Review Article

Computed Tomography in the Early Diagnosis of Mesenteric Ischemia: Diagnostic Performance and Clinical Utility

María José Víquez Angulo[1*], Marco Andrey Vega Chaves[2], Stefanny Nikole Trejos Castro[3], Fernando Rodríguez Flores[4], Dilan Javier Cambronero Vega[5], Ariel Francisco Carranza Villalobos[6]

[1]Medical Doctor, Independent Researcher, San José, Costa Rica
[2]Medical Doctor, Carlos Luis Valverde Vega Hospital, Alajuela, Costa Rica
[3]Medical Doctor, México Hospital, San José, Costa Rica
[4]Medical Doctor, Centro Integrado en Salud de Coronado, San José, Costa Rica
[5]Medical Doctor, Independent Researcher, Puntarenas, Costa Rica
[6]Medical Doctor, Víctor Manuel Monseñor Sanabria Hospital. Puntarenas, Costa Rica
[*]Corresponding author email: **marijose_v.a@hotmail.com**

International Archives of Integrated Medicine, Vol. 13, Issue 5, May, 2026.
Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 15-5-2026 **Accepted on:** 26-5-2026
Source of support: Nil **Conflict of interest:** None declared.

DOI: **10.5281/zenodo.20542093**

How to cite this article: María José Víquez Angulo, Marco Andrey Vega Chaves, Stefanny Nikole Trejos Castro, Fernando Rodríguez Flores, Dilan Javier Cambronero Vega, Ariel Francisco Carranza Villalobos. Computed Tomography in the Early Diagnosis of Mesenteric Ischemia: Diagnostic Performance and Clinical Utility. Int. Arch. Integr. Med., 2026; 13(5): 76-90.

Abstract

Computed tomography has become the cornerstone of early diagnosis in acute mesenteric ischemia because it offers rapid, highly accurate evaluation of both vascular compromise and bowel injury in a condition where diagnostic delay is strongly associated with bowel necrosis, sepsis, and death. Acute mesenteric ischemia comprises several etiologic subtypes, including arterial embolism, arterial thrombosis, mesenteric venous thrombosis, and non-occlusive mesenteric ischemia, each with distinct pathophysiological mechanisms and imaging patterns. Contrast-enhanced computed tomography angiography is particularly valuable because it can identify arterial or venous filling defects, assess bowel wall enhancement, and detect mesenteric and peritoneal abnormalities that reflect the extent of ischemic injury. Early findings such as mural hypoenhancement, bowel wall thickening or thinning,

mesenteric fat stranding, vascular engorgement, and ascites may indicate potentially reversible disease, whereas pneumatosis intestinalis, portomesenteric venous gas, and free intraperitoneal air strongly suggest transmural necrosis and the need for urgent surgical intervention. The diagnostic performance of computed tomography angiography is high, especially when biphasic arterial and portal venous acquisition protocols are used, but accuracy depends on appropriate technique, radiologist expertise, and timely clinical suspicion. Beyond establishing the diagnosis, computed tomography directly influences treatment decisions by distinguishing etiologic subtypes and helping determine whether patients are best managed with anticoagulation, endovascular revascularization, surgery, or hemodynamic optimization. Nevertheless, limitations remain in very early ischemia, non-occlusive disease, and cases with subtle or nonspecific findings. For this reason, computed tomography findings must always be interpreted in conjunction with the clinical presentation and within a multidisciplinary framework to optimize early recognition, guide therapy, and improve outcomes in this high-mortality emergency.

Key words

Mesenteric ischemia, computed tomography angiography, early diagnosis, diagnostic performance, bowel ischemia, clinical decision-making.

Introduction

Early diagnosis is essential in mesenteric ischemia because it is a rare but serious condition associated with a high mortality rate, often exceeding 50% [1]. Prompt recognition significantly improves outcomes, and early revascularization has been associated with better survival rates [2]. However, early detection remains challenging because the clinical presentation is often nonspecific, with manifestations such as generalized abdominal pain and vomiting, which makes imaging a crucial component of diagnosis [1, 3].

In this context, computed tomography angiography has emerged as the preferred diagnostic tool because of its high sensitivity, reported at 92.0%, and specificity, reported at 98.8%, for the detection of acute mesenteric ischemia. Its value lies in its ability to identify vascular occlusions and assess bowel ischemia, both of which are critical for determining the most appropriate intervention. In addition, computed tomography can detect related intra-abdominal complications, although it may have limitations in accurately demonstrating intestinal necrosis [1, 4].

Beyond diagnosis, computed tomography angiography has substantial clinical value in decision-making because it facilitates the timely diagnosis and management of acute mesenteric ischemia and supports decisions regarding endovascular or surgical interventions. It is particularly useful for distinguishing between occlusive and non-occlusive mesenteric ischemia, which require different management strategies [2]. Furthermore, imaging findings can help determine the extent of surgical resection required, thereby potentially preventing unnecessary bowel loss and its associated complications [4].

Despite these advantages, computed tomography also has important challenges and limitations. Imaging alone may not reliably distinguish between different subtypes and stages of acute mesenteric ischemia, such as non-transmural and transmural ischemia. Although non-vascular findings, including absent bowel wall enhancement, may provide prognostic value, they are not sufficiently reliable when interpreted in isolation. Moreover, in some cases, computed tomography findings may not correlate with clinical outcomes, which underscores the importance of a multidisciplinary approach to diagnosis and management [2].

The objective of this study is to provide a structured and clinically integrated analysis of the role of computed tomography in the early diagnosis of mesenteric ischemia, with particular emphasis on its diagnostic performance and its utility in guiding clinical decision-making.

