Left Mandibular Pain: A Rare Initial Symptom of Acute Aortic Dissection Without Coronary Obstruction

Masaki Tago, Naoko E. Furukawa, Rika Yamaguchi, Yoshinori Tokushima, Hidetoshi Aihara and Shu-ichi Yamashita

Abstract

An 88-year-old woman experienced sharp pain in the left mandible for a few minutes 3 days prior to hospital presentation. On the day of hospital presentation, the patient experienced similar pain and cold sweating for more than an hour early in the morning. On arrival, there was only mild discomfort ranging from the left mandible to the neck, without definite pain. Computed tomography revealed Stanford type A acute aortic dissection. Blood vessel prosthesis implantation was performed. Intraoperatively, the coronary arteries were confirmed to be intact. Mandibular pain is a rare but potential symptom of aortic dissection without coronary artery obstruction.

Key words: acute aortic dissection, mandibular pain, vagus nerve, intact coronary arteries

(Intern Med 56: 1663-1665, 2017)  
(DOI: 10.2169/internalmedicine.56.8362)

Introduction

Acute aortic dissection is a potentially fatal disease, with an overall in-hospital mortality of 27.4% (1) and an incidence of 2.6 to 3.5 per 100,000 person-years (2-4). Given its high mortality rate, a rapid and accurate diagnosis is imperative. As the first clues to the diagnosis are usually patient-reported symptoms, non-specific complaints can cause a delayed diagnosis (5) and increase the mortality rate (6). While abrupt chest pain or back pain are the most common symptoms of aortic dissection, rare cases without such symptoms have been reported (6). Among those patients without chest or back pain, the symptoms and signs are commonly syncope, heart failure, cerebral infarction or neurologic defects; mandibular pain has been reported as the first complaint in only one previous case (6, 7).

We herein report a rare case of a patient with acute aortic dissection without complicating acute myocardial infarction presenting with mandibular pain as the initial symptom.

Case Report

The patient was an 88-year-old woman with chronic kidney disease. She had mild cognitive dysfunction, with only slight difficulty in properly expressing her symptoms. The patient was able to complete her daily activities without assistance but might have been in denial about her symptoms. Three days prior to hospital admission, she experienced sharp pain in the left mandible for a few minutes during rest. At 4:00 AM on the day of hospital presentation, the patient experienced sudden pain with a feeling of stiffness in the left mandible, cold sweating, nausea, vomiting and blurred vision, which left her unable to stand. The symptoms persisted for over an hour, and she was taken by ambulance to our hospital at 5:30 AM.

On arrival, the patient was alert and oriented. Her blood pressure was 145/105 mmHg, heart rate 99 beats/min and regular, body temperature 35.1°C and oxygen saturation 93% on room air. Her palpebral conjunctiva did not indicate anemia. Although the spontaneous pain in the left mandible had resolved, the patient had tenderness from the left mandible to the neck without swelling or redness. There was no cervical vascular bruit, and her heart and respiratory sounds were normal. She did not have Horner’s syndrome. The laboratory findings are shown in Table. The patient had renal dysfunction, anemia and elevated levels of aspartate aminotransferase, alkaline phosphatase and C-reactive pro-
Table. Laboratory Findings on Admission.

| Test                | Value   |
|---------------------|---------|
| Complete blood cells| ALT 24 U/L |
| WBC 8,700 /μL       | LDH 286 U/L |
| RBC 2.86x10^12 /μL  | ALP 878 U/L |
| Hb 8.5 g/dL         | AMY 85 IU/L |
| Ht 28.7 %           | Glu 174 mg/dL |
| Pt 20.2x10^11 /μL   | BUN 40.3 mg/dL |
| Biochemistry         | Cr 4.1 mg/dL |
| TP 6.1 g/dL         | Na 142 mEq/L |
| CPK 106 IU/L        | Cl 111 mEq/L |
| T-bil 1.0 mg/dL     | CRP 6.00 mg/dL |
| AST 58 U/L          |         |

WBC: white blood cells, RBC: red blood cells, Hb: hemoglobin, Ht: hematocrit, Pt: platelets, TP: total protein, CPK: creatine phosphokinase, T-bil: total bilirubin, AST: aspartate aminotransferase, ALT: alanine aminotransferase, LDH: lactate dehydrogenase, ALP: alkaline phosphatase, AMY: amylase, Glu: glucose, BUN: blood urea nitrogen, Cr: creatinine, Alb: albumin, Na: sodium, K: potassium, Cl: chloride, CRP: C-reactive protein.

