A better echocardiographic view to aortic dissection

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This editorial refers to ‘Impact of contrast-enhanced echocardiography on the diagnostic algorithm of acute aortic dissection’, by A. Evangelista et al. on page 472

Acute aortic dissection is one of the most dramatic vascular events.1 Early mortality is ~1.4%/h within the first 48 h in non-treated patients. The incidence of aortic dissection has been estimated as 10–20 patients/year per 1 million inhabitants.2 Acute aortic dissection is characterized by the rapid development of an intimal flap separating the true and false lumen. The diagnosis of classical aortic dissection is confirmed when these two lumina separated by an intimal flap can be visualized within the aorta.1 The localization of aortic dissections can be described using the well-known classifications of Stanford and DeBakey,3,4 describing the extent of the dissection and the usual site of the intimal tear. The increasing use of modern high-resolution imaging techniques has improved the understanding of the pathogenesis of acute aortic syndromes. This was taken into account by the recommendations of the European Society of Cardiology according to the classification of Svensson.1

Classical aortic dissection with an intimal flap between the true and false lumen (class 1) was distinguished from a medial disruption with formation of an intramural haematoma/haemorrhage (class 2), a discrete/subtle dissection of an aortic aneurysm without haematoma, but a localized eccentric bulge at the tear site (class 3), plaque rupture leading to aortic ulceration (class 4), and iatrogenic or traumatic dissection (class 5). This classification permits a thorough description of the morphology of aortic dissection in addition to the site and extent described by the classifications of Stanford and DeBakey.

Echocardiography is known to be a useful method for detecting aortic dissection.5 However, chest configuration, obesity, pulmonary emphysema, and mechanical respiration limit the diagnostic value of transthoracic echocardiography (TTE). Because of the close anatomical connection of the oesophagus with the thoracic aorta, transoesophageal echocardiography (TEE) has been introduced for imaging in patients with suspected aortic dissection.6 TEE has eliminated the limitations of TTE in most cases. Cross-sectional TEE allows imaging of the descending aorta in multiple scanning planes.6,7 Combination with colour Doppler has been found to be useful to differentiate between the true and false lumen in the aorta due to the characteristic flow pattern.8 Moreover M-mode echocardiography can detect widening of the aortic lumen and the presence of an intimal flap. The detection of spontaneous echocardiographic contrast, indicating slow flow, has been used to diagnose the false lumen.8

Any therapeutic intervention aims to occlude the entry tear, so that the localization of the tear is of major importance.1 A tear is defined as a disruption of the flap continuity with fluttering of the ruptured intimal borders.9 Colour Doppler may facilitate the localization of the tear, and pulsed wave Doppler measurements enable the estimation of the pressure difference between the false and true lumen.8

Computed tomography (CT) and echocardiography are regarded as the most successful approaches for immediate and accurate diagnosis of aortic dissection in patients with abrupt onset of chest pain. In 1979 Harris et al. produced the first report of the diagnosis of aortic dissection by CT.10 The detection of an intimal flap is probably the most specific sign, but the strong movement of the intimal flap during the cardiac cycle may explain the initial better results with TEE compared with CT. Since the introduction of helical CT and multislice CT the limitations have now been overcome.

Finally the greatest accuracy in analysing the full extent of the aortic dissection can be achieved with intravascular ultrasound or intraluminal phased-array imaging definitively eliminating the ‘blind spot’ in the ascending aorta or in the abdomen, as well as by visualization from the inferior and superior vena cava.9,11

In summary the most important diagnostic goals for all imaging techniques including MRI in acute or chronic aortic dissection are:1

(i) confirmation of the diagnosis by visualization of the intimal flap;
(ii) the differentiation of the true and false lumen (visualization of spontaneous echocardiographic contrast, thrombus formation, slow or reduced reversed flow, systolic diameter reduction, entry jet into the false lumen);
(iii) detection and localization of the entry tear, demonstrating communication by 2-D or colour Doppler echocardiography;
(iv) determination of the extent of the dissection with differentiation between a communicating or non-communicating dissection;
(v) detection of wall motion abnormalities as a sign of pre-existing coronary artery disease or myocardial ischaemia due to ostium occlusion by an intimal flap, coronary artery rupture, or collapse of the true lumen during diastole;
(vi) detection and grading of aortic insufficiency;
(vii) assessment of side branch involvement/malperfusion;
(viii) detection of pericardial or pleural effusion and mediastinal haematoma as signs of an emergency situation, indicating a suspending rupture.

Transthoracic and suprasternal echocardiography is a fast and easily available procedure in detecting an intimal flap, but often fails in the descending aorta, especially in patients with emphysema and adipositas. Therefore, TTE should be followed by another definitive diagnostic procedure before surgical treatment. With the combination of TTE and TEE it is possible to gain a high sensitivity and specificity in the diagnosis of aortic dissection. However, in daily practice TEE is rarely used as the first imaging test in community hospitals. The explanation may be its lower availability compared with CT, lack of experienced physicians, and the fear of a rise in arterial blood pressure in critically ill patients. Therefore, the International Registry of Acute Aortic Dissection (IRAD) demonstrated that CT was the initial imaging modality in 61.1% of patients.

