

Vasopressor-Associated Non-Occlusive Mesenteric Ischemia in Critically Ill Adults: Mechanisms, Recognition, and Management

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ABSTRACT

Non-occlusive mesenteric ischemia is an acute failure of intestinal perfusion that arises without obstruction of the mesenteric vessels, and in the intensive care unit it has become the most common form of acute mesenteric ischemia. It develops almost exclusively in patients with circulatory shock who are receiving vasopressor support, and its mortality remains high largely because the diagnosis is made late. This narrative review examines how vasopressor therapy and the shock state interact to injure the gut, why the condition is so often missed in critically ill adults, and how it can be prevented and treated more effectively. The review shows that the intestine is unusually vulnerable in shock because its circulation is among the first to be sacrificed when blood flow falls, and this protective reflex can persist even after blood pressure has been restored. Vasopressors are essential for maintaining perfusion pressure, but when they are escalated in a patient whose volume status or cardiac output has not been corrected, they can deepen intestinal vasoconstriction and turn a reversible reduction in flow into bowel infarction. The risk therefore reflects the way these drugs are used as much as the choice of drug itself. Recognition is difficult because sedation and mechanical ventilation mask the abdominal pain and tenderness on which diagnosis traditionally depends, so clinicians must instead respond to indirect signals of deterioration, particularly a rise in lactate or vasopressor requirement that cannot otherwise be explained. Early contrast-enhanced computed tomography is the key diagnostic step, and catheter angiography adds the ability to deliver vasodilator therapy directly into the mesenteric circulation. Effective management depends on restoring blood flow rather than pressure alone, limiting vasoconstrictor exposure, resting and protecting the gut, and proceeding to surgery with planned reassessment when bowel necrosis is suspected. The review also finds that outcomes depend on wider system factors, including reliable access to imaging and interventional services, a secure supply of critical medicines, and coordinated multidisciplinary pathways.

The review concludes that vasopressor-associated non-occlusive mesenteric ischemia is largely preventable and treatable when it is anticipated. Careful vasopressor stewardship, a high index of suspicion, a low threshold for imaging and prompt multidisciplinary action offers the best opportunity to reduce its mortality, while prospective research on risk prediction, diagnostic biomarkers and catheter-directed therapy is needed to strengthen practice.

Keywords: non-occlusive mesenteric ischemia; vasopressors; norepinephrine; vasopressin; septic shock; cardiogenic shock; splanchnic circulation; intensive care; computed tomography angiography; papaverine

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INTRODUCTION

Acute mesenteric ischemia (AMI) is an uncommon but devastating cause of abdominal catastrophe, with mortality that has remained between 50 and 80 percent in many series despite advances in imaging and intervention (Clair & Beach, 2016; Kärkkäinen & Acosta, 2017; Schoots et al., 2004). Four mechanisms are classically described: arterial embolism, arterial thrombosis, mesenteric venous thrombosis

and non-occlusive mesenteric ischemia (NOMI). NOMI is defined by inadequate intestinal blood flow resulting from vasoconstriction of the mesenteric circulation, typically in the setting of reduced cardiac output or systemic hypotension, with patent major vessels (Björck et al., 2017; Trompeter et al., 2002). Although it accounts for a minority of AMI in emergency departments, it predominates among patients who develop intestinal ischemia while already in the ICU (Bourcier et al., 2016; Bourcier et al., 2022; Leone et al., 2015).

Critically ill patients with NOMI share a common pathway: shock, compensatory splanchnic vasoconstriction and, in almost all cases, exogenous vasopressor therapy. Vasopressors are essential to restore perfusion pressure and are recommended as first-line therapy for vasodilatory shock that persists after initial fluid resuscitation (Evans et al., 2021). At the same time, their effects on regional blood flow are heterogeneous, and the gut is among the organs most vulnerable to an imbalance between pressure and flow (Reilly & Bulkley, 1993; Woolsey & Coopersmith, 2006). The term vasopressor-associated NOMI is used in this review to describe NOMI developing during vasoactive therapy, recognizing that causality is rarely provable in an individual patient and that the shock state itself is a necessary contributor.

The purpose of this narrative review is to synthesize the physiological, pharmacological and clinical evidence regarding vasopressor-associated NOMI in critically ill adults, with emphasis on mechanisms, early recognition and practical management. Previous reviews have generally addressed acute mesenteric ischemia as a whole, or NOMI from a surgical or radiological perspective; the present review instead centers on the intensivist, integrating vasopressor pharmacology, bedside recognition, management and the system-level factors that determine whether the diagnosis is made in time. Particular attention is given to the decisions intensivists make every day, such as choice and dose of vasopressor, blood pressure targets, timing of enteral nutrition and thresholds for imaging, that may modify risk.

RESEARCH METHOD

This is a narrative, non-systematic review. Relevant literature was identified through searches of MEDLINE (PubMed), Embase and the Cochrane Library for articles published in English up to December 2022, using combinations of the terms non-occlusive mesenteric ischemia, nonocclusive mesenteric ischemia, NOMI, acute mesenteric ischemia, intestinal ischemia, splanchnic perfusion, vasopressor, norepinephrine, epinephrine, vasopressin, angiotensin II, dopamine, phenylephrine, septic shock, cardiogenic shock, cardiac surgery and intensive care. Reference lists of major guidelines and reviews were hand-searched. Priority was given to clinical practice guidelines, randomized trials of vasoactive agents reporting gastrointestinal outcomes, multicenter cohort studies of ICU mesenteric ischemia, and mechanistic studies of splanchnic perfusion in humans and large-animal models. Because the evidence base consists predominantly of observational studies and expert opinion, no formal grading or quantitative synthesis was attempted; the strength of the underlying evidence is indicated in the text where relevant. To place clinical findings within the wider systems in which ICU care is delivered, the search was supplemented by a curated collection of related and adjacent literature published up to 2022 on health system infrastructure, laboratory and diagnostic capacity, health informatics and predictive analytics, safety

science in high-risk industries, and pharmaceutical supply and quality; this material is synthesized in the discussion of health system and implementation considerations.

RESULTS AND DISCUSSION

The literature retained for this review comprised international clinical practice guidelines, randomized controlled trials and meta-analyses of vasoactive agents and nutrition, multicenter and population-based cohort studies of mesenteric ischemia, mechanistic studies in humans and large-animal models, and related health systems and implementation literature. The findings are presented and discussed thematically, moving from definitions and epidemiology through pathophysiology, risk factors, recognition and management to special populations, system-level considerations, and the limitations of the current evidence.

Definitions, Epidemiology and Outcomes

NOMI is characterized angiographically by narrowing at the origins of the branches of the superior mesenteric artery (SMA), alternating dilatation and narrowing of intestinal branches (the so-called string of sausages sign), spasm of the mesenteric arcades and impaired filling of intramural vessels (Siegelman et al., 1974). The clinical spectrum ranges from reversible mucosal ischemia to transmural infarction with perforation and peritonitis. NOMI may affect the small bowel, the colon, or both; isolated ischemic colitis in the ICU frequently shares the same non-occlusive mechanism (Brandt et al., 2015). The European Society of Vascular Surgery (ESVS) and the World Society of Emergency Surgery (WSES) treat NOMI as a distinct entity whose management centers on treatment of the precipitating condition and restoration of flow rather than revascularization (Bala et al., 2022; Björck et al., 2017).

