CT and MRI of Acute Thoracic Cardiovascular Emergencies

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A wide spectrum of acute cardiovascular disorders is seen in patients who are hospitalized in a critical care setting. These consist of several acquired conditions, including aortic dissection, venous thromboembolism, pericardial compromise, myocardial infarction, and acute coronary syndrome. Imaging plays a central role in the diagnosis and management of these conditions. The most frequently used imaging remains chest radiography; however, more advanced modalities, including coronary angiography, echocardiography, and radioisotope scintigraphy, have well established roles in the assessment of patients in the critical care setting. More recently, multidetector row CT (MDCT) and MRI are being used increasingly for evaluation of coronary artery disease (CAD), cardiac structure and function, coronary artery anomalies, cardiac masses, pericardial disease, valvular disease, postoperative cardiovascular abnormalities, venous thromboembolism, and acute aortic syndromes, often with other ancillary findings that can provide important clinical information [1]. Cardiac MRI can evaluate cardiac function accurately by cine gradient echo imaging of the ventricles and flow analysis across cardiac valves and the great vessels and evaluation of cardiac wall motion, ventricular volumes, and ventricular mass [2]. Although MR angiography techniques are well established for evaluating the aorta, CT is preferred in unstable patients. MDCT is readily available in most places around the clock, often with in-house CT technologists, and provides rapid imaging assessment of cardiovascular structures in the thorax.

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The three most common life-threatening cardiovascular processes in which advanced imaging plays a role, particularly CT, are discussed, including pulmonary embolism (PE), aortic dissection, and CAD.

Acute pulmonary embolism

Acute PE is associated with high morbidity and mortality, particularly in the acute care setting. It is the third most common cause of cardiovascular death after myocardial infarction and stroke [3]. At postmortem examination, PE is found in 7% to 27% of patients who had been in the ICU and contributes to or is the cause of mortality in up to 12% of patients [4].

The incidence of PE has remained constant, with age- and sex-adjusted rates of 117 cases per 100,000 person-years [5]. The incidence increases sharply after age 60 years in men and women [6]. The mortality associated with PE is highest in the first 3 months following the event and exceeds 15% [7]. The initial clinical manifestation is sudden death in almost one fourth of patients who have acute PE [5].

Although there are a myriad of risk factors associated with acute PE, many of them are common in an acute care or intensive care setting, some predating the ICU admission and others developing over the course of the ICU stay. These include prolonged immobilization, increased age, surgery, trauma, shock, stroke, malignancy, pancreatitis, and coagulation abnormalities, such as polycythemia, platelet abnormalities, and history of venous thrombosis. Pregnancy, oral contraceptive use, and smoking also are associated with a higher risk for PE. Patients in the ICU have more baseline risk factors for PE than do patients who are not in the ICU. These risk factors include age older than 70 years, bed rest for 5 days or longer, and a diagnosis of cancer, chronic obstructive pulmonary disease, or congestive cardiac failure [8,9]. The prevalence of deep vein thrombosis (DVT), at 13% to 33%, also is higher in patients who are admitted to the ICU than in patients who are not, regardless of whether they are receiving DVT prophylaxis [4,10,11]. In one study, a DVT rate of 33% was reported, despite DVT prophylaxis in 61% of the patients [10], whereas in another study of 102 patients in the ICU who specifically were defined as high risk for DVT and all were receiving prophylaxis, the rate of DVT was 12% [12].

Diagnosis of acute pulmonary embolism in the critical care setting

The diagnosis of acute PE in patients in the ICU can be challenging for many reasons and requires an integrated approach using clinical history, physical examination, laboratory data, and imaging. The clinical signs and symptoms are nonspecific and may be absent or masked by other disease processes. The diagnosis is complicated by coexisting diseases. Patients commonly present with dyspnea or tachypnea, often associated with pleuritic pain. Nonproductive cough and hemoptysis can occur if there

has been pulmonary infarction; however, this is uncommon. Syncope may occur with massive PE, but also with a lesser extent of PE in patients who have impaired cardiopulmonary reserve. On physical examination, tachypnea is a common finding. If cyanosis is present, it usually indicates massive PE. With smaller emboli, pleural effusions, pleural rub with wheeze, and crackles may be present. Lower extremity edema is found in only a third of patients who have acute PE. The major differential diagnoses to consider in this setting include acute myocardial infarction, heart failure, pneumonia, pneumothorax, and an acute aortic syndrome.

When normal, the D-dimer assay has a high negative predictive value of 95.6% to 96.7% for the absence of venous thromboembolism (VTE) [13]. An elevated D-dimer has a low specificity for VTE, ranging from 35% to 77% [14]. Elevated D-dimer can be seen in many acute systemic conditions that may be present in patients in the ICU, including myocardial infarction, pneumonia, sepsis, cancer, and after surgery [14,15]. Chest radiography—although the most frequently performed imaging examination in patients in the ICU—is of little value in the diagnosis of PE with its low specificity, and it often is confounded by coexisting infection, edema, or acute respiratory distress syndrome (ARDS) [16].

Traditionally, ventilation/perfusion (V/Q) scanning has been the mainstay of evaluation, with catheter pulmonary angiography serving as the gold standard or reference test. The presence of pulmonary disease in most critically ill patients makes V/Q scanning limited in its diagnostic value. In the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study, most patients (73%) had indeterminate (34%) or low (39%) probability V/Q scans, of which 33% and 12%, respectively, had PE. Only 13% of patients had a high probability scan result, in which the prevalence of PE was 88% [17]. From the abstract of that publication, "Almost all patients with pulmonary embolism had abnormal scans of high, intermediate, or low probability, but so did most without pulmonary embolism (sensitivity, 98%; specificity, 10%)." Most of these patients were not patients in the ICU. Coexisting lung disease increases the likelihood of an indeterminate test by virtue of the interpretation criteria. When a perfusion defect is present in the setting of a radiographic opacity, a low probability test result is converted into an intermediate result. In the ICU setting, only a combination of a low clinical and a low or very low scintigraphic probability renders the diagnosis of PE highly unlikely [18]; the only advantage is that it is possible to perform scintigraphy at the bedside of unstable patients. Catheter angiography has been recognized to be an imperfect gold standard, with considerable interobserver variability at the small artery level [19].

Over the last decade, intravenous contrast-enhanced CT pulmonary angiography (CTPA) has emerged as the single most important imaging modality for the diagnosis of acute PE. CTPA is readily available, and the images are available for review in a matter of minutes. This reduces the time to make the diagnosis and management. The sensitivity and specificity of MDCT

pulmonary angiography combined with indirect lower extremity CT venography, as reported recently in the PIOPED 2 study—the largest study of MDCT accuracy for PE—are 90% and 95%, respectively [20]. Several other studies showed a high sensitivity and specificity for CTPA of 90% to 100% and 89% to 94%, respectively, and a high negative predictive value of 98% to 99% [21–23]. Baile and colleagues [24] compared CT and catheter angiography in a porcine model for detecting subsegmental emboli, finding no difference in the sensitivity and specificity of the two modalities for detecting PE.

