Abstract—Acute aortic dissection is a rare but devastating condition with high mortality. Unfortunately, there is no sensitive screening indicator of disease in common use. The objective of this study was to assess the sensitivity and utility of the serum D-dimer as a test for acute aortic dissection. A pooled analysis was performed of all original research studies testing the sensitivity of serum D-dimer for acute aortic dissection. A search of MEDLINE, EMBASE, and the Cochrane Register using the terms “aortic dissection” and “d-dimer” was made of all English language publications. All original reports of consecutively enrolled patients with acute aortic dissection and a measured serum D-dimer were included. Case reports were excluded. A value of 0.5 microgram per milliliter was defined as the threshold for a positive D-dimer. The primary outcome was the pooled sensitivity of the D-dimer test for acute aortic dissection. There were 21 original reports of patients with acute aortic dissection and D-dimer measurements. Eleven studies were included and a total of 349 acute aortic dissection patients were described. The sensitivity of the D-dimer test was 327/349, 94% (95% confidence interval 91–96), and the point estimate was essentially unchanged in a sensitivity analysis, 183/192, 95% (95% confidence interval 91–98). Specificity ranged from 40% to 100%. Serum D-dimer is sensitive for acute aortic dissection and potentially represents a useful test for patients who present with a low likelihood of this disease. © 2008 Elsevier Inc.

Keywords—acute; aorta; dissection; D-dimer; sensitivity

INTRODUCTION

Acute aortic dissection is a rare but important emergency condition due to its high mortality. Survival is possible with emergent surgical and medical treatment, but reduction in mortality is critically dependent on rapid Emergency Department (ED) diagnosis (1). The most sensitive clinical test of acute aortic dissection is pain in the chest, back, or upper abdomen, and aortic dissection should be in the differential diagnosis of all patients who present with such pain (2,3). The pain is usually severe with a sudden onset. It is sometimes described as sharp, ripping, tearing, or migrating. Associated asymmetric blood pressure or pulses and focal neurologic deficits are strongly suggestive of aortic dissection, but these findings are usually absent. Chest X-ray study findings of an abnormal aortic contour or widened mediastinum are also suggestive, but at least 10% of patients with acute aortic dissection have a normal chest X-ray study (2,4).

Ultimately, emergency, cardiac, and critical care physicians must consider possible acute aortic dissection in a large number of patients. To varying degrees, the tests used to make a definitive diagnosis, such as computed tomography (CT) scan, transesophageal echocardiography (TEE), magnetic resonance imaging (MRI), and aortography, are expensive, are uncomfortable, have morbidity, or have limited availability. Thus, physicians often rely on clinical findings and diagnostic tests—such as the electrocardiogram (ECG) and chest X-ray study—
with limited sensitivity, and the diagnosis of acute aortic dissection is often missed, with disastrous consequences (1,3). To achieve the goal of diagnosing all patients with acute aortic dissection, physicians must either perform more advanced imaging studies or use better screening tests to identify patients at higher risk.

Aortic dissection exposes underlying vascular tissue to blood and would be expected to activate the coagulation cascade via the extrinsic pathway. There may be subsequent formation of fibrin and fibrinolysis. D-dimer is a product of plasmin fibrinolysis of cross-linked fibrin. Investigators have postulated and shown that D-dimer is elevated in the setting of acute aortic dissection in all but one of multiple series (5–15). The purposes of this investigation were to define the sensitivity of D-dimer for acute aortic dissection by pooling data from all relevant series, and to assess the potential of the serum D-dimer as a test for patients who present with a low likelihood of acute aortic dissection.

**METHODS AND MATERIALS**

**Study Design**

This study was a meta-analysis of all consecutive case series of patients with acute aortic dissection and a measured D-dimer. Local institutional review board approval was obtained in the respective included studies as per regional regulations.

**Setting**

Data were collected from emergency and critical care units from around the world.

**Selection of Participants**

A comprehensive search using the terms “aortic dissection” and “D-dimer” was made of all English-language publications involving human subjects through February 2007. Databases searched included: MEDLINE, EMBASE, and the Cochrane Central Register of Controlled Trials. All original reports of consecutively enrolled patients with acute aortic dissection and a measured serum D-dimer were included. Case reports and studies where D-dimer was measured only after surgical intervention were excluded. Bibliographies of the identified reports and recent review articles were also checked for relevant studies for potential inclusion.

