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General Review

Nonocclusive Mesenteric Ischemia in Aortic Surgery: What You Need to Know

Ali Murtada,[1] Matti Jubouri,[2] Mohamed Refaie,[1] and Idhrees Mohammed,[3] Rhyl and York, United Kingdom; and Chennai, India

Background: Nonocclusive mesenteric ischemia (NOMI), a subtype of acute mesenteric ischemia (AMI), is primarily caused by mesenteric arterial vasoconstriction and decreased vascular resistance, leading to impaired intestinal perfusion.Commonly observed after cardiac surgery, NOMI affects older patients with cardiovascular or systemic diseases, accounting for 20−30% of AMI cases with a mortality rate of ∼50%. This review explores NOMI's pathophysiology, clinical implications in aortic dissection, and the unmet needs in diagnosis and management, emphasizing its prognostic significance.

Methods: A comprehensive literature review was conducted using multiple electronic databases to extract relevant data and information.

Results: NOMI is a life-threatening condition characterized by mesenteric vasoconstriction and reduced splanchnic blood flow, often triggered by cardiac surgery, hemodialysis, or hypotensive episodes. Epidemiological studies highlight its prevalence in intensive care unit settings, with a high mortality rate linked to delayed diagnosis and systemic hypoperfusion. Risk factors include advanced age, vasopressor use, and inflammatory markers. Biomarkers such as intestinal fatty acid binding protein, citrulline, and D-lactate show potential for early detection but lack robust clinical validation. Management includes fluid resuscitation, vasodilators, and surgical intervention for bowel necrosis. Emerging endovascular approaches show promise but are limited to select cases without bowel infarction. This review underscores the critical need for timely diagnosis, risk factor identification, and tailored interventions to improve outcomes.

Conclusion: NOMI remains poorly understood despite advances in surgical and perioperative care. Its pathophysiology, linked to cardiopulmonary bypass and intraoperative factors, requires heightened clinical vigilance. Limited evidence underscores the need for a multidisciplinary approach involving surgeons, radiologists, and anesthetists to improve diagnosis, management, and outcomes in aortic surgery patients.

INTRODUCTION

Acute mesenteric ischemia (AMI) syndromes include three related processes: occlusive mesenteric ischemia, nonocclusive ischemia, and sepsis-induced SI. Nonocclusive mesenteric ischemia (NOMI) is primarily due to mesenteric arterial vasoconstriction and decreased vascular resistance, ushering in decreased intestinal perfusion and malfunction. NOMI was first described in patients with heart failure.[1]

Although the NOMI incidence rate following aortic surgery is undetermined, its predilection following cardiac surgery has been reported to be

[1]*Department of General Surgery, Glan Clwyd Hospital, Rhyl, UK.*

[2]*Hull York Medical School, University of York, York, UK.*

[3]*Institute of Cardiac and Aortic Disorders, SRM Institutes for Medical Science (SIMS Hospitals), Chennai, India.*

Correspondence to: Ali Murtada, BSc, MD, MRCS FRCS(Eng), Department of General Surgery Wales, Ysbyty Glan Clwyd, 22 Beaconsfield, Chester CH3 5Az, United Kingdom; E-mail: ali.murtada@wales.nhs.uk

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as high as 90%.[2,3] It generally affects patients over 50 years of age suffering from myocardial infarction, congestive heart failure, aortic insufficiency, renal or hepatic disease and patients following -cardiac surgery. Nonocclusive disease accounts for 20−30% of all cases of AMI with a mortality rate of the order of 50%.[4] Nonocclusive intestinal ischemia in aortic patients is a poorly understood entity and is likely to be a lesser-known complication with an incompletely understood pathophysiology. The significance of NOMI is that it is an essential element in the recovery and prognostication of our diseased population cohort.

Patients with aortic dissection are known to have hemodynamic instabilities, including lumen wall shear stress and pressure, geometrical factors such as entry tear location and size, and the distorted composition of the aortic wall (Fig. 1).[5] The instability and interval of acute hypotension can overwhelm local bowel autoregulation protective mechanisms. At a molecular level, the colon's mucosal layer becomes increasingly susceptible to ischemia. The splanchnic blood circulation is vital in regulating systemic blood pressure. Hence, blood flow to vital organs is maintained by shifting the flow away from the splanchnic vessels.

