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Research Article

Aortic Dissection is a Very Serious Health Condition

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Abstract

Aortic dissection is a very serious health condition, often fatal, in which the inner shell of the aortic wall ruptures while the outer shell remains intact. Blood penetrates the aortic wall through the cleft, further cleaves the middle shell, and creates a new channel in the aortic wall. Damage to the aortic wall in most cases is responsible for the occurrence of aortic dissection. The most common cause of such damage is high blood pressure, and it has been found in more than two-thirds of patients in whom aortic dissection occurs. Other causes are hereditary connective tissue disorders, especially Marfan and EhlerDunlos syndrome. Further causes are congenital heart and blood vessel defects such as aortic coarctation, open ductus arteriosus, aortic valve defects, arteriosclerosis and injuries. In rare cases, dissection occurs accidentally, during catheterization when a doctor inserts a catheter into an artery or during heart and blood vessel surgery. Aortic dissection should be suspected in any patient with chest or shoulder pain, unexplained syncope or abdominal pain, stroke, sudden heart failure, especially if pulse or limb pressures are different. Chest X-rays should be performed in these patients; in 60–90% there is an enlarged shadow of the mediastinum, usually with a localized protrusion indicating the source of dissection.

Keywords: Aortic Dissection, Pathophysiology, Pain, Imaging

Introduction

An aortic dissection occurs when blood enters into the wall of the artery dissecting between the layers and creating a cavity or false lumen in the vessel wall [1]. Dissecting aneurysms are classified into two types according to the Stanford classification scale. Dissecting aneurysms involving the ascending aorta are classified as Type A. Those involving only the descending aorta are Type B.

The peak incidence occurs in the sixth and seventh decades of life. Males are more commonly affected than females. Approximately 60% of dissecting aneurysms are Type A and 40% are Type B. The most common predisposing condition is hypertension. Other predisposing factors include Marfan syndrome, coarctation, bicuspid aortic valve, aortitis, and pregnancy. Aortic dissection may be iatrogenic and result at the site of an aortic cannulation, bypass grafting, cross-clamping, or during a catheterization procedure.

Aortic dissection has been classified into type A and type B [2]

- Type A dissection affects the ascending aorta and arch and occurs in two-thirds of cases.
- Type B dissection has an initial tear distal to the origin of the left subclavian artery and only the descending aorta is affected. It occurs in one-third of cases.

Aortic dissection (AD) is characterized by a separation of the aortic media creating an intimal medial complex, which separates from the remaining aortic wall [3]. Blood flowing between the intimal medial complex or flap and the remaining wall is considered to be within a false lumen, whereas blood flow bounded by the intima is considered to be within a true lumen. Multiple communication points can be observed between the true and false lumen. The proximal most communication is considered to be the "entry tear" with the remaining points of communication are considered to be "exit tears" implying flow directionality from true to false lumen and from false to true lumen, respectively. The actual incidence of AD is difficult to define, since AD involving the ascending aorta is often a fatal disease and patients frequently die prior to hospitalization. Likewise, AD is sometimes misdiagnosed on initial presentation, and these patients are also at risk for death outside of the hospital. Nevertheless, various population-based studies suggest that the incidence of AD ranges from 2 to 4 case per 100,000 patients.

The temporal definition of acute AD is a dissection that is identified less than 2 weeks after the onset of symptoms, while subacute ranges from 2 to 6 weeks following initial painful episode, and a chronic AD more than 6 weeks after the onset of pain.

The true prevalence and incidence of aortic dissection are unknown, but it has been reported to have an annual occurrence of 5–10 cases per million and to affect between 10,000 and 25,000 patients annually in the United States [4]. Autopsy studies in the United States and Denmark report dissections in 0.2–0.8% and 0.2% of cases, respectively. An age-adjusted mortality rate from aortic dissections of 0.5–2.7% per 100,000 inhabitants was calculated from 1950 to 1981. The overall incidence of aortic disse

tions consequently is in the same range or possibly up to two to three times greater than that for ruptured abdominal aortic aneurysm. It is two to five times more common in men than in women, and maximum occurrence is in the 5th decade of life. Still, in our era of modern diagnostic methods, a majority of patients probably die with this disease undetected. Aortic dissection is a dramatic and dangerous condition with a very high mortality: 20–50% of patients die within the first 24–48 h, and up to 75% within the first 2 weeks. It is considered as one of the "great masqueraders," with a wide range of presenting symptoms. Because the diagnosis is difficult, awareness of aortic dissection in the differential diagnosis is essential, as is rapid and correct management.

