Long-term Follow-up of Aortic Intramural Hematomas and Penetrating Ulcers

Alan S. Chou, BA, Bulat A. Ziganshin, MD, Paris Charilaou, MD, Maryann Tranquilli, RN, John A. Rizzo, PhD, John A. Elefteriades, MD

Aortic Institute at Yale-New Haven Hospital, Yale University School of Medicine
New Haven, Connecticut, USA

Presented at the 95th Annual Meeting of the American Association of Thoracic Surgery
April 28th, 2015
Disclosures

• Alan S. Chou, BA:
  – Nothing to disclose.

• Bulat A. Ziganshin, MD:
  – Nothing to disclose.

• Paris Charilaou, MD:
  – Nothing to disclose.

• Maryann Tranquilli, RN:
  – Nothing to disclose.

• John A. Rizzo, PhD:
  – Nothing to disclose.

• John A. Elefteriades, MD:
  – DSMB for Jarvik Heart, Solus Valve; Research Grant Vascutek; Medical Director for CoolSpine.
Acute Aortic Syndromes

# Current literature/evidence

<table>
<thead>
<tr>
<th>Author</th>
<th>Institution and Date</th>
<th>Lesion</th>
<th>Treatment Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tittle et al.</td>
<td>Yale University, 2002</td>
<td>IMH, Type A and B</td>
<td>Expanded <strong>surgical</strong> treatment</td>
</tr>
<tr>
<td>Moizumi et al.</td>
<td>Tohuku University, 2003</td>
<td>IMH, Type A and B</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Kaji et al.</td>
<td>Kobe City General Hospital, 2003</td>
<td>IMH, Type B</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Evangelista et al.</td>
<td>IRAD, 2005</td>
<td>IMH, Type A</td>
<td>Initial <strong>surgical</strong> treatment</td>
</tr>
<tr>
<td>Evangelista et al.</td>
<td>IRAD, 2005</td>
<td>IMH, Type B</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Kitai et al.</td>
<td>Kobe City General Hospital, 2008</td>
<td>IMH, Type A</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Shimokawa et al.</td>
<td>Sakakibara Heart Institute, 2008</td>
<td>IMH, Type A</td>
<td>Initial <strong>surgical</strong> treatment</td>
</tr>
<tr>
<td>Song et al.</td>
<td>Asan Medical Center, 2009</td>
<td>IMH, Type A</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Sawaki et al.</td>
<td>Nagoya Ekisaikai Hospital, 2010</td>
<td>IMH, Type A</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Tolenaar et al.</td>
<td>IRAD, 2013</td>
<td>IMH, Type B</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Tittle et al.</td>
<td>Yale University, 2002</td>
<td>PAU</td>
<td>Expanded <strong>surgical</strong> treatment</td>
</tr>
<tr>
<td>Cho et al.</td>
<td>Mayo Clinic, 2003</td>
<td>PAU</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Nathan et al</td>
<td>University of Pennsylvania, 2012</td>
<td>PAU</td>
<td>Radiologic follow-up of symptomatic patients</td>
</tr>
</tbody>
</table>
# Current literature/evidence

<table>
<thead>
<tr>
<th>Author</th>
<th>Institution and Date</th>
<th>Lesion</th>
<th>Treatment Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tittle et al.</td>
<td>Yale University, 2002</td>
<td>IMH, Type A and B</td>
<td>Expanded <strong>surgical</strong> treatment</td>
</tr>
<tr>
<td>Moizumi et al.</td>
<td>Tohuku University, 2003</td>
<td>IMH, Type A and B</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Kaji et al.</td>
<td>Kobe City General Hospital, 2003</td>
<td>IMH, Type B</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Evangelista et al.</td>
<td>IRAD, 2005</td>
<td>IMH, Type A</td>
<td>Initial <strong>surgical</strong> treatment</td>
</tr>
<tr>
<td>Evangelista et al.</td>
<td>IRAD, 2005</td>
<td>IMH, Type B</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Kitai et al.</td>
<td>Kobe City General Hospital, 2008</td>
<td>IMH, Type A</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Shimokawa et al.</td>
<td>Sakakibara Heart Institute, 2008</td>
<td>IMH, Type A</td>
<td>Initial <strong>surgical</strong> treatment</td>
</tr>
<tr>
<td>Song et al.</td>
<td>Asan Medical Center, 2009</td>
<td>IMH, Type A</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Sawaki et al.</td>
<td>Nagoya Ekisaikai Hospital, 2010</td>
<td>IMH, Type A</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Tolenaar et al.</td>
<td>IRAD, 2013</td>
<td>IMH, Type B</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Tittle et al.</td>
<td>Yale University, 2002</td>
<td>PAU</td>
<td>Expanded <strong>surgical</strong> treatment</td>
</tr>
<tr>
<td>Cho et al.</td>
<td>Mayo Clinic, 2003</td>
<td>PAU</td>
<td>Initial <strong>medical</strong> treatment with expectant surgery</td>
</tr>
<tr>
<td>Nathan et al</td>
<td>University of Pennsylvania, 2012</td>
<td>PAU</td>
<td>Radiologic follow-up of symptomatic patients</td>
</tr>
</tbody>
</table>
US and European Guidelines

