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Emerging Biomarkers for Early Diagnosis of Acute Mesenteric Ischemia: A Systematic Review

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Abstract

Background: Acute mesenteric ischemia (AMI) is a life-threatening condition caused by occlusive or non-occlusive compromise of intestinal blood flow. Early diagnosis is challenging because clinical signs and routine laboratory tests lack sensitivity. Emerging biomarkers (I-FABP, citrulline, D-lactate, GLP-1/2, SM22, adropin, HIF-1α, volatile organic compounds) show promise for earlier detection and disease staging. Methods: A systematic literature search was conducted across PubMed, Embase, and the Cochrane Library from inception through March 2025. Inclusion criteria were: 1) Original studies or meta-analyses evaluating diagnostic accuracy of one or more biomarkers for AMI in human subjects; 2) Reporting of diagnostic performance metrics (sensitivity, specificity, AUC, or 2 × 2 contingency data); 3) English-language publications. Exclusion criteria were animal-only studies, case reports, editorials, and studies evaluating chronic mesenteric ischemia exclusively. Results: I-FABP demonstrated the strongest and most consistent diagnostic performance. Conclusion: No single biomarker currently replaces CTA or clinical judgment. I-FABP is the most validated plasma marker for vascular AMI and intestinal necrosis. Combinatorial panels including I-FABP, SM22, villin-1, and novel markers (GLP-1/2, VOCs) show promise to improve early diagnosis and risk stratification. Prospective multicenter validation and integration with imaging pathways are required before routine clinical implementation.

Keywords

I-FABP, Citrulline, D-Lactate, GLP-1/2, SM22, Adropin, HIF-1α

1. Introduction

Acute mesenteric ischemia (AMI) is caused by occlusive or non-occlusive compromise of the blood supply to the intestine, leading to cellular damage, intestinal necrosis, and death if untreated. The incidence of the condition compared to other surgical conditions is 0.09% - 0.2% [1].

The etiology of AMI involves acute arterial thrombosis due to atherosclerosis or embolism from atrial fibrillation, mesenteric venous thrombosis, and non-occlusive mesenteric ischemia (NOMI) in the setting of low-flow states. Patients typically present with severe abdominal pain out of proportion to physical examination findings, nausea, vomiting, diarrhea, and hematochezia [1].

There are no specific laboratory parameters that enable a reliable diagnosis of AMI [1]. Several biomarkers have been investigated, each reflecting different aspects of ischemic injury pathophysiology:

Citrulline is a non-essential amino acid derived from glutamine that is released from enterocytes. Its plasma level reflects functional enterocyte mass and decreases when enterocyte function is compromised [2]. Intestinal fatty acid-binding protein (I-FABP) is a cytosolic protein released from damaged enterocytes; its level is elevated in intestinal ischemia [3]. D-lactate is a marker of intestinal barrier dysfunction and microbial translocation, produced by intestinal bacteria and leaking into the bloodstream due to mucosal barrier injury and bacterial overgrowth [3].

Glucagon-like peptides (GLP-1 and GLP-2) are produced from proglucagon by L-cells of the intestine in response to dietary fat and glucose. GLP-1 functions to stimulate insulin secretion and produce an anorexic effect. GLP-2 enhances hexose transport through upregulation of sodium-dependent glucose transporter-1 and glucose transporter-2 in the brush border membrane and regulates mucosal physiology [4]. Glicentin is also derived from proglucagon and produced in L-cells; its physiological role remains incompletely characterized [2].

Smooth muscle protein 22-alpha (SM22) is a marker released by intestinal smooth muscles and renally cleared. It is released in patients with damaged intestinal smooth muscle layers [5]. Villin-1 (VIL-1) is a cytoskeletal protein found in the brush border cells of the intestine. Villin-1 leaks into the bloodstream when enterocytes are damaged and serves as a plasma biomarker for mucosal injury [6].

Other markers include serum alpha-glutathione S-transferase (α-GST), cobalt-albumin binding assay (CABA), adropin, and hypoxia-inducible factor 1-alpha (HIF-1α) [1] [7]. Adropin is secreted by vascular endothelium and functions to promote neovascularization and regulate energy homeostasis. HIF-1α is produced by hypoxic cells and is involved in cell adaptation to low oxygen pressure, cell survival, metabolism, proliferation, angiogenesis, vascular tone, and iron metabolism [7].

