



Interdisciplinary Coordination in the Diagnosis and Management of Acute Abdomen: Radiological Assessment, Laboratory Correlation, and Administrative Support in Emergency Care

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Abstract

Background: Acute abdominal pain is a common and challenging presentation in emergency medicine, encompassing a wide spectrum of conditions from benign, self-limiting issues to life-threatening surgical emergencies. Its diverse etiologies include inflammatory, obstructive, vascular, and infectious processes, making accurate and timely diagnosis critical.

Aim: This article aims to synthesize an interdisciplinary framework for the diagnosis and management of the acute abdomen, emphasizing the integration of clinical assessment, radiological imaging, laboratory correlation, and administrative support to optimize patient outcomes.

Methods: A comprehensive review of the evaluation process is presented, covering the pathophysiology of abdominal pain, systematic history-taking, and physical examination. The analysis details the strategic use of diagnostic tools, including point-of-care ultrasound, laboratory tests, and advanced cross-sectional imaging like CT scans, tailored to the pain's location and patient-specific risk factors.

Results: Successful management hinges on a structured, team-based approach. Rapid resuscitation, timely analgesia, and early surgical consultation are paramount for unstable patients. Diagnostic accuracy is greatly enhanced by correlating clinical findings with targeted imaging and lab results, allowing for the swift identification of time-sensitive conditions such as mesenteric ischemia, ruptured aortic aneurysm, and appendicitis.

Conclusion: The effective management of acute abdominal pain requires a coordinated, multidisciplinary effort. Disciplined clinical evaluation, guided by an understanding of pain mechanisms and supported by appropriate diagnostics, ensures that critical conditions are rapidly identified and treated, thereby reducing morbidity and mortality.

Keywords: Acute Abdomen, Emergency Medicine, Diagnostic Imaging, Interdisciplinary Care, Surgical Emergency, Clinical Assessment.

1. Introduction

Acute abdominal pain is among the most frequent reasons for presentation to emergency departments and spans a spectrum from self-limited conditions to immediately life-threatening disease. The term “acute abdomen” denotes a clinical constellation in which rapid diagnostic triage and, at times, operative management are necessary to avert morbidity and mortality. Etiologies are diverse and

encompass infectious and inflammatory processes, vascular catastrophes such as mesenteric occlusion or aortic dissection, mechanical obstruction, and hollow-viscus perforation; less obvious sources include occult neoplasia or rupture of a previously undiagnosed viscus, each of which may initially manifest with non-specific symptomatology [1][2][3]. Patients typically describe abrupt pain onset, variably accompanied by fever, nausea, emesis, or progressive distension, yet

the initial clinical picture can be confounded by analgesic use, age-related hypoalgesia, or immunosuppression, all of which may blunt classic findings and delay recognition [4]. High-quality evaluation begins with a meticulous history that characterizes the pain's location, radiation, temporal pattern, quality, and intensity, and explores precipitating and palliating factors alongside systemic and gastrointestinal review. This history frames a hypothesis-driven examination that must, at minimum, include vital sign assessment and a complete abdominal examination—inspection for distension or scars, auscultation for bowel sounds, percussion for tympany or shifting dullness, and gentle then deep palpation to elicit focal tenderness, guarding, or rebound. The remainder of the physical examination is tailored by the history to detect extra-abdominal sources or consequences, such as cardiopulmonary pathology, hernias, or genitourinary disease [5][6]. In its most severe form, the acute abdomen is signaled by systemic toxicity or shock and peritoneal irritation; equally important are subtle clues—orthostasis, dehydration, dysuria, emesis pattern, or altered stooling—that refine the differential diagnosis when dramatic peritonitis is absent [5][6].

Early identification of “red flags” mandates escalation. Patients with hemodynamic instability, peritoneal signs, or clinical suspicion for entities such as high-grade bowel obstruction, mesenteric ischemia, gastrointestinal perforation, or ectopic pregnancy require urgent surgical or specialty consultation and expedited diagnostics. Likewise, the triad of fever, jaundice, and right upper quadrant pain warrants prompt assessment for hepatobiliary sepsis. Yet clinicians must not equate the absence of overt distress with low risk: seemingly non-acute presentations still merit systematic inquiry and targeted testing, because natural history and symptom expression vary by age, comorbidity, and anatomic site [7]. Decisions about laboratory evaluation and imaging should be individualized to the evolving problem representation, integrating pretest probability with the risks and benefits of each modality. Crucially, contemporary evidence supports the ethical and clinical imperative that analgesia not be withheld during diagnostic evaluation; appropriate pain control improves patient experience and does not obscure key findings when examination is performed thoughtfully and serially [7].

Etiology:

The acute abdomen represents a syndromic presentation arising from diverse gastrointestinal, genitourinary, vascular, infectious, and gynecologic processes, and accurate etiologic attribution hinges on an iterative synthesis of history, physical examination, laboratory studies, and targeted imaging. Gastrointestinal sources remain the most prevalent and include inflammatory, obstructive, ischemic, and perforating conditions. Classic examples are acute

appendicitis and perforated peptic ulcer, each capable of rapid clinical deterioration if unrecognized; acute pancreatitis and acute cholecystitis, which may present with overlapping epigastric or right upper quadrant pain; and colonic pathology such as diverticulitis or a ruptured diverticulum, where localized peritonitis can evolve to generalized sepsis without timely treatment [1][2][3]. Mechanical catastrophes—including small bowel obstruction and volvulus—manifest with colicky pain, vomiting, distension, and obstipation, while traumatic or spontaneous solid-organ injury, such as a lacerated spleen or liver, may present with peritoneal signs or hemodynamic instability. Mesenteric ischemia and ischemic bowel occupy a particularly lethal niche, with pain often out of proportion to examination and rapid progression to necrosis if revascularization or resection is delayed [1][2][3]. Peritonitis constitutes a common final pathway for many acute abdominal conditions and is most frequently bacterial in origin, arising via translocation, perforation, or direct inoculation. Secondary peritonitis may follow hollow-viscus perforation from ulcer disease, diverticular rupture, or appendiceal perforation, whereas tertiary or persistent peritonitis can complicate postoperative courses or malignancy. Intra-abdominal infection also develops in the setting of pelvic inflammatory disease, cirrhotic ascites with spontaneous bacterial peritonitis, abdominal tuberculosis, traumatic breaches, or device-related contamination from feeding tubes and peritoneal dialysis, each with distinct microbiologic profiles and therapeutic implications [8][9][10]. The clinician's challenge is to differentiate localized from generalized peritoneal irritation and to identify precipitating lesions that demand source control, balancing early resuscitation with prompt imaging and, when indicated, surgical consultation.

Catastrophic vascular events are prototypic causes of the acute abdomen and demand high diagnostic vigilance. Acute mesenteric ischemia—arterial embolic, thrombotic, or nonocclusive—produces severe visceral pain with initially modest tenderness, advancing quickly to bowel infarction without reperfusion or laparotomy, while rupture of an abdominal aortic aneurysm can present with abdominal or back pain, hypotension, and a palpable mass, but may be protean in older or anticoagulated patients [11][12]. Obstetric and gynecologic disorders are similarly time-sensitive: a ruptured ectopic pregnancy threatens hemorrhagic shock; ovarian torsion compromises ovarian viability; and pelvic inflammatory disease generates lower abdominal pain with fever and cervical motion tenderness, occasionally culminating in tubo-ovarian abscess formation if not rapidly treated [13]. Urologic etiologies, notably ureteral colic from obstructing calculi and acute pyelonephritis, can mimic intra-abdominal sources, producing flank-to-groin radiation, costovertebral angle tenderness, and

systemic inflammatory responses that blur traditional anatomic boundaries. Age and developmental context strongly modulate the differential diagnosis. Newborns and infants may present with necrotizing enterocolitis, midgut volvulus from malrotation, or intussusception, entities that can progress precipitously from nonspecific irritability and feeding intolerance to bowel ischemia and perforation without expeditious imaging and intervention [14]. In the broader pediatric population, acute appendicitis remains the most frequent surgical cause of the acute abdomen, yet early presentations can be atypical, necessitating careful serial examinations and judicious use of ultrasound or cross-sectional imaging to minimize diagnostic delay [14]. Across all age groups, clinicians must maintain a broad, hypothesis-driven perspective, continuously updating pretest probabilities as new data emerge, because timely recognition of the specific etiology—whether inflammatory, infectious, obstructive, vascular, or gynecologic—directly determines the need for antibiotics, source control, endovascular or operative therapy, and level of care [5][6][7].

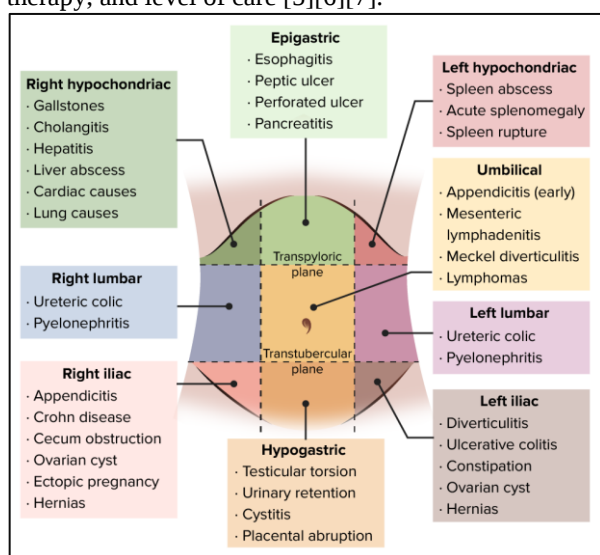


Figure-1: Etiology of acute abdomen.

