- Does anything make the pain better?

E.g. Leaning forward e.g. pericarditis
- Does anything make the pain worse?
E.g. Food (biliary colic), breathing in (pleuritic - pneumonia)

Severity – How would you rate the pain on a scale of 1-10? 1 being not too bad and 10 being the worst pain imaginable.
ICE

- You've given me a lot of information, thank you. I'd like to hear a little about what you think could be going on. Do you have any idea what could be causing the pain?
- Is anything concerning you that you'd particularly like to discuss?
- Is the main thing you seek from this consultation the resolution of the pain? Or reassurance?

PMHx
- Do you have any medical conditions?
- Specifically, I'd like to ask if you've ever been diagnosed with:

Reflux, Gallstones, Kidney stones, IBS, IBD, Atrial fibrillation (DO NOT miss ischaemic colitis as a DDx), Previous procedures/ abdominal instrumentation

DHx
- Are you on any medication at the moment?
- Consider asking about over-the-counter or alternative/ herbal medicine
- Do you have any allergies?

FHx
- Are you aware of any conditions that run in the family?
- Has anyone in the family ever gone through anything similar to what you're going though?

SHx –
- **S**moking/ Alcohol/ Recreational drugs
- **H**ome environment? Support?
- **O**ccupation – impact of the pain on life and on occupation?

Summarise and Thank
- I'd just like to summarise back to you to make sure I haven't missed anything
- Is there anything you'd like to talk about that we haven't quite addressed?
- Thank you for talking to me and I wish you all the best

Investigations
Examination Full abdominal examination +/-DRE, PV exam
Bedside Urine dip, blood glucose, pregnancy test
Bloods
FBC, U&E, LFTs, CRP, beta-HCG, amylase, lipase, lactate (ischaemic bowel), VBG, ketones,
Imaging (Depending on DDx): AXR, erect CXR, USS abdomen, CT abdomen
Special Oesophageal pH, urea breath test, endoscopy, gastrografin follow through.

The Acute Abdomen

Owing to the wide range of conditions that can present with acute abdominal pain, in what is often referred to as the "acute abdomen", it is useful to narrow down your differential diagnosis based on the location of the pain. In addition, its important you can distinguish between medical and surgical cause of abdominal pain. Familiarise yourself with the conditions included in the table below:

Right Hypochondriac Epigastrium Left Hypochondriac

Medical: Hepatitis Pneumonia

Medical: GORD
Peptic Ulcers

Medical: Pneumonia Gastric Ulcer

Surgical: Cholecystitis

Surgical: Pancreatitis Cholecystitis Thoracic AAA

Surgical:
Ruptured spleen
Right Flank Umbilical Left Flank
Medical: Pyelonephritis

Surgical:
Renal Colic
Retrocaecal Appendicitis

Surgical:
AAA
Pancreatitis
Diverticulitis
Early Appendicitis Small Bowel Obstruction

Medical: Pyelonephritis
Surgical: Renal Colic
Right Iliac Fossa Suprapubic Left Iliac Fossa

Medical: UTI
Crohn’s PID

Surgical:
Appendicitis Ureteric Colic Ovarian Torsion Ectopic Pregnancy Strangulated Hernias

Medical:
UTI
Endometriosis Crohn’s and UC Acute Urinary Retention

Surgical:
Urethral Stones Large Bowel Obstruction

Medical: UC
UTI
PID

Surgical:
Diverticulitis Ureteric Colic

Ovarian Torsion Ectopic Pregnancy Strangulated Hernias

Generalised

Surgical Causes: Peritonitis secondary to bowel perforation & ruptures (spleen, aorta, ectopic pregnancy) **Common Medical Causes:** Gastroenteritis, Diabetic Ketoacidosis, Spontaneous Bacterial Peritonitis **Rare Medical Causes:** Henoch–Schönlein Purpura, Acute Intermittent Porphyria and Sickle Cell Crisis

Top Tip! Small bowel and large bowel obstruction have different causes, risk factors, investigation findings and treatment. Make sure you understand the difference

Differential Diagnosis - SURGICAL

Diagnosis	Features In History	Features In Investigations	Management
Cholecystitis			
Appendicitis			
Bowel Obstruction			
Pancreatitis			
AAA			

RUQ pain, especially after fatty meal, associated nausea, may be colicky, jaundice. Likely had similar pains before, selfresolving. RFs: “4 Fs” fat, female, forties, fertile
Murphy’s positive on
examination, guarding.
Raised WCC and CRP, Raised ALP and GGT. USS shows cholecystitis or cholelithiasis Antibiotics (ceftriaxone and Metronidazole) and planned cholecystectomy in 4-6 weeks if stable.

Pain starts in umbilicus and migrates to RLQ, associated diarrhoea and fever. **NB:** consider mesenteric adenitis as DDx if recent viral illness and negative tests.

Abdominal pain associated with constipation, obstipation, abdominal distension, nausea and vomiting. May be ileus after abdominal surgery or obstructing cancer (ask about red flags, B symptoms, previous colonoscopies)

Sudden onset epigastric pain, severe, radiates to back, better leaning forward, nausea and vomiting. Most commonly history of alcohol excess, gall stones, hyperlipidaemia.

History of insidious back/ loin pain which may suddenly have gotten worse with new abdominal pain. May get early satiety, nausea, lower urinary tract symptoms, DVTs from local compression. Risk Factors include IDH, diabetes, family history, smoker.
Very tender over McBurney's point, Rovsing positive – guarding and peritonism may be present. USS shows appendicitis. Raised WCC and CRP, raised amylase (abdominal inflammation)

Abdominal distension, generalised tenderness, absent or "tinkling" bowel sounds. AXR: dilated bowel loops or air under diaphragm in CXR. Gastrografin follow through may show apple core sign in cancer.
Generalised pain, no peritonism, no masses. Pyrexia and hypoxaemia.
Raised amylase and lipase (more sensitive and specific), raised CRP and WCC
Surgical opinion for intervention. IV fluids, antibiotics (ceftriaxone and Metronidazole)

"Drip and suck" – IV fluids, NG tube on free drainage and treat underlying cause of obstruction.

Score prognosis on Glasgow and Ranson's criteria, Admit, NBM and IV fluids (ITU if severe).

Unwell patient, low blood pressure, tachycardia, pulsatile and expansile mid abdominal mass.
USS to confirm. FAST scan for blood in abdomen.
ABCD! Urgent fluid resuscitation (ideally blood) and surgical involvement.

Diverticulitis

LLQ pain and diarrhoea, with fevers and rigors. Usually elderly with a history of constipation. Stool may be mixed with blood. Poor diet.
Tender LLQ, no guarding unless complications
(perforation / abscess). Raised WCC and CRP.
NBM, IV fluids,
ceftriaxone and
Metronidazole. Re
introduce low fibre diet then long term for high fibre diet and education.

Strangulated Hernia

Abdominal pain, nausea and
vomiting (feculent in late
obstruction) associated with
constipation, and abdominal
distension. May have history of lump that usually goes back in but now stuck, tender and red. RF:
Constipation, abdominal surgery, obesity.
Abdominal distension,
generalised tenderness, absent or “tinkling” bowel sounds. AXR shows dilated bowel loops. (Look for perforation: Rigler’s sign or air under diaphragm in erect CXR). Doppler USS: for hernia to evaluate blood flow.
“Drip and suck” – IV fluids, NG tube on free drainage and urgent surgical referral for repair.

Differential Diagnosis - UROLOGICAL
Diagnosis Features In History Features In Investigations Management
Renal Colic
Acute Urinary Retention
Urinary Tract Infection

Sudden severe pain from “loin to groin” on one side, find it hard to get comfortable, haematuria, previous history of kidney stones. Fever and rigors may indicate pyelonephritis.

Gradual onset, progressive, severe suprapubic pain with a history of not passing urine. History of BPH (nocturia, slow stream, frequency, terminal dribbling). May have recently started new drugs (anticholinergics), or had recent surgery.

Suprapubic pain associated with dysuria associated with urinary frequency, fevers, rigors. Urine may appear cloudy / bloody and be odorous.
RF: Female, recent catheterisation, elderly are risk factors
Tender over renal angle.
Blood leucocytes on urine dip. USS KUB shows stone in ureter

Acutely painful suprapubically, palpable bladder, dull to
percussion (mass that you can’t get under).
Bladder scan shows full
bladder.
Stones < 5mm likely pass spontaneously. >5mm likely to need urological intervention.
Tamsulosin can help. Urinary catheter and TWOC at later date after treating underlying cause (e.g. tamsulosin and
finasteride for BPH, change of
medications)

Examination: suprapubic tenderness, pyrexia.
Bedside: urine positive for leucocytes and nitrites. Bloods: raised WCC, CRP
Antibiotics –
trimethoprim 3 days or nitrofurantoin for 5 days.

Differential Diagnosis - GYNAECOLOGICAL
Diagnosis Features In History Features In Investigations Management
Ectopic
Pregnancy
Ovarian Torsion or Cyst Rupture

Pelvic

Inflammatory Disease

Gradually worsening pain in lower right/left quadrant, may have shoulder tip pain, PV bleeding and/or discharge. Last menstrual period > 4 weeks ago and sexually active, no contraceptive used. Red flags – shoulder tip pain, faintness (implies rupture)

Sudden onset right/left lower quadrant pain, constant, may have nausea and vomiting. No change in bowel or bladder habits.
Previous history of ovarian
pathology / PCOS.

Lower abdominal pain, worsening, fevers, feeling unwell. PV
discharge, deep dyspareunia, abnormal bleeding, history of STIs, risky sexual practices (no barrier protection, multiple sexual partners)
Severe lower quadrant
tenderness. Urine and blood. bHCG positive.
Red flags – haemodynamic compromise, PV bleed
FAST scan for fluid in
peritoneum.
USS for intrauterine
pregnancy.
Very tender lower quadrant, may have guarding, no mass. Adnexal tenderness on PV exam.
USS pelvis shows cyst with reduced blood flow or rupture. Tender in lower abdomen, usually no guarding or
peritonism.
PV exam – cervical excitation (perhaps adnexal tenderness), discharge present.
Positive endocervical swabs If compromised:
Immediate
gynaecology consult, admit, IV fluids, cross match blood, NBM and prepare for
surgery.

Analgesia,
gynaecology referral.

Analgesia, prolonged course of antibiotics (triple therapy) and counselling about complications of PID (sterility) and safe sexual practice.

Differential Diagnosis – MEDICAL

Diagnosis	Features In History	Features In Investigations	Management
Diabetic Ketoacidosis	Generalised abdominal pain associated with polyuria, polydipsia, nausea, vomiting, weight loss, lethargy and weakness. May have altered consciousness/ confusion, tiredness. PMH: T1DM with poor control		
Gastritis / GORD	Burning epigastric pain, worse lying down and leaning forward, worse after meals, obesity, may radiate up into chest.		
Peptic Ulcer Disease	Epigastric pain associated with GORD, chronic NSAID / corticosteroid use. Worse before meals (gastric) or after (duodenal). May get vomiting with moderate amounts of fresh red blood or “coffee grounds” in vomit.		
IBD	Generalised abdominal pain associated with frequent, intermittent, chronic, loose stools (may be bloody / have mucous), weight loss, anorexia, lethargy. Extra intestinal: eyes, joint pains, skin rashes, perianal abscess.		
Gastroenteritis	Sudden onset, generalised pain, diarrhoea and vomiting with urgency, sick contacts or close living quarters (halls / nursing home / recent admission), fever, myalgia, lethargy.		
Hepatitis	RUQ pain, fever, jaundice, malaise and return from endemic country for Hepatitis A – faeco oral risk factors (street food, non-filtered water).		

Hepatitis B – travel to endemic areas. Preceded by vomiting and fever.
Look unwell, dehydrated (poor capillary refill, rapid weak pulse, low BP, dry membranes) High blood glucose level, glycosuria, ketonuria.
Acidosis on blood gas
Raised blood ketones

CXR may show hiatus hernia. Urea breath test for *H. pylori.* Endoscopy to look for
Barrett's or hiatus hernia. Admit. Immediate IV fluids and fixed rate insulin infusion with potassium
replacement. Look for trigger (e.g. infection, MI)

Simple antacids, PPI, *H. pylori* eradication. Weight loss.

Examination: epigastric tenderness, blood in vomit. Positive urea breath test.
Ulcer on endoscopy.
H. pylori eradication and PPI therapy. If haematemesis: admit, inpatient endoscopy, may need intervention if actively bleeding.

Generalised abdominal
tenderness, may have RIF mass (ileocecal)
Raised WCC, CRP. Faecal calprotectin raised. AXR may show thumb printing.
Colonoscopy for biopsy (?transmural inflammation) Rehydrate, immunosuppression (prednisolone,
azathioprine,
infliximab etc.
depending on
response)

Raised WCC and CRP may be present.
Positive stool cultures if
parasitic / bacterial.
Usually viral and selfterminating.

Exam: jaundice, dehydration from vomiting.
Bloods: abnormal LFTs, raised bilirubin.
Positive serology for viral hepatitis.

Acute Hep A & B: Self-limiting,
supportive treatment. Chronic Hep B:
Antivirals, monitor for HCC.
Hep C: interferon
therapy in acute and antivirals in chronic.

Spontaneous Bacterial Peritonitis

Severe pain and unwell patient with history of chronic liver disease and ascites. Fevers, encephalopathy, diarrhoea, ileus. Exam: tender ascitic abdomen, may have rebound and
guarding. Signs of chronic liver disease.
Bloods: deranged LFTs
Ascitic tap: raised protein, raised WCC
Admit, empirical
antibiotic therapy after ascitic tap (e.g. IV cefotaxime) until MC&S known.

Marking Criteria ABDOMINAL PAIN
Marks

Awarded Available Washed hands at the start of the station 1
Introduced themselves – Including First name, last name and role 1
Patient details confirmed: Full name, Age/ D.O.B. 1
Explained purpose of consultation 1
Open question about what brings the patient in today + Clarification of any ambiguity 1
Site – Enquires about exact location of abdominal pain 1
Onset (Timeline) – Asks questions to provide a clear understanding of onset and progression
- Onset/ Circumstance
- Sudden vs. gradual3- Fluctuations

- Progression
- Past episodes
Character 1- Obtains an accurate description of the abdominal pain (Ex. Stab, ache, colic, etc.)
Radiation 1- Asks about the pain moving anywhere else

Associated symptoms
Symptoms elicited are relevant and clearly directed at either arriving at a diagnosis or excluding other plausible diagnoses
- Meals
- Fatty food6- Jaundice

- Periods
Systems queried are relevant to the complaint and adequate questions are asked for each symptom
- Cardiorespiratory; Gastrointestinal; Genitourinary; Gynaecological; Constitutional

Timing 0- This has been done
Exacerbation/ Relief
- Clearly asks if the patient has noticed any relieving/ exacerbating factors, providing 1 appropriate examples if prompted (Ex. Food; breathing; sitting forwards)

Severity 1- Subjective quantitative assessment of chest pain severity
Explores **Ideas, Concerns and Expectations**3 Elicits relevant **Past Medical History**

- Reflux; Gallstones; Kidney stones; IBS; IBD; AF; Previous procedures/ abdominal 2 instrumentation
Elicits relevant **Drug History** including **Allergies** 2
Elicits relevant **Family History** 2
Elicits relevant **Social History** – including **S**moking/ alcohol/ recreational drugs; **H**ome2environment and support; **O**ccupation and impact on life

Closes consultation appropriately allowing the patient to ask any questions 1
Presentation: structured, concise 2
Appropriate Differential Diagnosis ± Investigations ± Management Plan 3
Examiner mark – professionalism and rapport 5
Patient mark – professionalism and rapport 5

Consultation Presentation Global marks patient Global marks examiner
Total 35F presents with abdominal pain 1 day of abdominal pain, started in the middle /generalised but now is more on the lower right side of the abdomen, now 8/10, not radiating anywhere, associated with nausea and vomiting, has had 2x loose stools (no blood or mucous), feeling feverish for the last 2 hours, not wanting to eat. No**HPC** relieving factors. Pressing on RIF makes it worse. Was well before this.
No abdominal distension / ill contacts / travel / previous episodes / associated with new food / dysphagia / odynophagia / haematemesis / chance of pregnancy

PMH GORD, rotator cuff injury
DH Omeprazole, paracetamol, combined contraceptive pill. NKDA.
FH Father - Colon cancer
SH Lives with partner who is well. Social smoker 10/week, 6 pints at weekends. No drugs. **ICE** “Please make the pain better!”
Dx Appendicitis
Ix USS abdomen, bloods incl. FBC, CRP, lactate and lipase, erect CXR to

rule out perforation **Mx** Analgesia, nil by mouth, IV fluids, surgical referral.

57F presents with abdominal pain

Abdominal pain started 3 hours ago after dinner (fried chicken and chips), top middle and right of tummy, 7/10 pain, sometimes comes and goes in waves, but is also there all the time, nothing helps the pain. Has had pain before, usually after meals (especially after fish and chips) – on and off for

HPC the last 6 months. Felt a bit sweaty today with nausea, but no fevers. Does think her skin looks more yellow.
No rigors / weight loss ("I wish!") / fevers / lethargy / diarrhoea / vomiting / dysphagia / change in bowel habit / blood in stool / abdominal distension / alcohol excess / previous liver disease

PMH T2DM, obesity (BMI = 38), hysterectomy, osteoarthritis
DH Metformin, gliclazide, paracetamol, codeine. NKDA
FH Mother – diabetes, hysterectomy. Father – MI.
SH Lives alone. Works as receptionist. Smokes 10/day, 25 years. One glass of wine a day. **ICE** "Is it my liver, because of all the wine?"
Dx Biliary colic on a background of cholecystitis
Ix Bloods incl. LFTs, CRP, FBC. USS abdomen, ?MRCP
Mx Analgesia, antibiotics (ceftriaxone and metronidazole), nil by mouth, IV fluids, surgical referral

23M presents with abdominal pain
Abdominal pain for the last 6 hours, generalised in abdomen, worsening steadily, now 7/10 pain. Has had nausea and vomiting (2 times, no blood, no bile), no diarrhoea. Feeling very unwell, tired,

HPC drowsy, reduced appetite. Has had a chest infection over the last few days – productive cough, fevers, and lethargy. Poor appetite - not eating and drinking much. Feels very thirsty, but also passing lots of urine.

PMH T1DM since age 9, asthma
DH Insulin (novomix BD) – has been taking it if he eats, salbutamol. Allergic to penicillin. **FH** Father and sister- diabetes
SH Works as mechanic. Lives alone. Smoker, 15/day for 5 years. Social

alcohol – 5 pints at weekends. **ICE** “I was thinking it might be appendicitis, do I need surgery?”
Dx Diabetic Ketoacidosis
Ix Blood glucose, ketones and gas to look at pH, urinalysis, bloods incl. FBC, U&E

Mx
Aggressive IV rehydration, fixed rate insulin, potassium replacement, regular blood glucose and neuro observations, treat underlying cause (intercurrent infection) – HDU care ideally.