Figure 1. Chest radiograph showing cardiac enlargement and a widened mediastinum.

The reasons for the absence of chest pain in our case were considered to be old age and the location of the aortic dissection in the ascending aorta (6). Painless aortic dissection can reportedly occur with syncope, heart failure, cerebral infarction and neurologic symptoms (6, 7). The presence of such symptoms may indicate painless aortic dissection and help with the diagnosis; however, remarkably, our patient had none of these indicators. Cardiac effusion is shown in 30% of aortic dissection, regardless of the presence of pain (6). We considered the cardiac effusion to have been caused by aortic dissection, as the effusion was confirmed to be bloody during the operation.

The mandibular pain in the present case was considered to be radiating pain elicited by aortic dissection. Cardiogenic pain is known to radiate to the neck, jaw, tooth, arm and shoulder. It is widely accepted that this cardiogenic pain radiation occurs because the cardiac visceral afferent fibers and sensory neurons that innervate the area of pain radiation have a common origin (terminal) in the spinal dorsal horn (9). Furthermore, jaw pain is reportedly the most frequent side effect of vagus nerve stimulation, followed by throat pain (10). The vagus nerve has recurrent branches running around the aortic arch, which contains visceral afferent fibers carrying sensory information from the chest and abdominal organs. These branches of the vagus nerve may be the route of radiating pain caused by aortic dissection and cardiac diseases.

A previous case of aortic dissection with mandibular pain, similar to the present case, reported intermittent left mandibular pain of sudden onset without myocardial ischemia indicated by electrocardiography (11). However, unlike the present case, the mandibular pain in the previously reported patient began with left hemiplegia due to cerebral infarction. As our patient had only nausea and vomiting with mandibular pain, it was more difficult to suspect aortic dissection in our case. Fortunately, aortic dissection was detected by CT performed to rule out any possible abnormalities in the cervical vascular system that could have caused the mandibular pain.

Discussion

Over 90% of patients with acute aortic dissection experience some kind of pain; about 80% of this pain has a sudden onset, and 70-90% occurs in the chest or back (1, 8). However, 4-6% of acute aortic dissection patients have no pain (1, 6), and their mortality rate is higher than that of patients with pain (6). Acute aortic dissection patients without pain generally have characteristic features, including older age, dissection located in the ascending aorta, and/or underlying diseases such as diabetes mellitus, aortic aneurysm, or previous cardiovascular surgery (6). Our patient’s mandibular pain without chest or back pain and the prodromal appearance of mandibular pain 3 days before hospital presentation are very atypical presentations of acute aortic dissec-

tein. Chest radiography showed heart enlargement and a widened mediastinum (Fig. 1). Electrocardiography showed atrial fibrillation without ST segment elevation or depression. Chest and neck computed tomography (CT) revealed cardiac effusion and slightly attenuated lesions in the vascular walls of the ascending and proximal aortic arch, brachiocephalic artery and left common carotid artery. Thus, an acute aortic dissection of Stanford type A was diagnosed (Fig. 2).

After the diagnosis, the patient was immediately transferred to another hospital with higher-order function for surgical reparation of proximal cervical vascular and hemi-arch replacement with a 26 mm J-graft. The coronary arteries were found to be intact without myocardial infarction. Post-operatively, the patient experienced disturbance of consciousness and a convulsion due to cerebral infarction and exacerbation of renal dysfunction; however, she became stable enough to be transferred to another hospital for rehabilitation two months later.
The number of elderly patients presenting as emergency medical cases is increasing, especially in Japan (12-16). Elderly people can have atypical complaints, symptoms, and physical findings. Additionally, detailed descriptions of diseases and/or symptoms can sometimes be elusive because of impaired cognitive function, which consequently makes it difficult to assess the need for a more detailed examination, such as a CT scan with contrast enhancement; as such, the morbidity and mortality rates of these patients tend to be higher than in younger patients. Although our patient described only atypical symptoms of aortic dissection, the sudden onset prompted us to perform an enhanced CT scan, which enabled the correct diagnosis and saved the patient’s life. When evaluating elderly patients in medical practice, physicians should take keywords such as “sudden” into serious account.

