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Nonocclusive mesenteric ischemia secondary to antihypertensive drug overdose successfully managed with early extracorporeal membrane oxygenation

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Case Report

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Abstract

Background: Nonocclusive mesenteric ischemia (NOMI) is a life-threatening complication of systemic hypoperfusion.

Case Presentation: A woman in her 50s presented with refractory hypotension after intentional overdose of amlodipine (150 mg), candesartan (240 mg), and imeglimin (24,500 mg). Computed tomography revealed portal venous gas and intestinal edema, findings consistent with NOMI. Despite vasopressor support, circulatory instability persisted, and venoarterial extracorporeal membrane oxygenation (VA-ECMO) was initiated. After stabilization on VA-ECMO, contrast-enhanced CT on day 2 showed reduced enhancement of the small bowel. Exploratory laparotomy revealed edematous but viable intestine, allowing us to avoid resection. The patient recovered fully and was transferred to psychiatry without complications.

Conclusion: This case highlights the potential role of early ECMO initiation as a bridge to recovery in stabilizing hemodynamics and preserving intestinal viability in NOMI secondary to drug-induced hypotension, particularly when instituted before irreversible intestinal necrosis develops.

Background

Nonocclusive mesenteric ischemia (NOMI) is a life-threatening condition resulting from systemic hypoperfusion, often seen in septic shock or postoperative low-flow states [1, 2]. While antihypertensive drug overdose is a known cause of persistent hypotension [3], NOMI associated with such overdose is rarely reported. We present a unique case of NOMI following intentional overdose of antihypertensive agents, successfully managed without bowel resection by early initiation of venoarterial extracorporeal membrane oxygenation (VA-ECMO).

Case Presentation

A woman in her 50s was brought to our hospital after intentional overdose of amlodipine (150 mg), candesartan (240 mg), and imeglimin (24,500 mg). She had experienced recent psychotic symptoms and had previously attempted overdose with hypnotics one week earlier.

On arrival, her Glasgow Coma Scale score was E3V3M5, and blood pressure was 52/30 mmHg despite high-dose norepinephrine (0.3 μg/kg/min). Arterial blood gas analysis revealed severe metabolic acidosis (pH 7.06, base excess −21 mEq/L), profound lactic acidosis (lactate 19.3 mmol/L), and an elevated anion gap (33.5 mEq/L). While no portal venous gas was noted on imaging at the referring hospital, non-contrast abdominal CT performed on admission to our hospital revealed new portal venous gas (Figure 1A) and bowel wall edema (Figure 1B), findings suggestive of nonocclusive mesenteric ischemia (NOMI). However, profound circulatory collapse precluded both contrast-enhanced CT and immediate exploratory laparotomy. Therefore, circulatory stabilization with VA-ECMO was prioritized.

The patient was intubated and mechanically ventilated at the emergency department. Despite administration of maximal catecholamines, she remained profoundly hypotensive, consistent with drug-induced, catecholamine-resistant distributive shock. Given the potential reversibility of the shock state due to antihypertensive overdose, VA-ECMO was urgently initiated (flow 4.0 L/min). Circulatory instability persisted despite adjunctive treatment with norepinephrine, vasopressin, calcium chloride, sodium bicarbonate (Meylon®), midodrine (Calthro®), meropenem, and dobutamine. Given the risk of hypoglycemia following imeglimin ingestion, continuous insulin infusion was not administered. Continuous renal replacement therapy was started for acute kidney injury with severe lactic acidosis.

On hospital day 2, her hemodynamics improved, and ECMO was successfully weaned. Repeat contrast-enhanced abdominal CT revealed reduced enhancement in segments of the small intestine (Figure 1C), prompting exploratory laparotomy. Intraoperative findings included large amounts of odorless, serous ascites and a 20 cm segment of mildly edematous and discolored jejunum distal to the ligament of Treitz (Figure 1D). However, there were no signs of necrosis, and bowel resection was not required.

The patient was extubated on day 8, tolerated oral intake, and ambulated independently. She was transferred to psychiatric care on hospital day 12 without complications.

Discussion and Conclusions

Overdose of antihypertensive agents can lead to prolonged hypotension and bradyarrhythmia [4], but NOMI is a largely unrecognized complication [5]. In the present case, the shock state was likely multifactorial, involving profound vasoplegia and myocardial depression secondary to calcium channel blocker and angiotensin receptor blocker overdose. This resulted in catecholamine-refractory hypotension and global hypoperfusion, predisposing the patient to the development of NOMI. Sustained low-flow states may result in global hypoperfusion, precipitating mesenteric ischemia [1]. NOMI remains difficult to diagnose early due to nonspecific clinical symptoms, but imaging findings such as portal venous gas, bowel wall edema, and decreased enhancement on contrast-enhanced CT are highly suggestive. In this case, early surgical intervention was initially contraindicated due to profound circulatory collapse. VA-ECMO served as a bridge to recovery by maintaining systemic and mesenteric perfusion until ischemic damage reversed. Importantly, VA-ECMO was initiated before irreversible intestinal necrosis developed. By promptly restoring systemic circulation and mesenteric perfusion, ECMO may have halted the progression from functional ischemia to transmural necrosis, thereby allowing bowel preservation. Although ECMO use for antihypertensive overdose has been reported in previous case reports[6], its role in preventing the progression of NOMI has not been well described. This case highlights that early ECMO deployment can sustain mesenteric perfusion, preserve bowel viability, and potentially avert the need for resection in NOMI secondary to drug-induced hypotension. Further investigation is needed to clarify indications, timing, and outcomes of ECMO use in similar scenarios. Early initiation of VA-ECMO in cases of antihypertensive drug overdose complicated by NOMI may stabilize hemodynamics, limit intestinal ischemic injury, and prevent bowel resection.