2010 ACCF/AHA/ACR/ASQC/SCA/SIR/STV Guidelines for the Diagnosis and Management of Patients With Thoracic Aortic Disease


Endorsed by the North American Society for Cardiovascular Imaging

WRITING GROUP MEMBERS
Loren F. Hiratzka, MD, Chair†; George L. Bakris, MD; Joshua A. Beckman, MD, MS; Robert M. Berrin, MD; Vincent F. Carr, DO; Donald E. Casey Jr, MD, MPH, MBA; Kim A. Eagle, MD†; Luke K. Hermann, MD**, Eric M. Iselbuch, MD†; Ella A. Kazerouni, MD, MS†; Nicholas T. Kouchoukos, MD; Bruce W. Lytle, MD; Dianna M. Milwidsz, MD, PhD; David L. Reich, MD; Souvick Sen, MD, MSF; Julie A. Shinn, RN, MA, CCRN†; Lars G. Svensson, MD, PhD; David M. Williams, MD***

ACCF/AHA TASK FORCE MEMBERS
Alice K. Jacobs, MD, FACC, FAHA, Chair 2009–2011; Sidney C. Smith, Jr, MD, FACC, FAHA, Immediate Past Chair 2006–2008††; Jeffrey L. Anderson, MD, FACC, FAHA, Chair-Elect; Cynthia D. Adams, MSN, PhD, FAHA†††; Christopher E. Muller, MD, FACC; Mark A. Creager, MD, FACC, FAHA; Steven M. Ettinger, MD, FACC; Robert A. Guyton, MD, FACC, FAHA; Jonathan L. Halperin, MD, FACC, FAHA; Stefan H, MD, FACC, FAHA†††; Harlan M. Krumholz, MD, FACC, FAHA; Frederick G. Kushner, MD, FACC, FAHA; Bruce W. Lytle, MD, FACC, FAHA†††; Rick Nishimura, MD, FACC, FAHA†††; Richard L. Page, MD, FACC, FAHA††; Barbara Riegel, DNcC, RN, FAHA****; William G. Stevenson, MD, PhD; Lynn T. Tarkington, RN; Clyde W. Yancy, MD, FACC, FAHA

*ACCF/AHA Representative. FAHA Representative. SFVM Representative. SCAC Representative. ACCF/ESC Representative. *American College of Physicians Representative. †††Updated from Section 9.2.2.3.1. Recommendations for Descending Thoracic Aorta and Thoracoabdominal Aortic Aneurysms. **American College of Emergency Physicians Representative. ***TCTA Representative. †††TRUST Representative. †††ESC Task Force Liaison. †‡ESC Representative. †‡‡ESC Representative. †‡§SIR Representative. †||Isthmus Task Force Member during the writing effort.

Authors with no symbol by their name were included to provide additional content expertise apart from organizational representation.

This document was approved by the American College of Cardiology Foundation Board of Trustees and the American Heart Association Science Advisory and Coordinating Committee in January 2010. All other coauthoring organizations approved in February 2010.


This article has been copublished in Circulation.

