Warm up angina / Pre-conditioning
Warm up angina / Pre-conditioning
Figure 2 Factors affecting preconditioning and its impact on cardiac events

Chest Pain

High Risk Features
Duration 20-30 mins
Diaphoresis / SOB
After Food / Shower
Pre-conditioning
Diffuse pain
Radiation to arms / jaws
Severe
Urgent referral

Low risk features
Short Duration < 5 mins
Focal Pain
With Oily Food
Likely non-cardiac
Symptomatic Treatment

Consider referral if persistent
Acute Chest Rest Pain Syndrome

High Risk Features

OBVIOUS STEMI / NSTEMI

STEMI / NSTEMI pathways

ECG

Normal
Non-acute Abnormalities

Hs-TnT at 0 and 3 hours Abnormal

Hs-TnT at 0 and 3 hours Normal / Non-trending

Life Threatening
Aortic Dissection
Ruptured Viscus
Internal Haemorrhage

Not ACS
Consider Alternative Diagnosis

Non-Life Threatening
Gastritis
MSK pain
Herpes Zoster

Non-Urgent Screening for Stable IHD if needed
<table>
<thead>
<tr>
<th></th>
<th>0h/3 h algorithm</th>
<th>0h/1 h algorithm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative predictive value for acute MI</td>
<td>98–100%</td>
<td>98–100%</td>
</tr>
<tr>
<td>Positive predictive value for acute MI</td>
<td>Unknown, depending on delta change and assay</td>
<td>75–80%</td>
</tr>
<tr>
<td>Effectiveness^a</td>
<td>++</td>
<td>+++</td>
</tr>
</tbody>
</table>
| Feasibility             | ++
|                         | requires GRACE score               | +++                               |
| Challenges              | Pain onset cannot be reliably quantified in many patients | Cut-off levels are assay-specific and different from the 99th percentile |
| Validation in large multicentre studies | +                                 | +++                               |
| Additional advantages   | Already used clinically           | Shorter time to decision          |

GRACE = Global Registry of Acute Coronary Events; MI = myocardial infarction.

^aEffectiveness is quantified by the percentage of consecutive chest pain patients clearly classified as rule-out or rule-in of acute MI (i.e., approximately 60% for the 0 h/3 h algorithm and approximately 75% for the 0 h/1 h algorithm).
Acute Rest Pain Syndrome

High Risk Features

OBVIOUS STEMI / NSTEMI

STEMI / NSTEMI pathways

ECG

Normal Non-acute Abnormalities

Hs-TnT at 0 and 3 hours Abnormal

Hs-TnT at 0 and 3 hours Normal / Non-trending

Life Threatening
Aortic Dissection
Ruptured Viscus
Internal Haemorrhage

Not ACS
Consider Alternative Diagnosis

Non-Life Threatening
Gastritis
MSK pain
Herpes Zoster!

Non-Urgent Screening for Stable IHD if needed
Acute Rest Pain Syndrome

High Risk Features

OBVIOUS STEMI / NSTEMI

STEMI / NSTEMI pathways

ECG

Normal Non-acute Abnormalities

Hs-TnT at 0 and 3 hours Abnormal

Hs-TnT at 0 and 3 hours Normal / Non-trending

Life Threatening
Aortic Dissection
Ruptured Viscus
Internal Haemorrhage

Not ACS
Consider Alternative Diagnosis

Non-Life Threatening
Gastritis
MSK pain
Herpes Zoster!

Non-Urgent Screening for Stable IHD if needed
Acute aortic syndrome

Although the chest pain of acute aortic dissection is widely recognised, less consideration has been given to pain associated with other aortic pathologies. In light of contemporary concepts in aortic pathology we would like to present the pathology of a new cardiovascular syndrome—acute aortic syndrome (AAS).

This syndrome embraces a heterogeneous group of patients with a similar clinical profile that includes penetrating atherosclerotic aortic ulcer, intramural aortic haematoma, and the classic aortic dissection (fig 1). The physiopathological mechanism that precipitates the appearance of each of these entities is different. However, occasionally some patients exhibit several or all of these lesions, demonstrating the existence of a link between them. In such cases it is difficult to know which was the initiating event.

AAS is characterised clinically by aortic pain in a patient with a coexisting history of hypertension. In acute coronary syndromes, the existence of a typical chest pain that, since Heberden, has been called angina pectoris is well

Figure 2  Histological section (Mason’s technique) from a patient with aortic dissection. Muscle is stained in red and collagen in green. The aortic media (stained in red) is partitioned in two (arrows); one forms part of the dissection flap, the other forms the outer wall of the false channel. Large arrow indicates the dissection flap. LF, false lumen; LV, true lumen.
Figure 1  Acute aortic syndrome (AAS). Arrows indicate the possible progression of each of these aortic lesions.
De Bakey Type I  Type II  Type III

Stanford  Type A  Type B

**De Bakey**

Type I  Originates in the ascending aorta, propagates at least to the aortic arch and often beyond it distally.

