Current perspectives

Aortic dissection: diagnosis, state-of-the-art of imaging and new management acquisitions

Massimo A. Mariani, Alessandro D'Alfonso, Carmela Nardi, Riccardo Codecasa, Riccardo Cocchieri, Jan G. Grandjean

Department of Cardio Thoracic Surgery, University Hospital, Pisa, Italy

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A prompt diagnosis is the cornerstone of effective treatment of aortic dissection and it is the single most important determinant of survival in this patient population. New imaging modalities such as transesophageal echocardiography, magnetic resonance imaging, helical computed tomography and electron-beam computed tomography have been introduced during the last decade. These new imaging techniques allow for a better and earlier diagnosis of aortic diseases even in emergency situations.

Bearing in mind the high overall mortality of aortic dissection, the role of prevention cannot be overstressed. The main risk factor for aortic dissection/rupture is the aortic diameter; therefore we would like to stress the role of aortic replacement as an effective preventive method for aortic dissection/rupture. Determining the right time for elective surgery, when the operative risk is lower than the risk of dilation-related complications, could contribute to a decrease in urgent surgical procedures on the ascending aorta.

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Address:

Dr. Massimo A. Mariani

Dipartimento Cardio Toracico
Ospedale Cisanello
Via Paradisa, 2
56124 Pisa
E-mail:
massimo_mariani@hotmail.com

Introduction

As reported by the European statistics for cardiovascular mortality and morbidity[1], aortic diseases make a significant contribution to the high overall cardiovascular mortality. Aortic dissection has a prevalence of 0.5 to 2.95/100 000/year and it ranges from 0.2 to 0.8 per 100 000 per year in the United States, resulting in roughly 2000 new cases per year[2]. Interestingly, at 4.04/100 000/year, the highest rate has been reported in Italy[3]. The overall mortality of aortic dissection is reported to range between 3.25-3.6/100 000/year[4]. Due to the high mortality of aortic dissection in the acute stage, the survival rate in both type A and B (type I-III) dissection is very low.

Forty years ago, the 24-hour mortality was 21%. After 30 days only 8% of patients remained alive and only 2% after 1 year[5]. Ten years later, the 48-hour mortality was still reported to be 50% (about 1%/hour). Up to 20% of the patients died before reaching the hospital[2].

Even recently, a mortality of 68% within 48 hours and of 1.4%/hour was reported in a survey spanning 27 years. In this population-based longitudinal study with an incidence of 2.95/100 000/year, the crucial observation was that the diagnosis was established in only 15% before autopsy, and that the primary cause of death was aortic rupture (80% of the patients)[6].

Therefore, a prompt diagnosis is the cornerstone of an effective treatment of aortic dissection and it is the single most important determinant of survival in this patient population.

A prompt diagnosis of aortic dissection is by far more successful in saving human lives that any major step forward in surgical technique achieved in the last 20 years.

New imaging modalities, such as transesophageal echocardiography (TEE), magnetic resonance imaging (MRI), helical computed tomography (CT) and electron-beam CT have been introduced during the last decade. These new imaging techniques allow for a better and earlier diagnosis of aortic diseases even in emergency situations, and they have changed patient management during recent years, leading to a more rapid diagnosis and decision-making[7-9].

Despite this rapid progress in diagnostic tools, global consensus regarding the diagnostic strategy for patient management has not yet been reached. At present, the diagnostic process should be primarily based on clinical assessment and the available imaging techniques should be used to establish a rapid diagnosis whilst avoiding diagnostic overkill.

Aortic dissection staging

The Stanford classification of aortic dissection distinguishes between type A and type B[10]: type A involves the ascending aorta while type B does not.

The De Bakey classification further subdivides aortic dissection: a type I dissection involves the entire aorta, a type II dissection involves the ascending aorta, and a type III dissection the descending aorta[10]. The first attempt to further subdivide the De Bakey classification was made by Reul et al.[11], differentiating between thoracic and abdominal type III dissection. Subdivision of aortic dissections into proximal and distal or ascending and descending is also common.