Study details, design, and method of patient identification and enrollment were tabulated (Table 1). Determination of optimal data collection to compare studies and to assess study quality was guided by the STARD statement for diagnostic studies, the MOOSE checklist for reporting meta-analyses of observational studies, and the QUADAS tool for systematic review of diagnostic studies (16–19). Aortic dissection was defined as acute using the common criterion of symptom onset within 14 days of presentation (2,20). Aortic dissection was diagnosed using standard criteria and modalities including TEE, transthoracic echocardiogram (TTE), CT, MRI, aortography, or postmortem examination (Table 1).

**Methods and Measurements**

Data and measurements recorded from each study included: the method of serum D-dimer measurement and its manufacturer, the number of aortic dissection patients in each study who did and did not have a serum D-dimer measurement during the study period, the average D-dimer value for patients with acute aortic dissection, and the number of these patients with a value greater or less than the threshold chosen for a positive test, 0.5 micrograms per milliliter. This value was empirically chosen as the threshold cutoff because it is the value commonly used to determine a positive test for venous thromboembolic disease, it was the test value used in a number of these studies, and it is the cutoff value for the semiquantitative latex agglutination test used (5,7,9,11). Other measurements included: the duration of symptoms at presentation, the distribution of dissection types according to the Stanford criteria, and the number of patients with a patent or thrombosed dissection lumen. If the study reported a control group, then control group characteristics were recorded, including: the criteria used for enrollment, the number of patients, the average D-dimer value, and the number of patients with D-dimer value greater or less than 0.5 μg/ml.

**Data Collection and Processing**

All data were abstracted from the original publications by a single unblinded investigator. Primary authors of the publications were contacted to provide clarification or additional data as indicated.

**Outcome Measures**

The primary outcome measure was the pooled sensitivity of the D-dimer test for acute aortic dissection. Additionally, a sensitivity analysis of the test sensitivity was
performed that included only those studies in which all acute aortic dissection patients had a D-dimer test and were enrolled. Two studies by the same group enrolled all patients who presented within 4 and 24 h of symptom onset, respectively, and they were included in the sensitivity analysis (9,13). Secondary outcomes included the specificity of the D-dimer test for acute aortic dissection in individual studies, and estimated positive and negative likelihood ratios. Specificity was not combined due to heterogeneous control groups.

### Primary Data Analysis

Excel (Office XP, Microsoft Corp., Redmond, WA) was used to tabulate data, Statxact software (Statxact 3, Version 3.0.2, Cytel Software Corporation, Cambridge, MA) was used to perform Fisher’s exact test, and Meta-DiSc (MetaDiSc, Version 1.3, Clinical Biostatistics, Ramon y Cajal Hospital, Madrid, Spain) was used to calculate the pooled sensitivity, its 95% confidence interval (CI), and to generate forest plots (21). The funnel plot was generated using Matlab (Matlab 6.5, The Mathworks, Natick, MA).

This analysis used a fixed-effect meta-analytic model. Sensitivity and specificity measurements were separately tested for heterogeneity using the Freeman-Halton extension of Fisher’s exact test. Sensitivity data from divergent studies were combined using the pooling meta-analytic method and a single diagnostic threshold value (21–23). When calculating a meta-analytic summary statistic, it is most common to use a weighted mean statistic where the weights are the inverse of the variance of each

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### Table 1. Publication Information