Consequently, any significant reduction of splanchnic blood flow exacerbates the severity of nonocclusive mesenteric ischemic events, usually observed as early as 10 min after the onset of hypotension.[6] The intestine can compensate for approximately a 75% acute reduction in mesenteric blood flow for up to 12 h without substantial injury, partly because of increased oxygen extraction.[7] However, attempts at interventions can yield an ischemia-reperfusion injury that predominantly affects the intestinal mucosa and submucosa due to oxidative stress and inflammatory cascade, leading to impairment of the mechanistic process that prevents the translocation of bacteria from the intestinal lumen. This is the exactness of why, during recovery, the bowel may not be functional, and attempts at enteral feeding may result in intestinal distension, osmotic diarrhea, and additional intestinal damage, leading to ischemic colitis and irreversible colonic necrosis.

NOMI has a subtle clinical presentation, and its timing and severity remain unknown in aortic practice. This review aims to dwell on the known, highlighting the unmet clinical and surgical needs and gaps in the evidence. The review also highlights the correlations and pertinent of NOMI to cardiovascular surgical emergencies.

METHODS

Literature Search Strategy

Electronic searches were performed on PubMed, Scopus, EMBASE, and Google Scholar databases with no date limits. Search terms included nonocclusive mesenteric ischemia, thoracic aortic aneurysms, size, risk factors, survival rates, medical therapy, aneurysm growth, dissection, rupture, and mortality. Search terms were charted to MeSH terms and combined using Boolean operations. They were also used as keywords. Papers were selected based on title and abstract. The reference lists of selected papers were reviewed to identify any relevant papers suitable for inclusion in the study.

Selection Criteria

The inclusion and exclusion criteria were used, considering fundamentals to minimize ambiguity and reduce the possibility of poor reproducibility due to many decisions regarding what to include to eliminate random error in study selection using a stringent protocol. Hence, the criteria were a priori based on the analytic framework or conceptual model. Research papers were not excluded based on study design except for case reports. Comments, opinions, or editorials were not included in our selection to provide an unbiased view. Papers were selected to offer primary endpoints of and/or information regarding NOMI. Papers were not excluded based on patient population age. Inclusion criteria for the population(s) of interest were defined in terms of relevant demographic variables, disease variables (i.e., variations in diagnostic criteria, disease stage, type, or severity), risk factors for disease, cointerventions, and coexisting conditions. The inclusion and exclusion criteria were identified to describe outcomes, outline any restrictions on measurement methods or timing of outcome measurement, and provide guidance for handling composite outcomes.

Epidemiological Perspective of NOMI

The reports of severe events related to nonocclusive mesenteric ischemia (NOMI) are predominantly derived from case series and retrospective cohort studies that focus on selected populations. The Swedish population-based study conducted between 1970 and 1982 indicated a population-level incidence of fatal NOMI at 2/100,000 person-years. From 23,446 systematic autopsies conducted

