

Review Article

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A Paradigm Shift in Acute Mesenteric Ischemia: A Comprehensive Guide from Anatomical Staging to Microvascular Thromboinflammation, Advanced Revascularization Strategies, And Long-Term Survival Dynamics

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Abstract

Background & Aim

Acute mesenteric ischemia (AMI) is a highly time-sensitive clinical syndrome and one of the most lethal cardiovascular emergencies, with mortality rates ranging from 50% to 80% [1]. Historically viewed merely as a mechanical issue of a blocked lumen (macrovascular occlusion), this condition is now redefined as a complex spiral of microvascular dysfunction and immunothrombosis [2]. This review aims to dismantle traditional dogmas in AMI management by synthesizing the latest Q1-level evidence on the pathophysiological role of venular thromboinflammation, anatomical occlusion staging (Stages A-C), advanced radiological and biochemical risk stratification, highly specific catheter-directed thrombolytic protocols, and the impact of modern “endovascular-first” strategies alongside rescue hybrid surgical techniques on long-term survival.

Evidence Synthesis

Translational studies have proven that even when macroscopic arterial recanalization is achieved, microvascular stasis-termed “venular-centered thromboinflammation”-sustains intestinal injury [2]. In terms of time management, while irreversible necrosis takes up to 12 hours in mesenteric ischemia compared to 6 hours in renal artery ischemia, this window is compressed to just 5 hours in proximal (Stage A) occlusions. Thin-slice Computed Tomography Angiography (CTA) remains the absolute gold standard in diagnostic algorithms [3]. Concurrently, specific clinical trials, scoring systems such as RADIAL and HALP, and host-response biomarkers like SeptiCyte RAPID are revolutionizing clinical risk stratification [4]. Therapeutically, the ESVS 2025 guidelines dictate an “endovascular-first” strategy; targeted t-PA infusions via pigtail or peripheral microcatheters (CDT) and hybrid/autogenous techniques such as Retrograde Open Mesenteric Stenting (ROMS) and Superior Mesenteric Artery (SMA) transposition serve as lifesaving modalities in the presence of heavily calcified lesions or peritonitis [5].

Conclusion

AMI is no longer just a traditional gastrointestinal emergency; it is a systemic cardiovascular crisis that demands multidisciplinary management under the umbrella of dedicated “Intestinal Stroke Centres” [6]. For patients who survive the initial AMI episode and are discharged, the 1-year survival rate reaches a remarkable 81.3%; long-term prognosis is integrated not with the ischemic subtype, but with the patient’s age-adjusted comorbidity burden (aCCI) and dependency on chronic parenteral nutrition [7].

Introduction

Acute Mesenteric Ischemia is a catastrophic syndrome where diagnostic uncertainties and therapeutic delays exact a heavy toll in the form of transmural bowel necrosis and multiple organ failure [1]. Although historical data attributed 40–50% of AMI cases to cardiac emboli, aging population dynamics and the global atherosclerotic pandemic have driven a pronounced upward trend in the incidence of in-situ arterial thrombosis (25–40%) [8].

Furthermore, the epidemiological profile exhibits a sharp age dichotomy. While arterial thrombosis and embolism dominate geriatric cohorts over 65, the primary mechanism in young adults aged 20–39 is mesenteric venous thromboembolism (MVT), accounting for 44% of cases in this demographic [9]. This epidemiological shift explains why a “one-size-fits-all” treatment approach inherently fails in AMI management. The lethal nature of the disease hinges not merely on reopening an occluded vessel, but on how effectively ischemia is anatomically contained, how microvascular stasis is prevented, and how systemic inflammation is modulated [2].

