

NAOSITE: Nagasaki University's Academic Output SITE



| | |
|-------------|---|
| Title | Thrombosed Stanford Type A Dissection of the Aorta in an Elderly Patient Following a Fall |
| Author(s) | Kawano, Hiroaki; Ishizaki, Masahiko; Koide, Yuji; Toda, Genji; Yano, Katsusuke |
| Citation | Internal Medicine, vol.44(6), pp.603-606; 2005 |
| Issue Date | 2005-06 |
| URL | http://hdl.handle.net/10069/22551 |
| Description | |
| Rights | Copyright (c) 2005 (社)日本内科学会 |
| Version | publisher |

Thrombosed Stanford Type A Dissection of the Aorta in an Elderly Patient Following a Fall

Hiroaki KAWANO, Masahiko ISHIZAKI, Yuji KOIDE, Genji TODA and Katsusuke YANO

Abstract

Physical or mental exertion is an important antecedent to dissection. A fall is one of the causes of hip fracture in the elderly population. We report the case of a 78-year-old woman who was diagnosed to have a thrombosed aortic dissection after a fall. We wish to emphasize with this case report that aortic dissection should be considered in the differential diagnosis of patients who complain of chest discomfort after a tumbling over. (Internal Medicine 44: 603–606, 2005)

Key words: aortic dissection, thrombosis, elder patient, hip fracture

Introduction

Acute aortic dissection is an uncommon, but potentially catastrophic condition (1). Previous reports have indicated that physical and/or mental exertion is an important antecedent to dissection (2–4). Hip fractures (fracture of the proximal femur) in the elderly represent a global issue associated with significant mortality and morbidity (5–8), and are often caused by a fall including mild fall. However, it is unknown whether or not a fall can induce acute aortic dissection in elderly people. We report the case of a patient diagnosed to have developed a thrombosed-type aortic dissection, in addition to a hip fracture, following a fall.

Case Report

A 78-year-old woman with recurrence of an oral cavity tumor and lymph node metastasis was admitted to our hospital on October 6, 2003, for re-operation. The operation was performed on October 30, 2003. While her condition im-

proved after the operation, except for mild chylorrhea from the neck wound, she fell at 5 AM on November 14, 2003.

Immediately after the fall, she started to complain of pain in the left leg, and a plain radiograph of the hip showed fracture of the left femur (Fig. 1). She also complained of mild chest discomfort and dyspnea about 3 hours after the fall, however, these symptoms abated within an hour. An electrocardiogram was normal. Surgery was scheduled for the hip fracture, and chest roentgenography and computed tomography (CT) were performed as part of the preoperative evaluation because the patient continued to have chylorrhea from the neck wound after resection of the cervical lymph nodes. The chest radiograph did not reveal any mediastinal widening, however, splitting of calcification of the aortic arch was seen, which had not been detected in the chest roentgenogram of the day before (Fig. 2). A plain chest CT showed a crescent-shaped high-density lesion in the region of the ascending to descending aorta, without any other clear evidence of aortic dissection (Fig. 3). A contrast-enhanced CT showed absence of opacification of the high-density region observed on the plain CT (Fig. 3). The patient was diagnosed to have thrombosed Stanford type A aortic dissection, and was admitted to our ward for the management at 4 PM on November 14, 2003. Physical examination at admission revealed a pulse of 77/min and blood pressure of 108/62 mmHg. Echocardiography revealed normal left ventricular function with no abnormal findings, including the pericardial echo-free space. Laboratory examination revealed a hematocrit of 10.5% and white blood cell count (WBC) of 9,600/mm³. The blood urea nitrogen was 20 mg/dl, serum creatinine 0.5 mg/dl, total protein 5.9 mg/dl, total bilirubin 0.6 mg/dl, aspartate aminotransferase 16 IU/l, alanine aminotransferase 8 IU/l, lactate dehydrogenase 196 IU/l, creatine kinase 35 IU/l, γ -glutamyltransferase 9 IU/l, glucose 103 mg/dl, total cholesterol 162 mg/dl, triglyceride 48 mg/dl, HDL-C 36 mg/dl, sodium 143 mEq/l, potassium 3.9 mEq/l, chloride 107 mEq/l, and C-reactive protein (CRP) 0.04