New studies have shown that intramural hemorrhage, intramural hematoma and aortic ulcers may be signs of evolving dissections or dissection subtypes. Consequently, a new differentiation has been proposed[12]:

• class 1: typical aortic dissection with an intimal flap between the true and false lumen. Acute aortic dissection is characterized by the rapid development of an intimal flap separating the true and false lumen. Due to the pressure difference, the true lumen is usually smaller than the false lumen. Intimal flap tears characterize communicating dissections[7]. However, tears are not always found and non-communicating dissections are not uncommon[7,13];

• class 2: medial disruption with formation of an intramural hematoma/hemorrhage. An intramural hematoma is probably the initial lesion in the majority of cases of cystic medial degeneration. This leads to aortic dissection in which the intimal tear seems to be secondary to a preceding intramural dissection[14]. An intramural hematoma/hemorrhage may be the result of ruptured normal-appearing vasa vasorum which are not supported by the surrounding aortic media or the result of rupture of diseased vasa vasorum[15]. Just as a dissection, the hematoma may extend along the aorta. The prevalence of intramural hemorrhage and hematoma in patients with suspected aortic dissection, as observed by various new imaging techniques, seems to be in the range of 10-30%[13,14,16]. The fact that intramural hemorrhage and hematoma can lead to aortic dissection has been demonstrated in follow-up studies[13,14,16] and in our experience (Figs. 1 and 2). Acute aortic dissection as a consequence of intramural hemorrhage and hematoma develops in 28-47% of patients and it is associated with aortic rupture in 21-47%. Regression occurs in about 10% of patients[13,14,16];

• class 3: discrete/subtle dissection without hematoma and with an eccentric bulge at the site of tear. The structural weakness may lead either to clinically unapparent disease or to minor forms of aortic dissection. Subtle dissection has been described as a partial stellate or linear tear of the vessel wall, covered by thrombus. When the partial tear forms a scar, this constellation is called an abortive, discrete dissection;

• class 4: plaque rupture leading to aortic ulceration and a penetrating aortic atherosclerotic ulcer with a surrounding hematoma, usually subadventitial. Ulceration of atherosclerotic aortic plaques may lead to aortic dissection or aortic perforation[17]. The continuous erosion of the atherosclerotic plaque may eventually violate the internal elastic membrane;

• class 5: iatrogenic and traumatic dissection. Blunt chest trauma usually causes dissection of the ascending aorta and/or the region of the ligamentum Botalli at the aortic isthmus. Iatrogenic dissection of the aorta rarely occurs during heart catheterization. It is frequently seen following angioplasty of an aortic coarctation (in adults), but it may also occur after cross-clamping of the aorta and after intra-aortic balloon pumping[18,19]. Most catheter-induced dissections are retrograde. They will usually decrease in size as the false lumen thromboses[19]. Proximal progression of the coronary dissection into the aortic root has also been described[19].

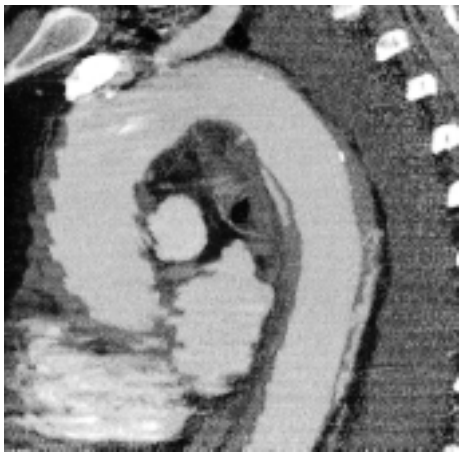

Figure 1. *Computed tomography showing an intramural hemorrhage and hematoma leading to aortic dissection.*

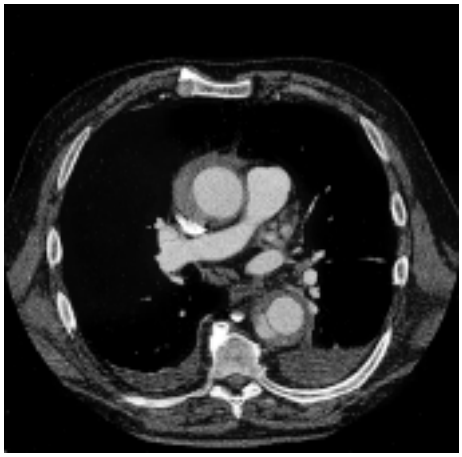

Figure 2. *Computed tomography showing an intramural hemorrhage and hematoma leading to aortic dissection.*

Time-based definition. All classes of dissection may be seen in their acute and chronic stages; chronic dis-

sections are considered to be present if > 14 days have elapsed since the acute event or if they are found at routine clinical evaluation.

