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Relationship between subarachnoid hemorrhage and nonocclusive mesenteric ischemia as a fatal complication: patient series

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BACKGROUND Nonocclusive mesenteric ischemia (NOMI) causes intestinal necrosis due to irreversible ischemia of the intestinal tract. The authors evaluated the incidence of NOMI in patients with subarachnoid hemorrhage (SAH) due to ruptured aneurysms, and they present the clinical characteristics and describe the outcomes to emphasize the importance of recognizing NOMI.

OBSERVATIONS Overall, 7 of 276 consecutive patients with SAH developed NOMI. Their average age was 71 years, and 5 patients were men. Hunt and Kosnik grades were as follows: grade II, 2 patients; grade III, 3 patients; grade IV, 1 patient; and grade V, 1 patient. Fisher grades were as follows: grade 1, 1 patient; grade 2, 1 patient; and grade 3, 5 patients. Three patients were treated with endovascular coiling, 3 with microsurgical clipping, and 1 with conservative management. Five patients had abdominal symptoms prior to the confirmed diagnosis of NOMI. Four patients fell into shock. Two patients required emergent laparotomy followed by second-look surgery. Four patients could be managed conservatively. The overall mortality of patients with NOMI complication was 29% (2 of 7 cases).

LESSONS NOMI had a high mortality rate. Neurosurgeons should recognize that NOMI can occur as a fatal complication after SAH.

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KEYWORDS nonocclusive mesenteric ischemia; subarachnoid hemorrhage; vasospasm; complication

Nonocclusive mesenteric ischemia (NOMI) was first reported as a complication of cardiac surgery by Ende in 1958.[1] NOMI causes intestinal necrosis due to irreversible ischemia of the intestinal tract despite the absence of organic obstruction in the mesenteric artery. NOMI has no characteristic symptoms, so the diagnosis is generally made at progression to severe disease. Therefore, NOMI is associated with a high mortality rate of ~50%–70%.[2,3] No definite diagnostic criteria for NOMI have yet been established. Suggested risk factors include age older than 70 years, low cardiac output state, sepsis, use of vasospastic drugs and diuretic therapy, digitalis, hemodialysis, major heart or abdominal surgery, and critical illnesses.[4–6] Therefore, the incidence of NOMI is expected to increase with the aging of the population.

NOMI has recently been stressed as a life-threatening complication of cardiac surgical procedures, so it should be recognized as a rare but serious complication following cardiac surgery.[6,7] In contrast, NOMI presenting as a severe complication following subarachnoid hemorrhage (SAH) is only known in 2 cases, so NOMI is still unfamiliar to most neurosurgeons.[8,9] The incidence of NOMI after SAH is likely to increase because the average age of onset of SAH is rapidly increasing. Therefore, neurosurgeons must be aware of the risk of onset of NOMI in patients with SAH, especially elderly patients with various underlying diseases. To expand the awareness of the onset of NOMI following SAH, in the present study, we analyzed all patients with SAH complicated by NOMI.

Study Description

Study Population

This retrospective, single-institution study included all patients with SAH hospitalized at the Kitasato University Hospital Emergency and Disaster

ABBREVIATIONS 3D-CT = three-dimensional computed tomography; AKA = Adamkiewicz artery; CT = computed tomography; HPVG = hepatic portal venous gas; HK = Hunt and Kosnik; IC-PComA = internal carotid–posterior communicating artery; ICU = intensive care unit; MCA = middle cerebral artery; MDCT = multidetector-row computed tomography; MIP = maximum-intensity projection; NOMI = nonocclusive mesenteric ischemia; SAH = subarachnoid hemorrhage; SMA = superior mesenteric artery.

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Medical Center over almost a 6-year period between January 2016 and October 2021. This study was approved by the Kitasato University Hospital Institutional Review Board for Human Research in Sagamihara City, Kanagawa, Japan. This retrospective observational study adopted the opt-out method of our hospital.

SAH Management

Initial admission and treatment of the patients with SAH were performed in the emergency intensive care unit (ICU). Ruptured aneurysms were commonly identified by digital subtraction angiography or 3-dimensional computed tomography (3D-CT) angiography with further treatment allocation to either endovascular coiling or surgical neck clipping, which was performed as soon as possible. Subsequently, hydrocephalus was treated with external ventricular drainage, allowing continuous monitoring of intracranial pressure. Conservative management of cerebral vasospasm included administration of cilostazol and fasudil and maintenance of normovolemia. Endovascular vasospasm treatment was initiated for refractory cerebral vasospasm. Chronic hydrocephalus was treated with ventriculoperitoneal shunt placement. Initial computed tomography (CT) was performed at admission, then additional CT was performed within 24 hours after aneurysm treatment and on day 4, day 7, and day 10 during the period of cerebral vasospasm, as well as after any clinical deterioration. The management protocols for SAH underwent no relevant changes during this study. All microsurgical clipping was performed by H.K., and all endovascular treatment was provided by D.Y.