Methodology

This manuscript was developed as a structured narrative review aimed at providing an updated and clinically integrated analysis of the role of computed tomography in the early diagnosis of mesenteric ischemia, with particular emphasis on its diagnostic performance and its utility in guiding clinical decision-making. The review was conducted in accordance with the SANRA (Scale for the Assessment of Narrative Review Articles) framework and followed a predefined methodological protocol established prior to literature screening. Given the clinical heterogeneity of mesenteric ischemia, including its multiple etiologic subtypes and variable clinical presentations, a narrative interpretative synthesis was selected over quantitative pooling in order to integrate pathophysiological, radiological, and clinical considerations into a coherent and clinically applicable framework. Special attention was given to early imaging findings, differentiation of ischemic subtypes, identification of prognostic indicators of bowel viability, and the role of computed tomography in directing therapeutic strategies, including endovascular, surgical, and conservative approaches. The objective was to provide a structured synthesis capable of supporting multidisciplinary decision-making in emergency and critical care settings.

A comprehensive literature search was conducted in PubMed, Scopus, and Web of Science, including peer-reviewed articles published in English or Spanish between January 2020 and December 2025. The final search was performed in March 2026. This timeframe was selected to capture contemporary advances in computed tomography angiography protocols, diagnostic accuracy studies, imaging-based prognostic markers, and evolving management strategies in acute mesenteric ischemia. Foundational studies were incorporated when necessary to contextualize pathophysiological mechanisms and the historical evolution of imaging approaches. The search strategy combined MeSH terms and free-text keywords using Boolean operators related to mesenteric ischemia, acute mesenteric ischemia, computed tomography, CT angiography, bowel ischemia, vascular occlusion, diagnostic imaging, early diagnosis, bowel wall enhancement, pneumatosis intestinalis, and portomesenteric venous gas. Searches were conducted across titles, abstracts, and indexed subject headings to maximize sensitivity.

The initial search yielded 148 records. After removal of duplicates, 90 articles remained for title and abstract screening. Of these, 44 underwent full-text evaluation, and 15 studies were included in the final synthesis. Selection was performed independently by two authors, with disagreements resolved through discussion and consensus. Exclusion criteria comprised non–peer-reviewed publications, isolated case reports, editorials without diagnostic or outcome data, purely technical imaging descriptions without clinical correlation, redundant datasets, and studies not directly addressing diagnostic performance, imaging findings, or clinical utility of computed tomography in mesenteric ischemia.

Eligible studies included randomized controlled trials, large observational cohorts, systematic reviews, meta-analyses, expert consensus statements, and contemporary international guidelines from radiology, emergency medicine, vascular surgery, and critical care societies. Priority was assigned to multicenter investigations, studies with standardized imaging protocols, and research evaluating diagnostic accuracy, sensitivity, specificity, prognostic imaging markers, and clinical outcomes. Extracted variables included study design, patient population, etiology of mesenteric ischemia, computed tomography protocol

characteristics, vascular and bowel imaging findings, diagnostic performance metrics, therapeutic approach, and reported outcomes, including bowel viability, need for surgery, and mortality. Methodological quality and internal validity were assessed narratively, considering risk of bias, sample size, imaging protocol consistency, timing of imaging, and reproducibility of findings. In cases of conflicting evidence, greater interpretative weight was assigned to higher-level evidence and guideline-supported recommendations.

Reference lists of included studies were manually screened to identify additional relevant publications. Given its narrative design, this review is subject to potential selection bias and does not provide pooled quantitative estimates. Artificial intelligence–based tools were used exclusively to assist in literature organization and structural coherence, whereas critical appraisal, synthesis, and final interpretation were conducted independently by the authors to preserve methodological rigor.

Pathophysiology and Etiologic Classification of Mesenteric Ischemia

Mesenteric ischemia develops when intestinal oxygen delivery falls below the metabolic threshold required to sustain mucosal integrity. Because the villous tips receive blood through a countercurrent oxygen-exchange mechanism that creates physiological hypoxia at their apices, they are the first to necrose typically within one to three hours of flow interruption. Submucosal and muscular layers follow; by six hours of complete occlusion, transmural infarction may be established, gut barrier failure occurs, bacterial translocation ensues, and systemic inflammatory activation drives the rapid progression toward sepsis and death that defines the clinical urgency of this condition [5, 6]. Reperfusion, whether spontaneous or therapeutic, adds a second wave of injury mediated by reactive oxygen species and neutrophil activation, producing bowel wall edema and hemorrhagic transformation that have direct correlates on CT [5, 6].

Four etiologic subtypes underlie virtually all cases, each producing a distinct vascular and bowel wall injury pattern [5, 6, 7]. Arterial embolism, the most common cause at 40–50% of cases, originates predominantly from intracardiac thrombi in the setting of atrial fibrillation or recent myocardial infarction. Emboli lodge characteristically in the mid-portion of the superior mesenteric artery, beyond the middle colic branch, abruptly interrupting flow to the distal jejunum, ileum, and right colon while sparing the proximal jejunum. Because occlusion is sudden and collateral pathways are absent, ischemia progresses rapidly; CT shows a discrete intraluminal filling defect in the superior mesenteric artery, with absent or diminished bowel wall enhancement in the affected distribution, and concomitant emboli to the spleen or kidneys may confirm the cardiac source [6, 7]. Arterial thrombosis, responsible for 15–25% of cases, arises on a substrate of longstanding atherosclerosis and typically occludes the superior mesenteric artery at or near its aortic origin. The distribution of ischemia is more extensive than embolism often encompassing the entire small bowel but the subacute development of occlusion may allow partial collateral recruitment; CT reveals dense calcification at the vessel origin, origin-level occlusion of the superior mesenteric artery trunk, and diffuse bowel changes across a broad territory [5, 6]. Mesenteric venous thrombosis accounts for 5–15% of cases and follows a fundamentally different mechanism: obstruction of venous outflow raises intraluminal pressure and produces bowel wall congestion and edema before ischemia supervenes. Predisposing conditions include thrombophilia, portal hypertension, abdominal malignancy, and oral contraceptive use. CT demonstrates a low-attenuation thrombus within the superior mesenteric vein or portal vein, associated with pronounced bowel wall thickening often greater than in arterial occlusion mesenteric fat stranding, ascites, and vascular engorgement, while bowel wall enhancement is typically preserved in the early phase, reflecting the

venous rather than arterial nature of the insult [6, 7]. Non-occlusive mesenteric ischemia accounts for the remaining 20–30% of cases and occurs without anatomical vascular obstruction. Systemic hypoperfusion from cardiogenic shock, sepsis, or vasopressor therapy activates splanchnic vasoconstrictors catecholamines, vasopressin, angiotensin that redistribute blood away from the intestinal bed. Ischemia is diffuse and multisegmental, and CT characteristically reveals a patent superior mesenteric artery whose branches appear narrowed and pruned without a discrete filling defect, accompanied by patchy, poorly enhancing bowel wall changes across multiple segments [5, 6].