Diagnostic path

Essential information: confirming the diagnosis. The basis for diagnosing aortic dissection is the demonstration of an intimal flap separating two lumina. If the false lumen is completely thrombosed, central displacement of the intimal flap, calcification or separation of the intimal layers may all be regarded as definitive signs of aortic dissection[2]. In the large International Registry of Aortic Dissection (IRAD), the first diagnostic step was transthoracic echocardiography (TTE) and TEE in 33%, CT in 61%, MRI in 2%, and angiography in 4%[20]. TTE/TEE, CT, MRI and angiography were used as secondary techniques in 56, 18, 9 and 17% of patients, respectively. Therefore an average of 1.8 methods was employed to diagnose aortic dissection. CT was used in 40%, MRI in 30%, and angiography in 21% of those cases where three diagnostic modalities were necessary[20].

The decision for a specific technique depends on two major factors: its availability in emergency situations and the experience of the emergency room and imaging staff.

Classification of aortic dissection. Communicating and non-communicating aortic dissections may be distinguished on the basis of the presence of flow in the false lumen or of tears in the intimal flap. Communication is present when forward, reversed or delayed flow is detected in the false lumen. In acute communicating dissections, the intimal flap may display strong excursions during the cardiac cycle. Such movement as well as the flow are reduced or absent when no communication is present. Depending on the degree of communication, thrombus formation may or may not be present[7].

Spontaneous contrast is an echocardiographic indicator of a reduced or absent flow in the false lumen[7]. Non-communicating aortic dissections should be differentiated from intramural hematomas. Intramural hematomas (class 2 aortic dissection) are more often localized and restricted to one or two aortic segments; they are frequently characterized by multiple wall layers with shearing of the different components during pulsation. A class 2 dissection may progress to a class 1 dissection.

Antegrade dissection is present when the tear is proximal to the distal end of the dissection. Retrograde progression is said to occur when the tear is located distally to the proximal end of the dissection. The new imaging techniques allow the differentiation of these different types of dissection with a high accuracy. Retrograde dissection with involvement of the ascending aorta and with the presence of a tear at the aortic isthmus is found in up to 20% of type A (type I) patients[7]. This means that a type B (type III) retrograde dissection may extend to the ascending aorta and evolve to a type I or type A dissection.

Plaque ulceration following plaque rupture is typically visualized at TEE, CT or MRI[17,21]. It has to be taken into account that multiple lesions are often present. Each one has to be carefully checked for signs of penetration or rupture[21].

Class 5 dissection due to iatrogenic injury will usually be evident when the catheter follows a false route during aortic contrast injection. Class 5 traumatic aortic dissections are best visualized at immediate TEE performed in the emergency room.

Indicators of emergency. When the pressure exceeds a critical limit and rupture occurs, blood extravasation into the pericardium, pleural space and/or mediastinum are indicators of emergency. Separation of the epi- and pericardium is indicative of pericardial effusion. An effusion as small as 30 ml may be detected at echocardiography, but CT and MRI are sensitive methods as well.

Fluid around the aorta is a sign of ongoing penetration or perforation, not uncommonly combined with intramural hemorrhage[7]. Fluid within the pleural space may be detected by means of echocardiography, CT and MRI. It is usually left-sided.

Mediastinal hematomas increase the distance between the aorta and esophagus and between the aorta and left atrium. A distance > 1 cm is a clear indicator of mediastinal hematoma, which may even compress the left atrium. This may be detected at TEE, CT and MRI. Fluid around the aorta may be a sign of ongoing penetration, eventually resulting in rupture. It is important to identify these signs of emergency as they are associated with a mortality > 50%[7].

Severity and etiology of aortic regurgitation. An elevated pressure amplitude as well as a diastolic murmur are often the presenting clinical signs of aortic dissection. An increased left ventricular contraction with or without dilation may be regarded as an indirect sign of aortic insufficiency.