It is important to consider the specificity of CTPA (95%–97%) [22,23] when PE is found, which allows treatment with a high degree of confidence in the diagnosis, as well as the high negative predictive value and the clinical outcome after a normal CT result. Patel and Kazerooni [25] summarized 18 studies in which 4233 patients with a normal CTPA examination were followed from 3 to 12 months. The weighted average occurrence of venous thromboembolic disease was 1.3%, and the weighted average of fatal PE was 0.4%. By comparison, the rate of PE after a normal catheter pulmonary angiogram is 1.6% to 1.7% [26]. Many thoracic radiologists consider CTPA, not catheter angiography, to be the reference standard for evaluating the pulmonary arteries. This is because catheter angiography is a projectional technique in which a limited number of views are obtained because of the contrast volume required for each injection and radiation concerns, small filling defects are difficult to detect, and even with expert readers, there is considerable interobserver variation when interpreting the subsegmental and smaller arteries [14,19]. In one porcine model study, catheter angiography had a false negative rate of 20%, attributed in many cases to partially occluding thrombi [27].

The use of CTPA in the ICU setting has been questioned [28,29], as has the accuracy of CTPA when there is coexisting lung disease, such as pneumonia, edema, or ARDS. Imaging is complicated further by factors such as tubes and lines, metallic hardware, and impaired cardiopulmonary function, causing streak artifacts and suboptimal contrast delivery. Remy-Jardin and colleagues [30] demonstrated that CTPA performed equally well in patients who did and did not have coexisting lung disease. In a study by Kelly and colleagues [11] specifically of patients in the ICU undergoing CTPA using 4-row MDCT scanners, diagnostic quality images were obtained in most patients (76%); images in the remaining 24% were nondiagnostic, highlighting the challenges in this population. Advances in scanner technology since that time, particularly 16- and 64-row scanners that allow the examination in be acquired in as little as 5 seconds, improve image quality by reducing respiratory motion. Additional strengths of CT are that it can evaluate the lung and pleural disease, which often coexists in patients in the ICU and may be the actual cause of an acute clinical deterioration, as well as the aorta and heart in the same acquisition.

In these high-risk patients, a normal CTPA effectively rules out an acute thromboembolic event. Bourriot and colleagues [31] evaluated the clinical

outcomes following a normal CTPA in 117 patients: 70% had a known cardiopulmonary disease and 36% had impaired cardiopulmonary reserve. The rate of recurrent PE in these patients was 1.8% to 4.9%, depending on the defining criteria used. This low recurrence confirms the usefulness of CTPA in excluding PE in patients who are being managed in the critical care setting.

CT pulmonary angiography: technique, image reconstruction, and interpretation

CT angiography of the pulmonary arteries is performed with 80 to 130 mL of iodinated contrast material injected through an antecubital vein at a rate of 4 mL per second. Using a 16-detector row CT scanner, this takes 10 to 12 seconds; with a 64-detector row scanner, it takes less than 5 seconds to complete a high-resolution examination of the entire thorax with collimation of approximately 1 mm. This means that the examination can be performed in a single breath hold, minimizing respiratory motion artifact. For an intubated patient, this minimizes the time that the ventilator is suspended for the image acquisition. With optimal enhancement of the pulmonary arteries, emboli in the main trunk down to subsegmental arteries can be visualized easily (Fig. 1). In situations in which the visualization of pulmonary artery filling defects is doubtful or difficult because of breathing or streak artifacts, multiplanar reformats can be generated on the workstation to review the artery in any desired plane, which may enhance diagnostic confidence (Fig. 2).

After the thoracic part of the examination, the veins of the pelvis and thighs are scanned after an additional 2 to 3 minutes as an indirect CT venogram (CTV) to identify DVT (Fig. 3). Scans are obtained from the iliac crests to the tibial plateaus as a contiguous acquisition using 5- or 7.5-mm
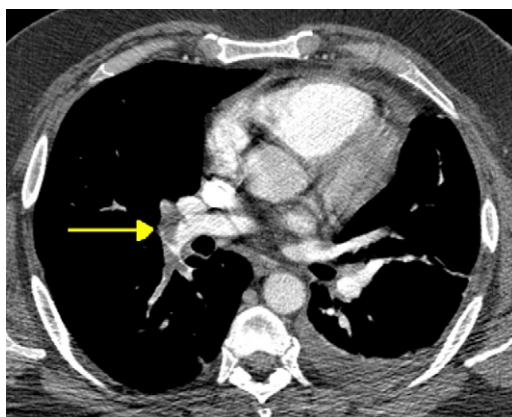

Fig. 1. A 55-year-old man who had sudden-onset chest pain. CTPA demonstrates emboli in right upper lobe lobar, segmental, and subsegmental arteries (*arrow*).
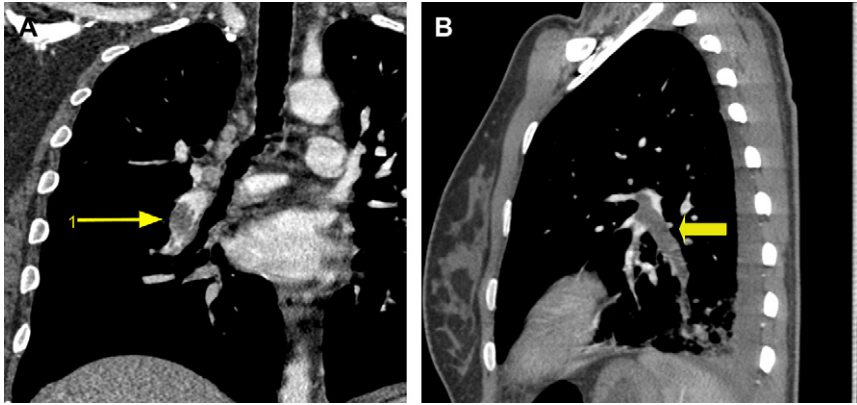
Fig. 2. Multiplanar reformatted images in the coronal (*A*) and sagittal (*B*) planes demonstrate a large embolus in the left lower lobar pulmonary artery and distal branches (*arrow*).