Pathophysiology and Anatomical Staging: The Venular Thromboinflammation Paradigm

One of the greatest frustrations in clinical practice is losing patients in the post-operative period to bowel necrosis and Systemic Inflammatory Response Syndrome (SIRS) despite successful surgical or endovascular recanalization (achieving TIMI 3 flow) of the occluded SMA [2]. Recent translational research utilizing intravital microscopy has illuminated the microvascular origins of this “no-reflow” phenomenon [2]. Initiated during ischemia and exacerbated by the re-entry of oxygen (reperfusion injury), this process is defined as “venular-centered thromboinflammation” [2]. Immunofluorescence analyses prove that these venular thrombi are not simple clots; they are organized immunothrombotic structures featuring a core of platelet/fibrin networks, surrounded by a periphery of neutrophils and Neutrophil Extracellular Traps (NETs) [2].

Macrovascular Anatomical Staging and Collateral Dynamics:

At the macrovascular level, the singular factor determining the severity of ischemia and bowel survival time is the anatomical level of the thrombosis. In this context, SMA thrombosis is evaluated in three critical stages:

- **Stage A (Proximal Occlusion):** The most lethal scenario, where the thrombus is located in the SMA trunk, proximal to the right colic and middle colic arteries. Protective collateral flow cannot form at this level, and fulminant necrosis typically develops within a short window of 5 hours.
- **Stage B (Mid-Segment Occlusion):** The thrombus is located between the right colic artery and the ileocolic artery.
- **Stage C (Distal Occlusion):** The thrombus is located distal to the ileocolic artery.

In Stage B and C lesions, collateral networks connecting the Inferior Mesenteric Artery (IMA) to the middle colic artery of

the SMA via the Arc of Riolan are rapidly engaged. Thanks to this anatomical lifeline, the intestines can be perfused for extended periods without necrosis, and in appropriate clinical scenarios, the window for endovascular thrombolytic therapy can extend up to 3 days.

Ischemic Thresholds, Time Management, and the Systemic Transfer Penalty

The rule “Time is gut” governs AMI management. Ischemia tolerance times vary according to organ metabolic rates and parenchymal structures. As a general rule; while irreversible necrosis takes approximately 12 hours in mesenteric artery ischemia (dropping to 5 hours in Stage A occlusions), this window is compressed to a mere 6 hours in highly metabolically active renal artery ischemia. Large-scale retrospective cohort analyses have unequivocally established that 48 hours from symptom onset to revascularization is a critical systemic inflection point [3]. Exceeding 48 hours skyrockets perioperative mortality from 14% to 39% (HR 2.95), increases recurrent thrombosis risk 6.3-fold, and surges the development of short bowel syndrome due to massive resection from 12% to 39% [3].

However, the primary barrier closing this golden window is systemic logistical hurdles rather than diagnostic difficulties. The interhospital transfer of patients presenting to centres lacking advanced angiographic and hybrid surgical infrastructure can turn into a ticking time bomb. A massive analysis of 39,690 patients from the US National Inpatient Sample (NIS) reveals that interhospital transfer-as an independent factor regardless of patient age and comorbidity-increases the risk of in-hospital mortality by 67% (aOR 1.67) [10].

Clinical Trials, Biomarkers, And Precision Medicine

There is no room for a “wait-and-see” approach in diagnosing AMI. Thin-slice (<1 mm) Computed Tomography Angiography (CTA), encompassing both arterial and venous phases, remains the absolute gold standard (ESVS 2025 Class I Evidence) [3]. Normal levels of traditional markers such as lactate, D-Dimer, or leukocytosis absolutely do not exclude early-stage AMI; elevated lactate typically reflects advanced-stage transmural necrosis and systemic hypoperfusion rather than early ischemia [11]. CTA decisions must never be delayed while waiting for laboratory results [12].

In clinical practice, differential diagnosis begins with organ-specific triads:

- **Mesenteric Ischemia Profile:** Characterized by postprandial abdominal pain, nausea, vomiting, a high risk of thromboembolic events, and elevated LDH levels, strongly guiding clinical suspicion.
- **Renal Artery Thrombosis Triad:** A must-consider in differential diagnosis; an absolute cornerstone featuring a high thromboembolic risk, sudden-onset severe flank pain, and elevated LDH paired with macroscopic/microscopic hematuria.

