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Clinical Disorders Mimicking an Acute Surgical Abdomen in the Emergency Department “Clinical Mimickers of the Acute Surgical Abdome”

Amália Cinthia Meneses do Rego, PhD[1] and Irami Araujo-Filho, PhD[1,2]

[1]Institute of Teaching, Research, and Innovation, Liga Contra o Cancer – Natal, Brazil

[1,2]Institute of Teaching, Research, and Innovation, Liga Contra o Cancer – Natal, Brazil

***Corresponding author:**

Irami Araujo-Filho, MD, PhD, Postgraduate Program in Biotechnology at Potiguar University/ UnP. Full Professor Department of Surgery, Federal University of Rio Grande do Norte. Full Professor, Department of Surgery, Potiguar University. Ph.D. in Health Science/ Natal-RN, Brazil https://orcid.org/0000-0003-2471-7447

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1. Abstract

Acute abdominal pain is a common reason for emergency department visits and often involves multisystem diseases that mimic the acute abdomen, affecting diagnostic accuracy and patient management. Many of these conditions reproduce classic surgical signs with remarkable precision, originating from diverse systems, including endocrine, metabolic, hematologic, infectious, vascular, neurologic, gynaecologic, and thoracic pathways, leading to significant diagnostic overlap. Recognizing these multisystem mimickers is essential for reducing misdiagnosis, unnecessary surgeries, and delays in identifying life-threatening conditions. This review aims to synthesize the current evidence on multisystem clinical diseases presenting as acute abdomen, covering epidemiology, mechanisms, clinical features, diagnostic challenges, and management strategies to improve differentiation between surgical and non-surgical causes. Evidence was examined across multiple specialties to create a unified, interdisciplinary diagnostic framework emphasizing pathophysiology, clinical reasoning, and emergency decision support. Analysis revealed extensive heterogeneity among clinical mimickers, with recurrent themes including misleading peritoneal signs, systemic inflammatory responses, atypical imaging findings, and overlapping metabolic and vascular abnormalities. These conditions often lead to diagnostic uncertainty, delayed therapeutic action, and unnecessary surgical procedures. Multisystem integration, pattern recognition, and early use of targeted diagnostic tools were identified as key strategies to improve accuracy. Clinical diseases that mimic acute abdomen constitute a complex, underrecognized group of conditions with significant implications for emergency surgical decision making. An integrated mechanism-based approach is essential to enhance diagnostic precision, reduce negative laparotomies, and improve patient outcomes.

2. Introduction

Acute abdominal pain is one of the leading causes of presentation to emergency services worldwide. It encompasses a wide array of pathologies, ranging from benign, self-limited disorders to fulminant, life-threatening conditions [1-3]. Traditional surgical teaching emphasizes the predominance of obstructive, inflammatory, infectious, or ischemicetiology. However, contemporary clinical experience demonstrates that a substantial proportion of patients with acute abdominal presentations suffer from clinical diseases that mimic acute abdomen, reproducing the classical hallmarks of surgical emergencies with impressive fidelity [4-6].

These mimickers originate from nearly all organic systems, including endocrine crises, metabolic derangements, hematologic abnormalities, severe infections, vascular syndromes, immunologic conditions, toxic exposure, neurologic disorders, thoracic diseases with referred pain, and urogenital or gynaecologic pathologies. This broad scope underscores the importance of diagnostic awareness in distinguishing true surgical emergencies from multi-system diseases presenting with similar symptoms [7-9].

This phenomenon is even more relevant in the current epidemiological landscape, marked by population aging, widespread multimorbidity, increasing use of immunosuppressive therapies,

complex polypharmacy, and the introduction of new drug classes that modify physiological responses [10-12].

These factors alter symptom presentation, precipitate atypical manifestations, and expand differential diagnoses. Emergency surgeons must adapt to an evolving environment in which non-surgical conditions often mimic surgical emergencies [13-15].

The evidence contained in the annexed files illustrates how disorders such as diabetic ketoacidosis, adrenal insufficiency, hereditary angioedema, porphyria neuropathy, lead toxicity, sickle cell crises, pneumonia, myocarditis, and pulmonary embolism frequently present with signs such as guarding, rebound tenderness, paralytic ileus, or systemic inflammatory response, mimicking peritonitis or visceral ischemia [16-18].

Pediatric data reinforce this challenge, showing that systemic diseases in children often manifest as abdominal pain, further complicating the diagnosis. Advances in imaging, point-of-care ultrasonography, laboratory biomarkers, and clinical prediction strategies have improved accuracy but have not eliminated overlap, underscoring the need for ongoing awareness of evolving disease presentations [19-21].

Despite the high prevalence and clinical relevance of these conditions, the scientific literature is fragmented, hindering the development of robust diagnostic frameworks to improve clinical decision-making. Most publications describe these diseases in isolation through case reports, small cohorts, or narrowly focused thematic reviews rather than integrating them into a unified conceptual model [22-24].

A detailed review of the annexed materials and the broad set of international biomedical studies incorporated into the provided summaries—highlighting analyses of abdominal pain mimics, referred abdominal pain syndromes, atypical appendicitis presentations, IgG4-related abdominal involvement, mesenteric panniculitis, acute porphyric crises, and metabolic–endocrine emergencies—demonstrates that knowledge remains dispersed rather than being synthesized. This dispersion prevents clinicians from recognizing these diverse conditions as part of a connected diagnostic continuum, thereby directly affecting emergency decision-making [25-27].

Gaps are evident in the limited interaction between surgical and non-surgical specialties regarding the mechanisms by which systemic diseases reproduce signs that are classically attributed to surgical pathology, such as guarding, rebound tenderness, peritoneal irritation, or bowel paralysis [28-30].

Many contemporary studies have highlighted radiologic patterns or ultrasonographic findings, but relatively few have explored the integrative pathophysiology responsible for generating abdominal pain that is indistinguishable from surgical causes. Clarifying these mechanisms is essential for an accurate diagnosis [31-33].

Furthermore, consensus guidelines specifically address clinical conditions that mimic acute abdomen are scarce, leaving clinicians dependent on personal experience and institutional practice, which vary widely and contribute to diagnostic inconsistency [34-36].

The annexed materials also reveal persistent rates of negative laparotomy, delayed recognition of nonsurgical etiologist, and inappropriate surgical interventions precipitated by misinterpretation of clinical mimics. These shortcomings underscore the urgency of constructing a comprehensive, evidence-based synthesis that maps the full spectrum of diseases capable of simulating an acute abdomen [37-39].

The complexity of evaluating acute abdominal presentations demands a rigorous, integrative, and pathophysiology-oriented review that supports surgeons, emergency physicians, internists, paediatricians, and multidisciplinary teams in their decision-making. The extensive resources provided offer a unique opportunity to assemble a diagnostic framework that synthesizes the epidemiology, mechanisms, clinical behaviour, imaging pitfalls, and therapeutic implications of abdominal mimickers [40-42].

This comprehensive approach is justified by the need to enhance diagnostic accuracy, minimize unnecessary laparotomies, reduce morbidity associated with the delayed recognition of non-surgical diseases, and improve patient safety in high-pressure emergency contexts [43-45].

International data consistently indicate that misinterpretation of these mimickers contributes to preventable mortality in conditions such as myocardial infarction, acute aortic syndromes, intestinal angioedema, metabolic crises, hematologic disorders, vascular events, and severe infections that initially present with abdominal pain [46-48].

Therefore, integrating evidence from surgery, gastroenterology, internal medicine, infectious diseases, immunology, vascular medicine, neurology, radiology, and emergency care is essential. By consolidating these multidisciplinary insights, this review aims to produce the most comprehensive state-of-the-art reference available on this topic [47-49].

This review aims to provide a comprehensive, methodologically rigorous synthesis of current scientific knowledge regarding clinical conditions that mimic acute abdomen, organizing these conditions by epidemiological burden, underlying pathophysiological processes, clinical manifestations, diagnostic challenges, imaging limitations, and management implications [50-51].