collimation. This one-stop CTPA combined with CTV essentially eliminates the need for a separate ultrasound of lower extremities, reducing the cost and time for the diagnostic workup. Furthermore, it increases the diagnostic yield of the CT examination for disease. In the PIOPED II study, the sensitivity for VTE increased from 83% to 90% when CTV was considered with CTPA [20]. Several other studies showed an excellent correlation between indirect CTV and ultrasound in studies in which patients prospectively underwent both tests, with sensitivity and specificity ranging from 89% to 100% and 94% to 100%, respectively, and a negative predictive value of 97% to 100% [32–38]. The reported interobserver agreement also is good to excellent, with kappa values of 0.59 to 0.88 [35,37,39]. Therefore, combining CTV with CTPA increases confidence in the diagnosis of venous thromboembolic disease. This is particularly useful in patients in the ICU
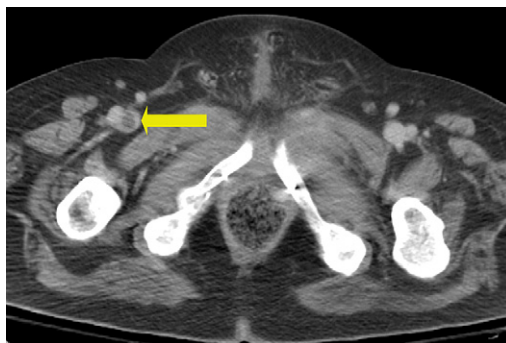

Fig. 3. Indirect CT venography in a patient who recently underwent abdominal surgery and developed lower extremity swelling demonstrates a thrombus in the right superficial femoral vein (*arrow*).

who may not need anticoagulation treatment when results for the pulmonary arteries and leg veins are normal.

Aortic dissection

Aortic dissection occurs most commonly in adults between the ages of 40 and 70 years, with an incidence of 1 to 6 cases per 100,000 per year. It is two to five times more common in men. Risk factors for aortic dissection include hypertension, pregnancy, coarctation of the aorta, bicuspid aortic valve, Marfan syndrome, Ehlers-Danlos syndrome, Bechet's disease, and prior cardiac/aortic surgery [40]. Given the 1% to 2% per hour mortality after symptom onset for the first 24 hours for type A dissection and a 30-day mortality of 10% for type B dissection, early diagnosis is imperative to avoid significant morbidity and mortality.

An aortic dissection is produced when there is penetration of circulating blood into the wall of aorta, through a tear of the intima, for a varying degree. Any mechanism that weakens the media of the aorta may result in aortic dilatation and aneurysm formation and, eventually, intramural hemorrhage, aortic dissection, or rupture [40]. The vessel walls can be affected by congenital connective tissue disorders, such as Marfan's syndrome and Ehlers-Danlos syndrome. Acquired conditions, such as chronic hypertension, may cause aortic aneurysm and dissection [41,42]. Inflammatory processes of the aortic wall or autoimmune processes involving the vasa vasorum supplying the aortic wall, such as Takayasu arteritis, giant cell arteritis, syphilis, and Behcet's disease, lead to weakening, expansion, and dissection, [40]. Iatrogenic aortic dissection can be caused by valve surgery, graft anastomosis, and the cannulation sites, as well as catheter placements [43]. Deceleration trauma, like car accidents and fall from height, can cause aortic dissection, pseudoaneurysm, and rupture, usually at the distal aortic arch just beyond the origin of left subclavian artery. Intramural hematomas of the aorta can lead to a secondary tear on the intima and communicate with the aortic lumen [44].

Aortic dissections generally are classified with respect to what part of the aorta is involved. In the DeBakey classification, type I involves the ascending and descending aorta, type II involves the ascending aorta only, and type III involves the descending aorta only, distal to left subclavian artery [45]. In the more commonly used Stanford classification, a type A dissection is defined as involving the ascending aorta, and type B dissection spares the ascending aorta.

Clinical presentation

Acute-onset chest pain in the midline, radiating to the back, is the most common presenting complaint. The onset usually is sudden and reaches maximal intensity immediately. This abruptness is the most specific characteristic

of the pain. The pain is characteristically described as ripping, tearing, choking, or stabbing; it does not commonly radiate to the neck, shoulder, or arm and may be absent in 5% to 10% of cases. Many patients are hypertensive because of preexisting hypertensive disease or increased sympathetic drive.

Clinical findings vary depending on branch artery involvement of different organ systems, due to ischemia secondary to obstruction of branches of aorta, direct compression of organ by expanding false lumen, or leak or rupture of false lumen into surrounding structures. The most common findings are due to cardiovascular and neurologic involvement when coronary arteries or aortic arch branches are involved. Cerebral ischemia and stroke is the most common feature. Syncope and myocardial infarction may be seen with coronary artery involvement. Spinal cord lesions are more common with distal dissections and can cause paraplegia. There may be pulse and blood pressure differential between the two arms when the dissection extends into or obliterates the arch vessels [46]. Similarly, acute renal failure can occur with renal artery involvement, and mesenteric ischemia can occur with celiac axis and mesenteric arterial involvement.

Differential diagnosis mainly includes acute myocardial infarction and PE. These can be evaluated easily with a single MDCT scan. Other conditions to consider in the differential include mesenteric arterial or venous thrombosis, peptic ulcer, acute appendicitis, intestinal obstruction, pancreatic/peritoneal cyst, and acute cholecystitis. Conditions associated with aortic dissection, such as hypertension and connective tissue disorders, can be helpful in narrowing down the diagnosis.

Diagnosis and advanced imaging

Aortic dissection should be considered in any patient presenting with sudden-onset severe chest pain. A chest radiograph may show a widened mediastinum, irregular aortic contour, deviation of the trachea or the nasogastric tube in the esophagus, and displacement of calcified intima; however, chest radiograph alone does not confirm the diagnosis and is nonspecific [47]. Historically, invasive catheter aortography was the definitive diagnostic modality. With advances in CT imaging, CT has replaced catheter angiography in the diagnostic evaluation of the aorta; it is best performed as an ECG-gated CT on a 16- or more detector row scanner, which eliminates aortic pulsation artifact. CT can quickly and noninvasively evaluate the true and false lumens and the intimal flap, including entry and reentry tears, as well complications, such as pericardial and pleural effusions and branch artery involvement. MDCT also is helpful in identifying causes of mediastinal widening as seen on chest radiograph other than dissection, such as mediastinal hematomas secondary to central line placement, mediastinal masses, and aortic aneurysms.

Several studies demonstrated that CT is a highly accurate and reliable imaging modality for aortic dissection. In a study by Yoshida and colleagues

[48], the accuracy of CT for the detection of aortic dissection or intramural hematoma of the thoracic aorta was 100%. The sensitivity, specificity, and accuracy, respectively, were 82%, 100%, and 84% for locating the entry tear; 95%, 100%, and 98% for arch branch vessel involvement; and 83%, 100%, and 91% for pericardial effusion. All of these values were 100% for aortic arch anomalies. In a more recent study, Hayter and colleagues [49] evaluated 373 patients who had suspected aortic dissection with MDCT. There were no false positives, 1 false negative, 76 true positives, and 304 true negatives, yielding a sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy of 99% (67 of 68), 100% (304 of 304), 100% (67 of 67), 99.7% (304 of 305), and 99.5% (371 of 373), respectively. Other studies have reported 100% sensitivity and specificity of MDCT to detect aortic dissections [50,51].

