A Case of Superior Mesenteric Artery Occlusion Caused by Delayed Administration of Anticoagulants in a Patient with Nonvalvular Atrial Fibrillation

Kazuhide TAKEYAMA[*1], Masaaki MIURA[*2], Keiko NAKAYAMA[*3] and Toshiyasu SUZUKI[*4]

[*1] *Department of Anesthesiology, Tokai University School of Medicine, Tokyo Hospital*
[*2] *Hiratsuka City Hospital*
[*3] *Hadano Red Cross Hospital*
[*4] *Department of Anesthesiology, Tokai University School of Medicine*

(Received August 19, 2015; Accepted November 25, 2015)

A 86-year-old female with nonvalvular atrial fibrillation (NOVAF) who did not receiveprophylactic anticoagulant treatment visited our hospital because of gastrointestinal symptoms. At first, acute gastroenteritis was suspected, but later she developed ileusand she was diagnosed with superior mesenteric artery occlusion (SMAO). We successfully performed the anesthetic management of this patient and subtotal resection of the small intestine was performed. Heparin was initiated after surgery, but she developed cerebral infarction later, and finally she died due to infection and anemiacaused by melena. Although this patient was at high risk of thrombosis, she did not receive anticoagulant treatment. It might result in developing SMAO, and once SMAO occurred, thrombosis recurred even on anticoagulant treatment. This case suggested the importance of primary prevention of thrombosis in patients with NVAF.

Key words: Nonvalvular atrial fibrillation, Superior mesenteric artery occlusion, Anticoagulants

INTRODUCTION

Thrombosis is a serious complication in patients with NVAF. Prophylactic Anticoagulants (novel oral anticoagulant or warfarin [WF]) are recommended in Patients with NVAF, whose CHADs scores are 2 or higher [1, 2] but some of patients with NVAF do not receive anticoagulants because of a patients' or physicians' choice. Here we report a patient with NVAF, who developed superior mesenteric artery occlusion (SMAO) possibly as a result of the developed administration of anticoagulants.

CASE REPORT

The patient was an 86-year-old woman, who was 150 cm in height and 45 kg in weight. Her previous medical history included sinus node dysfunction (for which she had a permanent pacemaker implanted), hypertension, diabetes mellitus, and stroke (for which she was taking aspirin). On October 2014, she was referred to our hospital owing to a diagnosis of atrial fibrillation with rapid ventricular response(Fig. 1: Left). On examination, 12-lead electrocardiography displayed sinus rhythm (Fig. 1: Right) and Holter electrocardiography was performed. Seven days later, she visited our hospital complaining of palpitations and gastrointestinal symptoms, such as stomach ache, diarrhea, and vomiting. She was diagnosed as having acute gastroenteritis and went home. Next day, she developed abdominal tenderness and revisited our hospital. Computerized tomography (CT) scanning of her abdomen displayed intestinal dilatation and air-fluid level (Fig. 2: Left). She was diagnosed as having ileus and was immediately admitted to hospital. Her C-reactive protein (CRP) was 23.01 mg/ml and her serum creatinine (Cr) was 1.63 mg/dl. Her symptoms did not improve even after inserting ileus tube, and 3 days later, she received emergency surgery for intestinal strangulation. Laboratory findings included decreased renal function (Cr = 2.30 mg/dl), decreased blood coagulation efficiency (APTT = 57 msec), and a very strong inflammatory reaction (CRP = 45 mg/L). Arterial blood gas analysis demonstrated overt acidosis (pH = 7.4111/ PaCO2 = 16.8 mmHg/PaO2 = 81.1 mmHg/BE = -11.7). Transthoracic echocardiography (TTE) displayed spontaneous echo contrast in the left atrium (Fig. 2: Right). <The process of anesthesia> The patient had NVAF from the time of entry into the operating room. We assumed that her circulating blood volume was insufficient, and gave her administration of colloidal solution (5% albumin: 250 ml and Hespander fluid: 500 ml). Anesthesia was induced by an intravenous injection of propofol (50 mg), fentanyl (50 μg), and rocuronium (50 mg) under oxygen administration, followed by tracheal intubation. The patient's blood pressure decreased after the intubation, which was dealt with by the continuous intravenous administration of noradrenaline, as well as phenylephrine administration as appropriate. Anesthesia was maintained using oxygen (2 L/ min), air (2 L/min), and the continuous intravenous

