Dear editor,

Although the average age of diagnosis for aortic dissection is 63,[1] this case demonstrates the need to consider AD in younger patients, particularly in the presence of risk factors or in the absence of another reasonable diagnosis. This case suggests PCP use as a precipitant for hypertension and sympathomimetic stress on the aorta. Despite high sensitivity, false negative CTA imaging for AD can occur. If pretest probability remains high, further imaging must be obtained. During resuscitation in the ED, focused point-of-care ultrasound can also assist with medical decision making.

CASE

A 43-year-old male presented to the emergency department via EMS with his wife because of altered mental status and chest pain. The patient was intermittently agitated and his wife provided most of the history. The patient used PCP chronically and had been using PCP daily for two weeks after losing his job. His wife stated that on the morning of presentation he inhaled PCP then went to a local department store, where he complained of sudden onset chest pain and dyspnea. He became anxious and walked into the parking lot, where he collapsed. Per bystanders, he was arousal but altered, without seizure activity. His wife stated that he had not complained of chest pain prior to this episode, nor had he had any fevers, cough, headache, or other illness over the past few weeks. The patient stated he felt as though someone was stepping on his chest, with gradually worsening pain. He admitted to heavy alcohol use the prior night as well. Past medical history was significant for hypertension as well as a prior hospitalization one year ago for pneumonia, during which PCP use was also noted. His wife stated he was noncompliant with his amlodipine and didn’t take any other medications or other illicit drugs. There was no history of recent trauma, surgeries, or travel.

The patient was initially hypotensive with a blood pressure of 69/46 mmHg, HR of 80–110 beats/minute, temperature of 36.3 °C, and SpO2 of 96% on room air. He was confused and agitated, intermittently alert and answering simple questions but with waxing and waning responsiveness. He was mildly diaphoretic. The trachea was midline. Work of breathing was normal with equal, clear breath sounds. Cardiac examination demonstrated regular rate and rhythm without murmurs or extra heart sounds. There was no JVD or pitting edema. He was moving all extremities with normal strength and coordination; no focal neurologic findings were detected. Other systemic physical examinations were unremarkable.

An IV fluid bolus started immediately. The patient was treated with oxygen, IV morphine, and IV diazepam, with some improvement in his agitation. ECG revealed sinus tachycardia (rate 112) and a borderline prolonged QTc interval (502 ms) with otherwise normal intervals and no ischemic changes or ectopy. Glucose was 167 mg/dL. Portable chest X-ray demonstrated widened mediastinum and prominent right mediastinal and hilar contour (Figure 1), along with patchy interstitial opacity of the right midlung. Broad spectrum antibiotics were initiated, although the chest X-ray was consistent with residual changes from pneumonia on previous admission. The basic metabolic and coagulation profile was unremarkable. The complete blood count was significant for a white blood cell count of 12.8 thousand/mcL with a hemoglobin of 12 gm/dL. Urine toxicology screen was positive for PCP.

After 3 liters of IV normal saline the patient remained tachycardic to 110 and hypotensive with systolic blood
pressures in the 80s. He was still agitated, leading to clinical concern for PCP intoxication versus a central nervous system event. He was intubated for hemodynamic instability, airway protection, and agitation interfering with further diagnostic testing. Central venous access was obtained via the right internal jugular vein, dopamine and midazolam drips were initiated, and a right femoral arterial line was placed for accurate hemodynamic monitoring. Post-intubation ABG demonstrated acidosis (7.23/45/118) and a lactate of 1.9 mmol/L. Head CT was unremarkable. There was concern for aortic dissection given the chest pain, chest X-ray, sympathomimetic use, and hemodynamic instability, so an emergent CTA of the chest, abdomen, and pelvis was obtained. Although there was no aortic dissection flap or irregularity of contour in the chest or abdomen, this study demonstrated a prominent aortic root and moderate pericardial effusion. The pericardial effusion was hyperdense, consistent with blood, inflammatory, or infectious material.

The pericardial effusion heightened suspicion for aortic dissection. An emergent bedside transthoracic cardiac echo (TTE) did not demonstrate evidence of dissection although large pericardial effusion was confirmed, along with right ventricular diastolic collapse, consistent with tamponade (Figure 2). Concurrently, pulsus paradoxus was noted on the arterial line tracing. A stat formal echo was obtained at bedside, which confirmed large pericardial effusion with diastolic right ventricular and atrial collapse, along with moderate eccentric aortic regurgitation jet (Figure 3).