Epidemiology:

Acute abdominal pain remains one of the most frequent and diagnostically challenging complaints in emergency and primary care medicine. In the United States alone, abdominal pain accounts for 7% to 10% of all emergency department visits, representing an estimated 5 to 10 million patient encounters annually—a burden that continues to rise with population growth and aging demographics [15]. The clinical presentation spans all ages and socioeconomic strata, though etiologic patterns differ markedly between pediatric, adult, and geriatric populations. In pediatric practice, nearly 9% of all primary care visits involve complaints of acute abdominal pain, reflecting the high prevalence of gastrointestinal and genitourinary disorders in younger patients [4]. Recent large-scale retrospective studies

have demonstrated that nonspecific abdominal pain and renal colic together account for approximately 60% of acute abdominal pain diagnoses in emergency settings [16]. These data highlight that, while many patients present with severe discomfort, the majority ultimately have self-limited or conservatively managed conditions. However, a smaller but critical subset of patients harbors life-threatening pathology requiring urgent surgical or interventional treatment, emphasizing the importance of rapid diagnostic triage and multidisciplinary collaboration in emergency departments. Age strongly influences both the incidence and etiology of acute abdominal conditions. In individuals older than 65 years, diverticulitis and cholecystitis are among the most frequent causes, reflecting the cumulative effects of chronic inflammation, gallstone disease, and age-related vascular compromise [15]. Conversely, in younger adults, renal colic and appendicitis are the leading diagnoses, with appendicitis particularly prominent among patients in their second to third decade of life. In this group, acute appendicitis, acute cholecystitis, and acute pancreatitis collectively constitute the majority of acute abdomen presentations [17].

Epidemiologic analyses indicate that approximately 14% of patients presenting with nontraumatic abdominal pain are ultimately diagnosed with an acute abdomen requiring immediate intervention. Interestingly, gender-based differences have been consistently observed: males represent 62.61% of cases, whereas females comprise 37.39%—a disparity that may partly reflect the inclusion of male-predominant conditions such as appendicitis and bowel obstruction, as well as diagnostic overlap in females with gynecologic disorders [17]. In pediatric populations, the distribution of causes varies with developmental stage. Among infants, the most frequent etiologies include incarcerated inguinal hernia and intussusception, both of which can rapidly lead to bowel ischemia if not promptly addressed. In contrast, acute appendicitis predominates in children older than one year, maintaining its status as the leading surgical cause of pediatric acute abdomen [18]. Despite modern imaging techniques, diagnostic delays in children remain a concern, underscoring the importance of heightened clinical vigilance and timely referral. Overall, the epidemiology of the acute abdomen reveals a condition that is both common and clinically heterogeneous, with patterns shaped by age, sex, lifestyle, and comorbidity. While the majority of patients present with benign or nonspecific causes, a significant proportion require emergent imaging, laboratory evaluation, and surgical consultation to prevent morbidity and mortality. This underscores the enduring relevance of comprehensive clinical assessment, evidence-based imaging algorithms, and interprofessional coordination in the effective management of patients presenting with acute abdominal pain [15][16][17][18].

Pathophysiology

The mechanisms underlying pain perception in patients with an acute abdomen are complex, involving intricate interactions among visceral, somatoparietal, and referred pain pathways. Understanding these mechanisms is essential for clinicians, as the quality, localization, and radiation of abdominal pain provide valuable diagnostic clues to the source and nature of pathology. Pain arising from the abdomen reflects the activation of specialized receptors distributed throughout visceral and somatic structures, each with distinct neuroanatomic and physiological features that contribute to characteristic clinical patterns [19].

Visceral Pain Receptors

Visceral pain, the hallmark of early intra-abdominal disease, originates from receptors located within the serosal surfaces, mesentery, intestinal muscle layers, and mucosal linings of hollow organs. The primary mechanical stimulus for visceral pain is stretch, although other stimuli—such as distention, traction, compression, torsion, ischemia, or inflammation—can also activate these receptors. Chemical mediators including prostaglandins, bradykinin, serotonin, and hydrogen ions sensitize these nociceptors, lowering their activation threshold during tissue injury or ischemia. Unlike somatic pain, which is sharply localized, visceral pain is typically diffuse, dull, and poorly localized. This difference arises from the physiology of the afferent fibers involved. Visceral nociceptive signals are transmitted through unmyelinated C-fibers, which conduct impulses slowly and enter the spinal cord bilaterally at multiple levels, resulting in imprecise localization and broad midline perception. The autonomic nervous system also plays an integral role in visceral pain transmission, explaining the frequent association of abdominal pain with autonomic symptoms such as nausea, vomiting, pallor, or diaphoresis. The distribution of visceral pain correlates with embryologic origin and innervation of abdominal organs, leading to characteristic regional pain patterns. Pain from foregut-derived structures—such as the distal esophagus, stomach, proximal duodenum, liver, pancreas, and biliary tree—is typically perceived in the epigastric region. In contrast, midgut-derived structures, including the distal duodenum, jejunum, ileum, and proximal colon, produce pain centered around the periumbilical region. Meanwhile, hindgut-derived structures, such as the distal colon and rectum, refer discomfort to the suprapubic or lower abdominal area [19]. This anatomical mapping helps clinicians localize disease processes based on early pain patterns, before peritoneal irritation leads to more localized somatic pain.

Somatoparietal Pain Receptors

As inflammation or infection progresses to involve the parietal peritoneum, a transition from visceral to somatoparietal pain occurs, marking the

evolution from vague discomfort to sharply localized tenderness. Somatoparietal pain receptors reside in the parietal peritoneum, abdominal musculature, and skin, and are activated primarily by inflammation, stretching, or tearing of these tissues. The pain signals from these receptors are transmitted through myelinated A- δ fibers, which conduct impulses rapidly and synapse with specific dorsal root ganglia at well-defined spinal cord segments. This precise topographic organization allows the brain to accurately localize the pain to a particular quadrant or structure. Clinically, somatoparietal pain is described as sharp, intense, and well localized, and it often corresponds to physical findings such as guarding, rebound tenderness, and rigidity. For example, localized lower quadrant tenderness in appendicitis reflects peritoneal inflammation adjacent to the inflamed appendix, while left lower quadrant pain in diverticulitis indicates parietal irritation from colonic inflammation. The evolution of pain from diffuse visceral discomfort to localized somatic pain often parallels the disease's progression, making this transition a valuable diagnostic marker for conditions that breach the peritoneal cavity or extend inflammation to its parietal layer [19].

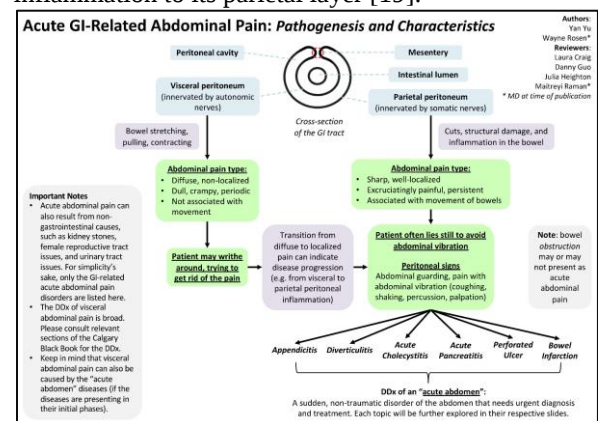


Figure-2: Pathology of acute abdomen.

Referred Pain

Referred pain is another important aspect of abdominal pain physiology and results from convergence of visceral and somatic afferent fibers onto the same spinal neurons within the dorsal horn. Because the brain more frequently interprets input from somatic structures, visceral pain signals entering the same segment are misperceived as originating from somatic areas. This neural overlap explains why pain from intra-abdominal organs is often felt at distant, seemingly unrelated sites. For example, cardiac ischemia—a condition of visceral origin—may manifest as discomfort in the neck, left shoulder, or arm, reflecting shared cervical and thoracic spinal pathways. Similarly, irritation of the diaphragm from gallbladder inflammation (cholecystitis) or subphrenic abscess may cause pain perceived in the right scapular or shoulder region, due to the shared innervation between the phrenic nerve (C3–C5) and the

supraclavicular nerves of the shoulder [19]. The phenomenon of referred pain can complicate diagnosis, as the apparent pain site may not correspond to the anatomical source. However, recognizing common referral patterns remains an essential clinical skill that can guide early diagnostic imaging and intervention.

