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TIME-CRITICAL SURGICAL ACUTE ABDOMEN: A NARRATIVE REVIEW OF DIAGNOSTIC PATHWAYS, ESCALATION PROCESSES, AND MULTIDISCIPLINARY CARE

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ABSTRACT

Introduction: Acute abdominal pathology remains a common and diagnostically challenging emergency presentation. A clinically important subset requires urgent surgical or interventional management, and delays may lead to bowel necrosis, sepsis, shock, multi-organ failure, and death.
Methods: A structured search of PubMed/MEDLINE, PubMed Central, Scopus, and Google Scholar was performed. International guidelines, systematic reviews, meta-analyses, and clinically relevant observational studies were prioritised, with a focus on obstruction, ischaemia, perforation, intra-abdominal infection, biliary emergencies, haemorrhage, and trauma-related abdominal pathology.
Results: The reviewed evidence shows that the surgical acute abdomen is mainly driven by obstruction, ischaemia, perforation/infection, and haemorrhage. Early diagnosis remains difficult because symptoms may be non-specific and presentations atypical, especially in older or comorbid patients. Repeated reassessment, risk-based imaging, early recognition of red-flag features, and timely surgical consultation are consistently associated with improved outcomes.
Conclusions: Effective management of the acute abdomen requiring surgical intervention depends on early recognition of time-critical pathology, structured diagnostic pathways, and prompt source control. Further research should focus on standardised care pathways that reduce time-to-intervention while maintaining patient safety.

KEYWORDS

Acute Abdomen, Emergency Surgery, Diagnostic Delay, Risk Stratification, Source Control, Emergency Laparotomy

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Introduction

Acute abdominal pathology remains one of the most frequent and diagnostically demanding presentations in emergency medicine and general surgery (Rogers & Kirton, 2024). Although many cases of abdominal pain are benign or self-limiting, a clinically important subset is driven by underlying surgical disease in which missed or delayed recognition may lead to irreversible organ injury, septic deterioration, or death (Rogers & Kirton, 2024; Tilsed et al., 2016). The aetiological spectrum is broad and includes bowel obstruction, mesenteric and intestinal ischaemia, gastrointestinal perforation, complicated biliary disease, intra-abdominal infection, and abdominal bleeding (Di Saverio et al., 2020; Pisano et al., 2020; ten Broek et al., 2018; Sartelli et al., 2020; Tarasconi et al., 2020; Tilsed et al., 2016). These entities often share non-specific features, may present atypically in older adults, and can be obscured by comorbidity or attenuated inflammatory responses, complicating early differentiation between surgical and non-surgical causes (Rogers & Kirton, 2024).

Time-sensitive abdominal emergencies are particularly unforgiving. In acute mesenteric ischaemia, strangulating obstruction, or perforated viscus, even short diagnostic delays may translate into bowel necrosis, diffuse peritonitis, or multi-organ failure (Tilsed et al., 2016; Murray et al., 2021). Evidence from emergency laparotomy pathways suggests that prolonged time to theatre in perforated and ischaemic gastrointestinal pathology is associated with increased morbidity and mortality (Murray et al., 2021). Beyond clinical harm, diagnostic delay also carries measurable system-level consequences, including increased hospital costs in common surgical emergencies such as appendicitis (Kulasekere et al., 2024). For these reasons, modern acute care frameworks increasingly emphasise structured risk stratification, early imaging with timely reassessment, and prompt interdisciplinary collaboration between emergency physicians, radiologists, anaesthetists, and surgeons (Rogers & Kirton, 2024; Grimes, 2025).

Methodology

This study was conducted as a narrative literature review based on a structured search of PubMed/MEDLINE, PubMed Central, Scopus, and Google Scholar. The search focused on publications addressing acute abdominal pathology requiring urgent surgical or interventional management, with particular attention to bowel obstruction, mesenteric and intestinal ischaemia, gastrointestinal perforation, complicated biliary disease, intra-abdominal infection, abdominal bleeding, and trauma-related abdominal emergencies.