To determine which patients will benefit from aggressive interventions, modern medicine has developed composite scoring systems and molecular host-response biomarkers (Table 1).

Table 1: Groundbreaking Prognostic Scoring Systems and Biomarkers in Vascular Emergencies.

Biomarker / Score Model	Measured Parameters	Impact on Clinical Strategy and Predictive Power
RADIAL Score	Absence of Rectal Bleeding, Age >65, pH <7.3, Creatinine >1.7 mg/dL, Hypotension	With an AUC of 0.78, it stratifies patients into low (30-40%), moderate (50-60%), and high (80%) mortality risk groups. Supports decisions to avoid “futile” invasive procedures in geriatric cohorts [13].
HALP Score	Hemoglobin, Albumin, Lymphocyte, Platelet (Combined Index)	By quantifying profound systemic inflammation and devastating malnutrition, a low score serves as a strong, independent predictor of mortality in the post-operative course of AMI [14].
LMR	Lymphocyte-to-Monocyte Ratio	Indicates the collapse of the immune response in acute ischemic crises. A low LMR possesses high discriminative power in detecting fatal AMI cases [14].
SeptiCytE RAPID	POC test profiling host mRNA expression (4 genes)	Delivers results in under 60 minutes in an operative setting. Differentiates secondary peritonitis and bacterial translocation from sterile ischemia with a 100% Negative Predictive Value (NPV) at a cut-off <7.4 [15].
Dual-Energy CT (DECT)	Bowel Wall Iodine Concentration (BWIC) Quantification	Spectral reconstructions enhance early ischemia detection and transmural necrosis exclusion, increasing the diagnostic accuracy of standard CTA from 73% to 86% [16].

A Renaissance in Revascularization: “Endovascular-First”, CDT, and Hybrid Approaches

The decades-long debate of “Open Surgery vs. Endovascular?” has largely concluded with the ESVS 2025 guidelines. The guidelines definitively dictate an “endovascular-first” revascularization strategy for AMI patients with suitable anatomy, regardless of age and etiology (Class I) [1]. Moreover, to preserve stent patency in focal lesions <25 mm without sacrificing side branches, the use of covered stents rather than bare-metal stents (BMS) is highly encouraged (Class IIa) [1]. Primary endovascular approaches (aspiration thrombectomy systems), applied on a 24/7 basis by interventional cardiologists utilizing reflexes similar to acute myocardial infarction protocols, reduce laparotomy requirements to 40% levels and report 100% technical success in non-calcified ostial lesions [17].

Catheter-Directed Thrombolysis (CDT) Protocols

In both mesenteric and renal ischemia, slow-infusion thrombolytic therapies represent a revolutionary organ-saving modality in appropriately selected patients. While pigtail catheters are classically employed for proximal infusions, fibrinolytic therapy can also be effectively administered as a continuous infusion via peripheral microcatheters. This microcatheter-directed approach enables highly selective, targeted delivery directly into the thrombus bed.

- a) Mesenteric Artery Thrombolysis: Generally applied in patients presenting within the first 24 hours without peritonitis.

The clinical boundary is peritonitis; once peritonitis develops, medical therapy is terminated, and surgery (laparotomy) is immediately pursued. However, in Stage B and C occlusions where collateralization from the IMA via the Arc of Rioloan is established, this thrombolytic window can extend up to 3 days depending on the clinical scenario.

- Standard Dosage: Following a 5 mg IV bolus of t-PA via the catheter, a continuous infusion at a rate of 1 mg/hour for 24 hours is a widely accepted standard in the literature.

b) Renal Artery Thrombolysis: Due to the narrow 6-hour ischemic tolerance, initiating fibrinolytic therapy within the first 90-180 minutes is strongly recommended; however, individualized assessments based on patient status can stretch this window up to 24 hours.