Diagnostic Criteria of NOMI

The diagnosis and management of NOMI rely on a multidisciplinary approach involving intensivists, gastroenterologists, radiologists, surgeons, and neurosurgeons. Multidetector-row computed tomography (MDCT) and/or 3D-CT angiography may be performed if NOMI is suspected, depending on the symptoms. Findings suggestive of NOMI include absence of frank obstruction and, supported by narrowing of the origin of the superior mesenteric artery (SMA), irregularities of the intestinal branches of the SMA (beading sign, strings of sausage sign), spasm of the vascular arcades, and impaired filling of intramural vessels. The need for surgery for diagnosis or treatment is discussed with all the specialists involved.

Patients with suspected NOMI were classified as having confirmed NOMI if the findings of CT, angiography, and/or surgery confirmed the diagnosis. CT indications of NOMI include pneumatosis intestinalis defined by the presence of gas inside the bowel walls, bowel dilation, portal venous gas, aortic or mesenteric atherosclerosis, and absence or heterogeneity of contrast-induced enhancement of the bowel walls.

NOMI Management

NOMI management at our institution is described below.[10] The primary management for NOMI includes treatment of the underlying medical condition and discontinuing the pharmacological agents contributing to the low-flow state. Fluid resuscitation was initiated, and broad-spectrum antibiotics were started because of mucosal compromise and the possibility of bacterial translocation. NOMI is secondary to vasospasm rather than occlusion, so treatment is medically focused and relies upon reversing the underlying cause of the low-flow state. Reinstating circulatory volume and administration of vasodilatory agents directly into the SMA both help improve mesenteric perfusion. Catheter placement into the SMA for infusion of prostaglandin E1 (alprostadil) is the most commonly used vasodilatory agent. Papaverine is an alternative infusible medication. Patients with an excellent response to infusion of prostaglandin E (5–10 μg) are administered continuous regional arterial infusion of papaverine (30–60 mg/h). Patients with peritoneal signs require emergent laparotomy with resection of only grossly necrotic or perforated bowel. Questionably viable bowel is observed until revascularization is complete and subsequently reevaluated at a second-look laparotomy within 48 hours.

Results

Clinical Presentation of SAH

A retrospective chart review identified 452 patients treated under a definitive diagnosis of SAH, of whom 176 patients with cardiopulmonary arrest on arrival, with SAH of unknown etiology, or untreatable SAH were excluded. NOMI occurred in 7 (2.5%) of the 276 patients indicated for treatment of SAH due to ruptured aneurysm (Fig. 1). Table 1 shows the chronological events relating to patient characteristics and presentation. The 5 men and 2 women ranged in age from 56 to 86 (mean 71 ± 13) years. Hunt and Kosnik (HK) grades were as follows: grade II, 2 patients; grade III, 3 patients; grade IV, 1 patient; and grade V, 1 patient. Fisher grades were as follows: grade 1, 1 patient; grade 2, 1 patient; and grade 3, 5 patients. The aneurysm locations were as follows: anterior communicating artery, 2 cases; anterior cerebral artery, 1 case; internal carotid–posterior communicating artery (IC-PComA), 2 cases; middle cerebral artery (MCA), 1 case; and basilar artery, 1 case. Three patients were treated with endovascular coiling, 3 with microsurgical clipping, and 1 with conservative management.

Clinical Presentation of NOMI

Five patients had abdominal symptoms, such as diarrhea, abdominal bloating, vomiting, and melena, prior to the diagnosis of NOMI. The duration from SAH onset to NOMI onset was, on average, 7 (range, 5–19) days. Four patients experienced shock.

Treatment for NOMI

Patients were treated according to the clinical treatment protocols of our institution for NOMI. Four patients were treated conservatively as nonsurgical cases, and 2 patients were surgically treated.