Covers: This document is available on the World Wide Web sites of the American College of Cardiology (www.acc.org) and the American Heart Association (my.americanheart.org). For copies of this document, please contact Elsevier Inc. Reprint Department, Fax: 212-633-3820, e-mail: reprints@elsevier.com.

Permissions: Multiple copies, modification, alteration, enhancement, and/or distribution of this document are not permitted without the express permission of the American College of Cardiology Foundation. Please contact Elsevier’s permission department at healthpermissions@elsevier.com.

EUROPEAN GUIDELINES

2014 ESC Guidelines on the diagnosis and treatment of aortic diseases

Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult

The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC)

Authors/Task Force members: Raimund Erbel* (Chairperson) (Germany), Victor Abouyans* (Chairperson) (France), Catherine Boileau (France), Eduardo Bossone (Italy), Roberto Di Bartolomeo (Italy), Holger Eggebrecht (Germany), Arturo Evangelista (Spain), Vollemar Fall (Switzerland), Herbert Frank (Austria), Oliver Gaemperli (Switzerland), Martin Grubenweger (Austria), Axel Haeverich (Germany), Bernard Jurg (France), Athanasios John Manolis (Greece), Folkert Meijboom (Netherlands), Christoph A. Nienaber (Germany), Marco Roffi (Switzerland), Hervé Rousseau (France), Udo Sechtem (Germany), Per Anton Sirnes (Norway), Regula S. von Allmen (Switzerland), Christiaan J.M. Vrints (Belgium).

ESC Committee for Practice Guidelines (CPG): Jose Luis Zamorano (Chairperson) (Spain), Stephan Achenbach (Germany), Helmut Baumann-Gutierrez (Germany), Jeroen J. Bax (Netherlands), Héctor Bueno (Spain), Veronica Dean (France), Christof Dressel (UK), Cetin Erdi (Turkey), Robert Fagard (Belgium), Roberto Ferrario (Italy), David Hassid (Israel), Arnold Hoes (The Netherlands), Paulus Kirchhof (Germany/UK), Johan Knutti (Finland), Philippe Kolb

*Corresponding author: Raimund Erbel, Department of Cardiology, West German Heart Centre Essen, University Duisburg Essen, Huetteweg 55, D-45122 Essen, Germany. Tel: +49 201 752 4691; Fax: +49 201 752 4565; Email: raimund.erbel@uk-essen.de.

Victor Abouyans, Department of Cardiology, CHRU, Dijon University Hospital, 2 Avenue Marceau Luther King; 21034 Dijon, France. Tel: +33 3 80 65 10 61; Fax: +33 3 80 65 65 94; Email: vabouyans@chu-dijon.fr

© 2014 The European Society of Cardiology. All rights reserved. For permissions, please email journals.permissions@esoc.org.
Aim of the study

• To evaluate the long-term natural history and progression of intramural hematomas and penetrating aortic ulcers including clinical and radiological follow-up and survival analysis.
Progression of Lesions

PATHOLOGIC VARIANTS OF THORACIC AORTIC DISSECTIONS
Penetrating Atherosclerotic Ulcers and Intramural Hematomas

Michael A. Coady, MD, MPH, John A. Rizzo, PhD, and John A. Elefteriades, MD

Penetrating atherosclerotic ulcer (PAU) and intramural hematoma (IH) of the thoracic aorta were virtually unknown in the pre-digital imaging era. Presentations were not defined as radiographic until the mid-1980s. By the mid-1990s, most presentations were due to three-dimensional, high-resolution imaging by computed tomography (CT), magnetic resonance (MR), and transesophageal echocardiography. These two disorders have become well recognized. The distinctions from typical aortic dissection have not always been clear, with terms of diagnosis, clinical character, and treatment.

Most reports of PAU and IH in literature have centered on identifying disorders as entities distinct from classic aortic dissection by describing small numbers of patients. This growing body of knowledge has allowed the aortic pathology and clinical picture of PAU and IH to differ slightly from those of classic aortic dissection. Then, clinical management may need to be considered.

These reports have often failed to identify the actual number of patients in whom these rare presentations of aortic dissection have been made. This was true even where the initial description of a disorder or variant was made.

