Clinical Prediction of Acute Aortic Dissection

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Background: Clinical criteria for aortic dissection are poorly defined. Thus, 35% of aortic dissections remain unsuspected in vivo, and 99% of suspected cases can be refuted.

Objective: To identify independent predictors of acute aortic dissection and create a prediction model for facilitated estimation of the individual risk of dissection.

Methods: Two hundred fifty patients with acute chest pain, back pain, or both; absence of an established differential diagnosis of the pain syndrome; and clinical suspicion of acute aortic dissection were evaluated for the presence of 26 clinical variables in a prospective, observational study. Multivariate analysis was performed to create a prediction model of aortic dissection.

Results: Aortic pain with immediate onset, a tearing or ripping character, or both; mediastinal widening, aortic widening, or both on chest radiography; and pulse differentials, blood pressure differentials, or both (P<.001 for all) were identified as independent predictors of acute aortic dissection. Probability of dissection was low with absence of all 3 variables (7%), intermediate with isolated findings of aortic pain or mediastinal widening (31% and 39%, respectively), and high with isolated pulse or blood pressure differentials or any combination of the 3 variables (≥83%). Accordingly, 4% of all dissections were assigned to the low-probability group, 19% to the intermediate-probability group, and 77% to the high-probability group of aortic dissection.

Conclusions: Assessment of 3 clinical variables permitted identification of 96% of the acute aortic dissections and stratification into high-, intermediate-, and low-probability groupings of disease. With better selection for prompt diagnostic imaging, this prediction model can be used as an aid to improve patient care in aortic dissection.

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PATIENTS AND METHODS

Three clinical criteria were applied to selected patients admitted to the emergency department for inclusion in the study group: (1) Patients with onset of chest pain, back pain, or both within 2 weeks of admission were subjected to history taking, physical examination, routine laboratory evaluation, and a 12-lead electrocardiogram performed on arrival in the emergency department. (2) Evidence of an acute coronary syndrome or another established diagnosis explaining the chest pain, back pain, or both was an exclusion criterion. In uncertain cases, additional diagnostic procedures were performed using a strategy similar to that described elsewhere before definite exclusion. (3) All other patients were included when jointly considered clinically suspicious of acute aortic dissection by 2 experienced emergency department physicians. Patients with a history of recent trauma, cardiovascular intervention, or gastrointestinal surgery and comatose patients with no history available were excluded.

All study patients were evaluated for 26 clinical variables, which were assessed unblinded to each other. A plain chest radiograph was obtained as frontal and lateral projections in 190 patients and as frontal bedside examination in 60 patients. After clinical evaluation, each study patient was subjected to contrast-enhanced computed tomography, magnetic resonance imaging, transesophageal echocardiography, or digital angiography. Diagnostic findings on these imaging procedures and findings at surgery or autopsy were used to establish the final diagnosis in each patient (Table 1).

Table 1. Characteristics of 250 Study Patients

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Type A (n = 78)</th>
<th>Type B (n = 50)</th>
<th>None (n = 122)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean ± SD, y</td>
<td>50 ± 15</td>
<td>57 ± 12</td>
<td>55 ± 17</td>
</tr>
<tr>
<td>Sex, M/F</td>
<td>61/17</td>
<td>39/11</td>
<td>73/49</td>
</tr>
<tr>
<td>Acute aortic dissection</td>
<td>67 (86)</td>
<td>38 (76)</td>
<td>NA</td>
</tr>
<tr>
<td>Subacute aortic dissection</td>
<td>11 (14)</td>
<td>12 (24)</td>
<td>NA</td>
</tr>
<tr>
<td>Chronic arterial hypertension</td>
<td>58 (74)</td>
<td>41 (82)</td>
<td>52 (43)</td>
</tr>
<tr>
<td>Marfan syndrome</td>
<td>9 (12)</td>
<td>NA</td>
<td>2 (2)</td>
</tr>
<tr>
<td>Dissection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overt dissection</td>
<td>71 (91)</td>
<td>41 (82)</td>
<td>NA</td>
</tr>
<tr>
<td>Intramural hemorrhage</td>
<td>7 (9)</td>
<td>9 (18)</td>
<td>NA</td>
</tr>
<tr>
<td>Subintimal hemorrhage</td>
<td>7 (9)</td>
<td>9 (18)</td>
<td>NA</td>
</tr>
<tr>
<td>Death =30 d</td>
<td>17 (22)</td>
<td>7 (14)</td>
<td>7 (6)§</td>
</tr>
</tbody>
</table>

*Data are given as number (percentage) of patients except where noted otherwise. NA indicates not applicable.
†Includes 31 patients with surgery for aortic aneurysm or aortic dissection, 5 patients with surgery for coronary artery disease, and 4 additional patients with abdominal (noncoronary vasculitis) surgery.
‡Includes 4 patients with intraoperative or postoperative death associated with thoracic aortic repair and 1 patient each with pulmonary embolism, acute coronary syndrome, and pancreatitis.
§Includes 4 patients with intraoperative or postoperative death associated with thoracic aortic repair and 1 patient each with pulmonary embolism, acute coronary syndrome, and pancreatitis.

