INTERNAL MEDICINE

□ CASE REPORT □

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

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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

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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℃ 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-

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Table. **Laboratory Findings on Admission.**

Complete blood cell counts		Biochemistry	
WBC	8,700 /μL	TP	6.1 g/dL
RBC	2.86×10^6 /μL	CPK	106 IU/L
Hb	8.5 g/dL	T-bil	1.0 mg/dL
Ht	28.7 %	AST	58 U/L
Plt	20.2×10^4 /μL	ALT	24 U/L
		LDH	286 U/L
		ALP	878 U/L
		AMY	85 IU/L
		Glu	174 mg/dL
		BUN	40.3 mg/dL
		Cr	4.1 mg/dL
		Na	142 mEq/L
		K	3.6 mEq/L
		Cl	111 mEq/L
		CRP	6.00 mg/dL

WBC: white blood cells, RBC: red blood cells, Hb: hemoglobin, Ht: hematocrit, Plt: 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

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. Postoperatively, 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.

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 dissection. 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.

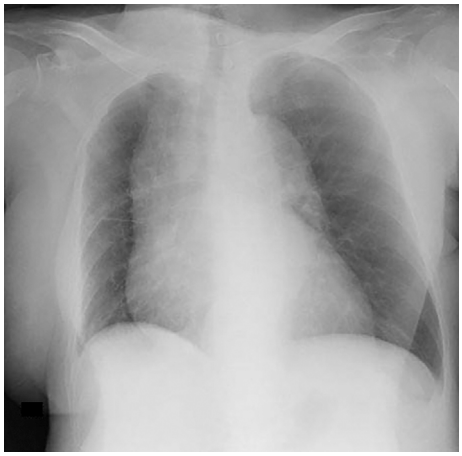

Figure 1. **Chest radiograph showing cardiac enlargement and a widened mediastinum.**

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.

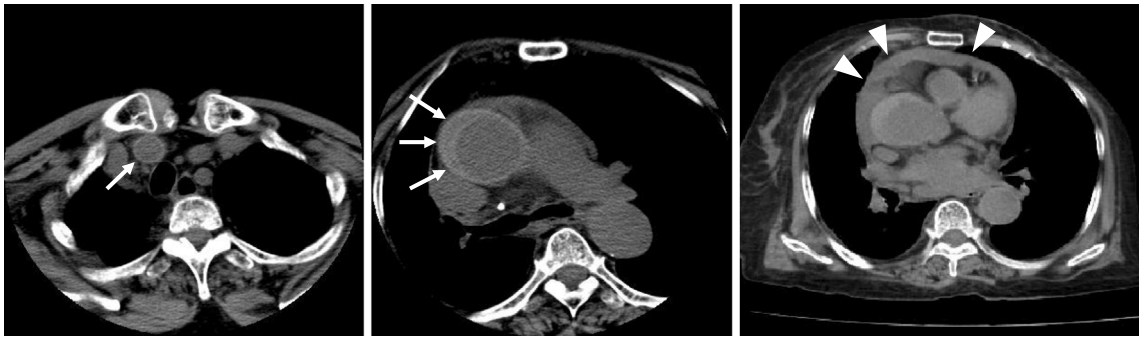

Figure 2. **Chest and neck computed tomography revealed an area of low attenuation in the pericardium, indicating cardiac effusion (arrowheads), and continuous weak high-attenuation areas in the walls of major arteries, ranging from the ascending aorta to the proximal aortic arch, brachiocephalic artery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

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).

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