Population-level estimates suggest that NOMI represents roughly 20 to 30 percent of all AMI (Clair & Beach, 2016; Kärkkäinen & Acosta, 2017; Trompeter et al., 2002). In a Swedish population-based autopsy study, fatal NOMI occurred at about 2 per 100,000 person-years, rising steeply after the age of 80, with heart failure, atrial fibrillation and recent surgery as independent risk factors (Acosta et al., 2006). A Finnish hospital-based study found AMI to be a more common cause of acute abdomen than appendicitis in patients older than 75 years (Kärkkäinen et al., 2015). In ICU cohorts the proportion is considerably higher. In a French multicenter retrospective study of 780 ICU patients with AMI, a large share of cases were non-occlusive and most patients were receiving vasopressors at the time of diagnosis (Leone et al., 2015). The prospective multicenter DIAGOMI study, published in 2022, confirmed that non-occlusive mechanisms account for the majority of mesenteric ischemia diagnosed in ICU patients and highlighted the frequency of delayed diagnosis (Bourcier et al., 2022). After cardiac surgery, NOMI is reported in under 1 to approximately 2 percent of patients, with substantially higher rates among those with prolonged cardiopulmonary bypass,

low cardiac output syndrome or mechanical circulatory support (Chaudhuri et al., 2006; Ghosh et al., 2002; Groesdonk et al., 2013; Guillaume et al., 2017; Lim et al., 2017; Sakamoto et al., 2020). In a registry of nearly 19,000 cardiac operations, intestinal ischemia was rare but carried a mortality of 59 percent (Nilsson et al., 2013), and in an earlier series mesenteric ischemia accounted for about one in nine postoperative deaths, predominantly through non-occlusive, low-output mechanisms (Venkateswaran et al., 2002). Among patients receiving veno-arterial extracorporeal membrane oxygenation for refractory shock, mesenteric ischemia developed in approximately 9 percent and was uniformly fatal in one series (Renaudier et al., 2021). In a post hoc analysis of NUTRIREA-2, AMI, mostly non-occlusive, occurred in 1 percent of ventilated patients with shock (Piton et al., 2022). Because confirmation requires imaging, endoscopy, surgery or autopsy, and because many patients die of multiorgan failure without definitive investigation, all of these figures are likely to underestimate the true incidence (Bourcier et al., 2021; Stahl et al., 2020a).

In a systematic review of survival after AMI by etiology, mortality associated with NOMI was approximately 70 percent, comparable with arterial thrombosis and higher than mesenteric venous thrombosis (Schoots et al., 2004). In ICU series, mortality between 50 and 80 percent is typical, and outcome is determined by the severity of the underlying shock, the extent of bowel necrosis, the delay to diagnosis and the ability to restore flow (Bourcier et al., 2022; Leone et al., 2015; Stahl et al., 2020a). Factors associated with transmural necrosis include organ failure, elevated lactate and bowel wall changes on CT such as reduced enhancement or dilatation (Nuzzo et al., 2017). A meta-analysis of observational studies confirmed persistently high in-hospital mortality, greater for arterial and non-occlusive forms than for venous thrombosis (Adaba et al., 2015). Survivors of extensive resection may develop short bowel syndrome and chronic intestinal failure requiring long-term parenteral support and intestinal rehabilitation (Corcos et al., 2013; Pironi et al., 2016). Post-cardiac surgery NOMI carries particularly high mortality, frequently above 50 percent (Groesdonk et al., 2013; Klotz et al., 2001; Sakamoto et al., 2020).

Pathophysiology

Physiology of the splanchnic circulation

The splanchnic circulation receives roughly one quarter of cardiac output at rest and contains a substantial fraction of total blood volume, much of it in the compliant venous capacitance system (Takala, 1996). Within the intestinal wall, most of the flow is distributed to the mucosa and submucosa, where metabolic demand is highest. The villus microvasculature is organized as a countercurrent exchanger in which oxygen diffuses from the arteriole to the adjacent venule at the villus

base. As a result, the villus tip normally operates at a low oxygen tension and is the first structure to become hypoxic when flow falls (Haglund & Bergqvist, 1999; Hasibeder, 2010).

Under physiological conditions the intestine maintains oxygen uptake over a wide range of flows by increasing oxygen extraction and recruiting capillaries. Below a critical delivery threshold, however, oxygen consumption becomes supply-dependent and mucosal injury begins within minutes to hours (Haglund & Bergqvist, 1999; Takala, 1996). The mesenteric vascular bed is richly innervated by sympathetic fibers and expresses abundant alpha-1 adrenergic, angiotensin type 1 and vasopressin V1a receptors, making it one of the most reactive vascular territories in the body (Holmes et al., 2001; Reilly & Bulkley, 1993). Splanchnic organs also differ in their vulnerability: during experimental hemorrhage, jejunal mucosal flow was relatively preserved by autoregulation whereas pancreatic flow fell sharply (Krejci et al., 2000), and the hepatic arterial buffer response that normally protects the liver fails in endotoxemia and low-flow states (Jakob, 2002).

The endogenous response to shock

In hypovolemic and cardiogenic shock, the body redistributes blood away from the splanchnic bed to preserve cerebral and coronary perfusion. Splanchnic vasoconstriction mobilizes blood into the central circulation, and arteriolar constriction reduces mesenteric arterial inflow disproportionately to the fall in cardiac output (Reilly & Bulkley, 1993; Takala, 1996). Classic experimental work in porcine cardiogenic shock demonstrated that this selective mesenteric vasoconstriction is mediated predominantly by the renin-angiotensin axis and could be attenuated by angiotensin blockade (Bailey et al., 1987). Circulating vasopressin and catecholamines contribute further (Holmes et al., 2001; Reilly & Bulkley, 1993). Critically, mesenteric vasoconstriction can persist after systemic hemodynamics are restored, so that the gut may remain hypoperfused even when blood pressure and cardiac output appear adequate (Bourcier et al., 2021; Trompeter et al., 2002).

In septic shock the picture is more complex. Total splanchnic blood flow is often normal or increased, yet mucosal perfusion may be heterogeneous with microcirculatory shunting, capillary dropout, endothelial dysfunction, microthrombosis and increased metabolic demand of the hepatosplanchnic region (Hasibeder, 2010; Takala, 1996). The gut therefore experiences an oxygen supply and demand mismatch even without a fall in global flow, and the addition of intestinal edema and raised intra-abdominal pressure further compromises perfusion (Kirkpatrick et al., 2013; Reintam Blaser et al., 2020).