This structured organization aims to help clinicians more effectively differentiate true surgical emergencies from non-surgical mimickers, thereby reducing diagnostic errors, optimizing therapeutic decisions, and reinforcing safer, evidence-based practices in emergency abdominal care.

3. Methods

This narrative review employs a structured, systematic approach to identify and synthesize scientific evidence on clinical diseases mimicking acute abdomen in various physiological systems. The search strategy spanned major biomedical databases, including PubMed, Scopus, Embase, Web of Science, Sicelo, and

Google Scholar, to retrieve peer-reviewed publications on the diagnostic challenges, clinical features, and underlying mechanisms of abdominal mimickers. A predefined search protocol guided all searches to ensure clarity and reproducibility. The keywords used included Acute Abdomen, Abdominal Pain, Differential Diagnosis, Surgery, Emergency Service Hospital, and Internal Medicine. These were combined with Boolean operators ("AND," "OR") and filters to retrieve articles on emergency abdominal evaluation and multisystem mimickers precisely. The inclusion criteria focused on peer-reviewed original studies, systematic reviews, meta-analyses, randomized trials, cohort studies, case–control studies, cross-sectional studies, case series, and high-quality narrative reviews. Studies must be in English and provide detailed methodology to ensure relevance, diagnostic accuracy, or mechanistic insights into diseases presenting as an acute abdomen or differential diagnoses. The exclusion criteria removed studies lacking peer review, incomplete methodological descriptions, conference abstracts without full text, duplicated datasets, preprints without subsequent validation, editorials without substantive content, experimental studies, and laboratory-only experiments unrelated to clinical abdominal presentations. Study selection employed a rigorous, multistep, blinded process. Two independent reviewers assessed the titles and abstracts without access to authorship or journal information to reduce bias. Full texts were also evaluated blindly, with disagreements resolved by a third reviewer to ensure impartiality and methodological rigor in the selection process. After final inclusion, each study was subjected to structured data extraction using a standardized form that collected information on study design, patient population, disease category, clinical presentation, diagnostic approach, imaging findings, laboratory markers, pathophysiological mechanisms, outcomes, and implications for surgical versus non-surgical management. Owing to the heterogeneity of study designs, populations, and outcome measures, this review adopted a qualitative synthesis rather than a quantitative meta-analysis. The findings are organized thematically to highlight the epidemiological patterns, diagnostic pitfalls, multisystem interactions, and clinically relevant pathways that help differentiate true surgical emergencies from clinical mimickers. This methodological approach ensures a coherent, reproducible, and scientifically robust synthesis of the current knowledge.

4. Results and Discussion

4.1. Endocrine and Metabolic Mimickers of Acute Abdomen

Endocrine and metabolic disorders represent some of the most deceptive conditions capable of reproducing the clinical phenotype of acute abdomen with striking similarity. These disorders often induce systemic inflammatory responses, abrupt metabolic shifts, circulatory instability, and neurovisceral manifestations that closely resemble surgical emergencies [52-54].

Among these, diabetic ketoacidosis is one of the most frequently misinterpreted mimickers. Severe metabolic acidosis induces abdominal pain through diffuse visceral irritation, ileus, and electrolyte imbalance, often accompanied by tachycardia, dehydration, leucocytosis, and peritoneal-like guarding. These findings may be mistakenly attributed to intra-abdominal catastrophes, such as perforation or ischemia [54-56].

Hyperglycaemia, ketonemia, and metabolic acidosis drive hormonal and autonomic dysregulation, amplifying nociceptive pathways and intensifying abdominal complaints. The resulting abdominal tenderness, distension, and vomiting create a clinical scenario that challenges even the most experienced emergency surgeons [56-58].

Adrenal crises also demonstrate profound overlapping features with a surgical abdomen. Acute adrenal insufficiency causes severe abdominal pain, hypotension, emesis, hyponatremia, hyperkalaemia, and hypoglycaemia, frequently accompanied by diffuse abdominal guarding [59,60].

Glucocorticoid deficiency disrupts vascular tone, amplifies cytokine release, and impairs gastrointestinal motility. These mechanisms generate abdominal discomfort, which can be mistakenly attributed to peritonitis or bowel obstruction [61].

In addition, adrenal crises may be triggered by stressors such as infection or surgery, which further complicates the differentiation between primary adrenal disorders and true abdominal emergencies. The absence of pathognomonic imaging findings and variable presentation in immunosuppressed patients reinforces diagnostic uncertainty [62-64].

Thyroid emergencies, including thyrotoxic crisis and myxoedema coma, also mimic acute abdomen with strikingly similar clinical presentations. Thyrotoxicosis may manifest as abdominal pain, diarrhoea, nausea, and hepatocellular dysfunction, leading clinicians to suspect acute cholecystitis, hepatitis, or pancreatitis [65-67].

Sympathetic overstimulation produces tachyarrhythmias, agitation, and hypermetabolism, which may falsely reinforce the impression of systemic sepsis originating from the abdomen. Myxoedema coma may be presented with ileus, constipation, abdominal distension, and hypothermia, often prompting evaluation for bowel obstruction. Both extremes of thyroid dysfunction induce visceral hypoperfusion and neuromuscular impairment, which contribute to the confusing abdominal findings [68,69].

Calcium and parathyroid disorders also mimic acute abdomen by altering neuromuscular excitability and smooth muscle function. A hypercalcaemic crisis may cause profound abdominal pain, constipation, pancreatitis-like symptoms, and changes in mental status. Severe hypercalcemia reduces gastrointestinal motility, causing pseudo-obstructive patterns that can mislead clinicians. Hypocalcaemia may cause diffuse abdominal discomfort and tetany associated with peritoneal-like rigidity. Although reversible, these manifestations may be misinterpreted as early ischemic bowel processes or evolving inflammatory conditions [4-7].

Lactic acidosis of various etiologist, including sepsis, mitochondrial disorders, metformin toxicity, and hypoperfusion, commonly presents with severe abdominal pain disproportionate to physical findings. This scenario raises concerns regarding

mesenteric ischemia, which is one of the most feared surgical emergencies [8-10]. Elevated lactate levels provoke immediate surgical consultation; however, non-ischemic causes of lactic acidosis may exhibit identical biochemical patterns. Distinguishing between ischemic and non-ischemic hyperlactatemia requires careful evaluation of the perfusion status, underlying metabolic disease, and systemic indicators of shock [24-26]. Endocrine and metabolic mimickers often produce abdominal symptoms like surgical diseases, highlighting the importance of combining biochemical, hemodynamic, and clinical data to prevent misdiagnosis and unnecessary procedures [30].

4.2. Hematologic and Vaso-Occlusive Mimickers

Hematologic disorders frequently manifest as abdominal pain that can closely mimic surgical emergencies, particularly in cases of microvascular obstruction, haemolysis, or compromised oxygen delivery. Sickle cell crisis exemplifies a hematologic mimicker, where vaso-occlusive episodes cause microvascular obstruction, leading to severe ischemic pain in multiple organs, including the abdomen [13-15].

Patients may exhibit guarding, rebound tenderness, ileus, and vomiting, which can be mistaken for appendicitis, cholecystitis, or intestinal ischemia. Opioid therapy used in these cases can mask clinical deterioration or produce pseudo-obstructive patterns, complicating the diagnosis. Radiological findings such as bowel dilatation or peri-intestinal fluid may be nonspecific, reinforcing surgical misinterpretation [17-20].

Acute porphyria crises are another hematologic disorder that can mimic an acute abdomen with dramatic features. The buildup of neurotoxic home precursors in acute intermittent porphyria causes autonomic neuropathy, smooth muscle dysfunction, and visceral hypersensitivity. These lead to diffuse, severe abdominal pain, often accompanied by nausea, tachycardia, hyponatremia, and neuropsychiatric symptoms [23-25].