Color flow Doppler has a high sensitivity and specificity in the detection of aortic regurgitation. In addition, aortic regurgitation may be recognized and graded by means of cine-MRI but not at CT. Aortic regurgitation may be graded at angiography. The etiology of the regurgitation and the diameter of the aortic ring are important to the surgeon, as a decision for aortic value replacement or resuspension has to be made.

TEE is currently the best technique for the assessment of the morphology and function of the valve and surrounding structures[22].

Signs of myocardial ischemia. In the presence of chest pain, it will not be possible to differentiate between myocardial ischemia and aortic dissection. The ECG will show ST-segment elevation and typical signs of acute

transmural myocardial infarction when an ostium of the coronary arteries is occluded by the intimal flap or hematoma. These ECG signs may be so typical that thrombolytic therapy is started[23]. Signs of previous myocardial infarction have to be taken into account in order to consider coronary artery disease.

Wall motion abnormalities detected at echocardiography or MRI are indicative of a reduced myocardial perfusion. Another sign of ischemia may be ST-segment depression indicating a non-transmural infarction or coronary insufficiency secondary to deteriorating hemodynamics in coronary artery disease, to coronary ostium occlusion by flaps or to collapse of the true lumen during diastole.

Tear localization and disease extent. Any therapeutic intervention aims to occlude the entry tear (e.g. by stent implantation or grafting). Hence the importance of accurately detecting and localizing the tear. It is common to see not just one entry and one reentry, but multiple tears. Intimal tears may be directly visualized at TTE, TEE, CT and MRI, but also at angiography. The full extent of aortic dissection may be determined by means of ultrasonic techniques alone, if echocardiography is combined with duplex sonography, abdominal sonography or intravascular ultrasound. Due to its low image quality, duplex abdominal sonography cannot be used for unequivocal decision-making.

Having eliminated the blind spot in the ascending aorta or in the abdomen which used to limit examinations in the past, intravascular ultrasound has become the most accurate among ultrasonic techniques[24].

The full extent of the dissection may be visualized either non-invasively at CT and MRI or invasively at angiography.

Assessing flow in the false and true lumen. It is important to differentiate between true and false lumen. The true lumen is usually compressed by the false lumen, and because of the pressure changes expands during systole and collapses during diastole.

Flow and pulsation in the true lumen may be attenuated when there is no abdominal reentry. The false lumen exhibits systolic compression. It is usually adjacent to the outer segment of the aorta, is characterized by a constantly slow flow and may contain a thrombus[7,25]. If the false lumen is completely occluded it may be helpful to evaluate the aortic structure in different scan fields, and to look for centrally displaced calcifications[16,24,25]. Color flow and pulsed Doppler may be employed if further differentiation is necessary[7].

In addition, communicating dissection intimal flaps show stronger pulsation than non-communicating dissection. The extent of flow visualization in the false lumen depends on the degree of communication. In patients with no or reduced communication between the true and false lumina, imaging shows absent or only reduced flow in the false lumen. If at TEE no flow is present, communication may safely be ruled out.

Imaging modalities

Transthoracic/transesophageal echocardiography. The diagnosis of aortic dissection is confirmed when two lumina, separated by an intimal flap, can be visualized within the aorta. Positive criteria are complete obstruction of a false lumen, centrally displaced intimal calcification, separation of intimal layers from the thrombus, and shearing of the different wall layers during aortic pulsation[7].

Differentiation of the true from the false lumen is based on M-mode, two-dimensional, and Doppler echocardiographic signs[26]. Criteria for identifying the true lumen are systolic expansion and diastolic collapse, the absence or low intensity of spontaneous echocardiographic contrast, systolic jets directed away from the lumen, and systolic forward flow. On the other hand, diagnostic criteria for the false lumen are an increase in diastolic diameter, spontaneous echocardiographic contrast, reversed, delayed, or absent flow and thrombus formation[7,26].

A thrombus is diagnosed when a mass separate from the intimal flap and the aortic wall is imaged in the free space of the false, or rarely the true lumen[7]. Echo-free spaces resulting from the presence of free fluid around the aorta are judged to be signs of penetration due to periaortic hematoma[7].

The European Cooperative Study Group[25] showed that the sensitivity of TTE and of single plane, occasionally biplane, but not multiplane TEE reaches 99%, the specificity 89% and the positive predictive accuracy and negative predictive accuracy 89 and 99% respectively (Figs. 3 and 4).