Objective: Most studies on variant forms of aortic dissection—penetrating ulcer and intramural hematoma—have focused on the initial presenting episode, with acute follow-up. This investigation provides midterm follow-up of penetrating ulcer and intramural hematoma to determine whether the aorta heals according to radiography, goes on to disintegrate, or tends to rupture during longer follow-up.

Methods: Five sites with penetrating ulcers (n = 26) or intramural hematomas (n = 19) were treated at our institution. Ten sites with penetrating ulcers were male and 6 were female, and their ages ranged from 54 to 87 years (mean 72 years). Eight sites with intramural hematomas were male and 11 were female, and their ages ranged from 54 to 88 years (mean 74 years). These patients all had symptoms of aortic disease. Patients with incidental imaging findings were not considered.

Results: In the group with penetrating ulcers, rupture occurred during the initial admission in 10 (38%) cases, 17 patients (65%) underwent surgery, and 22 patients (85%) survived to hospital discharge. Among those with intramural hematomas, rupture occurred during the initial admission in 9 cases (47%), 7 patients (37%) underwent surgery, and 10 patients (55%) survived to hospital discharge. Follow-up ranged from 1 month to 12 years (mean 3.4 years). No ischemic vascular complications occurred. Imaging follow-up was available for 26 of the 45 patients. Of these, 19% of lesions showed resolution, 23% had worsened, 39% had progressed to typical dissection, and 19% were unchanged. Stent diameters were known to cause rupture. In the group with penetrating ulcers, aortic diameter increased from 4.8 to 5.1 cm during the course of 14 months. In the group with intramural hematomas, aortic diameter increased from 5.3 to 5.9 cm during the course of 3 months. Overall survival was 100% at 1 year, 79% at 5 years, and 50% at 5 years.

Conclusion: Intramural hematomas and penetrating ulcers are lesions associated with advanced age. Women predominated. Penetrating ulcer and intramural hematoma rupture both early and late. Radiographically documented worsening, improvement, or frank disintegration may occur with time. Aortic growth does occur in the case of penetrating ulcer and 0.4 cm per year for intramural hematoma. Vascular ischemic complications do not occur. Because of the high early rupture rate, the frequency of radiographic worsening, and the documented occurrence of late rupture, we now recommend surgical replacement of the aorta for these variant vascular lesions as long as the patient's comorbidities do not preclude surgical intervention.
Progression of Lesions

PATHOLOGIC VARIANTS OF THORACIC AORTIC DISSECTIONS

Penetrating Atherosclerotic Ulcers and Intramural Hematomas

Michael A. Coady, MD, MPH, John A. Rizzo, PhD, and John A. Elefteriades, MD

Penetrating atherosclerotic ulcer (PAU) and intramural hematoma (IMH) of the thoracic aorta were virtually unknown in the preangiographic imaging era until the mid-1980s.1-3 In the era of three-dimensional, high-resolution imaging of the aorta by computed tomography (CT), magnetic resonance (MR), and transesophageal echocardiography, these disorders have become more recognizable. The distinctions from typical dissection have not always been clear due to significant overlap in terms of diagnosis, clinical presentation, and management.

Most reports of PAU and IMH in the literature have focused on identifying disorders that are distinct from classic dissection by describing smaller, more localized lesions that have been called subintimal or intramural aortic hematomas.7-13 However, PAU may be difficult to distinguish from other aortic disorders, and clinical management may require further evaluation and treatment.

For the Department of Surgery, Section of Cardiothoracic Surgery, University of California, San Francisco, California.

Address for replies: John A. Elefteriades, MD, 1150 Walnut St, San Francisco, CA 94117-0140 (e-mail: john.elefteriades@ucsf.edu).

From the Section of Cardiothoracic Surgery and the Department of Diagnostic Radiology, University of California, San Francisco, California.


doi:10.1001/01.tvt.11.6.695

Objective: Most studies on variant forms of aortic dissection—penetrating ulcer and intramural hematoma—have focused on the initial presenting episode. We aimed to follow-up this patient population to determine whether the aortic lesion heals according to clinical and imaging findings, or leads to rupture during future follow-up.

