

**Acute type A aortic dissection mimicking hemorrhagic stroke**Youichi Yanagawa<sup>1</sup>, Kazuhiko Omori<sup>1</sup>, Yumi Kando<sup>2</sup>, Soichiro Kitamura<sup>2</sup><sup>1</sup>Department of Acute Critical Care Medicine, Shizuoka Hospital, Juntendo University, Japan<sup>2</sup>Numazu City Hospital, Japan**\*Corresponding author**

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**Abstract:** An 87-year-old female became comatose after complaining of severe headache. Upon arrival, her Glasgow Coma Scale was a sum score of 6, with a systolic blood pressure of 60 mmHg and heart rate of 120 beats per minute. She had isocoric non-reactive pupils 3 mm in diameter. Other her results of a physical examination were negative. Although plain head CT showed no intracranial hemorrhage, enhanced trunk CT disclosed Stanford A type aortic dissection (AD) with cardiac tamponade without expansion of dissection to either or both the carotid or vertebral artery. After immediate pericardiocentesis, she achieved stable circulation and was thereafter transferred to another hospital for emergency operation. Headache without chest or back pain as the initial manifestation of acute AD is extremely rare, and examination of the literature revealed three previous cases with the following patterns of presentation: 1) the headache is referred pain from the AD, 2) the expansion of dissection to either or both the carotid or vertebral artery from the AD causes a headache, and 3) the headache is a complication of hemorrhagic stroke and the AD due to hypertension. Atypical cases often lead to misdiagnosis and poor outcome. One point to note in detecting AD with atypical presentation is that patients presenting with headache due to AD may have hypotension, which is unusual in standard stroke cases. Given the high morbidity and mortality after misdiagnosis of the AD, patients with headache and shock should be evaluated by enhanced truncal computed tomography.**Keywords:** aortic dissection; hemorrhagic stroke; diagnosis

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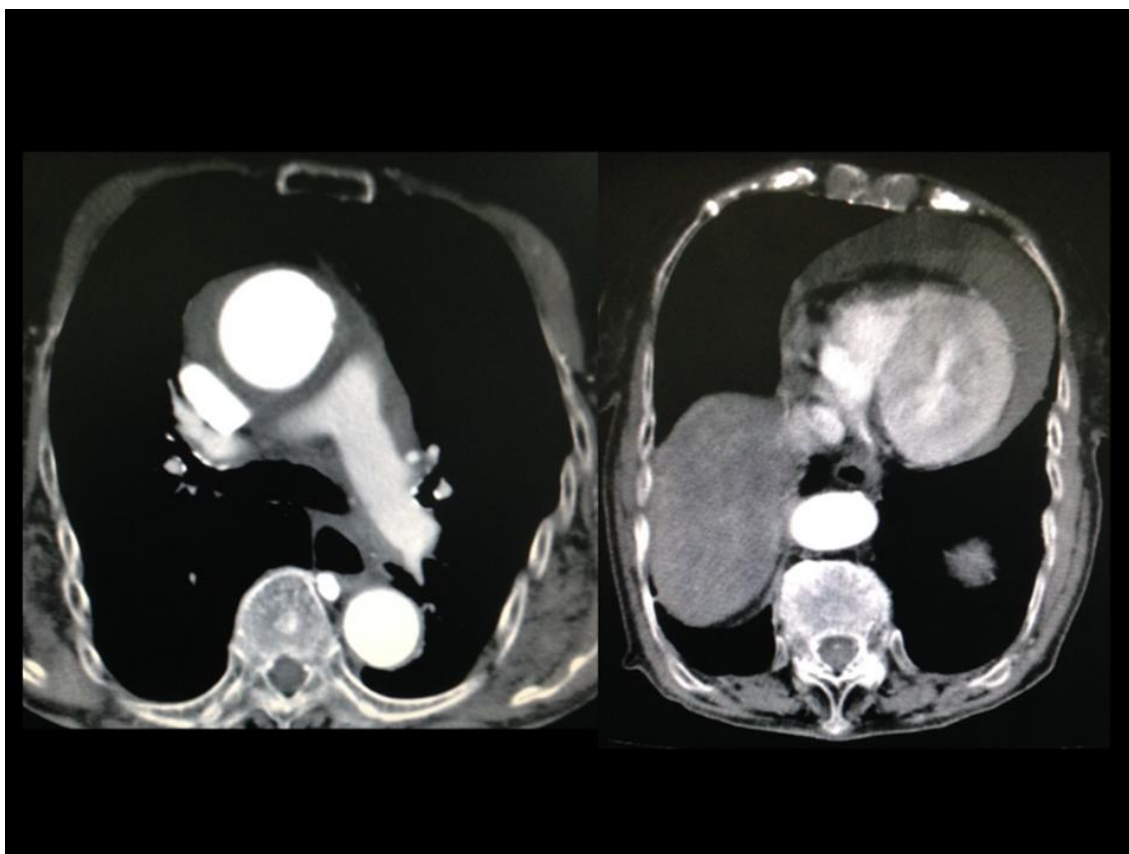
**INTRODUCTION**