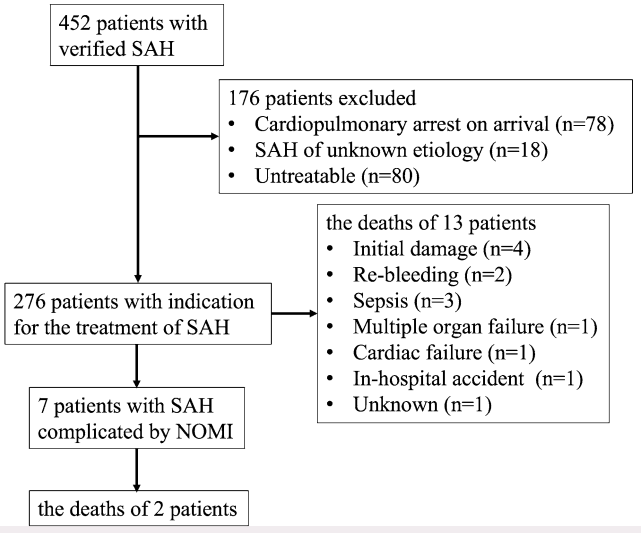

FIG. 1. Flowchart of the study population.

TABLE 1. Patient characteristics and clinical presentation

Case No.	Age (yrs)	Sex	Neuro HK Grade	Fisher Grade	Aneurysm Location	Tx Modality	No. of Days From SAH Onset to NOMI Onset	Abdominal Sxs	Shock Caused by NOMI	Tx for NOMI	Outcome
1	56	M	II	3	AComA	Clipping	7	Abdominal bloating, vomiting	–	Conservative mgmt	Survival
2	81	F	V	3	BA	Conservative mgmt	19	Vomiting	–	Conservative mgmt	Survival
3	81	M	III	3	ACA	Coil	6	None	+	Op & arterial drug infusion	Death
4	52	M	IV	3	MCA	Clipping	5	Vomiting	+	None	Death
5	66	M	III	3	AComA	Clipping	9	None	–	Conservative mgmt	Survival
6	80	F	III	2	IC-PComA	Coil	15	Diarrhea, melena	+	Conservative mgmt	Survival
7	86	M	II	1	IC-PComA	Clipping	7	Melena	+	Op & arterial drug infusion	Survival

ACA = anterior cerebral artery; AComA = anterior communicating artery; BA = basilar artery; mgmt = management; Neuro = Neurological; Op = surgery; Sx = symptom; Tx = treatment.

Survival After Onset of NOMI

Fifteen of 276 patients with indications for the treatment of SAH were not discharged from the hospital and eventually died. The overall mortality under indications for the treatment of SAH was 5.4% (15 of 276 cases). Causes of death were as follows: NOMI, 2 cases; initial damage, 4 cases; rebleeding, 2 cases; sepsis, 3 cases; multiple organ failure, 1 case; cardiac failure, 1 case; in-hospital accident, 1 case; unknown, 1 case. Two of 7 patients with NOMI had died at discharge. NOMI accounted for 13.3% of the total number of fatalities. The overall mortality of patients with NOMI was 29% (2 of 7 cases).

Illustrative Cases

Case 4

A 52-year-old male experienced sudden onset of headache and deterioration of consciousness while driving. CT revealed SAH with hematoma predominantly in the right sylvian fissure (Fisher grade 3) (Fig. 2A). His neurological status was HK grade III. CT angiography identified a saccular aneurysm of the right MCA (Fig. 2B). Therefore, we performed clipping and decompressive craniectomy indicated for space-occupying intracerebral hematoma on CT. The postoperative course was uneventful except for slight fever. However, he began vomiting on postoperative day 4 and experienced shock. Dynamic CT to identify the cause of shock showed massive hepatic portal venous gas (HPVG), a poorly enhanced region of the small intestines, and the presence of gas inside the bowel walls (Fig. 2C and D). CT detected no thrombus in the SMA or superior mesenteric vein, which indicated that the cause of ischemia was nonocclusive. These radiological findings led to a clinical diagnosis of NOMI. The patient died within a few hours due to progression of multiple organ failure.

Case 6

An 80-year-old female was transferred to our institution with SAH at 4 days after the onset of headache. Neurological examination revealed mild consciousness disturbance (HK grade III). CT demonstrated SAH in the basal cisterns, sylvian fissure, and lateral ventricle. CT angiography revealed a 4-mm left IC-PComA aneurysm. Endovascular coiling was selected to treat the aneurysm because of her advanced age and was performed with no complications. She was drowsy but could follow commands after surgery. Tube feeding was started on postoperative day 2. No abnormal neurological findings due to cerebral vasospasm were subsequently observed. During the ICU stay, she had persistent diarrhea, but her vital signs were stable. She suddenly developed melena and fell into shock on day 15. Dynamic CT showed the same findings as in Case 4, and coronary maximum-intensity projection (MIP) demonstrated narrowing of the SMA, irregularities of the intestinal branches of the SMA, and spasm of the vascular arcades (Fig. 3A). However, HPVG was alleviated by conservative treatment (rehydration and antibiotics), and coronary MIP showed radiological improvement of the narrowing of the SMA (Fig. 3B) and spasm of the vascular arcades. The patient survived this episode of NOMI and recovered.