How vasopressors interact with the shock state

Vasopressors increase perfusion pressure, which may improve mesenteric flow if the bed is pressure-passive and hypotension was the limiting factor. Conversely, if vasoconstriction increases mesenteric vascular resistance proportionally more

Table 1: Vasoactive agents and their reported effects on the splanchnic circulation

<i>Agent</i>	<i>Main receptors</i>	<i>Splanchnic effects (human and experimental data)</i>	<i>Clinical implications</i>
Norepinephrine	Alpha-1 > beta-1	At moderate doses with adequate volume status, splanchnic flow and gastric mucosal perfusion are generally preserved; experimental high doses reduce mucosal flow (De Backer et al., 2003; Krejci et al., 2006)	First-line in septic shock (Evans et al., 2021); risk rises with high dose, hypovolemia and low cardiac output
Epinephrine	Beta-1, beta-2, alpha-1	Reduced splanchnic fractional flow, increased gastric mucosal-arterial PCO ₂ gap and lactate compared with norepinephrine or norepinephrine plus dobutamine (De Backer et al., 2003; Levy et al., 1997; Meier-Hellmann et al., 1997)	No mortality difference in trials (Annane et al., 2007; Myburgh et al., 2008); prefer as second-line; monitor lactate interpretation carefully
Dopamine	Dopaminergic, beta-1, alpha-1 (dose dependent)	Increased total splanchnic flow in some studies but heterogeneous mucosal effects (De Backer et al., 2003)	More arrhythmias than norepinephrine; not recommended as first-line (De Backer et al., 2010; Evans et al., 2021)
Phenylephrine	Pure alpha-1	Experimental reductions in mucosal microcirculatory flow in sepsis (Krejci et al., 2006)	Limited role; caution in low cardiac output states
Vasopressin	V1a (vascular), V2	Potent mesenteric vasoconstrictor; dose-dependent reductions in gastric mucosal perfusion reported (Holmes et al., 2001; van Haren et al., 2003); ischemic skin lesions at higher doses (Dünser et al., 2003)	Low-dose adjunct reduces norepinephrine requirement; trials and meta-analysis show more digital ischemia without a clear excess of mesenteric ischemia (Gordon et al., 2016; Nagendran et al., 2019; Russell et al., 2008)
Angiotensin II	AT1	Mediates endogenous splanchnic vasoconstriction in shock (Bailey et al., 1987)	Raises MAP in refractory vasodilatory shock (Khanna et al., 2017); limited gut-specific data; thromboembolic events reported, prophylaxis advised
Dobutamine	Beta-1, beta-2	May improve splanchnic flow when cardiac output is low (Annane et al., 2007; Levy et al., 1997)	Consider when low cardiac output contributes to hypoperfusion

MAP, mean arterial pressure; PCO₂, partial pressure of carbon dioxide.

than it increases driving pressure, flow falls. The net effect depends on at least four variables: the receptor profile of the drug, the dose, the patient's intravascular volume and cardiac output, and the integrity of local autoregulation (Krejci et al., 2006; Woolsey & Coopersmith, 2006). Vasopressor exposure is therefore best understood as an amplifier of an already vulnerable state rather than as an isolated cause of NOMI (Table 1). The highest-risk situation is the patient with uncorrected hypovolemia or low cardiac output in whom a vasoconstrictor is titrated upward to meet a pressure target, producing an acceptable mean arterial pressure at the cost of a falling splanchnic flow (Groesdonk et al., 2013; Trompeter et al., 2002).

Norepinephrine

Norepinephrine is the recommended first-line vasopressor in septic shock (Evans et al., 2021). In patients with septic shock, its effects on hepatosplanchnic flow are usually neutral or favorable when cardiac output is maintained, and it compares favorably with epinephrine on markers of gut

perfusion (De Backer et al., 2003; Meier-Hellmann et al., 1997). In experimental endotoxic shock, norepinephrine preserved mucosal adenosine triphosphate content even though it blunted the rise in mucosal flow produced by fluids alone (Revelly et al., 2000). However, raising arterial pressure above about 65 mmHg with vasopressors does not reliably improve microcirculatory or splanchnic perfusion (Boerma & Ince, 2010). Nevertheless, norepinephrine is the vasopressor most commonly being administered when NOMI is diagnosed in ICU cohorts, and high or rising doses are repeatedly identified as risk markers (Groesdonk et al., 2013; Leone et al., 2015; Lim et al., 2017). After cardiovascular surgery, norepinephrine doses above 0.10 micrograms per kilogram per minute and mechanical ventilation beyond 24 hours independently predicted NOMI (Sato et al., 2018). Systematic reviews and a Cochrane review of vasopressor trials found no mortality difference between agents and too few data to compare them on gut ischemia, which was reported inconsistently (Avni et al., 2015; Gamper et al., 2016). This association reflects both

the drug and the severity of shock for which high doses are needed, and the two cannot be separated in observational data.

Epinephrine. In septic shock, epinephrine reduced splanchnic blood flow relative to cardiac output and widened the gastric mucosal-arterial PCO₂ gap compared with norepinephrine with dobutamine (Levy et al., 1997; Meier-Hellmann et al., 1997). In patients with severe shock, epinephrine was associated with lower splanchnic fractional flow than norepinephrine (De Backer et al., 2003). Its beta-2 mediated aerobic glycolysis also raises lactate, which can obscure the lactate signal that might otherwise alert clinicians to gut ischemia (Levy et al., 1997). Large randomized trials did not show differences in mortality between epinephrine and norepinephrine-based regimens, but they were not designed to detect mesenteric complications (Annane et al., 2007; Myburgh et al., 2008).

Vasopressin and its analogues

Vasopressin acts on V1a receptors that are densely expressed in the splanchnic vasculature, and it is used therapeutically to reduce portal flow in variceal hemorrhage (Holmes et al., 2001). In catecholamine-dependent septic shock, vasopressin infusion was associated with deterioration in gastric mucosal perfusion at higher doses (van Haren et al., 2003), and ischemic skin lesions were frequent in early series using doses above current recommendations (Dünser et al., 2003). In septic pigs, vasopressin raised arterial pressure but more than halved superior mesenteric flow and reduced gastric and jejunal mucosal microcirculatory flow (Hiltebrand et al., 2007), and low-dose vasopressin roughly halved portal venous flow (Krejci et al., 2007). In the VASST and VANISH trials, low-dose vasopressin added to or replacing norepinephrine did not significantly increase serious mesenteric ischemia (Gordon et al., 2016; Russell et al., 2008). An individual patient data meta-analysis found more digital ischemia with vasopressin, while differences in mesenteric ischemia were not statistically robust (Nagendran et al., 2019). Earlier meta-analyses found that vasopressin or terlipressin spared catecholamines without an overall excess of serious adverse events, although ischemic complications were inconsistently reported and bolus terlipressin may cause excessive splanchnic vasoconstriction (Belletti et al., 2015; Polito et al., 2012; Serpa Neto et al., 2012). In a randomized trial, continuous terlipressin did not reduce mortality compared with norepinephrine and caused more serious adverse events, mainly digital ischemia (Liu et al., 2018). The selective V1a agonist selepressin performed well in ovine septic shock (He et al., 2016), but isolated ischemic adverse reactions were reported in a phase IIa trial and it did not improve ventilator- and vasopressor-free days in the SEPSIS-ACT trial (Laterre et al., 2019; Russell et al., 2017). Current guidance supports adding vasopressin at low fixed

doses when norepinephrine requirements are moderate to high (Evans et al., 2021); clinicians should nevertheless monitor the gut closely, avoid escalation beyond standard doses, and be particularly cautious with terlipressin, whose longer duration of action makes its effects harder to reverse.