<table>
<thead>
<tr>
<th>Ref. #</th>
<th>Authors</th>
<th>Publication Date</th>
<th>Location</th>
<th>Acute Aortic Dissection Inclusion Criteria</th>
<th>Acute Aortic Dissection Diagnostic Modality</th>
<th>Study Design</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>Moriyama et al.</td>
<td>1998</td>
<td>Kagoshima, Japan</td>
<td>Diagnosed acute Stanford type B aortic dissection</td>
<td>CT, MRI</td>
<td>Case series</td>
</tr>
<tr>
<td>6</td>
<td>Weber et al.</td>
<td>2003</td>
<td>Wels, Austria</td>
<td>Diagnosed acute aortic dissection (10 prospective, 14 retrospective)</td>
<td>TTE, TEE, CT, MRI, angiography, or autopsy</td>
<td>Case-control</td>
</tr>
<tr>
<td>7</td>
<td>Eggebrecht et al.</td>
<td>2004</td>
<td>Essen and Munster, Germany</td>
<td>Chest pain with onset within 48 h and aortic dissection diagnosed</td>
<td>At least two modalities from: TEE, aortography, CT, or MRI</td>
<td>Prospective aortic disease cohort study</td>
</tr>
<tr>
<td>8</td>
<td>Perez et al.</td>
<td>2004</td>
<td>Hartford, CT, USA</td>
<td>Previously diagnosed acute aortic dissection (all confirmed acute presentations per personal communication – A. Perez)</td>
<td>CT, echocardiogram, angiogram</td>
<td>Retrospective cohort</td>
</tr>
<tr>
<td>9</td>
<td>Hazui et al.</td>
<td>2005</td>
<td>Osaka, Japan</td>
<td>Diagnosed ascending acute aortic dissection with symptom onset within 4 h</td>
<td>CT</td>
<td>Case-control</td>
</tr>
<tr>
<td>10</td>
<td>Akutsu et al.</td>
<td>2005</td>
<td>Tokyo, Japan</td>
<td>Chest or back pain and non-diagnostic ECG, and aortic dissection diagnosed</td>
<td>CT</td>
<td>Prospective cohort</td>
</tr>
<tr>
<td>11</td>
<td>Ohlmann et al.</td>
<td>2006</td>
<td>Strasbourg, France</td>
<td>Previously diagnosed acute aortic dissection</td>
<td>TEE, CT, MRI, angiography, autopsy</td>
<td>Retrospective cohort</td>
</tr>
<tr>
<td>12</td>
<td>Weber et al.</td>
<td>2006</td>
<td>Wels, Austria</td>
<td>Prospective cohort of patients with acute aortic dissection</td>
<td>CT, echocardiogram, autopsy</td>
<td>Prospective cohort</td>
</tr>
<tr>
<td>13</td>
<td>Hazui et al.</td>
<td>2006</td>
<td>Osaka, Japan</td>
<td>Diagnosed acute aortic dissection with symptom onset within 24 h</td>
<td>CT</td>
<td>Retrospective cohort</td>
</tr>
<tr>
<td>14</td>
<td>Monaco et al.</td>
<td>2006</td>
<td>Naples, Italy</td>
<td>Prospective cohort study of patients with Type B acute aortic dissection</td>
<td>CT</td>
<td>Prospective cohort</td>
</tr>
<tr>
<td>15</td>
<td>Sbarouni et al.</td>
<td>2007</td>
<td>Athens, Greece</td>
<td>Prospective cohort study</td>
<td>CT, echocardiogram</td>
<td>Prospective cohort</td>
</tr>
</tbody>
</table>

CT = computed tomography; MRI = magnetic resonance imaging; TTE = transthoracic echocardiography; TEE = transesophageal echocardiography.
individual value (24). In 7 of 11 studies in this analysis, the sensitivity was 100%. Consequently, the variances in these studies would be zero and the inverse would be infinity. In this situation, and particularly when combining sensitivity and specificity data, the statistic may be approximated by pooling the data where the weight of each study is essentially its sample size (21–23,25). No test for a threshold effect was performed due to heterogeneous control groups and specificity.

**RESULTS**

Twenty-one original studies were identified in the search. Eleven investigations were included, and five case reports, three reports of chronic or uncertain duration dissection, and two reports of post-operative patients were excluded (5–15,26–33). Study characteristics and quality information are reported in Tables 1–3. Table 1 displays the publication information, study design, acute aortic dissection inclusion criteria, and diagnostic modalities used for each study. Table 2 displays information about the acute aortic dissection patients, including: the duration of symptoms, number of patients with and without the D-dimer test during the study period, D-dimer methodology and manufacturer, results and test sensitivities, and acute aortic dissection characteristics. Table 3 displays control group information, including: the inclusion criteria, number of patients, D-dimer results, and test specificities. All studies were consistent with regard to
the following quality issues: all acute aortic dissection cases were verified with a reference test such as CT independent of the D-dimer test, but none of the radiologic assessments were explicitly blinded to the D-dimer results or other case information.

