It is a medical emergency! Act fast: a case report of painless aortic dissection

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Background
A painless presentation of aortic dissection is rare in about 5% of cases. A few case reports discussed about involvement of coronary arteries causing bradycardia. In our case, we are presenting a rare presentation of aortic dissection with initial presentation as bradycardia without involvement of coronary arteries and without cardiac symptoms.

Case summary
A 56-year-old man with history of hypertension presented with acute onset of altered mental status and right hand numbness. Initial vital signs were remarkable for bradycardia with heart rate 36/min. Physical exam was only significant for decreased sensation for light touch on right hand. Electrocardiogram showing marked sinus bradycardia. Initial computed tomography (CT) head did not show any acute abnormalities. On Day 2, patient complained of new weakness in left upper extremity. On exam, he had dysmetria, left upper extremity drift and cold to touch. Blood pressure was significantly different in both arms with 141/83 mmHg on right and 44/23 mmHg on left. He underwent immediate CT angiography chest showing Type 1 aortic dissection with extension into the brachiocephalic artery and right common carotid artery (RCCA) with thrombosis in RCCA. Patient was emergently taken for surgical repair of aortic dissection and resuspension of aortic valve.

Discussion
In our case, the initial presentation of aortic dissection included isolated sinus bradycardia without cardiac symptoms and no evidence for atrioventricular block. The mechanism may have been involvement in the arterial supply of the carotid body receptors. Patients with coexisting symptoms such as bradycardia and neurological deficits should be evaluated for possible aortic dissection.

Keywords
Aortic dissection • Bradycardia • Painless aortic dissection • Case report

Learning points
• Patients with coexisting symptoms such as bradycardia and neurological deficits should be evaluated for possible aortic dissection. Early imaging in addition to adequate physical exam including measurement of blood pressure in both arms in these patients will improve the diagnosis.
• Role of tissue plasminogen activator (tPA) in patients presenting with unexplained bradycardia with coexisting neurological deficits should be cautiously evaluated as providing tPA in masked cases of aortic dissection further increases mortality.

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Introduction

Aortic dissection classically presents with sudden onset, severe tearing chest pain often radiating to back. A painless presentation is a rare occurrence in about 5–10% of cases with syncope and stroke being most common presentation in these patients. Often the occurrence of syncope or stroke in these cases is attributed to involvement of one of arteries supplying the part of heart causing heart block or brain causing acute stroke. Presentation with isolated bradycardia without any other cardiac symptoms is very rare. There are a few published case reports describing an aortic dissection involving the coronary artery that supplies the atrioventricular (AV) node causing bradycardia. In our case, we highlight a rare presentation of aortic dissection with initial manifestation as bradycardia with possible involvement of arterial supply for carotid receptors causing bradycardia. In this case, we report a patient with aortic dissection involving right common carotid artery (RCCA) with thrombosis in RCCA. The patient had persistent neurological deficits with bilateral hemiparesis, slurred speech and was discharged to an acute rehabilitation facility.

Timeline

Upon presentation to emergency room
- Acute onset of altered mental status, slurred speech, and right hand numbness of 2 h duration.
- Electrocardiogram showing sinus bradycardia with heart rate 36/min.
- Decreased sensation for light touch on right hand with National Institute of Health (NIH) scale of 1.
- Computed tomography (CT) head: no acute abnormalities.
- Patient complained of new weakness in left upper extremity.
- On examination, dysmetria and pronator drift were noted in left upper extremity and bilateral upper limbs were cold to touch.
- Significant blood pressure difference noted in both arms.
- Immediate CT angiography chest: Type 1 aortic dissection with extension into the brachiocephalic artery and right common carotid artery (RCCA) with thrombosis in RCCA.
- Emergent surgical repair of aortic dissection with placement of Dacron graft and resuspension of aortic valve.
- Follow-up brain imaging confirmed multiple ischemic stroke in bilateral hemispheres.
- Continued management in intensive care unit.
- Persistent neurological deficits with bilateral hemiparesis, slurred speech.
- Patient was discharged to an acute rehabilitation facility.

Case presentation

A 56-year-old man with history of hypertension presented with acute onset of altered mental status, slurred speech, and right hand numbness of 45 min duration. Patient was not using any medications at home. Initial vital signs were remarkable for bradycardia with heart rate 36/min, respiratory rate 20, saturating 95% on 5 L nasal cannula and blood pressure 136/72 mmHg. Physical exam was only significant for decreased sensation for light touch on right hand with National Institute of Health (NIH) scale of 1. Transient confusion resolved. Electrocardiogram showing marked sinus bradycardia with prolonged PR interval, prolonged QRS duration, a right bundle branch block, and left axis deviation. No evidence of ischemic changes or ST elevation (Figure 1). Chest X-ray was unremarkable.