Integration of Pain Mechanisms in the Acute Abdomen

In many cases of acute abdominal pathology, these mechanisms coexist or evolve sequentially. For instance, in acute appendicitis, early luminal distention produces visceral periumbilical pain, which later shifts to localized somatoparietal pain in the right lower quadrant as the inflammatory process extends to the peritoneum. Similarly, bowel obstruction initially generates diffuse visceral discomfort that may later become sharp and localized with ischemia or perforation. Understanding these transitions allows clinicians to interpret the temporal and qualitative evolution of pain as a dynamic reflection of disease progression. Thus, the pathophysiology of abdominal pain in the acute abdomen represents a sophisticated interplay of visceral, somatoparietal, and referred pathways, mediated by distinct neural networks and influenced by embryologic origin, receptor distribution, and inflammatory extension. Accurate recognition of these mechanisms enhances diagnostic precision, enabling clinicians to distinguish medical from surgical emergencies and to prioritize timely, targeted interventions that can significantly reduce morbidity and mortality [19].

History and Physical

The evaluation of acute abdominal pain begins with a thorough and structured history and physical examination, both of which are indispensable in distinguishing benign, self-limiting conditions from potentially life-threatening causes that require urgent surgical or medical intervention. While many cases of abdominal pain are resolved spontaneously, the clinician's primary objective is to rapidly identify those at risk of catastrophic intra-abdominal pathology. Recognizing a patient with an established acute abdomen—characterized by peritoneal irritation, guarding, and systemic instability—is often straightforward. However, the true diagnostic challenge lies in detecting incipient abdominal catastrophes in patients whose symptoms remain subtle or nonspecific. In these situations, meticulous attention to history and physical findings becomes the cornerstone of accurate diagnosis and timely management.

History

A detailed history of the present illness provides essential diagnostic clues and guides further evaluation. The location, onset, character, severity, and radiation of the pain are particularly informative, as they often correspond to the embryologic origin of affected organs. Patients should be asked to describe where the pain began and whether it has migrated—

for example, periumbilical pain that shifts to the right lower quadrant is typical of acute appendicitis, while epigastric pain radiating to the back suggests pancreatitis. The onset (sudden versus gradual) can also differentiate causes—sudden, severe pain may point to perforation or vascular catastrophe, while progressive pain may indicate inflammation or obstruction. The duration and frequency of the pain episodes help distinguish acute from chronic conditions, while the quality of pain—whether dull, cramping, burning, gnawing, or stabbing—can indicate the underlying mechanism. For instance, burning or gnawing pain is characteristic of peptic ulcer disease, whereas colicky pain that waxes and wanes typically arises from hollow viscus obstruction. Precipitating factors, such as meals, motion, or certain body positions, may further narrow the differential diagnosis. Pain that worsens after eating may indicate mesenteric ischemia or biliary colic, whereas relief after eating could point to duodenal ulcer disease. The associated symptoms provide further diagnostic guidance. Nausea and vomiting may occur with bowel obstruction, gastroenteritis, or pancreatitis; fever and chills suggest infection or inflammation; diarrhea or constipation may indicate bowel pathology; and hematochezia or melena raise suspicion for gastrointestinal bleeding. Dysuria, hematuria, or flank pain suggest urinary tract involvement, whereas weight loss and anorexia may signal malignancy. Cardiopulmonary symptoms, such as cough, dyspnea, or chest pain, must also be elicited, as myocardial infarction, pneumonia, or pulmonary embolism can masquerade as abdominal pain.

Reproductive and sexual history is vital, particularly in women of reproductive age. Questions should include the date of the last menstrual period, the possibility of pregnancy, and a history of dyspareunia, dysmenorrhea, or vaginal discharge, which may indicate pelvic inflammatory disease, ovarian torsion, or ectopic pregnancy. For men, evaluation for testicular pain or swelling may uncover torsion or epididymitis that can mimic intra-abdominal pathology. Past medical and surgical history must be reviewed carefully. Prior abdominal or pelvic surgery raises the risk of adhesive bowel obstruction, while a history of hypertension, atherosclerosis, or smoking may suggest abdominal aortic aneurysm or mesenteric ischemia. Conditions such as diabetes, peptic ulcer disease, chronic pancreatitis, and liver disease also predispose to specific causes of abdominal pain. The medication history is equally important. Nonsteroidal anti-inflammatory drugs (NSAIDs) can lead to peptic ulcer disease or gastritis, antibiotic use can cause *Clostridioides difficile* colitis, corticosteroids can mask infection or perforation, and opioids or anticholinergics may induce constipation or ileus. Alcohol use should always be addressed due to its association with hepatitis and pancreatitis, while travel history can identify exposure to infectious

enteropathogens in cases of suspected gastroenteritis or colitis [20].

Potential Causes of Pain Based on Location

The anatomic location of pain is among the most valuable clues for differential diagnosis. Pain in the left upper quadrant frequently reflects splenic pathology, such as splenomegaly, splenic infarct, splenic abscess, or splenic rupture, conditions that may follow trauma or hematologic disease. Epigastric pain often suggests upper gastrointestinal or pancreatic pathology. Causes include myocardial infarction—a critical extra-abdominal mimic—acute or chronic pancreatitis, peptic ulcer disease, gastroesophageal reflux disease (GERD), gastritis, and gastroparesis. These entities share overlapping symptomatology but differ markedly in urgency and required intervention. Right upper quadrant pain classically arises from hepatobiliary disorders. The differential diagnosis includes biliary colic, acute cholecystitis, acute cholangitis, sphincter of Oddi dysfunction, acute hepatitis, perihepatitis, liver abscess, Budd–Chiari syndrome, and portal vein thrombosis. The presence of Murphy’s sign, jaundice, or fever helps narrow this spectrum—fever and jaundice, for example, may indicate ascending cholangitis, a surgical emergency [20][6]. Pain from other regions of the abdomen can similarly guide evaluation. Periumbilical pain suggests pathology involving the small intestine or appendix, whereas lower abdominal pain may reflect colonic, urinary, or reproductive organ involvement. Importantly, in elderly patients or those with comorbidities, pain localization may be less reliable due to blunted inflammatory responses or neuropathy. In summary, the history and physical examination remain the most critical components in evaluating the acute abdomen. A systematic approach—integrating pain characterization, associated symptoms, risk factors, and anatomical localization—allows clinicians to prioritize investigations and identify patients who require emergent imaging or surgical consultation. While modern imaging enhances diagnostic precision, no substitute exists for the nuanced clinical judgment derived from a thorough bedside assessment [20][6].

Physical Examination

A comprehensive and systematic physical examination is a critical component in the evaluation of a patient with acute abdominal pain. It must be conducted promptly, yet with deliberate attention to detail, as findings from the examination often provide the earliest clues to the underlying pathology. The clinician should begin by assessing the patient’s general appearance and vital signs, noting any abnormal findings such as tachycardia, hypotension, fever, or tachypnea, which may indicate systemic compromise. The patient’s posture, movement, and facial expression can also reveal valuable diagnostic information. For example, patients who remain completely still and experience sharp pain with

minimal movement—such as when the bed is bumped—often exhibit peritoneal irritation, whereas those who appear restless and writhing in pain typically have visceral or ischemic causes, such as biliary colic or renal colic [21][22]. A focused abdominal examination should proceed in the classical sequence of inspection, auscultation, percussion, and palpation. During inspection, the clinician should observe abdominal distension, surgical scars, visible peristalsis, hernias, or discoloration (such as Cullen’s or Grey–Turner’s signs, which may suggest retroperitoneal hemorrhage or pancreatitis). The position of the abdomen—whether scaphoid, flat, or distended—can also provide important diagnostic clues. Auscultation should precede palpation to avoid artificially altering bowel motility. The clinician should listen to bowel sounds in all quadrants, assessing their presence, frequency, and character. Hypoactive or absent bowel sounds may indicate peritonitis or paralytic ileus; while tinkling, high-pitched sounds suggest mechanical bowel obstruction. Bruits may occasionally be detected in patients with vascular causes, such as mesenteric ischemia or an abdominal aortic aneurysm [21][22].

Percussion is then performed lightly to assess tympany, dullness, or shifting dullness. Tympany over a distended area may indicate gas-filled loops of bowel, while localized dullness may signal a mass, fluid, or organomegaly. Shifting dullness, when present, confirms ascites. Gentle percussion that elicits significant pain suggests peritoneal inflammation, warranting further investigation [23]. Palpation should begin superficially and progress to deeper palpation, starting away from the area of maximal tenderness. The examiner should note guarding, either voluntary or involuntary, as well as rebound tenderness, both of which are classic indicators of peritonitis. Careful localization of tenderness helps narrow the differential diagnosis—right lower quadrant pain may indicate appendicitis, left lower quadrant pain may suggest diverticulitis, and right upper quadrant pain may point toward cholecystitis or cholangitis. Rigidity and board-like stiffness are ominous signs of diffuse peritoneal inflammation, often due to perforation or peritonitis. Although traditional teaching has long recommended a rectal examination in all patients presenting with abdominal pain, current evidence suggests that its diagnostic value is limited, particularly in cases such as appendicitis, where it rarely changes management. However, it remains essential when gastrointestinal bleeding, prostate pathology, or rectal masses are suspected. Similarly, a pelvic examination is imperative in females presenting with lower abdominal pain, as it allows assessment for pelvic inflammatory disease, ectopic pregnancy, or ovarian torsion. In males, a testicular examination is mandatory when testicular torsion or epididymitis is a diagnostic consideration. Examination for inguinal and femoral hernias should be routine, as incarcerated

or strangulated hernias may present subtly but require immediate surgical attention.