Across all subtypes, pneumatosis intestinalis and portomesenteric venous gas are the most specific CT markers of transmural necrosis and mandate urgent surgical reassessment regardless of etiology [6, 7]. Recognizing the etiologic mechanism from CT is clinically essential: embolism is treated by arterial catheter-directed thrombolysis or embolectomy; thrombosis requires revascularization and anticoagulation; venous thrombosis is managed with systemic anticoagulation; and non-occlusive ischemia demands correction of the underlying hemodynamic disorder. A pathophysiology-based approach to CT interpretation linking vascular anatomy, occlusion site, and bowel wall findings to the responsible mechanism is therefore the foundation of accurate early diagnosis and decisive therapeutic decision-making in this high-mortality condition [5, 6, 7].

Pathophysiology and Etiologic Classification of Mesenteric Ischemia

Contrast-enhanced multidetector CT angiography has become the unequivocal first-line imaging modality in suspected acute mesenteric ischemia, endorsed by the World Society of Emergency Surgery guidelines and the American College of Radiology Appropriateness Criteria alike [5, 8]. Its primacy in the emergency setting rests on a convergence of practical and diagnostic advantages that no other imaging modality currently replicates. CT is widely available around the clock in emergency departments, requires no specialized preparation, and delivers a complete examination from vascular anatomy to bowel wall and peritoneal assessment in seconds, making it uniquely suited to a condition whose outcome is critically time-dependent [7, 8, 9].

The diagnostic performance of CT angiography in mesenteric ischemia is well established. A recent systematic review and meta-analysis of 81 studies confirmed a sensitivity of 92% and specificity of 98.8% for CT angiography in diagnosing acute mesenteric ischemia, with non-angiographic CT protocols yielding substantially lower sensitivity (75.8%), underscoring that a biphasic acquisition arterial and portal venous phases is essential for adequate diagnostic yield [9]. The arterial phase resolves the mesenteric vasculature with sufficient detail to identify filling defects, occlusions, arterial caliber reduction, and venous thrombi; the portal venous phase optimizes bowel wall enhancement assessment, detects portomesenteric venous gas, and enables evaluation of the mesentery, peritoneum, and solid organs [6, 8]. Beyond its diagnostic accuracy for mesenteric ischemia itself, CT simultaneously surveys the entire abdomen for alternative or coexisting diagnoses including perforated viscus, pancreatitis, aortic pathology, and incarcerated hernia all of which can clinically mimic mesenteric ischemia and whose distinction is critical for correct emergency management [7, 8].

The clinical value of early CT acquisition ideally within minutes of clinical suspicion is substantial. Mesenteric ischemia is diagnosed by CT before the clinical syndrome reaches full expression in a significant proportion of cases, and studies have demonstrated that when AMI was not suspected prior to imaging, the diagnostic yield of radiology reports was significantly lower and rates of bowel resection were higher, confirming that clinical suspicion combined with timely CT is the optimal

diagnostic strategy [7]. CT findings encompass two complementary domains: direct vascular signs filling defects, vascular occlusion, caliber changes, venous thrombus that identify the responsible mechanism, and indirect bowel injury signs bowel wall thickening or thinning, abnormal wall enhancement, mesenteric stranding, ascites, pneumatosis intestinalis, and portomesenteric venous gas that reflect the depth of ischemic injury and determine urgency of intervention [6, 8]. Pneumatosis intestinalis and portomesenteric venous gas, the most specific markers of transmural necrosis, are reliably detected only with contrast-enhanced CT; unenhanced imaging alone misses the enhancement pattern abnormalities that are essential for detecting early reversible ischemia before necrosis supervenes [8, 9]. The ability of CT to differentiate pre-necrotic from transmural disease distinguishing cases still amenable to revascularization from those requiring immediate bowel resection directly informs surgical and endovascular decision-making and has direct implications for patient survival [8]. For all these reasons, CT is not merely a confirmatory test in mesenteric ischemia but the central tool around which time-sensitive clinical decisions are structured [5, 6, 8].

Computed Tomography Technique and Imaging Protocols

The recommended imaging protocol for suspected acute mesenteric ischemia centers on contrast-enhanced multidetector CT angiography performed with a multiphasic acquisition strategy that covers the abdomen and pelvis without oral contrast oral agents delay the examination, may be aspirated, and are not required for the vascular or bowel wall assessment that matters most [6, 8]. An optional non-enhanced acquisition prior to contrast injection provides a baseline for detecting intramural hemorrhage, high-attenuation emboli within the superior mesenteric artery lumen, and intramural or venous gas; it also enables quantitative comparison of bowel wall attenuation before and after contrast, which is particularly useful for identifying early wall hypoenhancement in equivocal cases [9, 10]. Intravenous administration of nonionic iodinated contrast at a concentration of 300–370 mg I/mL, delivered at a high flow rate of 4–5 mL/s through a large-bore peripheral cannula and followed by a saline flush of approximately 40 mL, produces the rapid, dense vascular opacification required to visualize small mesenteric vessel branches and bowel wall perfusion; lower flow rates result in suboptimal arterial enhancement and may miss subtle filling defects or wall hypoenhancement [8, 10]. Scan timing in the arterial phase is ideally controlled by bolus-tracking rather than fixed delays, with the trigger threshold placed in the aorta at approximately 100–150 Hounsfield units, initiating acquisition with a delay of 20–25 seconds after threshold this approach accommodates interpatient variability in cardiac output and contrast transit time, both of which are clinically relevant in the hemodynamically compromised patients who frequently present with mesenteric ischemia [8, 10]. The portal venous phase is acquired at a fixed delay of 60–70 seconds after contrast injection and constitutes the workhorse phase for bowel wall evaluation: it maximizes bowel wall enhancement in normal segments, making hypoenhancement or absent enhancement in ischemic segments visually conspicuous; it opacifies the portal vein and mesenteric veins to reveal venous thrombus; and it allows comprehensive evaluation of the solid organs, peritoneum, and mesenteric fat [6, 8].