Computed tomography. The diagnosis is based on the demonstration of an intimal flap which separates the true from the false channel. The flap is identified

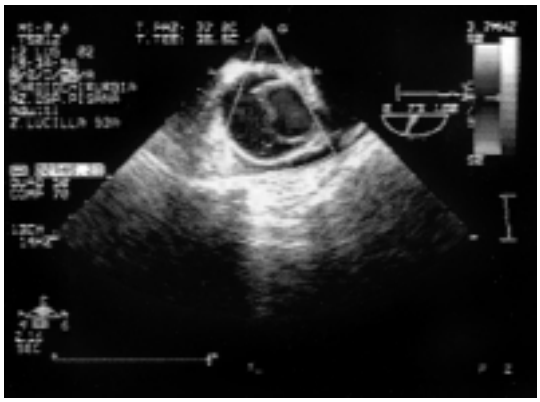

Figure 3. *Transesophageal echocardiography showing separation of the intimal layers in a patient with type A aortic dissection.*

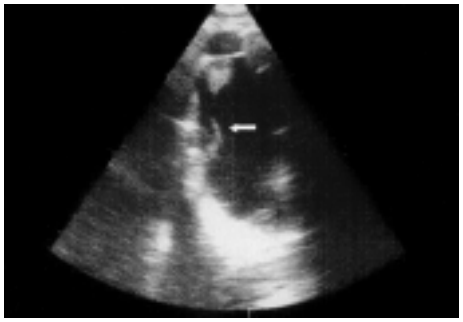

Figure 4. *Transthoracic echocardiography showing separation of the intimal layers in a patient with type B aortic dissection.*

as a low attenuation linear structure in the aortic lumen[27].

In the early 1990s large prospective studies on the evaluation of aortic dissections with conventional incremental CT have reported sensitivities of 83-94% and specificities of 87-100%[25].

The benefits of helical CT compared to incremental CT are well established and include shorter examination times and the potential for a better evaluation of the aorta, as all images are obtained during optimal contrast enhancement. The average sensitivity is > 95%. The sensitivity and specificity for diagnosing arch vessel involvement are 93 and 98% respectively, with an overall accuracy of 96%[28] (Fig. 5).

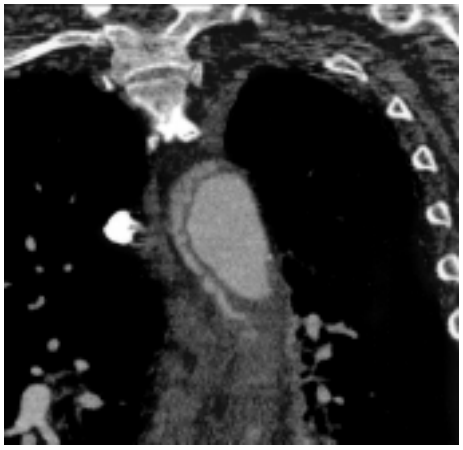

Figure 5. *Computed tomography showing medial disruption with formation of an intramural hematoma/hemorrhage in a patient with type A aortic dissection.*

Magnetic resonance imaging. MRI clearly demonstrates the extent of the disease and depicts the distal ascending aorta and the aortic arch in even more detail than TEE[29]. The localization of the sites of entry and reentry is nearly as accurate as with TEE and the sensitivity approaches 90%. Accordingly, classification of the disease into proximal and distal aortic dissection which is crucial for selecting the appropriate management may be easily accomplished with MRI. Besides, the presence of adverse prognostic signs such as the presence of pericardial effusion or aortic regurgitation may be accurately assessed[29] (Figs. 6 and 7).

Flow in the false and true lumina may be quantified using phase contrast cine-MRI or tagging techniques. With state-of-the-art MRI, the proximal coronary arteries and their involvement in the dissecting process may be clearly delineated[30]. However, no direct comparison between imaging techniques regarding the depiction of coronary involvement has been published yet.

MRI permits detection of acute and subacute aortic intramural hemorrhage[16]. Typical features of intramural hemorrhage include a thickened wall (> 7 mm) with a smooth surface which may contain areas of high signal intensity. The high signal intensity is the result of methemoglobin formation which occurs after several days and persists for several months, although it may be absent in the acute phase[31].