From the Department of Cardiovascular Medicine, Course of Medical and Dental Science, Graduate School of Biomedical Science, Nagasaki University, Nagasaki

Received for publication August 6, 2004; Accepted for publication February 4, 2005

Reprint requests should be addressed to Dr. Hiroaki Kawano, Department of Cardiovascular Medicine, Course of Medical and Dental Science, Graduate School of Biomedical Science, Nagasaki University, 1-7-1 Sakamoto, Nagasaki 852-8051

mg/dl. Medical management was determined because she had no complicating extravasation. She was treated with a calcium-channel antagonist, and blood pressure was controlled. On November 15, 2003, CRP was 2.39 mg/dl and WBC was 6,100 /mm³. Maximum level of CRP was 4.18 mg/dl on November 17, 2003. One week after the admission

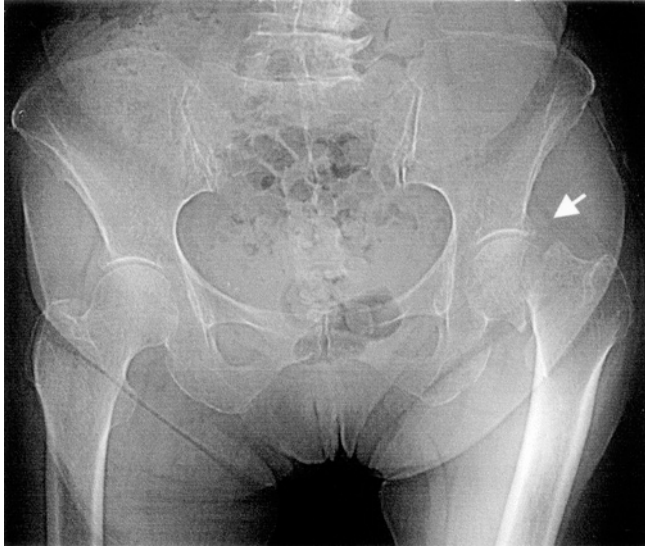


Figure 1. A plain radiograph of the hip showed fracture of the left femur (arrow).

(on November 21, 2003), she complained of mild chest discomfort again. A contrast-enhanced CT showed re-dissection at the site of thrombosed dissection of the ascending aorta (Fig. 4), and CRP was 3.95 mg/dl. She was transferred to the Department of Cardiovascular Surgery for an emergent operation. Operative findings were as follows; an intimal tear was located in the base of the ascending aorta, pseudolumen was closed with fresh thrombus, and moderate bloody pericardial effusion was seen. After ascending aortic replacement was performed, the patient underwent operation on for the hip fracture. Subsequently, her postoperative course was satisfactory, and she was discharged without complication.

Discussion

Although medial degeneration, i.e., degeneration of the medial collagen and elastin, is considered to be the main predisposing factor for aortic dissection (9), it is frequently absent in patients with aortic dissection, in whom the aortic wall changes are remarkably similar to those encountered in elder patients without dissection (10–13). These reports indicate that in addition to reduced resistance of the aortic wall, other factors may also predispose to the development of aortic dissection in these patients. It has been previously reported that physical and/or mental exertion is an important antecedent to dissection (2–4). The physical activities predisposing to aortic dissection could be as mundane as those associated with daily activities working, eating or drinking, defecation or urination, sports, housework, drinking, taking