45 year old male with abdominal pain and generally unwell
Patient reports 1 week history of chills, nausea and vomiting. Reports fevers and night sweats. Reports abdominal pain and loss of appetite. Patient does not report any dizziness or SOB. No chest pain. No **HPC** cough or colds. Patient reports some dysuria. Patient describes worsening abdominal distension and weight gain. Also reports some leg swelling over last two months.
PMH Hepatitis B
DH NKDA, Tenofovir
FHx Nil relevant
SH 50 units alcohol per week, current smoker. Ex-IVDU.
ICE “Doctor, this pain is really worrying me. I'm afraid my Tenofovir isn't working anymore.”
Dx Spontaneous Bacterial Peritonitis
Ix
FBC, U&E, LFT, CRP, blood cultures, aspiration and culture of ascitic fluid, USS liver, vitamin B and folate
Mx
Antibiotics, consider ascitic tab in case large amount of ascites present, alcohol and smoking cessation advice, refer to community alcohol team

25 year old female with abdominal pain
Patient presents with gradual onset abdominal pain. Pain limited to right iliac fossa but radiating to mid line and epigastric region. Patient says pain started 1 week ago and has gradually been getting **HPC** worse. She describes no distension in her abdomen. Patient reports no nausea or vomiting. Some diarrhoea and dysuria. No headaches, no dizziness, no palpitations. Patient does not report any fevers.
PMH Chlamydia infection 2 years ago, treated with antibiotics. Not on

contraception.
DH NKDA, nil regular
FH Nil relevant
SH Long term boy friend, drinks 20 units of alcohol per week. Non-smoker.
ICE “I can't handle this pain anymore doctor. Can’t you please give me some pain relief?”
Dx Ectopic pregnancy
Ix Pregnancy test, USS pelvis, FBC, U&E, LFT, CRP, β-hCG
Treatment choice depends on urgency of treatment. Methotrexate or surgical removal of pregnancy**Mx** and likely associated Fallopian tube.

Top Tip! Remember that many of the presentations above can lead to organ rupture (appendix, colon, aorta etc.) which can then lead to signs of peritonism: rigid abdomen, pain exacerbated by any movement. Similarly bowel obstruction due to cancer, volvulus, adhesions or strangulated hernias may lead to intestinal perforation which presents in a similar manner.

Signs of peritonism on examination: guarding, rigidity, rebound tenderness. These patients are a surgical emergency and require immediate surgical review and potentially prepping for urgent surgery.

DYSPHAGIA

GENERAL FRAMEWORK

“WIPE” – Introduce yourself, whilst gaining consent
- Wash hands
- Introduce self – “Hello, my name is X and I am a medical student”
- Patient details – “Could I ask your full name and age?”
- Explain –“I have been asked to speak with you about what brings you in, would that be alright?”

Open
- I understand you’ve had problems with your swallowing, would you mind telling me more about that?
- **Clarify** – Before you tell me some more, it’s important for me just to ask whether you struggle with swallowing

3

I have a patient with abdominal pain. How do I determine the cause?

Shobha W. Stack, PhD, MD

CHIEF COMPLAINT

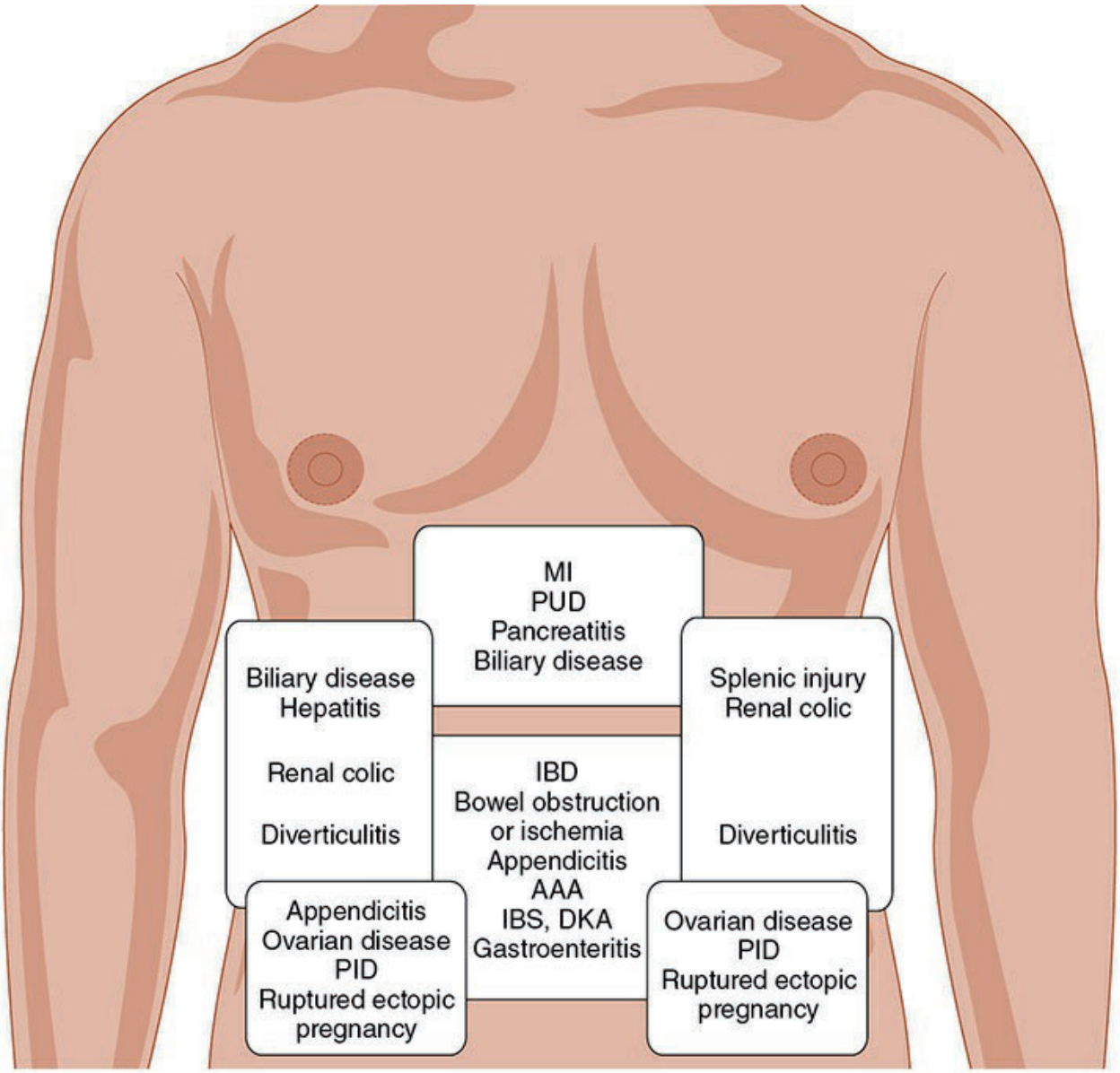
PATIENT 

Mr. C is a 22-year-old man who complains of diffuse abdominal pain.

What is the differential diagnosis of abdominal pain? How would you frame the differential?

CONSTRUCTING A DIFFERENTIAL DIAGNOSIS

Abdominal pain is the most common cause for hospital admission in the United States. Diagnoses range from benign entities (eg, irritable bowel syndrome [IBS]) to life-threatening diseases (eg, ruptured abdominal aortic aneurysms [AAAs]). The first pivotal step in diagnosing abdominal pain is to identify the **location** of the pain. The differential diagnosis can then be limited to a subset of conditions that cause pain in that particular quadrant of the abdomen (Figure 3-1).



AAA, abdominal aortic aneurysm; DKA, diabetic ketoacidosis; IBD, inflammatory bowel disease; IBS, irritable bowel syndrome; MI, myocardial infarction; PID, pelvic inflammatory disease; PUD, peptic ulcer disease.

Figure 3-1. The differential diagnosis of abdominal pain by location.

Several other pivotal points can help narrow the differential diagnosis including (1) the time course of the pain, (2) peritoneal findings on exam, (3) unexplained hypotension, and (4) abdominal distention. Each of these is reviewed below.

The time course of the pain is a pivotal feature. Some diseases present subacutely/chronically over weeks to months or years (eg, IBS) whereas others present acutely, within hours to days of onset (eg, appendicitis). In patients with their first episode of acute severe abdominal pain, a variety of life-threatening, must not miss diagnoses must be considered (eg, AAA). Many of these diseases that cause acute abdominal pain cannot recur because patients are either treated or

die of complications (eg, AAA, acute appendicitis, splenic rupture.) Since prior episodes are incompatible with many of these diagnoses, a history of such prior episodes narrows the differential diagnosis. Therefore, the differential diagnosis of abdominal pain can be organized based on whether patients are presenting with their (1) first episode of acute abdominal pain, (2) a recurrent episode of acute abdominal pain, or (3) chronic/subacute abdominal pain. Table 3-1 outlines the typical time course associated with different diseases causing abdominal pain. See Table 3-2 for a summary of abdominal pain organized by location, time course, and clinical clues.

Table 3-1. Differential diagnoses in abdominal pain organized by time course.

Acute Abdominal Pain		Subacute/Chronic Abdominal Pain
First episode	Recurrent episode	
AAA	Biliary disease	Chronic mesenteric ischemia
Acute mesenteric ischemia	Diverticulitis	IBD
Appendicitis	DKA	IBS
Biliary disease	Nephrolithiasis	Hepatitis
Diverticulitis	Pancreatitis	PUD
DKA	PID	
Ectopic pregnancy	Small or large bowel obstruction	
Gastroenteritis		
Ischemic colitis		
Myocardial infarction		
Ovarian torsion		
Nephrolithiasis		
Pancreatitis		
Peritonitis (from ruptured PUD, diverticulitis, etc)		
PID		
Small or large bowel obstruction		
Splenic rupture		

AAA, abdominal aortic aneurysm; DKA, diabetic ketoacidosis; IBD, inflammatory bowel disease; IBS, irritable bowel syndrome; PID, pelvic inflammatory disease; PUD, peptic ulcer disease.

Table 3-2. Summary table of abdominal pain by location, time course, and clinical clues.

Location	Differential Diagnosis	Time course: Acute 1 X	Time course: Acute > 1 X	Time course: Chronic	Unexplained Hypotension	Clinical Clues
Right upper quadrant	Biliary disease	✓	✓		✓[1]	Postprandial or nocturnal pain Dark urine
	Hepatitis	✓		✓		Alcohol use Injection drug use Jaundice Toxin/drug ingestion (acetaminophen)
	Pancreatitis	✓	✓	✓	✓[1]	Alcohol use Gallstones
	Renal colic: (Usually flank pain)	✓	✓			Hematuria (usually microscopic) Severe pain
Left upper quadrant	Renal colic: (Usually flank pain)	✓	✓			Hematuria (usually microscopic) Severe pain
	Splenic infarct or rupture	✓			✓	Endocarditis Trauma Shoulder pain
Epigastrium	Biliary disease	✓	✓		✓[1]	Postprandial or nocturnal pain Dark urine
	Pancreatitis	✓	✓	✓	✓[1]	Worse supine History of alcohol abuse or gallstones
	Peptic ulcer			✓	✓[2]	Melena History of NSAID use
Diffuse periumbilical	AAA	✓			✓	Smoking Male sex Hypotension, syncope or pulsatile abdominal mass
	Appendicitis	✓			✓[1]	Migration and progression
	Acute mesenteric ischemia	✓			✓[1]	History of AF Heart failure Valvular heart disease Catheterization Pain out of proportion to exam
	Bowel obstruction	✓				Inability to pass stool or flatus Prior surgery
	Chronic mesenteric ischemia			✓		PVD or CVD Pain brought on by food Food fear Weight loss
	Gastroenteritis	✓				Diarrhea Travel history
	Inflammatory bowel disease			✓		Family history Hematochezia Weight loss
	Irritable bowel syndrome			✓		Intermittent diarrhea or constipation Pain relieved by defecation
	Splenic rupture	✓			✓	Trauma
Right lower quadrant	Appendicitis				✓[1]	Migration and progression
	Ectopic pregnancy	✓			✓	Sexually active premenopausal woman
	Ovarian torsion	✓				
	PID	✓	✓		✓[1]	Sexually active woman Vaginal discharge Cervical motion tenderness
Left lower quadrant	Diverticulitis	✓	✓		✓[1]	Diarrhea Fever
	Ectopic pregnancy	✓			✓	Sexually active premenopausal woman
	Ovarian torsion	✓				
	PID	✓	✓		✓[1]	Sexually active woman Vaginal discharge Cervical motion tenderness

[1]If associated with sepsis.
[2]If associated with hemorrhage.
AAA, abdominal aortic aneurysm; AF, atrial fibrillation; CVD, cardiovascular disease; NSAIDs, nonsteroidal anti-inflammatory drugs; PID, pelvic inflammatory disease; PVD, peripheral vascular disease.

Peritoneal findings of rebound tenderness, rigidity, and guarding are pivotal features and suggest an intra-abdominal catastrophe. Typical causes include AAA, bowel infarction (due to

bowel obstruction or acute mesenteric ischemia), bowel perforation (due to appendicitis, peptic ulcer disease [PUD], diverticulitis), pancreatitis, or pelvic inflammatory disease (PID).

Unexplained hypotension is yet another potential pivotal clue. While many patients with abdominal pain experience hypotension due to dehydration from nausea, vomiting, or poor oral intake, some patients with abdominal pain present with *unexplained* hypotension. Unexplained hypotension can suggest sepsis, retroperitoneal hemorrhage, or other diseases. Table 3-3 lists diseases associated with abdominal pain and unexplained hypotension.

Table 3-3. Differential diagnoses in patients with abdominal pain and unexplained hypotension.

Mechanism of Hypotension	Differential Diagnosis
Intra-abdominal hemorrhage	Abdominal aortic aneurysm Ruptured ectopic pregnancy Splenic rupture
Sepsis	Acute mesenteric ischemia Appendicitis Ascending cholangitis Bowel obstruction (with infarction) Cholecystitis Diverticulitis Inflammatory bowel disease Ischemic colitis Nephrolithiasis (if accompanied by ascending infection) Pancreatitis Pelvic inflammatory disease Peptic ulcer disease (with perforation) Spontaneous bacterial peritonitis
Other	Adrenal insufficiency Diabetic ketoacidosis Myocardial infarction

Orthostatic vital signs should be taken in patients with abdominal pain. They may provide invaluable diagnostic and therapeutic information.

The final pivotal finding is significant abdominal distention, which may develop from excess air or fluid in the abdomen. Excess air may occur with bowel obstruction or bowel perforation (free air). Excess fluid may be seen in patients with ascites or hemorrhage. Percussion and shifting dullness can usually distinguish excess air from fluid in such patients. Table 3-4 lists the diagnostic considerations in patients with abdominal distention.

Table 3-4. Differential diagnosis in patients with abdominal pain and distention.

Air		Fluid	
Free air	**Luminal air**	**Ascites**	**Hemorrhage**
Appendicitis Bowel infarction (SBO, AMI) Diverticulitis PUD (with perforation)	LBO SBO	Pancreatitis SBP	AAA Ruptured ectopic pregnancy Ruptured spleen

AAA, abdominal aortic aneurysm; AMI, acute mesenteric ischemia; LBO, large bowel obstruction; PUD, peptic ulcer disease; SBO, small bowel obstruction; SBP, spontaneous bacterial peritonitis.

Other important historical points include factors that make the pain better or worse (eg, eating), its' quality, radiation of the pain, and associated symptoms (nausea, vomiting, anorexia, inability to pass stool and flatus, melena, hematochezia, change in color of the urine or stool, jaundice, fever, chills, weight loss, altered bowel habits, orthostatic symptoms, urinary symptoms) or prior abdominal surgeries (increasing the risk of small bowel obstruction [SBO]). Pulmonary symptoms or a cardiac history can be clues to pneumonia or myocardial infarction presenting as abdominal pain. In women, sexual and menstrual histories are important. The patient should be asked about alcohol consumption as well as prescription and over-the-counter medications and supplements.

A few final points about the physical exam are worth emphasizing. First, vital signs are just that, vital. Hypotension, fever, tachypnea, and tachycardia are critical clinical clues that must not be overlooked. The HEENT exam should look for pallor or icterus. Jaundice suggests either hepatitis or biliary disease. Careful heart and lung exams can suggest pneumonia or other extra-abdominal causes of abdominal pain.

The physical exam of a patient with abdominal pain includes more than just the abdominal exam.

Of course, the abdominal exam is key. Inspection assesses for distention as noted above. Auscultation evaluates whether bowel sounds are present. Absent bowel sounds may suggest an intra-abdominal catastrophe; high-pitched tinkling sounds and rushes suggest an intestinal obstruction. Palpation should be performed last. *It is useful to distract the patient by continuing to talk with him or her during abdominal palpation*. This allows the examiner to get a better appreciation of the location and severity of maximal tenderness. The clinician should palpate the painful area last. The rectal exam should be performed, and the stool tested for occult blood. Finally, the pelvic exam should be performed in adult women and the testicular exam in men.

Mr. C felt well until the onset of pain several hours ago. He reports that the pain is a pressure-like sensation in the mid/upper abdomen, which is not particularly severe. He had never had this symptom before. He reports no fever, nausea, vomiting, or diarrhea. His appetite is diminished, and he has not had a bowel movement since the onset of pain. He reports no history of urinary symptoms such as frequency, dysuria, or hematuria. His past medical history is unremarkable. On physical exam, his vital signs are temperature, 37.0°C; RR, 16 breaths per minute; BP, 110/72 mm Hg; and pulse, 85 bpm. His HEENT, cardiac and pulmonary exams are normal. Abdominal exam reveals a flat abdomen with hypoactive but positive bowel sounds. He has no rebound or guarding; although he has some mild diffuse tenderness, he has no focal or marked tenderness. There is no hepatosplenomegaly. Rectal exam is nontender, and stool is guaiac negative.

At this point, what is the leading hypothesis, what are the active alternatives, and is there a must not miss diagnosis? Given this differential diagnosis, what tests should be ordered?

RANKING THE DIFFERENTIAL DIAGNOSIS

The patient's history is not particularly suggestive of any diagnosis. The first pivotal point determines the location of the pain. Mr. C's pain is in the mid/upper abdomen, which limits the differential diagnosis. Common causes of mid/upper abdominal pain include appendicitis, IBS, PUD, pancreatitis, inflammatory bowel disease (IBD), SBO, large bowel obstruction, acute ischemia, AAA, myocardial infarction, diabetic ketoacidosis, and gastroenteritis (Figure 3-1). Several of these diagnoses are very unlikely and need not be considered further. AAA and myocardial infarction would be exceptionally rare in this age group and gastroenteritis is very unlikely in the absence of either vomiting or diarrhea. Acute ischemia is unlikely given the absence of pain out of proportion to exam. The lack of a history of diabetes would make diabetic

ketoacidosis unlikely unless this was the initial presentation. A simple blood sugar could help exclude this diagnosis. Other pivotal points in patients with abdominal pain include its time course (see Table 3-1), and if present, unexplained hypotension or abdominal distention (see Tables 3-3 and 3-4). The patient reports that this is an acute episode that has not occurred previously. This makes IBD and IBS very unlikely, focusing attention on the remaining possibilities of appendicitis, PUD, pancreatitis, and bowel obstruction. Appendicitis is the leading hypothesis as it is common and amenable to surgical cure (Table 3-5). He has neither unexplained hypotension nor distention to help focus the differential diagnosis further.