Aortography. The angiographic diagnosis of aortic dissection is based upon "direct" (diagnostic) angio-

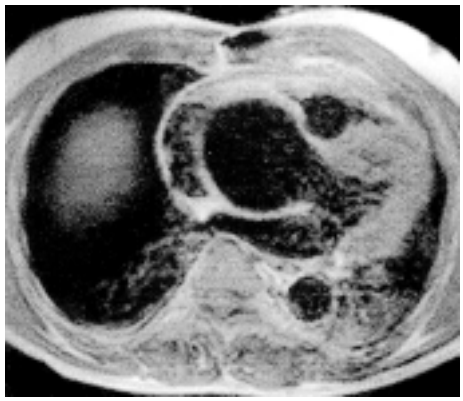

Figure 6. *Magnetic resonance image of a dissection occurring in a dilated ascending aorta.*

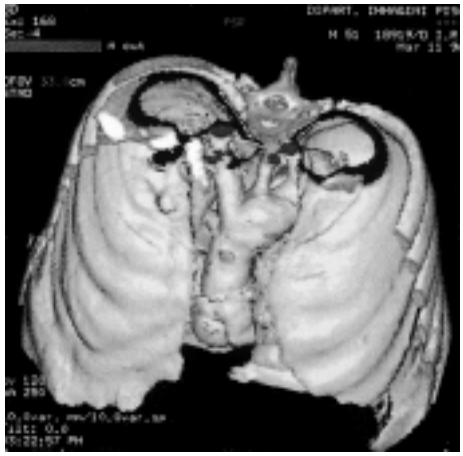

Figure 7. *Magnetic resonance image of a dissection of the ascending aorta (three-dimensional reconstruction).*

graphic signs, such as the visualization of the intimal flap (a negative, often mobile, linear image) or the recognition of two separate lumina, or "indirect" (suggestive) signs, including irregularities of the contour of the aortic lumen, rigidity or compression, branch vessel abnormalities, thickening of the aortic walls and aortic regurgitation[32] (Fig. 8).

Contrast aortography accurately identifies branch vessel involvement. In particular, angiography is an excellent technique for the definition of renal or mesenteric compromise[33]. The concomitance of aortic regurgitation and its severity may be readily identified during aortography.

The specificity of aortography for diagnosing aortic dissection is > 95% but its sensitivity may be lower than that of other techniques, especially in atypical forms of aortic dissection[34]. False-negative aortograms are mainly the result of the inability of the technique to differentiate the two lumina within the aorta[7,25,35]. The missed diagnosis can either be due to a completely thrombosed false lumen or to an intramural hematoma (class 2 dissection).

Some limitations of this technique deserve mention. It is invasive and therefore is laden with an inherent risk. In addition, this technique requires the administration of potentially nephrotoxic radio-opaque contrast media and ionizing radiation.

The catheters should be advanced with care and only by experienced angiographers. Furthermore, the intimal flap as well as the distal end of the dissection are not clearly defined when slow flow is present and the aortic wall thickness cannot be accurately visualized.

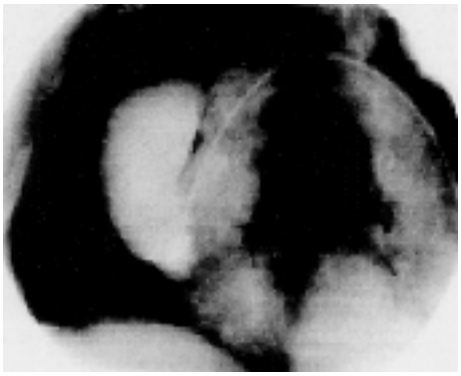

Figure 8. *Aortography showing dissection of the ascending aorta.*

Coronary angiography. In some patients preoperative delineation of the coronary anatomy is desirable. Although new imaging techniques are gaining an emerging role in the assessment of the coronary ostial involvement by the dissecting flap, coronary angiography remains the gold standard for the evaluation of the whole coronary tree[36]. This is important because chronic coronary atherosclerotic disease (present in 25% of patients), although not related to the process of aortic dissection, may worsen the surgical outcome.

Intravascular ultrasound. The use of intravascular ultrasound has been advocated as being complementary to angiographic information in the diagnosis of patients with aortic dissection[24].