A funnel plot (Figure 1) of the sensitivity data vs. sample size was suggestive of publication bias. In the absence of publication bias, the point estimates of smaller studies should be approximately symmetrically distributed, but with more widely variable estimates, around the larger studies. In this case, the majority of smaller studies had a sensitivity estimate greater than the two larger studies and the distribution is not symmetric. Smaller studies with lower sensitivity point estimates may not have been published for a variety of reasons. This phenomenon is not unusual in meta-analyses of diagnostic test accuracy (34).

The tests for heterogeneity revealed a p value of < 0.0001 for both sensitivity and specificity. However, after removal of a single study with highly discordant results described below, the p value for sensitivity was 0.39 (14). This suggested that it was reasonable to pool the sensitivity, but not the specificity (Figure 2). The pooled sensitivity of the D-dimer test for patients with acute aortic dissection was 327/349, or 94% (95% CI 91–96). Including only those studies in which all acute aortic dissection patients had the D-dimer test, the sensitivity analysis revealed a test sensitivity of 183/192, or 95% (95% CI 91–98) (7,9,10,12,13,15). The specificities ranged from 40% to 100%. Conservatively assuming a
sensitivity of 94% and specificity of 40%, the estimated negative likelihood ratio is 0.15 and the positive likelihood ratio is 1.6. Therefore, for example, for a patient with an estimated pre-test risk of aortic dissection of 2.0% (odds 1 to 50), a negative D-dimer would be associated with a post-test probability of about 0.3% (odds .15 to 50).

There were a total of 22 acute aortic dissection patients in four studies in whom the D-dimer was less than the cutoff for a positive test (9,11,13,14). Hazui et al. found the following characteristics to be associated with a false-negative D-dimer in a logistic regression analysis: completely thrombosed dissection lumen, shorter length of dissection, and younger patient age (9,13). Ohlmann et al. identified 2 patients with false-negative D-dimer. One had a localized hematoma of the ascending aorta without intimal flap and the other also had an intramural hematoma (11). Monaco et al. found the majority of patients with type B dissection had a negative D-dimer on pre-operative testing before endovascular repair (14). This patient sample had a long 5.6-day average duration of symptoms before D-dimer measurement. Patients suffering from thoracic pain not responsive to morphine and anti-hypertensive therapy and with increasing false lumen diameter were chosen for dissection repair. The criterion for endovascular as opposed to open repair of type B dissection was “anatomic suitability for device implantation” (14,35). There may have been characteristics, such as limited extent of dissection, in patients chosen for endovascular repair that were also associated with lower D-dimer values. In these four studies, false-negative results were obtained with the following distri-

<table>
<thead>
<tr>
<th>Reference</th>
<th>Criteria Used for Control Group</th>
<th>Number of Control Group Patients</th>
<th>Advanced Imaging Performed on All Control Patients</th>
<th>Average D-dimer Value in Control Group Patients (μg/mL)</th>
<th>D-dimer Test Outcome in Controls with 0.5 μg/mL Threshold: Positive/Negative</th>
<th>Specificity of D-dimer Test for Acute Aortic Dissection (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moriyama et al. (5)</td>
<td>No control group</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Weber et al. (6)</td>
<td>Acute chest pain Chest pain with onset within 48 h and acute aortic dissection ruled out</td>
<td>35/48</td>
<td>Unspecified/Unspecified</td>
<td>Unspecified/0.6</td>
<td>11/24/16/32</td>
<td>69/67</td>
</tr>
<tr>
<td>Eggebrecht et al. (7)</td>
<td>Chronic aortic dissection</td>
<td>32</td>
<td>Unspecified</td>
<td>0.3</td>
<td>5/27†</td>
<td>81</td>
</tr>
<tr>
<td>Perez et al. (8)</td>
<td>No control group</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Hazui et al. (9)</td>
<td>Acute myocardial infarction with symptom onset within 4 h</td>
<td>49</td>
<td>Unspecified</td>
<td>0.4</td>
<td>10/39</td>
<td>80</td>
</tr>
<tr>
<td>Akutsu et al. (10)</td>
<td>Sudden chest or back pain and acute aortic dissection ruled out (including 7 with TAA/AAA)</td>
<td>48</td>
<td>CT</td>
<td>0.4</td>
<td>22/26 (all 7 TAA/AAA patients tested positive)</td>
<td>54</td>
</tr>
<tr>
<td>Ohlmann et al. (11)</td>
<td>Symptoms suspicious for acute aortic dissection and dissection ruled out</td>
<td>94</td>
<td>CT or TEE</td>
<td>0.6 (median)</td>
<td>56/38‡</td>
<td>40</td>
</tr>
<tr>
<td>Weber et al. (12)</td>
<td>No control group</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Hazui et al. (13)</td>
<td>No control group</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Monaco et al. (14)</td>
<td>No control group</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Sbarouni et al. (15)</td>
<td>Normal subjects</td>
<td>8</td>
<td>None§</td>
<td>0.2</td>
<td>0/8</td>
<td>100</td>
</tr>
<tr>
<td>Sbarouni et al. (15)*</td>
<td>Chronic aortic dissection</td>
<td>21</td>
<td>CT</td>
<td>1.3</td>
<td>8/13§</td>
<td>62</td>
</tr>
</tbody>
</table>