Case 7

An 86-year-old man presenting with headache was admitted. His left eyelid showed complete ptosis, and his left pupil was mildly dilated and nonreactive to light. CT showed no apparent findings of SAH, but CT angiography revealed a 9-mm left IC-PcomA aneurysm. Clipping was performed, considering the threatened rupture of the aneurysm. Intraoperative examination discovered SAH, confirming that the left IC-PcomA aneurysm had ruptured. The patient

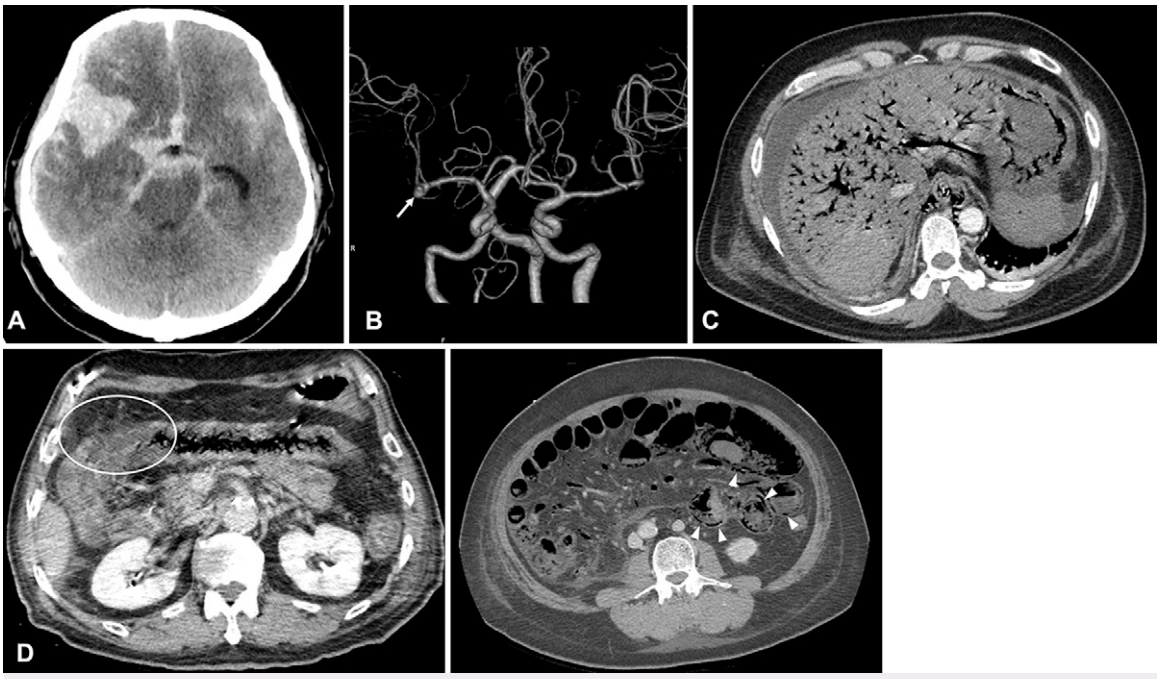

FIG. 2. Case 4. CT scan showing a predominantly right-sided SAH (**A**). 3D-CT angiogram showing a right MCA aneurysm (*white arrow*, **B**). CT scan of the abdomen showing hepatic portal venous gas (**C**). CT scans of the abdomen showing a poorly enhanced region of the small intestines (*white oval*) and the presence of gas inside the bowel walls (*white arrowheads*, **D**).

was awake, following commands, moving all extremities with full strength and had near resolution of his oculomotor nerve palsy with the left pupil 1 mm larger than the right pupil on admission to the ICU. However, he suddenly developed melena on day 7. He fell into shock, and abdominal CT was performed, which demonstrated HPVG and a poorly enhanced region suspected of intestinal ischemia, suggesting a diagnosis of NOMI. Partial intestinal resection was carried out to save the patient (Fig. 4A). Histopathological examination found various degrees of ischemic change in the bowel wall and mucosal necrosis, concordant with the clinical diagnosis of NOMI (Fig. 4B–E). Postoperative angiography of the SMA showed poor contrast enhancement in some areas of the small intestine with narrowing of the distal branches of the SMA (Fig. 4F). Continuous intraarterial infusion of papaverine was started via a catheter placed in the SMA, and SMA angiography demonstrated radiological improvement of narrowing of all SMA branches and poor blood flow (Fig. 4G). His symptoms gradually improved, and CT showed significant reduction of HPVG. He started oral intake and recovered with a modified Rankin scale score of 4.