Methods: Five patients with penetrating ulcers (n = 26) or intramural hematomas (n = 10) were treated at our institution. Ten patients with penetrating ulcers were male and 8 were female, and their ages ranged from 41 years to 78 years (mean 72 years). Eight patients with intramural hematomas were male and 2 were female, and their ages ranged from 41 years to 88 years (mean 74 years). Patients were monitored for symptoms of aortic disease. Patient outcomes were analyzed using Kaplan-Meier survival analysis.

Midterm follow-up of penetrating ulcer and intramural hematoma of the aorta

Dannen L. Tita, MD
Raymond J. Lynch, BS
Patricia E. Cole, MD
Herschel S. Singh, BS
John A. Rizzo, PhD
Gary S. Kaplan, MD
John A. Elefteriades, MD

Surgery for Acquired Cardiovascular Disease

Midterm follow-up of penetrating ulcer and intramural hematoma of the aorta

Objective: Most studies on variant forms of aortic dissection—penetrating ulcer and intramural hematoma—have focused on the initial presenting episode. We aimed to follow-up this patient population to determine whether the aortic lesion heals according to clinical and imaging findings, or leads to rupture during future follow-up.

Methods: Five patients with penetrating ulcers (n = 26) or intramural hematomas (n = 10) were treated at our institution. Ten patients with penetrating ulcers were male and 8 were female, and their ages ranged from 41 years to 78 years (mean 72 years). Eight patients with intramural hematomas were male and 2 were female, and their ages ranged from 41 years to 88 years (mean 74 years). Patients were monitored for symptoms of aortic disease. Patient outcomes were analyzed using Kaplan-Meier survival analysis.

Midterm follow-up of penetrating ulcer and intramural hematoma of the aorta

Dannen L. Tita, MD
Raymond J. Lynch, BS
Patricia E. Cole, MD
Herschel S. Singh, BS
John A. Rizzo, PhD
Gary S. Kaplan, MD
John A. Elefteriades, MD

Surgery for Acquired Cardiovascular Disease

Midterm follow-up of penetrating ulcer and intramural hematoma of the aorta

Objective: Most studies on variant forms of aortic dissection—penetrating ulcer and intramural hematoma—have focused on the initial presenting episode. We aimed to follow-up this patient population to determine whether the aortic lesion heals according to clinical and imaging findings, or leads to rupture during future follow-up.

Methods: Five patients with penetrating ulcers (n = 26) or intramural hematomas (n = 10) were treated at our institution. Ten patients with penetrating ulcers were male and 8 were female, and their ages ranged from 41 years to 78 years (mean 72 years). Eight patients with intramural hematomas were male and 2 were female, and their ages ranged from 41 years to 88 years (mean 74 years). Patients were monitored for symptoms of aortic disease. Patient outcomes were analyzed using Kaplan-Meier survival analysis.

Surgery for Acquired Cardiovascular Disease

Midterm follow-up of penetrating ulcer and intramural hematoma of the aorta

Objective: Most studies on variant forms of aortic dissection—penetrating ulcer and intramural hematoma—have focused on the initial presenting episode. We aimed to follow-up this patient population to determine whether the aortic lesion heals according to clinical and imaging findings, or leads to rupture during future follow-up.

Methods: Five patients with penetrating ulcers (n = 26) or intramural hematomas (n = 10) were treated at our institution. Ten patients with penetrating ulcers were male and 8 were female, and their ages ranged from 41 years to 78 years (mean 72 years). Eight patients with intramural hematomas were male and 2 were female, and their ages ranged from 41 years to 88 years (mean 74 years). Patients were monitored for symptoms of aortic disease. Patient outcomes were analyzed using Kaplan-Meier survival analysis.
Progression of Lesions

Resolution

No Change

(Stable)
PATHOLOGIC VARIANTS OF THORACIC AORTIC DISSECTIONS

Penetrating Atherosclerotic Ulcers and Intramural Hematomas

Michael A. Coady, MD, MPH, John A. Rizzo, PhD, and John A. Elefteriades, MD

Penetrating atherosclerotic ulcer (PAU) and intramural hematoma (IMH) of the aorta were virtually unknown in the preangiographic era. Recent advances in multidetector computed tomography, magnetic resonance imaging, and transesophageal echocardiography have made PAU and IMH readily identifiable. The classification and natural history of these entities are the topic of this review.