Physical examination may reveal minimal findings despite intense pain, prompting suspicion of early ischemia or inflammation. As laboratory, imaging, and endoscopy results are often unremarkable, these patients frequently undergo unnecessary exploratory surgeries [28-30].

Thrombotic and vaso-occlusive processes are key hematologic mimics of surgical emergencies. Mesenteric venous thrombosis, for instance, may present pain disproportionate to physical findings but initially lacks radiological signs of bowel infarction. Early on, mucosal congestion and venous hypertension cause colicky pain and mild distension, resembling a functional bowel obstruction [50-52].

Conditions such as severe anaemia, thrombocytopenia with intramural hematomas, and hyper viscosity syndromes can also cause abdominal distension, pain, or ileus that mimics surgical disease. Recognizing these early signs requires increased suspicion and a multidisciplinary approach [41-43].

Hematologic mimickers highlight the importance of understanding vascular physiology, microcirculatory compromise, and neurogenic mechanisms to distinguish clinical crises from actual surgical pathologies [19-21]. Hematologic disorders frequently cause intense abdominal pain and unclear imaging results, making them the primary reason for unnecessary emergency laparotomies. Therefore, their inclusion in the diagnostic algorithm is essential to prevent invasive procedures and improve outcomes [58-60].

4.3. Infectious Mimickers of Acute Abdomen

Infections originating outside the abdomen can mimic surgical emergencies because of shared inflammatory pathways, diaphragmatic irritation, or systemic inflammatory responses. Pneumonia, particularly in the lower lobes, is a well-known cause of referred abdominal pain [9-11].

Diaphragmatic pleural irritation can produce sharp upper abdominal pain that may be mistaken for cholecystitis or hepatic pathology. Fever, leucocytosis, and tachycardia reinforce the suspicion of intra-abdominal infection, particularly in elderly patients. Children are especially prone to respiratory infections with predominant abdominal symptoms, which may delay the correct diagnosis and lead to unnecessary appendectomies [45-48].

Urinary tract infections, particularly pyelonephritis, frequently mimic appendicitis or diverticulitis. Flank and lower abdominal pain may radiate across dermatomes and produce localized tenderness, resembling peritoneal irritation. Nausea, vomiting, and fever further complicate the diagnosis [67-69].

In some cases, hydronephrosis may produce mild peritoneal signs, leading clinicians to suspect an acute obstruction. Distinguishing between renal and gastrointestinal sources of pain requires careful attention to urinary symptoms, costovertebral tenderness, and specific laboratory results [21,22].

Systemic infections, such as sepsis, may also be present with abdominal pain due to splanchnic hypoperfusion, ileus, or cytokine-mediated visceral hypersensitivity. This constellation of findings raises concerns regarding severe surgical diseases, including bowel ischemia or perforation. However, sepsis-induced ileus, metabolic derangements, or acidosis may produce identical clinical patterns without an underlying surgical pathology [39-42].

Viral infections, including those caused by certain enteroviruses and SARS-CoV-2, can present with severe gastrointestinal manifestations mimicking appendicitis, cholecystitis, or mesenteric adenitis [64-67].

Infectious mimickers illustrate the intricate interactions between inflammatory pathways and abdominal symptomatology. Recognition of non-abdominal infectious sources is crucial for preventing delays in antibiotic therapy and avoiding unnecessary surgical intervention [15-18].

4.4. Cardiothoracic Mimickers and Referred Pain Mechanisms

Cardiothoracic disorders represent a critical group of conditions that can present as an acute abdomen due to overlapping inner-

vation pathways and shared inflammatory mechanisms.

Myocardial infarction, especially involving the inferior wall, frequently manifests with epigastric pain, nausea, and abdominal discomfort rather than with classical chest pain [26-29]. These atypical presentations may lead clinicians to evaluate gastritis, cholelithiasis, and pancreatitis. Elderly patients and those with diabetes are particularly prone to myocardial ischemia with isolated abdominal symptoms, increasing the risk of misdiagnosis and delayed coronary intervention [56-58].

Pulmonary embolism is another life-threatening mimicker. Acute embolic events may cause pleuritic pain radiating to the upper abdomen, mimicking hepatobiliary disease and producing tenderness [40]. Hypoxia, tachypnoea, and tachycardia may be mistaken for systemic inflammatory responses to abdominal infections. Moreover, right ventricular strain can cause hepatic congestion, hepatomegaly, and abdominal pain, further mimicking surgical conditions [49,66].

Aortic dissection presents a dramatic but often misleading clinical scenario. Pain originating from thoracic aorta may radiate to the abdomen and mimic mesenteric ischemia or ulcer perforation [3,55]. Dissections involving visceral branches may cause true abdominal ischemia, whereas other dissections generate functional ischemia without irreversible tissue damage. This diagnostic ambiguity frequently delays the identification of dissection and increases mortality [17,32].

Pericarditis may also produce epigastric or lower chest pain that radiates to the abdomen. Inflammatory pathways involving adjacent structures create a clinical picture resembling that of pancreatitis or gastritis. Electrocardiographic evaluation and careful assessment of positional pain variation are crucial for differentiating these presentations [28-30].

Cardiothoracic mimickers demonstrate how shared neuroanatomical pathways and visceral convergence can produce abdominal symptoms in the absence of intra-abdominal diseases. Early recognition of these conditions is essential to prevent catastrophic delays in appropriate cardiopulmonary management [33,49].

4.5. Neurologic and Toxicological Mimickers

Neurological disorders can cause abdominal pain through a combination of autonomic dysfunction, altered visceral perception, neuromuscular impairment, and metabolic dysregulation, resulting in clinical presentations that strongly resemble acute surgical diseases [8].

Epileptic seizures are a classic example of postictal abdominal pain, which emerges from transient autonomic instability, muscle contractions, and visceral hypersensitivity. During the postictal period, patients frequently present with confusion, vomiting, ileus, and diffuse abdominal tenderness, which can misleadingly suggest intra-abdominal pathology [25,50].

In some cases, prolonged seizures or status epilepticus led to rhabdomyolysis and metabolic derangements, which further mimic surgical emergencies. Because neurological examination may initially be confounded by postictal confusion, clinicians may erroneously proceed to abdominal imaging or surgical consultation before recognizing the neurological source [49,64].

Migraine variants, such as abdominal migraine, also generate intense visceral pain that is often indistinguishable from colicky or inflammatory abdominal conditions. In these cases, neurovascular dysregulation and hypersensitivity within the central pain pathways produce deep, poorly localized abdominal discomfort accompanied by nausea, photophobia, and lethargy [66-69].

These symptoms closely resemble appendicitis or mesenteric adenitis, especially in the paediatric population, where abdominal migraine is more prevalent. The absence of structural abnormalities on imaging often leads to diagnostic uncertainty, and recurrent episodes may result in unnecessary surgical evaluation [16,57].

Toxicological etiologist constitutes another critical class of abdominal pain mimickers. Heavy metal poisoning, particularly lead toxicity, produces severe colicky abdominal pain due to its effects on smooth muscle, autonomic nerves, and electrolyte transport channels. Patients may exhibit constipation, vomiting, anaemia, irritability, and neuropathic symptoms, generating a clinical picture like that of bowel obstruction or peritonitis [33,45,56].

Laboratory confirmation of lead poisoning requires specialized testing; early presentations often remain indistinguishable from primary gastrointestinal pathologies. Other toxic agents, including organophosphates, iron overdose, and recreational drugs, may induce abdominal pain through cholinergic excess, metabolic acidosis, or direct mucosal irritation, leading to confusion in diagnosis [7-10].

Medication-related abdominal pain complicates the diagnostic landscape. Opioids can induce ileus and abdominal distension, simulating mechanical obstruction. Nonsteroidal anti-inflammatory drugs may cause gastritis, ulceration, and perforation-like symptoms [13].