Table 3-5. Diagnostic hypotheses for Mr. C.

Diagnostic Hypothesis	Demographics, Risk Factors, Symptoms and Signs	Important Tests
Leading Hypothesis		
Appendicitis	Migration of pain from periumbilical region to right lower quadrant	Clinical exam CT scan
Active Alternatives-Most Common		
Peptic ulcer	NSAID use *Helicobacter pylori* infection Melena Pain relieved by eating	EGD Stool antigen for *H pylori*
Pancreatitis	Alcohol abuse Gallstones	Serum lipase
Active Alternatives-Must Not Miss		
Early bowel obstruction	Prior abdominal surgery Inability to pass stool or flatus Nausea, vomiting	Abdominal radiographs, CT scan Small bowel study Barium enema

EGD, esophagogastroduodenoscopy; NSAID, nonsteroidal anti-inflammatory drug.

Reviewing Table 3-5 for clues (risk factors and associated symptoms), Mr. C reports no history of nonsteroidal anti-inflammatory drug (NSAID), aspirin, or alcohol ingestion. He has no known gallstones and no prior history of abdominal surgery. He reports that he is passing flatus and denies vomiting.

Is the clinical information sufficient to make a diagnosis? If not, what other information do you need?

Leading Hypothesis: Appendicitis

Textbook Presentation

The classic presentation of appendicitis is abdominal pain that is initially diffuse and then intensifies and migrates toward the right lower quadrant (RLQ) to McBurney point (1.5–2 inches from the anterior superior iliac crest toward umbilicus). Patients often complain of bloating and anorexia.

Disease Highlights

A. Appendicitis is one of the most common causes of an acute abdomen, with a 7% lifetime occurrence rate.

B. It develops secondary to obstruction of the appendiceal orifice with secondary mucus accumulation, swelling, ischemia, necrosis, and perforation.

C. Initially, the pain is poorly localized. However, progressive inflammation eventually involves the parietal peritoneum, resulting in pain localized to the RLQ.

D. The risk of perforation increases steadily with age.

1. Ages 10–40, 10%
2. Age 60, 30%
3. Age > 75, 50%

Evidence-Based Diagnosis

A. The classic presentation of nausea and vomiting with pain migration from the periumbilical area to the RLQ is present in only 50–65% of patients.

B. RLQ pain is the most useful clinical finding; LR+, 7.3–8.5; LR–, 0.0–0.3

C. Most of the clinical findings have low sensitivity for appendicitis making it difficult to rule out the diagnosis.

1. In one study, guarding was completely absent in 22% of patients, and rebound was completely absent in 16% of patients with appendicitis.
2. Fever was present in only 40% of patients with perforated appendices.

Fever, severe tenderness, guarding, and rebound may be absent in patients with appendicitis.

D. Nonetheless, certain findings increase the likelihood of appendicitis when present (ie, rebound, guarding) (Table 3-6).

Table 3-6. Classic clinical and laboratory findings in appendicitis.

Finding	Sensitivity	Specificity	LR+	LR–
Clinical findings				
Guarding (moderate to severe)	46%	92%	5.5	0.59
Rebound (moderate to severe)	61%	82%	3.5	0.47
Vomiting	49%	76%	2.0	0.7
Pain migration to RLQ	54%	63%	1.5	0.7
RLQ tenderness	89–100%	12–59%	1.1–2.2	0–0.2
Fever > 38.1°C	15–67%	85%	1	1
Laboratory findings				
WBC > 7000/mcL	98%	21%	1.2	0.1
WBC > 11,000/mcL	76%	74%	2.9	0.3
WBC > 17,000/mcL	15%	98%	7.5	0.9

RLQ, right lower quadrant; WBC, white blood cell.

E. Symptoms are different in octogenarians than in patients aged 60–79 years.

1. Symptom duration is longer (48 vs 24 hours).
2. They are less likely to report that pain migrated to the RLQ (29% vs 49%).

F. History is particularly important in women to differentiate other causes of RLQ pain (eg, PID, ruptured ectopic pregnancy, ovarian torsion, and ruptured ovarian cyst). The most useful clinical clues that suggest PID include the following:

1. History of PID
2. Vaginal discharge
3. Cervical motion tenderness on pelvic exam

Rule out ectopic pregnancy in premenopausal women who complain of abdominal pain by testing urine for beta-HCG.

G. White blood cell (WBC) count and C-reactive protein (CRP)

1. The WBC and CRP neither identify nor rule out acute appendicitis.
2. WBC > 10/mcL
   - **a.** The sensitivity is only 82% with an LR– of only 0.4
   - **b.** In patients presenting in the first 24 hours
     - **(1)** Sensitivity is only 23%; specificity is 41%
     - **(2)** LR+, 1.4
3. CRP > 10 mg/L
   - **a.** Sensitivity, 77%; specificity, 37%
   - **b.** LR+, 1.2; LR–, 0.6

The WBC and CRP cannot reliably rule in nor rule out acute appendicitis.

H. Urinalysis may be misleading and reveal pyuria and hematuria due to bladder inflammation from an adjacent appendicitis.

I. Clinical decision rules:

1. There are 2 clinical decision rules.
    - **a.** The Alvarado score
        - **(1)** Most commonly validated rule
        - **(2)** However, has a wide sensitivity (68–96%) and specificity (58–89%)
    - **b.** The Appendicitis Inflammatory Response score
        - **(1)** Less commonly used (Table 3-7)

Table 3-7. Appendicitis Inflammatory Response (AIR) score.

Symptom/Sign	Score
Vomiting	1
Pain in right lower quadrant	1
Rebound	
Light	1
Medium	2
Strong	3
Temperature > 38.5° C	1
Leukocyte shift	
70–84%	1
≥ 85%	2
White blood cell	
> 10.0 – 14.9 K	1
≥ 15.0	2
C-reactive protein	
10–49 g/L	1
≥ 50	2
	Maximum score: 12 Score 0–4: low probability of appendicitis

(2) Its specificity is similar 62–85% (LR+, 2.4–6.2), but it has a higher sensitivity (90–94%) and lower LR– (0.08–0.16), suggesting it may be helpful in ruling out appendicitis.

(3) However, no prospective studies have incorporated the score into clinical decisions and documented its safety.

2. Neither is satisfactory for clinical use.

J. CT scanning

1. Test of choice to rule out appendicitis when the diagnosis is uncertain
2. Sensitivity, 92.7%; specificity, 96%; LR+, 24; LR−, 0.08 without contrast. Oral and IV contrast may increase sensitivity further.
3. Two randomized controlled trials demonstrated that CT scans reduce unnecessary surgery compared with clinical evaluation without CT (2.6% vs 13.9% and 6.7% vs 27%).
4. CT scanning lowers overall costs.

K. Ultrasonography (by both radiologist and trained emergency physicians)

1. Sensitivity is poor, ranging from 48% to 80% and may be particularly *insensitive* in patients with appendicitis but a low clinical suspicion, limiting its usefulness.
2. Operator and experience dependent: The sensitivity was only 33–66% when clinicians completed short training courses (1–4 hours).
3. Point-of-care ultrasound by trained emergency physicians is less sensitive than CT but fairly specific.
   a. Sensitivity 80% (75–83%); specificity, 90–94%
   b. LR+, 10.2 (8.2–12.7); LR–, 0.22 (0.19–0.26)
4. Should not be used to rule out appendicitis

L. Diagnosis in pregnancy

1. Although inferior to CT, ultrasonography is recommended due to the risk of radiation to the fetus.
2. Indeterminate studies in the third trimester are common.
3. Unenhanced MRI
   a. An alternative option for patients with indeterminate ultrasound; sensitivity, 91%; specificity, 98%; LR+, 45; LR−, 0.09
   b. Gadolinium is a pregnancy category C drug and is not advised.
4. Surgical and gynecologic consultation suggested.

Treatment

A. Observation is critical.

B. Monitor urinary output and vital signs.

C. IV fluid resuscitation

D. Broad-spectrum antibiotics, including gram-negative and anaerobic coverage.

E. Trials have compared surgery with nonsurgical therapy.

1. Patients were randomized to surgery vs. observation with IV antibiotics.
2. Patients were required to have uncomplicated appendicitis defined by CT scanning.

3. Patients who were immunocompromised, older, or had comorbidities were excluded.
4. 10% of patients in the observation group required emergent appendectomy.
5. An additional 30% of patients in the observation group needed surgery within the year for recurrences.

F. Surgery remains the therapy of choice.

MAKING A DIAGNOSIS

Mr. C's symptoms are consistent with—but certainly not diagnostic of—appendicitis. He has no risk factors for any of the alternative diagnoses of pancreatitis, PUD, or bowel obstruction (alcohol use, NSAID ingestion, or prior abdominal surgery, respectively). Diagnostic options include obtaining a complete blood count (CBC) (always done but clearly of limited value), continued observation and reexamination, surgical consultation, and obtaining a CT scan. Given the lack of evidence for any of the less concerning possibilities, you remain concerned that the patient has early appendicitis. You elect to observe the patient, obtain a CBC and lipase, order a CT scan and ask for a surgical consult.

Frequent clinical observations are exceptionally useful when evaluating a patient with possible appendicitis.

The CBC reveals a WBC of 8700/mcL (86% neutrophils, 0% bands) and an HCT of 44%. The lipase is normal. On reexamination the patient complains that the pain is now more severe in the RLQ. On exam, he is moderately tender but still without rebound or guarding.

The migration of pain to the RLQ is suggestive of appendicitis. Less likely considerations might include Crohn ileitis as well as diverticulitis or colon cancer (both unlikely in this age group). If the patient were a woman, PID and ovarian pathology (ruptured ectopic pregnancy, ovarian torsion, or ruptured ovarian cyst) would also need to be considered.

Diffuse abdominal pain that subsequently localizes and becomes more constant, suggests parietal peritoneal inflammation.

The CT scan reveals a hypodense fluid collection on the right side inferior to the cecum. An appendolith is seen. The interpretation is possible appendiceal perforation versus Crohn disease.

CASE RESOLUTION

The patient's symptom complex, particularly the pain's migration, localization, and intensification are highly suggestive of appendicitis. CT findings make this diagnosis likely. At this point, surgical exploration is appropriate.

The patient undergoes surgery and purulent material is found in the peritoneal cavity. A necrotic appendix is removed, and the peritoneal cavity is irrigated. The patient is treated with broad-spectrum antibiotics and does well postoperatively.

CHIEF COMPLAINT

PATIENT 

Ms. R is a 50-year-old woman who comes to the office complaining of abdominal pain. The patient reports that she has been having "episodes" or "attacks" of abdominal pain over the last month, with about 3 "attacks" during this time. The last attack was 4 days previously. She reports that the attacks of pain are in the epigastrium, last up to 4 hours, and often awaken her at night. The pain is described as a severe cramping-like sensation that is very intense and steady for hours. Occasionally, the pain radiates to the right back. The pain is associated with emesis. She reports that the color of her urine and stool are normal. On physical exam, her vital signs are stable. She is afebrile. On HEENT exam, she is anicteric. Her lungs are clear, and cardiac exam is unremarkable. Abdominal exam is soft with only mild epigastric discomfort to deep palpation. Murphy sign (tenderness in the right upper quadrant [RUQ] with palpation during inspiration) is negative. Rectal exam reveals guaiac-negative stool.

At this point, what is the leading hypothesis, what are the active alternatives, and is there a must not miss diagnosis? Given this differential diagnosis, what tests should be ordered?

RANKING THE DIFFERENTIAL DIAGNOSIS

The first pivotal feature of Ms. R's abdominal pain is its epigastric location. Common causes of epigastric pain include PUD, pancreatitis, and biliary colic (Figure 3-1). The second pivotal feature of Ms. R's abdominal pain is its time course, with multiple acute episodes. Many diseases cause well-defined recurrent discrete episodes of abdominal pain (Table 3-1) but of these, only pancreatitis and biliary colic tend to occur in the epigastrium. PUD is a common cause of epigastric abdominal pain and obviously needs to be considered. However, the pain in PUD is typically more chronic than acute, and not typically discrete or so severe, making this a less likely possibility. Ms. R does not have other pivotal clues such as peritoneal findings, unexplained hypotension, or abdominal distention that could focus the differential. The final clinical clue is the severe crampy quality of the pain. Severe intense crampy abdominal pain ("colicky") suggests obstruction of a hollow viscera, which can be caused by biliary, bowel, or ureteral obstruction (due to biliary stones, bowel obstruction, or nephrolithiasis, respectively). Taken together, the epigastric location, multiple discrete episodes, quality and intensity of the pain, make biliary colic the leading hypothesis. Table 3-8 lists the differential diagnosis.

Table 3-8. Diagnostic hypotheses for Ms. R.

Diagnostic Hypotheses	Demographics, Risk Factors, Symptoms and Signs	Important Tests
Leading Hypothesis		
Biliary colic	Episodic and crampy pain may radiate to back	Ultrasonography
Active Alternatives—Most Common		
Peptic ulcer disease	NSAID use *Helicobacter pylori* infection Melena Pain relieved by eating or by antacids	EGD *H pylori*, stool antigen assay
Pancreatitis	Alcohol abuse Gallstones	Serum lipase

EGD, esophagogastroduodenoscopy; NSAID, nonsteroidal anti-inflammatory drug.

Ms. R reports no history of alcohol binging, NSAID use, or known PUD. The pain does not improve with food or antacids. The pain is not relieved by defecation.

Is the clinical information sufficient to make a diagnosis? If not, what other information do you need?

Leading Hypothesis: Biliary Colic

Textbook Presentation

Gallstone disease may present as incidentally discovered asymptomatic cholelithiasis, biliary colic, cholecystitis, cholangitis, or pancreatitis. The pattern depends on the location of the stone and its chronicity (Figure 3-2). Biliary colic typically presents with episodes of intense abdominal pain that begin 1 hour or more after eating and commonly wake patients from sleep. The pain is usually located in the RUQ, although epigastric pain is also common. The pain may radiate to the back and may be associated with nausea and vomiting. The pain usually lasts for more than 30 minutes and may last for hours.

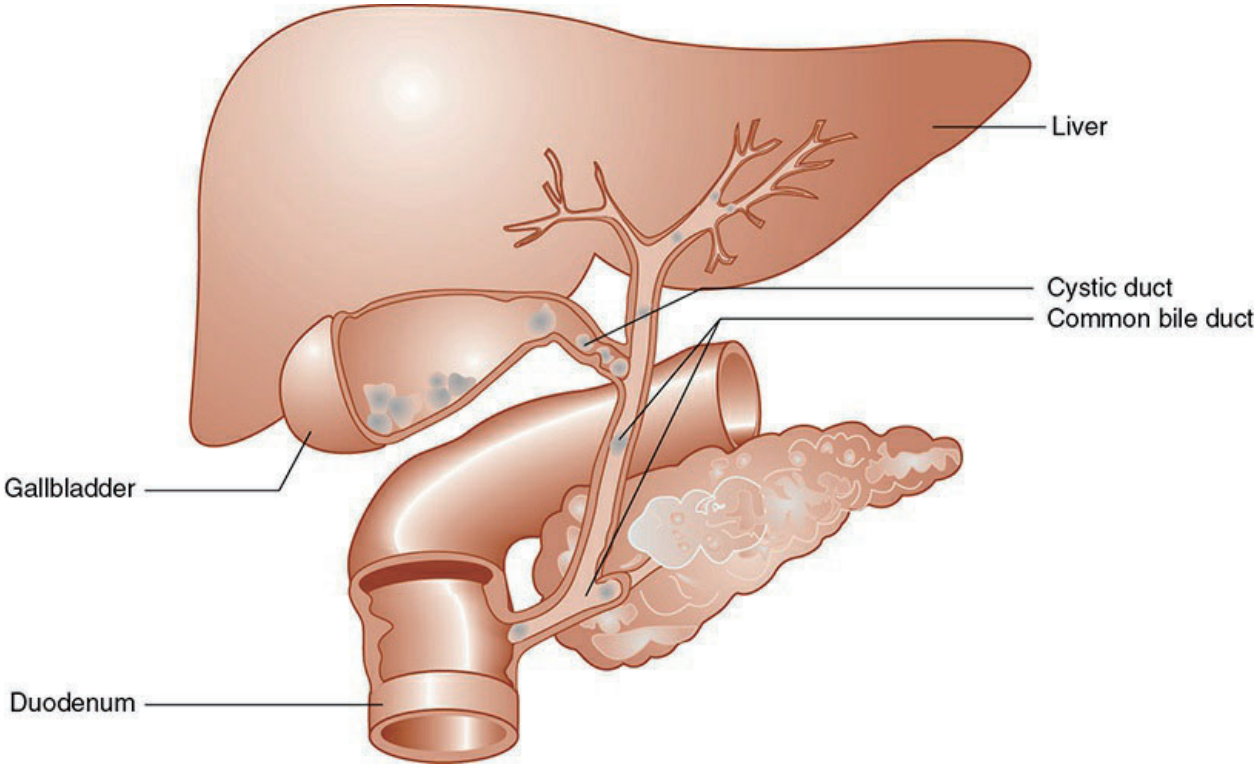


Figure 3-2. Calculi may lodge in several locations. In the cystic duct, they may cause biliary colic or cholecystitis. In the common bile duct, they may cause cholangitis and/or pancreatitis.

Disease Highlights

A. Biliary colic

1. Gallstones are commonly *asymptomatic*. Biliary colic occurs when a gallstone becomes lodged in the cystic duct and the gallbladder contracts against the obstruction.
2. Annual risk of biliary colic in patients with asymptomatic gallstones is 1–4%.
3. Risk factors for gallstone development
   - **a.** Increasing age is the predominant risk factor. The prevalence is 8% in patients older than 40 years and 20% in those older than 60 years (Figure 3-3).