Thin-section acquisition at 0.5–1 mm slice thickness throughout both phases is essential for diagnostic quality in mesenteric ischemia CT. Thin collimation allows the generation of high-resolution multiplanar reconstructions in coronal and sagittal planes, which are indispensable for tracing the full course of the superior mesenteric artery and its branches, identifying the precise location and extent of filling defects or thrombosis, and evaluating bowel loop distribution and mesenteric root involvement in three dimensions information that axial images alone cannot convey with equivalent clarity [8,

10]. Maximum intensity projection reconstructions of the arterial phase highlight the vascular tree and render mesenteric vessel occlusions, stenoses, and arterial caliber reduction immediately apparent, while volume-rendered images provide an intuitive overview of vascular anatomy useful for surgical or endovascular planning [7, 10]. Coronal multiplanar reconstructions of the portal venous phase are particularly valuable for assessing the extent of bowel involvement, identifying the transition between enhancing and non-enhancing segments, and detecting portomesenteric venous gas distributed along the peripheral branches of the portal system within the hepatic parenchyma findings that may be subtle on axial images but clearly visible on reformatted views [6, 7].

Several technical factors exert a decisive influence on diagnostic accuracy and are worth explicitly addressing in clinical practice. Inadequate arterial phase timing whether from scanning too early before peak mesenteric arterial enhancement or too late as opacification shifts to the venous system is the single most common cause of missed arterial filling defects and false-negative interpretations of bowel wall enhancement; bolus-tracking acquisition is therefore preferred over empirical fixed delays, especially in patients receiving vasopressors or with poor cardiac function [8, 10]. Low kilovoltage peak scanning typically 80–100 kVp rather than the standard 120 kVp increases iodine attenuation and enhances the visual conspicuity of both vascular filling defects and bowel wall enhancement differences, improving detection of early ischemia without requiring additional contrast volume; dual-energy CT acquisition achieves the same iodine conspicuity advantage while simultaneously generating virtual monoenergetic images and iodine maps that can quantify bowel wall perfusion in a way conventional CT cannot [10]. Finally, the role of radiologist experience and systematic structured image interpretation cannot be overstated: studies have demonstrated that the rate of missed diagnoses on CT is significantly higher when mesenteric ischemia is not clinically suspected before imaging, reflecting the perceptual challenges posed by subtle early findings such as mild bowel wall hypoenhancement or isolated mesenteric stranding findings that require active search and knowledge of their significance in context rather than incidental detection [7, 9].

Early Computed Tomography Findings in Mesenteric Ischemia

The early CT findings of mesenteric ischemia are most usefully organized into three domains vascular, bowel wall, and mesenteric/peritoneal because each domain reflects a distinct aspect of the underlying injury and carries different diagnostic weight in terms of specificity, severity, and therapeutic urgency [6, 10, 11].

The vascular findings are the most diagnostically specific and are often the first abnormality detectable on CT before bowel wall injury has advanced to irreversible stages [10]. In arterial embolism, the cardinal finding is a discrete intraluminal filling defect within the superior mesenteric artery on the arterial phase, characteristically located in the mid-vessel beyond the middle colic branch origin, typically eccentric and with an abrupt vessel cutoff; on unenhanced images a high-attenuation embolus may be apparent even before contrast administration [6, 7]. In arterial thrombosis, the occlusion is seen at or near the aortic origin of the superior mesenteric artery, frequently surrounded by dense atherosclerotic calcification, with absent opacification of the proximal trunk and its branches in the affected distribution [11]. In mesenteric venous thrombosis, the arterial phase appears normal but the portal venous phase reveals a hypodense filling defect within the superior mesenteric vein or portal vein, expanding the vessel lumen and occasionally extending into intrahepatic portal branches; associated mesenteric venous engorgement enlarged, tortuous mesenteric veins visible as prominent vascular structures within the mesenteric root is an important early ancillary sign that suggests venous outflow obstruction

before bowel wall injury has developed [6, 11]. In non-occlusive mesenteric ischemia, the vessels are patent but abnormally narrowed: the superior mesenteric artery branches appear pruned and irregularly constricted with reduced caliber across multiple generations, a finding best appreciated on multiplanar reformations of the arterial phase, without a focal filling defect but with diffuse underperfusion of the bowel [10, 11].

The bowel wall findings reflect the depth and mechanism of ischemic injury and vary systematically across etiologic subtypes [6, 11]. Mural hypoenhancement reduced or absent bowel wall opacification on the portal venous phase compared to adjacent normal segments is among the earliest and most specific signs of arterial ischemia and indicates compromised mucosal perfusion; it requires an optimized contrast protocol to detect confidently and may be subtle when ischemia is incomplete or collateral flow is partially preserved [10]. Bowel wall thickening, defined as a wall measuring more than 3 mm in a non-distended loop, is common but non-specific; it results from submucosal edema and hemorrhage and is characteristically prominent in venous thrombosis, where congestion drives progressive transmural edema, producing the halo or target sign concentric rings of inner mucosal enhancement, submucosal edema, and outer serosal enhancement which reflects preserved mucosal perfusion against a background of submucosal fluid accumulation [6, 11]. In severe arterial occlusion, paradoxically thin bowel wall the so-called paper-thin wall appearance results from the loss of mural volume and vascular tone in profoundly ischemic or infarcted segments, and represents a marker of more advanced injury than thickening alone [11]. Luminal dilatation of affected bowel loops a consequence of ileus from ischemia-induced bowel wall dysfunction is an early, if non-specific, sign that prompts attention to the vascular anatomy and enhancement pattern of the dilated segments; it is frequently the feature that first brings affected loops to the radiologist's notice before vascular findings are actively sought [10]. Segmental distribution of bowel changes rather than diffuse or random involvement is a key interpretive principle: involvement confined to a vascular territory helps confirm an ischemic rather than inflammatory etiology and guides the clinician toward the responsible vessel [6, 11].