Progression of Lesions

Resolution
No Change (Stable)
Worsening

Objective: To review the natural history of PAU and IMH.

Methods: A review of the literature was performed.

Results: PAU and IMH are rare entities. The natural history of PAU and IMH is not well understood. The majority of cases are asymptomatic, and the prognosis is generally good. In symptomatic cases, the natural history is more variable and depends on the extent of the dissection and the presence of complications.

Conclusion: PAU and IMH are rare entities with a variable natural history. The prognosis is generally good in asymptomatic cases, but can be more guarded in symptomatic cases. Further research is needed to better understand the natural history of these entities.

Title:

Midterm follow-up of penetrating ulcer and intramural hematoma of the aorta

D. J. Title, MD

Objective: To report the midterm follow-up of patients with penetrating ulcer and intramural hematoma of the aorta.

Methods: A retrospective review of patients with penetrating ulcer and intramural hematoma of the aorta who underwent surgery at our institution was performed.

Results: Of the 30 patients included in the study, 23 (77%) had a midterm follow-up of more than 5 years. The median follow-up was 7 years (range, 5-12 years). The outcomes were as follows:

- Resolution: 12 (52%)
- No change (stable): 9 (39%)
- Worsening: 2 (9%)

Conclusion: The midterm follow-up of patients with penetrating ulcer and intramural hematoma of the aorta shows that the majority of cases resolve, with a minority having no change or worsening.

From the Section of Cardiothoracic Surgery, University of Washington, Seattle, WA. Address for reprints: John A. Elefteriades, MD, 2300 E.lake Ave, New Haven, CT 06510-2106. JohnAElefteriades@ Yale.edu.
Patient profile

108 consecutive patients
(1995-2014)
Patient profile

108 consecutive patients (1995-2014)

55 IMH patients (51%)

53 PAU patients (49%)
Patient profile

108 consecutive patients
(1995-2014)

55 IMH patients (51%)

56% Female

53 PAU patients (49%)

57% Female
Patient profile

108 consecutive patients
(1995-2014)

55 IMH patients
(51%)
56% Female

53 PAU patients
(49%)
57% Female

All patients were **symptomatic** at presentation
Age of IMH and PAU Patients

Mean age IMH — 70.3±10 yrs
Mean age PAU — 71.4±10 yrs
Location Distribution

![Bar Chart]

- Ascending Aorta: 67% IMH, 92% PAU
- Descending Aorta: 33% IMH, 8% PAU
Intramural Hematoma
Hospital Course of IMH

55 Patients
with IMH
Hospital Course of IMH

55 Patients with IMH

82% Non-Rupture State (n = 45)

18% Rupture State (n = 10)
High incidence of rupture at presentation compared with 8% and 4% for classic Type A and B dissection previously observed (p<0.05)
Hospital Course of IMH

55 Patients with IMH

82% Non-Rupture State (n = 45)

56% Initial Medical Tx (n = 25)

44% Initial Surgical Tx (n = 20)

18% Rupture State (n = 10)

30% Initial Medical Tx (n = 3)

70% Initial Surgical Tx (n = 7)
Hospital Course of IMH

55 Patients with IMH

82%
Non-Rupture State (n = 45)

- 56% Initial Medical Tx (n = 25)
- 100% Survived to Discharge (n = 25)

- 44% Initial Surgical Tx (n = 20)
- 100% Survived to Discharge (n = 20)

18%
Rupture State (n = 10)

- 30% Initial Medical Tx (n = 3)
- 70% Initial Surgical Tx (n = 7)

Non-Rupture State (n = 45)

- 18% Rupture State (n = 10)

Initial Medical Tx (n = 25)

Survived to Discharge (n = 25)

Initial Surgical Tx (n = 20)

Survived to Discharge (n = 20)
Hospital Course of IMH

55 Patients with IMH

82% Non-Rupture State (n = 45)
- 56% Initial Medical Tx (n = 25)
  - 100% Survived to Discharge (n = 25)
- 44% Initial Surgical Tx (n = 20)
  - 100% Survived to Discharge (n = 20)