Beyond the abdomen, clinicians should perform a general systemic examination, including the eyes and skin for jaundice (suggestive of hepatic or biliary pathology) and mucous membranes for signs of dehydration or anemia. Cardiac and respiratory assessments are vital in patients presenting with upper abdominal pain, as myocardial infarction or pneumonia can mimic intra-abdominal disease. Finally, identifying extraintestinal manifestations such as arthralgia, rashes, or oral ulcers may suggest inflammatory bowel disease or autoimmune etiologies. In summary, the physical examination in acute abdominal pain serves as a crucial diagnostic bridge, linking clinical suspicion with targeted investigations. Each component—from inspection to systemic evaluation—contributes essential information that helps distinguish benign self-limited conditions from life-threatening emergencies. A meticulous, systematic, and patient-centered approach ensures that serious intra-abdominal pathology is neither overlooked nor misdiagnosed [21][22][23].

Classic Presentation of Abdominal Pain

Classic abdominal pain presentations, while frequently invoked at the bedside, are heuristics rather than rigid rules; nonetheless, recognizing them can shorten time to definitive imaging, consultation, and intervention. Clinicians must simultaneously maintain suspicion for atypical variants—particularly in older adults, pregnant patients, the immunosuppressed, and those receiving analgesics—because blunted inflammatory responses or altered pain perception can mask peritoneal or vascular emergencies. What follows synthesizes common archetypes of abdominal pain that often herald urgent or emergent disease, with emphasis on the features most likely to influence immediate management and disposition. Abdominal aortic aneurysm exemplifies this tension between silence and catastrophe. Most aneurysms remain clinically occult until incidentally detected. When symptomatic yet unruptured, patients frequently describe deep, noncolicky abdominal, back, or flank pain that may wax and wane over days. A ruptured aneurysm, by contrast, tends to present abruptly with severe pain, hypotension, and—classically—a pulsatile abdominal mass. The full triad is insensitive, especially in obesity, but any two elements in an older smoker with vascular disease warrant immediate aortic imaging and simultaneous resuscitation given the high prehospital and in-hospital mortality. Intestinal obstruction classically announces itself with colicky pain, vomiting, and obstipation. Examination often reveals distension, tympany, and either high-pitched, tinkling bowel sounds from hyperperistaltic proximal loops or, later, hypoactive or absent sounds with ileus. Pain character and timing correlate with the level and completeness of obstruction; small-bowel obstruction produces earlier vomiting and periumbilical pain, while large-bowel obstruction

classically causes more pronounced distension and delayed emesis. Closed-loop obstruction and strangulation convert intermittent cramping into continuous pain with peritoneal signs, mandating urgent operative evaluation.

Acute appendicitis remains the paradigmatic migratory pain syndrome. Early luminal obstruction generates visceral periumbilical discomfort that localizes over hours to the right lower quadrant at McBurney's point as the parietal peritoneum becomes inflamed. Anorexia, nausea, low-grade fever, and vomiting are common but not universal, and retrocecal or pelvic appendices can blunt or shift somatic tenderness. The combination of migratory pain, localized guarding, and peritoneal irritation in the right iliac fossa retains high diagnostic value, but atypical presentations are frequent in the very young and very old. Acute cholecystitis presents with severe, persistent right upper quadrant or epigastric pain lasting beyond four to six hours, often following a meal. Guarding, fever, leukocytosis, and a positive Murphy sign on examination support the diagnosis. Distinguishing prolonged biliary colic from evolving cholecystitis hinges on the duration of constant pain and the emergence of systemic inflammatory findings, both of which prompt early ultrasonography and antibiotic therapy while surgical consultation is arranged.

Acute cholangitis arises when an obstructing stone or stricture in the common bile or hepatic ducts permits ascending infection. The classic Charcot triad of fever, jaundice, and right upper quadrant pain is neither ubiquitous nor necessary for severe disease, but its presence should accelerate broad-spectrum antibiotics, biliary decompression planning, and hemodynamic monitoring. Modern criteria incorporate laboratory and imaging evidence of ductal dilation and cholestasis to refine diagnosis and guide the timing of endoscopic intervention [24]. Acute diverticulitis typically causes steady left lower quadrant pain in Western populations, though right-sided disease occurs, particularly in patients of Asian descent. Localized guarding, rigidity, and rebound tenderness reflect paracolic inflammation; microperforation increases the likelihood of peritoneal signs. A rectal examination may uncover occult blood or, rarely, mass effect or tenderness when a distal sigmoid abscess abuts the rectum. Imaging not only confirms the diagnosis but also stratifies severity and guides percutaneous drainage versus surgical consultation [25].

Acute pancreatitis most often manifests as severe, constant epigastric and left upper quadrant pain radiating posteriorly to the back, frequently accompanied by nausea and vomiting. Pain is typically exacerbated by supination and improved by sitting forward. Early recognition matters because aggressive fluid resuscitation and targeted etiologic therapy—biliary, alcoholic, hypertriglyceridemic, or medication-induced—attenuate systemic

complications and necrosis. Aortic dissection may mimic diverse intrathoracic and intra-abdominal conditions. Descending dissections more commonly produce back or abdominal pain, classically described as sharp, ripping, or tearing. Hypotension suggests rupture, hemothorax, or tamponade, though hypertension is more typical in subacute descending dissections. Pulse deficits, focal neurologic signs, mesenteric ischemia, or renal malperfusion reflect branch-vessel involvement and demand immediate imaging and blood pressure control while surgical or endovascular teams are mobilized [26][27]. Ectopic pregnancy should be presumed in any reproductive-age patient with first-trimester vaginal bleeding and abdominal pain six to eight weeks after the last menstrual period until proven otherwise. The combination of pain, bleeding, and a positive pregnancy test mandates urgent transvaginal ultrasonography and quantitative β -hCG interpretation to distinguish viable intrauterine gestation, pregnancy of unknown location, and ectopic implantation. Delays risk tubal rupture, hemorrhagic shock, and loss of future fertility.

Incarcerated hernias present with localized, exquisitely tender groin or ventral masses that may show overlying erythema and systemic signs when strangulation ensues. Because necrotic bowel can remain sequestered within the hernia sac, frank peritonitis may be absent until ill-advised forceful reduction or late perforation occurs. Gentle reduction after appropriate analgesia may be attempted in selected cases without ischemic features, but persistent tenderness, tachycardia, leukocytosis, or skin changes should prompt urgent operative evaluation [28]. Intestinal malrotation with midgut volvulus in children characteristically produces abdominal pain out of proportion to early examination, bilious emesis, distension, and rapid progression to peritonitis and hemodynamic instability. In adults, cecal or sigmoid volvulus often begins more insidiously with progressive pain, nausea, bloating, and constipation, though fulminant presentations also occur. Vomiting typically follows pain by days. The pain is generally constant and intense, punctuated by colicky exacerbations during peristaltic waves. Radiographic patterns and endoscopic reducibility help triage patients to detorsion versus immediate surgery when ischemia is suspected [29]. Intussusception in infants and toddlers classically presents with paroxysms of severe, intermittent abdominal pain every 15 to 20 minutes, during which the child pulls the knees to the abdomen and cries inconsolably. Vomiting is common and often progresses from nonbilious to bilious as obstruction evolves. A right-sided, sausage-shaped abdominal mass may be palpable. Approximately one quarter of patients pass grossly bloody stools; far more have guaiac-positive stools, and the often-quoted currant jelly stool is a late sign of mucosal sloughing.

Mesenteric ischemia presents with abrupt, severe, diffuse abdominal pain that is strikingly disproportionate to early physical findings. Embolic occlusion of the proximal superior mesenteric artery typically yields periumbilical pain, whereas left-sided cramping and hematochezia with tenesmus suggest acute colonic ischemia. Because laboratory tests lack sensitivity, a high index of suspicion and rapid imaging are essential to preserve bowel and reduce mortality; delays convert reversible ischemia into transmural infarction requiring extensive resection. Necrotizing enterocolitis, a disease of premature and medically fragile neonates, often first manifests as a sudden change in feeding tolerance. Additional signs—abdominal wall erythema, crepitus, induration, apnea, temperature instability, and lethargy—portend systemic involvement. Hypotension and septic shock signal advanced disease. Radiographic pneumatosis intestinalis, portal venous gas, or free air dictate escalation to bowel rest, antibiotics, and surgical consultation [39]. Ovarian torsion presents sudden, severe unilateral pelvic pain accompanied by nausea and vomiting in a patient with an adnexal mass or ovarian stimulation. Because venous outflow obstruction precedes arterial inflow compromise, pain may wax and wane early, misleading clinicians; Doppler flow does not exclude torsion, and prompt gynecologic evaluation preserves ovarian viability.

