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Review Article

Clinical and Diagnostic Clues for the Approach of Patients with Suspected Acute Aortic Dissection

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Abstract

Aortic dissection is a rare condition that frequently manifests suddenly as a catastrophic sickness with intense back or chest pain that is ripping, along with acute hemodynamic compromise. For survival, early and precise diagnosis and treatment are essential. An urgent need for heart surgery is acute ascending thoracic aortic dissection. In stable patients, acute descending thoracic aortic dissection is treated medically, but endovascular interventions may be needed if persistent pain, dissection progression and end-organ ischemia. Acute medical management includes pain control and anti-impulse therapy to reduce the velocity of LV contraction and decrease shear stress by giving antihypertensive therapy (target BP 100 to 120mmHg) typically using IV beta blockers with a target HR of less than 60.

Keywords: Aortic dissection; Ischemia; Turner syndrome; Trauma.

Introduction

Aortic dissection is a rare condition that frequently manifests suddenly as a catastrophic sickness with intense back or chest pain that is ripping, along with acute hemodynamic compromise. For survival, early and precise diagnosis and treatment are essential. Aortic dissection is associated with a high risk of death; however, improvements in surgical and endovascular procedures have led to a general decline in mortality rates for individuals who receive prompt diagnosis and treatment [1]. The ascending aorta (type A) is the site of genesis for two thirds of aortic intimal rips. Intimal tears of type B will begin close to the left subclavian artery and extend into the descending thoracic aorta in one-third of cases [2]. It is possible for an isolated abdominal aortic dissection to result through traumatic, spontaneous, or iatrogenic processes, with the infrarenal abdominal aorta being more frequently involved [3]. Aortic dissection is mostly brought on by aortic intima rips. These tears provide a major danger of cardiac tamponade or ischemia

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(coronary, cerebral, or spinal) if they expand proximally to disrupt the aortic valve and penetrate the pericardial space [4]. Ischemia can be caused in the celiac, mesenteric, renal, and femoral artery areas when it extends distally to affect branch arteries [5].

Acute aortic dissection patients are typically males between the ages of 60 and 80; in contrast to degenerative aortic dissection, family dissections affect individuals much more frequently when they are younger [6].

High-risk conditions defined by the presence of one or more of these factors:

- High Blood Pressure.
- Genetically mediated connective tissue disorders (Marfan, Ehlers-Danlos syndrome) [7].
- Preexisting aortic aneurysm.
- Aortic bicuspid valve.
- Aortic instrumentation or surgery [8].
- Aortic coarctation/Turner syndrome [9].
- Inflammatory diseases/vasculitis (giant cell arteritis, Takayasu, rheumatoid arthritis) [10].
- Trauma.
- Pregnancy and delivery [11].
- Fluoroquinolone use [12].

Discussion

Clinical clues

Acute aortic dissection can happen at any time of day; however, symptoms typically appear from 8 am to 12 pm while people are awake [13]. Over 90% of patients report some level of pain, usually sharp or knife-like, which can be present on its own or in combination with other symptoms including syncope, a cerebrovascular accident, acute coronary syndrome, or heart failure. Abdominal pain is a complication of 20% type A dissections and 40% type B dissections, raising the possibility of mesenteric vascular compromise to a considerable degree [14]. 10% of dissections are painless, primarily in elderly people who frequently experience ascending aortic dissection. They typically present with syncope, heart failure, or stroke symptoms and a history of diabetes, an aortic aneurysm, or cardiovascular surgery [15].

Only 25 to 35 percent of type A dissections and 70 percent of type B dissections have hypertension. On the other hand, 25% of ascending aorta dissections may result in hypotension from an aortic valve disruption that results in substantial aortic regurgitation and/or extravasation into the pericardial space that induces cardiac tamponade [16]. Ascending aorta involvement individuals who can present with neurologic impairments, coma, and hypotension may also experience pulse deficit, which is the presence of reduced or nonexistent blood flow to peripheral vessels. Males and younger patients are more likely to experience pulse deficit than female and older patients [17].

The number of pulse deficits (carotid, brachial, or femoral) is associated with increased mortality (9% with no deficit, 16% with one or 2, and 35% with 3 or more deficits) [18].

Another finding is a variation (>20 mmHg) in SBP when comparing the BP in the arms. A new diastolic murmur in the heart, which occurs in half to two thirds of ascending dissections more frequently in younger patients than in older ones, is an indication of acute aortic regurgitation [19].