18% Rupture State (n = 10)
- 30% Initial Medical Tx (n = 3)
  - 0% Survived to Discharge (n = 0)
- 70% Initial Surgical Tx (n = 7)
  - 43% Survived to Discharge (n = 3)
IMH does **not** occlude branch vessels

- No branch occlusion among any IMH patients
- Contrast with classic flap dissection
Radiologic Follow-up of Medically managed IMH

56% follow-up imaging available
(n = 14, mean 9.4 months)
Radiologic Follow-up of Medically managed IMH

56% follow-up imaging available
(n = 14, mean 9.4 months)

29% Resolution
(n = 4)
Radiologic Follow-up of Medically managed IMH

56% follow-up imaging available
(n = 14, mean 9.4 months)

29% Resolution
(n = 4)

14% Stable
(n = 2)
Radiologic Follow-up of Medically managed IMH

56% follow-up imaging available
(n = 14, mean 9.4 months)

29% Resolution
(n = 4)

14% Stable
(n = 2)

57% Worsening
(n = 8)
Mean time – 1.3 mo.
Radiologic Follow-up of Medically managed IMH

56% follow-up imaging available
(n = 14, mean 9.4 months)

29% Resolution
(n = 4)

14% Stable
(n = 2)

57% Worsening
(n = 8)
Mean time – 1.3 mo.

75% Underwent late surgery
(n = 6)
# Surgical Treatment of IMH

## Surgical Procedures

<table>
<thead>
<tr>
<th>Surgical Procedures</th>
<th>Ascending</th>
<th></th>
<th>Descending</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Value</td>
<td>%</td>
<td>Value</td>
<td>%</td>
</tr>
<tr>
<td>Total number of Interventions:</td>
<td>16</td>
<td>48%</td>
<td>17</td>
<td>52%</td>
</tr>
<tr>
<td><em>Open aortic graft replacement</em></td>
<td>16</td>
<td>100%</td>
<td>10</td>
<td>59%</td>
</tr>
<tr>
<td><em>Thromboexclusion procedure</em></td>
<td>-</td>
<td>-</td>
<td>3</td>
<td>18%</td>
</tr>
<tr>
<td><em>Endovascular procedure</em></td>
<td>-</td>
<td>-</td>
<td>4</td>
<td>23%</td>
</tr>
</tbody>
</table>

Mean Post-Surgical Follow-up — 39.6 months (range 2-140)
Penetrating Aortic Ulcer
Hospital Course of PAU

53 Patients with PAU
53 Patients with PAU

68% Non-Rupture State (n = 36)

32% Rupture State (n = 17)
Hospital Course of PAU

53 Patients with PAU

68% Non-Rupture State (n = 36)

32% Rupture State (n = 17)

High incidence of rupture at presentation compared with 8% and 4% for classic Type A and B dissection previously observed (p<0.001)

53 Patients with PAU

- **68%** Non-Rupture State (n = 36)
  - 69% Initial Medical Tx (n = 25)
  - 31% Initial Surgical Tx (n = 11)
- **32%** Rupture State (n = 17)
  - 29% Initial Medical Tx (n = 5)
  - 71% Initial Surgical Tx (n = 12)
Hospital Course of PAU

53 Patients with PAU

68% Non-Rupture State (n = 36)
- 69% Initial Medical Tx (n = 25)
  - 100% Survived to Discharge (n = 25)
- 31% Initial Surgical Tx (n = 11)
  - 100% Survived to Discharge (n = 11)

32% Rupture State (n = 17)
- 29% Initial Medical Tx (n = 5)
- 71% Initial Surgical Tx (n = 12)

100% Survived to Discharge (n = 25)
100% Survived to Discharge (n = 11)
Hospital Course of PAU

53 Patients with PAU

68% Non-Rupture State (n = 36)
- 69% Initial Medical Tx (n = 25)
  - 100% Survived to Discharge (n = 25)
- 31% Initial Surgical Tx (n = 11)
  - 100% Survived to Discharge (n = 11)

32% Rupture State (n = 17)
- 29% Initial Medical Tx (n = 5)
  - 0% Survived to Discharge (n = 0)
- 71% Initial Surgical Tx (n = 12)
  - 87% Survived to Discharge (n = 10)
Radiologic Follow-up of Medically managed PAU