Perforation of the gastrointestinal tract is suggested by the abrupt intensification of pain, often after instrumentation or steroid use. The clinical picture ranges from localized peritonitis to diffuse rigidity and septic physiology; free intraperitoneal air, retroperitoneal emphysema, or contained abscesses on imaging guide operative versus percutaneous source control. Sequelae include fistula formation when leakage remains walled off. Splenic rupture, typically traumatic but occasionally spontaneous in infectious mononucleosis or neoplasia, presents with left upper quadrant or left chest wall pain and Kehr sign—left shoulder pain exacerbated by inspiration—reflecting diaphragmatic irritation by hemoperitoneum. Hemodynamic instability mandates immediate resuscitation and hemorrhage control through operative or endovascular means. Testicular torsion, most common in adolescents but possible at any age, frequently presents as acute scrotal pain with associated nausea and lower abdominal discomfort. Because visceral afferents overlap, abdominal pain may predominate. Absent cremasteric reflex, high-riding testis, and horizontal lie heighten suspicion, but clinical uncertainty should not delay urologic consultation and rapid detorsion to prevent ischemic loss of the gonad. Across these entities, the value of “classic” presentations lies not in their perfection but in their capacity to shape pretest probability and prioritize actions. When clinicians pair these archetypes with vigilant reassessment and judicious imaging, they improve the chances of timely source

control, organ salvage, and survival—even when the presentation is anything but classic.

Evaluation

The evaluation of acute abdominal pain begins at the bedside and is driven by the synthesis of a careful history with a directed physical examination, immediately stratifying patients by hemodynamic stability and the likelihood of life-threatening disease. Patients with unstable vital signs, peritoneal signs, or suspected catastrophic etiologies—such as ectopic pregnancy, myocardial infarction, high-grade bowel obstruction, abdominal aortic aneurysm, acute mesenteric ischemia, or hollow-viscus perforation—require parallel processes of diagnostic assessment and resuscitation in an emergency setting. Initial actions emphasize rapid appraisal of general appearance and the ABCs, with prompt measurement of vital signs and correction of hypoxemia or hypotension as indicated. Point-of-care tests provide crucial early discriminants: a bedside capillary glucose helps identify or exclude diabetic ketoacidosis as a metabolic driver of pain, a 12-lead electrocardiogram screens for cardiac ischemia and electrolyte-related conduction disturbances, and focused ultrasonography accelerates detection of abdominal aortic aneurysm, hydronephrosis, pericardial effusion, or intraperitoneal free fluid compatible with hemoperitoneum. In parallel, analgesia should be administered; contemporary evidence supports humane pain control without compromising diagnostic accuracy when the examination is performed thoughtfully and, if needed, serially. When the clinical picture suggests perforation or intra-abdominal sepsis, an upright or decubitus portable chest radiograph is a pragmatic first study to detect subdiaphragmatic free air and to reassess cardiopulmonary status. If bedside ultrasound and chest radiography fail to yield a decisive clue, contrast-enhanced computed tomography of the abdomen and pelvis becomes the principal next step, offering high sensitivity for obstruction, ischemia, perforation, and abscess. In pregnant patients, ionizing radiation avoidance dictates an ultrasound-first approach to confirm intrauterine pregnancy and evaluate common etiologies such as appendicitis, nephrolithiasis, cholelithiasis, cholecystitis, and uterine rupture; when further anatomic detail is indispensable and sonography is nondiagnostic, magnetic resonance imaging without gadolinium is preferred. Even in nonpregnant patients, ultrasound retains primacy for biliary disease and selected gynecologic and renal disorders, while CT provides comprehensive cross-sectional problem solving in indeterminate cases. Throughout, laboratory testing is hypothesis-driven: a complete blood count and basic metabolic panel support resuscitative decisions and trend leukocytosis or electrolyte derangements, while targeted assays refine probability for specific diagnoses suggested by symptom location and risk factors. In less emergent presentations, the anatomic location of pain provides a

scaffold for tailored evaluation, mirroring the structure of the differential diagnosis. Age, comorbidity, and pregnancy status further condition pretest probabilities, and in children the likely causes vary with developmental stage; thus, imaging selection and sedation planning are adapted to minimize risk while preserving diagnostic yield. The principle remains constant across populations: align the next diagnostic step with the most dangerous plausible diagnosis that remains on the table, escalating promptly if new data suggest deterioration.

Right Upper Quadrant Pain

Right upper quadrant pain most commonly reflects disorders of the hepatobiliary system, recognizing that the liver parenchyma is relatively insensate and becomes painful primarily when capsular stretch occurs. Initial evaluation combines targeted laboratory testing with ultrasound imaging. A complete blood count, electrolytes, aminotransferases, alkaline phosphatase, total and direct bilirubin, lipase, and amylase help discriminate between cholestatic and hepatocellular patterns and screen for concurrent pancreatobiliary involvement. Abdominal ultrasonography is the imaging modality of choice because it rapidly detects gallstones, gallbladder wall thickening, pericholecystic fluid, and common bile duct dilation, and because it can elicit Murphy's sign under direct visualization to support acute cholecystitis. When ultrasound findings are equivocal and clinical suspicion remains high, secondary modalities such as hepatobiliary scintigraphy for cystic duct obstruction or cross-sectional imaging for complications may be pursued, but ultrasound remains the first-line test anchored by its availability, lack of radiation, and high specificity for cholelithiasis and biliary dilation [30][31][32]. In parallel, clinical features such as fever and jaundice guide early identification of ascending cholangitis, for which expeditious antibiotics and biliary decompression are time sensitive.



Figure-3: Acute abdomen imaging.

Epigastric Pain

The initial workup of epigastric pain parallels that for right upper quadrant pain because of the overlapping involvement of foregut structures, but it adds a cardiovascular dimension given the frequent mimicry between visceral and cardiac syndromes. For patients with risk factors or suggestive symptoms—dyspnea, exertional chest pressure, diaphoresis,

nausea, or vomiting—electrocardiography and cardiac troponin testing are obtained promptly, and a chest radiograph is considered to evaluate for pneumonia or free subdiaphragmatic air that might refer pain to the epigastrium. Abnormalities in these studies may trigger echocardiography, stress testing, coronary CT angiography, or invasive angiography depending on acuity and resource availability. When peptic ulcer disease, gastritis, or gastroesophageal reflux disease is suspected, acid suppression and *Helicobacter pylori* testing are initiated, with upper endoscopy reserved for alarm features such as gastrointestinal bleeding, anemia, dysphagia, weight loss, or persistent pain refractory to empiric therapy. Elevations in lipase support pancreatitis in the appropriate clinical context, but cross-sectional imaging is reserved for diagnostic uncertainty, suspected complications, or failure to improve after supportive care. Throughout, the clinician reappraises the trajectory of pain and associated symptoms to distinguish time-sensitive entities, such as perforated ulcer or gastric volvulus, from conditions amenable to outpatient management.

Left Upper Quadrant Pain

Pain localized to the left upper quadrant frequently arises from splenic disease or epigastric sources and demands careful history to elicit early satiety, abdominal fullness, and referred pain to the chest or left shoulder, which suggest splenic capsular stretch. Ultrasonography is the preferred initial imaging tool to determine splenic size and to screen for gross structural abnormalities, while laboratory evaluation begins with a complete blood count to identify cytopenias or leukocytosis that point toward hematologic disorders. Serum transaminases may be obtained to appraise concomitant hepatic involvement, and additional tests are individualized: HIV testing when risk factors or opportunistic infections are plausible, blood cultures in febrile patients to investigate splenic abscess, and a glucocerebrosidase assay when Gaucher disease is a consideration in the proper clinical context. Cross-sectional imaging by CT or MRI becomes necessary when focal lesions such as splenic infarction, abscess, laceration, or neoplasm are suspected, as these modalities delineate parenchymal architecture, vascular supply, and complicating collections with higher resolution. A chest radiograph is sometimes revealing, demonstrating a reactive pleural effusion that accompanies splenic abscess or subphrenic processes. When malignancy is suspected, definitive diagnosis may require bone marrow evaluation or lymph node biopsy, coordinated with hematology-oncology to mitigate bleeding risks and optimize diagnostic yield [33].

Across these regional syndromes, the unifying framework is probabilistic and iterative. Each new datum—vital signs, laboratory pattern, sonographic sign, CT finding—reshapes the diagnostic landscape and refines risk, prompting either de-escalation to observation and outpatient follow-up

or escalation to more invasive diagnostics and consultation. The best outcomes follow from disciplined adherence to resuscitation principles in unstable patients, judicious imaging aligned with the suspected pathology, and timely specialty involvement when an operative or procedural solution is likely. By integrating bedside assessment with targeted testing, clinicians can navigate the heterogeneity of acute abdominal pain, ensuring that high-risk conditions are recognized and treated without delay while avoiding unnecessary radiation or interventions in those with self-limited disease [30][31][32][33].