Angiotensin II and other agents

Synthetic angiotensin II increased mean arterial pressure in refractory vasodilatory shock in the ATHOS-3 trial (Khanna et al., 2017). Given the central role of angiotensin in endogenous mesenteric vasoconstriction (Bailey et al., 1987), theoretical concern exists for splanchnic hypoperfusion, although in porcine fecal peritonitis angiotensin II restored arterial pressure with regional perfusion comparable to norepinephrine (Corrêa et al., 2014). Gut-specific clinical outcome data are limited, and the drug should be used within its studied indications with attention to thromboprophylaxis. Dopamine increased arrhythmias without improving survival compared with norepinephrine (De Backer et al., 2010), and phenylephrine reduced gastrointestinal microcirculatory flow in experimental sepsis (Krejci et al., 2006), although in a small randomized trial first-line phenylephrine did not worsen hepatosplanchnic perfusion markers compared with norepinephrine (Morelli et al., 2008). Non-vasopressor drugs that cause mesenteric vasoconstriction, including digoxin, ergot alkaloids, cocaine and some serotonergic agents, may add to vasopressor effects and should be reviewed in at-risk patients (Clair & Beach, 2016; Trompeter et al., 2002).

From mucosal hypoxia to transmural infarction

Mucosal hypoxia leads to epithelial cell death, loss of tight junction integrity and increased permeability within hours. Restoration of flow generates reactive oxygen species, neutrophil recruitment and complement activation, so that reperfusion injury may extend damage beyond that caused by ischemia alone (Haglund & Bergqvist, 1999; Schoenberg & Beger, 1993). Human experimental models show that the epithelium can shed damaged cells and rapidly restore barrier integrity after brief ischemia, whereas longer ischemia leads to barrier loss and inflammation (Grootjans et al., 2016). Loss of the mucosal barrier permits translocation of bacteria, endotoxin and gut-derived mediators into the mesenteric lymph and portal circulation, propagating systemic inflammation and distant organ failure (Clark & Coopersmith, 2007). This concept of the gut as a motor of multiorgan dysfunction explains why NOMI frequently presents not as an abdominal emergency but as unexplained deterioration in a patient who was otherwise stabilizing. Progression to transmural necrosis results in perforation, peritonitis and septic shock that becomes refractory to further vasopressor escalation, thereby establishing a vicious circle (Acosta, 2015; Trompeter et al., 2002).

Risk Factors

Risk factors for NOMI reflect three domains: the severity and

Table 2: Reported risk factors for non-occlusive mesenteric ischemia in critically ill adults

<i>Domain</i>	<i>Factors</i>
Shock and hemodynamics	Cardiogenic shock and low cardiac output syndrome; septic shock; hypovolemia or hemorrhage; prolonged hypotension; high or escalating vasopressor doses; need for multiple vasopressors; elevated lactate at baseline (Groesdonk et al., 2013; Leone et al., 2015; Lim et al., 2017; Stahl et al., 2020a)
Cardiac surgery and mechanical support	Prolonged cardiopulmonary bypass; postoperative low cardiac output; intra-aortic balloon pump; extracorporeal life support; postoperative atrial fibrillation; norepinephrine above 0.10 micrograms/kg/min; prolonged ventilation (Groesdonk et al., 2013; Guillaume et al., 2017; Klotz et al., 2001; Lim et al., 2017; Nilsson et al., 2013; Renaudier et al., 2021; Sakamoto et al., 2020; Sato et al., 2018; Venkateswaran et al., 2002)
Patient substrate	Advanced age; peripheral and mesenteric atherosclerosis; heart failure; chronic kidney disease and hemodialysis; diabetes mellitus and adverse cardiometabolic risk profile (John et al., 2000; Kärkkäinen & Acosta, 2017; Okwah, 2022; Trompeter et al., 2002)
Concurrent drugs	Digoxin; ergot derivatives; cocaine and other sympathomimetics; diuretics producing hypovolemia (Clair & Beach, 2016; Trompeter et al., 2002)
ICU interventions	Enteral nutrition during unstable shock; dobutamine exposure; anemia; intra-abdominal hypertension; aggressive fluid removal or ultrafiltration; high positive end-expiratory pressure with low cardiac output (John et al., 2000; Kirkpatrick et al., 2013; Malbrain et al., 2005; Piton et al., 2022; Reignier et al., 2018)

type of shock, the patient's vascular and comorbid substrate, and iatrogenic exposures in the ICU (Table 2). Most derive from retrospective case-control or cohort designs, and the relative contributions of individual factors are uncertain.

Hemodialysis deserves particular mention. Intradialytic hypotension and rapid ultrafiltration can provoke mesenteric vasoconstriction, and patients with end-stage kidney disease have a recognized predisposition to NOMI (John et al., 2000). In the ICU, intermittent hemodialysis or high net ultrafiltration rates in patients receiving vasopressors may create comparable conditions.

Enteral nutrition increases intestinal oxygen demand. In NUTRIREA-2, a randomized trial of early enteral versus parenteral nutrition in ventilated adults with shock, the enteral group had a small but significant excess of bowel ischemia and acute colonic pseudo-obstruction, without a mortality difference (Reignier et al., 2018). This finding supports the physiological concern that full-target enteral feeding during unstable shock may precipitate ischemia in a borderline-perfused gut.

CLINICAL RECOGNITION AND DIAGNOSIS

Clinical presentation

Classical teaching describes abdominal pain out of proportion to physical findings in AMI (Clair & Beach, 2016). In the ICU this cue is largely unavailable: patients are sedated, intubated, delirious or receiving opioids, and pain may be absent in up to a large proportion of NOMI cases (Stahl et al., 2020a; Trompeter et al., 2002). Presentation is often insidious and is better framed as a clinical syndrome of unexplained deterioration. Features that should prompt consideration of NOMI include:

- Rising or persistent hyperlactatemia that is not explained by global hemodynamics, epinephrine, liver failure or

other identifiable causes (Bourcier et al., 2016; Bourcier et al., 2022).

- Increasing vasopressor requirements or new organ failure after a period of apparent stabilization.
- New enteral feeding intolerance, high gastric residual volumes, vomiting, ileus or abdominal distension (Reintam Blaser et al., 2012).
- Gastrointestinal bleeding, particularly bloody diarrhea or hematochezia suggesting colonic involvement (Brandt et al., 2015).
- Rising intra-abdominal pressure, new peritoneal signs, or unexplained fever, leukocytosis or bacteremia with enteric organisms.
- Metabolic acidosis with an increasing anion gap or base deficit.

In post-cardiac surgery patients, the combination of low cardiac output, high-dose vasopressor or inotrope support, and an unexplained increase in lactate during the first postoperative days is particularly suggestive (Groesdonk et al., 2013; Guillaume et al., 2017; Klotz et al., 2001). More broadly, gastrointestinal symptoms during the first ICU week are associated with higher mortality, and graded scores of gastrointestinal dysfunction have been developed to standardize their recognition and documentation (Reintam Blaser et al., 2013; Reintam Blaser et al., 2021).