The search strategy combined terms such as “acute abdomen,” “surgical acute abdomen,” “bowel obstruction,” “mesenteric ischaemia,” “gastrointestinal perforation,” “complicated diverticulitis,” “acute cholecystitis,” “appendicitis,” “abdominal trauma,” “diagnostic delay,” “source control,” and “emergency laparotomy.” Priority was given to international guidelines, systematic reviews, meta-analyses, and higher-quality observational studies published within the last five years. Older landmark articles were also included when they remained directly relevant to pathophysiology, diagnostic reasoning, escalation thresholds, or time-critical management.

Sources were selected on the basis of clinical relevance, methodological value, and applicability to emergency and surgical practice. Studies focused primarily on non-surgical causes of abdominal pain, highly selective case reports, or publications with limited relevance to acute decision-making were not prioritised. The final selection was intended to provide a clinically focused overview of the major mechanisms, presentations, diagnostic pathways, and management principles in abdominal pathology requiring urgent surgical evaluation.

Results

Pathophysiology

Most surgical acute abdominal emergencies can be conceptualised through a limited set of dominant pathophysiological mechanisms—obstruction, ischaemia, perforation/infection, and haemorrhage—each capable of evolving rapidly from localised injury to systemic decompensation (Rogers & Kirton, 2024; Grimes, 2025). Although these categories frequently overlap, the dominant mechanism usually determines urgency, escalation strategy, and the optimal method of source control (operative or interventional) (Rogers & Kirton, 2024; Di Saverio et al., 2020; Pisano et al., 2020; ten Broek et al., 2018; Sartelli et al., 2020; Tarasconi et al., 2020; Tilsed et al., 2016).

Obstruction-driven pathology is primarily mediated by progressive luminal distension and rising intraluminal pressure. The typical cascade is: obstruction → distension → venous congestion → bowel wall oedema → impaired microcirculatory perfusion → ischaemia → necrosis → perforation (ten Broek et al., 2018; Grimes, 2025). Time sensitivity increases substantially in closed-loop obstruction and strangulation, where simultaneous compromise of inflow and outflow accelerates transmural ischaemia and necrosis. These mechanisms provide the physiological basis for prioritising early recognition of strangulation features and rapid escalation when ischaemia is suspected (ten Broek et al., 2018; Grimes, 2025).

Ischaemic injury reflects a mismatch between intestinal oxygen demand and perfusion. Mechanistically, ischaemia progresses via reduced arterial inflow or impaired venous outflow → mucosal hypoxia → barrier breakdown → bacterial translocation → systemic inflammatory response → shock and multiorgan dysfunction (Tilsed et al., 2016; Grimes, 2025). A key clinical implication is that symptoms may precede peritoneal signs: early ischaemia may produce severe pain while abdominal examination remains relatively non-specific until transmural infarction develops. This pathophysiological dissociation underpins guideline emphasis on maintaining high suspicion for acute mesenteric ischaemia in appropriate clinical contexts (Tilsed et al., 2016).

Perforation and intra-abdominal infection represent a transition from local contamination to diffuse peritoneal inflammation and systemic inflammatory dysregulation. The core sequence is: wall disruption → peritoneal contamination → peritoneal inflammation → capillary leak and third spacing → (relative/absolute) hypovolaemia → sepsis and shock (Tarasconi et al., 2020; Grimes, 2025). Perforated peptic ulcer disease is a prototypical example, where a rapid shift from chemical to bacterial peritonitis can precipitate haemodynamic compromise; consequently, early recognition and timely source control are central management principles (Tarasconi et al., 2020).

Haemorrhagic pathology compromises systemic stability primarily through hypovolaemia and impaired oxygen delivery. The cascade is: bleeding → reduced preload and cardiac output → tissue hypoperfusion → anaerobic metabolism → progressive acidosis and coagulopathy, which can become self-perpetuating, particularly in major intra-abdominal haemorrhage or severe trauma (Coccolini et al., 2020; Smyth et al., 2022). These

mechanisms explain why haemodynamic assessment and resuscitation are inseparable from diagnostic reasoning in suspected haemorrhagic abdominal emergencies (Coccolini et al., 2020; Smyth et al., 2022).