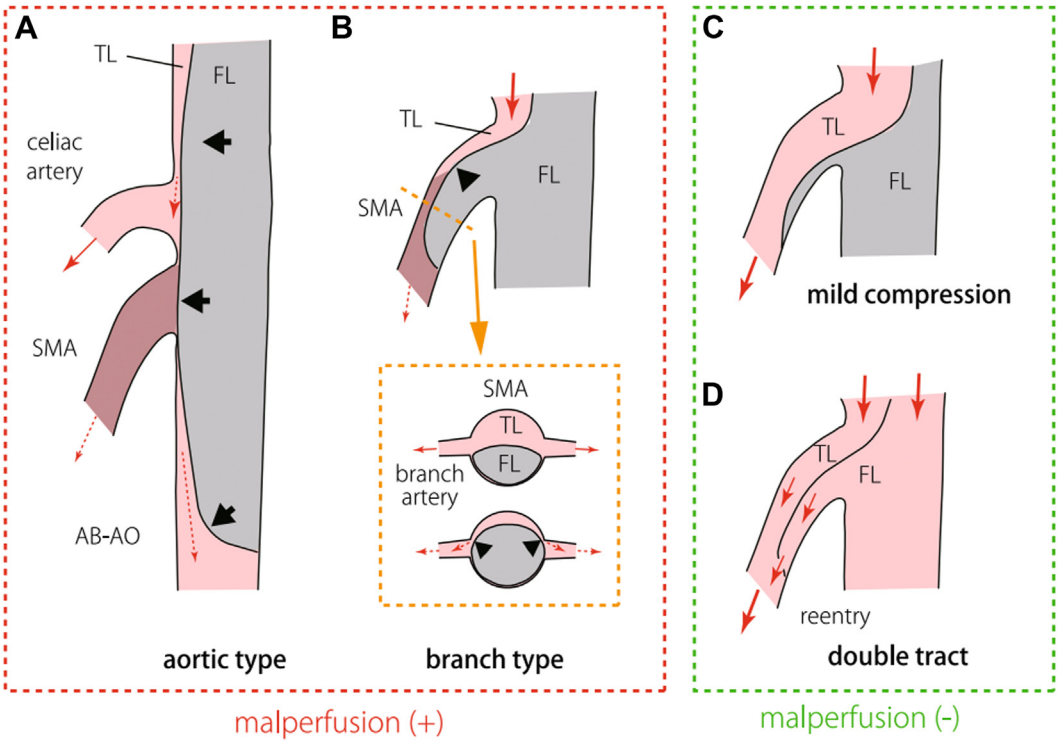

Fig. 1. Schematic illustration of morphological and hemodynamic patterns of mesenteric ischemia. The aortic type **(A)** and branch type **(B)** cause significant malperfusion, while mild compression of the true lumen or double tract perfusion do not cause malperfusion. AB-AO, abdominal aorta; FL, false lumen; SMA, superior mesenteric artery. Reproduced from Orihashi et al. [REF] with copyright permission obtained.

during this period, 62 fatal NOMI cases were documented. Upon thorough investigation of clinical data, it was found that these patients exhibited a higher likelihood of experiencing fatal cardiac failure, atrial fibrillation, and recent surgical interventions. In addition, necroptotic examinations frequently revealed concurrent infarctions in other visceral organs, including the liver, spleen, and kidneys, pointing toward a systemic state of global organ hypoperfusion. Within the framework of a nonrecent retrospective monocentric surgical case series, the prevalence of NOMI as a causative factor for AMI varies significantly, with reported rates ranging from 4% to 60%, contingent upon the specific case mix involved.[8,9]

Several other smaller cohorts also reported hemodialysis as a setting associated with a risk of NOMI.[10] In a retrospective study of 57 cases occurring in the first 12 hours after the last hemodialysis session, all cases were preceded by hypotension during hemodialysis, and investigations found diffuse ($\geq$3) ischemic areas in 20% of cases.

In the intensive care unit (ICU), while described for decades, interest in NOMI is growing, as shown by an increase in reporting in the last few years.[11,12] To date, the most significant retrospective multicenter study gathered 780 AMI diagnoses in ICU patients, reporting in-ICU mortality of 58%.[13] Of note, the occlusive or nonocclusive origin of AMI was not investigated. NOMI appears prevalent in 91% of cases in a study of 101 acute mesenteric ischemic syndrome patients, with similar rates in other cohorts. This increased prevalence in ICU may be explained by the fact that many conditions leading to ICU admission may be associated with a NOMI onset.

Several studies reported NOMI as a complication of cardiopulmonary bypass (CPB) surgery, occurring in less than 1% of patients, often in patients with peripheral artery disease.[14–16] As a result, NOMI should be suspected in patients suffering from multiple organ failure after cardiac surgery, as suggested by Guillaume et al.[17] in a cohort study of 320 patients in which the NOMI rate was 10% and reported a median of 7 days between cardiac surgery and NOMI diagnosis.

Understanding Risk Factors

Identifying the risk factors for NOMI during the preoperative and perioperative periods may help

early detection and intervention, which leads to improved clinical outcomes. Many risk factors, such as age, using an intraaortic balloon pump, vasopressors, and increases in inflammatory markers, are implicated in NOMI. The clinical threshold, suspicion, and timing of the suspicion of NOMI with risk factors remain unclear. Moreover, the patient's clinical profile, often associated with prolonged surgery, makes it difficult to understand what affects the results of increasingly challenging procedures.

Splanchnic blood flow is subject to various influences, including the autonomic nervous system, hormonal dynamics, endogenous mediators, and pharmacological agents. Reperfusion injury refers to the secondary damage that occurs in ischemic tissues following the restoration of blood flow. This condition is marked by profound vasospasm and ischemic injury to the intestinal mucosa, which may result from cellular damage mediated by oxygen-derived free radicals. The nonocclusive transformation of what is initially occlusive ischemia underscores the potential for vasodilatory therapy in managing occlusive splanchnic ischemia.

NOMI has been documented in various clinical scenarios, including shock syndromes, low-flow states, drug-induced vasoconstriction, and hypotensive episodes exacerbated by vasodilator therapy, among other conditions. Individuals with preexisting vascular diseases, such as diabetes complicated by small vessel disease or arteriosclerosis, are particularly predisposed to nonocclusive ischemia.