Laboratory investigations

No laboratory test is sufficiently accurate to confirm or exclude NOMI. Serum lactate has high sensitivity but low specificity for intestinal ischemia, and a normal value does not exclude early ischemia because portal lactate is cleared by the liver (Cudnik et al., 2013; Evennett et al., 2009; Lange & Jäkel, 1994). In patients receiving epinephrine, lactate is confounded by beta-2 adrenergic stimulation (Levy et al.,

Table 3: Computed tomography features suggestive of non-occlusive mesenteric ischemia

<i>Feature</i>	<i>Comment</i>
Small-caliber SMA or reduced SMA diameter	Diffuse narrowing of the SMA trunk or reduced SMA to SMV diameter ratio (Nakamura et al., 2013; Perez-Garcia et al., 2018; Woodhams et al., 2010)
Irregular, beaded mesenteric branches	CT correlate of the angiographic string of sausages sign (Kammerer et al., 2018; Woodhams et al., 2010)
Patent mesenteric vessels	Absence of embolus, thrombus or venous occlusion is required for the diagnosis (Björck et al., 2017)
Reduced or absent bowel wall enhancement	Suggests significant ischemia; patchy, segmental or watershed distribution is typical (Nuzzo et al., 2017; Perez-Garcia et al., 2018)
Bowel dilatation and wall thinning	Associated with transmural necrosis (Nuzzo et al., 2017)
Pneumatosis intestinalis, portal or mesenteric venous gas	Late signs of transmural injury; not specific (Acosta, 2015; Bourcier et al., 2021)
Free fluid, free intraperitoneal air	Suggest advanced disease or perforation

SMA, superior mesenteric artery; SMV, superior mesenteric vein.

1997). D-dimer was first proposed as a sensitive marker of acute bowel ischemia in occlusive disease (Acosta et al., 2001) but is nonspecific in critical illness (Cudnik et al., 2013). A systematic review identified D-dimer, alpha-glutathione S-transferase and intestinal fatty acid binding protein as the more promising plasma markers, while lactate, white cell count and base excess performed poorly (Derikx et al., 2017). Novel markers of enterocyte injury, including intestinal fatty acid binding protein (I-FABP), alpha-glutathione S-transferase and plasma citrulline, have shown promise but have heterogeneous accuracy and are not widely available for clinical decisions, particularly in resource-limited laboratories where quality systems for novel assays are still developing (Peoc'h et al., 2018; Treskes et al., 2017; Yusuf & Abdulrazaq, 2018). Pooled sensitivity and specificity of serum I-FABP were about 0.80 and 0.85 (Sun et al., 2016; Thuijls et al., 2011), yet a prospective cohort found that citrulline, I-FABP and D-lactate did not discriminate AMI from other causes of acute abdominal pain (Nuzzo et al., 2021). In NOMI specifically, adding I-FABP to clinical variables improved discrimination in a Japanese cohort (Matsumoto et al., 2019). In practice, laboratory trends are most useful as triggers for imaging rather than as diagnostic tests. Integrated laboratory information systems that flag serial changes automatically, linked to real-time clinical analytics, can shorten the interval between an abnormal result and clinical action (Abdulrazaq et al., 2019; Nnaji & Akinlolu, 2022). In the DIAGOMI cohort, combinations of clinical, biological and radiological features performed better than any single marker (Bourcier et al., 2022).

Computed tomography

Contrast-enhanced multidetector computed tomography (CT) is the first-line imaging investigation recommended by current guidelines for suspected AMI, including NOMI (Bala et al., 2022; Björck et al., 2017; Tilsed et al., 2016). A

biphasic protocol, with arterial and portal venous phases and without oral contrast, allows assessment of the mesenteric arteries, excludes occlusive causes and characterizes bowel wall perfusion. A meta-analysis reported pooled sensitivity of about 93 percent and specificity of 96 percent for multidetector CT in AMI overall (Menke, 2010), and the American College of Radiology recommends CT angiography as the first-line test (Ginsburg et al., 2018). Its yield depends heavily on the radiologist being alerted to the clinical suspicion (Lehtimäki et al., 2015), and common findings are nonspecific while the most specific signs, such as absent wall enhancement and pneumatosis, are uncommon (Wiesner et al., 2003). CT findings associated with NOMI are summarized in Table 3.

Sensitivity of CT for NOMI is lower than for occlusive AMI, because early vasospasm may be subtle and vasopressor therapy itself can narrow mesenteric vessels (Bourcier et al., 2021; Kammerer et al., 2018). Concerns about contrast-associated kidney injury should not delay imaging in a patient with suspected ischemic bowel, given the far higher mortality of missed infarction (Bala et al., 2022). Transport of an unstable patient carries risk, and the decision requires bedside judgment, but the threshold for CT should be low when the clinical picture is compatible.

Angiography and other modalities

Selective SMA angiography remains the reference standard for diagnosing NOMI, using the criteria described by Siegelman and colleagues (Siegelman et al., 1974). Its major advantage is the opportunity to start intra-arterial vasodilator therapy immediately (Björck et al., 2017; Boley et al., 1977; Brandt & Boley, 2000; Lam et al., 2022; Trompeter et al., 2002). Angiography is invasive and requires transport to the interventional suite, and angiographic appearances may be influenced by systemic vasopressors. It is most useful when CT

Table 4: Practical approach to suspected vasopressor-associated NOMI

Step	Actions
1. Recognize	Maintain suspicion in high-risk patients; act on unexplained lactate rise, increasing vasopressor need, feeding intolerance, distension or bleeding; measure intra-abdominal pressure
2. Resuscitate flow, not only pressure	Assess and correct hypovolemia using dynamic indices; evaluate cardiac output with echocardiography or advanced monitoring; consider dobutamine or mechanical support in low output states
3. Vasopressor stewardship	Use the lowest dose achieving an individualized MAP target; prefer norepinephrine; add low-dose vasopressin rather than escalating catecholamines indefinitely; avoid epinephrine where alternatives exist; stop non-essential vasoconstrictive drugs
4. Protect the gut	Withhold or stop enteral nutrition; decompress the stomach; treat intra-abdominal hypertension; start broad-spectrum antimicrobials covering enteric organisms
5. Image early	Biphasic contrast-enhanced CT; involve radiology, surgery and interventional radiology at once
6. Restore mesenteric flow	Selective SMA angiography with intra-arterial vasodilator infusion when no immediate indication for surgery; consider systemic anticoagulation
7. Operate when indicated	Laparotomy for peritonitis, perforation or CT evidence of necrosis; resect non-viable bowel; damage control and planned second-look within 24 to 48 hours
8. Reassess	Repeat clinical, laboratory and imaging assessment; follow-up angiography during vasodilator therapy; continue optimization of hemodynamics

MAP, mean arterial pressure; SMA, superior mesenteric artery.

is suggestive or inconclusive and the patient has no indication for immediate laparotomy.