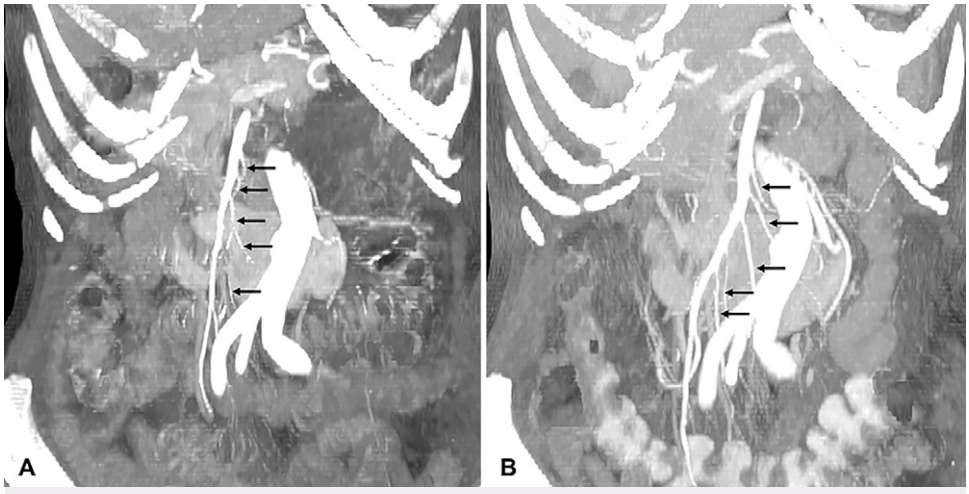

FIG. 3. Case 6. Coronary MIP showing narrowing of the SMA, irregularities of the intestinal branches of the SMA, and spasm of the vascular arcades (**A**). Coronary MIP showing radiological improvement of the narrowing of the SMA (*black arrows*) and spasm of the vascular arcades (**B**).