On CT, the primary finding in aortic dissection is the presence of two distinct lumens with a visible intimal flap, which is seen in most cases (Fig. 4); in other cases, the two lumens are identified only by their differing rates of opacification with contrast material or the low attenuation of the false lumen if it is completely thrombosed. An intramural hematoma is a variant of a classic dissection in which only a thickened wall is present, and there are no entry or reentry tears (Fig. 5). One explanation for this is rupture of the vaso vasora that supply the aortic wall. CT examinations done for acute aortic syndromes routinely include noncontrast images first, because acute blood in the aorta appears higher in attenuation than does the blood in the aortic lumen, whereas long-standing hematoma will not (Fig. 6). Indirect signs of dissection include compression of the true lumen by the false lumen, spiraling of a thrombosed false lumen, displaced intimal calcification, widening of the aortic lumen, and ulcerlike projections of contrast material [52]. Three-dimensional reconstructions of the CT data are routine in evaluating the morphology of the dissection and relation to branch vessels
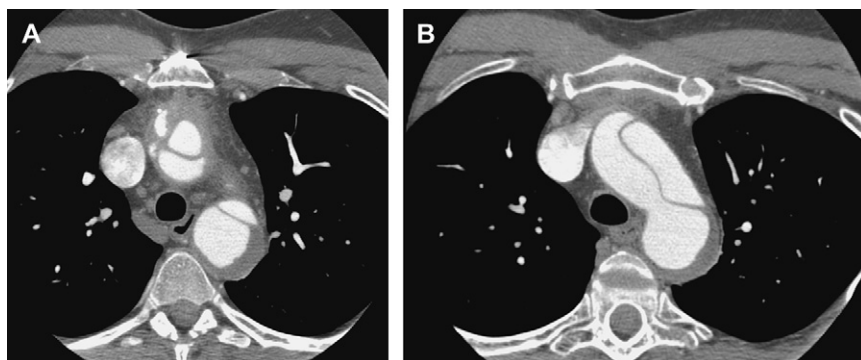

Fig. 4. Type A aortic dissection with intimal separating the true and false lumens at the ascending aorta (*A*) and at the aortic arch (*B*).
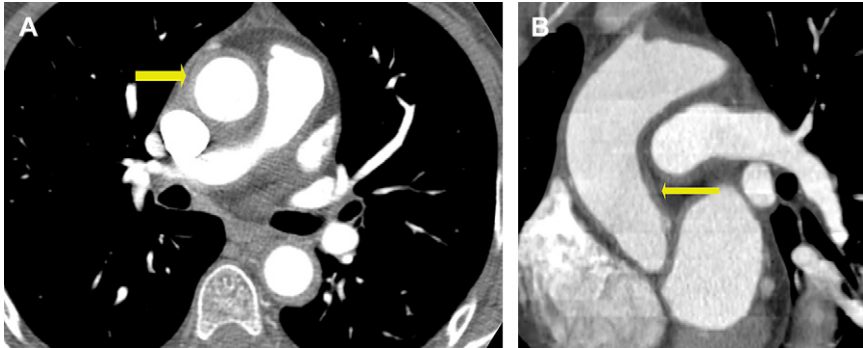
Fig. 5. Acute intramural hematoma manifests as thickening of the ascending and descending thoracic aortic wall (*arrow*), as shown on axial (*A*) and oblique coronal reformatted (*B*) images.

(Fig. 7), which are critical in clinical decision making, particularly when open or endovascular repair is necessary.

Patients who are unable to receive intravenous iodinated contrast for CT can be evaluated with MRI. It also is an accurate noninvasive technique for examining patients who are suspected of having aortic dissection, aortic intramural hematoma, or penetrating aortic ulcer; however, it generally is reserved for stable patients or follow-up imaging, because of the longer examination time and the logistics of doing an MRI examination in a patient who is unstable and requires close monitoring by the health care team. Although MRI has high sensitivity (95%–100%) and specificity (94%–98%) for the detection of aortic dissection, a sensitivity of 100% for detection of aortic intramural hematoma, and a sensitivity of 86% for detection of penetrating aortic ulcer, it has serious limitations [53–55]. Most importantly,
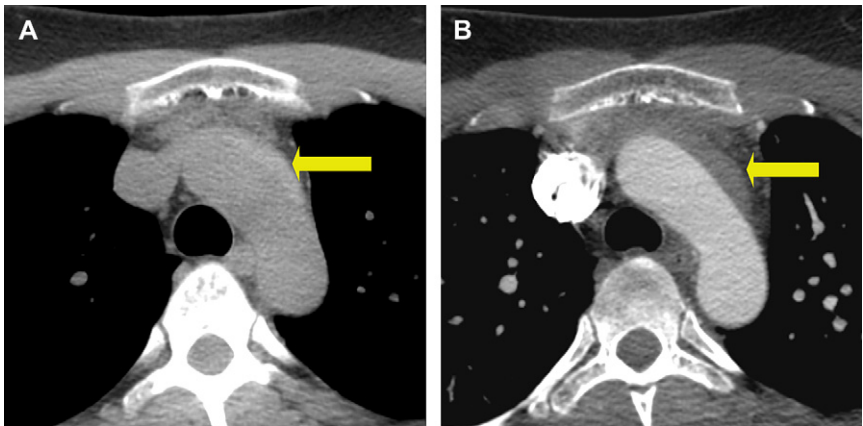

Fig. 6. Intramural hematoma of the aortic arch appears as high attenuation on a noncontrast enhanced image (*A*, *arrow*) relative to blood in the aortic lumen, indicating that it is acute, and as low attenuation thickening on a contrast-enhanced image (*B*, *arrow*).
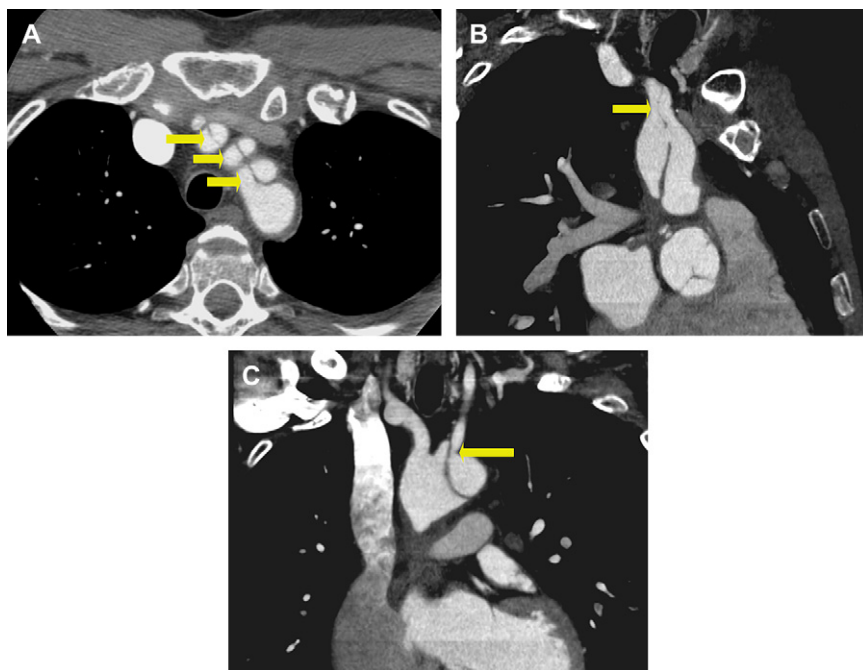
Fig. 7. Acute aortic dissection involving the branches of the aortic arch, as demonstrated on an axial image with flap extending into all three great vessels (*A*, *arrows*), flap extending into the innominate artery (*B*), and on left common carotid artery on oblique sagittal reconstructions (*C*).