Bedside colonoscopy or flexible sigmoidoscopy with minimal insufflation can identify colonic mucosal ischemia and is useful when bloody diarrhea suggests colonic involvement, but it does not evaluate the small bowel or the depth of injury reliably (Brandt et al., 2015). Diagnostic laparoscopy or laparotomy may be required when non-invasive investigations are inconclusive and suspicion remains high, or when peritonitis is present (Bala et al., 2022; Yanar et al., 2007). Gastric tonometry, measurement of the mucosal-arterial PCO₂ gap, and indocyanine green techniques have been studied as markers of splanchnic perfusion, but none is established for routine screening (Bourcier et al., 2021; Hasibeder, 2010).

MANAGEMENT

Management of vasopressor-associated NOMI has two components: prevention through optimized hemodynamic care, and treatment once ischemia is suspected or established (Table 4).

Prevention: hemodynamic optimization and vasopressor stewardship

The most effective intervention is likely to be avoidance of the conditions that lead to NOMI. Adequate intravascular volume should be established before or alongside vasopressor initiation, while avoiding fluid overload that contributes to bowel edema and intra-abdominal hypertension (Evans et al., 2021; Kirkpatrick et al., 2013). Cardiac output should be assessed rather than assumed, particularly in cardiogenic shock and after cardiac surgery, and low-output states should be treated with inotropes or mechanical support rather than

escalating vasoconstrictors alone (Groesdonk et al., 2013; Guillaume et al., 2017). Levosimendan improved microvascular flow more than dobutamine in a small septic shock trial, although gut perfusion was not measured (Morelli et al., 2010), and dobutamine exposure was independently associated with AMI in ventilated patients with shock (Piton et al., 2022), so inotrope choice and dosing also warrant attention. Bedside echocardiography is well suited to this purpose and is increasingly used for cardiovascular risk stratification even in settings with limited access to advanced monitoring (Okwah, 2022).

Blood pressure targets influence cumulative vasopressor exposure. The Surviving Sepsis Campaign recommends an initial mean arterial pressure target of 65 mmHg in septic shock (Evans et al., 2021). In SEPSISPAM, targeting 80 to 85 mmHg did not reduce mortality and was associated with more atrial fibrillation (Asfar et al., 2014). In the 65 trial, permissive hypotension in patients aged 65 years and older reduced vasopressor exposure without significant harm (Lamontagne et al., 2020). Although none of these trials was powered for mesenteric outcomes, they support avoiding unnecessarily high pressure targets and the accompanying vasoconstrictor load. Norepinephrine should be the default first-line agent, with low-dose vasopressin added as norepinephrine requirements rise, rather than continued unlimited catecholamine escalation (Evans et al., 2021). Epinephrine is reasonable as an additional agent but its unfavorable splanchnic profile and confounding effect on lactate should be considered (De Backer et al., 2003; Meier-Hellmann et al., 1997). Vasopressor doses should be reviewed regularly and weaned as soon as perfusion permits. Agent selection is also shaped by availability: during a national

norepinephrine shortage in the United States, substitution with other vasopressors, mainly phenylephrine, was associated with higher mortality in septic shock (Vail et al., 2017). Drug shortage early warning systems and resilient pharmaceutical supply chains therefore have direct relevance to vasopressor stewardship (Awulu et al., 2022b; Eze et al., 2022a).

Enteral nutrition

European guidance recommends delaying enteral nutrition in uncontrolled shock and in overt bowel ischemia, and starting low-dose enteral feeding once shock is controlled with fluids and vasopressors (Reintam Blaser et al., 2017; Singer et al., 2019). North American guidance similarly advises withholding enteral feeding during hemodynamic instability or escalating vasopressor requirements (McClave et al., 2016), and enteral feeding was independently associated with AMI in the NUTRIREA-2 post hoc analysis (Piton et al., 2022). Signs of feeding intolerance in a patient receiving vasopressors should prompt reduction or cessation of feeding and evaluation for ischemia rather than prokinetic escalation alone (Reignier et al., 2018; Reintam Blaser et al., 2012). Trophic feeding is a reasonable compromise when vasopressor doses are stable and decreasing.

Intra-abdominal pressure

Intra-abdominal hypertension reduces mesenteric perfusion pressure and venous outflow. Intra-abdominal hypertension developing during the ICU stay independently predicts mortality (Malbrain et al., 2005), and regular bladder pressure measurement is recommended in patients with risk factors, which include shock, large-volume resuscitation and ileus (Kirkpatrick et al., 2013). Medical measures include gastric and rectal decompression, optimization of sedation and analgesia, avoidance of positive fluid balance and drainage of ascites; decompressive laparotomy is indicated for abdominal compartment syndrome (Kirkpatrick et al., 2013).

Medical treatment of established NOMI

Once NOMI is suspected, the immediate priorities are to restore cardiac output and volume status, reduce vasoconstrictor exposure to the minimum compatible with adequate perfusion pressure, and discontinue other vasoconstrictive drugs (Bala et al., 2022; Björck et al., 2017; Trompeter et al., 2002). Broad-spectrum antimicrobial therapy covering enteric gram-negative and anaerobic organisms is recommended because of mucosal barrier failure and translocation (Bala et al., 2022; Björck et al., 2017). In a prospective intestinal stroke center cohort, early oral antibiotics were independently associated with less transmural necrosis and fewer resections (Nuzzo et al., 2019). Empirical regimens should be informed by local antimicrobial resistance surveillance data, and assured medicine quality matters because substandard antimicrobials contribute to resistance (Aneke et al., 2022b; Yusuf et al., 2020). Gastric decompression and cessation of enteral feeding are appropriate. Systemic anticoagulation with unfractionated heparin is often

used, particularly during catheter-directed therapy, but specific evidence in NOMI is limited and the decision should account for bleeding risk (Bala et al., 2017; Björck et al., 2017).

Intra-arterial vasodilator therapy

Selective infusion of a vasodilator into the SMA aims to reverse persistent mesenteric vasospasm. Papaverine, a phosphodiesterase inhibitor, is the most widely reported agent; continuous infusion through an angiographic catheter positioned in the SMA, typically for 24 hours or more with repeat angiography to assess response, has been described in multiple series (Boley et al., 1977; Klotz et al., 2001; Trompeter et al., 2002). Papaverine precipitates with heparin, so separate infusion lines are required. Prostaglandin E1 has been used both intra-arterially and intravenously, with favorable outcomes reported from Japanese experience when combined with early CT diagnosis (Mitsuyoshi et al., 2007). Other agents described include tolazoline and nitroglycerin. In a retrospective study, local intra-arterial vasodilator infusion was associated with significantly improved survival compared with patients not receiving this treatment (Winzer et al., 2020). A meta-analysis found that intra-arterial vasodilator therapy reversed vasospasm in about three quarters of cases with few complications (Stahl et al., 2020b), and in a Japanese nationwide database, papaverine or prostaglandin E1 in surgically treated NOMI was associated with lower in-hospital mortality (Takiguchi et al., 2020). In a prospective study of NOMI with severe shock, an early fall in lactate during infusion predicted survival, and lower baseline norepinephrine doses predicted response (Rittgerodt et al., 2022). Guidelines from the ESVS and WSES suggest intra-arterial vasodilator therapy in NOMI patients without signs of peritonitis or bowel necrosis, while acknowledging that the evidence is of low quality (Bala et al., 2022; Björck et al., 2017). Systemic hypotension during infusion is uncommon with selective delivery but requires monitoring, and vasodilator therapy must be weighed against concurrent vasopressor requirements.