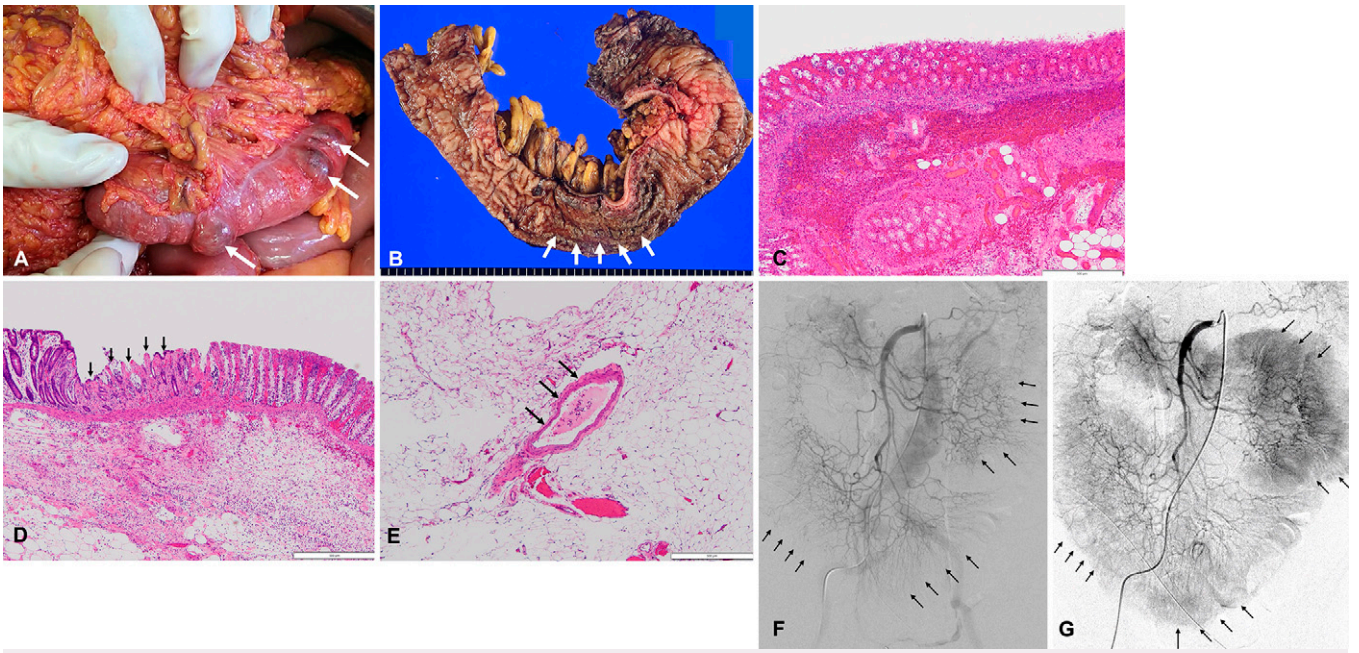

FIG. 4. Case 7. Intraoperative findings at the laparotomy. Edema and partial necrosis of the bowel wall is present (*white arrows*, **A**). The resected small intestine shows hemorrhagic necrosis (*white arrows*, **B**). Pathological examination revealed infiltration of many inflammatory cells into all layers and advanced edema of the submucosa on the luminal surface (**C**; hematoxylin and eosin, original magnification ×40). Pathological examination of the intestinal specimen showed ghostlike appearance of crypt (*black arrows*, **D**) characterized in the borderline region by destroyed mucosal epithelium and necrosis (hematoxylin and eosin, original magnification ×40). No thrombus is observed in the mesenteric vessels (*black arrows*, **E**; hematoxylin and eosin, original magnification ×40). Selective SMA angiogram showing narrowing of all SMA branches and poor blood flow (*black arrows*, **F**). SMA angiogram showing radiological improvement of severe NOMI after intramesenteric papaverine treatment (*black arrows*, **G**).

Discussion

Observations

Only 2 cases of the complication of NOMI following SAH have been reported.[8,9] Our study clarified that NOMI is a life-threatening complication in patients with SAH using a relatively large case series in a single institute. NOMI occurred in 2.5% of all patients with indications for the treatment of SAH, which may not seem so high, but NOMI caused 13.3% of all fatalities, so the mortality rate was definitely not low.

Diagnosis of NOMI

Previously, angiography was the gold standard for the diagnosis of NOMI. However, MDCT is an equally useful modality based on analysis of the diameter of the SMA on multiplanar reconstructed images.[11] MDCT improves the diagnostic accuracy, but the early onset of NOMI is characterized by the absence of specific clinical symptoms, which delays the treatment.[12] The diagnosis is challenging because patients with SAH are frequently ventilated, sedated, and have impaired consciousness and so are unable to verbally communicate. However, more than half of the patients in our series experienced some type of digestive symptoms, such as abdominal distension, melena, vomiting, and diarrhea. Over the course of the SAH treatment, during the period of cerebral vasospasm in particular, such digestive symptoms may suggest progression of intestinal ischemia, which should indicate the possibility of NOMI and the need for detailed work-up. Therefore, awareness of the seriousness of NOMI among neurosurgeons could potentially achieve early diagnosis.

Pathogenic Mechanism of NOMI

The main causative mechanism of NOMI is thought to occur as follows. Systemic hypoperfusion will probably result in sacrifice of the blood flow to organs within the abdomen and the lower extremities to maintain adequate blood flow to the vital organs, such as the brain and heart. Peripheral vasospasm due to excessive reaction of humoral factors, such as the sympathetic nerves, vasopressin, and angiotensin, is associated with decreased cardiac output and extremely low circulating plasma volume. NOMI is caused by extremely low and imbalanced intestinal perfusion.[12] More than half of our patients fell into shock with hypotension and were later diagnosed with NOMI. Three cases of mesenteric ischemia after endovascular coiling of ruptured cerebral aneurysms suggested that catheter-related vascular injury is a potential etiology.[9] However, our series included cases of NOMI after clipping and conservative management, so the causative mechanism of NOMI is not as simple a problem as only catheter-related vascular injury.

