

An Uncommon Cause of New-Onset Heart Failure



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A 50-year-old woman with hypertension was admitted to the hospital for evaluation of a 6-week history of effort dyspnea and recent orthopnea. Her blood pressure was 207/113 mm Hg, and bibasilar crepitations and leg edema were noted on physical examination. Chest radiography revealed cardiomegaly, pulmonary congestion, and small bilateral effusions. A widening of the mediastinum was noted (Figure 1). Echocardiography identified marked left ventricular hypertrophy, preserved function, mild calcific aortic stenosis/regurgitation, and a dilated ascending aorta (7.5 cm) and aortic arch (5.5 cm). Computed tomographic angiography confirmed an aneurysm of the ascending aorta (8.9×8.1 cm) and revealed aortic dissection (AoD) (Figure 2). The patient did not recall any pain. Hypertension normalized with intravenous nitropruside, and the patient was transferred to the intensive care unit before vascular surgical intervention.

Our patient's risk factors were hypertension and aortic aneurysm, seen in 69% and

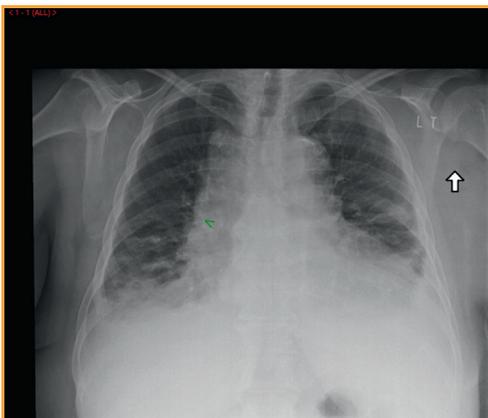


FIGURE 1. Chest radiograph at presentation showing cardiomegaly, pulmonary congestion, and small bilateral effusions. Note the widening of the mediastinum.

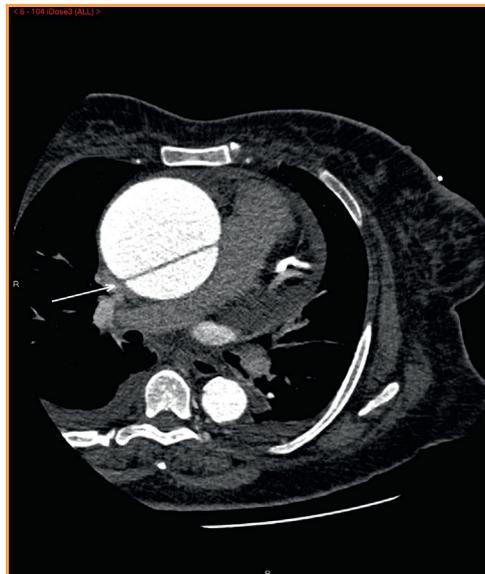


FIGURE 2. Single axial image from chest computed tomographic angiography showing an intimal flap in an aneurysmal ascending aorta (arrow), consistent with Stanford type A dissection. The intimal flap extended into the right common carotid artery (not shown). Small bilateral pleural and pericardial effusions were present. Calcifications were evident in the abdominal aorta and its branches.

12% of patients with proximal (type A) AoD.¹ The potential involvement of any part of the aorta and the dynamic nature of AoD underlie the great diversity in presentation, which often mimics other conditions. Severe pain (90%) and abrupt onset (85%) are characteristic.¹ Some patients, however, may present with symptoms of more than 2 weeks' duration (termed *chronic AoD*). Only approximately 6% do not have pain.^{1,2}

Our patient's dissection was chronic and painless and presented as congestive heart failure (CHF)—each a highly atypical feature that may triple the time to diagnosis and increase mortality risk to 33%.² Among 1069



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patients in the International Registry of Acute Aortic Dissection,³ 6% presented with acute CHF and were less likely to have chest pain. Varied mechanisms underlie CHF symptoms in AoD: new aortic regurgitation, coronary artery involvement, hypertension, anemia, dissection into the left atrium, and compression causing supra-avalvular aortic stenosis or increased left ventricular afterload. Physicians should be cognizant of painless AoD presenting as CHF because this atypical presentation of catastrophic disease impacts timely diagnosis and is an independent predictor of surgical delay.³ The common occurrence of a widened mediastinum on admission chest radiography may be a valuable clue ([Supplemental Figure](#), available online at <http://www.mayoclinicproceedings.org>).

SUPPLEMENTAL ONLINE MATERIAL

Supplemental material can be found online at: <http://www.mayoclinicproceedings.org>. Supplemental material attached to journal articles has not been edited, and the authors take responsibility for the accuracy of all data.

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