PATIENTS

Between January 1, 1988, and December 31, 1996, 41 495 consecutive patients presented to the emergency department of the University Hospital Eppendorf, Hamburg, Germany, with acute chest pain, back pain, or both; of these, 250 constituted the study group (77 women and 173 men; mean±SD age, 54±15 years). Dissection was confirmed in 128 study patients, including 16 diagnosed in the stage of intramural hematoma; 105 patients had acute dissection with onset of symptoms within 48 hours and 23 had subacute dissection with onset of pain within 2 weeks of emergency department admission. Seventy-eight patients with dissection in the ascending aorta were identified as type A (61%) and 50 with no affliction of the ascending aorta were identified as type B (39%) dissections (Table 1). Chronic arterial hypertension was prevalent in 151 patients (60%), and Marfan syndrome was present in 11 (4%).

Dissection was refuted in the other 122 study patients. The remainder of 41 245 patients with chest pain, back pain, or both (20 062 women and 21 183 men; mean±SD age, 55±16 years) did not meet the inclusion criteria. The final diagnoses in the 2 cohorts of the excluded 41 245 patients and in the 122 study patients with refuted dissection were acute coronary syndrome (24.4% vs 14.7%; P < .001); neurovascular syndrome (17.1% vs 0.8%; P < .001); pulmonary disease (15.0% vs 0.8%; P < .001); cardiac arrhythmia (6.5% vs 0%; P < .001); hypotension with vagal reaction (6.2% vs 0%; P < .001); heart failure (5.0% vs 0%; P < .001); hyperventilation (3.3% vs 0%; P < .001); hypertensive crisis (2.2% vs 9.0%; P < .03); chest wall syndrome (1.8% vs 14.7%; P < .001); gastrointestinal diseases such as esophagitis, peptic ulcer, gastritis, and pancreatitis (1.2% vs 9.8%; P < .001); pneumothorax (0.6% vs 1.6%; P = .67); pulmonary suspicion of acute aortic dissection in the remaining 250 patients eligible for our study (0.6%) (Table 1).

Study patients presented to the emergency department 49±44 hours after initial onset of symptoms. Clinical evaluation and emergency diagnostic imaging were completed within 1.8±1.5 hours, confirming aortic dissection in 128 patients (51%) and excluding dissection in 122 (49%). At the time of initial presentation, no patient had electrocardiographic signs of acute myocardial infarction; 5 patients, however, developed new ST-segment elevation (4%). Five patients with dissection were falsely diagnosed as having acute coronary syndrome: 1 survived fibrinolytic therapy and the other 4 died and were diagnosed at autopsy.

Univariate analysis identified 13 variables associated with acute dissection (Table 2). The final model included aortic pain with immediate onset of pain, tearing or ripping pain, or both (odds ratio, 6.41; 95% confidence interval, 3.06-13.46); mediastinal widening, aortic widening, or both (odds ratio, 57.86; 95% confidence interval, 7.04-475.47); and pulse differentials, blood pressure differentials, or both (odds ratio, 10.19; 95% confidence interval, 4.92-21.11; P < .001 for all) as independent predictive variables of acute aortic dissection. Each of these 3 variables was confirmed as an independent predictor separately for both subtypes of dissection. In ad-
embolism (0.4% vs 4.9%; \( P < .001 \)); pleuritis (0.2% vs 4.0%; \( P < .001 \)); and pericarditis (0.1% vs 5.7%; \( P < .001 \)). In 5.9% and 3.3%, respectively, a definite diagnosis could not be established (\( P < .002 \)), and in the remainder miscellaneous diagnoses were made.

