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CASE REPORT

ADVANCED

HEART CARE TEAM/MULTIDISCIPLINARY TEAM LIVE

Dissecting Into the 2021 Chest Pain Guidelines

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ABSTRACT

Although acute coronary syndromes remain crucial diagnoses of chest pain that cannot be missed, there are several other potentially fatal diagnoses that can manifest similarly. This case report applies the 2021 chest pain guidelines emphasizing the importance of considering alternative nonischemic but still serious presentations under the umbrella of chest pain. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2022;4:21–26) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 70-year-old man presented to the emergency department with sudden onset midsternal chest pain at rest that started approximately 6 hours earlier. He used tramadol 50 mg once, but this did not help with the pain. The chest pain was aching and constant, located over his midsternum without radiation. This pain was associated with dyspnea at rest and nausea with nonbilious emesis. It was not worse with activity. He denied previous chest pain episodes.

LEARNING OBJECTIVES

- To recognize other serious causes of acute chest pain on initial presentation aside from ACS.
- To understand the different types of imaging modalities for the diagnosis of aortic dissection and the optimal protocols for those modalities.
- To increase knowledge about both the medical and surgical management of aortic dissection patients, especially for those who are not candidates for open repair.

PAST MEDICAL HISTORY

His past medical history included heart failure with a reduced ejection fraction of 37% attributed to ischemic cardiomyopathy, coronary artery disease, hypertension, moderate mitral regurgitation, moderate tricuspid regurgitation, paroxysmal atrial fibrillation, hyperlipidemia, diabetes mellitus type 2 complicated by retinopathy, peripheral arterial disease, and benign prostatic hyperplasia.

His past surgical history was relevant for coronary artery bypass grafting (CABG) with an in situ left internal thoracic artery to the left anterior descending artery, a reverse saphenous vein graft to the right coronary artery (RCA), a reverse saphenous vein graft to the second diagonal artery, and a reverse saphenous vein graft to the left circumflex artery, along with repair of the mitral valve with a #28 profile 3-dimensional complete rigid annuloplasty ring and repair of the tricuspid valve with a Kay annuloplasty

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ABBREVIATIONS AND ACRONYMS

ACC/AHA = American College of Cardiology/American Heart Association

ACS = acute coronary syndrome

CABG = coronary artery bypass grafting

CMR = cardiac magnetic resonance

CTA = computed tomography angiography

CTAG = Conformable Gore Tag Thoracic Endoprosthesis

ECG = electrocardiogram

LOE = Level of Evidence

RCA = right coronary artery

TEE = transesophageal echocardiogram

TTE = transthoracic echocardiogram

stitch performed a month before this presentation. He also had undergone percutaneous coronary intervention in the right coronary artery (RCA) 7 years earlier and laminectomy 4 years earlier.

QUESTION 1: WHAT IS THE DIFFERENTIAL DIAGNOSIS AT THIS POINT?

With his recent history of CABG, acute coronary syndrome (ACS) should be investigated because 1 of his grafts may have occluded, or he have may had a new coronary plaque rupture or erosion. As the new 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR chest pain guidelines stress,[1] there are several etiologies of cardiac but nonischemic chest pain that need to be quickly identified such as perimyocarditis, valvular disease, anomalous coronaries or aortic dissection (**Figure 1, Table 1**). Other differential diagnoses that should not be missed include stress-induced cardiomyopathy, arrhythmias, pulmonary embolism, pneumothorax, and esophageal spasm or rupture.

QUESTION 2: WHAT INVESTIGATIONS SHOULD BE DONE AT THIS TIME?

For cardiac causes, serial electrocardiogram (ECGs) and troponin level determinations should be done. A bedside echocardiogram should also be performed if available to evaluate for cardiac tamponade, regional wall motion abnormalities, valvulopathies, and aortic dissection. A chest radiograph and/or computed tomography (CT) of the chest with contrast can be done to examine for pulmonary causes while also looking for dissection flaps, crepitus suggestive of esophageal rupture, or musculoskeletal abnormalities.