the MRI examination requires approximately 30 minutes or more, compared with the 10 to 30 seconds to acquire the CT data on an MDCT scanner, for which more time is spent moving the patient in and out of the room than on the scan itself. CT scan data are reconstructed so quickly on modern consoles that a physician can review the images and make an assessment before a patient from the ICU and medical team are ready to leave the CT suite. The time it takes for an MRI scan (30 minutes) is a serious limitation, particularly in patients from the ICU who may be unstable, ventilated, or need constant monitoring and who may have MRI incompatible hardware. Also, MRI is less readily available and commonly is at a site at great distance from the ICU or in a remote area of a medical complex, which further limits its role in the diagnosis of an aortic syndrome in the acute setting [49].

Coronary disease and advanced imaging in the critical care setting

A wide variety of cardiac disorders can be found in patients in the ICU; however, CAD with myocardial infarction is among the most common acute conditions and contributes to a significant proportion of the mortality in these patients. The clinical signs and symptoms of an acute cardiac event

can be nonspecific (eg, atypical chest pain, nausea, shortness of breath, fatigue, cough, and diaphoresis) and masked by other disease processes. In patients in the ICU who frequently have a complicated clinical presentation, accurate and rapid diagnosis of an acute cardiac event can be challenging. Historically, the diagnosis of an acute coronary syndrome has been based on the ECG and cardiac enzymes, often with a noninvasive stress modality (eg, echocardiography or radionuclide single photon emission computed tomography [SPECT] imaging), with catheter coronary angiography as the final arbiter when the noninvasive test results are not conclusive or discordant with the pretest clinical probability of disease.

The clinical applications of cardiovascular nuclear imaging techniques in the intensive care setting have been well established. These include thallium[201] and Tc[99m]-sestamibi SPECT and Multigated (MUGA) studies to provide quantitative information concerning myocardial perfusion, acute myocardial ischemia, and left ventricular function. These techniques provide objective guidelines for therapy and prognosis [56]. The reported sensitivity and specificity of Tc[99m]-sestamibi SPECT to predict acute coronary ischemia are 94% and 84%, respectively [57,58]; however, the lack of on-site, around-the-clock availability and long examination times are real concerns for its use in patients from the ICU [59].

Rapid advances in MDCT technology over the last few years have greatly facilitated the accurate and rapid evaluation of the coronary arteries using 64-detector MDCT scanners to perform CT coronary angiography. Several studies reported high sensitivity and specificity of MDCT for detecting coronary artery stenoses of 50% or greater, ranging from 90% to 95% and 82% to 98%, respectively [60–65]. Perhaps the most important characteristic of CT coronary angiography is its consistently high NPV of 97% to 99% [22,23,61,62,66]. Mollet and colleagues [66], using vessel-based analysis with 64-slice computerized tomographic angiography (CTA) to detect stenoses of 50% or greater, reported sensitivity, specificity, PPV, and NPV ranging from 97% to 100%, 92% to 99%, 78% to 80%, and 99% to 100%, respectively, depending on the calcium score. The sensitivity for detecting significant disease in the left anterior descending was 96%, whereas in other main coronary arteries the sensitivity was 100%. There was good correlation between CTA and coronary angiography, with a kappa value of 0.85. Ehara and colleagues [67] evaluated 64-slice MDCT for detecting angiographically significant coronary artery stenosis in an unselected consecutive patient population and compared it with conventional invasive angiography. Fifty-seven percent of the patients already had coronary artery stents. They reported that sensitivity for diagnosing significant stenosis ($\geq 50\%$) was 90%, specificity was 94%, PPV was 89%, and NPV was 95%. For the stented arteries, the sensitivity, specificity, PPV, and NPV were 93%, 96%, 87%, and 98%, respectively.

Raff and colleagues [23] evaluated the diagnostic accuracy of 64-slice coronary CT angiography in 70 consecutive patients undergoing invasive

coronary angiography, including patients with high heart rates (23% > 70 beats per minute [bpm], range up to 96 bpm), obesity (50% with body mass index > 30 kg/m^2), and coronary calcification (26% had Agatston score > 400 with range up to 1804), reflecting a more "real world" group of patients. They demonstrated a high NPV of 98% by segment and 97% by artery. They also observed improved image quality with smaller voxel size provided by the 64-slice scanner, which reduced, but did not eliminate, the calcium blooming and beam hardening artifacts. The sensitivity and specificity were 97% and 95%, respectively, with heart rate of less than 70 bpm and 88% and 71%, respectively, with heart rate of 71 to 85 bpm, reinforcing the need to pharmacologically reduce the heart rate during the CT examination. This is done routinely with β-blocker and sometimes calcium channel blockers if there is a contraindication to the former. A high or irregular heart rate decreases the image quality of coronary CT angiography. Achenbach and colleagues [69] evaluated a new dual-source MDCT with the advantage of higher temporal resolution than other 64-detector MDCT scanners, which has made it possible to obtain good quality images at higher heart rates and has reduced, but not eliminated, the need to premedicate patients completely. They reported visualization of 98% of coronary artery segments free of cardiac motion artifacts.

There has been preliminary work on the use of coronary CT angiography in patients who have chest pain presenting to emergency rooms, who are stable clinically, low risk for CAD, and have a normal ECG and cardiac enzymes for at least 4 hours. Patients with a normal scan can be discharged early, leading to a reduction in length of stay and cost of care; unfortunately, the sample sizes have been too small to determine the impact of this strategy on coronary event rates, such as myocardial infarction and intervention [57,68,69]. In a recent study, Rubinshtein and colleagues [70] demonstrated emergency department (ED) MDCT sensitivity of 100%, specificity of 92%, PPV of 87%, and NPV of 100% in a cohort of 58 patients. They concluded that 64-slice cardiac MDCT represents a valuable diagnostic tool in patients in the ED who have chest pain of uncertain origin, providing early direct noninvasive visualization of coronary anatomy.

