Fulminant non-occlusive mesenteric ischemia after head trauma: report of two cases

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Abstract
There have been no case reports of non-occlusive mesenteric ischaemia (NOMI) following head trauma. Our two patients with non-surgical traumatic intracerebral hemorrhage succumbed to NOMI one week after the injury. Both were women over age 80 years and were clinically improving before NOMI occurred. One patient had been eating since admission, while the other had not, which prompted initiation of enteral nutrition on day 5. The patients shared many characteristics: 1) over age 80 years; 2) minor brain contusion; 3) constipation for a week; 4) minimal abdominal symptoms; 5) rapidly developing leukocytosis, hyperglycemia, hypernatremia, and elevated blood urea nitrogen; 6) massive diarrhea with a small amount of blood on the same day that laboratory data became abnormal; and 7) fever and shock developed shortly after diarrhea appeared. Due to the fulminant worsening of the condition, surgical intervention was not performed in either patient because of their rapid progression to a fulminant disease stage and advanced age. In retrospect, if NOMI was diagnosed earlier when the acute pancreatitis-like symptoms began, surgical intervention may have saved their lives. Clinicians should be aware that NOMI can occur after relatively minor head trauma, which can cause death if the diagnosis is delayed.

Categories: Gastroenterology, Neurosurgery, Emergency Medicine
Keywords: acute pancreatitis, diarrhea, hyperglycemia, head trauma, non-occlusive mesenteric ischemia

Introduction
Non-occlusive mesenteric ischemia (NOMI) is a life-threatening disorder with an extremely high mortality rate [1, 2]. The cause is insufficient blood supply to the intestine, which may result from circulatory failure (cardiogenic shock), septic shock, hypovolemic shock, or cardiac arrest [1-5]. To the best of our knowledge, NOMI has not been previously reported after head trauma. We present here two patients who died of NOMI, although they were under conservative treatments after a mild traumatic intracerebral hemorrhage and were recovering from the trauma.

Case Presentation
Case 1
An 86-year-old woman with a history of hypertension struck her head on the living room floor during a family visit to her home. On hospital arrival, her height and weight were 152 cm and 48.5 kg, respectively. She was amnestic to the event and disoriented. Glasgow Coma Scale (GCS) score was 14 (E4V4M6). Her vital signs on arrival (Day 0) and throughout hospitalization are shown in Figure 1a. Head computed tomography (CT) demonstrated a left subdural hygroma and subependymal areas of high signal density along the right lateral ventricle. The patient was admitted for observation. Follow-up CT on Day 2 showed enlargement of the left subdural hygroma and blurring of the high-density areas. On Day 5, she complained of decreased appetite and no bowel movements since admission. Oral intake had previously been adequate. Abdominal CT was unremarkable. Laboratory data on Day 7 showed leukocytosis (26.6 × 103/mL), high C-reactive protein (CRP) concentration (11.6 mg/dL), high blood urea nitrogen (BUN) concentration (62.0 mg/dL), hypernatremia (153 mmol/L) and hyperglycemia (554 mg/dL) (Table 1). Her vital signs remained stable. Acute pancreatitis was suspected, but she had no abdominal or back pain and concentrations of amylase and lipase levels were normal (Table 1). An intravenous insulin drip was initiated. That evening (Day 7), after defecating watery stools with a tiny amount of blood, the patient’s level of consciousness deteriorated rapidly. On examination, her GCS score was 6 (E2V1M3) and she grimaced when her abdomen was palpated. Head CT showed no change. Abdominal CT demonstrated massive ascites, gas in the hepatic portal venous system, and edema throughout the small intestine, which strongly suggested NOMI. Her family declined emergency surgery because of her advanced age. Palliative treatment and supportive care were administered. On Day 8, her serum glucose and sodium concentrations normalized to 125 mg/dL and 144 mmol/L, respectively; her leukocytosis had worsened (70.4 × 103/mL), however. The patient died later that day. An autopsy was not performed. (Fig. 1a-f)
FIGURE 1: Case 1

a. Vital signs over time: Until Day 6, the patient’s vital signs were stable. Triggered by diarrhea at Day 7, her body temperature suddenly became high and subsequently went into shock status.