Kazuhide TAKEYAMA, Department of Anesthesiology, Tokai University School of Medicine, Tokyo Hospital, 1-2-5 Yoyogi, Shibuya-ku, Tokyo 151-0053, Japan Tel: +81-3-3370-2321 Fax: +81-3-3379-1955 E-mail: takeyama@is.icc.u-tokai.ac.jp

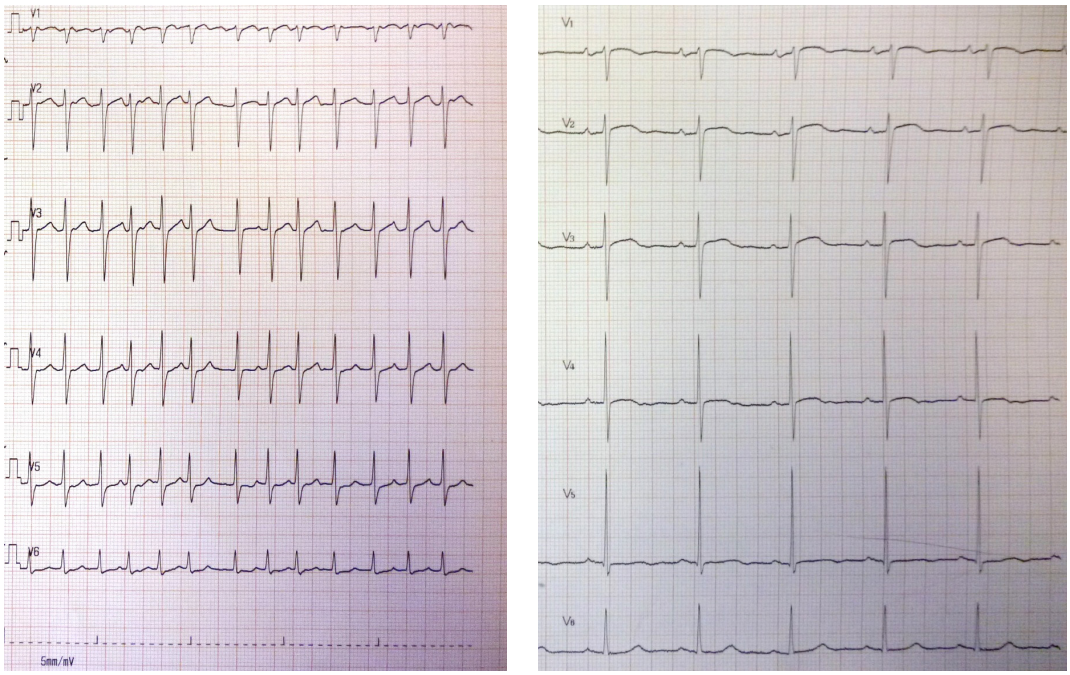


Fig. 1 Left: atrial fibrillation with tachycardia is displayed on echocardiography.
Right: sinus rhythm is displayed on echocardiography.