Technique

Cardiac imaging using CT is a technically demanding procedure, requiring high temporal and spatial resolution to visualize small coronary arteries while the heart is beating continuously. This is achieved using retrospective ECG gating, segmentation, and tailored reconstruction algorithms. Respiratory motion also must be eliminated for cardiac imaging, so scanning is optimally performed in a single breath hold, easily achievable with the 5- to 10-second acquisition times for the examination using 64-slice MDCT scanners.

A stable heart rate of 65 bpm or less is important to obtain diagnostic image quality and to decrease radiation dose and shorten the time for image processing and evaluation. For example, a study of 94 patients reported an inverse correlation between the number of analyzable vessel segments and heart rate [71]. Vessel visibility was highest when the heart rate was less than 65 bpm [71]. If the heart rate is more than 65 bpm or is irregular, β-blocker medication can be administered orally or intravenously before the scan. A single puff (0.4 mg) of sublingual nitroglycerin also is given a few minutes before the scan to dilate the coronary arteries and exaggerate the difference between normal and abnormal segments, as is done before catheter coronary angiography.

A noncontrast enhanced scan using prospective ECG gating is performed through the heart, from which the calcium score is generated and the location of the coronary arteries confirmed. The coronary calcium score is a sensitive marker of CAD; the higher the score, the greater the likelihood of significant coronary event. A large coronary calcium load can potentially degrade the image quality of CTA, however. A timing bolus of 15 to 20 mL of intravenous contrast agent is used to determine the optimum time of arterial peak enhancement for the specific patient, by placing a region of interest in the aortic root. Following this, the contrast-enhanced CTA of the coronary arteries is performed with 70 to 80 mL of low osmolar iodinated contrast material injected intravenously at a rate of 5 mL/s through an
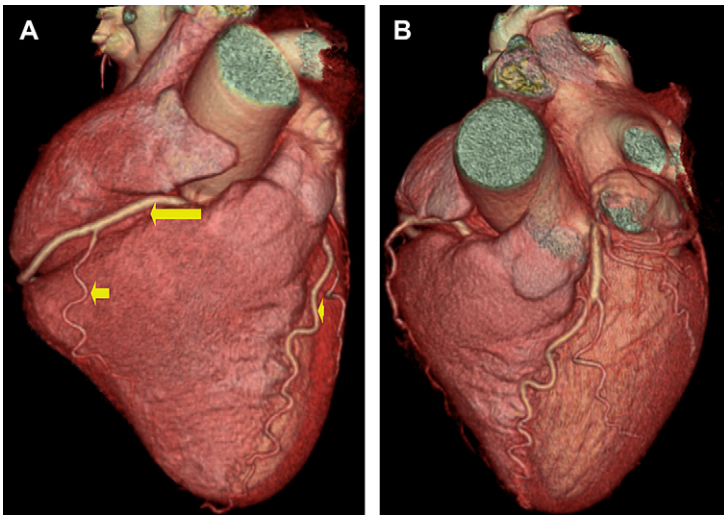

Fig. 8. Volume-rendered images demonstrate excellent visualization of normal coronary anatomy, including the right coronary artery (*arrow*), acute marginal artery (*short arrow*), and left anterior descending coronary artery (*arrowhead*) (*A*) as well as the left main, left anterior descending, and left circumflex coronary arteries and their branches (*B*).
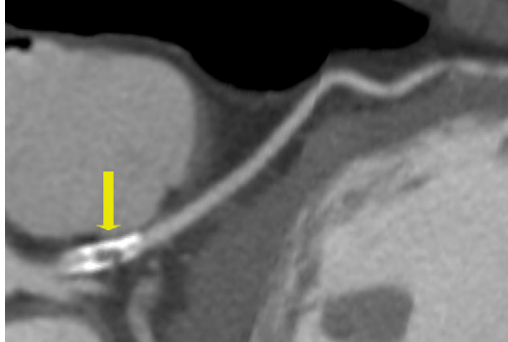
Fig. 9. CT coronary angiography with a high-grade narrowing of the proximal left anterior descending coronary artery due to plaque that has calcified and noncalcified components (*arrow*), as demonstrated on a multiplanar reformatted image.

antecubital vein. During the later part of the contrast injection, the contrast is mixed with saline using a dual-headed power injector, so that the contrast in the right cardiac chambers is not so bright as to cause artifacts in the deep atrioventricular groove where the right coronary artery resides. With 64-slice scanners, the collimation used ranges from 0.5 to 0.625 mm; gantry rotation time is less than 500 milliseconds. The typical scan time is 5 to 10 seconds, short enough to complete the study in a single breath hold.

Following the scan acquisition, processing is performed using retrospective reconstruction of the images at end diastole (70%, 75%, and 80% of the R-R interval), a time of little cardiac motion and the greatest coronary blood flow. The images are reviewed on advanced processing workstations using specialized software for evaluation of the coronary arteries. In a study that compared axial, virtual angioscopic, volume-rendered, and multiplanar reformatted images, the most stenoses were detected on axial images followed by virtual angioscopic, volume-rendered (Fig. 8), and multiplanar reformatted images (Figs. 9 and 10) [72]. Use of all four techniques gave the highest sensitivity.
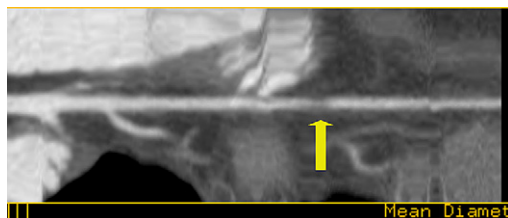

Fig. 10. CT coronary angiography with high-grade stenosis in the mid to distal left anterior descending coronary artery due to circumferential noncalcified plaque (*arrow*).

Summary

ED MDCT is a rapid and accurate test for evaluation of patients who have chest pain in the ED setting. Our understanding about the usefulness and limitations of ED MDCT will improve as more data are made available from ongoing studies.

References

[1] Coche EE, Muller NL, Kim KI, et al. Acute pulmonary embolism: ancillary findings at spiral CT. Radiology 1998;207:753–8.

[2] Greenberg SB. Assessment of cardiac function: magnetic resonance and computed tomography. J Thorac Imaging 2000;15:243–51.

[3] White RH. The epidemiology of venous thromboembolism. Circulation 2003;107:I4–8.

[4] Geerts W, Selby R. Prevention of venous thromboembolism in the ICU. Chest 2003;124:357S–63S.

[5] Heit JA. The epidemiology of venous thromboembolism in the community: implications for prevention and management. J Thromb Thrombolysis 2006;21:23–9.