The mesenteric and peritoneal findings complete the CT assessment by reflecting the indirect consequences of bowel and vascular injury on the surrounding compartments [10, 11]. Mesenteric fat stranding increased attenuation and haziness of the mesenteric fat adjacent to affected bowel loops results from transudation of fluid across a compromised bowel wall and mesenteric vascular permeability; it is more prominent and extensive in venous thrombosis due to the congestive mechanism but can be seen in any subtype when ischemia is sufficiently advanced [10]. Mesenteric vascular engorgement prominently visible mesenteric vessels with increased caliber in the distribution of affected bowel is an early and reliable sign of venous obstruction that appears before significant bowel wall edema develops and provides early spatial guidance toward the affected vascular territory [11]. Small-volume free peritoneal fluid between bowel loops and in the pelvic dependent recesses is common and, in the correct clinical and imaging context, supports the diagnosis of advanced bowel wall permeability; it is sensitive but not specific on its own and gains diagnostic significance when combined with other findings in the same distribution [10, 11].

The clinical importance of systematically recognizing these early findings vascular, bowel, and mesenteric before irreversible necrosis develops cannot be overemphasized. Bowel wall hypoenhancement, mesenteric engorgement, and filling defects in the absence of pneumatosis or portomesenteric venous gas represent a pre-necrotic window during which revascularization whether by catheter-directed thrombolysis, surgical embolectomy, or correction of the

underlying hemodynamic state can restore perfusion and preserve bowel viability [5, 6, 10]. The recognition of these early CT signs, before the irreversible markers of transmural necrosis appear, is therefore the most meaningful contribution CT can make to the clinical management of this condition, transforming a historically late and fatal diagnosis into one in which salvage intervention remains possible [7, 9].

Diagnostic Performance of Computed Tomography

The diagnostic performance of CT angiography in acute mesenteric ischemia is among the highest of any imaging modality in emergency abdominal radiology, and its evidence base now extends across multiple large systematic reviews and meta-analyses encompassing thousands of patients across diverse study designs and geographic settings [9, 12]. The most comprehensive recent meta-analysis, incorporating 81 studies identified through a systematic search of four major databases, confirmed a pooled sensitivity of 92.0% and a pooled specificity of 98.8% for CT angiography in the diagnosis of acute mesenteric ischemia, with substantially lower performance for CT protocols that omit the dedicated arterial phase non-angiographic acquisitions yielded sensitivity of 75.8% and specificity of 90.5% underscoring that protocol quality is itself a determinant of diagnostic accuracy rather than a fixed property of the modality [9]. These figures position CT angiography as the only non-operative investigation with sufficient accuracy to confirm or exclude acute mesenteric ischemia in lieu of diagnostic laparotomy, a role that no clinical scoring tool, laboratory biomarker, or ultrasound examination has been able to replicate with comparable reliability [5, 9].

Diagnostic performance is not, however, a fixed quantity and varies systematically across several clinical and technical dimensions that the interpreting radiologist must understand. Disease stage at the time of imaging has a pronounced effect: CT performs best when vascular findings filling defects, vessel occlusion, venous thrombus are present and unmistakable on the arterial phase, as occurs in the early or intermediate phases of arterial embolism and venous thrombosis; performance diminishes in very early ischemia when the only manifestation is subtle bowel wall hypoenhancement that requires both an optimized acquisition and an experienced reader to detect confidently [10, 11]. Conversely, in late-stage disease with established transmural necrosis, CT sensitivity approaches 100% because pneumatosis intestinalis, portomesenteric venous gas, and free peritoneal air are highly conspicuous findings that are rarely missed; the diagnostic challenge at this point has shifted from detection to management, as the clinical implication is immediate surgical intervention regardless of etiology [6, 9]. Etiology also influences performance: arterial embolism and venous thrombosis both produce focal, vessel-specific vascular findings that are reliably identified on CT angiography; non-occlusive mesenteric ischemia, which lacks a discrete filling defect and presents only with diffuse arterial caliber reduction and non-specific bowel changes, is the most diagnostically challenging subtype and carries a higher risk of under-recognition, particularly when it occurs as a secondary phenomenon in patients with septic shock or following cardiac surgery who are being imaged for other reasons [7, 10]. Radiologist expertise represents an equally important and modifiable determinant: the rate of correctly reported key imaging findings in confirmed cases of acute mesenteric ischemia was found to be 97% when AMI was specifically mentioned in the clinical referral, compared to 81% when no clinical suspicion was communicated a statistically significant difference with direct consequences for patient outcomes, as cases in which the diagnosis was not clinically suspected prior to imaging were more likely to undergo bowel resection [7]. This observation reflects the perceptual dependency of AMI diagnosis on active search: findings such as subtle bowel wall hypoenhancement, early mesenteric venous

engorgement, or mild mesenteric fat stranding are reliably recognized when looked for but easily overlooked during a non-directed scan review aimed at other potential diagnoses [7, 9].