Exhaled volatile organic compounds (VOCs) analysis is a promising non-invasive diagnostic tool for detection of metabolic changes in AMI, detected using gas chromatography [6].

Computed tomography angiography (CTA) should be performed without delay in patients suspected of having AMI. CTA findings include intestinal dilatation

and wall thickening, reduction or absence of visceral enhancement, pneumatosis intestinalis, and portal venous gas [1]. Mortality rates exceed 50% if a patient is not managed promptly, and mortality doubles every 6 hours if the diagnosis is delayed. Correction of fluids and electrolytes should be initiated, broad-spectrum antibiotics should be started, and emergency laparotomy should be performed after initial resuscitation [1].

This systematic review aims to evaluate the diagnostic accuracy and clinical utility of emerging biomarkers for AMI, synthesize the current evidence, and identify directions for future research.

2. Methods

Eligibility Criteria: Studies were included if they: 1) evaluated the diagnostic accuracy of one or more biomarkers for AMI in human subjects; 2) reported diagnostic performance metrics including sensitivity, specificity, or AUC; 3) used a clearly defined reference standard (CTA, angiography, surgical findings, or histopathology); 4) were published in English. Exclusion criteria were animal-only studies, case reports, editorials, studies evaluating chronic mesenteric ischemia exclusively, and studies with fewer than 10 patients. Quality assessment of included studies was performed using the QUADAS-3 tool [8].

Study Selection and Data Extraction: Two reviewers independently screened titles and abstracts, followed by full-text review of potentially eligible studies. Disagreements were resolved by consensus. Data extraction was performed using a standardized form and included: study design, sample size, patient population, biomarker(s) evaluated, assay method, reference standard, diagnostic thresholds, sensitivity, specificity, AUC, positive and negative predictive values, and key findings.

Data Synthesis: Due to substantial heterogeneity in biomarker types, assay methods, cut-off values, patient populations, and AMI etiologies across studies, a narrative synthesis was performed rather than a pooled meta-analysis. Biomarkers were grouped by pathophysiologic mechanism, and diagnostic performance was compared across studies.

Reporting: This review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses of Diagnostic Test Accuracy Studies (PRISMA-DTA) guidelines [9] (**Table 1** & **Table 2**).

PRISMA 2020 flow diagram for new systematic reviews which included searches of databases and registers (**Figure 1**).

Table 1. Biomarkers and study results.

Biomarker	Source/Mechanism	Marker Type	Diagnostic Performance
GLP-1	Proglucagon-derived; L-cells	Mucosal injury	5.3 vs. 2.3 pmol/L (p = 0.01)
GLP-2	Proglucagon-derived; L-cells	Mucosal injury	2.8 vs. 0.9 pmol/L (p = 0.023)
I-FABP	Enterocyte cytoplasm	Mucosal damage	AUC 0.80 - 0.88 (vascular ischemia); 0.97 (post-cardiac surgery)

Continued

Villin-1	Enterocyte brush border	Mucosal damage	Under investigation (TACTIC study)
Citrulline	Enterocyte synthesis from glutamine	Enterocyte mass	AUC 0.68; 15.3 vs. 23.3 μmol/L (p = 0.001)
D-lactate	Bacterial fermentation in ischemic gut	Bacterial translocation	AUC 0.40
Adropin	Vascular endothelium	Hypoxia marker	AUC 0.69 (weak)
HIF-1*α*	Cellular hypoxia response	Hypoxia marker	AUC 0.71 (moderate)
VOCs	Exhaled breath metabolites	Metabolic signature	Under investigation (TACTIC study)

Table 2. Summary of included studies.