Condition

Aortic dissection is a potentially catastrophic condition that is due to spread within the wall of the aorta of a subintimal hematoma [5]. The hematoma may begin with a tear in the intima of the aorta or with rupture of the vasa vasorum within the aortic media. This syndrome can occur with trauma to the aorta, including motor vehicle accidents or medical procedures in which catheters or intraaortic balloon pumps damage the intima of the aorta. Nontraumatic aortic dissections are rare in the absence of hypertension and/or conditions associated with deterioration of the elastic or muscular components of the media within the aorta's wall. Cystic medial degeneration is a feature of several inherited connective tissue diseases, including Marfan and Ehlers-Danlos syndromes. About half of all aortic dissections in women under 40 years of age occur during pregnancy.

Almost all patients with acute dissections present with severe chest pain, although some patients with chronic dissections are identified without associated symptoms. Unlike the pain of ischemic heart disease, symptoms of aortic dissection tend to reach peak severity immediately, often causing the patient to collapse from its intensity. The adjectives used to describe the pain reflect the process occurring within the wall of the aorta—"ripping" and "tearing"—and the location usually correlates with the site and extent of the dissection. Thus, dissections that begin in the ascending aorta and extend to the descending aorta tend to cause pain in the front of the chest that extends into the back, between the shoulder blades.

Physical findings may also reflect extension of the aortic dissection that compromises flow into arteries branching off the aorta. Thus, loss of a pulse in one or both arms, cerebrovascular accident, or paraplegia can all be catastrophic consequences of aortic dissection. Hematomas that extend proximally and undermine the coronary arteries or aortic valve apparatus may lead to acute myocardial infarction or acute aortic insufficiency. Rupture of the hematoma into the pericardial space leads to pericardial tamponade.

Pathophysiology

An aortic dissection is a progressive separation of the aortic wall that results from a tear in the intima that progresses into the media, essentially splitting the aorta into an inner layer of intima and inner media and an outer layer of outer media and adventitia [6]. This produces two lumens: a true lumen and a false lumen. The true lumen is where the blood usually flows, while the false lumen is the blind sac that is formed between the intimal flap and the outer media. As blood flows into the false lumen, the tear propagates and the false lumen enlarges. Eventually secondary tears may develop, which allow the blood to reenter the true lumen. The tear is a direct consequence of aortic wall shear stress and most often happens in areas exposed to high mechanical forces (such as the aortic arch and proximal descending aorta just distal to the left subclavian artery).

Dissection of the thoracic aorta is among the most feared entities in all of medicine because of the exceedingly high mortality and the speed with which its injuries become irreversible [7]. An intimal tear of the aorta allows blood flow to exit the lumen and travel a variable distance within the media. Depending on the location of the intimal tear and the direction in which blood travels, aortic dissections can be categorized using two prominent classification schemes. The DeBakey classification describes the location and the extent, whereas the more simplified Stanford classification looks at location alone. A Stanford type A dissection involves the ascending aorta; a Stanford type B involves the arch or descending aorta.

The precise etiology of aortic dissections is unclear, but there is an unambiguous association with a variety of conditions, including aortic aneurysms, hypertension, smoking, pregnancy, and recent intravascular trauma. Once blood enters the medial plane, the intima "floats" within the aortic lumen and has a characteristic appearance on CT scans. The blood within the false lumen reenters the true lumen through any number of naturally created fenestrations or holes, either from disruption of the side branches or from reentry at the termination of the false lumen. Blood flow to any of the side branches off of the aorta can be compromised by the intimal flap, causing ischemia, a condition referred to as malperfusion. Malperfusion can involve the coronary arteries, branches of the aortic arch to the brain, the renal arteries, the mesenteric arteries, or branches to the lower extremities or the spinal cord. Blood can also exit the false lumen pathway into the adventitial space, causing free rupture. Rupture is more likely in Stanford type A dissections because of the intrapericardial location of the ascending aorta and the increased mechanical forces related to the proximity to the left ventricular outflow tract. Type B dissections in the descending aorta do not experience the same mechanical forces, because the more proximal aorta absorbs a significant amount of the kinetic energy. In addition, the extrapleural tissue helps reinforce the weakened adventitia, containing potential ruptures.