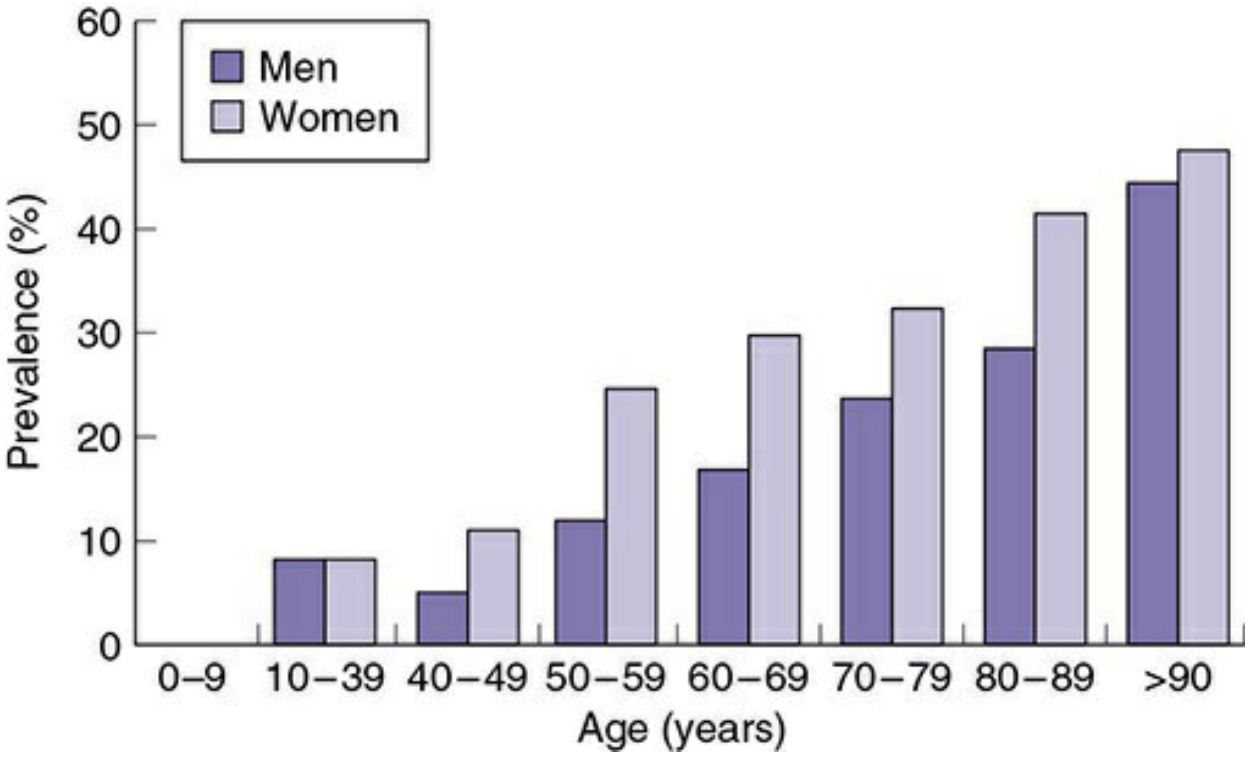


Figure 3-3. Prevalence of asymptomatic gallstones by age. (Reproduced with permission from Bateson MC. Forttnightly review: Gallbladder disease, BMJ. 1999;June 26;318(7200):1745–1748.)

b. Obesity

c. Sex: women are affected more than men (risk increases during pregnancy), although the incidence in men is still significant (Figure 3-3).

d. Gallbladder stasis (due to rapid weight loss, which may occur in patients on very low-calorie diets, on total parenteral nutrition, and after surgery)

e. Other less common risk factors include family history, Crohn disease, and hemolytic anemias (eg, thalassemia, sickle cell disease), which can lead to increased bilirubin excretion and bilirubin stones.

4. Presents as one of the classic visceral obstructive syndromes with severe, crampy waves of pain that incapacitate the patient.
5. Characterized by episodes of pain with pain-free intervals of weeks to years.
6. Pain begins 1–4 hours after eating or may awaken the patient during the night. May be precipitated by fatty meals.
7. The pain is usually associated with nausea and vomiting.
8. The pain usually lasts < 2–4 hours. An episode that lasts longer than 4–6 hours and is accompanied by fever or marked RUQ tenderness suggests cholecystitis has developed.
9. Resolution occurs if the stone comes out of the cystic duct. The intense pain improves fairly rapidly, although mild discomfort may persist for 1–2 days.
10. Prognosis

a. Biliary colic recurs in 50% of symptomatic patients.

b. Acute cholecystitis develops if the stone remains lodged in the cystic duct.

c. Complications (eg, pancreatitis, acute cholecystitis, or ascending cholangitis) occur in 25% of patients who have experienced biliary colic.

11. Colic occasionally develops in patients without stones secondary to sphincter of Oddi dysfunction or scarring leading to obstruction.

Evidence-Based Diagnosis

A. Pain localized to the RUQ in 54% of patients and epigastrium in 34% of patients. It also may present as a band-like pain across the entire upper abdomen, or rarely in the mid-abdomen. Pain may radiate to back, right scapula, right flank, or chest.

B. Laboratory tests (liver biochemical tests, lipase, urinalysis) are normal in uncomplicated biliary colic. Abnormalities suggest other diagnoses or complications (eg, stone migration into the common bile duct [CBD]).

C. Ultrasonography is the test of choice

1. Sensitivity, 89%; specificity, 97%

2. LR+, 30; LR−, 0.11

D. CT scan is only 79% sensitive.

E. Endoscopic ultrasound (EUS) is 100% sensitive and is useful in patients with a negative transabdominal ultrasound but in whom biliary colic is still strongly suspected.

Treatment

A. Cholecystectomy is recommended for patients with *symptomatic* disease.

B. Cholecystectomy not advised for patients with asymptomatic cholelithiasis.

Make sure the gallstones are *causing the pain* before advising cholecystectomy.

C. Lithotripsy is not advised

D. Dissolution therapies (eg, ursodiol or chenodiol) are reserved for patients who are not surgical candidates for surgery.

MAKING A DIAGNOSIS

Ms. R's history suggests biliary colic. You order an ultrasound of the RUQ.

A RUQ ultrasound reveals multiple small gallstones within the gallbladder. The CBD is normal, and no other abnormalities are seen. The serum lipase and liver biochemical tests are normal, and stool antigen for *Helicobacter pylori* is negative.

Have you crossed a diagnostic threshold for the leading hypothesis, biliary colic? Have you ruled out the active alternatives? Do other tests need to be done to exclude the alternative diagnoses?

Alternative Diagnosis: PUD

See Chapter 32, Unintentional Weight Loss.

Alternative Diagnosis: Acute Pancreatitis (see below)

CASE RESOLUTION

Ms. R discussed her case with her primary care physician and surgeon. Both agree that her symptom complex and ultrasound suggest biliary colic. Furthermore, there was no evidence of any of the alternative diagnoses. The normal lipase effectively rules out pancreatitis, and the combination of no NSAIDs and a negative *H pylori* stool antigen makes PUD very unlikely. They recommend surgery, which she schedules for the end of the summer.

FOLLOW-UP

Ms. R returns 3 weeks later (and prior to her scheduled surgery) in acute distress. She reports that her pain began last evening, is in the same location as her previous bouts of pain, but unlike her previous episodes, the pain has persisted. She is very uncomfortable. She reports that her urine has changed color and is now quite dark, “like tea.” In addition, she complains of “teeth chattering” chills. On physical exam, Ms. R is febrile (38.5°C). Her other vital signs are stable. Sclera are anicteric and cardiac and pulmonary exams are all completely normal. Abdominal exam reveals moderate tenderness in the epigastrium and RUQ. Murphy sign is positive.

At this point, what is the leading hypothesis, what are the active alternatives, and is there a must not miss diagnosis? Given this differential diagnosis, what tests should be ordered?

RANKING THE DIFFERENTIAL DIAGNOSIS

This episode of abdominal pain raises several possibilities. The first is that the current symptom complex is in some way related to her known cholelithiasis. The persistent pain suggests either cholecystitis (due to a stone lodged in the cystic duct), choledocholithiasis, ascending cholangitis, or pancreatitis. Of note, her dark urine is a pivotal clinical clue. Both hematuria and bilirubinuria can cause dark urine. Bilirubinuria only occurs in patients with conjugated hyperbilirubinemia which, in turn, is due to either CBD obstruction or hepatitis. In Ms. R, the preexistent biliary colic, persistent RUQ pain, and dark urine make the most likely diagnosis CBD obstruction due to migration of a stone into the CBD (choledocholithiasis) (Figure 3-2). Conversely, this symptom complex is not consistent with cholecystitis. In uncomplicated cholecystitis, only the cystic duct is obstructed. Since the *CBD* is *not* obstructed, bile flows unimpeded into the bowel, and does not back up into the liver. Therefore, such patients do not develop hyperbilirubinemia, bilirubinuria, dark urine, or significant increases in ALT or AST. Finally, Ms. R's fever suggests that the CBD obstruction has been complicated by ascending infection and taken together suggests ascending cholangitis, a life-threatening condition.

Dark urine suggests bilirubinuria and may precede icterus.

Rigors (defined as visible shaking or teeth chattering chills) suggests bacteremia and should increase the suspicion of a life-threatening bacterial infection.

Other considerations include hepatitis or pancreatitis (which may be caused by concomitant pancreatic duct obstruction). While hepatitis can cause RUQ pain, hyperbilirubinemia, and bilirubinuria, it would also require giving Ms. R another unrelated diagnosis and is therefore less likely. Table 3-9 lists the differential diagnosis.

Table 3-9. Diagnostic hypotheses for Ms. R on follow-up.

Diagnostic Hypothesis	Demographics, Risk Factors, Symptoms and Signs	Important Tests
Leading Hypothesis		
Ascending cholangitis	RUQ or epigastric pain Dark urine Fever Rigors	Ultrasonography Endoscopic ultrasonography ERCP MRCP CBC ALT, AST, bilirubin Blood cultures
Active Alternatives—Most Common		
Acute cholecystitis	RUQ pain Fever	Ultrasonography
Pancreatitis	Alcohol abuse Gallstones	Serum lipase Ultrasonography
Hepatitis	Alcohol abuse Injection drug use RUQ pain Nausea Dark urine	Elevated ALT and AST Viral serologies

ALT, alanine transaminase; AST, aspartate transaminase; CBC, complete blood count; ERCP, endoscopic retrograde cholangiopancreatography; MRCP, magnetic resonance cholangiopancreatography; RUQ, right upper quadrant.

2

Laboratory results include WBC 17,000/mcL (84% neutrophils, 10% bands). HCT is 38%; lipase, 17 units/L (nl 11–65 units/L); alkaline phosphatase, 467 units/L (nl 30–120); bilirubin,

4.2 mg/dL; conjugated bilirubin, 3.0 mg/dL (nl 0 – 0.3); GGT, 246 units/L (nl 8–35); ALT, 100 units/L (nl 15–59). Ultrasound shows sludge and stones within the gallbladder. No CBD dilatation or CBD stone is seen. Blood cultures are ordered and you initiate broad-spectrum IV antibiotics (ie, piperacillin/tazobactam).

Is the clinical information sufficient to make a diagnosis of ascending cholangitis? If not, what other information do you need?

Leading Hypothesis: Choledocholithiasis & Ascending Cholangitis

Textbook Presentation

Patients typically have some form of biliary obstruction (biliary colic, acute cholecystitis or gallstone pancreatitis). The presence of jaundice, increased liver biochemical tests or lipase suggest concomitant CBD obstruction. When accompanied by fever, it also suggests ascending cholangitis.

Disease Highlights

A. 5–20% of patients with symptomatic gallstones have stones within the CBD (choledocholithiasis).

B. Patients with choledocholithiasis may be asymptomatic.

C. Complications of choledocholithiasis may be the presenting manifestations.

1. Obstruction and jaundice may be present.
2. Fever, jaundice, and leukocytosis may be present due to ascending infection from the duodenum (ascending cholangitis). Ascending cholangitis may also occur when the CBD is obstructed due to tumors or strictures.
3. Pancreatitis may occur if there is concomitant obstruction of the pancreatic duct.

Evidence-Based Diagnosis

A. Ascending cholangitis

1. Clinical findings in patients with cholangitis include jaundice, 79%; temperature ≥ 38.0°C, 77%; and RUQ pain, 68%. In various studies 42–75% of patients had all 3 (Charcot triad).
2. There is leukocytosis in 73% of patients and elevated alkaline phosphatase and bilirubin in 91% and 87%, respectively.
3. 74% of patients are bacteremic

Bacteremia is exceptionally common in ascending cholangitis. Antibiotics should be administered promptly to patients in whom this diagnosis is suspected.

B. Choledocholithiasis

1. Any of the following suggests choledocholithiasis and warrants CBD evaluation (Table 3-10):

Table 3-10. Test characteristics for choledocholithiasis.

Finding	Sensitivity	Specificity	LR+	LR–
Cholangitis	11%	99%	18.3	0.93
Jaundice	36%	97%	10.1	0.69
Dilated CBD on ultrasound	42%	96%	6.9	0.77
Elevated alkaline phosphatase	57%	86%	2.6	0.65
Elevated amylase	11%	95%	1.5	0.99

CBD, common bile duct.
Modified with permission from Paul A, Millat B, Holthausen U, et al: Diagnosis and treatment of common bile duct stones (CBDS). Results of a consensus development conference, Surg Endosc. 1998 Jun;12(6):856–864.

a. Cholangitis

b. Jaundice

c. Dilated CBD on ultrasound

d. Elevated alkaline phosphatase

e. Elevated amylase or lipase

2. CBD stones are present in 5–8% of patients without any of the aforementioned risk factors.
3. Transabdominal ultrasound is *insensitive* for choledocholithiasis as opposed to its excellent performance in cholelithiasis. A dilated CBD is seen in only 25% of patients.
4. CT scanning is only 75% sensitive for choledocholithiasis and not the test of choice.
5. Endoscopic retrograde cholangiopancreatography (ERCP), magnetic resonance cholangiopancreatography (MRCP), and EUS are highly accurate in detecting CBD stones. These techniques share high sensitivity (90–100%) and specificity (90–100%).
   a. ERCP
      (1) Invasive procedure
      (2) Requires sedation
      (3) Both diagnostic and *therapeutic* because it allows for simultaneous imaging, sphincterotomy, and stone extraction
      (4) > 90% sensitive, 99% specific for diagnosis
      (5) Complicated by pancreatitis in 1–5% of patients and therefore reserved for

patients with a high pretest probability of disease in whom stone extraction is likely (eg, those with jaundice and fever)

b. MRCP

(1) *Noninvasive* scan visualizes CBD and adjacent structures

(2) Highly accurate for CBD stones: 90–100% sensitive, 88–100% specific

c. EUS

(1) Both sensitive (94–99%) and specific (94–95%) for CBD stones.

(2) One study reported that EUS was more sensitive than ERCP (97% vs 67%).

(3) A normal EUS or MRCP would obviate the need for a more invasive ERCP.

(4) EUS followed by selective ERCP in patients with documented CBD stones is an appropriate strategy in patients with suspected choledocholithiasis without cholangitis. Reserving ERCP for the subset of patients with *documented* choledocholithiasis decreases the need for ERCP by 67% and the complication rate by 12%, compared with performing ERCP in all patients with suspected choledocholithiasis.

d. Summary: The American Society of Gastrointestinal Endoscopy recommends the following based on the risk of choledocholithiasis:

(1) High-risk patients (> 50% risk): ERCP is recommended in patients with

(a) Clinical ascending cholangitis

(b) Documented CBD stones

(b) Bilirubin > 4 mg/dL

(d) Bilirubin levels of 1.8–4 mg/dL and dilated CBDs

(2) Moderate-risk patients (10–50% risk): MRCP or EUS is recommended followed by selective ERCP in patients with *documented* choledocholithiasis.

(a) Patients with dilated CBD on ultrasound

(b) Elevated bilirubin levels (1.8–4 mg/dL)

(c) Gallstone-associated pancreatitis (GAP)

(d) Elevated liver biochemical tests

(e) Age > 55 years

(f) An alternative approach is laparoscopic cholecystectomy with intraoperative cholangiogram and postoperative ERCP in patients with choledocholithiasis.

(3) Low-risk patients (< 10% risk): In patients with gallstone disease and none of the aforementioned risk factors, laparoscopic cholecystectomy without other preoperative CBD evaluation is recommended.

Treatment

A. Ascending cholangitis

1. Blood cultures, IV broad-spectrum antibiotics, and IV hydration should begin immediately.

2. Biliary drainage with ERCP should be performed urgently in patients with moderate to severe disease. (Organ dysfunction or 2 or more of the following: abnormal WBC, fever, age > 75 years, total bilirubin > 5 mg/dL, albumin < 0.7 times the lower limit of normal.)
3. In patients with mild disease, biliary drainage should be performed if there is no response to initial treatment within 24 hours.
4. If ERCP is unavailable, percutaneous transhepatic drainage or surgical decompression can be used.

B. In patients who have choledocholithiasis without ascending cholangitis, CBD stones can be removed via intraoperative CBD exploration or ERCP.

C. Cholecystectomy is recommended after inflammation has resolved in patients with ascending cholangitis.

MAKING A DIAGNOSIS

Neither dilation of the CBD nor CBD stone can be seen on ultrasound (but is only 25% sensitive). You still suspect choledocholithiasis because of the jaundice and increased transaminases.

Twenty-four hours later, blood cultures are positive for *Escherichia coli* (consistent with ascending cholangitis).

Have you crossed a diagnostic threshold for the leading hypothesis, ascending cholangitis? Have you ruled out the active alternatives? Do other tests need to be done to exclude the alternative diagnoses?

Alternative Diagnosis: Acute Hepatitis

See Chapter 26, Jaundice & Abnormal Liver Enzymes.

Alternative Diagnosis: Acute Cholecystitis

Textbook Presentation

Typical symptoms of acute cholecystitis include *persistent* RUQ or epigastric pain, fever, nausea, and vomiting.

Disease Highlights

A. Secondary to prolonged cystic duct obstruction (> 4–6 hours)

B. Persistent obstruction results in increasing gallbladder inflammation and pain. Necrosis, infection, and gangrene may occur.

C. Jaundice and marked elevation of liver enzymes are seen *only* if the stone migrates into the CBD and causes obstruction.

Evidence-Based Diagnosis

A. No clinical finding is sufficiently sensitive to rule out cholecystitis.

1. Fever: (sensitivity, 29–44%; specificity, 37–83%)
2. Marked RUQ tenderness (sensitivity, 60–98%; specificity, 1–97%; LR+, 2.7; LR–, 0.4)
3. Murphy sign
   - **a.** Sensitivity, 65%; specificity, 87%
   - **b.** LR+, 5.0; LR−, 0.4

B. Laboratory findings

1. Leukocytosis (> 10,000/mcL) is present in 52–63% of patients.
2. Cholecystitis does *not* typically cause significant increases in lipase or liver biochemical tests. Such findings suggest complications of pancreatitis and choledocholithiasis.

C. Ultrasound

1. Test of choice due to speed, cost, ability to image adjacent organs and lack of radiation.
2. Sensitivity, 81%; specificity, 83%; LR+, 4.8; LR−, 0.23
3. Cholelithiasis is usually present (84–99%) but is not in and of itself diagnostic of acute cholecystitis.
4. Additional findings that suggest acute cholecystitis include gallstones *with* gallbladder wall thickening, pericholecystic fluid, sonographic Murphy sign, or gallbladder enlargement > 5 cm. However, more specific findings may be less sensitive (27–38%).
5. If ultrasound is normal, and clinical suspicion is high, consider HIDA (see below).