The Role of Biomarkers

Biomarkers associated with mucosal ischemia are intestinal fatty acid binding protein (I-FABP), D-lactate, or citrulline. There is no substantial evidence to validate their use in current practice or their generality. To date, limited studies have delineated the efficiency and effectiveness of biomarkers in NOMI.

Fatty Acid-Binding Protein

Fatty Acid-Binding Protein (FABP) represents a group of proteins with an intestinal isoform (intestinal fatty acid-binding protein (I-FABP)). In ischemic events, a surge is released into circulation, which can potentially be measured.

The circulating levels are potentially valid to reflect the severity of ischemia-reperfusion injury.[18] FABP sensitivity and specificity for mesenteric ischemia diagnosis are approximately (80–90% and 85–89%), respectively.[19] In one of the recent studies, the association between I-FABP, multiple organ dysfunction, and 30-day mortality was observed. In a cohort of 50 patients, elevated admission I-FABP levels (38 ng/L) were associated with a higher incidence of multiple organ dysfunction and mortality. Conversely, the mean I-FABP values at admission in patients with a better prognosis were 18.3 ng/L.[20]

Citrulline. Plasma citrulline is a nonprotein amino acid. Enterocytes of the small intestinal mucosa predominantly produce it, making it a functional enterocyte mass marker. Low plasma citrulline is mainly associated with elevated I-FABP concentrations and bacterial translocation.[21]

D-Lactate. D-lactate is an isomeric form of lactate produced by colic bacteria as a typical result of bacterial metabolism. However, during ischemia, as the usual mucosal barrier is injured and permeability rises, D-lactate is released into the circulation. A higher blood concentration can be detected since the liver cannot metabolize D-lactate dehydrogenase due to a lack of D-lactate dehydrogenase, which may also reflect the intensity of bacterial translocation.

Endotoxin. Endotoxin (lipopolysaccharide or LPS) is a significant component of Gram-negative bacterial membranes and is common in the human intestine. The average plasma concentration is approximately 3 pg/mL. If released into the circulation, it causes multiple toxic effects, primarily by activating toll-like receptor 4. The most significant reactions are leukocyte and immune system activation to produce pro-inflammatory cytokines and activation of the complement and coagulation systems. Endotoxemia, sepsis, or the exacerbation of the systemic inflammatory response results from the release of a large amount of endotoxin [68]. In the case of intestinal barrier injury, increased motility of the gastrointestinal tract as a consequence of nutritional administration may increase endotoxin translocation. Higher biomarkers, such as I-FABP or citrulline, have been observed in patients after CA associated with endotoxemia.[22]

Correlation of NOMI with Aortic Repair

The duration of CPB and deep hypothermic circulatory arrest has significant implications for NOMI; however, there is no consensus in the literature regarding the ideal time that leads to more excellent surgical safety. However, decreasing aortic cross-clamping time and CPB is one of the most challenging issues in cardiac surgery. Their prolonged use and their association with NOMI are correlated with increased intra and postoperative complications after cardiac surgery. Those complications, caused by myocardial damage and the increased inflammatory response, can lead to low

cardiac output syndrome, renal dysfunction, vasoplegia, neurological deficit, and increased ventilation time. It's also presumed that a state of low cardiac output and bleeding are potential aggravators of NOMI and mesenteric injury; hence, intraoperative and postoperative are needed to assess and quantify the severity of NOMI.

The risk that CPB correlates to NOMI is associated with a nonobstructive decrease in oxygen delivery that falls below the critical threshold; at this point, oxygen consumption becomes dependent on supply, leading tissues to resort to anaerobic metabolism. Visceral hypoxia is marked by a decrease in adenosine triphosphate synthesis and an increase in catabolism. Ischemia ultimately arises from either a reduction in oxygen consumption, excessive oxygen demand, or a combination of both factors. Diminished mesenteric oxygen consumption typically results from decreased oxygen delivery but may also occur due to significantly impaired cellular oxygen uptake or abnormalities in the oxygen utilization mechanisms. Anaerobic metabolism generates excess acid, which is buffered and increases carbon dioxide (CO2) production. This rise in CO2 and impaired CO2 removal due to substantially reduced or absent blood flow underlies the phenomena of cellular, tissue, and venous hypercarbia.

How to Manage NOMI?