Malperfusion

Malperfusion is one of the most important conditions that could significantly increase the risk of mortality and need to be diagnosed as soon as possible when assessing ATAAD (Acute Type A Aortic Dissection) patients [8]. Malperfusion is defined as diminished or lack of blood perfusion to an organ caused by arterial obstruction due to the dissection. Malperfusion syndrome is defined as irreversible organ dysfunction with infarction due to an arterial blood supply obstruction. The incidence of malperfusion in ATAAD patients is reported in the range from 16% to 33%, and malperfusion syndrome significantly increases the mortality rate in ATAAD patients. Malperfusion could occur in any organ in the body leading to a variety of organ ischemia including heart, brain, spinal cord, viscera, or limbs. Theoretically, early peripheral intervention to selectively reperfuse the ischemic organ is critical to avoid malperfusion syndrome. However, as the bottom line, it is also important to perform a proximal aortic repair as soon as possible to save the patient's life. Clinical outcomes of ATAAD are time-dependent from the onset. It is well known that the mortality increases by 1–2% per hour after the onset without surgical intervention (i.e. proximal aortic repair), and up to 90% of patients die within 7 days.

Iatrogenic Aortic Dissection

Iatrogenic dissection of the ascending aorta and arch is a rare complication with an incidence of less than 0.1% for cases in which cardiopulmonary bypass is used [9]. The primary risk factor for aortic dissection from cannulation or cross-clamping of the ascending aorta is calcification. The overall mortality rate associated with this complication is greater than 35%. Management requires prompt recognition and action. If dissection occurs at the site of cannulation, the cannula must be immediately removed and replaced at another site (e.g., the femoral artery). The ascending aorta must then be repaired utilizing deep hypothermic cardiopulmonary bypass or even circulatory arrest. Pledgeted sutures may be used to repair small, local dissections at the cannulation site, but a more complex repair with replacement of the ascending aorta and/or arch with a tube graft may be necessary for extensive injury.

There are several strategies to minimize the risk of an aortic dissection.

TransesophagealTransesophageal echocardiography and epiaortic ultrasound should be used routinely to identify areas with prominent calcification and thus avoided. Lowering the systolic blood pressure to less than 100 mm Hg prior to aortic cannulation and decreasing pump flow prior to cross-clamping of the aorta also reduce risk. If the aorta cannot be cannulated or crossclamped safely due to the presence of extensive calcification, the operative plan must be altered or abandoned. In these situations, coronary artery bypass procedures may be completed using off-pump techniques or hypothermic fibrillatory arrest.

Risk Factors

The risk factors for aortic dissection are hypertension, connective tissue disease (e.g., Marfan's, Ehlers-Danlos), advanced age, atherosclerosis, pregnancy, cocaine use, aortic injury (e.g., trauma, cardiac catheterization), bicuspid aortic valve), and aortic coarctation [6].

Pain

Aortic dissection is far more likely to present as an emergency than is a thoracic aortic aneurysm [10]. Aortic dissection is the cause of acute mortality almost twice as often as acute rupture of an abdominal aortic aneurysm. Although it does not fit the definition of an aneurysm as given above, the term dissecting aortic aneurysm frequently is applied. The underlying aorta may be normal in character or aneurysmal prior to the onset of the dissection. Aortic dissection always must be considered in the emergency setting, as it can be difficult to distinguish from a myocardial infarction. Thrombolytic agents used to treat a myocardial infarction may lead to death when the etiology is aortic dissection.

A dissecting aortic aneurysm frequently presents with the acute onset of severe pain. The pain may be similar to angina with a crushing type of pressure or pain. If the ascending aorta is involved and the dissection continues distally, the pain may migrate to the back. Some patients may describe a tearing pain between the scapulae and refer to it as the worst pain they have ever felt. The pain may be localized to the upper abdomen at times and may be confused with an abdominal aneurysm rupture, perforated ulcer, or cholecystitis. Because of the nature of the problem, associated symptoms can be multiple and often are related to the loss of blood supply to major organs due to the shearing off or occlusion of major side branches. Patients may present with signs of stroke, renal failure, bowel ischemia, or limb ischemia. If the ostium of a main coronary artery is involved, there may be signs of ischemia present on the ECG. This is most common involving the right coronary. New-onset aortic insufficiency is the final sign that should not be overlooked in a patient suspected of aortic dissection. The association of chest pain with loss of one or more peripheral pulses or new-onset aortic insufficiency murmur is the sine qua non of aortic dissection.