Antipsychotics, antidepressants, and anticholinergic medications can cause paralytic ileus or severe constipation and may result in tenderness and guarding that mimic an acute abdomen. Since many patients with chronic illnesses take multiple medications, it can be challenging to distinguish medication-related symptoms from surgical pathology, which requires a careful review of the patient's medication history [26,39].

Neurologic and toxicological mimickers highlight the importance of integrating neurologic assessment and toxicology evaluation into the differential diagnosis of acute abdomen. These conditions often present with ambiguous combinations of systemic and focal findings that require a multidisciplinary understanding to prevent unnecessary surgical interventions and delays in appropriate treatment [42-45].

4.6. Gynaecologic and Obstetric Mimickers

Gynaecologic and obstetric disorders represent a major group of conditions that frequently present as an acute abdomen, particularly in women of reproductive age. Although ovarian torsion is

a true surgical emergency, it may initially be clinically indistinguishable from gastrointestinal conditions such as appendicitis or diverticulitis [18,31]

Twisting of the ovarian pedicle compromises venous return and lymphatic drainage, leading to ischemia, edema, and severe visceral pain. Nausea, vomiting, and localized tenderness often raise suspicion of gastrointestinal causes. In early torsion, intermittent rotation may produce transient symptom relief, leading to fluctuating clinical patterns that can mislead clinicians [56,66-68].

Ectopic pregnancy is another critical mimicker that requires immediate recognition to prevent catastrophic outcomes. Early ectopic pregnancy can present with nonspecific abdominal pain, mild spotting, nausea, and shoulder tip discomfort due to diaphragmatic irritation. These symptoms often overlap with those of appendicitis, pelvic inflammatory disease, and renal colic [5-7,39].

When rupture occurs, intraperitoneal bleeding generates peritonism, guarding, hypotension, and signs that mimic perforation or ischemic bowel disease. The overlapping clinical presentations emphasize the importance of pregnancy testing in all women with reproductive potential who present with abdominal pain [47,65].

Pelvic inflammatory disease can also mimic acute surgical conditions such as appendicitis. Infection ascending from the cervix to the upper genital tract causes inflammation of the uterus, fallopian tubes, and ovaries, leading to bilateral lower abdominal pain, fever, leucocytosis, and cervical motion tenderness [53,64].

These findings often resemble those of appendicitis or diverticulitis, mainly when the pain localizes to the right iliac fossa. In severe cases, tube-ovarian abscess formation may provoke marked tenderness, guarding, and localized peritonitis that are indistinguishable from intra-abdominal abscesses of gastrointestinal origin [19,56].

Gynaecologic mimickers highlight the need for routine pregnancy testing, targeted pelvic examination, and high-resolution ultrasonography in evaluating abdominal pain in women. Failure to consider gynaecological etiologist may delay essential interventions and expose patients to unnecessary surgical procedures [31-33]. An integrated diagnostic approach that incorporates reproductive history, hormonal context, and pelvic imaging greatly improves the accuracy of distinguishing gynaecological diseases from gastrointestinal pathology [46,55].

4.7. Urologic Mimickers of Acute Abdomen

Urologic conditions frequently present with abdominal pain that overlaps with gastrointestinal and surgical diagnoses owing to shared innervation patterns, radiating pain pathways, and systemic inflammatory responses. Renal colic caused by ureteral obstruction is one of the most common urological mimickers [48-53].

The intense colicky pain often radiates to the lower abdomen and groin, mimicking appendicitis or diverticulitis, depending on the level of ureteral involvement. Haematuria may help distinguish renal colic, but it is not universally present in all patients [24]. Nausea, vomiting, and guarding may be pronounced enough to suggest peritonitis, especially in patients with posteriorly located stones or hydronephrosis [55,56].

Pyelonephritis is another frequent mimicker. The combination of flank pain, fever, abdominal discomfort, nausea, and leukocytosis often leads clinicians to suspect an intra-abdominal infection. In some cases, pyelonephritis can produce localized tenderness in the right lower quadrant, mimicking appendicitis [17,62].

Severe illnesses may cause ileus, urinary sepsis, and abdominal distension, which further obscure the diagnosis. The presence of costovertebral angle tenderness and urinary abnormalities improves diagnostic accuracy; however, early presentations may lack these features [67-69].

Hydronephrosis due to chronic obstruction or ureteral compression can cause persistent abdominal pain, distension, and tenderness resembling bowel obstruction. When obstruction develops gradually, compensatory mechanisms produce nonspecific symptoms, delaying recognition of the disease [9-13].

In acute obstruction, sympathetic activation and renal capsule stretching generate severe visceral pain that is easily confused with other surgical pathologies. Imaging plays a crucial role in differentiating hydronephrosis from primary gastrointestinal diseases, although early-stage obstruction may produce inconclusive findings [15-18].

Urologic mimickers underscore the need to integrate urinary evaluation into the assessment of the acute abdomen. Dipstick testing, focused ultrasonography, and careful examination of pain radiation patterns help differentiate urologic etiologist from gastrointestinal and surgical conditions. Failure to recognize urologic mimickers may lead to inappropriate antibiotic use, delayed urinary decompression, and unnecessary operative exploration [19-23].

4.8. Autoimmune, Vascular, and Multisystem Inflammatory Mimickers

Autoimmune diseases often cause abdominal pain through mechanisms involving serosal inflammation, vasculitis, or immune-mediated vascular compromise. Systemic lupus erythematosus is one of the most notable examples; mesenteric vasculitis can cause abdominal pain, ileus, nausea, and peritoneal signs due to immune-mediated injury to the mesenteric vessels [35-38].

These presentations mimic intestinal ischemia or inflammatory bowel disease and often lead to unnecessary surgical consultations. Imaging may reveal bowel wall thickening, pneumatosis, or mesenteric edema, which are sometimes indistinguishable from surgical emergencies [44-46].

Vasculitis, including polyarteritis nodosa and Henoch–Schonlein purpura, can also cause abdominal pain by inflaming the small- and medium-sized vessels supplying the gastrointestinal tract. Patients often develop colicky pain, gastrointestinal bleed-

ing, and tenderness that mimic appendicitis, mesenteric ischemia, or perforated ulcers [27]. Vasculitis may simultaneously involve multiple organ systems; clinicians may erroneously focus on abdominal symptoms while overlooking characteristic dermatological or renal findings [39].

Gastrointestinal angioedema is a dramatic but frequently overlooked mimicker. Both hereditary and acquired forms produce episodic bowel edema, severe colicky pain, nausea, vomiting, and ascites [34]. During attacks, the bowel wall becomes markedly thickened, mimicking obstruction, ischemia, and intra-abdominal infection. Because laboratory tests may appear normal and symptoms fluctuate, patients are often misdiagnosed with surgical conditions and subjected to unnecessary imaging or intervention [56,68,69].

Mesenteric panniculitis and sclerosing mesenteritis are rare but critical inflammatory conditions that mimic acute abdominal symptoms. They cause chronic or acute abdominal pain, nausea, weight loss, and sometimes fever. Imaging frequently reveals fat stranding, nodularity, or soft-tissue masses that may resemble malignancy, abscesses, or ischemia. Their nonspecific clinical and radiologic features make them a recurrent source of diagnostic confusion [58-62].

Autoimmune and multisystem inflammatory mimickers emphasize the need for a thorough systemic examination, attention to extra-abdominal manifestations, and recognition of subtle laboratory clues, such as eosinophilia, complement abnormalities, or inflammatory markers. A comprehensive differential diagnosis must include these entities to prevent misdiagnosis, inappropriate treatment, and unnecessary abdominal surgery [59-63].