In the emergency department, our patient's notable vital signs included a blood pressure of 209/105 mm Hg with a heart rate of 99 beats/min, whereas the rest of his vital signs were within normal ranges. On physical examination, he was in acute distress because of pain, his jugular venous pulsation was below his clavicle, and his heart was tachycardic but in regular rhythm without murmurs, rubs, or gallops. His lungs sounded clear, and his abdomen was soft and nontender without bruits. In addition, he had no lower extremity edema, and his radial and dorsalis pedis pulses were 2+ bilaterally. He was alert and oriented to self, place, and time.

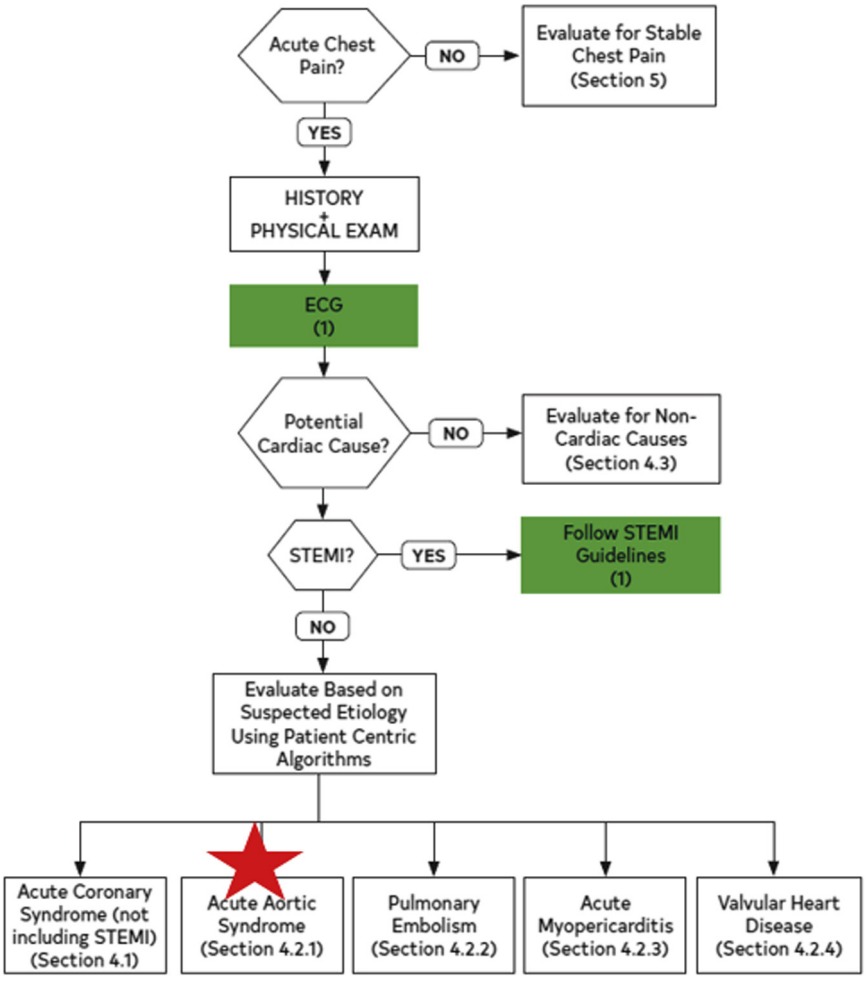
An ECG performed within 10 minutes of arrival showed normal sinus rhythm with left-axis deviation, anterior and inferior Q waves, and poor R-wave progression (**Figure 2**). A repeat ECG 15 minutes later showed similar findings. His conventional troponin T value was 0.08 ng/mL, and a repeat troponin measurement 3 hours later was 0.07 ng/mL (normal range, 0.000-0.029 ng/mL). His other laboratory values were unremarkable. His chest radiograph showed bibasilar atelectasis and intact sternotomy wires with aortic tortuosity.