Type II  Originates in and as confined to the ascending aorta.

Type III  Originates in the descending aorta and extends distally down the aorta or, rarely retrograde into the aortic arch and ascending aorta.

**Stanford**

Type A  All dissections involving the ascending aorta, regardless of the site of origin.

Type B  All dissections not involving the ascending aorta.
Stanford Type A “Aortic Dissection”

Stanford Type A “Intramural Hematoma”
Stanford Type B
“Penetrating Arterial Ulcer”
53 year old man
- chest pain
  - rest
  - 5 minutes each
  - not with exertion

ECG
- CK / Mb / TnT – normal x 1
Stanford Type A “Aortic Dissection”
66 year old man

Chest Pain 1 hour
BP 87/50
Radiate to back
Nausea SOB diaphoresis
E-COROS– minor CAD
Stanford Type A “Intramural Hematoma”
Diagnosis

History

CXR
ECG
D-dimers
D-Dimers

Diagnosis of Acute Aortic Dissection by D-Dimer
The International Registry of Acute Aortic Dissection Substudy on Biomarkers (IRAD-Bio) Experience

Tori Suzuki, MD; Alessandro Dianate, MD; Antonella Zizza, MS; Santi Trimarchi, MD; Massimo Villani, MD; Jorge Antonio Salerno Uriarte, MD; Luigi De Luca Tuppini Schinos, MD; Attilio Renzulli, MD; Federico Sabino, MD; Richard Nowak, MD; Robert Birkland, MD; Judd E. Holland, MD; Francis Counselman, MD; Ravi Vijayendran, PhD; Eduardo Bossone, MD; Kim Eagle, MD; for the IRAD-Bio Investigators

Background—D-dimer has been reported to be elevated in acute aortic dissection. Potential use as a "rule-out" marker has been suggested, but concerns remain given that it is elevated in other acute chest diseases, including pulmonary embolism and ischemic heart disease. We evaluated the diagnostic performance of D-dimer testing in a study population of patients with suspected aortic dissection.

Methods and Results—In this prospective multicenter study, 220 patients with initial suspicion of having acute aortic dissection were enrolled, of whom 87 were diagnosed with acute aortic dissection and 133 with other final diagnoses, including myocardial infarction, angina, pulmonary embolism, and other uncertain diagnoses. D-dimer was markedly elevated in patients with acute aortic dissection. Analysis according to control disease, type of dissection, and time course showed that the widely used cutoff level of 500 ng/mL for ruling out pulmonary embolism may also reliably rule out acute dissection, with a negative likelihood ratio of 0.07 throughout the first 24 hours.

Conclusions—D-dimer levels may be useful in risk stratifying patients with suspected aortic dissection to rule out acute dissection if used within the first 24 hours after symptom onset. (Circulation. 2009;119:2702-2707.)

Key Words: aorta ■ diagnosis ■ peripheral vascular disease

A acute aortic dissection (AD) remains a potentially catastrophic cardiovascular disease. Recent advancements in imaging methods (eg, computed tomography, magnetic resonance imaging) and the development of novel biochemical diagnostic methods (eg, smooth muscle myosin heavy chain) have made possible improved diagnosis of the disease to allow early and optimized treatment. However, the disease at times remains overlooked or misdiagnosed because of its relatively uncommon nature. A diagnostic test that can reliably identify or exclude this disease in a cost-effective and resource-efficient manner such as a blood assay would be very useful.

Clinical Perspective on p 2707

D-dimer, a fibrin fragment seen in coagulopathic disorders and now commonly used in the diagnosis of pulmonary embolism (PE), has recently been reported to be elevated in acute AD. D-dimer has been suggested to be useful as a "rule-out" diagnostic tool. Because most of the early studies used samples from selected patients, however, investigation of the performance of the assay in a clinically relevant population being investigated for suspected AD is necessary to accurately describe the usefulness of the assay.

The International Registry of Acute Aortic Dissection Substudy on Biomarkers (IRAD-Bio study) was established to investigate and develop biomarkers of acute AD. In the present study, we evaluated the diagnostic performance of D-dimer in acute AD in a population suspected of having the disease.