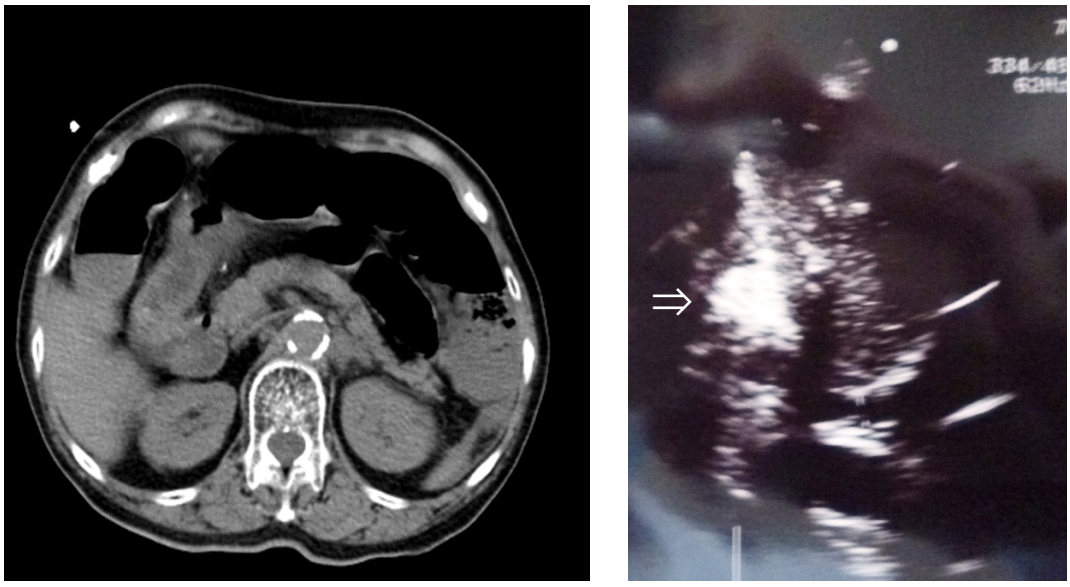


Fig. 2 Left: air-fluid level is displayed on abdominal CT.
Right: spontaneous echo contrast (arrow: ⇒) is displayed in the left atrium on transthoracic echocardiography.

administration of remifentanil (0.1–0.2 μg/kg/min). The NVAF continued even after transfusion of the colloidal solution, and hence β-blockers and calcium antagonists were administered (Fig. 3). <The process of operation> Intraoperative findings included necrosis in a wide area of the small intestine. There was no evidence of physical strangulation, and from the findings of the spontaneous echo contrast in the left atrium, the patient was diagnosed with SMAO caused by a left atrial thrombus, and subtotal resection of the small intestine was performed (Fig. 4: Left & Right). After the surgery, the patient was taken back to the ICU with continued intubation. In the ICU, anticoagulation therapy (heparin 10,000 units/day) was started. APTT at the start of heparin administration was 56 sec. On November 2014, the patient complained of weakness of the left side of the body, and a head CT lead to a diagnosis of cerebral infarction in the left parietal region (Fig. 5). On December 2014, her swallowing function recovered, and hence oral administration of WF (2 mg/day) was started. Three days later, melena was observed and hence the administration of WF and heparin was discontinued. Four days later, heparin administration was restarted; however, the patient developed complications of anemia and infection, and died on 22 days later.