He was sent for chest CT to evaluate for a pulmonary embolism study. Although there was no pulmonary embolism seen, the scan did show a type A aortic dissection starting from the sinotubular junction posteriorly and extending along the right posterior arch of the ascending aorta. The false lumen extended into the abdominal aorta. The coronary arteries, brachiocephalic artery, left common carotid artery, left subclavian artery, and celiac artery came off the true lumen (**Figures 3A to 3D**). Afterward, a bedside echocardiogram showed a type A dissection extending from the sinotubular junction as far as the supramesenteric aorta. The echocardiogram also showed a left ventricular ejection fraction of 55%, severe hypertrophy, normal right ventricular function and size, biatrial dilation, trivial mitral regurgitation, and trivial tricuspid regurgitation. Trace aortic regurgitation and no pericardial effusion were observed (Videos 1A and 1B).

QUESTION 3: WHAT INVESTIGATIONS ARE WARRANTED FOR TYPE A DISSECTION ACCORDING TO THE NEW 2021 CHEST PAIN GUIDELINES?

Applying our patient's presentation to the 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR chest pain guidelines,[1] he did have a bedside thoracic echocardiogram (TTE), which the guidelines give a Class 1, Level of Evidence (LOE): C-EO recommendation to evaluate for nonischemic causes of chest pain. Given his recent surgery and thus high risk for pulmonary embolism, his CT scan showed the dissection before our facility obtained an echocardiogram. In general, when aortic dissection is suspected, these patients should undergo either CT angiography (CTA) of the chest, abdomen, and pelvis (Class 1, LOE: C-EO) or a transesophageal echocardiogram (TEE) or cardiac

FIGURE 1 **Chest Pain Algorithm Highlighting Other Acute, Potentially Life-Threatening Causes of Chest Pain**



Evaluation of acute chest pain of nonischemic etiologies with our case **(star)** shown here. ECG = electrocardiogram; STEMI = ST-segment elevation myocardial infarction. Adapted from Gulati et al.[1]

TABLE 1 **Differential Diagnosis of Cardiovascular Nonischemic and Fatal Noncardiac Causes of Chest Pain in the Emergency Department**

Cardiovascular nonischemic causes
Pericarditis or pericardial effusion
Perimyocarditis
Aortic dissection: intramural hematoma or perforating arterial ulcer
Severe aortic stenosis
Anomalous coronary disease
Noncardiac fatal causes
Pulmonary embolism
Pneumothorax
Esophageal spasm or rupture

magnetic resonance scan if CT is contraindicated or unavailable (Class 1, LOE: C-EO).[1]

The 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for thoracic aortic disease states recommend that if CT is used, one should use helical CT scanners for superior spatial resolution with slices of 3 mm or less and ECG gating to avoid aortic root motion artifact.[2] CT imaging should extend from the root and at least until the bifurcation of the aortoiliac tree.

For echocardiography, TEE is superior to TTE for the thoracic aorta. To diagnose dissection, imaging must show the dissection flap, and artifacts including

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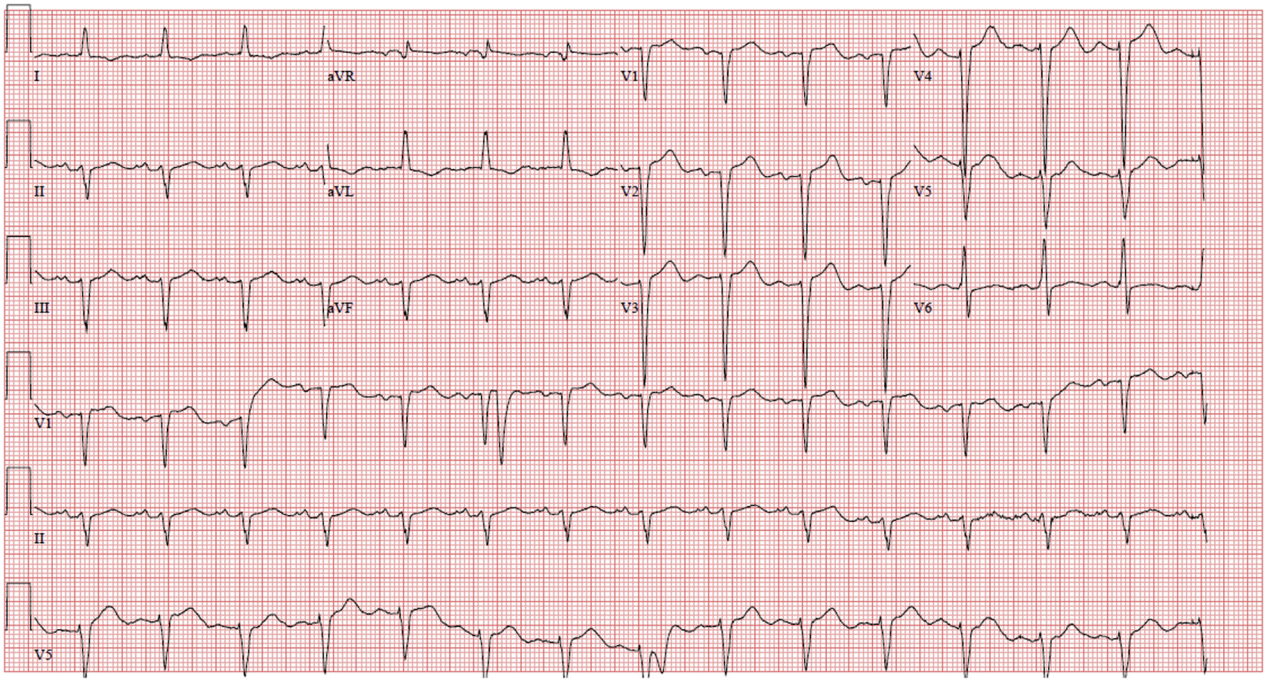
FIGURE 2 Initial Electrocardiogram on Presentation