Clinical interpretation of abdominal pain is further refined by the underlying pain pathways, which can be categorised as visceral, somatic (parietal), and referred (Martin & Rossi, 1997; Mayumi et al., 2016). Visceral pain arises from distension, traction, ischaemia, and inflammation of abdominal organs; it is typically dull, poorly localised, and often midline (Martin & Rossi, 1997; Mayumi et al., 2016). Somatic pain reflects irritation of the parietal peritoneum and is usually sharp and well localised, frequently associated with guarding and peritoneal signs (Martin & Rossi, 1997; Mayumi et al., 2016). Referred pain results from segmental convergence of visceral and somatic afferents, producing pain perceived at a distant somatic site (Martin & Rossi, 1997; Mayumi et al., 2016). This framework explains the evolution from vague visceral pain to localised peritonism as inflammation progresses or perforation occurs (Grimes, 2025; Martin & Rossi, 1997; Mayumi et al., 2016).

Within emergency general surgery, several entities illustrate these mechanisms. Acute appendicitis is classically obstruction-driven: luminal obstruction (e.g., lymphoid hyperplasia or faecolith) leads to mucus retention and distension, bacterial overgrowth, rising intraluminal pressure, venous congestion, and compromised perfusion, progressing to transmural inflammation, necrosis, and potential perforation (Di Saverio et al., 2020). The transition from visceral to somatic pain often corresponds to increasing peritoneal involvement and a higher probability of complicated disease, consistent with modern guideline frameworks distinguishing uncomplicated from complicated appendicitis (Di Saverio et al., 2020; Grimes, 2025; Martin & Rossi, 1997; Mayumi et al., 2016).

Acute calculous cholecystitis is similarly initiated by obstruction—most commonly cystic duct obstruction by gallstones—followed by gallbladder distension, wall ischaemia, and inflammatory mediator release, with a subset progressing to secondary infection, gangrene, empyema, or perforation when uncontrolled (Pisano et al., 2020). This obstruction–ischaemia–inflammation axis explains why delayed recognition may coincide with advanced pathology, particularly in older or comorbid patients with atypical clinical profiles (Pisano et al., 2020; Grimes, 2025).

Adhesive small bowel obstruction spans a spectrum from partial obstruction responsive to conservative therapy to strangulation requiring urgent surgery. Pathophysiologically, the central danger is progression from distension and oedema to perfusion compromise and infarction, paralleling the obstruction cascade described above. Guideline-based pathways therefore emphasise early recognition of clinical and radiological signs of ischaemia and strangulation, as these findings indicate transition towards irreversible tissue injury (ten Broek et al., 2018; Grimes, 2025).

Complicated diverticulitis typically begins with microperforation and localised inflammation, which may remain contained or progress to abscess formation. In some cases, free perforation develops, resulting in diffuse peritonitis and sepsis (Sartelli et al., 2020). CT-based staging aligns closely with this pathophysiological spectrum and informs escalation from conservative management to drainage or operative source control when contamination cannot be contained (Sartelli et al., 2020).

Collectively, these mechanisms highlight that acute abdominal emergencies are defined less by a single diagnosis than by the dominant pathophysiological process and its propensity for rapid systemic destabilisation. A mechanistic framework integrating obstruction, ischaemia, perforation/infection, and haemorrhage—together with an understanding of visceral and somatic pain evolution—provides a coherent basis for subsequent sections on clinical presentation, diagnostic strategies, and time-critical management pathways (Rogers & Kirton, 2024; Tarasconi et al., 2020; Tilsed et al., 2016; Grimes, 2025; Martin & Rossi, 1997; Mayumi et al., 2016).

Clinical presentation

Acute abdominal pathology of surgical origin presents along a broad clinical spectrum, ranging from localised pain with minimal systemic disturbance to rapidly progressive peritonitis, sepsis, or haemorrhagic shock (Rogers & Kirton, 2024; Grimes, 2025). Because early symptoms may be non-specific and overlap across aetiologies, clinical evaluation should focus on pain characteristics and evolution, signs of peritoneal irritation, and physiological instability, with repeated reassessment when the diagnosis remains uncertain (Rogers & Kirton, 2024; Mayumi et al., 2016).