[6] Silverstein MD, Heit JA, Mohr DN, et al. Trends in the incidence of deep vein thrombosis and pulmonary embolism: a 25-year population-based study. Arch Intern Med 1998;158:585–93.

[7] Piazza G, Goldhaber SZ. Acute pulmonary embolism: Part II: treatment and prophylaxis. Circulation 2006;114:e42–7.

[8] Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet 1999;353:1386–9.

[9] Flordal PA, Berggvist D, Burmark US, et al. Risk factors for major thromboembolism and bleeding tendency after elective general surgical operations. The Fragmin Multicentre Study group. Eur J Surg 1996;162:783–9.

[10] Hirsch DR, Ingenito EP, Goldhaber SZ. Prevalence of deep venous thrombosis among patients in medical intensive care. JAMA 1995;274:335–7.

[11] Kelly AM, Patel S, Carlos RC, et al. Multidetector row CT pulmonary angiography and indirect venography for the diagnosis of venous thromboembolic disease in intensive care unit patients. Acad Radiol 2006;13:486–95.

[12] Marik PE, Andrews L, Maini B. The incidence of deep venous thrombosis in ICU patients. Chest 1997;111:661–4.

[13] Palareti G, Legnani C, Cosmi B, et al. Risk of venous thromboembolism recurrence: high negative predictive value of D-dimer performed after oral anticoagulation is stopped. Thromb Haemost 2002;87:7–12.

[14] Sadosty AT, Goyal DG, Boie ET, et al. Emergency department D-dimer testing. J Emerg Med 2001;21:423–9.

[15] Goldhaber SZ. The perils of D-dimer in the medical intensive care unit. Crit Care Med 2000;28:583–4.

[16] Worsley DF, Alavi A, Aronchick JM, et al. Chest radiographic findings in patients with acute pulmonary embolism: observations from the PIOPED Study. Radiology 1993;189:133–6.

[17] Value of the ventilation/perfusion scan in acute pulmonary embolism. Results of the prospective investigation of pulmonary embolism diagnosis (PIOPED). The PIOPED investigators. JAMA 1990;263:2753–9.

[18] Jolliet P, Slosman DO, Ricou B, et al. Pulmonary scintigraphy at the bedside in intensive care patients with suspected pulmonary embolism. Intensive Care Med 1995;21:723–8.

[19] Stein PD, Henry JW, Gottschalk A. Reassessment of pulmonary angiography for the diagnosis of pulmonary embolism: relation of interpreter agreement to the order of the involved pulmonary arterial branch. Radiology 1999;210:689–91.

[20] Stein PD, Fowler SE, Goodman LR, et al. Multidetector computed tomography for acute pulmonary embolism. N Engl J Med 2006;354:2317–27.

[21] Qanadli SD, Hajjam ME, Mesurolle B, et al. Pulmonary embolism detection: prospective evaluation of dual-section helical CT versus selective pulmonary arteriography in 157 patients. Radiology 2000;217:447–55.

[22] Leschka S, Alkadhi H, Plass A, et al. Accuracy of MSCT coronary angiography with 64-slice technology: first experience. Eur Heart J 2005;26:1482–7.

[23] Raff GL, Gallagher MJ, O'Neill WW, et al. Diagnostic accuracy of noninvasive coronary angiography using 64-slice spiral computed tomography. J Am Coll Cardiol 2005;46:552–7.

[24] Baile EM, King GG, Muller NL, et al. Spiral computed tomography is comparable to angiography for the diagnosis of pulmonary embolism. Am J Respir Crit Care Med 2000;161:1010–5.

[25] Patel S, Kazerooni EA. Helical CT for the evaluation of acute pulmonary embolism. AJR Am J Roentgenol 2005;185:135–49.

[26] Henry JW, Relyea B, Stein PD. Continuing risk of thromboemboli among patients with normal pulmonary angiograms. Chest 1995;107:1375–8.

[27] Moser KM, Harsanyi P, Rius-Garriga G, et al. Assessment of pulmonary photoscanning and angiography in experimental pulmonary embolism. Circulation 1969;39:663–74.

[28] Velmahos GC, Vassiliu P, Wilcox A, et al. Spiral computed tomography for the diagnosis of pulmonary embolism in critically ill surgical patients: a comparison with pulmonary angiography. Arch Surg 2001;136:505–11.

[29] Velmahos GC, Toutouzas KG, Vassiliu P, et al. Can we rely on computed tomographic scanning to diagnose pulmonary embolism in critically ill surgical patients? J Trauma 2004;56:518–25 [discussion: 525–6].

[30] Remy-Jardin M, Tillie-Leblond I, Szapiro D, et al. CT angiography of pulmonary embolism in patients with underlying respiratory disease: impact of multislice CT on image quality and negative predictive value. Eur Radiol 2002;12:1971–8.

[31] Bourriot K, Couffinhal T, Bernard V, et al. Clinical outcome after a negative spiral CT pulmonary angiographic finding in an inpatient population from cardiology and pneumology wards. Chest 2003;123:359–65.

[32] Loud PA, Katz DS, Bruce DA, et al. Deep venous thrombosis with suspected pulmonary embolism: detection with combined CT venography and pulmonary angiography. Radiology 2001;219:498–502.

[33] Loud PA, Katz DS, Klippenstein DL, et al. Combined CT venography and pulmonary angiography in suspected thromboembolic disease: diagnostic accuracy for deep venous evaluation. AJR Am J Roentgenol 2000;174:61–5.

[34] Begemann PG, Bonacker M, Kemper J, et al. Evaluation of the deep venous system in patients with suspected pulmonary embolism with multi-detector CT: a prospective study in comparison to Doppler sonography. J Comput Assist Tomogr 2003;27:399–409.

[35] Garg K, Kemp JL, Wojcik D, et al. Thromboembolic disease: comparison of combined CT pulmonary angiography and venography with bilateral leg sonography in 70 patients. AJR Am J Roentgenol 2000;175:997–1001.

[36] Cham MD, Yankelevitz DF, Shaham D, et al. Deep venous thrombosis: detection by using indirect CT venography. The Pulmonary Angiography-Indirect CT Venography Cooperative Group. Radiology 2000;216:744–51.

[37] Ghaye B, Szapiro D, Willems V, et al. Combined CT venography of the lower limbs and spiral CT angiography of pulmonary arteries in acute pulmonary embolism: preliminary results of a prospective study. JBR-BTR 2000;83:271–8.

[38] Duwe KM, Shiau M, Budorick NE, et al. Evaluation of the lower extremity veins in patients with suspected pulmonary embolism: a retrospective comparison of helical CT venography

and sonography. 2000 ARRS Executive Council Award I. American Roentgen Ray Society. AJR Am J Roentgenol 2000;175:1525–31.

[39] Coche EE, Hamoir XL, Hammer FD, et al. Using dual-detector helical CT angiography to detect deep venous thrombosis in patients with suspicion of pulmonary embolism: diagnostic value and additional findings. AJR Am J Roentgenol 2001;176:1035–9.