mirroring and reverberation can lead to false positive results. Thus, one must confirm the diagnosis in several views, by looking for independent motion of the flap from the adjacent anatomy. Color flow Doppler can be used to show differential flow as well.[2]

QUESTION 4: WHY CAN IT BE DIFFICULT TO DISTINGUISH AORTIC DISSECTIONS FROM ACS?

Aortic dissections can be difficult to distinguish from ACS given the clinical and risk factor overlap because dissections are associated with hypertension and older age.[3] Our patient's lack of troponin elevation and trend argued against ACS. However, aortic dissection can be associated with elevated troponins through several different mechanisms, including catecholamine surge, kidney injury, and dissection of coronary arteries, most commonly the RCA.[4]

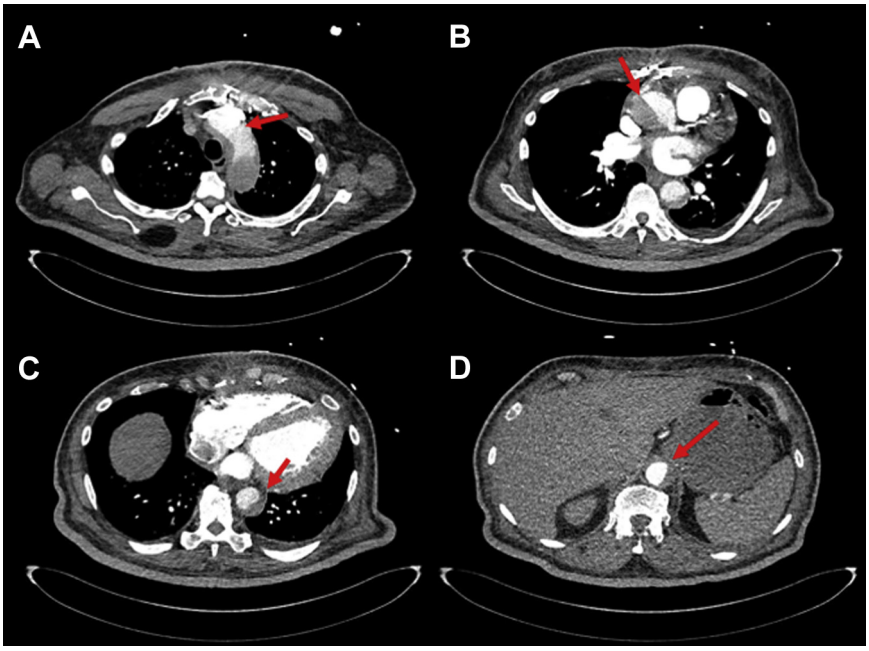
QUESTION 5: WHAT IS THE MORTALITY OF TYPE A DISSECTIONS, AND WHAT ARE RISK FACTORS FOR DELAYS IN DIAGNOSIS?