The diagnostic performance of CT in mesenteric ischemia is maximized when vascular, bowel wall, and mesenteric findings are systematically interpreted in combination rather than evaluated in isolation, because no single CT sign achieves both high sensitivity and high specificity alone [6, 11]. Vascular findings filling defects and vessel occlusion are highly specific but may be absent in non-occlusive disease; bowel wall thickening and mesenteric stranding are sensitive but non-specific; mural hypo enhancement is specific for arterial ischemia but requires protocol optimization to detect; pneumatosis and portomesenteric venous gas are highly specific for necrosis but appear only in advanced disease [6, 9, 11]. Combining findings across these three domains vascular anatomy, bowel wall, and mesenteric compartment in the context of a consistent clinical presentation and the appropriate etiologic subtype substantially increases diagnostic confidence and reduces both false-negative and false-positive interpretations [6, 10]. Beyond confirming ischemia, CT provides an additional layer of clinical value by simultaneously evaluating for alternative diagnoses that can produce an identical clinical presentation of severe acute abdominal pain with hemodynamic instability including acute pancreatitis, perforated viscus, aortic dissection, and incarcerated hernia all of which require fundamentally different and often equally urgent management strategies [7, 8]. In clinical practice, a significant majority of patients presenting with suspected acute mesenteric ischemia will not have ischemia, making the capacity of CT to confidently exclude the diagnosis and redirect management toward the true etiology an equally important component of its diagnostic value in the acute abdomen [7, 9].

Clinical Utility in Therapeutic Decision-Making

The clinical value of CT in acute mesenteric ischemia extends well beyond establishing the diagnosis: by characterizing the etiologic subtype, quantifying the extent and depth of bowel injury, and identifying the presence or absence of irreversible complications, CT provides the information necessary to select between fundamentally different treatment pathways anticoagulation, endovascular revascularization, surgery, or hemodynamic optimization that cannot be appropriately chosen on clinical grounds alone [5, 8, 12]. This treatment-guiding function makes CT not merely a confirmatory test but the operational foundation of acute mesenteric ischemia management.

Etiology, as revealed by CT, determines the primary treatment modality [5, 7]. When the arterial phase demonstrates a discrete filling defect consistent with embolism in a vessel with otherwise normal caliber, and the bowel wall remains enhancing without pneumatosis, endovascular embolectomy or catheter-directed thrombolysis is the preferred approach; CT not only identifies the target vessel and level of occlusion but also evaluates the proximal vascular anatomy for access planning and detects any distal branch involvement that might necessitate combined endovascular and surgical strategies [5, 8]. In atherosclerotic arterial thrombosis, the proximal and calcified nature of the occlusion identified on CT influences the choice between open surgical revascularization and hybrid endovascular repair, and the extent of bowel involvement on the venous phase guides the urgency of the decision [5, 7]. When mesenteric venous thrombosis is diagnosed identified as a low-attenuation filling defect within the superior mesenteric vein on the portal venous phase, with associated bowel wall edema but preserved mucosal enhancement anticoagulation is the primary treatment in the absence of necrosis; CT findings at this stage can support a conservative, non-operative approach when the bowel wall appears viable [5, 7]. In non-occlusive mesenteric ischemia, where CT

reveals a patent but diffusely constricted arterial tree, the treatment is directed at correcting the precipitating hemodynamic disturbance vasodilator infusion, correction of hypovolemia, reduction of vasopressor dose rather than vascular intervention, a distinction that CT uniquely enables by excluding a focal mechanical cause [5, 10].

The differentiation between potentially reversible ischemia and established transmural infarction is the most critical treatment-relevant assessment CT can provide, and it is one for which a systematic meta-analysis of radiological predictive factors has generated clear, quantified evidence [13]. A systematic review and meta-analysis of 11 studies including 1,037 patients identified the CT features most strongly associated with transmural necrosis: bowel wall thinning carried the highest diagnostic odds ratio (DOR 13.10; 95% CI 3.71–46.25), reflecting the loss of mural volume in irreversibly infarcted bowel; absent or severely reduced bowel wall enhancement had a DOR of 5.77 (95% CI 2.95–11.30), reflecting transmural ischemia; pneumatosis intestinalis showed a DOR of 5.78 (95% CI 2.24–14.95) and portomesenteric venous gas a DOR of 5.36 (95% CI 2.14–13.40), both reflecting gas-producing bacterial invasion of an already necrotic wall; bowel dilatation contributed a DOR of 3.23 (95% CI 2.03–5.15), reflecting the paralytic ileus of infarcted bowel [13]. The practical implication of these findings is a CT-based triage: a patient with an arterial filling defect and mildly reduced but present bowel wall enhancement in the absence of pneumatosis, portomesenteric venous gas, or bowel dilatation is a candidate for urgent revascularization with potential bowel salvage; a patient in whom pneumatosis and portomesenteric venous gas coexist with absent wall enhancement and paper-thin bowel loops requires immediate surgical exploration and resection, as revascularization of established necrosis risks reperfusion-mediated hemorrhage and systemic endotoxemia without bowel salvage [5, 6, 13].

Pneumatosis intestinalis and portomesenteric venous gas deserve particular attention as CT complications that immediately and unambiguously shift clinical management toward the operating room [5, 6]. Pneumatosis, visible as curvilinear or bubbly intramural gas best appreciated on lung windows, represents bacterial fermentation of the infarcted wall and is associated with transmural necrosis in approximately 78–81% of cases when seen in the context of clinical mesenteric ischemia [6]. Portomesenteric venous gas distributed as peripheral branching hypoattenuation within the hepatic parenchyma, or as gas bubbles within the mesenteric venous radicles carries a similar association with full-thickness infarction and demands urgent surgical triage [6, 13]. Free intraperitoneal air, when present, indicates frank perforation and is an absolute surgical indication requiring no further imaging workup [5]. The concurrent identification of these three findings pneumatosis, portomesenteric venous gas, and free air on a single CT examination defines the highest urgency tier of mesenteric ischemia management and allows the emergency team to mobilize surgical resources before the patient reaches the theater [5, 8].

Beyond treatment selection, CT plays a pivotal role in facilitating multidisciplinary coordination between emergency medicine, vascular surgery, interventional radiology, and intensive care a coordinated approach that is associated with improved outcomes in specialized centers [7, 8]. The CT report communicates in a structured way the etiologic subtype, the affected vascular territory, the presence or absence of irreversible findings, and the most likely treatment pathway, enabling each specialty to prepare its role before the patient arrives. In institutions where mesenteric ischemia is managed within dedicated intestinal stroke or mesenteric ischemia units, the CT findings serve as the shared clinical language around which multidisciplinary decisions are built, supporting both individualized treatment and systematic quality improvement [7, 8].