- Standard Dosage: More aggressive bolus regimens are preferred to dissolve the thrombus burden in the renal bed. An infusion protocol of 1 ml/hour for 24 hours following a 10 mg bolus is applied, and this cycle can be repeated up to 3 times for complete thrombus dissolution depending on clinical need.

c) Alternative Aggressive Regimens: In both organ ischemias, high-dose short-duration protocols such as 25 mg/6 hours or 50 mg/6 hours t-PA via catheter can also be successfully administered depending on the severity of the thrombus burden.

When endovascular access fails due to anatomical constraints, the zenith of modern vascular surgery-“hybrid and autogenous rescue surgical techniques”-comes into play (Table 2).

Table 2: Comparative Clinical Advantages and Operative Risk Profiles of Modern AMI Revascularization Strategies.

Revascularization Strategy	Optimal Patient Profile / Indication	Clinical & Systemic Advantages	Primary Limitations & Operative Risks
Primary Endovascular (Aspiration / Stenting)	Focal embolus or acute in-situ thrombus; hemodynamically stable patient without peritonitis	Minimally invasive. Dramatically reduces hospital and ICU length of stay. Minimizes major resection risk [18].	Wire crossing difficulty in chronic calcific occlusions. Risk of missing masked transmural necrosis in advanced peritonitis [19].
Catheter-Directed Thrombolysis (CDT)	Presenting <24h (up to 3 days in Stage B/C), no peritonitis, low bleeding risk	Dissolves clots without surgical trauma. Peripheral microcatheters allow highly targeted therapy directly into the thrombus bed.	Bleeding complications. Absolutely contraindicated once peritonitis develops , mandating immediate surgery.

ROMS (Retrograde Open Mesenteric Stenting)	Acute-on-chronic MI; ostial stenosis; patient requiring obligatory laparotomy	Combines open bowel viability assessment with endovascular speed. Spares the patient from aortic cross-clamping shock [20].	Requires adequate exposure of the distal SMA for retrograde puncture and a hybrid operating room infrastructure [21].
SMA Transposition	Heavily calcified proximal SMA occlusion; contaminated surgical field due to necrotic bowel	Eliminates prosthetic graft infection risk in contaminated fields. Provides rapid, autogenous revascularization with a single end-to-side anastomosis [22].	Strictly dependent on the presence of a healthy, non-calcified, soft segment of the infrarenal aorta suitable for anastomosis [23].
Traditional Open Surgery (Bypass / Embolectomy)	Advanced widespread bowel necrosis, failure of endovascular or hybrid methods	Ensures direct assessment of bowel viability, excision of necrosis, and simultaneous definitive macroscopic occlusion control [24].	Exceptionally high perioperative mortality, prolonged ICU and mechanical ventilation needs, high risk of repeated "second-look" cycles [25].

Clinical Decision and Management Algorithm

A standardized flowchart is essential to prevent fatal delays in AMI treatment. The presence or absence of peritonitis is the sharpest boundary determining the transition from medical

thrombolysis to surgery. Intraoperative quantification of bowel viability with Indocyanine Green Fluorescence Imaging (ICGFI) (ESVS Class IIb) is currently the most powerful intraoperative tool to prevent unnecessary massive resections and short bowel syndrome based on subjective observations [26] (Figure 1).