Atypical symptoms such as mandibular pain without chest or back pain may be a presenting symptom of acute aortic dissection without obstruction of the coronary arteries.

The authors state that they have no Conflict of Interest (COI).

References

1. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA 283: 897-903, 2000.
2. Bickerstaff LK, Pairolero PC, Hollier LH, et al. Thoracic aortic aneurysms: a population-based study. Surgery 92: 1103-1108, 1982.
3. Meszaros I, Morocz J, Szlavi J, et al. Epidemiology and clinicopathology of aortic dissection: a population-based longitudinal study over 27 years. Chest 117: 1271-1278, 2000.
4. Clouse WD, Hallett JW, Schaff HV, et al. Acute aortic dissection: population-based incidence compared with degenerative aortic aneurysm rupture. Mayo Clinic Proc 79: 176-180, 2004.
5. Hsu YC, Lin CC. Paraparesis as the major initial presentation of aortic dissection: report of four cases. Acta Neurol Taiwan 13: 192-197, 2004.
6. Park SW, Hutchison S, Mehta RH, et al. Association of painless acute aortic dissection with increased mortality. Mayo Clin Proc 79: 1252-1257, 2004.
7. Gaul C, Dietrich W, Friedrich I, Sirch J, Erbguth FJ. Neurological symptoms in type A aortic dissections. Stroke 38: 292-297, 2007.
8. Pape LA, Awais M, Woznicki EM, et al. Presentation, diagnosis, and outcomes of acute aortic dissection: 17-year trends from the International Registry of Acute Aortic Dissection. J Am Coll Cardiol 66: 350-358, 2015.
9. Lee TH. Chest discomfort. In: Harrison's Principles of Internal Medicine. 18th ed. Longo DL, Kasper DL, Jameson JL, Fauci AS, Hauser SL, Loscalzo J, Eds. McGraw-Hill, New York, 2012: 102-107.
10. Myers DE. Vagus nerve pain referred to the craniofacial region. A case report and literature review with implications for referred cardiac pain. Br Dent J 204: 187-189, 2008.
11. Li CH, Hsu YC, Hsu YC. Left mandibular pain in a patient of aortic dissection presenting with acute ischemic stroke: a case report. Acta Neurol Taiwan 23: 129-133, 2014.
12. Ota B. Programs for Continuing Medical Education: A session; 9. Pitfall in geriatric emergency care. Nihon Naika Gakkai Zasshi 104: 526-531, 2015 (in Japanese).
13. Kuhn JH, Magauran B. Trends in geriatric emergency medicine. Emerg Med Clin North Am 24: 243-260, 2006.
14. Walsh B, Roberts H. Older people’s use of Accident & Emergency services. Age Ageing 34: 535-536, 2005.
15. Lowthian J, Curtis A, Stoelwinder J, McNeil J, Cameron P. Emergency demand and repeat attendances by older patients. Intern Med J 43: 554-560, 2013.
16. Yim VW, Graham CA, Rainer TH. A comparison of emergency department utilization by elderly and younger adult patients presenting to three hospitals in Hong Kong. Int J Emerg Med 2: 19-24, 2009.

The Internal Medicine is an Open Access article distributed under the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License. To view the details of this license, please visit (https://creativecommons.org/licenses/by-nc-nd/4.0/).

© 2017 The Japanese Society of Internal Medicine
http://www.naika.or.jp/imonline/index.html

Figure 2. Chest and neck computed tomography revealed an area of low attenuation in the pericardium, indicating cardiac effusion (arrowheads), and continuous weak high-attenuation areas in the walls of major arteries, ranging from the ascending aorta to the proximal aortic arch, brachiocephalic artery and left common carotid artery (arrows).