Lower Abdominal Pain

Lower abdominal pain arises from a broad anatomic corridor that includes the distal colon, pelvic organs, urinary tract, and, in male patients, the scrotal contents. Because missed pregnancy can catastrophically alter management, exclusion of pregnancy is the first priority in all patients with childbearing potential; a timely serum or urine β -hCG aligns subsequent testing and imaging with maternal–fetal safety. The clinical differential remains wide even after pregnancy status is clarified. Pelvic sources—pelvic inflammatory disease, ectopic pregnancy, tubo-ovarian abscess, and ovarian torsion—often coexist with gastrointestinal or urinary pathology, and appendicitis, incarcerated hernia, diverticulitis, and ureteral obstruction remain common mimics. Accordingly, evaluation proceeds from the history and examination to a tiered set of laboratory tests and imaging studies that are selected to adjudicate the most dangerous plausible diagnosis while minimizing unnecessary radiation and delay. Baseline laboratory assessment frequently begins with a complete blood count to evaluate leukocytosis in infection or anemia in hemorrhage and, when bleeding is suspected, type and cross-match to prepare for transfusion. Urinalysis helps distinguish cystitis and pyelonephritis from nonurological causes of pelvic pain and can reveal hematuria in nephrolithiasis, while urine culture is reserved for complicated or febrile urinary infections. In patients with fever or systemic toxicity, blood cultures may be obtained prior to antibiotics to support later organism-directed therapy. In sexually active individuals with lower abdominal pain, targeted testing for *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, *Trichomonas vaginalis*, and bacterial vaginosis refines the probability of pelvic inflammatory disease, informs antimicrobial selection, and mitigates the risk of long-term sequelae such as infertility.

Imaging selection reflects the suspected organ system and the patient's pregnancy status. In patients with a positive pregnancy test, transvaginal and transabdominal ultrasonography are foundational, both to locate an intrauterine pregnancy and to detect free intraperitoneal fluid consistent with ruptured ectopic pregnancy or ovarian cyst, as well as to

evaluate adnexal torsion. When pregnancy is excluded, contrast-enhanced computed tomography of the abdomen and pelvis becomes the principal cross-sectional study for suspected appendicitis, diverticulitis, small-bowel obstruction, or nephrolithiasis, particularly when sonography is unrevealing or nonconclusive. Colonoscopy is not an acute diagnostic tool for peritonitis but, in subacute presentations of abdominal pain with iron-deficiency anemia or altered bowel habits, it is critical to evaluate for colon cancer or inflammatory bowel disease and to obtain histopathology that guides long-term therapy. In males with lower abdominal pain and a suggestive scrotal examination—tender swelling, high-riding testis, or a horizontal lie—prompt scrotal ultrasonography with Doppler interrogation assesses testicular perfusion and distinguishes torsion from epididymo-orchitis; because imaging can be falsely reassuring early in torsion, urologic consultation should not be delayed when clinical suspicion is high.

Diffuse Abdominal Pain

Diffuse abdominal pain poses an additional challenge because it may reflect intrathoracic disease, metabolic derangements, or generalized intraabdominal pathology. Initial laboratory testing therefore emphasizes broad discrimination. A complete blood count with differential quantifies leukocytosis or bandemia and assesses anemia from occult bleeding. Serum electrolytes and calculation of the anion gap identify dehydration, lactic acidosis, or toxin-mediated syndromes; blood urea nitrogen and creatinine gauge prerenal azotemia or obstructive uropathy; and serum glucose screens for diabetic emergencies that can present with abdominal pain. Amylase and lipase are obtained to detect pancreatitis, whereas aminotransferases, alkaline phosphatase, and bilirubin help separate hepatocellular from cholestatic patterns and direct attention to biliary obstruction or hepatitis. Calcium abnormalities may precipitate pain via pancreatitis or renal colic. As in focal lower abdominal pain, pregnancy testing remains mandatory in all patients with childbearing potential because it directly alters imaging algorithms and therapeutic thresholds.

Adjunctive imaging is selected according to the evolving bedside impression. A chest radiograph is useful in patients with diffuse upper abdominal pain and respiratory symptoms to identify lower-lobe pneumonia or to reveal subdiaphragmatic free air from perforated viscus on upright films. Plain abdominal radiographs retain value for rapid detection of pneumoperitoneum and for classic signs of bowel obstruction, including air–fluid levels and diffuse distension, although cross-sectional imaging has largely supplanted them for definitive diagnosis. When pulmonary embolism is suspected in the setting of pleuritic upper abdominal discomfort or unexplained tachycardia and hypoxemia, CT pulmonary angiography provides fast, definitive vascular imaging. Contrast-enhanced CT of the

abdomen and pelvis is the workhorse for suspected volvulus or mechanical obstruction and is also integral in hemodynamically stable patients when abdominal aortic aneurysm remains a concern after bedside assessment. In hemodynamically unstable patients with suspected ruptured aneurysm, focused ultrasonography can confirm aneurysm presence rapidly at the bedside and guide emergent operative or endovascular decisions without delaying resuscitation. For patients in whom mesenteric ischemia or aortic dissection is a leading diagnosis, an intravenous contrast-enhanced CT angiogram of the chest, abdomen, and pelvis provides comprehensive arterial mapping, identifies branch-vessel compromise, and detects bowel hypoenhancement or pneumatosis that signal transmural ischemia. To support judicious test selection, clinicians can consult the American College of Radiology Appropriateness Criteria, which synthesize evidence and expert consensus to align imaging modality with specific clinical questions in abdominal pain.

Children

In children, the diagnostic framework parallels that of adults but must be adapted to developmental anatomy, radiation stewardship, and the unique epidemiology of pediatric disease. Bilious emesis in an infant younger than one year represents malrotation with volvulus until proven otherwise and mandates emergent evaluation to prevent midgut necrosis. Because cumulative radiation exposure carries a higher lifetime risk in children, CT scanning is used sparingly; instead, initial imaging emphasizes modalities without ionizing radiation. For bilious vomiting, severe pain, pronounced distension, or peritoneal signs, an abdominal radiograph is commonly the first step to identify free air from perforation or to demonstrate obstruction through air–fluid levels and dilated loops. Ultrasonography is highly sensitive for intussusception, offering real-time visualization of the target sign, while a contrast enema both confirms the diagnosis and, in many cases, therapeutically reduces the intussusception. For suspected malrotation with volvulus, an upper gastrointestinal contrast series remains the test of choice because it delineates the position of the ligament of Treitz and the abnormal corkscrew configuration of the twisted midgut. Children who present with a classic history and examination for appendicitis should be evaluated by a pediatric surgeon early, as prompt surgical assessment can obviate imaging when the pretest probability is high and the risks of delay are greater than the benefits of confirmatory studies. When the presentation is atypical or equivocal, graded-compression abdominal ultrasonography is preferred as the first-line test; CT is reserved for nondiagnostic ultrasound in the context of high clinical suspicion or complications where cross-sectional detail will alter management.

Necrotizing enterocolitis requires particular vigilance in premature and medically fragile neonates.

Abdominal radiographs are the cornerstone, revealing hallmark features such as pneumatosis intestinalis, portal venous gas, and, in advanced cases, free intraperitoneal air. Some centers increasingly employ ultrasonography to detect free air, focal fluid collections, increased bowel wall thickness, and echogenicity, which can refine prognostication and the timing of surgical consultation [34]. A concurrent sepsis evaluation is essential and typically includes a complete blood count, serum electrolytes, blood urea nitrogen, creatinine, and glucose levels to characterize systemic involvement. Laboratory trajectories contribute prognostic information: persistent hyponatremia, hyperglycemia, and metabolic acidosis suggest necrotic bowel and evolving sepsis, while serial serum lactate levels provide a dynamic marker of perfusion and can be trended to monitor disease progression and response to therapy [35]. Across pediatric presentations, close collaboration with pediatric surgery and careful choice of imaging that balances diagnostic yield with safety are central to optimizing outcomes. In sum, the evaluation of lower and diffuse abdominal pain rests on disciplined bedside assessment, early exclusion of pregnancy in those at risk, hypothesis-driven laboratory testing, and imaging algorithms tailored to the suspected pathology and patient context. By prioritizing the most time-sensitive diagnoses, leveraging ultrasound where appropriate, reserving CT for cases in which cross-sectional clarity will change management, and adapting strategies for children to minimize radiation exposure, clinicians can expedite accurate diagnosis and align interventions with the anatomic source of pain. This approach ensures timely recognition of life-threatening disease while avoiding unnecessary testing, thereby improving patient safety and resource stewardship across diverse emergency and acute care settings [34][35].