Patients with aortic dissection (AD) present with a variety of complaints and symptoms [1-3], with major complaints being severe chest and back pain, which can shift with the progression of AD. However, AD can be painless and lead to hoarseness, heart failure, syncope, stroke, paraplegia, anuria, or sudden death [3]. AD can also induce toothache and left shoulder pain without typical chest or back pain [1]. We herein present a patient with acute aortic dissection type A mimicking hemorrhagic stroke, whose initial complaint was headache.

**CASE PRESENTATION**

An 87-year-old female became comatose after complaining of severe headache. She had a history of kyphosis and osteoarthritis in her knee. When emergency technicians reached her, she was in deep coma state and was transferred to Numazu City Hospital. Upon arrival, her Glasgow Coma Scale was a sum score of 6, with a systolic blood pressure of 60 mmHg and heart rate of 120 beats per minute. She had isocoric non-reactive pupils 3 mm in diameter. Other her results of a physical examination were negative. Arterial blood gas analysis (O<sub>2</sub> 10 L/minute) findings

were pH: 7.36, PCO<sub>2</sub>: 21.4 mmHg, PO<sub>2</sub>: 278.5 mmHg, HCO<sub>3</sub><sup>-</sup>: 11.9 mmol/l, and base excess: -13.5 mmol/l. A chest roentgen revealed only cardiomegaly, and her electrocardiogram was negative. A complete blood cell count showed a white blood cell level of 4,900/mm<sup>3</sup>, a hemoglobin level of 12.3 g/dl, and a platelet level of 22.7 × 10<sup>4</sup>/mm<sup>3</sup>. Serum biochemical analysis demonstrated an aspartate aminotransferase level of 176 international units (IU)/L, alanine aminotransferase level of 94 IU/L, blood urea nitrogen level of 14.9 mg/dl, creatinine level of 0.52 mg/dl, creatine phosphokinase level of 72 IU/L, sodium level of 140 mEq/L, potassium level of 4.1 mEq/L, chloride level of 104 mEq/L, CRP level of 0.1 mg/dl, international normalized ratio of prothrombin time of 0.92, activated partial thromboplastin time of 29.4 sec, D-dimer 5.4 µg/mL, and negative findings for troponin T. Although plain head CT showed no intracranial hemorrhage, enhanced trunk CT disclosed Stanford A type aortic dissection at the ascending aorta with cardiac tamponade without expansion of dissection to either or both the carotid or vertebral artery (Figure 1). After immediate pericardiocentesis, she achieved stable circulation and was thereafter transferred to another hospital for emergency operation.



**Fig 1: Enhanced trunk CT on arrival. The CT shows Stanford type A aortic dissection at the ascending aorta with cardiac tamponade**

## DISCUSSION

Headache without chest or back pain as the initial manifestation of acute aortic dissection type A is extremely rare, and examination of the literature revealed three previous cases with the following patterns of presentation: 1) the headache is referred pain from the AD [4-6], 2) the expansion of dissection to either or both the carotid or vertebral artery from the AD causes a headache [7,8], and 3) the headache is a complication of hemorrhagic stroke and the AD due to hypertension [9]. The 1) fulfills this case.

Myocardial ischemia also rarely causes headache. While cardiac cephalalgia and headache angina are recognized phenomena, the pathophysiological mechanism remains unclear [10]. The cervical ganglia share communicating branches with spinal cervical nerves and all sympathetic cardiac nerves [11]. The branches of the cervical ganglia also include the internal and external carotid nerves, sympathetic trunk, and branches communicating with the glossopharyngeal nerve, pharyngeal branch of the vagus nerve, and hypoglossal nerve [12]. The presence of these communications between the aorta and somatic nerves via the autonomic nervous system offers a potential explanation of the mechanism underlying the referred pain observed in this case. Furthermore, previous studies have hypothesized that chemical mediators, such as bradykinin, serotonin and histamine,

are hypothesized to induce pain in the shoulders, arms, neck, and in the present case, in the head [10].

Atypical cases often lead to misdiagnosis and poor outcome. Indeed, Matsumoto et al. reported fatal cases of acute AD where the initial complaints were headache [13]. One point to note in detecting AD with atypical presentation is that patients presenting with headache due to AD may have hypotension, which is unusual in standard stroke cases. Given the high morbidity and mortality after misdiagnosis of the AD, patients with headache and shock should be evaluated by enhanced truncal computed tomography.

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