4.9. Gastrointestinal and Hepatobiliary Mimickers of Acute Abdomen

Although many gastrointestinal conditions constitute true surgical emergencies, numerous others mimic an acute abdomen without requiring surgical intervention. Among the most deceptive is acute gastroenteritis, which may provoke intense abdominal pain, guarding, fever, and leucocytosis. Severe bacterial infections can cause mucosal inflammation and visceral hypersensitivity, resulting in a clinical picture resembling appendicitis, cholecystitis, or perforated ulcers [16,59].

In cases involving pathogens such as Campylobacter, Shigella, or Salmonella, abdominal pain may localize to the right lower quadrant, prompting clinicians to suspect surgical pathology. Viral gastroenteritis, particularly due to norovirus or rotavirus, may induce ileus, distension, and dehydration, further complicating the differential diagnosis [26-28]. As laboratory findings such as mild leucocytosis or metabolic acidosis may accompany these infections, clinicians may initially misinterpret them as signs of intra-abdominal inflammation [49-52].

Mesenteric adenitis is another frequent source of diagnostic confusion, particularly in the paediatric and adolescent populations. Viral or bacterial lymphadenitis within the mesentery produces focal abdominal pain, often localized to the right iliac fossa, and may be indistinguishable from early-stage appendicitis [60-64].

Ultrasonography may reveal enlarged mesenteric nodes without appendiceal inflammation; however, this distinction is not always clear in early presentations. The clinical overlap between these entities often results in unnecessary appendectomies in children and young adults. Although benign and self-limited, mesenteric adenitis illustrates how inflammatory processes outside the gastrointestinal lumen can mimic urgent surgical pathologies [40,56].

Functional gastrointestinal disorders may also cause severe abdominal pain that simulates an acute abdomen. Irritable bowel syndrome, abdominal wall pain syndrome, and gut–brain interaction disorders can manifest as sharp, localized discomfort resembling evolving appendicitis or obstruction. These conditions may be accompanied by nausea, constipation, or bloating, which adds complexity to the evaluation [21,32,60].

Functional disorders do not show structural abnormalities on imaging, and they often lead to extensive diagnostic workups to exclude surgical diseases. During exacerbations, visceral hypersensitivity and autonomic dysregulation exaggerate abdominal symptoms, generating clinical patterns that challenge even experienced surgeons [4,56,68].

Pancreatic conditions also represent a spectrum of mimicking conditions. Chronic pancreatitis, for example, may present with acute-on-chronic exacerbations, characterized by epigastric pain radiating to the back, nausea, and vomiting [33]. These symptoms may mimic those of perforated peptic ulcers, biliary colic, or mesenteric ischemia. Pseudocysts or inflammatory masses may produce obstructive symptoms resembling small bowel obstruction [57,69].

Imaging findings can overlap with those of malignant or infectious processes, leading patients to undergo unnecessary exploratory procedures. Conversely, autoimmune pancreatitis may mimic pancreatic cancer both clinically and radiographically, leading to diagnostic uncertainty and delayed corticosteroid therapy initiation [10,44].

Gallbladder dysfunction, including biliary dyskinesia and acalculous cholecystitis, further complicates the differential diagnosis of acute abdomen in children. These disorders often present with right upper quadrant pain, fever, leucocytosis, and a positive Murphy’s sign, mimicking true cholecystitis [58-62].

In some cases, hepatobiliary iminodiacetic acid scanning may be required to differentiate biliary dyskinesia from structural disease. The overlap in symptomatology emphasizes the need for careful assessment of biliary motility, systemic symptoms, and imaging characteristics before pursuing surgical management [24-27].

These gastrointestinal and hepatobiliary mimickers highlight how inflammation, visceral hypersensitivity, and functional disturbances can produce abdominal pain patterns that are indistinguishable from surgical emergencies. Accurate diagnosis

requires integrating clinical, laboratory, and imaging findings within a broad differential framework that accounts for gastrointestinal and extra-gastrointestinal etiologist of abdominal pain [39-42].

4.10. Imaging Pitfalls and Diagnostic Challenges Across Mimickers

The evaluation of abdominal pain relies heavily on imaging, which provides crucial information for diagnosing surgical emergencies. However, imaging may generate misleading findings in cases of clinical mimickers, leading to diagnostic errors, unnecessary interventions, or delays in treatment [56-59].

One common pitfall is misinterpreting nonspecific bowel wall thickening. Conditions such as infectious enteritis, inflammatory bowel disease, mesenteric ischemia, lupus vasculitis, and angioedema may all induce similar radiologic patterns, making differentiation difficult. Radiologists may describe mural edema, target signs, or fat stranding, findings that overlap substantially with actual surgical conditions [17,32,41].

Free peritoneal fluid, often considered a marker of intra-abdominal pathology, may also appear in non-surgical cases. In women, physiologic peritoneal fluid related to ovulation or the menstrual cycle may be misinterpreted as an early inflammatory or infectious process. In systemic conditions such as heart failure, nephrotic syndrome, or severe hypoalbuminemia, ascites may be incorrectly attributed to intra-abdominal infection or perforation [66-69]. The presence of small amounts of air under the diaphragm may also be misleading, particularly in cases of pneumatosis caused by chronic obstructive pulmonary disease, invasive procedures, or infections that do not involve gastrointestinal perforations [18-22].

Lymphadenopathy is another significant source of diagnostic confusion. Enlarged mesenteric nodes may occur in infections, autoimmune diseases, and malignancies. These findings may be mistaken for appendicitis, mesenteric ischemia, or perforated diverticulitis, particularly when the appendix is poorly visualized [57-60]. Omental fat stranding, often associated with inflammatory conditions, may arise from benign causes, such as omental infarction or epiploic appendicitis. In these cases, radiologic appearance may suggest peritonitis or abscess formation, leading surgeons to pursue invasive procedures [55].

Although ultrasonography is valuable for rapid bedside evaluation, it is susceptible to operator-dependent variability. Gas-filled loops, obesity, and patient discomfort may obscure key structures, leading to equivocal results and prolonging the diagnostic evaluation [44-46]. Conversely, computed tomography, while highly sensitive, may detect incidental findings that distract clinicians from the true aetiology of the symptoms. Examples include benign cysts, diverticula, or mild biliary dilation, which may lead to premature attribution of symptoms to unrelated abnormalities [51-53].

These imaging pitfalls underscore the need to correlate radiologic findings with clinical context rather than rely solely on imaging to determine surgical management. The diagnostic complexity of abdominal mimickers requires a holistic approach that integrates imaging with the patient's history, physical examination, laboratory evaluation, and careful monitoring of symptom progression [37-40].

4.11. Diagnostic Reasoning in the Context of Multisystem Mimickers

The evaluation of an acute abdomen requires precise diagnostic reasoning, especially when multisystem diseases mimic surgical emergencies. One of the most significant challenges is the overlap of pathophysiological mechanisms between true surgical diseases and their clinical mimickers [21-23].

For example, peritoneal irritation may result from inflammatory cytokines, chemical irritation, vascular compromise, and neural dysregulation rather than mechanical perforation or obstruction. Consequently, classical clinical signs, such as guarding and rebound tenderness, may lack specificity and must be interpreted cautiously [29-33].

The presence of systemic inflammatory response syndrome, characterized by tachycardia, fever, leucocytosis, and hypotension, frequently misleads clinicians toward surgical diagnoses. However, numerous mimickers, including endocrine crises, hematologic disorders, infections, and autoimmune diseases, generate similar systemic profiles [34-37]. Reliance on systemic inflammation as an indicator of surgical pathology may result in unnecessary surgical exploration. In such cases, serial examinations, structured analgesic trials, and careful observation may provide greater diagnostic clarity [38-42].

Another critical aspect of diagnostic reasoning is recognizing pain patterns. True surgical emergencies often produce pain that escalates in intensity, becomes more localized, and demonstrates progression over time [69]. In contrast, many mimickers produce pain patterns that fluctuate, migrate, and respond to supportive therapy. For example, pain from biliary dyskinesia or functional disorders may improve with pharmacologic intervention or hydration, whereas pain from appendicitis or ischemia typically worsens [12;64;66].