![Figure 2. Tamponade on bedside TTE. Emergent ED transthoracic cardiac echo (subxyphoid view) visualized a pericardial effusion (>) and RV collapse (*) during diastole.](image1)

![Figure 3. Aortic regurgitation confirmed by formal TTE. Formal transthoracic echo (parasternal long view) visualizing moderate eccentric aortic regurgitation. The aortic outflow tract (>) is shown to have bidirectional flow with color Doppler.](image2)

![Figure 4. Dissection flap on TEE. Dissection flap (<) and false lumen (*) of the aortic root seen on transesophageal echo in cath lab. The aorta (A) and aortic valve (AV) are also shown.](image3)

![Figure 5. Severe aortic regurgitation on TEE. The transesophageal echo also demonstrated severe aortic regurgitation.](image4)

Figure 1. Widened mediastinum on CXR. In the appropriate clinical context, a widened mediastinum (more than 8 cm at the level of the aortic knob) on CXR strongly suggests the need for additional aortic imaging. However, 40% of CXR in acute aortic dissection lack a widened mediastinum, and as many as 12%–37% are normal.[15,16] Other CXR findings suggestive of AD include pleural effusion, cardiomegaly, aortic contour abnormalities, inwards displacement of aortic wall calcification >10 mm, displacement of the trachea, mainstem bronchi, or esophagus (nasogastric tube), or aortic intimal calcification.[16]
Given the tamponade physiology, aortic regurgitation, and prominent aortic root on CTA, interventional cardiology was consulted for emergent drainage of the pericardial effusion in the cardiac catheterization laboratory, with concomitant transesophageal echo (TEE) to clarify the potential diagnosis of aortic dissection. TEE did reveal a flap of the aortic root and proximal aorta (Figures 4, 5) and the patient was emergently transferred to the OR for prosthetic aortic valve and root replacement. The patient was transferred to the ICU and was discharged home after an uncomplicated hospital course.

DISCUSSION

To our knowledge, there are no other published cases of aortic dissection in a patient using PCP. PCP is a noncompetitive antagonist of the NMDA receptor and also blocks the reuptake of biogenic amines, including dopamine and norepinephrine. The sympathomimetic toxidrome is a possible mechanism for aortic dissection, as cases of AD in patients using cocaine, methamphetamine, MDMA, and caffeine have been described. Cocaine has been proposed to increase the risk for dissection due to chronic atherosclerosis, however this has not been described with PCP. Another contributor to hypertension in acute PCP intoxication may be psychomotor agitation, present in 34% of patients with PCP intoxication.

Risk factors for aortic dissection include conditions associated with increased aortic wall stress and aortic media abnormalities. The former include hypertension, pheochromocytoma, cocaine or other stimulant use, weight lifting or valsalva, trauma, aortic coarctation, and bicuspid aortic valve. The latter include connective tissue disorders, inflammatory and infectious conditions, and polycystic kidney disease. Other predisposing factors include pregnancy, family history of AD, and aortic surgery or balloon pump. At the time of presentation, our patient’s known risk for AD was hypertension, which is a predisposing condition in 50%–70% of patients with aortic dissection. His PCP use also raised concern for a vascular catastrophe. Family history was later discovered to be significant with aortic aneurysm in his father and two brothers. Of the 7% of patients diagnosed with AD under 40 years of age, 50% have a history of Marfan syndrome, and 13% to 19% of patients without an identified genetic syndrome had first-degree relatives with thoracic aortic aneurysms or AD. Younger patients in general are less likely to have a history of hypertension and significantly more likely to have Marfan syndrome, bicuspid aortic valve, or a history of prior aortic surgery.

CTA (multidetector computed tomography with arterial contrast), magnetic resonance imaging (MRI), and TEE offer comparable sensitivities and specificities for the diagnosis of aortic dissection. Imaging modality is chosen based on the individual patient and institutional capabilities, with CTA as the recommended initial imaging choice in the ED due to its availability, rapid imaging of the entire aorta, and ability to detect alternative diagnoses. Diagnostic features of AD on CTA include intimal flap (a lucent strip or displacement of intimal calcification), double lumen (true and false), and dilation of the aorta (aneurysm). CTA is helpful for surgical planning as it can visualize the origin and extent of the dissection flap, including valve or branch vessel involvement or other distal complications. Sensitivity and specificity reports range from 94%–100% and 77%–100% respectively.