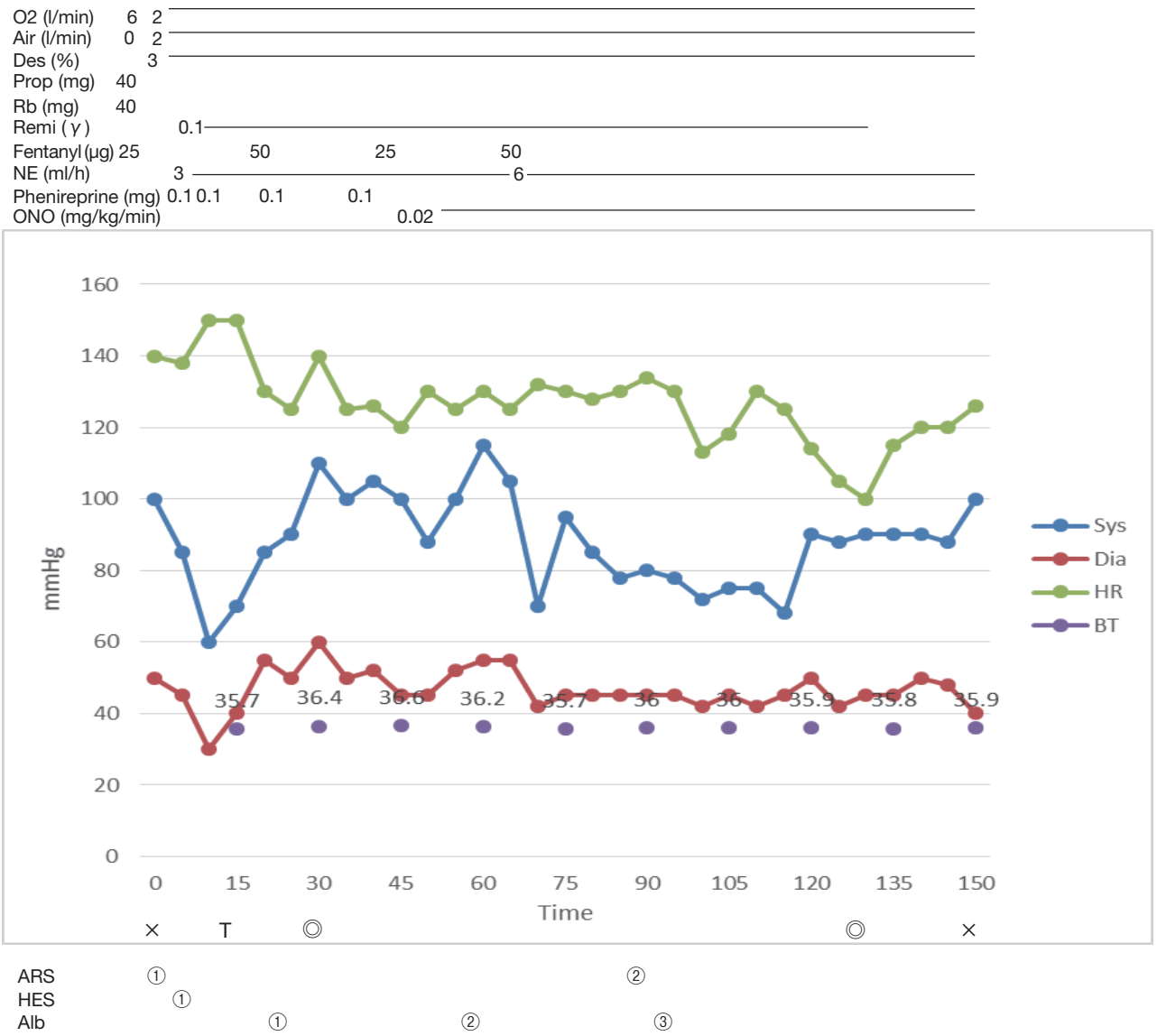
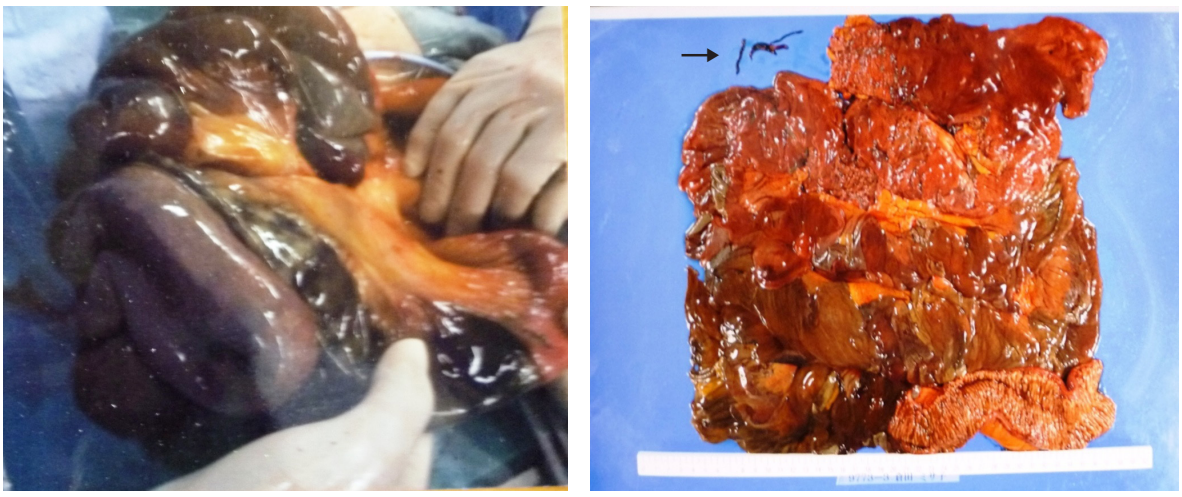


Fig. 3 Course of anesthesia.



Left Right

Fig. 4 Photographs demonstrating a necrotic intestinal tract (left) and thrombosis (right, arrow: →).

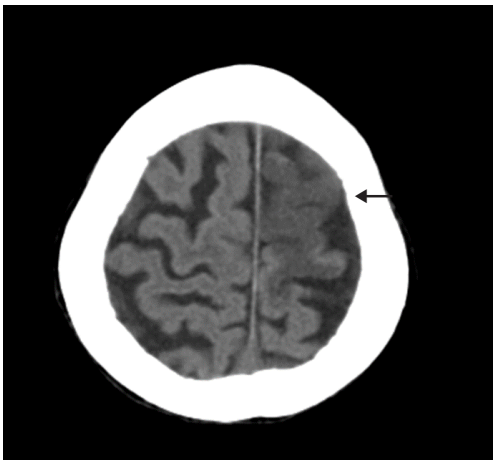


Fig. 5 A Low density area (arrow: ←) in the left parietal lobe is displayed on head CT.