Abbreviations

ECMO
extracorporeal membrane oxygenation
NOMI
nonocclusive mesenteric ischemia
VA-ECMO
venoarterial extracorporeal membrane oxygenation

Declarations

Ethics approval and consent to participate

In Japan, approval from an ethics committee is not required to report the cases. This case was reported in accordance with ethical guidelines for medical and health research involving human subjects established by the Japanese government.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests

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Authors' contributions

YW, YK, SK, HA, ET, TM, TF, YK, HO, KS, TM, SY, and HO treated the patient. YW, YK, and HO wrote the manuscript. YK and HO revised and edited the manuscript. All authors have read and approved the final manuscript.

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Figures

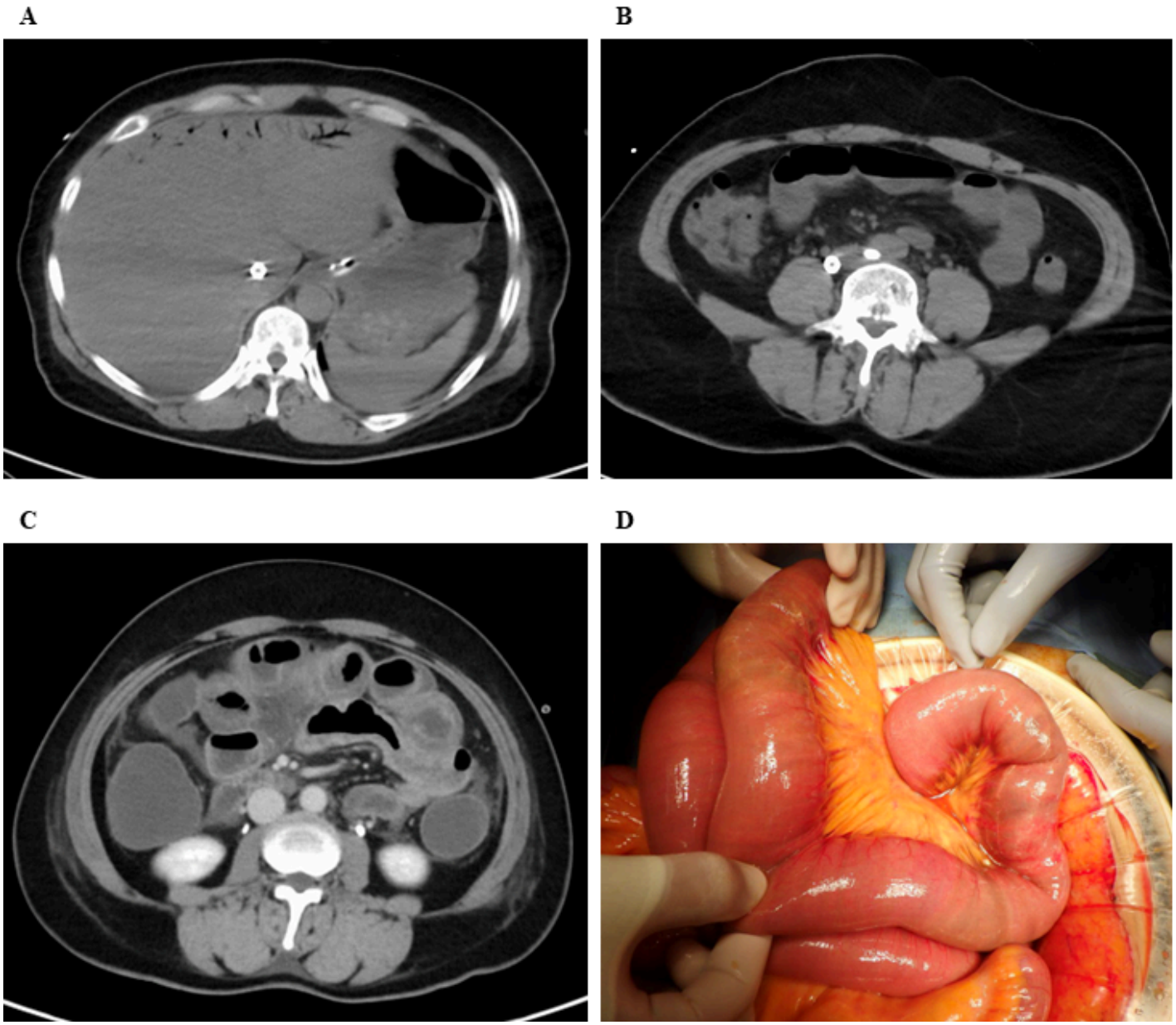


Figure 1

Sequential imaging and intraoperative findings in a patient with nonocclusive mesenteric ischemia (NOMI) secondary to antihypertensive drug overdose.

(A) Initial non-contrast abdominal CT demonstrating portal venous gas, suggesting early mesenteric ischemia.

(B) A subsequent slice from the same CT showing diffuse bowel wall edema.

(C) Contrast-enhanced CT on hospital day 2 revealing decreased enhancement of the small intestine, consistent with progression of ischemia.

(D) Intraoperative findings showing mild edema and segmental discoloration of the jejunum without definitive necrosis, allowing bowel preservation.

Collectively, these findings suggest the reversibility of intestinal ischemia under timely restoration of systemic circulation with VA-ECMO, prior to the development of irreversible necrosis.