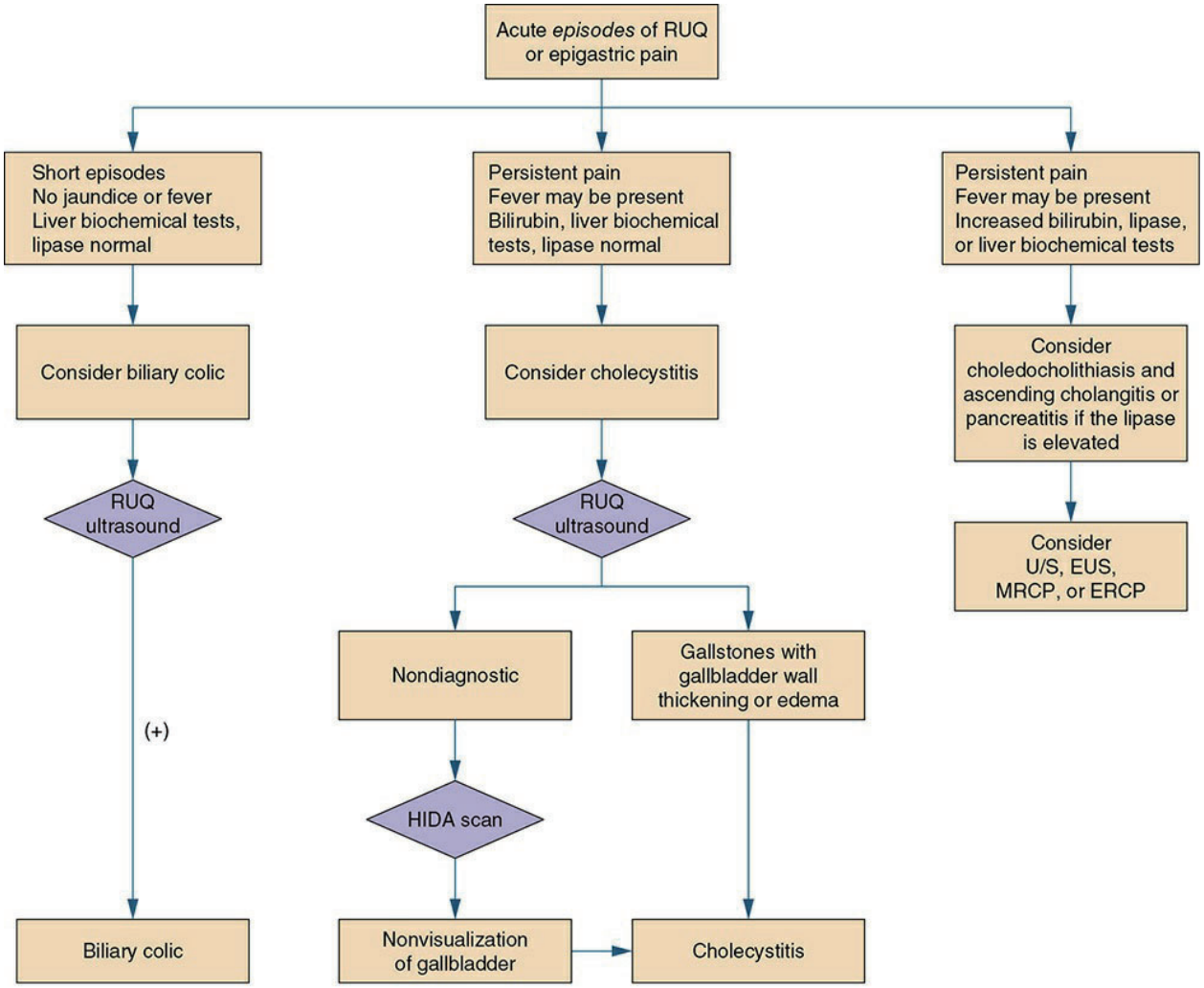
D. Bedside ultrasound by trained nonradiologists

1. Used with increasing frequency in emergency departments
2. One study reported good accuracy when performed by emergency department physicians with 5 hours of training; sensitivity, 91%; specificity, 66%; LR+, 2.7; LR–, 0.14.
3. Abnormal results should be confirmed with formal ultrasonography.
4. Normal results are probably adequate to rule out cholecystitis in patients with low pretest probabilities but not in those for whom there is a higher suspicion of acute cholecystitis.

E. Cholescintigraphy (HIDA) scans

1. Radioisotope is excreted by the liver into the biliary system. In normal patients, the gallbladder concentrates the isotope and is visualized. Visualization essentially excludes acute cholecystitis.
2. Nonvisualization of the gallbladder suggests cystic duct obstruction and is highly specific for acute cholecystitis (96% sensitive, 90% specific).
3. Nonvisualization can also be seen in prolonged fasting, hepatitis, alcohol abuse, and prior biliary sphincterotomy.
4. Useful when the pretest probability is high, due to persistent pain, and the ultrasound is nondiagnostic (ie, the ultrasound demonstrates stones within the gallbladder but no clear evidence of cholecystitis is seen, eg, no stones within the cystic duct nor evidence of gallbladder wall thickening or pericholecystic fluid).

F. A diagnostic algorithm to the approach of suspected gallstone disease is shown in Figure 3-4.



ERCP, endoscopic retrograde cholangiopancreatography; EUS, endoscopic ultrasound; HIDA, hepatobiliary iminodiacetic acid; LFTs, liver function tests; MRCP, magnetic resonance, cholangiopancreatography; RUQ, right upper quadrant; U/S, ultrasound.

Figure 3-4. Diagnostic approach: biliary disease.

Treatment

Patients with acute cholecystitis should be admitted, administered parenteral antibiotics, and undergo cholecystectomy. The timing of surgery depends on the severity of disease and medical risk assessment.

Alternative Diagnosis: Acute Pancreatitis

Textbook Presentation

Patients with acute pancreatitis often complain of a constant and boring abdominal pain of moderate to severe intensity that develops in the epigastrium and may radiate to the back. Associated symptoms may include nausea, vomiting, low-grade fever, and abdominal distention.

Disease Highlights

A. Etiology

1. Alcohol abuse (typically binge drinking) and choledocholithiasis (with concomitant obstruction of pancreatic outflow) cause 80% of acute pancreatitis cases.
2. 15–25% of cases are idiopathic, many of which may be due to microlithiasis or sphincter of Oddi dysfunction.
    - **a.** 34–67% of patients with idiopathic pancreatitis were found to have small gallstones on EUS or ERCP.
    - **b.** Sphincter of Oddi dysfunction may be particularly common in patients with prior cholecystectomy.
3. Post ERCP
4. Drugs commonly associated with pancreatitis include
    - **a.** Azathioprine
    - **b.** Didanosine
    - **c.** Estrogens
    - **d.** Furosemide
    - **e.** Hydrochlorothiazide
    - **f.** L-asparaginase
    - **g.** Metronidazole
    - **h.** Opioids
    - **i.** Pentamidine
    - **j.** Sulfonamides
    - **k.** Corticosteroids
    - **l.** Tamoxifen
    - **m.** Tetracycline
    - **n.** Valproate
5. Less common causes include
    - **a.** Trauma
    - **b.** Marked hypertriglyceridemia (> 1000 mg/dL)
    - **c.** Hypercalcemia
    - **d.** Ischemia
    - **e.** HIV infection, other infection

f. Pancreatic carcinoma

g. Pancreatic divisum

h. Autoimmune pancreatitis

i. Cystic fibrosis

j. Organ transplantation

6. Regardless of the inciting event, trypsinogen is activated to trypsin, which activates other pancreatic enzymes resulting in pancreatic autodigestion and inflammation (which may become systemic and lethal). Interleukins contribute to the inflammation.

B. Complications may be local or systemic. Severe pancreatitis develops in about 20% of patients and is complicated by significant morbidity and mortality.

1. Local complications

   a. Pancreatic pseudocyst

   b. Pancreatic necrosis

   c. Infections: A variety of infections may develop. Bacterial translocation from the bowel may infect pancreatic pseudocysts or necrotic pancreatic tissue. Ascending cholangitis may develop in patients with GAP.

2. Systemic complications

   a. Hyperglycemia

   b. Hypocalcemia

   c. Acute respiratory distress syndrome

   d. Acute kidney injury

   e. Disseminated intravascular coagulation

3. Death occurs in up to 4% of patients.

   a. Usually occurs in patients with infected pancreatic necrosis and in patients in whom multiple organ dysfunction develops.

   b. Several predictive scores have been developed including the Ranson criteria as well as the Glasgow and Apache II scores.

      (1) All use similar variables that increase the likelihood of organ failure, including increased age and elevated WBC, BUN, glucose, or lactate dehydrogenase.

      (2) Hypoxia and hypocalcemia are also associated with an increased risk.

   c. Hemoconcentration (HCT ≥ 50%) on admission predicts severe pancreatitis; LR+, 7.5 (compared to LR–, of 0.4 for patients with HCT ≤ 45%).

   d. CRP > 150 mg/L at 48 hours can also predict severe pancreatitis; sensitivity, 85–86%; specificity, 74–87%; LR+, 3.2–6.6; LR−, 0.16–0.2

Evidence-Based Diagnosis

A. History and physical exam

1. Low-grade fevers (< 38.3°C) are common (60%).
2. Pain may radiate to the back (50%) and may be exacerbated in the supine position.

3. Nausea and vomiting are usually present (75%).
4. Rebound is rare on presentation; guarding is common (50%).
5. Periumbilical bruising (Cullen sign) is rare.
6. Pancreatitis (as well as other diseases) can lead to retroperitoneal bleeding and flank bruising (Grey Turner sign), which is a rare but valuable clue when present.

B. Laboratory studies

1. Lipase > 3 times the upper limit of normal
   - **a.** 79% sensitive, 89% specific; LR+, 7.2; LR–, 0.2
   - **b.** Remains elevated longer than serum amylase
   - **c.** Marked elevations suggest pancreatitis secondary to gallstones.
2. Amylase: 72% sensitive, 93% specific; LR+, 10.3; LR–, 0.3
3. Liver biochemical tests
   - **a.** Studies suggest that *significant* elevations of the bilirubin, alkaline phosphatase, ALT, or AST in patients with pancreatitis suggest etiology secondary to gallstones (GAP). These enzymes increase due to concomitant obstruction of the CBD.
     - **(1)** ALT or AST elevations > 100 international units/L suggest GAP (sensitivity ≈ 55%, specificity ≈ 93%; LR+ 8–9)
     - **(2)** AST levels < 50 international units/L make GAP unlikely (sensitivity, 90%; specificity, 68%; LR−, 0.15).
     - **(3)** 10% of patients with GAP have normal levels of alkaline phosphatase, bilirubin, AST, and ALT.
   - **b.** Patients with GAP have a high risk of recurrent pancreatitis and require cholecystectomy.
4. Imaging: A variety of imaging techniques can be used in patients with acute pancreatitis.
   - **a.** Plain radiography is useful to rule out free air or SBO.
   - **b.** Transabdominal ultrasound is noninvasive and should be performed in *all* patients with pancreatitis to determine whether they have gallstones or CBD dilatation suggesting GAP.
   - **c.** Abdominal CT is 87–90% sensitive and 90–92% specific for the diagnosis of acute pancreatitis but insensitive for determining whether or not patients have GAP.
     - **(1)** Should be performed when the diagnosis is unclear or complications are suspected (pseudocysts or pancreatic necrosis)
     - **(2)** Pancreatic necrosis should be suspected in patients with severe pancreatitis, when signs of sepsis are present, and in patients who do not improve in the first 72 hours.
     - **(3)** IV contrast is required to demonstrate necrosis.
5. Detecting GAP
   - **a.** GAP should be suspected in any patient with pancreatitis that is not clearly secondary to alcohol.
   - **b.** Neither transabdominal ultrasound nor CT is sensitive at detecting

choledocholithiasis (21% and 40%, respectively).

c. MRCP is highly accurate for choledocholithiasis (80–94% sensitive) as are EUS and ERCP (≈ 96–98% sensitive).

d. ERCP

(1) Can cause pancreatitis and is therefore limited to patients with persistent obstruction or cholangitis.

(2) Its use in patients with severe pancreatitis is controversial.

c. Preoperatively, patients with GAP should have CBD evaluation with ERCP, MRCP, or EUS. A reasonable option in such patients is EUS followed by ERCP if stones are identified.

6. Imaging in idiopathic pancreatitis

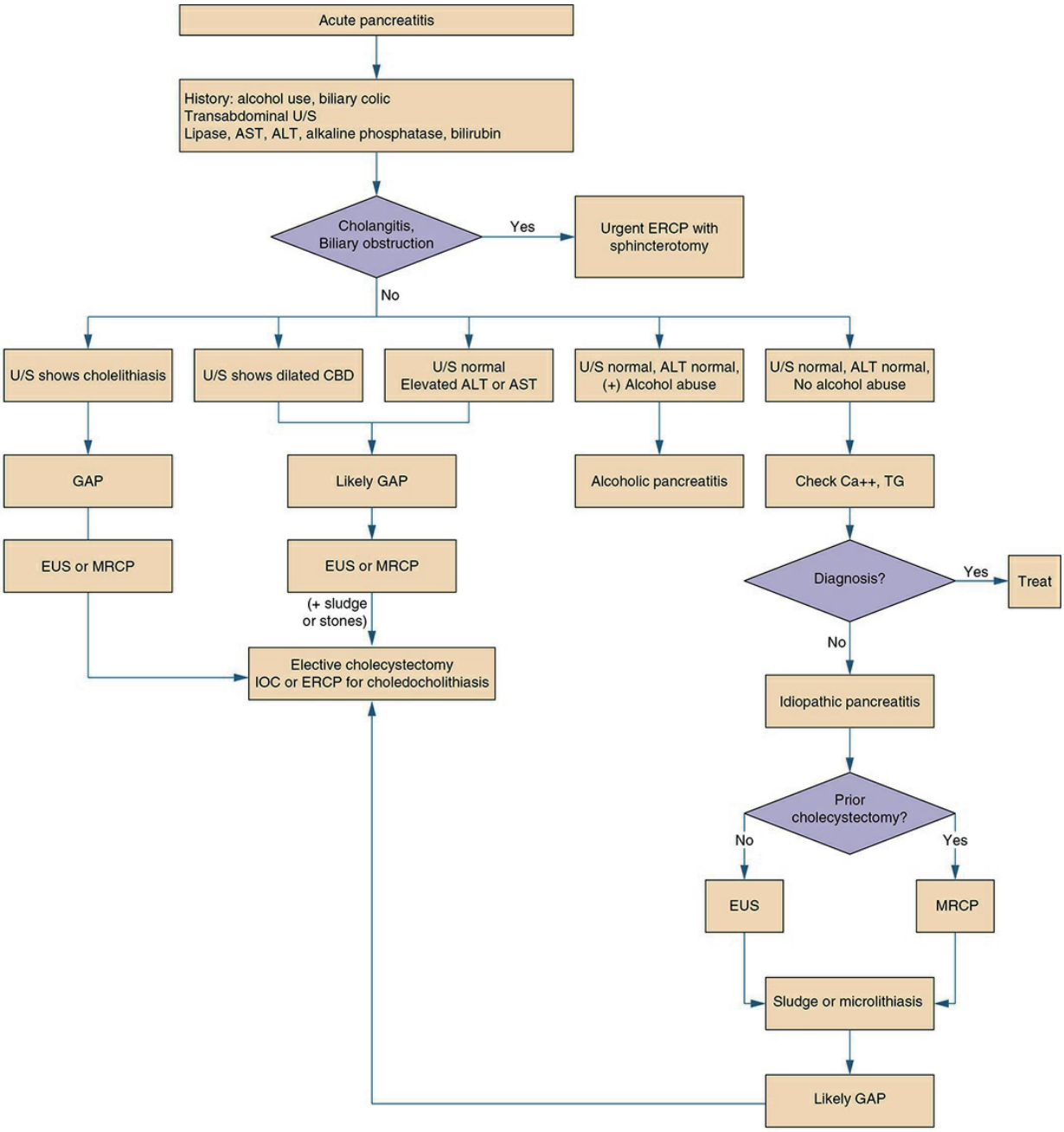
a. EUS is superior to MRCP (except in patients with prior cholecystectomy).

b. EUS is more sensitive for microlithiasis and sludge (suggesting GAP) than MRCP.

c. Sensitivity of EUS is 88–96%, compared with 24% of MRCP.

7. Calcium and triglycerides should be ordered to exclude less common causes of acute pancreatitis.

8. Figure 3-5 outlines an approach to evaluation of pancreatitis.



ALT, alanine transaminase; AST, aspartate transaminase; ERCP, endoscopic retrograde cholangiopancreatography; EUS, endoscopic ultrasound; GAP, gallstone-associated pancreatitis; IOC, intraoperative cholangiogram; MRCP, magnetic resonance cholangiopanereatography; TG, triglycerides; U/S, ultrasound.

Figure 3-5. Evaluation of pancreatitis.

Treatment

A. Vital signs, orthostatic BPs, and urinary output should be carefully monitored to assess intravascular volume.

B. Early aggressive IV fluid is critical, especially in the first 12–24 hours to both support the patient and avoid complications such as infection. BP and urinary output should be monitored. (Urinary output < 0.5 mL/kg/h suggests inadequate renal perfusion and

hypovolemia.)

1. Lactated Ringers is superior to normal saline because it is associated with a significant reduction in the incidence of systemic inflammatory response syndrome at 24 hours (absolute risk reduction ≈ 15%; NNT = 6.7)
2. Lactated Ringers should be avoided in patients with hypercalcemia.

C. Opioids for pain relief

D. Nasogastric tube if recurrent vomiting

E. Oxygen, electrolyte, and glucose monitoring

F. ICU admission for severe pancreatitis

G. Prophylactic antibiotics

1. Prophylactic antibiotics are not recommended in severe pancreatitis due to large number needed to treat (1429)
2. Antibiotics are recommended for extrapancreatic infection (present in 32% of cases) or suspected infected necrosis. For the latter, cultures and CT-guided fine-needle aspiration should be used to guide antibiotics.

H. ERCP and sphincterotomy (see above)

I. GAP: Cholecystectomy and ERCP/sphincterotomy

1. Surgery during the index admission is superior to delayed surgery or ERCP with sphincterotomy (ERCP/S) decreasing the rate of recurrent acute pancreatitis and other biliary events.
2. Recurrent GAP was seen in 1.7% of patients receiving surgery during their index admission vs 5.3% of patients having delayed ERCP/S and 13.2% in patients without surgery.
3. CBD evaluation (with intraoperative cholangiogram, MRCP, or EUS) is also required to ensure that the CBD is clear of stones.

J. Alcohol abstinence

K. Nutrition

1. Enteral feeding is superior to parenteral feeding and has been shown to decrease mortality.
2. Enteral feeding avoids a variety of IV catheter-related complications and decreases gut bacterial translocation, which may contribute to infection.

Alternative Diagnosis: Chronic Pancreatitis

See Chapter 32, Unintentional Weight Loss.

CASE RESOLUTION

An ERCP demonstrates multiple small stones within the CBD, which are extracted. Ms. R underwent cholecystectomy and recovered without incident.

CHIEF COMPLAINT

Mr. J is a previously healthy 63-year-old man with severe abdominal pain for 48 hours. The pain is periumbilical with severe crampy exacerbations that last for several minutes and then subside. He notes loud intestinal noises (borborygmi) during the periods of increased pain. The pain is associated with nausea and vomiting. He denies diarrhea. He reports decreased appetite with no oral intake in the last 48 hours. He denies having this pain previously.