DISCUSSION

Acute upper mesenteric artery occlusion (SMAO) is a serious type of acute abdomen,resulting in a low survival rate. Abdominal pain caused by SMAO is generally considered to be a severe periumbilical pain, which is followed a few days later by abdominal bloating and signs of peritoneal irritation. Our patient had very slight abdominal pain, and signs of peritoneal irritation were not observed. Because our patient had DM, it is possible that she had reduced sensory nerve function, and that the sense of pain was not transmitted efficiently. Although contrast-enhanced CT is considered to be effective for the diagnosis of SMAO, because our patient had a renal function disorder, contrast-enhanced CT could not be performed, and this was thought to be a reason why the patient's diagnosis was delayed. SMAO often occurs in elderly patients, who often have complications of the cardiovascular system as an underlying disease. According to a report by Matsui *et al.*, atrial fibrillation was the most common underlying complication [3]. This present case was also an elderly patient who had atrial fibrillation. Our patient had decreased cardiac output due to atrial fibrillation, acidosis due to intestinal ischemia, which was further accompanied by dehydration, and these symptoms made circulatory management during anesthesia very difficult. Atrial fibrillation in this patient was NVAF. The CHADS2 score has been recommended for the evaluation of the risk of cerebral infarction in NVAF patients [1, 2]. The CHADS2 score is an acronym of the 5 factors that are evaluated, namely, congestive heart failure (1 point), hypertension (1 point), age ≥ 75 years (1 point), diabetes mellitus (1 point), and stroke/transient ischemic attack (TIA) (2 points) [4, 5]. When a patient's total CHADS2 score is greater than 2 points, the administration of NOACs or WF is recommended. The following 3 factors were thought to be responsible for causing SMAO in our patient: 1) being at high risk of thrombosis (an elderly patient with a pacemaker), 2) no administration of anticoagulants, and 3) being in a dehydrated state owing to diarrhea and vomiting. For the 1), Elderly patients with implanted pacemakers as a general rule require anticoagulant therapy. For the 2), On her first visit to our outpatient clinic, our patient had a CHADS2 score of 5 points (hypertension, age ≥ 75 years, diabetes mellitus, and stroke/TIA). However, the patient showed sinus rhythm and hence Holter electrocardiography was given priority, and anticoagulants were not administered. Holter electrocardiography requires 10 days for analysis, and hence thrombus formation is thought to have occurred during this time. For the 3), elderly patients become easily dehydrated. When a patient with atrial fibrillation becomes dehydrated, the decreased circulation due to thickening of the blood leads to a high risk of developing left atrial thrombus. Therefore, we concluded that the patient should have been admitted when she developed diarrhea and vomiting, to provide the appropriate transfusions. Taken together, this patient was thought to have developed SMAO as a result of dehydration, in addition to the absence of the administration of anticoagulants. Our patient twice developed cardiogenic embolism (SMAO and cerebral infarction). The patient developed a cerebral infarction even though heparin was being administered after the SMAO had occurred, demonstrating the importance of preventing thrombosis. Therefore, on preoperative examination of patients with NVAF, it is important for anesthesiologists to pay close attention to the CHADS2 scores, as well as whether anticoagulants have been administered.

REFERENCES

1) Gage BF, Waterman AD, Shannon W, *et al.*: Validation of clinical classification schemes for predicting stroke: results from the National Registry of Atrial Fibrilation. JAMA 2001; 285: 2864–2870.
2) Inoue H, Nozawa T, Hirai T, *et al.*: Accumulation of risk factors increases risk of thromboembolic events in patients with nonvalubler atrial fibrillation. Circ J 2006; 70: 651–656.
3) Matsui M. A clinical study of acute superior mesenteric arterial occlusion. Masui 2007; 56: 1165–1167.
4) EAST (European Atrial Fibrillation Trial) Study Group. Secondary prevention in non-rheumatic atrial fibrillation after transient ischaemic attack or minor stroke. Lancet 1993; 342: 1255–1262.
5) Stroke Prevention in Atrial Fibrillation III randomized clinical trial investigators. Adjusted-dose warfarin versus low-intensity, fixed-dose warfarin plus aspirin for high-risk patients with atrial fibrillation: Stroke Prevention in Atrial Fibrillation III randomized clinical trial. Lancet 1996; 348: 633–638.