Study	Design	N	Biomarkers Studied	Primary Endpoint	Status
TACTIC Study (NCT05194527)	Multicenter prospective observational	120	I-FABP, villin-1, SM22, VOCs	Diagnosis of AMI stages	Recruiting
DIAGOMI Study	Multicenter prospective observational	61	Citrulline, I-FABP	Diagnosis of NOMI and intestinal necrosis	Completed 2022
El Hamwi *et al.* [4]	Single-center prospective	46	GLP-1, GLP-2, glicentin, I-FABP, citrulline	Differentiate AMI from controls	Completed 2025
Taghiyev *et al.* [14]	Prospective observational (post-cardiac surgery)	500 (48 high-risk)	I-FABP	Early detection of mesenteric ischemia	Completed 2025
Nuzzo *et al.* [3]	Cross-sectional diagnostic	129	Citrulline, I-FABP, D-lactate	Diagnosis of AMI	Completed 2021
Matsumoto *et al.* [13]	Prospective observational	208	I-FABP and traditional biomarkers	Diagnosis of vascular vs. non-vascular ischemia	Published
Kulu *et al.* [10]	Prospective observational	48	Citrulline, D-dimer	Diagnosis and prognosis of AMI	Published
Kurt *et al.* [7]	Prospective observational	60	Adropin, HIF-1*α*, apelin	Early detection of AMI	Published

3. Discussion

3.1. Citrulline as a Biomarker

Nuzzo *et al.* conducted a cross-sectional study to evaluate the accuracy of citrulline, I-FABP, and D-lactate in the diagnosis of AMI. The study showed lower median citrulline concentrations in AMI patients compared to controls (15.3 μmol/L vs. 23.3 μmol/L, p = 0.001) [3]. Kulu *et al.* conducted a study with 48 patients to evaluate the diagnostic and prognostic values of citrulline with those of D-dimer in patients with AMI. The study showed significantly higher citrulline levels in 23 patients with abdominal pain with AMI versus 25 patients with abdominal pain without AMI. Citrulline was also statistically significant for predicting mortality [10].

The apparent directional discrepancy between these two studies—lower citrulline in AMI [3] versus higher citrulline in AMI [10]—warrants explanation. Citrulline is synthesized by enterocytes, and its plasma level reflects functional

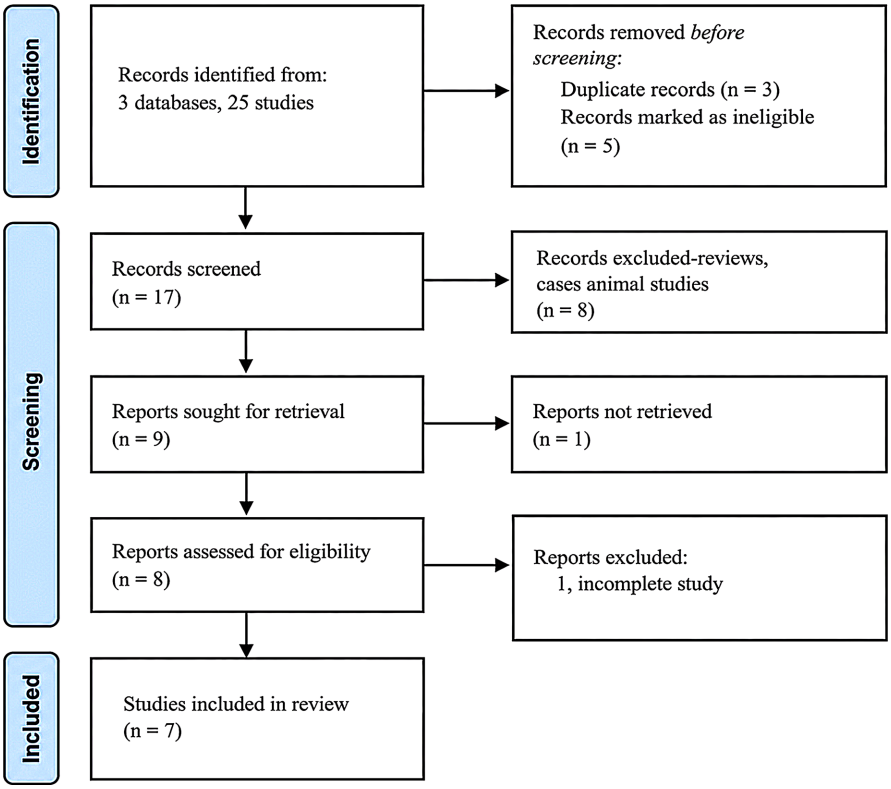

Figure 1. Identification of studies via databases and registers.

enterocyte mass; therefore, a decrease is the expected pathophysiological response to enterocyte loss in AMI, consistent with the findings of Nuzzo *et al.* and the broader literature [2]. Overall, citrulline shows modest standalone diagnostic utility for AMI.