Imaging

A CT of the chest, abdomen, and pelvis can rapidly (less than 30 seconds acquisition time) and accurately provide imaging of the entire aorta from the level of the aortic valve to the femoral vessels [11]. The primary finding is that of two lumens or channels with blood, as opposed to one distinct channel, and the visualization of the intimal or medial flap. CT with contrast will also allow for understanding the location and extent of the dissection. A noncontrast CT scan often provides important information about calcification in the walls of the aorta and whether intramural hematoma is present or thrombosis of the false lumen has occurred. Appropriate renal protective strategies should be employed for patients who have suspected or known diminution of their renal function. Hydration and alkalinization of the patient's urine are thought to be the most effective strategies to reduce the nephrotoxicity of most intravenous iodinated contrast agents.

The accuracy of multidetector CT with contrast for the detection of aortic dissection was 100% in several studies. In terms of locating the entry tear, CT has a 82% sensitivity and a 100% specificity. For defining involvement of the cephalic or visceral branch vessels, the sensitivity and specificity are 95% and 100%, respectively. Several other studies report nearly 100% sensitivity and specificity for CT scanning to detect aortic dissections.

Magnetic resonance imaging (MRI) is an alternative modality that is used when traditional contrast agents cannot be administered due to renal insufficiency or allergies. Gadolinium, the contrast agent used for most MRI, has been implicated in a many-fold increase in nephrogenic systemic fibrosis and has to be avoided in patients with chronic renal insufficiency. Furthermore, MRI is associated with longer image acquisition times (up to 30 minutes) and is often not readily available in many outlying medical centers, especially during off hours. MRI has been noted to have between 95% and 100% sensitivity and 94% and 98% specificity for the detection of aortic dissection.

Transesophageal echocardiography (TEE) is an invaluable resource when discrepancy exists regarding the diagnosis or if clarification is needed to understand whether an AAD involves the ascending aorta. Given that type A dissections necessitate immediate surgery, the confirmation of a true flap or dissection in the ascending aorta by TEE is mandatory if there is any doubt or equivocation on the CT images. Although its utility is limited in the descending thoracic aorta, and despite the modality being much more operator-dependent than CT or MRI, TEE can often clearly define the origin of the dissection as either distal or proximal to the subclavian artery and definitively reveal whether the ascending aorta is involved.

Management

Since high blood pressure will propagate the tear, it is critically important to maintain a low blood pressure, usually between 100 and 110 mmHg systolic [6]. This same management principle applies to all types of aortic dissection, regardless of location, and should be initiated as soon as the diagnosis is suspected. This is best done with intravenous beta-blockers. Beta-blockers decrease the shear forces on the aorta by decreasing the dp/dt (change in pressure/change in time).

Once the diagnosis is made, treatment depends largely upon the type of dissection [2].

Type A dissections should be managed surgically because of the risk that the dissection may extend back across the aortic root, resulting in tamponade, and to correct aortic incompetence where present. The surgery aims to interpose a prosthetic tube graft at the aortic root to prevent further dissection (and tamponade), and carries a high mortality.

Type B dissections are usually treated conservatively, and hypotensive drugs are used, reducing systolic pressure to 100–120 mmHg to prevent further extension of the dissection. The dissected portion may then thrombose. Any organ, limb or mesenteric ischaemia resulting from the dissection is treated by revascularization. An aneurysm resulting from a chronic dissection may require treatment if it enlarges or produces pressure symptoms. In cases where there is evidence of impending aortic rupture or non-perfusion of a visceral artery, endovascular placement of a covered stent (a stent-graft) is appropriate. The stent is placed to cover the proximal entry into the false lumen, and to reestablish blood flow through the collapsed true lumen.

Conclusion

Aortic dissection is a serious condition when the inner layer of a blood vessel ruptures and creates a new way for blood to pass, ie, it causes a flow disorder for vital organs. Aortic dissections are not common, but are very fatal because untreated can be fatal in the first 24-48 hours. On average, it affects men over 60 years of age. It can occur anywhere along the aorta and extend proximally or distally to other arteries. Symptoms and signs are the sudden appearance of tearing pains in the chest or back. Diagnosis is made by imaging methods (TEE, CTA, MR, contrast aortography). Treatment always includes aggressive blood pressure control and serial recording to

monitor dissection progression; surgical repair of the aorta, and insertion of a synthetic graft is necessary in ascending aortic dissection and certain descending aortic dissections.

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