Surgical management

Surgery is indicated for peritonitis, perforation, or imaging or endoscopic evidence of transmural necrosis (Bala et al., 2022; Tilsed et al., 2016). Because NOMI is often patchy and extensive, and because mesenteric perfusion may change after resuscitation, the operative strategy differs from that of occlusive disease. Damage-control principles are favored: resection of clearly necrotic bowel, deferral of anastomosis, temporary abdominal closure and a planned second-look procedure within 24 to 48 hours to reassess marginally viable segments (Bala et al., 2022; Weber et al., 2014; Yanar et al., 2007). In a multicenter NOMI cohort, planned and on-demand relaparotomy strategies produced similar mortality (Endo et al., 2021), suggesting that close reassessment matters more than a fixed schedule. Where imaging instead reveals an occlusive lesion, endovascular-first revascularization is associated with

lower mortality and less bowel resection than open surgery (Arthurs et al., 2011; Beaulieu et al., 2014). Intraoperative indocyanine green fluorescence angiography may help define viable margins and limit the extent of resection (Karampinis et al., 2018). Intra-arterial vasodilator infusion can be continued perioperatively. The decision to operate in a patient on high-dose vasopressors is difficult; in practice, an unrecognized necrotic segment is often the driver of refractory shock, and earlier surgical involvement is associated with better outcomes in specialized intestinal stroke programs (Nuzzo et al., 2017).

Multidisciplinary pathways

Several centers have developed dedicated intestinal stroke units in which gastroenterologists, vascular and digestive surgeons, interventional radiologists and intensivists manage AMI according to a standardized protocol (Corcos et al., 2013; Nuzzo et al., 2017; Roussel et al., 2015). A multimodal strategy combining gut protection, early revascularization and selective resection achieved high survival with limited intestinal failure (Corcos et al., 2013). Survivors of extensive resection or prolonged critical illness often leave hospital with reduced independence, and structured support for activities of daily living, delivered as a clinical intervention rather than an afterthought, can help restore function after discharge (Osinem et al., 2021). Although these models were developed mainly for occlusive disease, the same principles of rapid diagnosis, early revascularization or vasodilator therapy and conservative resection apply to NOMI. Hospitals without such units can establish local pathways specifying who to call, which imaging protocol to use and when to consider angiography.

Special Populations

Post-cardiac surgery

NOMI after cardiac surgery typically presents within the first postoperative days in patients with low cardiac output, prolonged bypass, intra-aortic balloon pump or extracorporeal support (Groesdonk et al., 2013; Guillaume et al., 2017; Lim et al., 2017). Early angiography with intra-arterial papaverine was associated with improved survival in an early series (Klotz et al., 2001). Prevention focuses on optimizing cardiac output, limiting vasoconstrictor doses and monitoring lactate trajectories with a low threshold for imaging.

Septic shock

In septic shock, NOMI may be difficult to distinguish from an intra-abdominal septic source, and the two may coexist. New abdominal findings or lactate rise during treatment of a non-abdominal infection should raise the possibility of secondary gut ischemia. Adherence to evidence-based vasopressor selection and pressure targets, and avoidance of high-dose epinephrine where alternatives exist, are reasonable preventive measures (Evans et al., 2021; Meier-Hellmann et al., 1997).

Kidney replacement therapy

Patients receiving intermittent hemodialysis or high-volume

ultrafiltration while on vasopressors are at increased risk (John et al., 2000). Continuous modalities with conservative net ultrafiltration rates are preferable in hemodynamically unstable patients.

Resource-limited settings

Most evidence on NOMI comes from high-income health systems. In many low- and middle-income settings, including much of Sub-Saharan Africa, access to multidetector CT, interventional radiology and round-the-clock laboratory services is uneven, particularly outside tertiary centers, and the uptake of newer diagnostic techniques is often slowed by shortages of trained staff, gaps in quality assurance and unreliable reagent supply (Abdulrazaq et al., 2021; Aminu-Ibrahim et al., 2020; Yusuf & Abdulrazaq, 2018). Interruptions in the supply of vasopressors, papaverine and other essential parenteral drugs can further limit options (Awulu et al., 2022b; Eze et al., 2022a). In these settings, the principles of this review still apply but must be adapted: serial clinical examination, intra-abdominal pressure measurement, lactate trends, bedside ultrasound and echocardiography, and early surgical consultation assume greater importance, and referral pathways to centers with imaging and interventional capacity should be defined in advance (Nnaji & Akinlolu, 2022; Okwah, 2022).

Health System and Implementation Considerations

Improving outcomes in NOMI depends not only on bedside knowledge but on the systems in which critical care is delivered. Scientific advances fail to improve patient outcomes when delivery infrastructure is weak (Ilodigwe & Adesemoye, 2019b), and this is especially relevant to a condition whose management requires rapid imaging, laboratory support, interventional radiology, reliable drug supply and coordinated teams (Corcos et al., 2013). Related literature from health systems research and from other high-risk sectors identifies system-level levers that complement bedside care.

Diagnostic, imaging and laboratory capacity

Timely diagnosis requires laboratory and imaging services that are available around the clock. Sustainable diagnostic laboratory models, capital delivery approaches for high-risk healthcare facilities and coordinated multi-site expansion programs have been described for emerging health systems (Aminu-Ibrahim et al., 2018; Aminu-Ibrahim et al., 2019; Aminu-Ibrahim et al., 2021). Laboratory spatial planning and regulatory-compliant design influence diagnostic accuracy, safety and throughput (Ogbete et al., 2018; Ogbete et al., 2019), and capacity built for pandemic preparedness, together with scalable blood collection networks, can be leveraged for critical care (Aminu-Ibrahim et al., 2022; Ogbete et al., 2022). Data quality and surveillance capacity in routine health information systems remain variable in some settings (Ekemezie et al., 2018), and comparative frameworks for health information governance show how data verification, risk screening and confidentiality safeguards can be maintained as these systems

are linked and expanded (Peteranaba et al., 2022). Barriers to adopting new diagnostic techniques in resource-limited laboratories, documented in other specialties, are likely to apply to novel ischemia biomarkers (Abdulrazaq et al., 2021), and conceptual models linking biomarkers to individual susceptibility illustrate how biomarker data may eventually support personalized risk assessment (Aladegba et al., 2021). Automated image analysis methods developed in other imaging fields show the potential of computer-assisted detection to support radiologists (Ejofodomi et al., 2014).

Data, informatics and decision support

Early recognition of NOMI is fundamentally a problem of detecting a weak signal within noisy, continuously generated ICU data. Real-time analytics, dashboards and standardized reporting workflows have been used to reduce decision delays and improve visibility in health and other settings (Alieme et al., 2022; Orise & Niniola, 2022; Owoade et al., 2022; Sanni & Atima, 2021). Predictive and big data models for outbreak anticipation and mobile monitoring of chronic disease illustrate approaches that could be adapted to ICU deterioration (Oparah et al., 2021b; Oparah et al., 2022). Experience with anomaly detection in financial systems (Atakpa, 2021; Atakpa & Abetoh, 2022), machine learning forecasting (Edivri & Oteri, 2022; Tonoyan et al., 2021), and the transition from reactive to predictive maintenance in engineering, including interpretable early failure prediction (Adaramola et al., 2018; Komi & Adamolekun, 2021; Sunday et al., 2020), is relevant to the design of early warning tools for gut ischemia. Human-in-the-loop designs keep clinicians central to the interpretation of such tools (Ladapo et al., 2022).