CTA requires exposure to radiation and IV iodinated contrast. In patients with significantly impaired renal function or known contrast allergy, TEE and MRI are alternatives, however these are less available and less efficient. ED TEE is infrequently available, but is an excellent choice for a hemodynamically unstable patient. Limitations of TEE include user-dependency, sedation requirement, relative contraindication in esophageal disease, and a 1-cm blind spot in the distal ascending aorta anatomically proximal to the innominate artery. Limitations of MRI in the acute setting include accessibility, longer imaging times, and monitoring challenges. MRI avoids radiation exposure, and is often used for serial imaging of known aortic disease.

Our CTA revealed a widened aortic root but no dissection flap. The diagnosis of aortic dissection cannot always be definitely excluded based on the results of a single imaging study. Although CTA, TTE, and MRI are all highly accurate, false negative studies do occur. In selected cases, especially when indirect signs of AD are present, a second imaging study should be strongly considered.

False negative CTA studies for AD can be caused by inadequate contrast opacification or a chronic aortic dissection with peripheral calcification simulating an atherosclerotic aneurysm. However, CTA is generally a very sensitive test and false negatives are rare. False positives occur more commonly and can be due to curvilinear artifacts in the aortic root and proximal ascending aorta caused by aortic wall motion, which is reduced by ECG-gating.
Bedside, point-of-care transthoracic echocardiography is not recommended to definitely diagnose AD. Limitations include inadequate sensitivity, user-dependency, and inability to image the entire descending aorta. The sensitivity and specificity of formal TTE for AD range from 77%–80% and 93%–96%, respectively, for the ascending aorta; sensitivity is 70% for distal dissection of the thoracic aorta. However, this case demonstrates that when used in conjunction with more definitive imaging, point-of-care TTE in the ED can be useful in clinical decision-making. Bedside TTE is efficient, repeatable, and allows the patient to stay in the acute resuscitation environment of the ED. This is necessary in the 6%–20% of ADs that present hemodynamically unstable from shock or heart failure secondary to severe aortic insufficiency, cardiac tamponade, myocardial ischemia or infarction, true lumen compression by a distended false lumen, or aortic hemorrhage in the thorax or abdomen. Bedside TTE can provide direct visualization of the flap via the parasternal long view of the aortic root, suprasternal view of the ascending and proximal descending arch including the origins of the head and neck vessels, and abdominal view of the descending aorta. Color Doppler flow can help identify flaps and false lumens or secondary findings such as aortic regurgitation or pericardial effusion causing tamponade, present in 32%–76% and 8%–10% of ADs, respectively. One study found that 93.5% of ADs are found to have dilated proximal aortic diameter >4 cm, of which emergency physician focused TTE identified 71.4%, resulting in faster diagnosis and less misdiagnosis.

CONCLUSION

AD may manifest as chest pain, back pain, abdominal pain, neurologic deficit, or syncope, as well as with evidence of other end-organ ischemic and mechanical dysfunction. AD is rare, but the risk for morbidity and mortality is great. The age distribution is bimodal; the most common risk factor for elderly patients is hypertension, while younger patients typically have another risk factor, such as medial wall abnormality or sympathomimetic toxidrome. This is the first case reported of AD in a patient using PCP. This patient had a history of hypertension and a family history of aortic disease so a direct association between PCP and the AD cannot be assumed, however PCP use on the day of the catastrophe and PCP’s known sympathomimetic properties suggest it could be a contributor. No single historical feature, physical examination finding, or laboratory test can safely rule out AD. CTA is usually the initial imaging test and is generally adequately sensitive, however false negatives do occur. ED ultrasound has several advantages, particularly in the setting of a suspected false negative CTA or a hemodynamically unstable patient that cannot go to the CT scanner. TTE can be performed before or after CTA for dynamic reassessment at the bedside, with special attention to indirect signs of AD which wouldn’t necessarily be appreciated on CTA such as pericardial effusion and aortic regurgitation.

ACKNOWLEDGEMENTS

The work should be attributed to Advocate Christ Medical Center, Department of Emergency Medicine.

Funding: None.
Ethical approval: Not needed.
Conflicts of interest: The authors declare that no competing interest and no personal relationships with other people or organizations that could inappropriately influence their work.
Contributors: DD proposed the study and wrote the first draft. All authors read and approved the final version of the paper.

REFERENCES


www.wjem.org

Received June 6, 2017
Accepted after revision October 26, 2017