b. Head CT on admission: High-density areas are identified at the subependymal area along the right lateral ventricle with low density areas around the lesions. Thin subdural hygroma appears in the left side.

c. Head CT on Day 2: The high-density areas along the right ventricle disappeared but left subdural space enlarges compressing the left hemisphere.

d. Abdominal CT on Day 5: Apart from moderate amount of air in the stomach, there is no intestinal edema, stool accumulation, or ileus.

e. Head CT on Day 7: No significant changes are observed, and the cause of the deteriorated consciousness is not of intracranial origin.

f. Abdominal CT on Day 7: Compared with the CT on Day 5, massive ascites is remarkable and marked edema of the small bowel is also identified.
Case 2

An 83-year-old woman with a history of hypertension and hyperlipidemia presented to the hospital complaining of headache and nausea after sustaining a head injury. Her height and weight were 145 cm and 49.0 kg, respectively. Her vital signs on arrival and throughout hospitalization are shown in Fig. 2a. Examination was notable for a GCS score of 14 (E4V4M6) and weakness of the right upper and lower limbs (3+/5 on manual muscle testing (MMT)). Head CT revealed traumatic subarachnoid hemorrhage in the right Sylvian fissure and frontal sulci as well as a small hemorrhage in the left cerebral peduncle. The patient was admitted for observation (Day 0). A few hours later, her weakness worsened to MMT 2/5. Repeat CT showed enlargement of the left cerebral peduncle hemorrhage and left subdural hygroma. Because of ongoing nausea and poor oral intake, intravenous fluids were initiated. On Day 4, her weakness improved to MMT 4+/5 but dysphagia was observed. A nasogastric tube was inserted to initiate enteral nutrition (EN) on Day 5, as bowel sounds were auscultated. The intravenous fluids were also continued. Vital signs were stable until Day 6. In the morning of Day 7, laboratory data demonstrated leukocytosis (15.0 × 10³/mL), hypernatremia (151 mmol/L), increased BUN concentration (47.7 mg/dL) and hyperglycemia (734 mg/dL) (Table 2). Acute pancreatitis was ruled out because of no abdominal pain and normal concentrations of amylase and lipase (Table 2). Insulin was added to her intravenous fluids and EN was continued. In the afternoon, the patient

<table>
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TABLE 1: Laboratory data in Case 1


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complained of abdominal pain one hour after an EN feeding had completed; she also defecated a large volume of watery stools with a small amount of blood. Thirty minutes later, her body temperature increased to 39.8°C, blood pressure fell to 96/61 mm Hg, and her level of consciousness deteriorated (GCS 5: E1V1M3). The glucose concentration had decreased to 461 mg/dL, but her hypernatremia and elevated blood urea nitrogen concentration had worsened (Table 2). Head CT showed no new lesions. Abdominal CT demonstrated dendritic hepatic portal venous gas, dilatation of the ileocecal region and marked enlargement of the ascending colon; no obvious necrosis of the small intestine was noted. Despite administration of albumin and catecholamines, the patient’s blood pressure dropped to 67/48 mm Hg. Her level of consciousness never recovered and body temperature increased further to 41.4°C. She died early the next morning (Day 8). The family consented to an autopsy, which revealed 500 mL of bloody ascites and necrosis throughout the small intestine. In the terminal portion of the ileum and the ileocecal region, intestinal wall thinning was observed. Microscopic examination findings in the small intestine are presented in Figure 2f-i. No obstruction of the mesenteric vessels was found, leading to a diagnosis of NOMI. (Fig. 2a-i)

**FIGURE 2: Case 2**

a. Vital signs over time: EN was initiated from Day 5. Soon after the diarrhea at Day 7, the patient demonstrated extremely high body temperature followed by irreversible rapid drop of blood pressure.

b. Head CT on admission: Traumatic SAH was identified at the right sylvian fissure and sulci in the right frontal, and small intraparenchymal hemorrhage in the left cerebral peduncle.