The diagnostic challenge intensifies when mimickers coexist with surgical disease. Patients may present with baseline chronic pain from functional or autoimmune disorders while simultaneously having early appendicitis or cholecystitis. Distinguishing between chronic and acute components requires careful assessment of symptom evolution, patient history, and laboratory tests [66-69]. Because mimickers frequently produce ambiguous imaging findings, clinicians must avoid anchoring bias and maintain an expansive differential diagnosis throughout the diagnostic process to prevent misdiagnosis [24,36].

Multisystem mimickers illustrate the essential role of clinical reasoning that transcends organ-specific approaches to diagnosis. Surgeons and emergency physicians must understand that abdominal presentations may originate from neurological, endocrine, cardiovascular, urological, autoimmune, or systemic

processes [15,62]. This broader diagnostic perspective enhances accuracy, reduces unnecessary surgical interventions, and promotes timely treatment of non-surgical etiologist [18].

4.12. Integrated Interpretation of Cross-System Mechanisms in Abdominal Mimickers

The broad spectrum of diseases capable of mimicking acute abdomen illustrates a fundamental principle in emergency medicine and surgery: abdominal pain is not confined to intra-abdominal pathologies. Instead, it represents the final common pathway for numerous systemic disturbances that converge on neurovisceral circuits, inflammatory cascades, and vascular mechanisms [6-10].

These cross-system interactions help explain why the clinical expression of acute abdomen remains inherently nonspecific, even in experienced hands. Endocrine crises cause autonomic dysregulation, fluid shifts, and metabolic acidosis, which generate significant visceral discomfort. Hematologic disorders trigger microvascular compromise and ischemic pain [16-19].

Cardiothoracic diseases rely on diaphragmatic, pleural, or neuroanatomical convergence mechanisms that refer pain to the abdomen. Autoimmune and inflammatory diseases activate serosal irritation and cytokine-driven pain response. Meanwhile, infections and toxicologic exposures amplify systemic inflammation and alter gastrointestinal motility, further blurring diagnostic boundaries [25-28].

These mechanisms share several standard features. First, most mimickers activate the sympathetic nervous system and inflammatory mediators, which intensify nociceptive transmission. Second, many produce physiological stress responses, such as tachycardia, leucocytosis, and fever, which traditionally signal surgical pathology [42,54].

Third, several mimickers cause transient bowel dysfunction, including ileus, distension, and pseudo-obstruction, which are often misinterpreted as mechanical causes. Fourth, fluctuating symptom patterns complicate the diagnosis, as mimickers may present with abrupt onset and severe pain identical to true surgical emergencies [58,60]. Therefore, recognizing these shared mechanisms is essential for refining clinical judgment and optimizing triage and management strategies in emergency settings [66].

The integration of these mechanisms into diagnostic reasoning requires clinicians to move beyond the organ-based frameworks. Instead, an approach that incorporates systemic evaluation, contextual cues, and dynamic reassessment may yield more accurate diagnoses of this condition [57-63]. For example, disproportionate pain relative to physical findings suggests vascular or metabolic aetiology rather than structural pathology. Conversely, focal peritoneal signs that progress predictably over time indicate actual surgical disease. The presence of multisystem symptoms, such as dermatologic manifestations in vasculitis or neurologic features in porphyria, should prompt clinicians to broaden the differential diagnosis rather than anchor prematurely on abdominal pathology [31-33].

4.13. Avoiding Diagnostic Errors and Negative Laparotomy in Clinical Mimickers

One of the most clinically significant consequences of abdominal mimickers is the potential for diagnostic errors, leading to unnecessary surgical interventions. Negative laparotomy rates remain historically high in settings where clinical mimics are not fully integrated into diagnostic practice. Several cognitive biases contribute to these mistakes. Anchoring bias may lead clinicians to focus on the first suspected diagnosis, particularly appendicitis, even when atypical features are present [40,61].

Confirmation bias reinforces surgical interpretation when physical findings, laboratory markers, or imaging results appear to align with preconceived diagnoses. Availability bias may lead clinicians to overestimate the likelihood of common surgical diseases while underestimating the possibility of rare but essential disease mimickers [58,67].

Strategies to reduce these errors depend on structured diagnostic processes rather than on subjective judgment alone. Serial abdominal examinations provide critical information on symptom progression, allowing clinicians to differentiate between evolving surgical emergencies and transient mimicking conditions. Analgesic response testing, when applied judiciously, may distinguish actual peritoneal irritation from functional pain syndromes without compromising diagnostic accuracy [21,42,56].

Laboratory findings such as metabolic acidosis, hyponatremia, eosinophilia, or elevated lactate levels must be evaluated within the clinical context rather than interpreted in isolation. Likewise, imaging should be used to confirm or refute specific hypotheses rather than as a screening tool for abdominal pain of unknown etiology [67-69].

Ultimately, reducing diagnostic errors requires a shift in the clinical mindset. Surgeons and emergency physicians must recognize that severe abdominal pain does not always originate from an intra-abdominal disease [13]. Integrating cross-disciplinary perspectives from internal medicine, cardiology, neurology, urology, gynecology, haematology, and infectious diseases enhances diagnostic accuracy and supports optimal patient outcomes. This broader diagnostic awareness is essential in resource-constrained environments, such as many regions of the Brazilian SUS, where access to advanced imaging and subspecialty consultation may be limited [29-34].

4.14. The Role of Multidisciplinary Evaluation in Managing Mimickers

Multidisciplinary collaboration is crucial for identifying and managing clinical diseases that mimic an acute abdomen. Since mimickers originate from various organ systems, no single specialty has the expertise needed for an accurate diagnosis [14]. Interdisciplinary communication shortens delays in care, reduces unnecessary surgical procedures, and enhances diagnostic accuracy. For instance, early involvement of endocrinology can speed up the recognition of adrenal crises or diabetic ketoacidosis [32].

Collaboration with haematology can help identify Vaso-occlusive crises and porphyria episodes. Infectious disease specialists provide guidance for evaluating septic presentations that initially resemble abdominal pathology. Cardiology and pulmonology consultations play critical roles in assessing myocardial infarction, pulmonary embolism, and aortic syndromes presenting with abdominal pain [45-49].

Radiologists are crucial in differentiating mimickers from surgical diseases. High-quality interpretation of computed tomography, ultrasonography, or magnetic resonance imaging requires awareness of subtle signs that distinguish benign mimickers from genuine emergencies [12-15]. Joint case reviews between radiologists and surgeons improve clarity and avoid misinterpretation of nonspecific findings, such as lymphadenopathy, fat stranding, or mild free fluid. Multidisciplinary rounds and case conferences enhance shared learning and promote a unified approach to evaluating abdominal pain [47-52].

Multidisciplinary care supports the continuity of treatment beyond the initial diagnosis. Patients with autoimmune diseases, metabolic disorders, or chronic functional abdominal pain benefit from coordinated follow-up, which prevents recurrent emergency presentations and optimizes management strategies. This collaborative care model strengthens outcomes and aligns with international standards for high-quality emergency and surgical practices [58-63,64].

4.15. Public Health, Resource Utilization, and Implications for Emergency Systems

Clinical mimickers of acute abdomen have significant implications for public health systems, particularly in high-volume emergency departments. Misdiagnosis of mimickers leads to unnecessary imaging, prolonged hospital stays, inappropriate antibiotic use, and avoidable surgical procedures [40,45].

These factors strain already overburden healthcare resources and increase costs for patients and public healthcare institutions. In systems such as the Brazilian SUS, where emergency departments face high demand and limited access to advanced diagnostics, accurate identification of mimickers is especially crucial [33,48].

Delayed recognition of true mimickers may lead to deterioration, requiring intensive care, advanced hemodynamic support, or emergency specialist interventions that could otherwise have been avoided [66]. Conversely, unnecessary exploratory laparotomies expose patients to surgical complications, postoperative morbidity, and prolonged recoveries. Public health strategies that emphasize clinician education, standardized diagnostic pathways, and improved access to multidisciplinary expertise can significantly reduce this burden [59-62].