Initial computed tomography (CT) head did not show any acute abnormalities. Due to the possibility of acute ischemia with initial neurological symptoms, tissue plasminogen activator (tPA) was initially considered for emergent thrombolysis in stroke. Patient declined tPA with improvement in symptoms and due to the risk of bleeding. The patient was admitted for telemetry monitoring. Overnight, during the first 12 h of hospitalization, there were no new neurological events and telemetry revealed persistent sinus bradycardia (heart rate 30–50) without AV block. Echocardiogram was performed 14 h after admission and was of limited quality due to poor echo windows and revealed normal chamber dimensions with an left ventricular ejection fraction of 60–65%. The aortic valve and aortic root were not well seen and no pathological aortic insufficiency was seen on Doppler. Sixteen hours after admission, the patient complained of new weakness in left upper extremity. On examination, dysmetria and pronator drift were noted in left upper extremity and bilateral upper limbs were cold to touch. Non-invasive blood pressure discrepancy was measured in the arms with right arm 44/23 and left 114/83 mmHg. He underwent immediate CT angiography chest showing Type 1 aortic dissection with extension into the brachiocephalic artery and right common carotid artery (RCCA) with thrombosis in RCCA (Figure 2). Patient was emergently taken for surgical repair of aortic dissection with placement of Dacron graft and resuspension of aortic valve. Post-operatively, no further pathological bradycardia or AV block was detected. Patient was not screened for inflammatory or infectious conditions, e.g. giant cell arteritis and syphilis as these causes seemed unlikely with the history and presentation. Follow-up brain imaging confirmed multiple ischemic stroke with large acute infarctions of the right cerebral hemisphere and basal ganglia predominantly within the internal watershed distribution and tiny acute infarctions of the left basal ganglia and left frontal lobe. The patient had persistent neurological deficits with bilateral hemiparesis, slurred speech and was discharged to an acute rehabilitation facility.

Discussion

Type I aortic dissection is a life-threatening emergency with mortality rates >50% in first 24 h, increasing up to 75% within 2 weeks of
the event. In our case, the initial presentation of aortic dissection included isolated sinus bradycardia. There were no other cardiac symptoms and no evidence for AV block. Patient had history of hypertension that was controlled at presentation but did not have any other known risk factors for aortic dissection including atherosclerosis, pre-existing aortic aneurysms, bicuspid aortic valve or aortic coarctation, and rare genetic diseases including Turner’s syndrome and Marfan syndrome. No family history of aneurysms of the aorta and other blood vessels or aortic dissections or connective tissue disorders including Ehlers–Danlos and Loeys–Dietz syndrome. Patient did not have history of engaging in any high-risk activity prior to presentation which may have precipitated the dissection, e.g. illicit drug use such as cocaine or high-intensity weightlifting. Echocardiogram did not show any obvious aortic valve abnormalities. The mechanism may have been pressure on the carotid body or involvement in the arterial supply of the carotid body receptors by the dissection. Carotid body pressure can be associated with bradycardia alone, hypotension alone, or both.4,5 In our patient, there was delay in diagnosis of this emergent condition because of this unusual and rare presentation. Standard testing including carotid ultrasound, echocardiogram, and further imaging of the cerebral vasculature were planned but not done on an emergent basis since the patients symptoms improved and no haemodynamic instability or usual symptoms for aortic dissection were present in first 12 h. Bilateral upper extremities blood pressures were not reported until 16 h after admission but exams reported no difference in pulses. Recognizing pulse differential in the setting of new focal neurological deficits prompted us for emergent evaluation for aortic dissection. Pulse or blood pressure differential, chest pain and mediastinal widening, or aortic widening on chest X-ray together form the high-risk clinical triad in clinical prediction of aortic dissection with a probability of 90–100% with presence of pulse or blood pressure differential alone and 83% with combination of chest pain and mediastinal widening.6 Mortality is high (43%) in painless presentation of aortic dissection compared with painful presentation (10%). Painless presentation is often challenging and a timely diagnosis can be crucial thus early recognition of pulse differential is extremely important. Patients with coexisting symptoms such as bradycardia and neurological deficits should be evaluated for possible aortic dissection. For patients with unexplained bradycardia and neurological findings, a thorough physical exam including measurement of blood pressure in both arms is essential followed by prompt imaging is important for confirmation of the diagnosis to enable prompt intervention, i.e. surgery. Early surgical intervention in patients with Type 1 aortic dissection decreases mortality.7 In patients presenting with unexplained bradycardia with coexisting neurological deficits or being considered for acute stroke, aortic dissection should be considered despite an absence of other cardiac symptoms as providing tPA would increase mortality.
Figure 2  Computed tomography angiography showing Type 1 aortic dissection. (A) Computed tomography angiography chest with contrast axial view showing Type 1 aortic dissection with true (small arrow) and false lumen (large arrow) involving ascending aortic arch. (B) Computed tomography angiography chest with contrast axial view showing extension of Type 1 aortic dissection into right common carotid artery with true (small yellow arrow) and false lumen (large yellow arrow) with occlusion and a patent left common carotid artery (red arrow). (C) Computed tomography angiography chest with contrast sagittal view showing Type 1 aortic dissection with true and false lumen involving ascending aortic arch. (D) Computed tomography angiography chest with contrast coronal view showing Type 1 aortic dissection with true (small yellow arrow) and false lumen (big yellow arrow) involving ascending aortic arch extending into brachiocephalic artery and right common carotid artery (red arrow). (E) Computed tomography angiogram head and neck with contrast showing occlusion with thrombosis in right common carotid artery.
Lead author biography

Anusha Yanamadala, MD, graduated from Government Medical College Anantapur, India. Currently, she is in final year residency in Internal Medicine at Loyola Medicine MacNeal hospital in Illinois, USA. She is a member of ACP (American college of Physicians) and AHA (American Heart Association). Interested in pursuing fellowship and further career in cardiology. Previous research and publications in PET/CT and venous thromboembolism.

Supplementary material

Supplementary material is available at European Heart Journal - Case Reports online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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