CHEST

A 43-Year-Old Man Presenting With Severe Chest Pain

Pablo Blanco, MD

VIDEO

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A 43-year-old man was brought to the ED with chest pain that had evolved for the previous 2 hours. His personal medical history revealed poorly controlled arterial hypertension and active smoking. Family history highlights included his father's sudden death at age 40 years.

On presentation, the patient's vital signs were a regular heart rate at 80/min, BP of 220/120 mm Hg, respiratory rate of 24/min, and temperature of 36°C. Pulse oximetry saturation was 95% on room air. The physical examination revealed a lucid but uncomfortable and poorly cooperative patient. He stated that the pain originated in the precordium but later radiated to the interscapular area. Pain was severe from the beginning; it was not accompanied by nausea, vomiting, or diaphoresis and did not reproduce with palpation. He looked pale but well perfused; a high-intensity second sound was heard on cardiac auscultation, and no murmurs were noted. Central and peripheral pulses were present and symmetric. Respiratory examination indicated clear lung fields. ECG showed regular sinus rhythm at 80/min. No Q waves, ST-segment deviation, or negative T waves were noted. High-voltage QRS complexes compatible with left ventricle (LV) hypertrophy were seen. A medical ICU consultation was called. After initial history, physical examination, and ECG review, bedside ultrasound (transthoracic echocardiogram [TTE]) was performed to evaluate the etiology of chest pain (Video 1).

Based on Video 1, the ECG, and the patient's clinical presentation, what is the most likely diagnosis?

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AFFILIATIONS: From the Intensive Care Unit, Hospital Dr Emilio Ferreyra, Necochea, Argentina.

CORRESPONDENCE TO: Pablo Blanco, MD, Intensive Care Unit, Hospital Dr Emilio Ferreyra, 4801, 59 St, Necochea 7630, Argentina; e-mail: ohtusabes@gmail.com

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Answer: Acute aortic dissection

Discussion

The images in Video 2 show the suprasternal notch of the ascending aorta, aortic arch, and proximal descending aorta to help rule out a proximal dissection. This patient presented with chest pain, the origin of which remained uncertain after clinical and ECG findings. Due to the presence of symptoms and signs of organ damage and extremely elevated BP, the diagnosis of hypertensive emergency was first considered. Possible diagnosis was acute myocardial ischemia or infarction (AMI), although ECG did not show a myocardial ischemic pattern. However, a normal ECG may be present in up to one-third of patients with coronary involvement.[1] Another diagnostic possibility was acute aortic dissection (AAD). Nevertheless, about 20% of patients with type A AAD have ECG evidence of acute ischemia or AMI[1] and/or wall motion abnormalities (WMAs) detected by TTE (particularly LV inferior hypokinesia). These figures highlight the need to rule out AAD, even in classic cases of AMI. Two other potentially fatal diagnoses could be pulmonary embolism and esophageal rupture. However, these two diagnoses are less likely due to the elevated BP, normal right ventricle [RV] size and function, no hypoxemia, and no evidence of pleural effusion on ultrasound, respectively.

As discussed in Video 3, a TTE, subcostal, four-chamber view (Video 1, Clip 1), although limited to some segments of basal-midventricular regions, suggested normal LV and RV size and systolic function. In the subcostal longitudinal view (Video 1, Clips 2, 3), an enlarged aorta was noted (2.65 cm [normal ≤ 2 cm]) with an intimal flap seen inside, compatible with aortic dissection. A parasternal long-axis view (Video 1, Clip 4) showed normal LV systolic function and no WMAs. The aortic root was dilated (4.3 cm [normal ≤ 3.5 cm]) with no intimal flap seen inside, and a trace aortic regurgitation (AR) jet was revealed on color Doppler imaging. A moderate to severe acute AR is present in nearly one-half of patients with type A aortic dissection. Accordingly, the absence of significant AR does not rule out the diagnosis of this type of dissection. Apical four- and two-chamber views (Video 1, Clip 5) showed normal RV and LV systolic function with no WMAs. In a modified apical two-chamber view (Video 1, Clip 6), the medium-distal portion of the descending thoracic aorta was seen behind the left atrium, with an intimal flap inside and two lumina noted. No pleural or pericardial effusion was noted. Pericardial effusion could be a complication of type A dissections and pleural effusion of type B dissections, both indicating aortic rupture into these cavities. Because the cranial portion of ascending aorta, aortic arch, and proximal thoracic descending aorta are not seen with the conventional TTE views, the next logical step would be to perform a suprasternal view to determine if a type A (involving the ascending aorta) or a type B dissection (not involving the ascending aorta) is present.

Video 2 shows the distal portion of the ascending aorta and aortic arch with no intimal flap inside. Color Doppler ultrasound revealed antegrade flow (red, toward the transducer) in the ascending aorta. Blue flow (away from the transducer) was noted corresponding to the venous brachiocephalic trunk flow. This flow should not be misinterpreted as a false lumen. A normal posterior aortic wall (arrow) must not be misinterpreted as an intimal flap. The aortic arch was normal in diameter (2.7 cm [normal ≤ 3 cm]) with no intimal flap inside. The proximal descending aorta was enlarged (3.17 cm [normal ≤ 2.5 cm]). No double lumen was noted, although echogenic material was seen in the medial side (arrows with question sign), suggestive of a hematoma formation.

Because the ascending aorta was not compromised, Stanford type B classification of a dissecting aortic aneurysm was presumably diagnosed. Thoracic and abdominal CT scans corroborated these findings (Figs 1-3).

AAD is a challenging clinical emergency. The incidence of AAD has been estimated to range from 2.9 to 3.5 occurrences per 100,000 person-years, whereas the

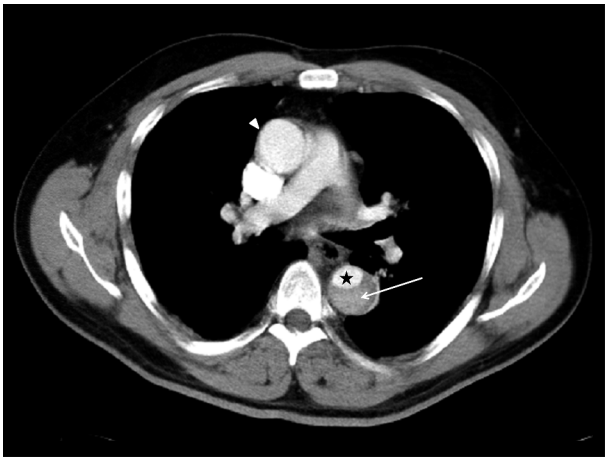

Figure 1 – *Chest CT scan in transverse section demonstrating a false lumen (arrow) and a true lumen (black star) within the proximal descending thoracic aorta. The ascending aorta is not involved (arrowhead).*