Limitations of a combined ultrasound technique, consisting of TTE and TEE, are related to the visualization of the ascending part of the aortic arch, which, because of the interposition of the trachea, cannot be visualized completely. Evangelista et al have demonstrated the feasibility of a simple solution to this problem by using contrast-enhanced echocardiography. To date only a few case reports have mentioned contrast echocardiography in the diagnosis of aortic dissection. Evangelista et al. present the first study validating the usefulness of this method in 128 consecutive patients with clinically suspected acute aortic dissection by comparing conventional and contrast-enhanced TTE and TEE. They validated the results against intraoperative findings in 45 patients and CT in 83 patients. The sensitivity and specificity of conventional TTE clearly increased after using contrast enhancement, almost reaching the values obtained with TEE. Contrast TTE was especially useful in identification of aortic dissection located in the upper part of the ascending aorta, known as the ‘blind spot’ for TEE, as already mentioned. Evangelista et al. have proposed a fast, non-invasive technique to reduce this limitation of TEE in the emergency setting. The limitation of this study is that patients with a poor acoustic window were excluded from the study; these represented ~5% of the primary study population, and this may be even higher in the general practice.

Furthermore they report three cases where conventional TEE yielded a false-positive diagnosis because of intraluminal aortic wall reverberations. After adding contrast, an intimal flap could easily be excluded. Ruling out this common pitfall, which is a problem particularly for non-experienced observers, may be one of the main advantages of contrast-enhanced TEE. Moreover the use of contrast enhancement resulted in a better interobserver agreement.

The use of harmonic imaging in combination with contrast enhancement has significantly increased the accuracy of TTE. Evangelista et al. propose contrast TTE as the initial imaging modality in the emergency setting when aortic dissection is suspected. However, even with contrast TTE, none of false lumen thrombosis or intramural haematoma were diagnosed. This means that TEE (or CT) remains necessary for diagnosis of descending aorta dissection, plaque ulceration, false lumen thrombosis, and intramural haematomas, and for therapeutic decision making. Last but not least TEE can be used immediately before surgery in the operating theatre, giving important information about the localization of the entry tear, which is still the best technique for identification and quantification of aortic regurgitation and its aetiology. Several reports have highlighted the usefulness of intraoperative TEE for guiding endovascular procedures, in particular monitoring hybrid techniques, i.e. a combination of stent graft placement and open visceral bypass grafting or stent graft placement. Evangelista et al. underline that contrast enhancement even in intraoperative TEE may be additionally useful for visualization of antegrade/retrograde false lumen flow and better/faster identification of entry tears.

The common goal of all studies concerning aortic dissection is that early diagnosis and prompt treatment should improve the prognosis in patients with aortic dissection. The decision on which diagnostic technique to use depends in particular on availability in emergency situations and the experience of the emergency room and imaging staff. Contrast TTE may be useful to provide a first glimpse, initiating further steps as the first diagnostic technique in awake patients with acute chest pain when aortic dissection is suspected. If it is positive it may accelerate the surgical treatment but if it fails TEE or CT remains essential to rule out acute aortic dissection definitively.

The main challenge in managing acute aortic dissection remains to diagnose the disease as early as possible.

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References
Severe ventricular arrhythmias in a patient with cardiac sarcoidosis: insights from MRI and PET imaging and importance of early corticosteroid therapy

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A 37-year-old male was referred to our institution for evaluation of exertional angina and non-sustained ventricular tachycardia. Physical examination and laboratory results were normal. The echocardiogram (Panel E) revealed left ventricular (LV) hypertrophy (interventricular septum 16 mm, posterior wall 13.8 mm) with normal ejection fraction. Coronary angiography was normal. Further exploration including cardiac magnetic resonance imaging showed perfusion defects in the basal septal and apical segments (Panel A) as well as a delayed enhancement in the same segments (Panel B).

99mTc-tetrofosmin scintigraphy demonstrated basal septal and apical LV hyperperfusion (Panel C) and cardiac 18F-Fluorodeoxyglucose Positron Emission Tomography (18F-FDG-PET) scanning revealed intense glucose metabolism in the same myocardial segments (Panel D). Whole body 18F-FDG scanning showed pathological uptake in the heart, thoracic, and abdominal lymph nodes, as well as in the lungs and the spleen.

Because of recurrent episodes of ventricular tachycardia with haemodynamic instability, an implantable cardioverter defibrillator was placed and the patient was started on oral amiodarone. Boluses of steroids were administered resulting in a prompt control of ventricular arrhythmia and a rapid regression (6 days) of left ventricular hypertrophy (interventricular septum 10.7 mm, posterior wall 11.1 mm) (Panel F) and of whole-body 18FDG uptake (2 months) (Panel H).

Cardiac sarcoidosis might present in young patients as left ventricular hypertrophy associated with severe ventricular arrhythmias. Magnetic resonance imaging and 18F-FDG-PET scan are extremely helpful in the diagnostic workup, showing the granulomatous lesions as contrast enhancement after gadolinium administration and a perfusion-metabolic mismatch, respectively. Early initiation of high-dose steroid treatment might result in a rapid clinical stabilization and regression of left ventricular hypertrophy.