3.2. D-Lactate as a Biomarker

Nuzzo *et al.* showed that plasma D-lactate concentrations were not statistically different between AMI and controls (0.042 mmol/L vs. 0.073 mmol/L, p = 0.28). ROC analysis showed an AUC of 0.438 (0.328 - 0.547) [3]. Thus, D-lactate fails to differentiate AMI from other causes of abdominal pain. Costello *et al.* conducted a scoping review to evaluate current diagnostic and management strategies, comparing endovascular and open surgical approaches. After analysis of biomarkers from 22 studies, lactate levels were substantially higher in open surgical patients (3.0 - 4.8 mmol/L) versus endovascular cases (1.6 - 2.2 mmol/L) [11].

3.3. I-FABP as a Biomarker

The cross-sectional study by Nuzzo *et al.* (2021) showed that plasma I-FABP concentrations had no statistically significant difference between AMI and other causes of abdominal pain (278 ng/L vs. 348 ng/L, p = 0.06). ROC analysis showed an AUC of 0.401 (0.291 - 0.512) [3]. I-FABP has a short plasma half-life (approximately 11 minutes), and levels may normalize if sampling occurs after the initial release phase or if ischemia is limited to the mucosa without significant enterocyte

necrosis. Sun *et al.* (2016) conducted a meta-analysis to evaluate the accuracy of serum I-FABP for diagnosis of AMI. The study showed pooled sensitivity of 0.80 (95% CI: 0.72 - 0.86), specificity of 0.85 (95% CI: 0.73 - 0.93), positive likelihood ratio of 5.5 (95% CI: 2.8 - 10.8), and negative likelihood ratio of 0.23 (95% CI: 0.15 - 0.35). The diagnostic odds ratio was 24 (95% CI: 9 - 65) and the AUC was 0.86 (95% CI: 0.83 - 0.89, p = 0.042), indicating high overall accuracy [12].

Matsumoto *et al.* conducted a study to evaluate the use of I-FABP and traditional biomarkers in the early diagnosis of acute intestinal ischemia of different causes. The study showed that levels of most biomarkers (including I-FABP) were significantly higher in the vascular ischemia group versus other groups ($p < 0.01$). The ROC curve showed that I-FABP had an AUC of 0.80 - 0.88 [13].

Taghiyev *et al.* conducted a prospective observational study of 500 patients undergoing cardiac surgery with cardiopulmonary bypass. ROC analysis showed an AUC of 0.973 at 36 hours post-ICU admission (95% CI: 0.764 - 0.997, $p < 0.001$) and an optimal cut-off of >1421 pg/mL. Thus, I-FABP is a highly accurate and early predictor of mesenteric ischemia after cardiac surgery [14]. Of note, this study evaluated a specific clinical context (NOMI after cardiopulmonary bypass), and results may not be directly generalizable to other AMI etiologies.

Bourcier *et al.* conducted the multicentric prospective observational DIAGOMI study on diagnosis and prognosis features in 61 patients with suspected mesenteric ischemia. Of the 33 patients with confirmed NOMI, 27 had intestinal necrosis. Plasma I-FABP was significantly increased in the presence of intestinal necrosis, with an AUC of 0.83 (0.70 - 0.96). At a threshold of 3114 pg/mL, sensitivity was 70%, specificity 85%, negative predictive value 58%, and positive predictive value 90% for the diagnosis of intestinal necrosis [15].

Taken together, these four studies demonstrate that I-FABP is the best-validated biomarker for diagnosing vascular intestinal ischemia and detecting intestinal necrosis, though its sensitivity may be limited in early or non-vascular forms of ischemia.