Correlation Between SAH and NOMI

On first impression, NOMI and SAH appear to be unrelated. However, animal experiments suggest that Onuf's nucleus S2 roots complex degeneration is important in mesenteric artery vasospasm and the development of intestinal ischemic mucosal changes following SAH.[13] Onuf's nucleus sends parasympathetic impulses to the distal colon, pelvic organs, and urethral and anal sphincters.[14] Blood flow to Onuf's nucleus and the inferior spinal nerves is provided by the Adamkiewicz artery (AKA). AKA vasospasm due to SAH causes ischemia of the inferior spinal cord and Onuf's nucleus.[15] Intestinal ischemic disorders

related to mesenteric artery spasm are thought to be causally related to spinal cord and Onuf's nucleus ischemia due to AKA spasm. Similarly, animal experiments showed that Onuf's nucleus–pudendal nerve ganglia complex degeneration secondary to AKA vasospasm may cause urinary retention after spinal SAH.[16] AKA vasospasm due to SAH has been clinically demonstrated.[17]

These studies might indicate a previously unknown mechanism of NOMI peculiar to SAH. Cerebral vasospasm usually occurs 3 to 14 days after aneurysm rupture, peaking on days 7 to 9 after bleeding. Cerebral vasospasm led to delayed cerebral ischemia and caused neurological deficits in patients with SAH. The condition of 2 of 3 previous patients deteriorated in the first 48 hours postoperatively, whereas the third patient died 12 days after hemorrhage.[9] Our patients experienced NOMI between 5 and 19 days (mean 9.7 days) after SAH. This timing is almost simultaneous with the period of cerebral vasospasm, which we do not think is coincidental.

Treatment and Prognosis for NOMI

The algorithm of the guidelines on intestinal ischemia produced by the American Gastroenterological Association in 2000 suggests that abdominal angiography and arterial infusion therapy are gold standards for the diagnosis and management of NOMI.[18] Recommendations for the treatment of acute nonocclusive intestinal ischemia are given in the guidelines for the management of patients with peripheral arterial disease produced by the American College of Cardiology and American Heart Association in 2006 as follows: (1) Treatment of the underlying shock state is the most important initial step in the treatment of nonocclusive intestinal ischemia (class I: level of evidence C); (2) laparotomy and resection of nonviable bowel are indicated in patients with nonocclusive intestinal ischemia who have persistent symptoms despite treatment (class I: level of evidence B); and (3) transcatheter administration of vasodilator medications into the area of vasospasm is indicated in patients with nonocclusive intestinal ischemia who do not respond to systemic supportive treatment and in patients with intestinal ischemia due to cocaine or ergot poisoning (class IIa: level of evidence B).[19] On the basis of these guidelines, our management for NOMI incorporates a certain level of treatment. NOMI is a disease characterized by high morbidity and low survival rates.

In our study, the overall mortality rate for NOMI was lower at 29% of patients with NOMI. Our institution has nearly a decade of excellent experience with the treatment of NOMI, and acute-phase management of SAH has been central in the training of intensivists involved in the treatment of NOMI.[10] Since learning the hard clinical lesson that our patients with SAH may die of complications with NOMI, we will pay more attention to abdominal symptoms and abdominal imaging to achieve early detection of this disease.[8] However, the fact that NOMI is a life-threatening disease remains constant. If the timing of the appearance of NOMI after SAH is understood and the risk factors and onset mechanism are identified, the accuracy of early diagnosis, as well as the effectiveness of early treatments and disease prevention, are expected to greatly improve.

Limitations of This Study

Our study has some limitations. First, this single-center, retrospective study has the potential for unavoidable bias in the incidence and mortality rate of NOMI in patients with SAH. Second, the protocol for this complication remains unclear. The overlap of occurrence time suggests some causal relationship between NOMI and cerebral vasospasm with SAH. However, we do not have the data to prove such relationships. Our future challenge is to clarify whether the occurrence of NOMI is associated with cerebral vasospasm. Therefore, we are planning a prospective study.

Lessons

We report our experience with 7 cases of NOMI that developed after SAH. NOMI has a high mortality rate, so all neurosurgeons must recognize NOMI as a fatal complication in patients with SAH. All neurosurgeons should be aware of NOMI as the cause of poor physical condition after treatment of SAH. If patients with SAH present with abdominal symptoms, abdominal imaging examination and specialist consultation should be obtained.