Treatment / Management

The management of patients presenting with an acute abdomen demands a structured, multidisciplinary approach focused on simultaneous diagnosis, stabilization, and targeted intervention. Resuscitation and diagnostic evaluation must occur concurrently, with early surgical or specialty consultation initiated based on clinical suspicion rather than delayed for confirmatory imaging. Clinicians should immediately place patients on cardiac monitoring to assess for arrhythmias or hemodynamic instability, which can accompany sepsis, electrolyte imbalance, or massive hemorrhage. Those presenting with peritonitis, hypotension, or a toxic appearance require rapid establishment of large-bore intravenous access for the administration of isotonic crystalloid fluids. Persistent hypotension despite adequate fluid resuscitation indicates septic or hemorrhagic shock, warranting the early initiation of vasopressor therapy, most commonly norepinephrine, titrated to maintain adequate mean arterial pressure

and organ perfusion [34]. In patients with adrenal insufficiency or chronic corticosteroid use, stress-dose steroids (such as hydrocortisone 100 mg IV every 8 hours) must be administered promptly to prevent adrenal crisis during acute physiologic stress. For patients with evidence of hypovolemia secondary to hemorrhage, immediate transfusion of blood products according to a massive transfusion protocol may be necessary, with a balanced ratio of red blood cells, plasma, and platelets to optimize oxygen delivery and coagulation. When infection, peritoneal contamination, or sepsis is suspected, clinicians must administer broad-spectrum antibiotics without delay. Empiric regimens should target gram-negative enteric bacilli, gram-positive cocci, and anaerobes, commonly including combinations such as piperacillin-tazobactam, carbapenems, or a third-generation cephalosporin plus metronidazole, adjusted according to local resistance patterns and culture results.

Pain management is a critical yet historically underemphasized component of care for the acutely painful abdomen. Contemporary evidence confirms that appropriate analgesia—using acetaminophen, parenteral ketorolac, or opioids—does not obscure diagnostic findings or delay accurate diagnosis. Clinicians must nonetheless exercise caution with nonsteroidal anti-inflammatory agents such as ketorolac, particularly in patients with suspected peptic ulcer disease, gastritis, or renal dysfunction, as these agents may exacerbate mucosal injury or precipitate acute kidney injury. Intravenous opioids, titrated carefully to effect, remain the most effective agents for rapid pain relief and should be administered judiciously, with ongoing reassessment of mental status and respiratory function [35]. Definitive treatment depends on the underlying diagnosis and often requires coordination among multiple specialties. General surgeons manage most cases of peritonitis, perforated viscus, appendicitis, and bowel obstruction; vascular surgeons handle ruptured or expanding abdominal aortic aneurysms and mesenteric ischemia; gynecologists are consulted for ectopic pregnancy, tubo-ovarian abscess, and ovarian torsion; urologists for obstructing infected stones or testicular torsion; and interventional radiologists for image-guided abscess drainage or angiographic embolization in selected bleeding cases.

Certain conditions require immediate operative or procedural intervention. These include a perforated hollow viscus with pneumoperitoneum visible on radiography, ruptured ectopic pregnancy, testicular torsion, ovarian torsion, obstructing infected ureteral calculus, and ruptured abdominal aortic aneurysm. Delayed recognition or transfer in such cases markedly increases mortality. Clinicians should also anticipate and treat complications of sepsis—such as acute kidney injury or disseminated intravascular coagulation—through early broad-spectrum coverage, aggressive resuscitation, and critical care support. In

summary, the management of the acute abdomen requires rapid resuscitation, timely diagnostic testing, early analgesia, and expeditious consultation with the appropriate specialists. A proactive, team-based strategy ensures that reversible causes are addressed promptly and that time-sensitive surgical emergencies receive definitive treatment without unnecessary delay.

Differential Diagnosis

The differential diagnosis of acute abdominal pain is broad and heterogeneous, spanning medical, surgical, gynecologic, urologic, vascular, metabolic, and even thoracic conditions. An organized, symptom-driven approach helps clinicians rapidly identify those who require immediate operative management or urgent medical therapy while minimizing unnecessary delays. Pattern recognition begins with the pain's location, quality, onset, temporal evolution, and associated features, but definitive triage often relies on early laboratories, point-of-care ultrasound, and targeted cross-sectional imaging to adjudicate competing possibilities in time-sensitive scenarios [6]. Age, pregnancy status, prior operations, immunocompromise, and cardiometabolic comorbidity further reshape pretest probabilities and should be incorporated into every diagnostic pass. Several high-consequence entities must remain at the forefront because missed or delayed diagnoses are catastrophic. Abdominal aortic aneurysm, intraabdominal hemorrhage with hemoperitoneum, mesenteric ischemia, and ruptured ectopic pregnancy often present with abrupt, severe pain and physiologic instability; prompt resuscitation proceeds in parallel with imaging, ideally with bedside ultrasound for unstable patients and contrast-enhanced CT angiography for those who are stable enough to leave the resuscitation bay [36]. Aortic dissection extending into visceral branches, portal vein thrombosis, Budd–Chiari syndrome, renal infarction, and splenic infarction are vascular mimics that can produce focal or diffuse abdominal pain out of proportion to early examination, and they demand rapid vascular imaging and specialty involvement. In children and older adults, intussusception and bowel volvulus, respectively, can evolve quickly to ischemia; in neonates and premature infants, necrotizing enterocolitis is a parallel emergency in which radiographic pneumatosis and metabolic acidosis portend transmural necrosis [37].

Gastrointestinal sources are common and diverse. Acute appendicitis remains the leading surgical cause across many age groups, but the presentation may be atypical with retrocecal or pelvic appendices. Acute cholecystitis and acute cholangitis anchor the right upper quadrant differential, with biliary colic, sphincter of Oddi dysfunction, and acute hepatitis as important alternatives. Acute pancreatitis presents with epigastric pain radiating to the back and may be triggered by gallstones, alcohol, hypertriglyceridemia, drugs, or rarely hypercalcemia;

acute peptic ulcer disease and frank perforation remain essential considerations in patients with risk factors or sudden peritonitis. In the left lower quadrant, acute diverticulitis predominates in many Western populations, while epiploic appendagitis, sclerosing mesenteritis, and colonic pseudo-obstruction mimic inflammatory pain without necessarily requiring surgery. Meckel's diverticulum, eosinophilic gastroenteritis, and celiac disease represent less frequent but important diagnoses in selected clinical contexts, as do helminthic infections in travelers or migrants. Familial Mediterranean fever and IgA vasculitis exemplify inflammatory disorders that produce recurrent abdominal pain, sometimes with fever, arthralgia, or rash, and they may masquerade as surgical abdomen until recognized [6][36]. Mechanical and functional processes also feature prominently. Small- or large-bowel obstruction from adhesions, hernias, malignancy, or volvulus presents with colicky pain, distension, vomiting, and obstipation, whereas narcotic bowel syndrome and gastroparesis reflect dysmotility and often coexist with chronic opioid exposure or diabetes. Rectus sheath hematoma mimics acute abdomen in anticoagulated or coughing patients and is frequently diagnosed by targeted ultrasound or CT. Lead poisoning and acute porphyrias belong to the metabolic and toxic differential; both cause severe abdominal pain with few localizing findings and should be considered when routine imaging is unrevealing. Hypercalcemia, hypothyroidism, and ketoacidosis can all precipitate abdominal pain through ileus, pancreatitis, or metabolic stress, and they are unmasked by basic chemistry panels and acid–base assessment [36][37].

Infectious and intraabdominal suppurative conditions require special vigilance. Acute peritonitis may be spontaneous in cirrhosis, secondary to hollow-viscus perforation, or postoperative. Liver abscess and splenic abscess present with fever, focal pain, and leukocytosis; modern imaging differentiates them from malignancy, while percutaneous or surgical source control complements broad-spectrum antibiotics. Pseudoappendicitis, often associated with *Yersinia enterocolitica*, can mimic right lower quadrant appendicitis; streptococcal pharyngitis, especially in children, may also provoke significant abdominal pain and merits a rapid antigen test when sore throat and fever coexist. Herpes zoster can produce segmental, burning abdominal pain preceding the rash, and clinician awareness prevents unnecessary laparotomy. Among thoracic masqueraders, lower-lobe pneumonia and pulmonary embolism frequently refer pain to the upper abdomen, underscoring the value of a chest radiograph or CT pulmonary angiography when respiratory symptoms, hypoxemia, or pleuritic features are present [38]. Gynecologic and urologic etiologies are central in lower abdominal pain. Ectopic pregnancy with tubal rupture, ovarian torsion, and tubo-ovarian abscess constitute surgical or interventional emergencies, whereas pelvic

inflammatory disease and perihepatitis (Fitz–Hugh–Curtis syndrome) require prompt broad-spectrum antibiotics and partner management. Urinary causes include acute ureteral colic, acute pyelonephritis, and, less commonly, renal infarction; hematuria and flank-to-groin radiation increase the likelihood of nephrolithiasis, while fever and costovertebral angle tenderness favor infection. Incarcerated or strangulated hernias can present subtly yet progress to ischemic pain and peritonitis; examination in standing and supine positions with attention to the groin and prior incision sites improves detection [36][38].

Rare but consequential conditions round out the differential. Wandering spleen predisposes to torsion and infarction with episodic left upper quadrant pain; paroxysmal nocturnal hemoglobinuria may cause abdominal pain via mesenteric thrombosis; celiac artery compression syndrome produces postprandial epigastric pain and weight loss; and malignancy—including gastrointestinal, hepatobiliary, pancreatic, gynecologic, or hematologic cancers—may present with subacute pain, anemia, or obstructive features. Thoracic duct–venous junction obstruction has been implicated in enigmatic abdominal pain syndromes through lymphatic congestion, though it remains uncommon. Finally, abdominal compartment syndrome, whether primary or secondary, should be considered in trauma, massive resuscitation, or postoperative patients with escalating ventilatory pressures, oliguria, and tense distension. Ultimately, the breadth of the differential diagnosis requires disciplined, iterative reasoning that integrates bedside clues with judicious testing. Clinicians refine probabilities with each new datum, relentlessly prioritizing diagnoses in which time to source control or reperfusion determines outcome. This structured approach—anchored in early recognition of vascular and gynecologic emergencies, prompt identification of surgical abdomens, and attention to medical mimics—reduces missed diagnoses, shortens time to treatment, and improves survival across the full spectrum of acute abdominal presentations [6][36][37][38].