* Chronic aortic dissection control group.
† Personal communication, Eggebrecht, 2006.
‡ Personal communication, Ohlmann, 2006.
§ Personal communication, Georgiadou and Sbarouni, 2007.
TAA = Thoracic aortic aneurysm; AAA = Abdominal aortic aneurysm; CT = computed tomography; TEE = transesophageal echocardiography.
DISCUSSION

Acute pain in the chest or back is the most sensitive symptom of acute aortic dissection, but it is often nonspecific. Furthermore, approximately 10% of patients present without pain, and they complain instead of syncope, focal neurologic deficits, or other symptoms (2). Other clinical signs and commonly used bedside tests are even less sensitive for acute aortic dissection. Clinicians must have a low threshold to consider this lethal disease, but they have limited screening tools to rule it out without resorting to advanced imaging.

A number of tests have been explored for diagnosing acute aortic dissection, including measurement of smooth-muscle myosin heavy-chain protein and soluble D-Dimer for Acute Aortic Dissection 373
elastin fragments. Unfortunately, these tests seem to be 90% sensitive at best and suffer from high variability as a function of dissection duration, patient age, or false lumen patency (36,37). Serum D-dimer holds promise as an inexpensive, widely available, rapid, and sensitive test for acute aortic dissection.

D-dimer seems relatively sensitive, but poorly specific for acute aortic dissection. Similar to the utility of the D-dimer test in patients with suspected pulmonary embolism, it may be most useful as a screening test for patients at clinically low probability for acute aortic dissection (38). The clinical evaluation, including history, physical examination, and chest X-ray study, cannot rule out acute aortic dissection, but it can be used to identify a patient group with relatively low probability of acute aortic disease (2,39). Von Kodolitsch et al. found that the probability of acute aortic dissection in a high risk group of patients with suspected acute aortic dissection could be decreased from 50% to 7% if acute tearing or ripping pain, pulse or blood pressure differentials on examination, and mediastinal or aortic widening on chest X-ray were absent (39). A negative D-dimer could eliminate the need for further testing for acute aortic dissection in patients at low risk for disease based on clinical and chest X-ray evaluation. Patients at moderate or high risk of acute aortic dissection would probably not benefit from a D-dimer test as they would require definitive advanced imaging regardless of the D-dimer result.

Use of the D-dimer in combination with the clinical and chest X-ray evaluation may not necessarily lower the use of CT or increase the diagnosis of acute aortic dissection. Introduction of the rapid ELISA D-dimer for the diagnosis of venous thromboembolism in our academic ED was associated with an increase in pulmonary vascular imaging but no change in the rate of pulmonary embolism diagnosis (40). Nevertheless, the intention would be to obviate the need for advanced imaging such as CT in a subgroup of patients and to increase the pretest probability of aortic dissection in those patients undergoing such imaging.