At this point, what is the leading hypothesis, what are the active alternatives, and is there a must not miss diagnosis? Given this differential diagnosis, what tests should be ordered?

RANKING THE DIFFERENTIAL DIAGNOSIS

The first pivotal point for Mr. J's abdominal pain is its periumbilical location. A variety of diseases present with pain in this location, including AAA, appendicitis (early), bowel ischemia, bowel obstruction, diabetic ketoacidosis, gastroenteritis, IBS, and IBD (Figure 3-1). The second useful pivotal point to consider is the time course of Mr. J's abdominal pain (Table 3-1). This allows us to further limit the differential diagnosis to those diseases causing *acute* periumbilical pain. Typically, IBS and IBD do not cause acute pain. Furthermore, diabetic ketoacidosis is unlikely (unless this is his presentation of diabetes). Gastroenteritis is also unlikely given the absence of diarrhea and the severity of the pain. Finally, Mr. J's severe crampy abdominal pain suggests some type of visceral obstruction. The syndromes associated with pain of this quality include ureteral obstruction secondary to kidney stones, biliary obstruction, or intestinal obstruction (large or small bowel). The associated nausea and vomiting can be seen with any of those diseases. However, the combination of the location of the pain and the loud intestinal sounds that accompany the pain makes bowel obstruction the leading hypothesis. It will also be important to determine whether he has unexplained hypotension or abdominal distention during his exam. Table 3-11 lists the differential diagnoses for Mr. J.

Table 3-11. Diagnostic hypotheses for Mr. J.

Diagnostic Hypothesis	Demographics, Risk Factors, Symptoms and Signs	Important Tests
Leading Hypothesis		
Bowel obstruction	Inability to pass stool or flatus Nausea, vomiting Prior abdominal surgery or altered bowel habits Hematochezia Abdominal distention, hyperactive bowel sounds (with tinkling) or hypoactive bowel sounds	Abdominal radiographs, CT scan
Active Alternatives—Must Not Miss		
AAA	Smoking history, male sex, family history of AAA Orthostatic hypotension Pulsatile abdominal mass Decreased lower extremity pulses	Abdominal CT scan Bedside emergency ultrasonography
Appendicitis	Migration of pain from periumbilical region to right lower quadrant	Clinical exam CT scan
Bowel ischemia: Acute mesenteric ischemia	Atrial fibrillation, valvular heart disease, heart failure, hypercoagulable state Abrupt onset pain Pain out of proportion to exam	CT angiography
Bowel ischemia: Ischemic colitis	Age > 60, vascular disease, hypotension (due to MI, sepsis), hematochezia, diarrhea	Colonoscopy

AAA, abdominal aortic aneurysm; MI, myocardial infarction.

Considering each hypothesis in turn (Table 3-11), Mr. J reports no change in his bowel habits until 4 days ago. Since that time, he has been constipated and unable to pass stool or flatus. He also notes that 3 weeks ago, he saw a small amount of blood on the stool. He has no prior history of intra-abdominal surgeries, hernias, or diverticulitis. He does not have a smoking history (which increases the risk for AAA) and states that the pain does not radiate to his back. He has no risk factors for acute mesenteric ischemia (no prior history of atrial fibrillation, valvular heart disease or known hypercoagulable state). On physical exam, he is intermittently very uncomfortable with episodes of severe diffuse cramping pain. Vital signs reveal orthostatic hypotension: supine BP, 110/75 mm Hg; pulse, 90 bpm; upright BP, 85/50 mm Hg; pulse, 125 bpm; temperature, 37.0°C; RR, 18 breaths per minute. He is anicteric. Cardiac and lung exams are unremarkable. On abdominal inspection there is prominent distention. Auscultation shows intermittent rushes. Percussion is tympanitic and on palpation there is mild diffuse tenderness to exam without rebound or guarding. Stool is brown and heme positive.

The constipation, absence of flatus, and rushing bowel sounds further increase the suspicion of bowel obstruction. The tympanitic abdominal distention is a pivotal finding suggesting accumulation of air in the abdomen, in this case most likely due to obstruction. Most cases of SBO are due to adhesions from prior surgery. Mr. J's negative surgical history makes SBO less unlikely. However, the hematochezia raises the possibility of a malignant obstruction and large bowel obstruction (LBO). The orthostatic hypotension is most likely due to dehydration from the vomiting and lack of oral intake and does not in and of itself suggest intra-abdominal hemorrhage from an AAA.

Laboratory findings are WBC 10,000/mcL (70% neutrophils, 0% bands); HCT, 41%. Electrolytes: Na, 141 mEq/L; K, 3.0 mEq/L; HCO_3, 32 mEq/L; Cl, 99 mEq/L; BUN, 45 mg/dL; creatinine 1.0 mg/dL. An abdominal upright radiograph is shown in Figure 3-6.

Is the clinical information sufficient to make a diagnosis? If not, what other information do you need?

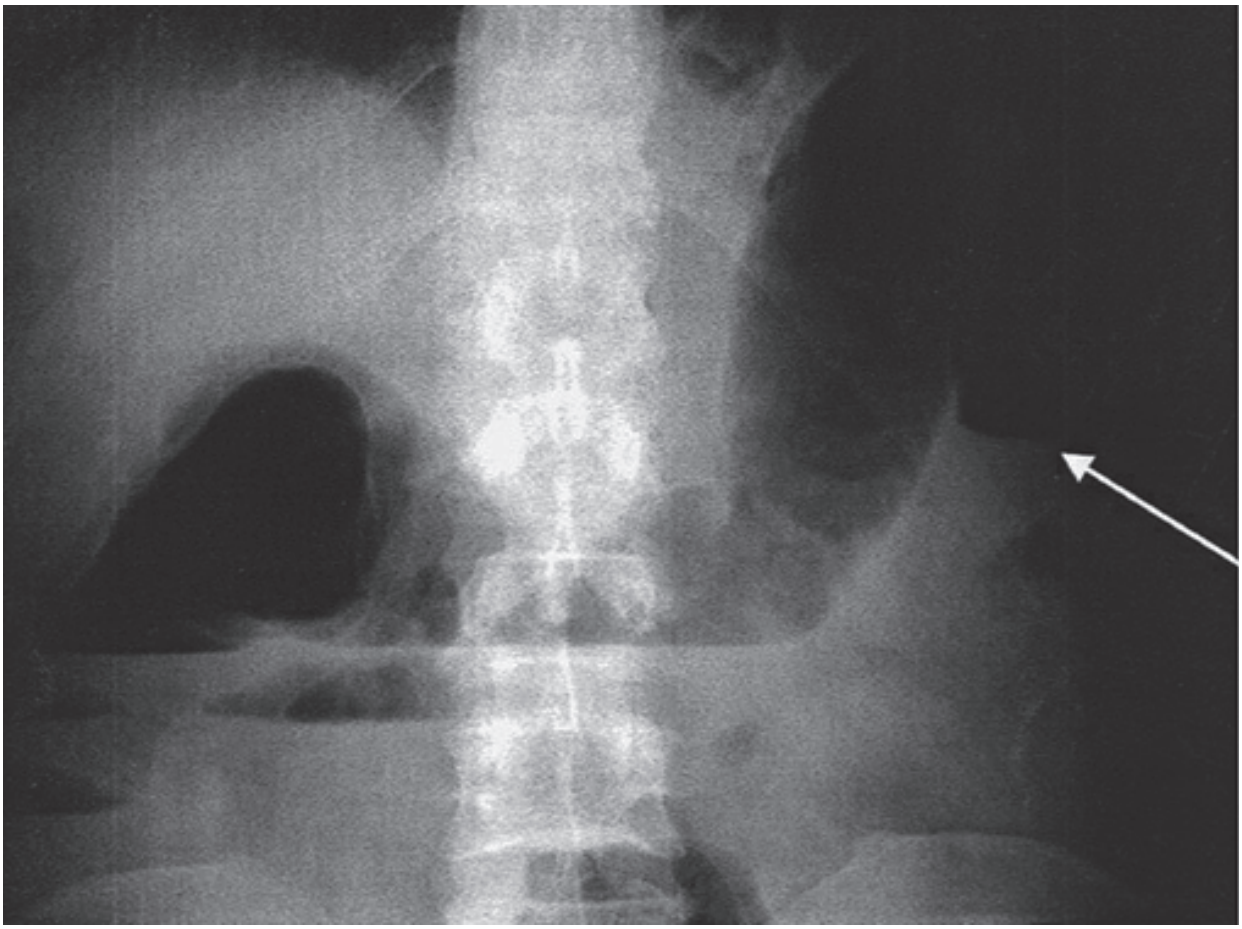


Figure 3-6. Plain radiography reveals grossly distended ascending colon, multiple air-fluid levels, and an abrupt termination of air in the transverse colon (arrow) suggestive of large bowel obstruction.

Leading Hypothesis: Large Bowel Obstruction (LBO)

Textbook Presentation

Bowel obstruction presents with waves of severe crampy abdominal pain that the patient finds incapacitating. Vomiting is common. The pain is often diffuse and poorly localized. Initially, the patient may have several bowel movements as the bowel *distal* to the obstruction is emptied in the first 12–24 hours. Bowel sounds are hyperactive early in the course. Abdominal distention is often present. (Distention is less prominent in proximal SBOs.) At first, the pain is intermittent; later, the pain often becomes more constant, bowel sounds may diminish and become absent, constipation progresses, and the patient becomes unable to pass flatus. If bowel infarction occurs, focal tenderness and peritoneal findings may be seen.

In patients with abdominal pain, the absence of bowel movements or flatus suggests bowel obstruction.

Disease Highlights

A. Bowel obstruction accounts for 4% of patients with abdominal pain.

B. LBO accounts for 24% of all bowel obstructions.

C. Etiology

1. Cancer (53%)
2. Sigmoid or cecal volvulus (17%)
3. Diverticular disease (12%)
4. Extrinsic compression from metastatic cancer (6%)
5. Other (12%) (adhesions rarely cause LBO)

Evidence-Based Diagnosis

A. History and physical exam in LBO and SBO (Table 3-12)

Table 3-12. Test characteristics for predicting bowel obstruction.

Finding	Sensitivity	Specificity	LR+	LR–
Visible peristalsis	6%	99.7%	20	0.94
Prior abdominal surgery	69%	94%	11.5	0.33
Constipation	44%	95%	8.8	0.59
Abdominal distention	63%	89%	5.7	0.42
Increased bowel sounds	40%	89%	3.6	0.67
Reduced bowel sounds	23%	93%	3.3	0.83
Colicky pain	31%	89%	2.8	0.78
Vomiting	75%	65%	2.1	0.38

Data from Böhmer H: Simple Data from History and Physical Examination Help to Exclude Bowel Obstruction and to Avoid Radiographic Studies in Patients with Acute Abdominal Pain, Eur J Surg 1998 Oct;164(10):777–784.

1. None of the expected clinical findings are very sensitive.
   - **a.** Vomiting, 75%
   - **b.** Abdominal distention, 63%
2. Certain findings are specific.
   - **a.** Constipation, 95%; LR+, 8.8

   - **b.** Prior abdominal surgery, 94%; LR+, 11.5
   - **c.** Abdominal distention, 89%; LR+, 5.7
3. Combinations are more specific (but less sensitive 27–48%)

a. Distention associated with increased bowel sounds, vomiting, constipation, or prior surgery are highly suggestive (LR+ ≈ 10).

b. Increased bowel sounds with a history of prior surgery or vomiting is also very suggestive of obstruction (LR+ of 11 and 8, respectively).

B. A CBC and electrolytes should be obtained: Anion gap acidosis suggests bowel infarction or sepsis.

Marked leukocytosis, left shift or anion gap acidosis in a patient with bowel obstruction is a *late* finding and suggests bowel infarction.

C. Plain radiography may show air-fluid levels and distention of large bowel (> 6 cm).

1. 84% sensitive, 72% specific for presence of LBO (not etiology)
2. Small bowel distention also occurs if ileocecal valve is incompetent.

D. CT scan is also accurate in the diagnosis of LBO.

1. 91% sensitive, 91% specific
2. LR+, 10.1; LR−, 0.1

E. Barium enema (water soluble)

1. Barium enema is highly accurate for LBO.
   a. 96% sensitive, 98% specific
   b. LR+, 48; LR−, 0.04
2. Can determine etiology preoperatively (if patient stable)
3. Can exclude acute colonic pseudo-obstruction (distention of the cecum and colon without mechanical obstruction). Colonoscopy can decompress pseudo-obstruction and prevent cecal perforation.

Treatment of LBO

A. Aggressive rehydration and monitoring of urinary output is vital.

B. Broad-spectrum antibiotics advised: 39% of patients have microorganisms in the mesenteric nodes.

C. Surgery, stents, and balloon dilatation have been used. Consultation is advised.

D. For patients with sigmoid volvulus, and no evidence of infarction, sigmoidoscopy allows decompression and elective surgery at a later date to prevent recurrence.

1. Emergent indications for surgery: perforation or ischemia
2. Nonemergent indications for surgery: increasing distention, failure to resolve

MAKING A DIAGNOSIS

After reviewing the plain films, you order an abdominal CT scan.

Have you crossed a diagnostic threshold for the leading hypothesis, LBO? Have you ruled out the active alternatives? Do other tests need to be done to exclude the alternative diagnoses?

Alternative Diagnosis: SBO

Textbook Presentation

The presentation is similar to that for LBO with the exception that patients are more likely to have a history of prior abdominal surgery.

Disease Highlights

A. Bowel obstruction accounts for 4% of patients with abdominal pain.

B. SBO accounts for 76% of all bowel obstructions.

C. Etiology

1. Postsurgical adhesions, 70%
2. Malignant tumor, 10–20%
   - **a.** Usually metastatic
   - **b.** However, 39% of SBOs in patients with a prior malignancy are due to adhesions or benign causes.
3. Hernia (ventral, inguinal, or internal), 10%
4. IBD (with stricture), 5%
5. Radiation
6. Less common causes of SBO include gallstones, bezoars, and intussusception.

D. SBOs may be partial or complete.

1. Complete SBO
   - **a.** 20–40% progress to strangulation and infarction. Strangulation may occur secondary to mesenteric twisting cutting off the blood supply or due to increasing intraluminal pressure directly compromising perfusion.
   - **b.** Clinical signs do *not* allow for identification of strangulation prior to infarction: Fever, leukocytosis, and metabolic acidosis are late signs of strangulation and suggest infarction.
   - **c.** 50–75% of patients admitted for SBO require surgery.
2. Partial SBO
   - **a.** Rarely progresses to strangulation or infarction
   - **b.** Characterized by continuing ability to pass stool or flatus (> 6–12 hours after symptom onset) or passage of contrast into cecum
   - **c.** Resolves spontaneously (without surgery) in 65–80% of patients

Evidence-Based Diagnosis

A. Ideally, tests for SBO should identify obstruction *and* ischemia or infarction, if present (since ischemia and infarction are indications for emergent surgery rather than further observation). Unfortunately, even tests that successfully differentiate complete from partial SBO do not reliably determine whether there is ischemia and infarction.

B. See test characteristics of history and physical exam under LBO.

C. Physical exam findings are *insensitive* at predicting infarction. However, localized tenderness, rebound, or guarding would all suggest infarction is present.

D. WBC may be normal even in presence of ischemia.

E. Plain radiographs may show ≥ 2 air-fluid levels or dilated loops of bowel proximal to obstruction (> 2.5 cm diameter of small bowel).

1. Sensitivity for obstruction 75%; specificity, 66%; LR+, 2.2; LR–, 0.37
2. Rarely determines etiology
3. Complete obstruction is unlikely in patients with air in the colon or rectum.

F. Ultrasound

1. Can show dilated bowel (≥ 25 mm) proximal to normal or collapsed distal bowel.
2. Formal ultrasound
   - **a.** Sensitivity, 90%; specificity, 96%
   - **b.** LR+, 14.1; LR–, 0.13
3. Bedside ultrasound
   - **a.** Has excellent accuracy
   - **b.** Sensitivity, 97%; specificity, 90%
   - **c.** LR+, 9.5; LR–, 0.04

G. CT scanning

1. Sensitivity for determining obstruction is 87%; Specificity, 81%; LR+, 3.6; LR–, 0.18
   - **a.** Obstruction is suggested by a transition point between bowel proximal to the obstruction, which is dilated, and bowel distal to the obstruction, which is collapsed.
   - **b.** CT scanning should be performed prior to nasogastric suction, which may decompress the proximal small bowel and thereby decreases the sensitivity of the CT scan for SBO.
2. May delineate etiology of obstruction
3. Test of choice to diagnose SBO (not ischemia)
4. Not reliably sensitive at determining the presence of ischemia and infarction (and the need for immediate surgery). Different studies have reported sensitivities ranging from 15% to 100% (specificity 85–94%).

The absence of CT signs of ischemia in patients with SBO does not rule out ischemia.

H. Gastrografin small bowel series

1. Diagnosis
   - **a.** Accurate in the diagnosis of SBO and useful to predict nonoperative resolution
   - **b.** 97%, sensitive; 96%, specific (Spontaneous resolution is likely in patients in whom

contrast reaches the colon.)

c. Unlike CT scanning, small bowel series cannot delineate etiology of SBO or demonstrate ischemic changes.

2. Therapy

a. Gastrografin is hyperosmolar and draws fluid into bowel lumen, potentially dilating bowel.

b. Randomized trials of patients with presumed adhesive SBO demonstrate reduced odds of surgical intervention in patients receiving gastrografin compared to controls OR (0.44–0.87).

Treatment

A. Fluid resuscitation

1. IV rehydration is important to correct the prominent intravascular dehydration from decreased oral intake, vomiting, *and* third spacing of fluid within the bowel.
2. Monitor vital signs, orthostasis, and urinary output carefully.

B. Careful, frequent observation and repeated physical exam over the first 12–24 hours

C. Nasogastric suction

D. Broad-spectrum antibiotics are recommended (59% of patients have bacterial translocation to mesenteric lymph nodes), although evidence is limited.

E. Water-soluble contrast for partial SBO that persists for 48 hours.

F. Indications for surgery include any of the following:

1. Signs of ischemia (increased pain, fever, tenderness, peritoneal findings, acidosis, or worsening leukocytosis)
2. CT findings of infarction
3. SBO secondary to hernia
4. SBO clearly not secondary to adhesion (no prior surgery)

Alternative Diagnosis: Ischemic Bowel Secondary to Acute Mesenteric Ischemia or Ischemic Colitis

Ischemic Bowel

Three distinct clinical subtypes of ischemic bowel include chronic mesenteric ischemia (chronic small bowel ischemia), acute mesenteric ischemia (acute ischemia of small bowel), and ischemic colitis (ischemia of the large bowel). Chronic mesenteric ischemia is discussed at the end of the chapter.