[40] Nienaber CA, Eagle KA. Aortic dissection: new frontiers in diagnosis and management: Part I: from etiology to diagnostic strategies. Circulation 2003;108:628–35.

[41] Reed D, Reed C, Stemmermann G, et al. Are aortic aneurysms caused by atherosclerosis? Circulation 1992;85:205–11.

[42] Larson EW, Edwards WD. Risk factors for aortic dissection: a necropsy study of 161 cases. Am J Cardiol 1984;53:849–55.

[43] Januzzi JL, Sabatine MS, Eagle KA, et al. Iatrogenic aortic dissection. Am J Cardiol 2002; 89:623–6.

[44] Nienaber CA, Sievers HH. Intramural hematoma in acute aortic syndrome: more than one variant of dissection? Circulation 2002;106:284–5.

[45] Chen K, Varon J, Wenker OC, et al. Acute thoracic aortic dissection: the basics. J Emerg Med 1997;15:859–67.

[46] Ganaha F, Miller DC, Sugimoto K, et al. Prognosis of aortic intramural hematoma with and without penetrating atherosclerotic ulcer: a clinical and radiological analysis. Circulation 2002;106:342–8.

[47] Jagannath AS, Sos TA, Lockhart SH, et al. Aortic dissection: a statistical analysis of the usefulness of plain chest radiographic findings. AJR Am J Roentgenol 1986;147:1123–6.

[48] Yoshida S, Akiba H, Tamakawa M, et al. Thoracic involvement of type A aortic dissection and intramural hematoma: diagnostic accuracy–comparison of emergency helical CT and surgical findings. Radiology 2003;228:430–5.

[49] Hayter RG, Rhea JT, Small A, et al. Suspected aortic dissection and other aortic disorders: multi-detector row CT in 373 cases in the emergency setting. Radiology 2006;238:841–52.

[50] Sommer T, Fehske W, Holzknecht N, et al. Aortic dissection: a comparative study of diagnosis with spiral CT, multiplanar transesophageal echocardiography, and MR imaging. Radiology 1996;199:347–52.

[51] Sebastia C, Pallisa E, Quiroga S, et al. Aortic dissection: diagnosis and follow-up with helical CT. Radiographics 1999;19:45–60, quiz 149–50.

[52] Tisnado J, Cho SR, Beachley MC, et al. Ulcerlike projections: a precursor angiographic sign to thoracic aortic dissection. AJR Am J Roentgenol 1980;135:719–22.

[53] Nienaber CA, von Kodolitsch Y, Petersen B, et al. Intramural hemorrhage of the thoracic aorta. Diagnostic and therapeutic implications. Circulation 1995;92:1465–72.

[54] Nienaber CA, von Kodolitsch Y, Nicolas V, et al. The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. N Engl J Med 1993;328:1–9.

[55] Nienaber CA, Spielmann RP, von Kodolitsch Y, et al. Diagnosis of thoracic aortic dissection. Magnetic resonance imaging versus transesophageal echocardiography. Circulation 1992;85:434–47.

[56] Ell PJ, Donaldson RM. Cardiovascular nuclear medicine and intensive care. Intensive Care Med 1978;4:119–22.

[57] Hoffmann U, Nagurney JT, Moselewski F, et al. Coronary multidetector computed tomography in the assessment of patients with acute chest pain. Circulation 2006;114:2251–60.

[58] Hilton TC, Thompson RC, Williams HJ, et al. Technetium-99m sestamibi myocardial perfusion imaging in the emergency room evaluation of chest pain. J Am Coll Cardiol 1994;23: 1016–22.

[59] White C, Read K, Kuo D. Assessment of chest pain in the emergency room: what is the role of multidetector CT? Eur J Radiol 2006;57:368–72.

[60] Nieman K, Cademartiri F, Lemos PA, et al. Reliable noninvasive coronary angiography with fast submillimeter multislice spiral computed tomography. Circulation 2002;106: 2051–4.

[61] Ropers D, Baum U, Pohle K, et al. Detection of coronary artery stenoses with thin-slice multi-detector row spiral computed tomography and multiplanar reconstruction. Circulation 2003;107:664–6.

[62] Hoffmann U, Moselewski F, Cury RC, et al. Predictive value of 16-slice multidetector spiral computed tomography to detect significant obstructive coronary artery disease in patients at high risk for coronary artery disease: patient- versus segment-based analysis. Circulation 2004;110:2638–43.

[63] Achenbach S, Ropers D, Hoffmann U, et al. Assessment of coronary remodeling in stenotic and nonstenotic coronary atherosclerotic lesions by multidetector spiral computed tomography. J Am Coll Cardiol 2004;43:842–7.

[64] Kuettner A, Beck T, Drosch T, et al. Diagnostic accuracy of noninvasive coronary imaging using 16-detector slice spiral computed tomography with 188 ms temporal resolution. J Am Coll Cardiol 2005;45:123–7.

[65] Mollet NR, Cademartiri F, Krestin GP, et al. Improved diagnostic accuracy with 16-row multi-slice computed tomography coronary angiography. J Am Coll Cardiol 2005;45:128–32.

[66] Mollet NR, Cademartiri F, van Mieghem CA, et al. High-resolution spiral computed tomography coronary angiography in patients referred for diagnostic conventional coronary angiography. Circulation 2005;112:2318–23.

[67] Ehara M, Surmely JF, Kawai M, et al. Diagnostic accuracy of 64-slice computed tomography for detecting angiographically significant coronary artery stenosis in an unselected consecutive patient population: comparison with conventional invasive angiography. Circ J 2006;70:564–71.

[68] Raff GL, Gallagher MJ, O'Neill WW, et al. Immediate coronary CTA rapidly and definitively excludes CAD in low-risk acute chest pain. Presented at the American College of Cardiology Annual Scientific Session. Atlanta (GA); 2006. p. 807–8.

[69] Achenbach S, Ropers D, Kuettner A, et al. Contrast-enhanced coronary artery visualization by dual-source computed tomography–initial experience. Eur J Radiol 2006;57:331–5.

[70] Rubinshtein R, Halon DA, Gaspar T, et al. Usefulness of 64-slice cardiac computed tomographic angiography for diagnosing acute coronary syndromes and predicting clinical outcome in emergency department patients with chest pain of uncertain origin. Circulation 2007;115:1762–8.

[71] Schroeder S, Kopp AF, Kuettner A, et al. Influence of heart rate on vessel visibility in noninvasive coronary angiography using new multislice computed tomography: experience in 94 patients. Clin Imaging 2002;26:106–11.

[72] Vogl TJ, Abolmaali ND, Diebold T, et al. Techniques for the detection of coronary atherosclerosis: multi-detector row CT coronary angiography. Radiology 2002;223:212–20.