**CLINICAL VARIABLES**

The 26 most frequently reported clinical features in aortic dissection were chosen as study variables. Among these, onset of pain within less than 2 minutes was considered immediate; a trigger of pain was antecedent to strain of an emotional or physical nature; and prodromal symptoms comprised giddiness, swaying, or apprehension before pain (Table 2). Intense severity of pain was present with the notion of excruciating, unbearable intensity, and tearing or ripping pain was considered with any tearing, ripping, or lacerating feature of pain, also described as “if something inside tore loose.” Shifting of pain location was considered migratory, whereas inspiratory aggravation of pain was considered pleuritic. Episodic interruption of consciousness without focal neurologic defect was considered syncope; prolonged loss of consciousness or coma and focal neurologic signs were defined by standard criteria. Systolic blood pressure greater than 150 mm Hg on hospital admission was considered hypertension and less than 100 mm Hg was considered hypotension; a difference in systolic blood pressure of more than 20 mm Hg between both arms was a blood pressure differential. Absence of a proximal extremity or carotid pulse was considered a pulse differential. Acute renal failure was diagnosed in the presence of anuria, oliguria, and/or increased creatinine levels with a systolic blood pressure greater than 100 mm Hg and no history of chronic renal failure. A murmur of aortic regurgitation was detected by auscultation. Pulsating abdominal mass and abdominal board-like tensesness were considered abdominal signs. Mediastinal and/or aortic widening (including the aortic knob) and pleural effusion on chest radiographs were diagnosed at the discretion of a board-certified radiologist. Electrocardiographic left ventricular hypertrophy, previous Q-wave infarction, and acute myocardial infarction syndrome were assessed by standard criteria.

**STATISTICAL METHODS**

Logistic regression analysis was performed with only 0.6% missing data. Risk of aortic dissection associated with the presence of each sign or symptom was initially evaluated using univariate, unconditional logistic regression models and statistical software. Risk factors significant for predicting risk at the 5% level were evaluated for inclusion in a multivariate model using stepwise procedures. To simplify the final model, a new variable was created in which anyone with pain of immediate onset or of a tearing or ripping nature (both variables were the only pain variables, which were independent predictors of aortic dissection) was labeled “aortic pain.” The final model obtained from the stepwise procedure was then refitted using this simplified pain variable. Estimates of risk (odds ratios) were calculated based on coefficients from the logistic models. Analyses were repeated for patients with type A and type B dissection. Probability of dissection was calculated as the number of patients with dissection and the clinical feature(s) divided by the number of patients presenting with the clinical feature(s). Comparison between groups and characteristics was performed using the Fisher exact test; significance was set at 5%. Data are given as mean ± SD.

**COMMENT**

This prospective, observational study of a selected group of patients with acute chest pain, back pain, or both identifies aortic pain; pulse differentials, blood pressure differentials, or both; and mediastinal widening, aortic widening, or both on chest radiography as independent predictors of acute aortic dissection. These variables permit identification of 96% of all acute dissections and rapid stratification according to high, intermediate, or low probability of disease. With better selection for prompt diagnostic imaging, this prediction model can be used as an aid to improve survival after dissection.

**SELECTION OF PATIENTS**

Selection of candidates for imaging of the aorta depends on the experience of the attending physician and his or her clinical impression, which was also used as a criterion for enrollment in the present study. Our data confirm that in specialized cardiovascular centers, such subjective clinical criteria might yield diagnostic confirmation of dissection in 50% of suspected cases. Identification of dissection in unselected outpatients, however, is much less efficient. Thus, use of defined clinical criteria for aortic dissection seems particularly helpful when dealing with unselected patients in settings without specialized clinical experience or adequate diagnostic or therapeutic facilities available for the management of potential acute aortic disease.

Patients without pain were not included in our study group. However, all documented cases of so-called pain-
less dissection revealed atypical chest pain along with other signs of dissection. Because such patients were included, we believe that our study group was not biased by the absence of completely painless dissections.

**CLINICAL SIGNS OF DISSECTION**

Chest pain is consistently reported in more than 90% of patients with aortic dissection. Its immediate, catastrophic onset of a ripping, tearing, and migrating nature is reported in more than 80% of dissections. Our study confirms the high incidence of pain and identifies other signs of dissection as independent predictors of dissection. However, focal neurologic signs and renal signs were found exclusively as independent predictors of dissection. However, focal neurologic signs and renal signs were found exclusively in patients with dissection and thus might be used as additional diagnostic clues of dissection.

**DIFFERENTIAL DIAGNOSES OF DISSECTION**

In 21 study patients with exclusion of dissection (17%), other aortic disorders, such as penetrating aortic ulcer and unstable nondisseminating aneurysms with rapid expansion, rupture, or severe aortic regurgitation, were diagnosed, more than half of which required immediate surgical intervention. Chest pain, mediastinal widening, and murmurs of aortic regurgitation make such patients difficult to separate from those with acute dissection.