As with ACS, it is important to diagnose aortic dissection quickly given its high mortality. On average, it takes 4.3 hours to diagnose dissections, with longer delays associated with female patients and with patients with atypical symptoms, a history of previous cardiac surgery, or initial presentation to a nontertiary care center.[3] Untreated patients have a mortality of 1%-2% per hour from the onset of symptoms.[5] Recognition is crucial because surgery drastically alters the high mortality rate. Patients who undergo surgery for type A dissections have a 1-year survival rate of 96% and a 3-year survival rate of 90.5%, although these numbers do not reflect sicker patients who did not qualify for surgical treatment.[6]

QUESTION 6: RECOGNIZING THAT SURGERY IS THE OPTIMAL TREATMENT, HOW IS TYPE A DISSECTION MANAGED MEDICALLY UP UNTIL THE POINT OF SURGERY?

Labetalol, 20 mg once intravenously, and a nitroprusside drip at 20 μg/min were administered in the emergency department, and he was transferred to the cardiac intensive care unit with an immediate cardiothoracic surgery consultation. While the patient was in the unit, the care team achieved a goal systolic blood pressure of <125 mm Hg and heart rate

FIGURE 3 **Computed Tomography Screening for Pulmonary Embolism Showing Aortic Dissection**



Aortic dissection **(A)** at the level of the ascending aorta **(arrow), (B)** at the level of the coronary arteries with the left main artery coming off the true lumen **(arrow), (C)** at the level of the descending aorta **(arrow),** and **(D)** extending into the abdomen **(arrow).**

of 50 to 60 beats/min with metoprolol, 5 mg intravenous pushes every 5 minutes as needed, and a nitroprusside drip at 40 μg/min. His pain subsided with these interventions, and he was given morphine, 1 mg every 15 minutes as needed for breakthrough pain.

HOSPITAL COURSE

He quickly developed acute kidney injury, with repeat creatinine values increasing from 1.15-1.83 mg/dL over 6 hours; these levels peaked at 4.32 mg/dL 3 days later. Thus, CT of the abdomen and pelvis with contrast was not pursued to define the extent of dissection. A renal vascular ultrasound examination performed on day 2 showed high resistive flow in both renal arteries and low flow within the renal parenchyma.

Given his age and comorbidities, he was deemed too high a risk for open repair, and the surgical team decided on stent grafting in discussion with the patient. In the operating room, intravascular ultrasound and minimal contrast injections showed an entry tear in the mid-distal ascending aorta with patent bypass grafts and another entry tear distal to the left subclavian artery. On day 5 of hospitalization, he underwent thoracic endovascular aortic repair with placement of a 34 mm × 15 cm Conformable Gore Tag Thoracic Endoprosthesis (CTAG, W.L. Gore & Associates) thoracic stent graft from the celiac artery to the renal artery and placement of a 40 mm × 20 cm CTAG thoracic stent graft from the left subclavian artery to the celiac artery. The mid-distal ascending aorta dissection was not addressed because of an aortogram showing an optimally expanded true lumen and concern about compromising the patent bypass graft. His creatinine improved to 1.41 mg/dL, and he was discharged to rehabilitation on day 13. To maintain impulse control with target heart rate of <60 beats/min and blood pressure <120/80 mm Hg, he was discharged on carvedilol 25 mg twice a day. For better blood pressure control, he was also receiving the following: amlodipine 10 mg daily, hydralazine 100 mg 3 times a day, isosorbide dinitrate 10 mg 3 times a day, and furosemide 20 mg daily.

Overall, our case demonstrates that it is crucial to recognize that not all chest pain is ischemic but still

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can be potentially fatal. The new guidelines[1] reaffirm the importance of timely identifications of these dangerous causes of chest pain to facilitate lifesaving treatments.

FOLLOW-UP

He was seen in the cardiothoracic surgery clinic and was noted to be doing well, without recurrence of chest pain.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS acute coronary syndrome, aortic dissection, chest pain

APPENDIX For a supplemental video, please see the online version of this paper.

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