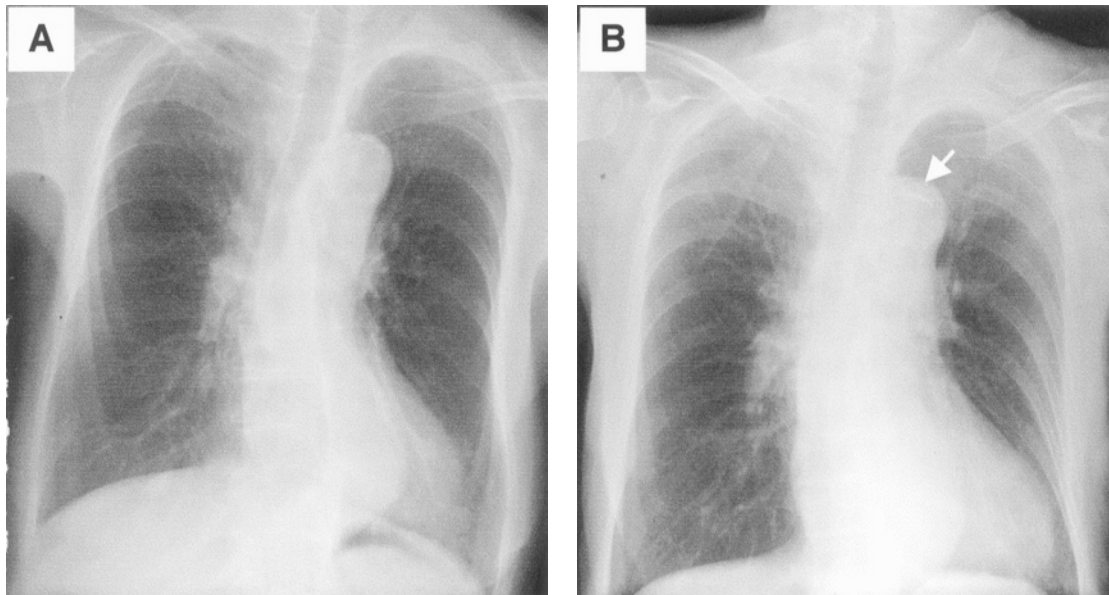


Figure 2. A. Thoracic aorta had kinks and calcification of aortic wall was seen in the chest roentgenogram performed on November 13, 2003. Lung fields were clear and cardiac size was normal. B. A chest roentgenogram showed no mediastinal widening but splitting of calcification of aortic arch was seen (arrow) on November 14, 2003 in addition to the previous findings.

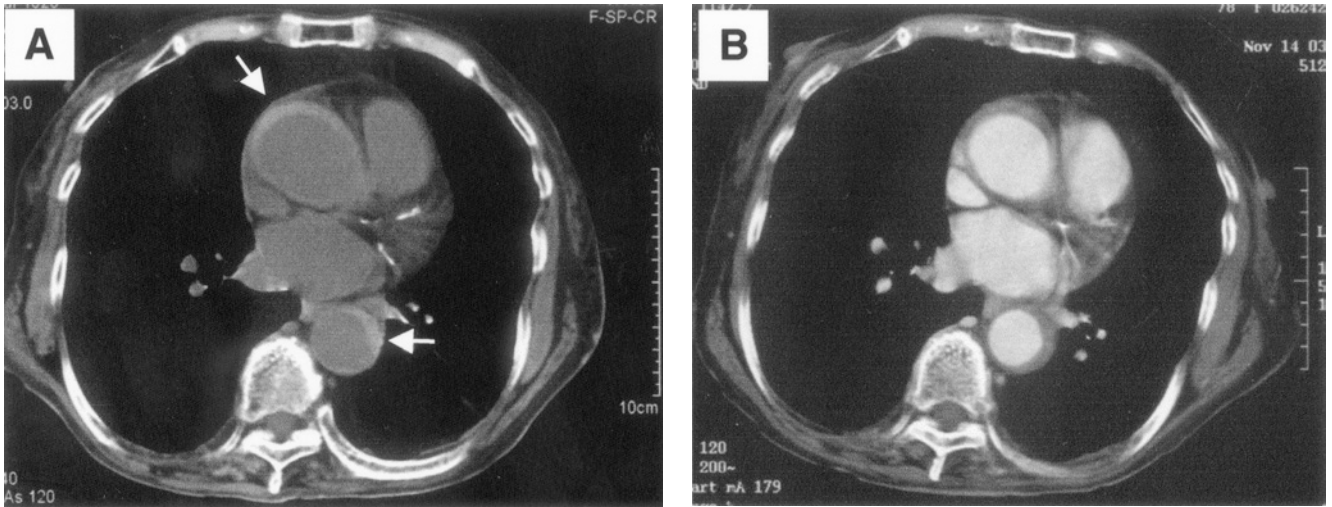


Figure 3. Computed tomography (CT) on November 14, 2003. **A.** A plain CT: Ascending aorta showed mild dilation (about 4.5 cm in diameter) and a crescent-shaped high density lesion was seen in the region of the ascending to descending aorta (arrows). **B.** A contrast-enhanced CT showed absence of opacification of the high-density region observed on the plain CT. It indicates a thrombosed-type aortic dissection.