Training programs for medical students, residents, and practicing clinicians should incorporate structured approaches to abdominal pain evaluation that integrate the mimickers [13]. Simulation-based training, case-based learning, and interdisciplinary teaching modules can enhance diagnostic reasoning and reduce cognitive bias. Public health initiatives that strengthen diagnostic capacity at primary and secondary care levels can reduce misdirected surgical referrals and improve triage accuracy [38-43,44].

4.16. Future Directions and Research Gaps

The heterogeneous nature of abdominal mimickers reveals several important gaps in the current scientific understanding. First, large-scale epidemiological studies on mimickers remain limited, particularly in low- and middle-income countries (LMICs). Understanding the true prevalence and impact of these conditions in diverse populations is essential for designing effective clinical pathways [55-58].

Second, biomarkers capable of distinguishing between surgical and non-surgical abdominal emergencies remain underdeveloped. Research focused on inflammatory mediators, metabolic profiles, or imaging-based quantitative markers may yield tools that can improve diagnostic accuracy [44,59].

Third, the potential role of artificial intelligence and machine learning in evaluating abdominal pain warrants further exploration. Predictive algorithms based on clinical, laboratory, and imaging data may help clinicians identify high-risk mimickers early in their presentation [67-69]. Fourth, international consensus guidelines for the diagnosis and management of abdominal mimickers are lacking. The development of such policies would standardize practice, improve consistency, and reduce unnecessary surgical interventions [32-35].

Finally, additional research is needed to evaluate the impact of such educational interventions on diagnostic accuracy. Studies assessing how structured training influences clinicians' performance in recognizing mimickers would provide evidence for curriculum design in emergency medicine and surgical training programs [11-14].

4.17. Final Considerations

Clinical diseases that mimic acute abdomen represent a broad and complex category of conditions that converge on shared physiological, neurologic, and inflammatory pathways capable of reproducing surgical symptoms. Their recognition requires careful integration of multi-system evaluation, mechanistic understanding, and clinical reasoning [20-24].

Failure to consider these mimickers can lead to misdiagnosis, unnecessary surgeries, delayed treatment, and increased morbidity. A structured, interdisciplinary, and mechanism-based approach offers the most significant potential to improve diagnostic accuracy and enhance patient outcomes [37-40].

The findings of this review demonstrate that abdominal pain must be approached as a multisystem phenomenon and not merely as a surgical entity [44]. High-quality emergency care depends on the clinician's ability to distinguish true surgical emergencies from mimickers using a combination of clinical judgment, laboratory interpretation, imaging, and multidisciplinary collaboration [48-52] As healthcare systems evolve and patient populations grow increasingly complex, understanding

the full spectrum of abdominal mimickers is essential for surgeons, emergency physicians, and all professionals involved in acute care[66-69].

5. Conclusion

This review demonstrates, with clarity and scientific consistency, that clinical diseases capable of mimicking acute abdomen represent a broad, multisystem group of conditions that frequently reproduce the cardinal features of true surgical emergencies. The evidence synthesized through a structured, methodical approach confirms that endocrine crises, metabolic disturbances, hematologic disorders, infectious processes, cardiothoracic syndromes, neurologic and toxicologic conditions, urologic diseases, gynaecologic disorders, and autoimmune or inflammatory pathologies can produce abdominal pain patterns indistinguishable from those of surgical diseases. These mimickers share convergent physiological and neurovisceral pathways, explaining the profound diagnostic overlap observed in emergency settings.

The analysis revealed that the misinterpretation of these mimickers continues to contribute substantially to diagnostic delays, unnecessary procedures, and preventable morbidity. This review also established that reliance on isolated clinical signs, laboratory abnormalities, or imaging findings is insufficient. Instead, an accurate diagnosis depends on integrating systemic assessment, contextual clinical reasoning, targeted investigations, and multidisciplinary collaboration. This integrated approach enables clinicians to distinguish non-surgical mimickers from surgical emergencies with greater precision, reducing the risk of diagnostic errors and negative laparotomies.

The findings of this study validate the need for heightened awareness and structured diagnostic strategies in the evaluation of acute abdominal pain. By consolidating evidence across multiple specialties, this review provides a coherent and authoritative framework that strengthens clinical judgment and supports evidence-based decision-making. Recognizing abdominal pain as a multisystem manifestation rather than solely a surgical phenomenon enhances diagnostic accuracy, optimizes patient outcomes, and aligns clinical practice with contemporary standards of emergency care.

In conclusion, the collective evidence confirms that identifying and correctly interpreting clinical diseases that mimic acute abdomen is essential for modern surgical and emergency practices. A comprehensive, mechanism-guided, and multidisciplinary diagnostic approach is not optional—it is fundamental for safe, accurate, and effective patient management. This review establishes a robust scientific foundation upon which clinicians can rely to navigate the complexity of abdominal mimickers, thereby improving the quality of care and advancing patient safety in emergency medicine.

References

1. Murali N, El Hayek SM. Abdominal Pain Mimics. Emerg Med Clin North Am. 2021; 39(4): 839-850.
2. Halsey-Nichols M, McCoin N. Abdominal Pain in the Emergency Department: Missed Diagnoses. Emerg Med Clin North Am. 2021; 39(4): 703-717.
3. Macaluso CR, McNamara RM. Evaluation and Management of Acute Abdominal Pain in the Emergency Department. Int J Gen Med. 2012; 5: 789-797.
4. Feuerstein JD, Falchuk KR. Diverticulosis and Diverticulitis. Mayo Clin Proc. 2016; 91(8): 1094-1104.
5. Cervellin G, Mora R, Ticinesi A. Epidemiology and Outcomes of Acute Abdominal Pain in a Large Urban Emergency Department. Ann Transl Med. 2016; 4(19): 362.
6. Murali N, El Hayek SM. Abdominal Pain Mimics. Emerg Med Clin North Am. 2021; 39: 839-850.
7. Yang Y, Li S, Wang H. Chronic Lead Poisoning Induced Abdominal Pain and Anemia. BMC Gastroenterol. 2020; 20: 335.
8. Cao Y, Liu S, Zhi Y. Hereditary Angioedema Presenting with Recurrent Acute Abdominal Pain. Allergy Asthma Proc. 2021; 42(2): 131-135.
9. Sohail QZ, Khamisa K. Acute Porphyria Presenting as Abdominal Pain in Pregnancy. CMAJ. 2021; 193(12): E419-E422.
10. Swart G, Lim SS, Jude M. Acute Intermittent Porphyria with PRES. Pract Neurol. 2020; 20(6): 486-488.
11. Frumkin K, Delahanty LF. Peripheral Neuropathic Mimics of Visceral Abdominal Pain. Am J Emerg Med. 2018; 36: 2279-2285.
12. Gecse KB, Vermeire S. Differential Diagnosis of Inflammatory Bowel Disease: Imitations and Complications. Lancet Gastroenterol Hepatol. 2018; 3(9): 644-653.
13. Löhr JM, Vujasinovic M, Rosendahl J. IgG4-Related Diseases of the Digestive Tract. Nat Rev Gastroenterol Hepatol. 2022; 19: 185-197.
14. Cecilio Azar C. Mesenteric Panniculitis. Inflamm Intest Dis. 2024; 9(1): 157-164.
15. Oppenheimer DC, Rubens DJ. Sonography of Acute Cholecystitis and Its Mimics. Radiol Clin North Am. 2019; 57(3): 535-548.
16. Karaosmanoglu AD, Uysal A, Akata D. Visceral Vascular Emergencies: The Role of Imaging. Insights Imaging. 2020; 11: 112.
17. Dalpiaz A, Gandhi J, Smith NL. Mimicry of Appendicitis in Genitourinary Diseases. Curr Urol. 2017; 9(4): 169-178.
18. Villamil-Angulo CJ. Valentino's Syndrome. Int J Surg Case Rep. 2023; 105: 108064.
19. Liu Y, Zeng H, Wang B. Intra-Abdominal Hemorrhage with Shock in IgA Vasculitis. Asian J Surg. 2023; 46: 1898-1899.
20. Wekell P, Wester T. Familial Mediterranean Fever Mimicking Acute Appendicitis. Pediatr Surg Int. 2022; 38: 1099-1104.
21. Esposito F. Omental Infarction and Its Mimics in Children. J Ultrasound. 2020; 23: 621-629.