Limitations, Pitfalls, and Differential Considerations

Despite its established primacy as the first-line diagnostic modality, CT carries intrinsic limitations that the clinician and radiologist must understand to avoid diagnostic failure particularly in the early and subtle stages of ischemia where intervention remains most beneficial [9, 14]. The most significant limitation is the detection of very early arterial ischemia before bowel wall changes have developed. In the first one to three hours after an embolic or thrombotic occlusion, the only CT abnormality may be the vascular filling defect itself, while the bowel wall remains morphologically normal and normally enhancing; if this vascular finding is not actively sought particularly in patients who present without a prior clinical suspicion of mesenteric ischemia CT may be reported as unremarkable and the diagnosis missed [7, 14]. The reduction in bowel wall enhancement that characterizes pre-necrotic arterial ischemia is a subtle, continuous, and reader-dependent finding: it requires optimal arterial phase timing, adequate contrast delivery, and active side-by-side comparison of the suspect and normal segments; under suboptimal protocol conditions or in the absence of a dedicated active search, this early sign is easily overlooked [10, 14]. Non-occlusive mesenteric ischemia presents an additional detection challenge, because the arterial vessels are patent and the bowel wall changes diffuse, patchy, and poorly demarcated may be attributed to ileus, inflammation, or bowel preparation artifact rather than ischemia, particularly when the clinical diagnosis has not been communicated to the radiologist [7, 10].

Diagnostic pitfalls in CT interpretation of mesenteric ischemia arise from the overlap between ischemic and non-ischemic bowel pathology across all three finding domains vascular, bowel wall, and mesenteric [14]. Bowel wall thickening, the most common CT sign of mesenteric ischemia, is also among the most non-specific findings in abdominal radiology: it occurs with equal frequency in Crohn's disease, infectious enteritis, radiation enteritis, angioedema, hypoalbuminemia, and intramural hemorrhage, none of which require vascular intervention [9, 14]. A non-distended bowel loop may simulate wall thickening by collapsed lumen alone the so-called pseudo-thickening pitfall leading to false-positive interpretation in patients with generalized bowel hypomotility [9]. Conversely, isolated bowel loop dilation without other findings may be attributed to non-ischemic ileus when in fact it represents early mesenteric ischemia causing paralytic bowel dysfunction before wall injury is radiologically apparent (14). The halo or target sign of bowel wall enhancement classically associated with venous thrombosis can also be produced by inflammatory bowel disease and infectious colitis, creating false-positive diagnostic impressions when the pattern is observed without corresponding venous filling defects [11, 14]. Portal venous gas and pneumatosis intestinalis, while highly specific in the ischemic clinical context, can occur in benign settings endoscopy, enterocolitis, blunt abdominal trauma, colonoscopy-related complications and must be interpreted strictly in conjunction with the clinical presentation rather than as isolated deterministic findings [6, 14]. Solid organ infarcts elsewhere in the abdomen splenic or renal infarcts visible on CT raise suspicion for an embolic source and should prompt active search for mesenteric arterial filling defects, serving as an indirect vascular finding that can elevate diagnostic suspicion before bowel wall changes appear [6, 14].

Clinically relevant constraints exist for CT use in certain patient subgroups [5, 8]. Patients with advanced chronic kidney disease or acute kidney injury face the risk of contrast-induced nephropathy from iodinated contrast media; however, the WSES guidelines and major expert groups emphasize that in suspected acute mesenteric ischemia where the consequence of diagnostic delay is bowel necrosis, sepsis, and death the risk of contrast nephropathy is substantially outweighed by the risk of missed

diagnosis, and CT angiography should not be withheld on renal grounds alone [5]. Documented contrast allergy may be managed with appropriate premedication, though the emergency urgency of mesenteric ischemia often limits the time available for conventional premedication protocols; in such cases, the benefits of performing the study with modified premedication or proceeding without it must be weighed against the catastrophic consequences of diagnostic delay [5, 8]. Severe hemodynamic instability and cardiogenic shock frequently the same conditions that predispose to non-occlusive mesenteric ischemia create competing priorities for the emergency team: the patient who is most hemodynamically compromised is both most likely to have non-occlusive ischemia and least able to be safely transported to the CT suite, requiring a clinical judgment about whether rapid resuscitation can safely enable imaging or whether operative decision-making must proceed empirically [5, 10].

The paramount practical implication of all these limitations is that CT findings in mesenteric ischemia must never be interpreted in isolation from the clinical context [7, 9, 14]. A structurally convincing CT report that attributes bowel wall thickening to inflammatory disease in a 70-year-old patient with atrial fibrillation and acute-onset periumbilical pain out of proportion to physical examination represents a diagnostic failure regardless of the formal imaging findings, because the clinical pretest probability mandates a vascular diagnosis until disproved. Equally, a normal CT in a patient with a very short symptom duration and compelling clinical features does not exclude early arterial ischemia; the WSES guidelines explicitly recommend that CTA should not delay treatment in patients with clear clinical indicators of peritonitis, and that clinical and imaging information must be integrated rather than treated sequentially [5]. The role of laboratory testselevated lactate, rising D-dimer, and leukocytosisas supporting contextual data rather than standalone diagnostic tools is well established; abnormal values increase the pretest probability that guides active CT search, while normal values do not reduce it sufficiently to reassure in the appropriate clinical setting [5, 7]. The single most effective intervention for reducing false-negative CT interpretations in mesenteric ischemia is ensuring that clinical suspicion is explicitly communicated to the interpreting radiologist before or at the time of image review a finding that has been quantified in retrospective studies demonstrating that 16% of confirmed cases were not correctly reported when AMI suspicion was absent from the clinical referral [7, 15].

Conclusions

Contrast-enhanced computed tomography angiography is the cornerstone of early diagnosis in acute mesenteric ischemia because it combines very high diagnostic accuracy with rapid evaluation of vascular, bowel wall, and mesenteric abnormalities, allowing both confirmation of ischemia and exclusion of alternative abdominal emergencies. Its diagnostic yield is greatest when biphasic protocols and structured interpretation are used.