Moreover, in a setting of acute chest pain, back pain, or both, acute coronary syndromes outweigh the frequency of acute aortic dissection by 80:1, with their diagnostic separation failing in 0.01%. Our data confirm a high prevalence of aortic regurgitation in type A dissection, emphasizing the usefulness of diastolic aortic murmurs to distinguish proximal from distal dissection. The scarce occurrence of malperfusion signs disqualifies them as independent predictors of dissection. However, focal neurologic signs and renal signs were found exclusively in patients with dissection and thus might be used as additional diagnostic clues of dissection.
confused with arterial embolism caused by acute myocardial infarction.32 More important, 4% of acute aortic dissections present with electrocardiographic signs of acute myocardial infarction;34 fibrinolysis was administered to 0.4% of patients with aortic dissection and 2% of reported dissections received fibrinolytic therapy, with lethal outcome in 64%.34 In our study, 5 patients with aortic dissection were misdiagnosed as having acute coronary syndromes, with atypical ST-segment elevations due to left ventricular hypertrophy in 2 and typical ST-segment elevations caused by aortic dissection with coronary artery involvement in 3. Thus, clinical evaluation for signs and symptoms of dissection in patients even with electrocardiographic evidence of acute myocardial infarction might avoid confusion of both diseases.

**CLINICAL ALGORITHM FOR DIAGNOSING ACUTE AORTIC DISSECTION**

Assessment of risk is likely to help (1) administer the appropriate initial medical therapy, (2) choose the optimal imaging modality, (3) assess the urgency for definitive diagnosis, and (4) define the need for potential emergency intervention. In patients at high risk for dissection, initiation of intravenous therapy with β-adrenergic blocking agents, precautions for surgical intervention, and diagnostic confirmation have priority, and transportation to a tertiary care center is warranted for adequate imaging.35 Patients with isolated aortic pain or mediastinal widening are at intermediate risk for dissection and should be treated as high-risk patients.

In patients with a low probability of dissection, the priority of diagnostic evaluation might be shifted to other diseases and imaging of the aorta conducted later in the differential workup. Knowledge of rare and variable alternative signs of dissection might help individualize the risk in each patient, and it should be emphasized that our prediction model is designed for more rapid identification of patients with dissection rather than as an instrument for clinical rule out.

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**REFERENCES**


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**Table 3. Risk for Types A and B Acute Aortic Dissection According to 3 Clinical Predictors**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Dissection (n = 128)</th>
<th>No Dissection (n = 122)</th>
<th>Probability of Dissection, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>No sign present</td>
<td>5 (4)</td>
<td>65 (53)</td>
<td>7</td>
</tr>
<tr>
<td>Aortic pain alone</td>
<td>13 (10)</td>
<td>29 (24)</td>
<td>31</td>
</tr>
<tr>
<td>Mediastinal widening, aortic widening, or both alone</td>
<td>11 (9)</td>
<td>17 (14)</td>
<td>39</td>
</tr>
<tr>
<td>Aortic pain + mediastinal widening, aortic widening, or both</td>
<td>50 (39)</td>
<td>10 (8)</td>
<td>83</td>
</tr>
<tr>
<td>Pulse differentials, blood pressure differentials, or both alone</td>
<td>2 (2)</td>
<td>0</td>
<td>100</td>
</tr>
<tr>
<td>Aortic pain + pulse differentials, blood pressure differentials, or both</td>
<td>11 (9)</td>
<td>1 (1)</td>
<td>92</td>
</tr>
<tr>
<td>Mediastinal widening + pulse differentials, blood pressure differentials, or both</td>
<td>2 (2)</td>
<td>0</td>
<td>100</td>
</tr>
<tr>
<td>Aortic pain + mediastinal widening, aortic widening, or both + pulse differentials, blood pressure differentials, or both</td>
<td>34 (27)</td>
<td>0</td>
<td>100</td>
</tr>
</tbody>
</table>

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**Table 4. Clinical Findings in 57 Patients With No Dissection but Presence of Clinical Predictors of Aortic Dissection**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Non dissecting Aortic Disease† (n = 21)</th>
<th>No Aortic Disease (n = 36)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediate onset of pain alone</td>
<td>8 (38)</td>
<td>25 (69)</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Tearing or ripping pain alone</td>
<td>2 (10)</td>
<td>1 (3)</td>
<td>.27</td>
</tr>
<tr>
<td>Immediate onset of pain and tearing or ripping pain</td>
<td>1 (5)</td>
<td>3 (8)</td>
<td>.61</td>
</tr>
<tr>
<td>Mediastinal widening, aortic widening, or both</td>
<td>17 (81)</td>
<td>10 (28)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Murmur of aortic regurgitation</td>
<td>14 (67)</td>
<td>5 (14)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Pulse differentials, blood pressure differentials, or both and aortic pain</td>
<td>1 (5)</td>
<td>0</td>
<td>...</td>
</tr>
</tbody>
</table>

*Data are given as number (percentage) of patients.
†Patients with no aortic dissection or intramural hemorrhage but with other aortic disease such as penetrating aortic ulcer and unstable nondissecting aneurysms with rapid expansion, rupture, or severe aortic regurgitation.*