Automated risk monitoring and alerting pipelines (Mbonu et al., 2019; Uchechi & Ominyi, 2020; Uchechi et al., 2022) depend on data that are reliable, secure and available when needed, which in turn requires attention to data protection governance, cybersecurity assurance and audit, and investment in healthcare cybersecurity (Dosunmu & Ogundele, 2019; Fadayomi et al., 2019; Mbonu et al., 2018; Ozowara et al., 2022). Platform governance in regulated healthcare environments and tamper-resistant recordkeeping support the integrity of clinical data used for audit and research (Anichukwueze et al., 2021; Badmus et al., 2019). Risk-based business intelligence architectures and technology-enabled audit frameworks (Akomolafe & Agu, 2018; Akomolafe & Agu, 2019; Mbonu et al., 2021) offer models for systematic clinical audit of NOMI cases and missed diagnoses, and data-informed digital learning platforms can support training of clinicians to recognize this uncommon complication (Orise & Niniola, 2020).

Lessons from safety science and high-reliability industries

Delayed recognition of NOMI is a patient safety event, and high-reliability industries offer transferable lessons. These include systematic hazard identification, near-miss reporting and analysis of human error causation (Arumosoye & Obriki,

2019; Obogo et al., 2019c; Obriki & Arumosoye, 2020); formal safety governance and safety management systems (Adeyelu, 2019; Obogo et al., 2019b; Obogo et al., 2020; Obogo et al., 2022); leadership-driven safety culture and organizational learning (Arumosoye & Obriki, 2021; Obogo et al., 2019a; Obriki & Arumosoye, 2019); incident prevention and the institutionalization of life-preserving practices that balance productivity with safety (Nwangwu et al., 2018; Obogo et al., 2021; Obriki & Arumosoye, 2021); data-driven risk control and predictive compliance monitoring (Adeyelu, 2018; Obriki & Arumosoye, 2018); and structured workforce training (Obriki et al., 2022a). Risk-based inspection approaches that concentrate surveillance on the highest-risk assets (Atta et al., 2020; Obetta, 2018), and risk modeling under data limitations (Lamidi & Olamide, 2018), parallel the targeted surveillance of high-risk ICU patients advocated in this review. Within hospitals, multidimensional compliance frameworks such as those developed for infection prevention and control (Omaghomi et al., 2022), structured emergency preparedness (Obriki et al., 2022b) and rapid incident response strategies (Dosunmu & Ogundele, 2021) provide models for building and auditing local NOMI pathways.

Medicines supply, quality and pharmacy practice

Vasopressors, inotropes, papaverine and prostaglandins are essential medicines whose availability cannot be assumed. Pharmacy-led delivery models, last-mile distribution strategies and Lean Six Sigma approaches to distribution bottlenecks address access gaps in developing settings (Asiedu & Asiedu, 2022; Ilodigwe & Adesemoye, 2020; Obukadata et al., 2021a). Supply chain resilience, risk management and predictive demand forecasting reduce the impact of disruptions (Agbabiaka et al., 2019; Aifuwa et al., 2020; Anene, 2021; Anene & Clement, 2022; Filani et al., 2022; Mbonu et al., 2020; Ogunwole et al., 2021), and regulatory-compliant procurement in high-risk environments is relevant to critical medicines (Okonkwo et al., 2021). Medicine quality depends on traceability and chain-of-custody integrity, risk-based good manufacturing practice, effective corrective and preventive action systems, regulatory readiness and interdiction of illicit products (Aneke & Adesemoye, 2019; Aneke & Adesemoye, 2020; Aneke & Adesemoye, 2021; Aneke et al., 2022a; Eze et al., 2022b), as well as on operational performance and digital transformation in pharmaceutical manufacturing (Awulu et al., 2022a; Obukadata et al., 2021b). At the bedside, systematic drug interaction screening, as proposed in other clinical contexts, could be adapted to identify concurrent vasoconstrictive medications in high-risk ICU patients (Asiedu, 2022).

Financing and translation

Financing mechanisms and provider payment reforms determine whether hospitals can sustain the imaging, interventional and critical care services on which NOMI management depends (Ilodigwe & Adesemoye, 2019a). The

persistent gap between scientific innovation and patient outcome underlines the importance of investment in delivery infrastructure alongside clinical research (Aminu-Ibrahim et al., 2019; Ilodigwe & Adesemoye, 2019b).

LIMITATIONS AND FUTURE DIRECTIONS

This review has limitations. It is narrative rather than systematic, so study selection may be subject to bias, and no formal appraisal or quantitative synthesis was undertaken. The evidence base for vasopressor-associated NOMI is also limited by its relatively low incidence, the difficulty of confirming the diagnosis, and confounding between vasopressor exposure and shock severity. Key research priorities include:

- Prospective multicenter cohorts with standardized diagnostic criteria to define incidence and risk factors, building on DIAGOMI (Bourcier et al., 2022).
- Validation of enterocyte injury biomarkers and multivariable prediction models, potentially incorporating machine learning on routinely collected ICU data, which in turn depends on interoperable, high-quality data infrastructure and transparent AI-based risk stratification (Bourcier et al., 2021; Ilodigwe & Adesemoye, 2021; Oparah et al., 2021a; Treskes et al., 2017).
- Reporting of mesenteric ischemia as a prespecified adverse event in randomized trials of vasopressors, blood pressure targets and nutrition (Reintam Blaser et al., 2020).
- Comparative studies of intra-arterial vasodilators, dosing, duration and the role of systemic anticoagulation (Björck et al., 2017; Winzer et al., 2020).
- Evaluation of bedside tools such as Doppler ultrasound of the SMA and intestinal perfusion imaging for screening in high-risk patients.
- Implementation research on multidisciplinary pathways and their effect on time to diagnosis and mortality.

CONCLUSION

Vasopressor-associated NOMI arises at the intersection of shock physiology and therapeutic vasoconstriction. The splanchnic circulation is uniquely vulnerable to this combination, and its failure can transform a recovering patient into one with refractory multiorgan dysfunction. Prevention depends on resuscitating flow as well as pressure, judicious selection and dosing of vasopressors, cautious enteral nutrition during unstable shock, and attention to intra-abdominal pressure. Recognition requires clinicians to look beyond classical abdominal signs and to act on unexplained deterioration with early CT and multidisciplinary consultation. Once diagnosed, restoration of mesenteric flow through hemodynamic optimization and intra-arterial vasodilator therapy, combined with timely resection of necrotic bowel, offers the best chance of survival. A structured, proactive approach to this complication should be part of routine care in every ICU that manages patients with shock, and prospective research on risk stratification, biomarkers and catheter-directed

therapy remains a priority.

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Ethics approval

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