Pain patterns and evolution

Abdominal pain can be conceptualised through visceral, parietal (somatic), and referred components, which helps explain changes in localisation and severity over time (Martin & Rossi, 1997; Mayumi et al., 2016). Visceral pain, such as in early appendicitis or early obstruction, is often dull and poorly localised, whereas irritation of the parietal peritoneum produces sharper, localised pain with guarding and rebound tenderness, features that should raise suspicion for perforation, ischaemia with transmural necrosis, or advanced inflammatory disease (Martin & Rossi, 1997; Mayumi et al., 2016). Sudden onset of severe, generalised pain may suggest perforation or major vascular catastrophe, while pain out of proportion to examination findings is a classic high-risk pattern for acute mesenteric ischaemia (Tilsed et al., 2016; Mayumi et al., 2016).

Peritoneal signs and systemic features

Peritoneal irritation, including localised or generalised guarding, rebound tenderness, and rigidity, often indicates advanced intra-abdominal pathology and should prompt early surgical involvement and expedited imaging and/or operative decision-making (Rogers & Kirton, 2024; Mayumi et al., 2016). Systemic manifestations such as fever, tachycardia, hypotension, altered mental status, and oliguria may signal evolving sepsis, shock, or significant haemorrhage and should immediately shift management priorities towards resuscitation and urgent source control (Rogers & Kirton, 2024; Coccolini et al., 2020; Smyth et al., 2022). Importantly, the absence of fever or leucocytosis does not exclude serious surgical pathology, particularly in older adults or immunocompromised patients (Rogers & Kirton, 2024; Mayumi et al., 2016).

High-risk groups and atypical presentations

Atypical or blunted presentations are common in older adults, patients with multiple comorbidities, and those with immunosuppression, in whom severe pathology may occur despite minimal tenderness or only modest inflammatory marker elevation (Rogers & Kirton, 2024; Mayumi et al., 2016). Similarly, analgesia may reduce apparent tenderness without altering the underlying disease process; therefore, persistent severe pain, progressive symptoms, or physiological deterioration should outweigh reassuring single-point findings and prompt reassessment and escalation (Rogers & Kirton, 2024; Grimes, 2025). In patients of reproductive age, gynaecological causes should be considered early and pregnancy testing should be incorporated into the diagnostic pathway; however, surgical aetiologies remain common and should not be deprioritised when red-flag features are present (Mayumi et al., 2016; Rogers & Kirton, 2024).

Clinical patterns in major surgical entities

Although presentations overlap, several patterns are clinically useful when integrated with risk stratification and imaging:

Acute appendicitis commonly evolves from vague periumbilical pain to right lower quadrant tenderness, with increasing peritoneal signs in complicated disease. Complicated appendicitis may present with systemic features, localised peritonism, or a palpable inflammatory mass or abscess; current guideline frameworks emphasise distinguishing uncomplicated from complicated disease because management pathways differ (Di Saverio et al., 2020; Mayumi et al., 2016).

Acute calculous cholecystitis typically presents with right upper quadrant pain, tenderness, often accompanied by inspiratory arrest on palpation, and variable fever or inflammatory marker elevation; progression to gangrene or perforation is more likely in delayed or high-risk presentations (Pisano et al., 2020).

Small bowel obstruction, including adhesive small bowel obstruction, commonly presents with colicky abdominal pain, vomiting, distension, and obstipation. Transition to constant severe pain, focal tenderness, fever, or peritonism should raise concern for strangulation or ischaemia and prompt urgent reassessment and escalation (ten Broek et al., 2018; Mayumi et al., 2016).

Complicated diverticulitis often presents with left lower quadrant pain and localised tenderness; abscess, free perforation, or generalised peritonitis may develop in more severe cases, in which imaging-based staging guides escalation to drainage or surgery (Sartelli et al., 2020).

Perforated peptic ulcer or other perforated viscus classically presents with sudden severe pain and early peritoneal signs; rapid progression to systemic compromise may occur as diffuse peritonitis develops, reinforcing the importance of early recognition and source control (Tarasconi et al., 2020; Mayumi et al., 2016).