1. Acute Mesenteric Ischemia

Textbook Presentation

Acute mesenteric ischemia is a life-threatening condition that virtually always presents with the abrupt onset of acute severe abdominal pain that is typically out of proportion to a relatively benign physical exam. Acute mesenteric ischemia usually occurs in patients with risk factors of systemic embolization (eg, atrial fibrillation) or arterial thrombosis. Unexplained metabolic acidosis can be an important clue.

Disease Highlights

A. Etiology: Usually due to superior mesenteric artery or celiac artery embolism (50%). Other causes include thrombosis (15–25%), low flow states without obstruction (15–30%) (nonobstructive mesenteric ischemia), and mesenteric venous thrombosis (5%).

1. Embolism
    - **a.** Risk factors include atrial fibrillation, acute myocardial infarction, valvular heart disease, heart failure, ventricular aneurysms, angiography of abdominal aorta, and hypercoagulable states.
    - **b.** The onset is often sudden without prior symptoms.
2. Thrombosis
    - **a.** Usually occurs in patients with atherosclerotic disease of the involved artery.
    - **b.** Approximately half of such patients have a prior history of chronic mesenteric ischemia with intestinal angina.
3. Nonobstructive mesenteric ischemia
    - **a.** May have an insidious onset
    - **b.** Often occurs in elderly patients with mesenteric atherosclerotic disease and superimposed hypotension (due to myocardial infarction, heart failure, dialysis, or sepsis). Alpha-agonists, digoxin, and beta-blockers may also increase the risk of nonobstructive mesenteric ischemia.
    - **c.** Also seen in critically ill patients after cardiopulmonary bypass or other major surgery
    - **d.** Other causes include cocaine use and following endurance exercise activities (eg, marathon, cycling).
4. Mesenteric venous thrombosis is often secondary to portal hypertension, hypercoagulable states, and intra-abdominal inflammation.

B. Patients have acute abdominal pain that is often out of proportion to their abdominal exam. If left untreated, bowel infarction and peritoneal findings will develop.

C. Incidence: 0.1–0.3% of hospital admissions, 1% of patients presenting with abdominal pain, and up to 10% of patients > 70-years-old presenting with abdominal pain.

D. Mortality is high and increases with delay in treatment.

Evidence-Based Diagnosis

A. Abdominal pain out of proportion to exam is a classic finding but is absent in 20–25%. Other

common presenting symptoms are vomiting (71%) and diarrhea (42%).

B. 50% of patients have a prior history of intestinal angina.

C. Laboratory studies are nonspecific.

1. The WBC is abnormal in 90% of patients and often markedly elevated (mean WBC 21.4 $\times 10^9$/mL).
2. Lactate level has a sensitivity of 86% and specificity of 44%; LR+, 1.5; LR–, 0.32

A normal lactate level does not rule out acute mesenteric ischemia.

D. Standard CT scanning is insensitive for acute mesenteric ischemia (64%). It *may* demonstrate superior mesenteric artery occlusion or findings suggesting ischemic and necrosis such as segmental bowel wall thickening or pneumatosis. One study reported 100% sensitivity but patients were studied 3 days after symptom onset, when infarction may have been easier to demonstrate.

E. CT angiography is very accurate (93.3% sensitive, 95.9% specific), rapidly available and fast. It is the initial study prior to angiography. Magnetic resonance angiography has also been used.

Routine CT scanning may not diagnose AMI. CTA is required.

F. Catheter angiography is the gold standard and can also be therapeutic. However, it is invasive, time consuming, and may not be available on an emergent basis.

G. Doppler ultrasonography is insensitive due to bowel distention.

Treatment

A. Emergent revascularization (via thromboembolectomy, thrombolysis, vascular bypass, or angioplasty) and surgical resection of necrotic bowel are the mainstays of therapy. Prompt surgical intervention (< 12 hours) reduces mortality compared with delayed intervention (> 12 hours) (14% vs 75%).

B. Broad-spectrum antibiotics

C. Volume resuscitation

D. Preoperative and postoperative anticoagulation to prevent thrombus propagation

E. For patients with nonobstructive mesenteric ischemia, improved perfusion is paramount.

F. Intra-arterial papaverine improves mesenteric blood flow by reducing reactive mesenteric

arteriolar vasoconstriction.

2. Ischemic Colitis

Textbook Presentation

Ischemic colitis typically presents with left-sided abdominal pain. Patients frequently have bloody or maroon stools or diarrhea. Profuse bleeding is unusual.

Disease Highlights

A. The most common form of intestinal ischemia

B. Usually due to nonocclusive decrease in colonic perfusion

C. Distribution

1. Typically involves the watershed areas of the colon, most commonly the splenic flexure, descending colon, and rectosigmoid junction.
2. Right colonic involvement occurs occasionally.
3. Rectal involvement is rare and suggests other diseases.

D. Precipitating events may include hypotension, myocardial infarction, sepsis, heart failure, or cardiac or aortic surgery but is usually not identified.

E. Uncommon causes include

1. Vasculitis
2. Hypercoagulable states
3. Vasoconstrictors
4. Cocaine
5. Vascular surgery
6. Drugs (eg, alosetron)
7. Long-distance running or bicycling (presumably due to shunting and hypoperfusion)

Evidence-Based Diagnosis

A. Abdominal pain (not usually severe) is reported by 68–84% of patients.

B. Hematochezia is a helpful diagnostic clue when present but not when absent. Sensitivity, 46%; specificity, 90.9%; LR+, 5.1; LR−, 0.6

C. Diarrhea is seen in approximately 40% of patients.

D. Abdominal tenderness is common (81%), but rebound tenderness is rare (15%).

E. Risk factors that increase the likelihood of ischemic colitis include

1. Age > 60 years
2. Hemodialysis
3. Cardiovascular disease
4. Hypertension
5. Diabetes mellitus
6. Hypoalbuminemia
7. Medications that induce constipation

F. Features that distinguish acute mesenteric ischemia (small bowel) from ischemic colitis are summarized in Table 3-13.

Table 3-13. Features that distinguish ischemic colitis from acute mesenteric ischemia.

Ischemic Colitis	Acute Mesenteric Ischemia
Usually due to nonocclusive decrease in colonic perfusion	Usually due to acute arterial occlusion of SMA or celiac artery
Precipitating cause often not identified	Precipitating cause typical (MI, atrial fibrillation etc.)
Patients are usually not severely ill	Patients appear severely ill
Abdominal pain usually mild	Abdominal pain usually severe
Abdominal tenderness usually present	Abdominal tenderness not prominent early
Hematochezia common	Hematochezia uncommon until very late
Colonoscopy procedure of choice, angiography not usually indicated	Angiography indicated

MI, myocardial infarction; SMA, superior mesenteric artery.

G. Colonoscopy (without preparation) is the preferred test to evaluate ischemic colitis.

H. Ultrasound may show segmental circumferential thickening of a long segment (> 10 cm) of the splenic flexure or sigmoid with sudden transition from abnormal to normal areas. Color flow is absent or greatly diminished in 80% of cases, helping distinguish this from IBD in which flow is increased.

I. CT scan may demonstrate segmental circumferential wall thickening (which is nonspecific) or be normal.

J. Vascular studies are usually normal and not indicated except in the unusual case of isolated right-sided ischemic colitis.

Treatment

A. Therapy is primarily supportive, with bowel rest, IV hydration, and broad-spectrum

antibiotics.

B. Colonic infarction occurs in a small percentage of patients (15–20%) and requires segmental resection.

C. Indications for surgery include peritonitis, sepsis, free air on plain radiographs, clinical deterioration (persistent fever, increasing leukocytosis, lactic acidosis), or strictures.

CASE RESOLUTION

The abdominal CT scan reveals marked dilatation proximal to a transition point in the sigmoid colon with collapse distally. There is a suggestion of a mass at this location. Mr. J underwent surgical exploration, which confirmed an obstructing colonic mass. The mass was resected and a colostomy created. Pathologic evaluation revealed adenocarcinoma of the colon.

CHIEF COMPLAINT

PATIENT 

Mr. L is a 65-year-old man who arrives in the emergency department complaining of 1 hour of excruciating constant diffuse periumbilical abdominal pain radiating to his left flank. He has never had pain like this before. He has suffered 1 episode of vomiting and feels light headed. The emesis was yellow. He has moved his bowels once this morning and continues to pass flatus. There is no change in his bowel habits, melena, or hematochezia. Nothing seems to make the pain better or worse. He was without any pain until this morning. His past medical history is remarkable for hypertension and tobacco use and appendectomy at age 12. On physical exam, he is diaphoretic and in obvious acute distress. Vital signs are BP, 110/65 mm Hg; pulse, 90 bpm; temperature, 37.0°C; RR, 20 breaths per minute. HEENT, cardiac, and pulmonary exams are all within normal limits. Abdominal exam reveals moderate diffuse tenderness, without rebound or guarding. Bowel sounds are present and hypoactive. Stool is guaiac negative.

At this point, what is the leading hypothesis, what are the active alternatives, and is there a must not miss diagnosis? Given this differential diagnosis, what tests should be ordered?

RANKING THE DIFFERENTIAL DIAGNOSIS

Mr. L has severe, diffuse, and acute abdominal pain. The first pivotal point evaluates the location of the pain. Mr. L's pain is diffuse, which limits the differential diagnosis. Common causes of diffuse mid abdominal pain include IBS; IBD; SBO or LBO; acute ischemia; AAA; diabetic ketoacidosis; gastroenteritis; and, given the radiation of pain to his back, pancreatitis, and nephrolithiasis (Figure 3-1). Although this is an extensive differential, it can be focused further. Several of these diagnoses are very unlikely and need not be considered further. Given the lack of diarrhea and the severity of pain, gastroenteritis is very unlikely. His continued bowel movements and flatus make bowel obstruction unlikely (although this can be seen early in obstruction.) The lack of a history of diabetes would make diabetic ketoacidosis unlikely, unless this was the initial presentation; a simple blood sugar could help exclude this diagnosis. The second pivotal point which can serve to narrow the differential diagnosis is the time course of the pain, which is hyperacute. Of the remaining hypotheses, AAA, bowel ischemia, pancreatitis, and nephrolithiasis can all present acutely, whereas this would be very unlikely presentation of IBD or IBS. The radiation to the left flank increases the likelihood of AAA, nephrolithiasis, and pancreatitis. Clearly, AAA is a must not miss diagnosis. Other pivotal findings that can help narrow the differential diagnosis include peritoneal findings on exam, unexplained hypotension and abdominal distention. Table 3-14 lists the differential diagnoses for Mr. L.

Table 3-14. Diagnostic hypotheses for Mr. L.

Diagnostic Hypotheses	Clinical Clues	Important Tests
Leading Hypothesis		
AAA	Orthostatic hypotension Pulsatile abdominal mass Decreased lower extremity pulses	Abdominal CT scan
Active Alternatives—Most Common		
Renal colic	Flank pain Radiation to groin Hematuria Costovertebral angle tenderness	Urinalysis Renal CT
Pancreatitis	Alcohol abuse Gallstones	Serum lipase
Bowel ischemia: Acute mesenteric ischemia	Atrial fibrillation, valvular heart disease, heart failure, hypercoagulable state Abrupt onset pain Pain out of proportion to exam	CT angiography
Bowel ischemia: Ischemic colitis	Age > 60, vascular disease, hypotension (due to MI, sepsis), hematochezia, diarrhea	Colonoscopy

Exploring each one of the hypotheses, Mr. L has no history of kidney stones or hematuria. He does not drink alcohol. He has no history of atrial fibrillation, valvular heart disease, or hypercoagulable states. On reexamination, orthostatic maneuvers reveal profound orthostatic hypotension. Supine BP and pulse were 110/65 mm Hg and 90 bpm. Upon standing his BP falls to 65/40 mm Hg and a pulse of 140 bpm. He remains afebrile. Again, you find that he lacks rebound or guarding and has moderate flank and back tenderness to percussion. His abdominal aorta cannot be palpated due to his abdominal girth. Lower extremity pulses are

intact. Plain abdominal radiographs do not demonstrate free air.

Is the clinical information sufficient to make a diagnosis? If not, what other information do you need?

The most dramatic and important pivotal physical finding is the presence of profound orthostatic hypotension. It is critical to appreciate that his hypotension is clearly out of proportion to dehydration since he has only vomited once and his decreased oral intake only began 1 hour ago. As noted above *unexplained* hypotension is a pivotal finding that must be explained. The lack of dehydration suggests that the orthostatic hypotension is due to either intra-abdominal hemorrhage or sepsis (Table 3-3). He has no fever or chills to suggest sepsis, but this is still possible. A serious consideration is therefore massive intra-abdominal hemorrhage, either within the gastrointestinal tract or intraperitoneal. Massive gastrointestinal hemorrhage always exits the bowel quickly resulting in obvious clinical findings, such as hematemesis, melena, or hematochezia, and is rarely subtle. Therefore, you are more concerned about intraperitoneal hemorrhage. Causes of massive intraperitoneal hemorrhage include AAA rupture, splenic rupture, or rupture of an ectopic pregnancy. The patient's history is most suggestive of AAA rupture. You call for a stat vascular surgery consult.

Orthostatic hypotension is always important. It significantly influences the differential diagnosis and the diagnostic and management decisions, and it may be marked despite a *normal supine* BP and pulse.

Leading Hypothesis: AAA

Textbook Presentation

Classically, patients are men with a history of smoking who have the triad of severe abdominal pain, a pulsatile abdominal mass, and hypotension.

Disease Highlights

A. Defined as an external diameter of the infrarenal abdominal aorta of ≥ 3 cm.

B. The lifetime risk of AAA in patients age 45 years through 85 years is 5.6%. The highest lifetime risk of AAA is 17% in white male current smokers.

C. 10,000 deaths per year in the United States

D. Risk factors

1. Smoking is the most significant risk factor (OR 5).
2. Men are affected 4 to 5 times more often than women.
3. Family history of AAA (OR 4.3)
4. Increased age
5. Hypertension (OR 1.2)

E. Presentation

1. Ruptured AAA
   - **a.** Mortality with rupture is 81%. Approximately 32% die before reaching a hospital.
   - **b.** Misdiagnosis occurs in 16% of cases. (Most common misdiagnosis is renal colic.)
   - **c.** Rupture into the duodenum is a rare complication, is more common in patients with prior AAA graft, and may result in gastrointestinal bleeding over weeks.
2. Symptomatic, contained
   - **a.** Rarely, patients present nonemergently with symptomatic contained rupture of the abdominal aorta.
   - **b.** Symptoms are primarily secondary to retroperitoneal hemorrhage and are occasionally present for weeks or even several years.
3. Inflammatory AAA
   - **a.** Comprise about 5–10% of AAAs and usually occurs at a slightly younger age
   - **b.** Typical features include chronic abdominal pain and weight loss.
   - **c.** Distinguishing characteristic is marked inflammation of aortic adventitia.
4. Limb ischemia
   - **a.** AAA can present when intraluminal thrombus within the aneurysmal sac embolizes, leading to limb ischemia.
   - **b.** Patients may present with blue toes from the embolization; this can occur with or without rupture.
5. Asymptomatic AAA (discovered incidentally or on screening)
   - **a.** The risk of rupture increases markedly as the diameter of the aneurysm increases.
   - **b.** See Table 3-15.

Table 3-15. Annual rupture rate in AAA.

AAA Diameter (cm)	Rupture Risk (%)
3.0–3.9	0
4.0–4.9	1
5.0–5.9	1–11
6.0–6.9	10–22
> 7.0	30–33

Data from Moll FL, Powell JT et al. Management of Abdominal Aortic Aneurysms Clinical Practice Guidelines of the European Society for Vascular Surgery. Eur J Vasc Endovasc Surg. 2011;Jan;41(41 Suppl 1):S1–58.

Evidence-Based Diagnosis

A. Ruptured AAA

1. Abdominal pain, distention, a large abdominal girth, and rupture all limit sensitivity of finding a pulsatile mass in patients with a ruptured AAA.
   - **a.** Sensitivity, 22–68%; specificity, 75–99%
   - **b.** LR+, 8.0; LR–, 0.6
2. Hypotension is a late finding
3. 50% of patients will not have the triad of hypotension, back pain, and a palpable mass.

Both hypotension and a palpable mass are *unusual* in patients with a ruptured AAA.

4. Bruits do not contribute to diagnosis.
5. Syncope may be present.

B. *Symptomatic contained* AAA

1. Abdominal pain 83%
2. Flank or back pain 61–66%
3. Syncope 26%
4. Abdominal mass on careful exam 52% (only 18% had abdominal mass noted on routine abdominal exam)

5. Hypotension or orthostasis 48%
6. Leukocytosis (> 11,000/mcL) 70%
7. Anemia (unusual)

C. *Inflammatory* AAA

1. Back pain or abdominal pain is usual presentation (80% of patients); rupture is rarely presenting manifestation.
2. Symptoms of inflammation (fever, weight loss) present in 20–50% of patients.
3. Erythrocyte sedimentation rate is elevated in 40–90% of cases.
4. CT or MRI reveal the aneurysm and marked thickening of the aortic wall. Periaortic fat stranding may be seen.

D. Asymptomatic AAA

1. Sensitivity of *focused* exam for *asymptomatic* AAA is poor overall (31–39%) and only 82% among patients with large AAA ≥ 5 cm.
2. The sensitivity of the physical exam is less in obese patients (53% in patients with waist circumference > 39 inches vs. 91% < 39 inches).

E. Imaging

1. CT angiography is very accurate for ruptured AAA: sensitivity, 98.3%; specificity, 94.9%; LR+, 19.3; LR–, 0.02
2. Bedside emergency ultrasound is accurate in symptomatic patients: sensitivity, 99%; specificity, 98%; LR+, 44.5; LR–, 0.01
3. For screening, ultrasound is preferred: sensitivity, 95%; specificity, 100%.
4. CT angiography is typically used for preoperative evaluation prior to repair of *asymptomatic* AAA.

Treatment

A. For ruptured AAA, proceed directly to the operating room.

B. Screening: See Chapter 2, Screening and Health Maintenance

C. Timing of elective surgery

1. Mortality with rupture exceeds 80%. Surgery is performed to minimize the risk of rupture.
2. The risk of rupture increases substantially with increasing AAA diameter.
3. The standard recommendation is to electively repair AAAs ≥ 5.5 cm or those that have increased in size by ≥ 1 cm in 1 year.
4. Other risk factors for rupture include smoking (hazard ratio 2.0, 1.3–3.1) and female sex (hazard ratio 3.8, 2.6–5.5). Hypertension also increases the risk.
5. Some authorities recommend repair in women when the AAA reaches 5.2 cm.

D. Options include open surgical repair or endovascular aneurysm repair (EVAR). Comparing open surgery to EVAR:

1. EVAR is more commonly performed and has a lower 30-day morbidity and mortality

compared to open surgery, but no significant difference in overall survival.

2. Open surgical repair is recommended for patients who cannot comply with periodic long-term surveillance or with aneurysms not amenable to EVAR.