References

1. Ende N. Infarction of the bowel in cardiac failure. *N Engl J Med.* 1958;258(18):879–881.
2. Suzuki S, Kondo H, Furukawa A, et al. Prognostic factors of preoperative examinations for non-occlusive mesenteric ischemia: a multicenter retrospective project study conducted by the Japanese Society for Abdominal Emergency Medicine. *World J Surg.* 2020; 44(11):3687–3694.
3. Mitsuyoshi A, Obama K, Shinkura N, Ito T, Zaima M. Survival in nonocclusive mesenteric ischemia: early diagnosis by multidetector row computed tomography and early treatment with continuous intravenous high-dose prostaglandin E_1. *Ann Surg.* 2007;246(2): 229–235.
4. Copin P, Zins M, Nuzzo A, et al. Acute mesenteric ischemia: a critical role for the radiologist. *Diagn Interv Imaging.* 2018;99(3): 123–134.
5. Gnanapandithan K, Feuerstadt P. Review article: mesenteric ischemia. *Curr Gastroenterol Rep.* 2020;22(4):17.
6. Groesdonk HV, Klingele M, Schlempp S, et al. Risk factors for nonocclusive mesenteric ischemia after elective cardiac surgery. *J Thorac Cardiovasc Surg.* 2013;145(6):1603–1610.
7. Klotz S, Vestring T, Rötker J, Schmidt C, Scheld HH, Schmid C. Diagnosis and treatment of nonocclusive mesenteric ischemia after open heart surgery. *Ann Thorac Surg.* 2001;72(5):1583–1586.
8. Koizumi H, Yamamoto D, Hanajima T, et al. A case of non-occlusive mesenteric ischemia after neuroendovascular treatment of aneurysmal subarachnoid hemorrhage. Article in Japanese. *Currently Pract Neurosurg.* 2019;29(10):1110–1115.
9. Kamel MH, Murphy M, Kamel MH. Mesenteric ischaemia after endovascular coiling of ruptured cerebral aneurysms. *Acta Neurochir (Wien).* 2008;150(4):367–370.
10. Kataoka Y, Shimada K, Kashimi F, et al. A study of the diagnosis and treatment of nonocclusive mesenteric ischemia (NOMI) with special reference to the effectiveness of arterial infusion therapy. Article in Japanese. *J Abdom Emerg Med.* 2011;31(7):1015–1019.
11. Woodhams R, Nishimaki H, Fujii K, Kakita S, Hayakawa K. Usefulness of multidetector-row CT (MDCT) for the diagnosis of non-occlusive mesenteric ischemia (NOMI): assessment of morphology and diameter of the superior mesenteric artery (SMA) on multiplanar reconstructed (MPR) images. *Eur J Radiol.* 2010;76(1): 96–102.
12. Trompeter M, Brazda T, Remy CT, Vestring T, Reimer P. Non-occlusive mesenteric ischemia: etiology, diagnosis, and interventional therapy. *Eur Radiol.* 2002;12(5):1179–1187.
13. Karadeniz E, Caglar O, Firinci B, et al. Predeterminative role of Onuf's nucleus ischemia on mesenteric artery vasospasm in spinal subarachnoid hemorrhage: a preliminary experimental study. *Asian J Surg.* 2019;42(8):797–804.
14. Holstege G, Griffiths D, de Wall H, Dalm E. Anatomical and physiological observations on supraspinal control of bladder and urethral sphincter muscles in the cat. *J Comp Neurol.* 1986;250(4):449–461.

15. Kieffer E, Fukui S, Chiras J, Koskas F, Bahnini A, Cormier E. Spinal cord arteriography: a safe adjunct before descending thoracic or thoracoabdominal aortic aneurysmectomy. *J Vasc Surg.* 2002;35(2):262–268.
16. Yolas C, Kanat A, Aydin MD, et al. The important liaison between Onuf nucleus-pudendal nerve ganglia complex degeneration and urinary retention in spinal subarachnoid hemorrhage: an experimental study. *World Neurosurg.* 2016;89:208–214.
17. Miyoshi Y, Kondo A, Yasuhara T, et al. Vasospasm of Adamkiewicz artery after subarachnoid hemorrhage. Article in Japanese. *Brain Nerve.* 2011;63(2):182–183.
18. American Gastroenterological Association. American Gastroenterological Association medical position statement: guidelines on intestinal ischemia. *Gastroenterology.* 2000;118(5):951–953.
19. Hirsch AT, Haskal ZJ, Hertzer NR, et al. ACC/AHA Guidelines for the Management of Patients with Peripheral Arterial Disease (lower extremity, renal, mesenteric, and abdominal aortic): a collaborative report from the American Associations for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (writing committee to develop guidelines for the management of patients with peripheral arterial disease)—summary of recommendations. *J Vasc Interv Radiol.* 2006;17(9):1383–1398.

Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Koizumi, Kataoka, Kumabe. Acquisition of data: Koizumi, Yamamoto, Maruhashi. Analysis and interpretation of data: Koizumi, Maruhashi, Kataoka. Drafting the article: Koizumi. Critically revising the article: Maruhashi, Kumabe. Reviewed submitted version of manuscript: Maruhashi, Kumabe. Administrative/technical/material support: Yamamoto, Maruhashi, Inukai. Study supervision: Asari, Kumabe.

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