Prognosis

The prognosis of patients presenting with an acute abdomen is heterogeneous and hinges on both the underlying etiology and the rapidity with which definitive therapy—medical, endoscopic, interventional, or surgical—is instituted [39][40]. Time-sensitive conditions such as ruptured abdominal aortic aneurysm, mesenteric ischemia, perforated viscus, and complicated biliary sepsis carry the highest mortality; in these entities, outcomes correlate closely with early recognition, hemodynamic resuscitation, targeted antibiotics when infection is suspected, and prompt source control. Age, frailty, immunosuppression, and multimorbidity amplify risk, as do delayed presentation, persistent hypotension, elevated serum lactate, acute kidney injury, and the need for vasopressor support, all of which portend

worse short-term survival and prolonged length of stay [39]. Conversely, benign or self-limited causes—such as nonspecific abdominal pain, biliary colic without cholecystitis, epiploic appendagitis, or uncomplicated gastroenteritis—typically resolve with conservative therapy and have an excellent prognosis when red-flag conditions have been conscientiously excluded [40]. Despite advances in imaging, laboratory diagnostics, and clinical decision support, diagnostic uncertainty remains common. Undifferentiated abdominal pain persists as the final diagnosis in roughly a quarter of patients discharged from emergency departments and in approximately 35% to 41% of those admitted to the hospital, reflecting both the protean nature of intraabdominal disease and the episodic resolution of symptoms before a definitive lesion is identified [2][6][7][8]. Reassuringly, longitudinal data indicate that about 80% of patients discharged with undifferentiated pain experience improvement or complete resolution within two weeks of the index visit, underscoring the importance of structured discharge instructions, return precautions, and timely outpatient reassessment to capture the minority who subsequently declare a specific pathology [2]. In admitted patients without an immediate explanation, a stepwise strategy of serial examinations, targeted repeat labs, and interval imaging often clarifies the diagnosis and guides definitive management, improving outcomes while minimizing unnecessary procedures [6][7][8].

Condition-specific trajectories vary markedly. For example, mortality in mesenteric ischemia escalates rapidly with each hour of diagnostic delay, whereas early endoscopic decompression in acute cholangitis or timely cholecystectomy in acute cholecystitis reduces complications and readmissions. In appendicitis, prompt surgery or carefully selected nonoperative management yields low complication rates, but perforation risk rises with delayed presentation; similarly, small-bowel obstruction outcomes depend on early differentiation between simple obstruction amenable to conservative therapy and strangulation requiring urgent operation [39][40]. Across these scenarios, prognosis improves when care is delivered within coordinated, protocolized systems that emphasize rapid triage, analgesia, resuscitation, risk-stratified imaging, and early specialty involvement. Ultimately, while many episodes of acute abdominal pain resolve favorably, the subset driven by time-critical pathology demands vigilant surveillance and expeditious intervention to optimize survival and functional recovery [39][40][2][6][7][8].

Complications

If unrecognized or inadequately treated, an acute abdomen can precipitate a cascade of systemic and local complications that substantially increase morbidity and mortality. Sepsis arises when uncontrolled intraabdominal infection drives a

dysregulated host response, progressing from bacteremia to septic shock with cardiovascular collapse, coagulopathy, and multiorgan dysfunction. Prolonged ischemia from strangulated obstruction or mesenteric hypoperfusion culminates in transmural necrosis or gangrene of the bowel, with subsequent bacterial translocation, perforation, and diffuse peritonitis. Loss of mucosal integrity and ongoing inflammation promote abnormal epithelial tract formation, resulting in external or enteroenteric fistulas that perpetuate fluid, electrolyte, and protein losses and complicate nutritional rehabilitation. Mortality increases sharply when source control is delayed, particularly in elderly or immunocompromised patients, underscoring the time-critical nature of definitive intervention. Even after operative management, secondary wound complications—such as surgical site infection, dehiscence, and incisional hernia—may occur, especially in the setting of contamination, malnutrition, or poorly controlled diabetes. Hemodynamic instability, third-spacing from peritonitis, and nephrotoxic exposures contribute to acute kidney injury, which portends longer hospitalization, greater need for renal replacement therapy, and worse long-term outcomes. Prevention hinges on early recognition, prompt resuscitation, targeted broad-spectrum antibiotics when infection is suspected, and timely source control. Meticulous postoperative care—including judicious fluids, glycemic and temperature control, venous thromboembolism prophylaxis, and early nutrition—mitigates complications and improves survival [40].

Enhancing Healthcare Team Outcomes

The management of an acute abdomen requires an integrated, multidisciplinary approach in which radiologists, laboratory personnel, and medical secretaries play pivotal yet often underrecognized roles. Each discipline contributes distinct expertise that collectively drives diagnostic precision, timely intervention, and efficient coordination of patient care. Given the acute abdomen's potentially life-threatening nature, seamless collaboration among these professionals ensures that diagnosis and treatment occur without delay, minimizing morbidity and mortality. Radiologists occupy a central position in the diagnostic process. The early and accurate interpretation of imaging—such as plain radiographs, ultrasound, CT, or MRI—determines both the urgency and direction of care. Radiologists assess findings consistent with bowel obstruction, ischemia, perforation, or vascular compromise, and their reports often dictate the need for surgical consultation or emergent intervention. In many institutions, interventional radiologists extend their role from diagnosis to minimally invasive treatment, performing image-guided drainage of abscesses, embolization for hemorrhage control, or percutaneous gastrostomy placement. Their ability to provide real-time imaging feedback and rapid communication with the surgical

and emergency teams directly influences patient outcomes. Adherence to the ACR Appropriateness Criteria and institution-specific imaging protocols allows radiologists to optimize diagnostic yield while reducing radiation exposure and resource overuse [40].

Laboratory professionals are equally essential to the early identification of life-threatening conditions. Rapid processing and validation of laboratory results—such as complete blood counts, electrolytes, liver function panels, amylase and lipase, lactate, and inflammatory markers—support the clinical team in distinguishing between inflammatory, ischemic, and infectious causes of abdominal pain. In suspected sepsis or perforation, prompt identification of leukocytosis, elevated lactate, or positive blood cultures guides antimicrobial therapy and resuscitation strategies. Laboratory technologists' precision, quality control, and efficiency underpin the reliability of diagnostic data, ensuring that clinical decisions are based on accurate information. Their collaboration with clinicians in specimen prioritization and critical value reporting is vital in emergencies. Medical secretaries serve as the administrative backbone of coordinated care. They ensure that patient records, imaging results, laboratory findings, and specialist consultations are efficiently documented and communicated among departments. In fast-paced emergency and surgical settings, secretaries manage scheduling for imaging studies, operating room availability, and interdepartmental communication, minimizing delays in diagnosis and treatment. By maintaining accurate electronic health records, they facilitate continuity of care, prevent duplication of investigations, and enhance compliance with institutional and legal documentation standards [40].

Ultimately, the success of acute abdomen management depends on the synergy between clinical expertise, diagnostic accuracy, and organizational coordination. Radiologists deliver critical visual insights, laboratories provide biochemical confirmation, and medical secretaries ensure operational coherence across the continuum of care. Together, these professionals form the diagnostic and logistical framework that enables physicians and surgeons to make timely, evidence-based decisions. Strengthening interprofessional communication, implementing standardized protocols, and promoting mutual respect among all team members lead to improved diagnostic efficiency, reduced complications, and enhanced patient satisfaction. In this integrated system, each discipline's contribution is indispensable, ensuring that patients presenting with acute abdominal pain receive comprehensive, coordinated, and high-quality care [40].

Conclusion:

In conclusion, the effective diagnosis and management of the acute abdomen demand a highly coordinated, interdisciplinary approach. This process begins with a meticulous clinical assessment,

integrating a detailed history and physical examination to form a preliminary differential diagnosis. This initial evaluation must then be systematically supported by targeted radiological imaging and laboratory investigations to confirm or rule out life-threatening conditions. The prognosis for patients hinges on the speed and accuracy of this diagnostic triage, followed by prompt intervention. Ultimately, optimal patient outcomes are achieved through seamless collaboration between emergency physicians, radiologists, surgeons, and nursing staff, all supported by efficient administrative systems. A protocol-driven strategy that prioritizes rapid resuscitation, timely analgesia, and early specialty consultation is essential. By uniting clinical acumen with advanced diagnostics within a structured, team-based framework, healthcare providers can ensure that critical, time-sensitive pathologies are identified and managed effectively, thereby significantly reducing morbidity and mortality associated with the acute abdomen.

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