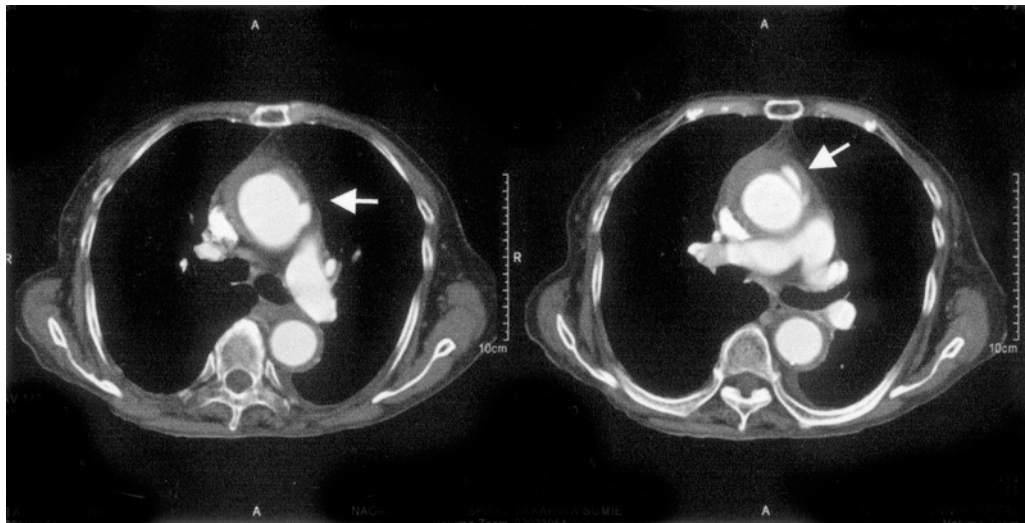


Figure 4. A contrast-enhanced computed tomography on November 21, 2003. Ascending aorta was dilated (about 6 cm in diameter). Thrombus in the pseudolumen was increased, and pseudolumen was dilated especially in the ascending aorta compared to the previous CT (Fig. 3). Ulcer-like projection was seen in the thrombosed pseudolumen of the ascending aorta (arrows). Left pleural effusion was seen.

a bath, and walking.

In the present case, thrombosed aortic dissection occurred just after the patient fell when she moved to the portable pot. While a fall is one of the most important causes of hip fracture in the elderly, it has never been previously reported to trigger the development of aortic dissection. Aging is also

associated with reduced resistance of the arterial wall, and is one of the most common predisposing factors to aortic dissection (14). Thus, the brief and mild dynamic impact to the body associated with a fall seems to be one of the triggers of aortic dissection in the elderly patients.

The main symptoms including back and/or chest pain are

References

observed at the onset of aortic dissection. However, it may be difficult to determine the time of onset of the aortic dissection, and failure to elicit a history of significant symptoms at the onset of dissection sometimes occurs (3). Aortic dissection is sometimes detected after a delay in cases of blunt trauma and direct injury during cardiac surgery (15, 16), and it may be difficult to determine the triggers of aortic dissection. The present patient had mild chest discomfort about 3 hours after she fell on her back, and the symptom disappeared within one hour. The stop of aortic dissection and thrombus formation in the pseudolumen of the aorta may be related to the disappearance of the chest symptom. Fortunately, in the present case, CT conducted for preoperative evaluation of the hip fracture revealed the thrombosed aortic dissection, and allowed for the surgery for the hip fracture to be put off.

The serum level of CRP is elevated in patients with aortic dissection, and elevated admission CRP values in patients with symptomatic aortic aneurysm/dissection are independently associated with poor prognosis (17). In the present case, admission CRP level was normal and CRP became elevated on the day after the admission to our ward, and bone fracture also increases serum level of CRP. Thus, CRP is not a perfect marker of acute aortic dissection in the elderly patients with a bone fracture.