In retrospect, the data set available to evaluate the D-dimer test for acute aortic dissection is relatively small and limited. Multiple questions and qualifications remain. The optimal D-dimer test methodology and associated threshold value to detect acute aortic dissection remain uncertain. Optimal threshold values may differ for different test methodologies (7,9,11,15). Beyond a single threshold for positivity, the quantitative value of a positive test may have further utility in predicting acute aortic dissection versus pulmonary embolism or elevation due to chronic disease. The D-dimer test may be less sensitive within the first 2 h of symptom onset, and it likely loses sensitivity as a function of acute aortic dissection duration (5–7,9,13,14,31,32,41). The size and characteristics of the dissection lesion may be important (6,11). Patients with isolated or thrombosed intramural hematomas may be particularly likely to have lower and false-negative D-dimer results (6,9,11,13). Dissection location seems to be less important (11).

Specificity of the D-dimer test for acute aortic dissection remains uncertain, but specificity of the D-dimer test generally decreases with advanced age and multiple chronic diseases. The additional benefit of D-dimer when added to a clinical diagnostic algorithm including chest X-ray study presumes independent predictive value of these tests. Available data suggest independence of the D-dimer test and radiographic mediastinal widening, but further verification of independence from this and other radiographic findings is needed (9).

Distinguishing acute myocardial infarction (MI) from acute aortic dissection can be both important and difficult. It may be critically important when considering heparin or thrombolytic therapy for acute MI as these therapies could be lethal to the acute aortic dissection patient. It may be difficult because approximately 7% of acute aortic dissection patients present with acute MI due to retrograde extension and involvement of the coronary artery ostia (2). D-dimer tends to be mildly elevated in acute MI, and the utility of this test to distinguish these two conditions remains uncertain (9,11).

**LIMITATIONS**

This pooled analysis and the individual studies upon which it is based have many limitations. Many of the studies were small with a retrospective cohort or case-control design. The funnel plot (Figure 1) suggests publication bias. There was potential sampling bias because in multiple studies the D-dimer test was ordered only on some acute aortic dissection patients, and the motivations for ordering the test in individual cases were generally unknown (5,6,8,11,14). Acute aortic dissection patients who did not have a D-dimer drawn may have had lower values. However, selective patient inclusion generally has not been found to significantly change the meta-analytic estimation of diagnostic accuracy (42). In this meta-analysis, the result of a sensitivity analysis that excluded these studies was essentially unchanged.

Advanced imaging was documented on all control patients in only two studies (10,11). It is possible that some patients with acute aortic dissection in the other studies were incorrectly diagnosed and labeled as controls. However, the morbidity and mortality of acute aortic dissection is high and increases over time. The likelihood of patients with this condition remaining undiagnosed without further symptoms or cardiovascular collapse seems low.
The D-dimer test was not standardized. ELISA and immunoturbidimetric D-dimer tests are generally more sensitive than latex agglutination tests for the diagnosis of deep venous thrombosis and pulmonary embolism (43,44). In this meta-analysis, the D-dimer test was particularly insensitive for acute aortic dissection in a single study that used the ELISA methodology. However, this may have been due to the markedly longer duration of patient symptoms and the types of dissection chosen for endovascular repair and D-dimer testing in this study (14). Overall, the robustness of the results despite the use of variable and less sensitive D-dimer tests in multiple diverse patient populations and presentations could be interpreted as a strength of this study and its conclusions.

No conclusions can be drawn on the specificity of the D-dimer test for acute aortic dissection due to the variable control groups employed. Only two studies enrolled patients with chest or back pain and suspected acute aortic dissection, and directly compared the results between patients diagnosed or ruled out for this disease (7,10). Even in these two studies, the control patients tended to have serious illnesses and did not represent all Emergency Department patients who present with chest pain. In one study, the investigators chose matched control patients in whom acute aortic dissection had been ruled out (11). Two studies enrolled patients with acute chest pain or myocardial infarction without aortic dissection as controls, but the procedure to rule out acute aortic dissection in each patient was not explicitly stated (6,9). Two studies also included a control group of patients with chronic aortic dissection (7,15).

CONCLUSIONS

There is much yet to be learned about the utility of the D-dimer test for acute aortic dissection, and further large studies are needed. Nevertheless, it seems to be a sensitive tool in assessing for acute aortic dissection and may aid the clinician in low probability patients where diagnostic uncertainty remains. Its use may help to address a difficult unsolved diagnostic emergency medical problem.

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