c. Head CT at 4 hours after admission: SAH in the right sylvian fissure was washed out and the SAH spread in to the right temporo-parietal sulci, and the hemorrhage in the left cerebral peduncle enlarged. Thin subdural hygroma appears at the left side.

d. Head CT at Day 7: No new lesions are recognized which can cause deterioration of consciousness.

e. Abdominal CT at Day 7: The CT demonstrated multiple dendritic hepatic portal venous gas, the ileocecal region is edematous (white circle), and the ascending colon is markedly enlarged due to gas retention (white arrows). But no obvious necrosis of the small intestine can be clearly recognized.

f. The mucosa of the intestinal tract is in a standing necrotic state (epithelial necrosis with cell denucleation, leaving the framework of the glandular ducts), with intramucosal haemorrhage and dilated blood vessels. (haematoxylin and eosin [HE], 40X)

g. The subepithelial mucosal lamina propria is oedematous, with a small neutrophilic infiltrate. (HE, 40X)

h. Vessels within the submucosa are also dilated and there is evidence of neutrophilic infiltration of the wall. (HE, 100X)

i. Small vessels within the mucosal intrinsic layer also show wall necrosis, with a hemorrhagic and edematous appearance in the surrounding area. (Masson-goldner, 100X)
### TABLE 2: Laboratory data in Case 2

**Discussion**

A critically ill state has been considered a precondition for developing NOMI [1-5]. EN may induce NOMI, but even then, it only has occurred in patients in poor general condition [6, 7]. In contrast, our two patients were not in critical condition and NOMI was not foreseeable. Furthermore, minimal abdominal symptoms before NOMI onset caused a delay in diagnosis. Even though the NOMI was fulminant, had it been diagnosed early, surgical intervention may have saved their lives.

Our patients shared many characteristics: 1) over age 80 years; 2) minor traumatic intracerebral hemorrhage; 3) constipation for a week; 4) minimal abdominal symptoms; 5) rapidly progressing leukocytosis, hyperglycemia, and hypernatremia; 6) massive diarrhea with a small amount of blood on the same day that laboratory data became abnormal; and 7) acute fever and shock developed shortly after diarrhea appeared. Old age is a risk factor for NOMI and atherosclerosis is a major contributor [8, 9].

We found no previous reports of NOMI developing after head trauma. We believe that NOMI would not have occurred in our patients had the head injuries not occurred, and that the head trauma and consequent cerebral hemorrhage indirectly resulted in NOMI. Constipation itself is not associated with mesenteric ischemia, but bloody diarrhea is [10, 11]. Our patients complained of little abdominal pain. Nonetheless, the disease rapidly progressed to such a fulminant stage that no aggressive treatment was performed. The rapid onset of leukocytosis, hyperglycemia, hypernatremia, elevated BUN concentration, and fever were clear.
signs of a clinical problem. Hyperglycemia appears to be a rare complication of NOMI and it has only been reported in one other case [12]. However, it is probably not a coincidence that our patients, who did not have a history of diabetes mellitus, demonstrated rapid elevation of serum glucose concentration. High fever and rapid drop in blood pressure after development of bloody diarrhea indicated septic shock from intestinal necrosis. At this point, surgical intervention in a patient over 80 years of age would likely have resulted in death. In retrospect, we had suspected NOMI and made the diagnosis earlier, surgery could have been performed before shock occurred. When acute pancreatitis-like signs such as leukocytosis, hyperglycemia, hypernatremia, and elevated BUN are observed in an older patient after a head injury, NOMI should be suspected in addition to acute pancreatitis. Clinicians should be aware that NOMI can occur after head trauma, which can be fatal if not diagnosed promptly.

Conclusions
Generally, NOMI is a complication in critically ill patients. However, as shown in this report, NOMI can also occur in mild head injury cases admitted for observation, and the most common cause of death due to NOMI is delayed diagnosis. When acute pancreatitis-like data are identified in a patient following head trauma, NOMI should be kept in mind.

Additional Information

Disclosures
Human subjects: All authors have confirmed that this study did not involve human participants or tissue. Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work. Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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