Focal neurologic impairments result from either the spread of the dissection or from the

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mass compression of nearby structures caused by the enlarging aorta [20]:

- Stroke/altered consciousness: decreased carotid flow, more prevalent in women.
- Compression of the superior cervical sympathetic ganglion causes Horner syndrome.
- Left recurrent laryngeal nerve compression: hoarseness/vocal cord paralysis.
- Spinal cord ischemia, which is more common in type B aortic dissections, causes acute paraplegia.

Para clinical clues

ECG is normal in 30%, nonspecific ST changes in 40%, ischemic changes in 15%. In type A aortic dissection (when dissection leads to coronary ischemia), it can show acute myocardial infarction signs in 5% of cases [21]. Chest radiographs are the most common finding is widening of the aortic silhouette, appearing in 60 to 90 percent of cases. Pleural effusion is found in 20% of cases mostly females [22].

CT angiography of the chest and abdomen for hemodynamically stable patients, has a sensitivity of 80 to 95% and specificity of 85 to 100%. It is the technique with the least operator dependence, offers helpful anatomic correlates for surgical and endovascular therapy, and gathers data for measurement and follow-up analysis [23].

TEE for hemodynamically unstable patients is the preferable initial study for suspected aortic dissection, with a sensitivity of 98% and a specificity of 60 to 90% [24].

TTE occasionally reveals the intimal flap at either the aortic root or descending aorta in addition to the consequences of TAD (pericardial effusion, aortic regurgitation) [25].

Laboratory clues

Troponin T and I are very sensitive and specific for myocardial necrosis but not for a single TAD, unless the dissection has reached the right coronary artery and produced myocardial infarction. A normal Troponin level cannot therefore be taken as evidence that TAD is absent.

A transmembrane and soluble isoform of ST2, a member of the interleukin-1 receptor family, is available. Solvable ST2 (sST2) may be helpful for distinguishing acute aortic dissection from other disorders that show with acute chest pain because it has a cutoff level of 34ng/mL (Sensitivity and NPV >95%) [26].

Using a threshold of 500 ng/mL, D-dimer (DD) is a good screening technique to identify patients who do not have acute aortic dissection, while its specificity and PPV are low (50%) and its NPV is above 90% [27]. D-dimer is not useful as a diagnostic tool because of:

- Low specificity: raised in many conditions and in pregnancy especially 2nd/3rd trimester [28].
- Low sensitivity: it can be falsely negative, for example due to Intramural hematoma.
- The lack of standardized testing procedures (ELISA testing is relatively sensitive, while latex agglutination is neither sensitive nor specific) [29-36]. Clinicians must therefore be familiar with the features of the assay used at

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their hospitals when utilizing D-dimer.

- Levels can vary depending on how long-ago symptoms started (typically negative within the first three hours), but sensitivity increases to 100% after 180 minutes, highlighting the potential value of serial DD assessment [30].

Diagnostic clues

A triad of findings is commonly present in TAD:

- Deficit or blood pressure differential
- Presence of mediastinal widening on CXR
- Chest or back pain

But the absence of these clinical features (7%) does not exclude aortic dissection [31].

Aortic dissection detection risk score (ADD-RS), presence of one or more (each 1 point) [32]:

- High-risk condition
- Pain in the chest, back, or abdomen
- Physical examination findings of perfusion deficit (pulse deficit, focal neurological deficit, aortic regurgitation murmur, hypotension/shock).

ADD-RS plus D-dimer, ADD-RS (0 to1) and negative d-dimer (<500 mg/dL) effectively ruled out AAS (aortic dissection, aortic rupture, aortic intramural hematoma) with a NPV >98% [33].

For ADD-RS>1, D-dimer was not discriminatory, requiring conclusive imaging.

For ADD-RS (2 or 3), 40 percent of patients have acute aortic syndrome.

Missed diagnoses (40%) are typically attributed to various causes of chest discomfort, most frequently acute coronary syndromes. Diagnostic mistakes cause treatment delays and incorrect therapy (such as the use of antithrombotic drugs) that raise the risk of consequences (including significant bleeding and greater mortality) [34].

Therapeutic clues

- An urgent need for heart surgery is acute ascending thoracic aortic dissection.
- In stable patients, acute descending thoracic aortic dissection is treated medically, but endovascular interventions may be needed if persistent pain, dissection progression and end-organ ischemia.
- Acute medical management includes pain control and anti-impulse therapy to reduce the velocity of LV contraction and decrease shear stress by giving antihypertensive therapy (target BP 100 to 120mmHg) typically using IV beta blockers with a target HR of less than 60 [3].

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