Conservative Approach. The management of NOMI primarily involves addressing the underlying precipitating factors. Essential initial interventions include fluid resuscitation, cardiac output optimization, and vasopressor use cessation. Additional therapeutic options may encompass systemic anticoagulation and catheter-directed administration of vasodilatory and antispasmodic agents, predominantly papaverine hydrochloride.[23]

Surgically. Surgical intervention is warranted in the presence of peritonitis, bowel perforation, or a significant decline in the patient's overall condition. In cases where patients exhibit signs of peritonitis, an exploratory laparotomy is imperative for the resection of necrotic bowel tissue. Unfortunately, the critical condition of these patients is often associated with a high mortality rate, which can range from 50% to 85%. Consequently, a damage control strategy is essential, given the severity of these cases.[24,25]

The presence of a nonviable intestine, if not promptly identified, can lead to multisystem organ dysfunction and ultimately result in mortality. An immediate laparotomy facilitates the direct evaluation of bowel viability.

Following initial resuscitation efforts, a midline laparotomy should be executed, which allows for the thorough assessment of all segments of the intestine. Decisions regarding resecting areas that are necrotic should be made at this stage. In situations where viability is uncertain, intraoperative Doppler assessment may prove beneficial; detecting Doppler signals over the distal branches of the superior mesenteric artery (SMA) supports bowel preservation and minimizes the risk of long-term disability. The SMA can be conveniently palpated by positioning fingers posterior to the root of the mesentery, where it presents as a firm tubular structure that may or may not exhibit a palpable pulse. Alternatively, the SMA can be localized by tracing the middle colic artery as it converges with the SMA at the mesentery. Adequate revascularization exposure necessitates direct, sharp dissection to isolate the artery from its surrounding mesenteric tissue. In cases of diagnostic ambiguity, arteriography is the preferred imaging modality, which can be conducted intraoperatively, particularly in hybrid surgical suites.

Restorative techniques for blood flow are selected based on the underlying pathophysiology of AMI. Combined with primary or patch angioplasty, embolectomy constitutes a well-established definitive intervention for SMA emboli. Conversely, thrombosis at the origin of the aorta, frequently associated with diffuse atherosclerosis, may necessitate a bypass procedure. However, this approach amplifies the complexity of surgical interventions and could necessitate using prosthetic materials in contaminated fields. One viable option includes a retrograde bypass from the iliac artery to the distal SMA utilizing the femoral vein or a synthetic graft.

The Current Role of Endovascular Revascularization

There are no randomized controlled trials comparing laparotomy versus endovascular treatment as a first-line strategy for managing mesenteric occlusion. The most important argument in favor of the early laparotomy approach is the ability to assess bowel viability directly, and thereby minimize delays in restoring mesenteric blood flow.

Several case series using endovascular techniques in combination with pharmacologic therapy have been reported recently.[26–28] However, any evidence of bowel ischemia or infarction precludes the use of thrombolytic therapy. Contraindications to thrombolytic therapy include recent surgery, trauma, cerebrovascular or gastrointestinal bleeding, and uncontrolled hypertension.

In a recent retrospective series of 679 patients with AMI and vascular intervention (both open and endovascular), endovascular treatment was performed in 24% (165 patients). The technique was successful in 87% of the patients, and in-hospital mortality was lower than among those who underwent open procedures (25 vs. 40%).[29] Again, this report emphasized that only patients who did not require open emergent intervention are suitable for this revascularization technical approach.

Endovascular embolectomy may be achieved by percutaneous mechanical aspiration or thrombolysis and permits percutaneous transluminal angioplasty, with or without stenting, in case series of patients with evidence of acute partial or complete occlusion of the SMA (either the main trunk or branch) and without no clinical or imaging evidence of advanced bowel ischemia. Complete technical success was achieved in 28% of cases; all had occlusion of the main SMA trunk.[30,31]

CONCLUSION

Despite advancements in surgical techniques, anesthesia, and perioperative management, the phenomenon of NOMI remains inadequately understood within the surgical community. The pathophysiology of NOMI, particularly its incidence following aortic surgical interventions, is predominantly linked to the effects of CPB and various intraoperative factors. Clinicians should maintain a heightened awareness of NOMI in scenarios characterized by the sudden onset of severe abdominal pain, metabolic acidosis, and evidence of systemic organ failure. A notable deficiency exists in robust clinical evidence regarding the incidence and outcomes of NOMI within the context of aortic surgery; much of the existing literature is derived from retrospective analyses that often yield inconclusive results. To enhance patient outcomes, a multidisciplinary approach, engaging acute care surgeons, radiologists, anesthetists, and cardiovascular surgeons, is paramount in the management of this complex condition

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

Ali Murtada: Conceptualization, Supervision, Writing – review & editing. **Matti Jubouri:** Resources, Writing – original draft, Data curation. **Mohamed Refaie:** Formal analysis, Resources, Methodology. **Idhrees Mohammed:** Visualization, Writing – review & editing.

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