E. Surveillance of small AAA (3.0–5.4 cm)

1. Patients should undergo surveillance by repeat ultrasonography every 3–12 months, with surgical referral for growth > 1.0 cm per year or a total diameter ≥ 5.5 cm.
2. Ultrasound may underestimate the diameter of a AAA. One study reported that 73% of patients found to have an AAA of 5–5.4 cm on ultrasound had an aneurysm that was ≥ 5.5 cm on CT.
3. AAA in females should be monitored by aneurysm diameter *indexed to body size*, as this is more predictive of rupture than the absolute diameter.

F. Nonsurgical management

1. Smoking cessation has been shown to slow AAA growth.
2. No medication definitively reduces AAA growth.
3. AAAs are considered a coronary equivalent. Aspirin and statins are recommended.

MAKING A DIAGNOSIS

Further evaluation (eg, with CT or ultrasonography) at this point depends on the index of suspicion. An emergent bedside ultrasound is the preferred test in this situation. If unavailable, and AAA is very likely and the patient unstable, many vascular surgeons proceed directly to the operating room without further studies in order to avoid the potential lethal delay of obtaining a CT scan. If AAA is less likely and the patient is stable, CT scanning is appropriate.

Have you crossed a diagnostic threshold for the leading hypothesis, AAA? Have you ruled out the active alternatives? Do other tests need to be done to exclude the alternative diagnoses?

Alternative Diagnosis: Nephrolithiasis

Textbook Presentation

Patients typically experience a rapid onset of excruciating back and flank pain, which may radiate to the abdomen or groin. The intensity of the pain is often dramatic as patients writhe and move about constantly (unlike peritonitis) in an unsuccessful attempt to get comfortable. The pain may be associated with nausea, vomiting, dysuria, or urinary frequency.

Abdominal *tenderness* is unusual in patients with nephrolithiasis and should raise doubt about the diagnosis.

Disease Highlights

A. Incidence: Symptomatic stones develop in 7–13% of people in the United States.

1. 35–50% recurrence at 5 years
2. Men affected 2–3 times more often than women
3. Positive family history increases the risk (relative risk 2.6)

B. Etiology

1. Calcium oxalate stones 75%
2. Calcium phosphate stones ($CaPO_4$) 5%
3. Uric acid stones 5–10%
4. Struvite stones ($MgNH_4PO_4$) 5–15%
5. Other: cystine and indinavir stones

C. Pathophysiology

1. Stones form when the concentration of salts (ie, calcium, oxalate, or uric acid) becomes supersaturated in the urine resulting in precipitation and crystallization.
2. Supersaturation is secondary to a combination of increased urinary salt excretion combined with inadequate diluting urinary volume. Numerous mechanisms can contribute to an increase in urinary mineral excretion including:
   - **a.** Calcium: idiopathic hypercalciuria, hypercalcemic disorders, primary hyperparathyroidism, immobilization, excessive sodium intake (which increases calcium excretion), systemic acidosis, and excessive vitamin D supplementation
   - **b.** Uric acid: Excessive dietary purines, myeloproliferative disorders, uricosuric agents (for the treatment of gout), and metabolic syndrome. Low urine pH also contributes to uric acid stone formation. Hyperuricosuria can lead to uric acid stones or calcium stones due to heterogeneous ossification.
   - **c.** Oxalate: Increased excretion may be secondary to excessive oxalate intake (rhubarb, spinach, chocolate, nuts, vitamin C) and/or increased oxalate absorption.
     - **(1)** Fat malabsorption *increases* oxalate absorption. The unabsorbed fat competes

with oxalate to bind calcium leading to more intraluminal oxalate that is not bound to calcium. Unbound oxalate is absorbed and excreted in the urine.

(2) Causes of fat malabsorption include short bowel syndrome, IBD, celiac disease, and bariatric surgery.

3. In some patients, a decrease in urinary stone inhibitors (urinary citrate) also contribute to stone formation.
4. Infection with urea-splitting organisms (ie, *Proteus*) plays a key role in the formation of struvite stones ($MgNH_4PO_4$).
5. Renal colic develops when stones dislodge from the kidney and obstruct urinary flow.

D. Complications

1. Ureteral obstruction
2. Pyelonephritis
3. Sepsis
4. Acute kidney injury is rare, occurring in patients with bilateral obstruction or obstruction of a solitary functioning kidney.

Evidence-Based Diagnosis

A. The evaluation is directed at establishing the diagnosis of nephrolithiasis *and* its underlying etiology so that measures to prevent its recurrence can be implemented.

B. Establishing the diagnosis

1. Gross or microscopic hematuria is neither very sensitive nor specific for symptomatic nephrolithiasis. Sensitivity, 80%; specificity, 41%; LR+, 1.4; LR–, 0.49

The absence of hematuria does not rule out nephrolithiasis.

2. Radiographs (kidneys, ureters, bladder) or ultrasound are not sufficiently sensitive to rule out nephrolithiasis (sensitivity 29–68% and 32–57%, respectively).
3. Noncontrast renal CT is the test of choice.
   a. Sensitivity, 95%; specificity, 98%; LR+, 48; LR−, 0.05
   b. Importantly, CT scan revealed alternative diagnoses in 33% of patients in whom nephrolithiasis was clinically suspected.
1. In pregnant women, ultrasonography is the test of choice.

C. Evaluation of documented nephrolithiasis

1. All patients should have a urinalysis and culture and basic serum chemistries, including several measurements of serum calcium. Urine culture, pH, and chemical analysis of any retrieved stones are also recommended.
2. A more comprehensive evaluation, including several 24-hour urine specimens for

analysis of calcium, oxalate, uric acid, sodium, creatinine, and citrate as well as submission of retrieved stones for chemical analysis, is recommended for patients with recurrent stones. Some experts recommend this for patients with their first stone.

Treatment

A. Pain control

1. NSAIDs
   - **a.** Treat pain and diminish spasm
   - **b.** To be avoided 3 days before lithotripsy due to antiplatelet effects
2. Opioids are second line.

B. Hydration (oral if tolerated, otherwise IV)

C. Hospitalization indicated for uncontrolled pain, persistent nausea or vomiting, acute kidney injury, or signs of infection.

D. Sepsis or acute kidney injury due to bilateral obstruction or unilateral obstruction in a solitary kidney

1. Necessitates emergent drainage (via percutaneous nephrostomy tube or ureteral stent)
2. For sepsis, broad-spectrum IV antibiotics to cover gram-negative organisms and enterococcus should also be administered.

E. Stone passage

1. Nifedipine and alpha-blockers have been shown to significantly increase the likelihood of stone passage.
2. Lithotripsy or ureteroscopy can be used to remove persistent ureteral stones.

F. Secondary prevention

1. Patients with a single calcium stone:
   - **a.** Increasing fluid intake to ≥ 2 L/day decreases the risk of recurrent stones (relative risk, 0.45; 0.24–0.84).
   - **b.** Reducing phosphate-containing soft drinks may decrease recurrences (relative risk, 0.83; 0.71–0.98).
2. For patients with recurrent idiopathic calcium stones other options include:
   - **a.** Thiazide diuretics decrease urinary calcium excretion (especially when combined with potassium supplementation) (relative risk, 0.52, 0.39–0.69).
   - **b.** Citrate supplementation is effective in patients with and without hypocitraturia (relative risk, 0.25, 0.14–0.44).
   - **c.** Allopurinol in patients with concomitant hyperuricemia or hyperuricosuria (relative risk, 0.59, 0.42–0.84).
3. More specific management (ie, dietary modification) is complex and depends on the underlying etiology of the patient’s nephrolithiasis.

CASE RESOLUTION

An emergent bedside ultrasound reveals a massively dilated aorta (9 cm) with 500 mcL of adjacent fluid (presumed blood). Emergent surgery reveals a leaking AAA that ruptures intraoperatively. The aorta is cross clamped, repaired, and the patient is stabilized.

REVIEW OF OTHER IMPORTANT DISEASES

Irritable Bowel Syndrome (IBS)

Textbook Presentation

Patients often complain of intermittent abdominal pain accompanied by diarrhea or constipation or both of *years'* duration. The diarrhea is often accompanied by cramps that are relieved with defecation. Pain cannot be explained by structural or biochemical abnormalities. Weight loss or anemia should alert the clinician to other possibilities.

New persistent *changes* in bowel habits (either diarrhea or constipation) should be thoroughly evaluated to exclude colon cancer, IBD, or another process. An assumption of IBS in such patients is inappropriate.

Disease Highlights

A. Affects 8.9% of men and 14% of women.

B. Etiology is a combination of altered motility, visceral hypersensitivity, autonomic dysfunction, and psychological factors.

C. Symptoms are often exacerbated by psychological or physical stressors.

D. Patients may have pain associated primarily with diarrhea (IBS-D), constipation (IBS-C) or a mixed bowel pattern (IBS-M).

Evidence-Based Diagnosis

A. There are no known biochemical or structural markers for IBS.

B. A variety of symptoms are common in patients with IBS including lower abdominal pain, passage of mucous, feeling of incomplete evacuation, loose or frequent stools at onset of pain, and pain relieved by defecation. However, none of these are very predictive (LR+, 1.3–2.1; LR–, 0.59–0.88).

C. Only abdominal pain is very sensitive (sensitivity, 90%; specificity, 32%) and is also required by the criteria.

D. Diarrhea pattern

1. One study suggested that patients with diarrhea-predominant IBS were more likely to have irregularly irregular diarrhea that fluctuated over days whereas patients with inflammatory diseases (IBD and celiac disease) were more likely to have persistent diarrhea that fluctuates over months.
2. Persistent diarrhea increased the likelihood of IBD (LR+, 4.2).
3. A more extensive work-up may be indicated in patients with persistent, constant diarrhea.

E. The diagnosis is usually made by a combination of (1) a consistent history, (2) the absence of alarm features, and (3) a limited work-up to exclude other diseases.

1. Consistent history

a. Although a variety of criteria have been developed (ie, Rome criteria), a recent review by the American College of Gastroenterology suggested a consistent history was abdominal pain or discomfort that occurs in association with altered bowel habits for at least 3 months.

b. Patients may also report a relief of pain with defecation.

2. Alarm symptoms (suggest alternative diagnosis and necessitate evaluation)

a. Positive fecal occult blood test or rectal bleeding

b. Anemia

c. Unintentional and unexplained weight loss

d. Fever

e. Family history of colorectal cancer, IBD, or celiac disease

f. Recent antibiotic use

3. Limited work-up

a. A CBC is appropriate to rule out anemia, which would suggest alternative diagnoses, and CRP to rule out inflammatory diseases.

b. Other diagnostic testing is not recommended for young patients without alarm features, with the exception of serologic testing for celiac disease in patients with IBS-D.

(1) Although recommended by the American College of Gastroenterology, one study found the incidence of confirmed celiac disease in IBS-D patients (without alarm features) to be very low (0.41%) and not different from asymptomatic patients.

(2) May best be reserved for the subset of IBS-D patients with other possible clues suggesting celiac disease (eg, anemia, autoimmune disease, or family history)

c. Lactose hydrogen breath testing can be considered if lactose intolerance remains a concern despite dietary lactose restriction.

d. There is no evidence that routine flexible sigmoidoscopy or colonoscopy is necessary in young patients without alarm symptoms.

e. Colonoscopy is recommended in patients with alarm symptoms and in those aged ≥ 50 years (if not already performed). Biopsy is also recommended in patients with IBS-D to rule out microscopic colitis.

f. The following should also be evaluated in patients with alarm symptoms:

(1) Stool for occult blood

(2) Thyroid-stimulating hormone levels

(3) Basic chemistries

(4) Stool for *Clostridium difficile* toxin and presence of ova and parasites

(5) A variety of serum and fecal markers, including ASCA, pANCA, fecal calprotectin, and fecal lactoferrin, are useful in selected patients and can suggest bowel inflammation or IBD.

Treatment

A. Nonspecific management. A variety of treatments have been shown to be effective in IBS including:

1. Eliminate foods which cause gas and aggravate symptoms such as lactose, beans, cabbage, onions, Brussels sprouts, cauliflower, and broccoli.
2. Physical activity
3. Antispasmodics (including hyoscine and peppermint oil) help decrease abdominal pain.
4. Tricyclic antidepressants and selective serotonin reuptake inhibitors may reduce persistent symptoms.
5. Dietary fiber (psyllium) may be effective. Studies are conflicting.

B. Specific therapy is based on predominant syndrome.

1. Diarrhea-predominant IBS (IBS-D)
   - **a.** Patients with IBS-D should have a trial of lactose-free diet. Such treatment in lactase-deficient individuals with IBS markedly reduces outpatient visits.
   - **b.** Loperamide reduces diarrhea (but not abdominal pain or bloating).
   - **c.** A single, short course of rifaximin (a nonabsorbed antibiotic) is helpful.
   - **d.** Alosetron is a $5HT_3$-receptor antagonist whose use is restricted to a risk management program by the US Food and Drug Administration due to the risk of ischemic colitis. Expert consultation recommended.
   - **e.** Probiotic treatment with *Bifidobacteria* may improve symptoms.
2. Constipation predominant IBS (IBS-C)
   - **a.** Lubiprostone
     - **(1)** A selective C-2 chloride channel activator is more effective than placebo in women.
     - **(2)** Premenopausal women should have a negative pregnancy test before starting the medication and maintain contraception while taking lubiprostone.
     - **(3)** Not yet recommended in men

Alternative Diagnosis: Diverticulitis

Textbook Presentation

Patients typically complain of a constant gradually increasing left lower quadrant abdominal pain, usually present for several days. Diarrhea or constipation and fever are often present. Guarding and rebound may be seen.

Disease Highlights

A. Diverticula are outpouchings of the colonic wall that may be asymptomatic (diverticulosis), become inflamed (diverticulitis), or hemorrhage.

B. Diverticulosis

1. Develops in 5–10% of patients aged > 45 years, 50% in persons aged > 60 years, and 80% in those aged > 85 years.
2. Low-fiber diets are believed to cause diverticula by decreasing stool bulk, resulting in increased intraluminal pressure causing the mucosa and submucosa to herniate through weakness in the colonic wall created by penetrating vessels.

C. Diverticulitis

1. Develops secondary to microscopic or frank perforation of diverticula.
2. 85–95% of diverticulitis occurs in sigmoid or descending colon
3. Mean age of onset is 63 years.
4. Complications of diverticulitis
   - **a.** Abscess
   - **b.** Peritonitis
   - **c.** Sepsis
   - **d.** Colonic obstruction
   - **e.** Fistula formation (colovesicular fistula most common)
5. Simultaneous diverticular hemorrhage and *diverticulitis* are unusual; diverticular hemorrhage is discussed in Chapter 19, GI Bleeding.

Evidence-Based Diagnosis

A. Left lower quadrant tenderness increases the likelihood of diverticulitis; LR+, 3.4; LR–, 0.41.

B. Neither fever nor leukocytosis is very sensitive for diverticulitis or diverticular abscess.

1. In patients with uncomplicated diverticulitis, only 45% had temperature of ≥ 38.0°C or WBC > 11,000/mcL.
2. In patients with diverticular abscess, only 64% of patients had temperature of ≥ 38.0°C and 62% had WBC > 11,000/mcL.

C. Plain radiographs may demonstrate free air or obstruction.

D. CT scan

1. Test of choice in men and nonpregnant women
2. Can confirm diverticulitis (diverticula with thickened bowel wall or pericolonic fat

stranding); evaluate the extent, severity, and complications (abscess formation and perforation); and diagnose other conditions.

3. 93–97% sensitive

E. Colonoscopy

1. Colon cancer can be mistaken for diverticulitis on CT.
2. All patients with diverticulitis should have follow-up colonoscopy (unless one has recently been performed) delayed 4–6 weeks until there is a resolution of acute inflammation.

Treatment

A. Outpatient management is appropriate for patients with a mild attack (ie, patients without marked fever or marked leukocytosis, pain manageable with oral analgesics, tolerating oral intake) and without significant comorbidities, immunocompromise, or advanced age.

1. Antibiotics
   - **a.** Antibiotics have routinely been administered to all patients with diverticulitis.
   - **b.** Recent guidelines have questioned the utility of this approach.
   - **c.** A recent randomized controlled trial of antibiotic therapy in *mild* diverticulitis proven by CT failed to demonstrate *statistically* significant improvement with antibiotics over observation.
   - **d.** However, every outcome measured *favored* antibiotic use over observation; days to recovery 12 versus 14, readmission rate within 6 months 12% vs. 17.6%, total number of readmissions 13.2% vs. 25%, complicated diverticulitis 2.6% vs. 3.8%, ongoing diverticulitis 4.1% vs. 7.3%, sigmoid resection 2.3% vs. 3.8%.
   - **e.** Until further data is available, antibiotic therapy is recommended.
2. Clear liquid diet
3. High-fiber diet after attack resolves
4. Follow-up colonoscopy

B. Moderate to severe attack (unable to tolerate oral intake, more severe pain) necessitates inpatient treatment.

1. Broad-spectrum antibiotics
2. No oral intake
3. CT-guided drainage for abscesses > 5 cm
4. Emergent surgery is recommended in patients with
   - **a.** Peritonitis
   - **b.** Uncontrolled sepsis
   - **c.** Clinical deterioration despite medical management
   - **d.** Obstruction or large abscesses that cannot be drained or are contaminated with frank fecal contents
5. The threshold for surgery should be lower in immunocompromised patients.

6. High-fiber diet once the attack has resolved

Chronic Mesenteric Ischemia

Textbook Presentation

Patients with chronic mesenteric ischemia typically complain of recurrent postprandial abdominal pain (often in the first hour and diminishing 1–2 hours later), food fear, and weight loss. Patients often have a history of tobacco use (75%), peripheral vascular disease (55%), coronary artery disease (43%), or hypertension (37%).

Disease Highlights

A. Usually secondary to near obstructive atherosclerotic disease of the superior mesenteric artery or celiac artery or both.

B. Arterial stenosis results in an imbalance between intestinal oxygen supply and demand that is accentuated after eating leading to intestinal angina resulting in food fear and weight loss.

C. Two or more vessels (ie, superior mesenteric artery *and* celiac artery) are involved in 91% of affected patients.

Evidence-Based Diagnosis

A. Abdominal pain

1. Occurs in 94% and is postprandial in 88%.
2. It is typically epigastric or periumbilical.
3. Typically develops in the first hour after eating and diminishes 1–2 hours later.

B. Weight loss occurs in 78% of patients and is due to food aversion.

C. Diarrhea occurs in 36% of patients.

D. An epigastric bruit has been reported in 63% of patients (17–87%).

E. The abdomen is typically nontender even during a severe episode of pain.

F. Although stenoses are common in older patients (18% of population over age 65 years), symptomatic chronic ischemia is rare, and documented stenosis does *not* confirm the diagnosis of mesenteric ischemia. It is important to exclude more common disorders (ie, PUD and gallstone disease).

G. Duplex ultrasonography is very sensitive (> 90%) and can be used as the initial diagnostic tool. Normal results make the diagnosis very unlikely.

H. CT angiography and magnetic resonance angiography have also been used. Angiography should be considered if the results of noninvasive testing suggest vascular obstruction.

Treatment

Revascularization (by surgical endarterectomy, bypass grafting, or percutaneous endovascular repair) is the only treatment.

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