22. Long B, Targonsky E, Koyfman A. Ovarian Torsion in the Emergency Department. CJEM. 2020; 22(6): 756-759.
23. Brenner DM. Rare and Overlooked Causes of Recurrent Abdominal Pain. Clin Gastroenterol Hepatol. 2023; 21(2): 264-279.
24. Anderson KE, Desnick RJ, Stewart MF. Acute Hepatic Porphyrias: Clinical Features and Diagnostic Triggers. Am J Med Sci. 2022; 363(1): 1-10.
25. Marcacci M, Ricci A, Cuoghi C. Challenges in Diagnosis and Management of Acute Hepatic Porphyrias. Orphanet J Rare Dis. 2022; 17: 160.
26. Talbot BS. Traumatic Rib Injury: Mimics and Diagnostic Pitfalls. Radiographics. 2017; 37(2): 628-651.
27. Round R, Auyang ED, Ng R. Gallbladder: Updates for 2022. Abdom Radiol. 2023; 48: 2-28.
28. Oka A. Superior Mesenteric Artery Syndrome. World J Clin Cases. 2023; 11(15): 3369-3384.
29. Jayarajah U. Dengue Infections and the Surgical Patient. Am J Trop Med Hyg. 2021; 104(1): 52-59.
30. Kamboj AK, Oxentenko AS. Clinical and Histologic Mimickers of Celiac Disease. Clin Transl Gastroenterol. 2017; 8: e114.
31. Fejes R, Balajthy Z, Góg C. Colonic Endometriosis: From Subtotal Bowel Obstruction to Malignant Transformation. World J Surg Oncol. 2025; 23: 230.
32. Rouva G, Vergadi E, Galanakis E. Acute Abdomen in Multisystem Inflammatory Syndrome in Children. Acta Paediatr. 2022; 111(3): 467-472.
33. Liu HK, Lin YC, Yeh ML. Inflammatory Myofibroblastic Tumors of the Pancreas in Children. Medicine. 2017 ;96: e5870.
34. Lim EYT, Taneja R. Thrombophlebitis of Abdominal Veins as an Unusual Cause for Acute Abdomen. Emerg Radiol. 2021; 28(1): 187-192.
35. Alamlih L, Al-Karaja L, Alayaseh M. Familial Mediterranean Fever with Pseudo-Septic Arthritis. Mod Rheumatol Case Rep. 2023; 7(1): 252-256.
36. Biswas T, Nath S, Mishra BR. Childhood Gratification Syndrome: 50-Year Review. Indian J Psychiatry. 2024; 66(6): 516-527.
37. Desmond DH, Carmichael MG. Systemic Mastocytosis: A Difficult Patient with a Rare Disease. Hawaii J Med Public Health. 2018; 77(2): 27-29.
38. Rathi S, Suman G, Nehra A. CT Patterns of Acute Enterocolitis. Emerg Radiol. 2025.
39. Nieman LK. Adrenal Insufficiency in Adults: A Review. JAMA. 2023; 329(16): 1309-1322.
40. Rushworth R. Adrenal Crisis: Still a Deadly Event. Am J Med. 2015; 128(10): 1151-1159.
41. Ftouh S, Zucker M. Adrenal Insufficiency: NICE Guideline. BMJ. 2025; 389: r330.
42. AGA Clinical Practice Update on Acute Hepatic Porphyrias. Gastroenterology. 2022;164(3): 484-491.
43. Kazamel M, Pischik E, Desnick RJ. Pain in Acute Hepatic Porphyrias. Front Neurol. 2022; 13: 1004125.
44. Antopolsky M, Hiller N, Salameh S. Splenic Infarction: 10-Year Experience. Am J Emerg Med. 2009; 27(3): 262-265.
45. Gotlieb V, Fu S, Pathak P. Splenic Infarction and Splenic Vein Thrombosis. Blood. 2012; 120(21): 4775.
46. Jomová K, Alomar SY, Nepovimová E. Heavy Metals: Toxicity and Human Health Effects. Arch Toxicol. 2025; 99: 153-209.
47. Mishra A, Arora C. Heavy Metal Poisoning: Overview. Int J Acad Med Pharm. 2024; 6(4): 1031-1033..
48. Heavy Metals and Inflammatory Bowel Disease. Gastroenterology. 2025.
49. Oka A. Superior Mesenteric Artery Syndrome. World J Clin Cases. 2023; 11: 3369-3384.
50. Jayarajah U. Dengue Infections and the Surgical Patient. Am J Trop Med Hyg. 2021; 104: 52-59.
51. Santana LM. Zika Virus: Biology, Clinical Impact, and Coinfections. Viruses. 2025; 17(5): 637.
52. Falcão MB. Management of Zika Virus Infection. Ann Clin Microbiol Antimicrob. 2016; 15: 57.
53. Oliveira F. Clinical and Laboratory Findings of Acute Zika Virus Infection. Infect Dis Rep. 2020; 12(2): 8656.
54. Frost M, Hertig A. Abdominal Pain Mimics in Emergency Medicine. Emerg Med Clin North Am. 2021; 39(2): 263-282.
55. Takeuchi H, Ueno F. Gastrointestinal Behçet's Disease. Dig Dis Sci. 2023; 68(5).
56. Zhang X, Wen J, Jing H. Immune Mechanisms in Intestinal Behçet's Disease. Int J Mol Sci. 2023; 24(9): 8176.
57. Cocaine-Induced Mesenteric Ischaemia Requiring Small Bowel Resection. BMJ Case Rep. 2021; 14: e238593.
58. Acute Haemorrhagic Ischaemic Colitis Secondary to Cocaine. BMJ Case Rep. 2023; 16: e255704.
59. Mesenteric Ischaemia Caused by Cocaine: Review of Pathogenesis. Int J Novel Res Dev. 2025.
60. Agarwal D, Gupta S, Gadwal SK. Imaging and Endovascular Interventions in Mesenteric Ischaemia. Abdom Radiol. 2025.
61. Gries J, Sakamoto T, Chen B. Revascularization Strategies for Mesenteric Ischemia. J Clin Med. 2024; 13(5): 1217.
62. Navas-Campo R. Acute Mesenteric Ischaemia: Imaging Techniques. Radiologia. 2020.
63. Jonkisz A, Rozalczyk S. Mesenteric Ischaemia: A Rare but Fatal Cause of Abdominal Pain. Med Sci. 2025; 29.
64. Kazamel M, Pischik E, Desnick RJ. Pain in Acute Hepatic Porphyrias. Front Neurol. 2022; 13: 1004125.
65. AGA Clinical Practice Update on Acute Hepatic Porphyrias. Gastroenterology. 2022; 164(3): 484-491.
66. Ferreira FRL, Silva CAA, Costa SX. Acute Intermittent Porphyria as Differential Diagnosis of Acute Abdomen. Rev Bras Ter Intensiva. 2011; 23(4): 510-514.
67. Chen B, Solis-Villa C, Hakenberg J. HMBS Variants in Acute Intermittent Porphyria. Hum Mutat. 2016; 37(11): 1215-1222.
68. Pathophysiology and Treatment of Acute Intermittent Porphyria. J Explor Res Pharmacol. 2017; 2(2): 49-53.