3.4. GLP-1 and GLP-2 as Biomarkers

El Hamwi *et al.* conducted a prospective study to assess whether circulating levels of proglucagon-derived peptides differ between patients with AMI and a control group. Twenty-three patients in the ischemia group and 23 in the control group were analyzed. Both GLP-1 and GLP-2 levels were higher in the ischemia group versus the control group: GLP-1 (5.3 vs. 2.3 pmol/L, p = 0.01) and GLP-2 (2.8 vs. 0.9 pmol/L, p = 0.023), respectively [4]. These findings are promising but limited by the small sample size (n = 46) and single-center design. ROC-derived diagnostic accuracy metrics (AUC, sensitivity, specificity) were not reported, precluding direct comparison with other biomarkers.

3.5. SM22 and Villin-1 as Biomarkers

Duivenvoorden *et al.* described the multicentre prospective observational TAC-

TIC study to evaluate a panel of biomarkers and volatile organic compound profiles in exhaled air to diagnose AMI. One hundred twenty patients are being recruited and recruitment is ongoing. I-FABP, villin-1, and SM22 plasma levels over time increase as mesenteric ischemic damage increases. Thus, these markers may be used to determine the severity of AMI [6].

3.6. Adropin and HIF-1α as Biomarkers

Kurt *et al.* studied whether adropin and HIF-1α may be used to aid in the early detection of AMI. The study showed higher levels of adropin and HIF-1α in 20 patients with AMI compared to both the abdominal pain group (n = 20) and the healthy control group (n = 20). ROC analysis showed that HIF-1α had an AUC of 0.705 (moderate diagnostic accuracy) while adropin had an AUC of 0.692 (weak accuracy) for AMI [7]. These results are limited by the very small sample size (n = 60 total), single-center design, and the inclusion of healthy controls in the comparator group, which may inflate diagnostic performance estimates compared to the clinically relevant comparison of AMI versus other causes of acute abdominal pain. The pathophysiological role of HIF-1α in intestinal ischemia-reperfusion injury has been further characterized in preclinical models [16].

3.7. Time-Dependent Biomarker Kinetics

The clinical utility of each biomarker is influenced by its temporal release pattern relative to ischemia onset. I-FABP, as a small cytosolic protein (15 kDa), is rapidly released upon enterocyte membrane disruption but has a short plasma half-life of approximately 11 minutes, meaning levels may peak early and decline if sampling is delayed. In the post-cardiac surgery setting, I-FABP levels peaked between ICU admission and 36 hours [14]. SM22 becomes elevated from 4 hours of ischemia onward, reflecting the later progression from mucosal to transmural damage [5]. HIF-1α increases significantly at 3 - 6 hours after ischemia onset. These kinetic differences suggest that a staged biomarker approach, early I-FABP for mucosal injury detection, followed by SM22 for transmural progression, may optimize diagnostic sensitivity across the time course of AMI. Prior systematic reviews and meta-analyses have similarly highlighted the complementary diagnostic value of combining multiple serological biomarkers [17].

4. Limitations

This review has several limitations. First, the included studies are heterogeneous in design, patient populations, AMI definitions, reference standards, and assay methods, precluding formal meta-analysis across biomarker categories. Second, sample sizes are generally small (range: 40 - 500 for individual biomarkers), limiting statistical power and generalizability. Third, most studies are single center, introducing potential selection bias. Fourth, standardized cut-off values have not been established for most biomarkers, and assay variability across laboratories may affect reproducibility. Fifth, the temporal relationship between biomarker

sampling and ischemia onset is inconsistently reported, which is critical given the time-dependent kinetics of biomarker release. Sixth, several promising biomarkers (villin-1, VOCs) lack published diagnostic accuracy data, and their evaluation relies on ongoing studies. Seventh, diagnostic performance metrics are reported inconsistently across studies; some report sensitivity/specificity, others only AUC, and some report only mean differences, limiting direct comparability. Finally, publication bias cannot be excluded, as negative results are less likely to be published.

5. Conclusion

No single biomarker currently replaces CTA or clinical judgment. I-FABP is the most validated plasma marker for vascular AMI and intestinal necrosis. Combinatorial panels including I-FABP, SM22, villin-1, and novel markers (GLP-1/2, VOCs) show promise to improve early diagnosis and risk stratification. Prospective multicenter validation and integration with imaging pathways are required before routine clinical implementation.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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