Methods

Patients and Samples
Fourteen centers in Europe, the United States, and Japan participated in the present study (see the Appendix in the online-only Data Supplement available with this article at http://circ.ahajournals.org). The online-only Data Supplement is available with this article at http://circ.ahajournals.org. Correspondence to Toru Suzuki, MD, Department of Cardiovascular Medicine, Graduate School of Medicine, The University of Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113-8655 Japan. E-mail tsnzumi@umin.ac.jp

© 2009 American Heart Association, Inc.

Circulation is available at http://circ.ahajournals.org

Downloaded from http://circ.ahajournals.org by guest on February 22, 2012
D-Dimers

Figure 2. Time course box plots for D-dimer levels in patients according to time from onset.
D-Dimers

Table 4. Diagnostic Performance of D-Dimer for Patients Presenting Within the First 6 Hours at a Cutoff of 500 ng/ml

<table>
<thead>
<tr>
<th>AD and Control</th>
<th>Sensitivity, %</th>
<th>Sensitivity 95% CI</th>
<th>Specificity, %</th>
<th>Specificity 95% CI</th>
<th>PLR</th>
<th>NLR</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>A and B</td>
<td>95.7</td>
<td>78.1–99.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>61.3</td>
<td>42.2–78.2</td>
<td>2.47</td>
<td>0.07</td>
<td>45.2</td>
<td>97.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MI only</td>
<td>55.6</td>
<td>21.2–86.3</td>
<td>2.15</td>
<td>0.08</td>
<td>41.8</td>
<td>97.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angina only</td>
<td>64.3</td>
<td>35.1–87.2</td>
<td>2.68</td>
<td>0.07</td>
<td>47.2</td>
<td>97.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PE only</td>
<td>50.0</td>
<td>1.3–98.7</td>
<td>1.91</td>
<td>0.09</td>
<td>38.9</td>
<td>97.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other only</td>
<td>66.7</td>
<td>22.3–95.7</td>
<td>2.87</td>
<td>0.07</td>
<td>48.9</td>
<td>97.9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations as in Table 2.
<table>
<thead>
<tr>
<th>Presenting Hemodynamics and Clinical Findings</th>
<th>Frequency/Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive</td>
<td>32%</td>
</tr>
<tr>
<td>Normotensive</td>
<td>45%</td>
</tr>
<tr>
<td>Hypotensive</td>
<td>14%</td>
</tr>
<tr>
<td>Shock</td>
<td>13%</td>
</tr>
<tr>
<td>Cardiac tamponade</td>
<td>5%</td>
</tr>
<tr>
<td>Murmur of aortic insufficiency</td>
<td>45%</td>
</tr>
<tr>
<td><strong>Pulse deficits</strong></td>
<td><strong>26%</strong></td>
</tr>
<tr>
<td>Pericardial friction rub</td>
<td>2%</td>
</tr>
<tr>
<td>Cerebrovascular accident</td>
<td>8%</td>
</tr>
<tr>
<td>Ischemic peripheral neuropathy</td>
<td>3%</td>
</tr>
<tr>
<td>Ischemic spinal cord damage</td>
<td>2%</td>
</tr>
<tr>
<td>Ischemic lower extremity</td>
<td>10%</td>
</tr>
<tr>
<td>Coma/altered consciousness</td>
<td>12%</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>5%</td>
</tr>
<tr>
<td>First blood pressure systolic, mean</td>
<td>130 mm Hg</td>
</tr>
<tr>
<td>First blood pressure diastolic, mean</td>
<td>75 mm Hg</td>
</tr>
</tbody>
</table>

Adapted from Pape et al.\textsuperscript{227}
High Risk Features

Clinical

Pain – severe and acute
Pulse deficit > 20 mm Hg
Evidence of Aortic regurgitation
Family History
Associated Connective Tissue (Marfan’s)
Tests

ECG – MI is much more common
CXR – normal does not exclude

TEE / CT / MRI depending on local resources
Repeat if negative and clinically suspicious
Chest Pain

Exertional Pain

Typical Angina
Not ACS
Possible Stable IHD

Aspirin
Nitrates
Beta-Blockers (HR < 75)
Statins

Refer for risk stratification
Acute Rest Pain Syndrome

High Risk Features

OBVIOUS STEMI / NSTEMI

STEMI / NSTEMI pathways

ECG

Normal Non-acute Abnormalities

Hs-TnT at 0 and 3 hours
Abnormal

Life Threatening
Aortic Dissection
Ruptured Viscus
Internal Haemorrhage

Not ACS
Consider Alternative Diagnosis

Non-Life Threatening
Gastritis
MSK pain
Herpes Zoster!

Non-Urgent Screening for Stable IHD if needed