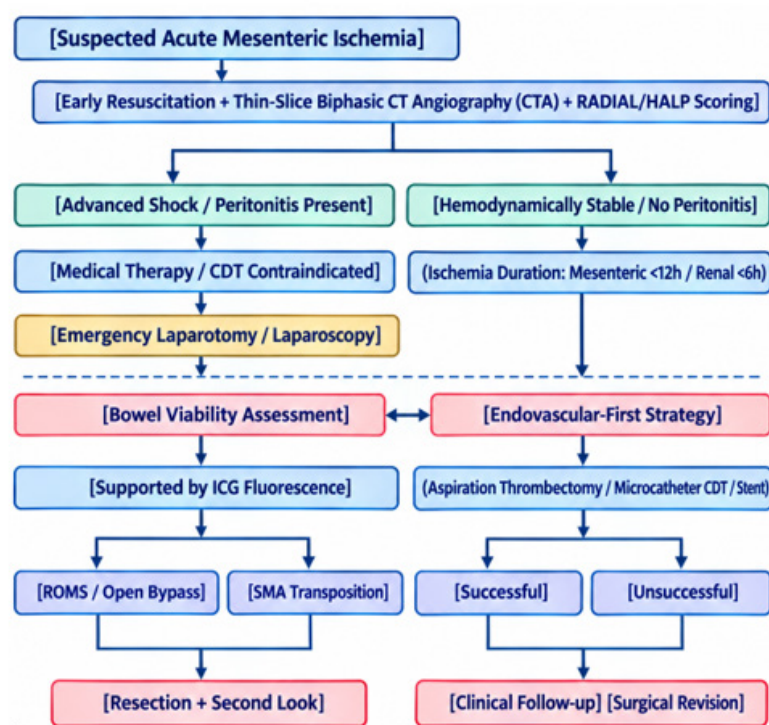


Figure 1: Multidisciplinary Clinical Decision Algorithm for Acute Mesenteric Ischemia.

Beyond Survival: Long-Term Survival, Quality of Life (QOL), and Intestinal Failure

For decades, AMI literature fixated almost exclusively on 30-day in-hospital mortality, cultivating a pessimistic bias that long-term management was a "futile" endeavor. However, the 1-year follow-up

results of the international, multicentre AMESI study have provided illuminating data that entirely overturn this grim narrative [7]. For AMI patients who survive the acute episode and are discharged alive, the 1-year overall survival rate stands at a highly satisfactory 81.3% [7]. Moreover, this survival rate is found to be statistically independent of the ischemic subtype (venous, arterial, or NOMI)

[7]. The singular independent factor determining long-term survival is not the AMI itself, but the patient's age-adjusted Charlson Comorbidity Index (aCCI) at the time of discharge (HR 1.24) [7]. Equally as important as survival is "Quality of Life" (QoL). When evaluated using the EQ-5D-5L scale, 46% of surviving patients reported no problems in any of the dimensions at the end of one year, and the median VAS score reached an impressive 75 [7].

Conversely, the only AMI-specific parameter that statistically and clinically decimates quality of life (RR 1.61) is dependency on parenteral nutrition (PN) at 1 year [7]. Data from the Paris SURVI ("Intestinal Stroke Center") demonstrate that while 60% of discharged patients initially required PN, this rate could be drastically reduced to 18.3% at 1 year through dedicated multidisciplinary rehabilitation and aggressive, bowel-sparing revascularization strategies [6]. The management of Acute Mesenteric Ischemia has emerged from its dark ages, propelled by the recognition of organ-specific ischemic thresholds (mesenteric 12 hours, renal 6 hours), angiographic evaluation of anatomical collateralization stages (Stages A-C), optimization of pigtail and microcatheter-based targeted CDT protocols, and the integration of an "Endovascular-First" paradigm into surgical practice. To prevent irreversible bowel necrosis, it is absolutely vital that the 48-hour "golden window"-before the peritonitis boundary is crossed-is not squandered on unnecessary hospital transfers or diagnostic indecision. Moving forward, the widespread establishment of 24/7 dedicated "Intestinal Stroke Centres" and the routine implementation of integrated algorithms (ROMS, SMA transposition, CDT) utilizing collaborative teams of interventional cardiologists, radiologists, and vascular surgeons will not only maximize acute survival but also guarantee patients a high-quality, long-term life independent of parenteral nutrition.

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