The greatest clinical value of computed tomography lies not only in detecting mesenteric ischemia, but also in defining its etiologic subtype and estimating bowel viability, which directly determines treatment strategy. By distinguishing embolic, thrombotic, venous, and non-occlusive mechanisms, and by identifying reversible versus transmural injury, computed tomography guides the choice between anticoagulation, revascularization, surgery, or hemodynamic optimization.

Despite its excellent performance, computed tomography has important limitations in very early ischemia, non-occlusive disease, and cases with subtle or nonspecific bowel findings, so imaging must always be interpreted together with clinical suspicion and multidisciplinary assessment. Early communication of diagnostic concern to the radiologist significantly improves

detection and may reduce delayed treatment and bowel loss.

References

1. Molyneux K, Beck-Esmay J, Koyfman A, Long B. High risk and low prevalence diseases: Mesenteric ischemia. The American Journal of Emergency Medicine [Internet]. 2023 Jan 4;65:154–61. Available from: https://doi.org/10.1016/j.ajem.2023.01.001
2. Blaser AR, Forbes A, Björck M. Acute mesenteric ischaemia. Current Opinion in Critical Care [Internet]. 2022 Aug 10;28(6):702–8. Available from: https://doi.org/10.1097/mcc.0000000000000972
3. Costello L, Duggan WP, Flanagan M, Toale C, Kavanagh DO. Current Approaches to Diagnosis and Management of Acute Mesenteric Ischaemia: A scoping review. Digestive Surgery [Internet]. 2025 Jul 15;42(5):229–46. Available from: https://doi.org/10.1159/000547297
4. Balci NC, Kumar S, Ansari HA. Editorial for "MRI for the Detection of Small bowel ischemic Injury in Arterial Acute mesenteric Ischemia: Pre-clinical Study in a Porcine model." Journal of Magnetic Resonance Imaging [Internet]. 2022 Jul 13;57(3):928–9. Available from: https://doi.org/10.1002/jmri.28341
5. Bala M, Catena F, Kashuk J, De Simone B, Gomes CA, Weber D, et al. Acute Mesenteric Ischemia: Updated Guidelines of the World Society of Emergency Surgery. World J Emerg Surg [Internet]. 2022 Oct 19;17(1):54. Available from: https://doi.org/10.1186/s13017-022-00443-x
6. Olson MC, Bach CR, Wells ML, Andrews JC, Khandelwal A, Welle CL, et al. Imaging of Bowel Ischemia: An Update, From the AJR Special Series on Emergency Radiology. AJR Am J Roentgenol [Internet]. 2023 Feb;220(2):173–85. Available from: https://doi.org/10.2214/AJR.22.28140
7. Garzelli L, Ben Abdallah I, Nuzzo A, Zappa M, Corcos O, Dioguardi Burgio M, et al. Insights into Acute Mesenteric Ischaemia: An Up-to-Date, Evidence-Based Review from a Mesenteric Stroke Centre Unit. Br J Radiol [Internet]. 2023 Nov;96(1151):20230232. Available from: https://doi.org/10.1259/bjr.20230232
8. Ronza FM, Di Gennaro TL, Buzzo G, Piccolo L, Della Noce M, Giordano G, et al. Diagnostic Role of Multi-Detector Computed Tomography in Acute Mesenteric Ischemia. Diagnostics [Internet]. 2024 Jun 7;14(12):1214. Available from: https://doi.org/10.3390/diagnostics14121214
9. Reintam Blaser A, Koitmäe M, Laisaar KT, Forbes A, Kase K, Kiisk E, et al. Radiological Diagnosis of Acute Mesenteric Ischemia in Adult Patients: A Systematic Review and Meta-Analysis. Sci Rep [Internet]. 2025 Mar 22;15(1):9875. Available from: https://doi.org/10.1038/s41598-025-94846-w
10. Yu H, Kirkpatrick IDC. An Update on Acute Mesenteric Ischemia. Can Assoc Radiol J [Internet]. 2023 Feb;74(1):160–71. Available from: https://doi.org/10.1177/08465371221094280
11. Nakamura Y, Kondo S, Narita K, Maeda S, Fonseca D, Honda Y, et al. Understanding CT Imaging Findings Based on the Underlying Pathophysiology in Patients with Small Bowel Ischemia. Jpn J Radiol [Internet]. 2023;41(4):353–66. Available from: https://doi.org/10.1007/s11604-022-01367-x

12. Garzelli L, Nuzzo A, Copin P, Calame P, Corcos O, Vilgrain V, et al. Contrast-Enhanced CT for the Diagnosis of Acute Mesenteric Ischemia. AJR Am J Roentgenol [Internet]. 2020 Jul;215(1):29–38. Available from: https://doi.org/10.2214/AJR.19.22625
13. Zeng Y, Yang F, Hu X, Zhu F, Chen W, Lin W. Radiological Predictive Factors of Transmural Intestinal Necrosis in Acute Mesenteric Ischemia: Systematic Review and Meta-Analysis. Eur Radiol [Internet]. 2023 Apr;33(4):2792–9. Available from: https://doi.org/10.1007/s00330-022-09258-5
14. Fitzpatrick LA, Rivers-Bowerman MD, Thipphavong S, Clarke SE, Rowe JA, Costa AF. Pearls, Pitfalls, and Conditions that Mimic Mesenteric Ischemia at CT. RadioGraphics [Internet]. 2020 Mar–Apr;40(2):545–61. Available from: https://doi.org/10.1148/rg.2020190122
15. Ojha V, Mani A, Mukherjee A, Kumar S, Jagia P. Mesenteric ischemia in patients with COVID-19: an updated systematic review of abdominal CT findings in 75 patients. Abdominal Radiology [Internet]. 2021 Nov 10;47(5):1565–602. Available from: https://doi.org/10.1007/s00261-021-03337-9