In patients with classic forms of aortic dissection, this catheter-based imaging tool provides crisp visualization of the intimal-medial flap, its movement (pulsatility), its circumferential and longitudinal extent and the degree of luminal compromise. This technique appears particularly well suited for the delineation of the most distal extent of abdominal aortic dissections[24]. Sensitivities and specificities approximating 100% have been reported[24].

Changes in the aortic wall due to hemorrhage into the media are visible by intravascular ultrasound because of the accompanying increase in wall thickness. Intravascular ultrasound is very accurate in displaying the circumferential and longitudinal extent of the hematoma.

A comparison of the diagnostic value of the various imaging techniques used for aortic dissection is reported in table I.

New management acquisitions

Bearing in mind the high overall mortality of an aortic dissection, the role of prevention cannot be overstressed. The common risk factors for aortic disease,

Table I. Comparison of the diagnostic value of the various imaging techniques used for aortic dissection.

	TTE/TEE	CT	MRI	Angiography	IVUS
Sensitivity (%)	60-80/90-99	80-100	98-100	80-90	94-100
Specificity (%)	80-96/85-98	90-100	98-100	88-95	97-100
Tear localization	+++	–	++	+	+
Aortic regurgitation	+++	–	++	++	–
Pericardial effusion	+++	++	++	–	–
Mediastinal hematoma	++	+++	+++	–	+
Coronary involvement	++	–	+	+++	++

CT = computed tomography; IVUS = intravascular ultrasound; MRI = magnetic resonance imaging; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography.

such as atherosclerosis and hypertension should be aggressively treated in any documented case of initial aortic disease. In addition, the main risk factor for aortic dissection/rupture remains the aortic diameter and this highlights the role of aortic replacement as an effective preventive method for aortic dissection/rupture.

However, the question regarding the right time to operate on a dilated ascending aorta before it progresses to aortic dissection has not yet been definitely answered. Currently, there is not complete agreement about which criteria should be used to solve this problem. By calculating the right time for elective surgery, when the operative risk is lower than the risk of dilation-related complications, it could be ideally possible to avoid urgent surgical procedures on the ascending aorta. Currently, the size of the aorta and the underlying aortic pathologic process are considered the most important factors in predicting complications. To reach this goal, we developed a new formula[37]:

$$R = e^{C \times [MD-PD]/MD}$$

where R = risk of dissection, C = coefficient to give the R value a unique significance in the presence of different underlying pathologic processes [this coefficient was computed as the ratio MD/(MD - PD) because, as reported by Ergin et al.[38], the critical aortic ratio (MD/PD) varies according to the pathology: for Marfan and chronic dissection (MD/PD = 1.3) the value of C is 4.3, for bicuspid aortic valve (MD/PD = 1.4) C is 3.5, and for other conditions (MD/PD = 1.5), C is 3.0], MD = measured diameter (cm) and PD = predicted diameter (upper 95% of the normal confidence limits of the aortic root diameters at the supra-aortic ridge depending on age and body surface area).

We prospectively studied the patients who were referred to our center for isolated dilation of the ascending aorta during a 6-month period using the above formula. Patients with R values of at least 3 were assigned to elective surgery, patients with R values between 2.7 and 3 were assigned to echocardiographic follow-up after 3 months, patients with R values between 2.0 and 2.7 were assigned to echocardiographic follow-up after 6 months, and patients with R values < 2.0 were assigned to echocardiographic follow-up after 12 months. To date, no patient assigned to echocardiographic follow-up presented with any complications.

In order to facilitate the application of these issues, an internet site has been created in which the formula is available on line; it includes a few fields which must be filled in (age, height, weight, comorbidity) and calculates the PD, the calculated risk (R), the critical aortic size and the indication for treatment (surgery or follow-up). This site is available at www.aortaonline.org.

Conclusion

Since the mortality of acute aortic dissection remains a major epidemiologic issue, prevention forms the cornerstone for a decrease in the incidence and therefore in the mortality of this seriously life-threatening condition.

However, when dissection ultimately occurs, prompt recognition of the clinical signs and symptoms (clinical screening) and the correct use of the available clinical tools are the major determinants of survival for these patients. An early diagnosis may improve patient survival to a greater extent than any major surgical advance proposed to date.

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