Acute mesenteric ischaemia should be suspected in patients with severe abdominal pain, sometimes disproportionate to early examination findings, particularly in the presence of vascular risk factors; delayed recognition may rapidly lead to infarction and sepsis (Tilsed et al., 2016).

Intra-abdominal bleeding and traumatic abdominal injury may present with abdominal pain accompanied by hypotension, tachycardia, syncope, or altered mental status; both missed bleeding and delayed contamination may have catastrophic consequences (Coccolini et al., 2020; Smyth et al., 2022).

Practical red flags for immediate escalation

Across aetiologies, several findings should trigger immediate escalation, including haemodynamic instability, generalised peritonitis, persistent severe pain with deteriorating physiology, signs suggestive of strangulation in bowel obstruction, suspicion of mesenteric ischaemia, including pain out of proportion to examination findings, and suspected perforation or major haemorrhage (Rogers & Kirton, 2024; Tilsed et al., 2016; ten Broek et al., 2018; Tarasconi et al., 2020; Coccolini et al., 2020; Smyth et al., 2022). These features justify early senior review, expedited imaging selection, and timely surgical consultation within standardised pathways (Rogers & Kirton, 2024; Grimes, 2025).

Diagnostic approach

The diagnostic approach to acute abdominal pain with potential surgical origin should be risk-based, time-sensitive, and iterative, integrating early physiological assessment with targeted laboratory testing and appropriately selected imaging (Rogers & Kirton, 2024; Mayumi et al., 2016). Because presentations often overlap and may be atypical in older or comorbid patients, evaluation should rely on parallel assessment and repeated clinical reassessment rather than on a single-point diagnostic impression (Rogers & Kirton, 2024; Grimes, 2025; Mayumi et al., 2016).

Initial triage and early risk classification

A pragmatic first step is classification according to haemodynamic stability and the presence or absence of peritoneal irritation, as these factors determine the urgency of escalation (Mayumi et al., 2016; Rogers & Kirton, 2024). Haemodynamic instability or sepsis requires immediate resuscitation while rapidly excluding haemorrhage, perforation, or ischaemia, with early surgical involvement (Rogers & Kirton, 2024; Coccolini et al., 2020; Smyth et al., 2022; Tilsed et al., 2016). Localised or generalised peritonitis should prompt expedited imaging and early operative planning (Tarasconi et al., 2020; Mayumi et al., 2016). Stable patients without peritonism may undergo staged evaluation, but only with clearly defined thresholds for reassessment and escalation if symptoms persist or evolve (Rogers & Kirton, 2024; Grimes, 2025; Mayumi et al., 2016).

Laboratory assessment

Laboratory testing supports risk stratification and imaging selection but should not delay escalation in high-risk presentations (Rogers & Kirton, 2024; Mayumi et al., 2016). A practical baseline panel includes a complete blood count, C-reactive protein, metabolic panel with lactate, liver function tests with or without lipase, urinalysis, pregnancy testing when applicable, and type and screen when haemorrhage or operative intervention is plausible (Pisano et al., 2020; Tilsed et al., 2016; Coccolini et al., 2020; Smyth et al., 2022; Mayumi et al., 2016).

Imaging strategy

Imaging is central to distinguishing surgical from non-surgical causes of acute abdominal pain and to defining operative versus non-operative pathways (Rogers & Kirton, 2024; Mayumi et al., 2016). Ultrasound is the preferred first-line modality in suspected biliary disease and remains useful for focused trauma assessment of free fluid (Pisano et al., 2020; Coccolini et al., 2020). Contrast-enhanced computed tomography is the cornerstone of evaluation in suspected bowel obstruction, perforation, complicated diverticulitis, and many undifferentiated high-risk abdominal presentations, and it also helps classify appendicitis phenotypes when management pathways diverge (Di Saverio et al., 2020; ten Broek et al., 2018; Sartelli et al., 2020; Tarasconi et al., 2020; Rogers & Kirton, 2024). In suspected mesenteric ischaemia, early vascular-phase CT is particularly important because radiological confirmation may otherwise be delayed until irreversible bowel injury has developed (Tilsed et al., 2016). Magnetic resonance imaging may be used selectively when radiation avoidance is important, provided that it does not delay definitive management (Mayumi et al., 2016).