The present case developed chest discomfort a second time, after one week, associated with aortic re-dissection, even though the systolic blood pressure was controlled at below 120 mmHg. The initial pain followed by a painless interval, a window in the spectrum of pain (18), lasting for one hour to several days has been reported in cases of aortic dissection. Meszaros et al (19) reported that 27 of 66 hospitalized patients with acute aortic dissection had a return of pain or died suddenly after this painless interval, suggesting that re-dissection may not be rare in patients with aortic dissection, even if the blood pressure is satisfactorily controlled.

In conclusion, a fall also seems to be one of the triggering factors of aortic dissection in the elderly population, and it is important to consider the diagnosis of aortic dissection in elderly patients who complain of chest discomfort after a fall.

- 1) Kouchochos NT, Dougenis D. Surgery of the thoracic aorta. *N Engl J Med* **336**: 1876–1888, 1997.
- 2) Kojima S, Sumiyoshi M, Nakata Y, Daida H. Triggers and circadian distribution of the onset of acute aortic dissection. *Circ J* **66**: 232–235, 2002.
- 3) Matsuo H. The thrombosed type of aortic dissection—Its clinical features and diagnosis. *Int J Angiol* **7**: 329–334, 1998.
- 4) Hirst AE Jr, Johns VJ Jr, Kime SW Jr. Dissecting aneurysm of the aorta: a review of 505 cases. *Medicine* **37**: 217–279, 1958.
- 5) Cummings SR, Kelsey JL, Nevitt MC, O'Dowd KJ. Epidemiology of osteoporosis and osteoporotic fractures. *Epidemiol Rev* **7**: 178–208, 1985.
- 6) Maggi S, Kelsey JL, Litvak J, Heyse SP. Incidence of hip fractures in the elderly: a cross-national analysis. *Osteoporos Int* **1**: 232–241, 1991.
- 7) Allander E, Lindahl B. The Mediterranean Osteoporosis Study (MEDOS): Theoretical and practical issues of a major international project on hip fracture epidemiology. *Bone* **14**: S37–S43, 1993.
- 8) Melton LJ 3rd. Hip fracture: A worldwide problem today and tomorrow. *Bone Mineral* **14**: S1–S8, 1993.
- 9) Marsalese DL, Moodie DS, Lytle BW, et al. Cystic medial necrosis of the aorta in patients without Marfan's syndrome: surgical outcome and long-term follow-up. *J Am Coll Cardiol* **16**: 68–73, 1990.
- 10) Wilson SK, Hutchins GM. Aortic dissecting aneurysms: Causative factors in 204 patients. *Arch Pathol Lab Med* **106**: 175–180, 1982.
- 11) Schlatmann TJ, Becker AE. Pathogenesis of dissecting aneurysm of the aorta—comparative histologic study of significant medial changes. *Am J Cardiol* **39**: 21–26, 1977.
- 12) Nakashima Y, Kurozumi T, Sueishi K, Tanaka K. Dissecting aneurysm: A clinicopathologic and histologic study of 111 autopsied cases. *Hum Pathol* **21**: 291–296, 1990.
- 13) Larson EW, Edwards WD. Risk factors for aortic dissection: A necropsy study of 161 cases. *Am J Cardiol* **53**: 849–855, 1984.
- 14) Pretre R, Von Segesser LK. Aortic dissection. *Lancet* **349**: 1461–1464, 1997.
- 15) Rogers FB, Osler TM, Shackford SR. Aortic dissection after trauma: Case report and review of the literature. *J Trauma* **41**: 906–908, 1996.
- 16) Still RJ, Hilgenberg AD, Akins CW, Daggett WM, Buckley MJ. Intraoperative aortic dissection. *Ann Thorac Surg* **53**: 374–379, 1992.
- 17) Schillinger M, Domanovits H, Bayegan K, et al. C-reactive protein and mortality in patients with acute aortic dissection. *Intensive Care Med* **28**: 740–745, 2002.
- 18) Crawford ES. The diagnosis and management of aortic dissection. *JAMA* **264**: 2537–2541, 1990.
- 19) Meszaros I, Morocz J, Szilavi J, et al. Epidemiology and clinicopathology of aortic dissection. *Chest* **117**: 1271–1278, 2000.