Reassessment and escalation thresholds

Dynamic reassessment is essential in the evaluation of acute abdominal pain. Escalation is warranted when pain worsens, peritoneal signs evolve, physiological or metabolic derangement progresses, or imaging suggests ischaemia, strangulation, or perforation; such findings should prompt early senior review and timely surgical consultation within standardised pathways (Rogers & Kirton, 2024; ten Broek et al., 2018; Tarasconi et al., 2020; Tilsed et al., 2016; Murray et al., 2021; Grimes, 2025).

Diagnostic and therapeutic management

Management of the surgical acute abdomen should proceed along parallel priorities: early stabilisation, rapid identification of time-critical pathology, and timely source control when indicated, whether operative or image-guided (Rogers & Kirton, 2024; Grimes, 2025). Because delayed diagnosis is consistently associated with worse outcomes in perforated and ischaemic gastrointestinal disease, contemporary emergency care places particular emphasis on streamlined escalation pathways and early senior surgical involvement in high-risk presentations (Murray et al., 2021; Grimes, 2025).

Initial stabilisation and early supportive care

Management should begin with immediate assessment and stabilisation of airway, breathing, and circulation, together with early analgesia and antiemetic treatment when required (Rogers & Kirton, 2024; Mayumi et al., 2016). In patients with haemodynamic instability, suspected haemorrhage, or evolving septic physiology, resuscitation, including fluid therapy, blood products when appropriate, and early critical care support, should proceed in parallel with diagnostic evaluation and surgical consultation (Coccolini et al., 2020; Smyth et al., 2022; Rogers & Kirton, 2024). Adequate pain control should not be withheld; instead, clinical reassessment should be repeated after analgesia to identify evolving peritoneal irritation or physiological deterioration (Mayumi et al., 2016).

Antimicrobial therapy and sepsis-oriented escalation

When intra-abdominal infection, perforation, or diffuse peritonitis is suspected, early antimicrobial therapy should be initiated as part of a time-sensitive sepsis-oriented approach while definitive source control is pursued (Tarasconi et al., 2020; Sartelli et al., 2020; Mayumi et al., 2016). In perforated peptic ulcer disease and complicated diverticulitis, antibiotics are adjunctive rather than definitive, because outcomes depend primarily on timely and adequate control of the septic source through surgery and/or image-guided drainage, particularly in the presence of generalised peritonitis or ongoing contamination (Tarasconi et al., 2020; Sartelli et al., 2020). Similarly, in complicated appendicitis, systemic features, abscess formation, or diffuse peritoneal irritation may require escalation beyond conservative pathways (Di Saverio et al., 2020; Mayumi et al., 2016).

Source control and operative versus non-operative pathways

A central management question is whether the patient requires urgent surgery, interventional radiology, or structured conservative treatment with close reassessment, as reflected in contemporary guidance on appendicitis, diverticulitis, perforation, obstruction, mesenteric ischaemia, and abdominal trauma (Rogers & Kirton, 2024; Di Saverio et al., 2020; Sartelli et al., 2020; Tarasconi et al., 2020; ten Broek et al., 2018; Tilsed et al., 2016; Coccolini et al., 2020; Smyth et al., 2022). In general, haemodynamic instability, generalised peritonitis, radiological evidence of perforation, strangulation or ischaemia, and uncontrolled haemorrhage favour urgent operative management, as consistently emphasised in guidelines addressing perforated peptic ulcer disease, mesenteric ischaemia, bowel obstruction, liver trauma, and bowel injury (Tarasconi et al., 2020; Tilsed et al., 2016; ten Broek et al., 2018; Coccolini et al., 2020; Smyth et al., 2022). In contrast, selected contained processes, such as diverticular abscess amenable to drainage or complicated appendicitis with a well-defined abscess in an otherwise stable patient, may be managed with antibiotics and image-guided drainage, provided that close monitoring and clear criteria for escalation are maintained (Sartelli et al., 2020; Di Saverio et al., 2020; Mayumi et al., 2016).

Time sensitivity and escalation thresholds

Time-sensitive abdominal emergencies have narrow therapeutic windows. Acute mesenteric ischaemia, strangulating bowel obstruction, and perforated viscus are prototypical examples in which delayed recognition and delayed source control may rapidly lead to bowel necrosis, sepsis, and death (Tilsed et al., 2016; Murray et al., 2021; Grimes, 2025). Murray et al. (2021) linked longer time to surgery with higher mortality in acute perforated and ischaemic gastrointestinal pathology, while Grimes (2025) highlighted the value of standardised pathways that shorten time-to-imaging, time-to-surgeon, and time-to-intervention.

Trauma-related abdominal emergencies and emergency decision-making constraints

Traumatic abdominal emergencies can be understood within the same overarching framework of early stabilisation, risk-based imaging, and timely source control when indicated (Coccolini et al., 2020; Smyth et al., 2022). Coccolini et al. (2020) noted that solid organ injury may be managed non-operatively in haemodynamically stable patients under protocolised monitoring, whereas Smyth et al. (2022) emphasised that hollow viscus and mesenteric injury carry a substantial risk of delayed contamination and often require heightened vigilance and timely operative management when suspected. Kituuka et al. (2023) further highlighted that emergency surgery is frequently undertaken under conditions of limited time and impaired decisional capacity, meaning that consent and ethical processes should support rather than delay life-saving treatment while remaining proportionate to clinical urgency.

Discussion

This review shows that the acute abdomen requiring surgical intervention remains a time-critical and diagnostically challenging condition. The main mechanisms—obstruction, ischaemia, perforation/infection, and haemorrhage—often overlap and may rapidly lead to systemic deterioration.

The reviewed evidence indicates that poor outcomes are strongly linked to delayed recognition and delayed source control. Early presentation may be misleading, with non-specific symptoms, modest laboratory abnormalities, and initially limited abdominal findings, particularly in older or comorbid patients. This supports repeated reassessment as a key principle of safe management.

The literature also supports a risk-based diagnostic approach integrating clinical assessment, laboratory testing, and appropriate imaging, especially contrast-enhanced CT in high-risk presentations. Once time-critical surgical pathology is suspected, timely escalation and definitive source control are central to improving outcomes.

This review is limited by its narrative design and the heterogeneous nature of the included evidence. Nevertheless, current data consistently support early recognition of red-flag features, repeated reassessment, and prompt progression to definitive management.

Conclusion

Acute abdominal pathology of surgical origin remains a major clinical challenge because of its heterogeneous aetiologies, overlapping symptom patterns, and potential for rapid progression to peritonitis, sepsis, bowel necrosis, or haemorrhagic shock (Rogers & Kirton, 2024; Tarasconi et al., 2020; Tilsed et al., 2016; Mayumi et al., 2016). A mechanistic framework centred on obstruction, ischaemia, perforation/infection, and haemorrhage, together with structured clinical risk stratification, may support earlier recognition of time-critical disease and more consistent escalation decisions (Rogers & Kirton, 2024; Tilsed et al., 2016; Grimes, 2025; Martin & Rossi, 1997; Mayumi et al., 2016). Modern diagnostic pathways that integrate repeated clinical assessment with targeted laboratory testing and appropriate imaging selection, particularly contrast-enhanced computed tomography where indicated, may reduce preventable diagnostic delay and improve triage into operative, interventional, or conservative management pathways (Rogers & Kirton, 2024; Di Saverio et al., 2020; ten Broek et al., 2018; Sartelli et al., 2020; Tarasconi et al., 2020; Tilsed et al., 2016; Mayumi et al., 2016). Ultimately, outcomes depend not only on diagnostic accuracy but also on timely source control and coordinated multidisciplinary care across emergency medicine, radiology, surgery, anaesthesia, and critical care services (Murray et al., 2021; Coccolini et al., 2020; Smyth et al., 2022; Grimes, 2025). Future research should prioritise high-quality evaluation of 8tilization8 acute care pathways and implementation strategies that 8tilizat time-to-intervention while balancing resource 8tilization and patient safety (Rogers & Kirton, 2024; Murray et al., 2021; Grimes, 2025).

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