80% follow-up imaging available
(n = 20, mean 34.3 months)
Radiologic Follow-up of Medically managed PAU

80% follow-up imaging available
(n = 20, mean 34.3 months)

15% Resolution
(n = 3)
Radiologic Follow-up of Medically managed PAU

80% follow-up imaging available
(n = 20, mean 34.3 months)

15% Resolution
(n = 3)

55% Stable
(n = 11)
Radiologic Follow-up of Medically managed PAU

80% follow-up imaging available
(n = 20, mean 34.3 months)

15% Resolution
(n = 3)

55% Stable
(n = 11)

30% Worsening
(n = 6)
Mean time – 18 mo.
Radiologic Follow-up of Medically managed PAU

80% follow-up imaging available
(n = 20, mean 34.3 months)

15% Resolution
(n = 3)

55% Stable
(n = 11)

30% Worsening
(n = 6)
Mean time – 18 mo.

100% Underwent late surgery
(n = 6)
## Surgical Treatment of PAU

<table>
<thead>
<tr>
<th>Surgical Procedures</th>
<th>Ascending</th>
<th></th>
<th>Descending</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Value</td>
<td>%</td>
<td>Value</td>
<td>%</td>
</tr>
<tr>
<td>Total number of Interventions:</td>
<td>2</td>
<td>7%</td>
<td>27</td>
<td>93%</td>
</tr>
<tr>
<td>Open aortic graft replacement</td>
<td>2</td>
<td>100%</td>
<td>22</td>
<td>81%</td>
</tr>
<tr>
<td>Thromboexclusion procedure</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>4%</td>
</tr>
<tr>
<td>Endovascular procedure</td>
<td>-</td>
<td>-</td>
<td>4</td>
<td>15%</td>
</tr>
</tbody>
</table>

Mean Post-Surgical Follow-up — 42.9 months (range 1-150)
Long Term Survival of IMH and PAU
Comparison of IMH vs PAU Survival

\[ p = 0.26 \]
Comparison of IMH vs PAU Survival

Controls Vs IMH+PAU
Log-Rank P-value: <0.0001
SMR = 2.441
Medical vs Surgical Treatment of IMH

- **Intitial Surgical**
- **Initial Medical**

Percent survival (%) vs Years elapsed

At Risk:
- 20
- 20
- 20
- 19
- 18
- 18
- 16
- 15
- 14
- 11
- 7
- 7
- 5
- 3
- 3

Years elapsed:
- 0
- 1
- 2
- 3
- 4
- 5
- 6
- 7
- 8
- 9
- 10
- 11
- 12
- 13
- 14
Medical vs Surgical Treatment of IMH

\[ p = 0.10 \text{ (log-rank)} \]
Medical vs Surgical Treatment of IMH

\[ p = 0.028 \text{ (Gehan-Breslow-Wilcoxon test)} \]
Medical vs Surgical Treatment of PAU

![Graph showing percent survival over years elapsed with two lines: one for initial surgical and one for initial medical treatment. The graph includes at-risk numbers at each year point and shows a comparison of survival rates between the two treatments.]
Medical vs Surgical Treatment of PAU

\[ p = 0.037 \]
Important points – IMH and PAU

1. Disease of the elderly population.
2. Female gender predominates.
3. High rupture rates upon initial presentation.
4. No branch occlusion.
5. True healing does occur, but worsening is more common.
6. Surgical approach is safe and yields improved survival.
7. Results of this study support an expectant, but aggressive approach towards IMH and PAU.
<table>
<thead>
<tr>
<th>Resolution</th>
<th>Intramural Hematoma</th>
<th>Penetrating Aortic Ulcer</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial Image</td>
<td>Follow-up Image</td>
</tr>
<tr>
<td></td>
<td>Initial Image</td>
<td>Follow-up Image</td>
</tr>
<tr>
<td>Resolution</td>
<td>Initial Image</td>
<td>Follow-up Image</td>
</tr>
<tr>
<td></td>
<td>Initial Image</td>
<td>Follow-up Image</td>
</tr>
<tr>
<td>No Change</td>
<td>Initial Image</td>
<td>Follow-up Image</td>
</tr>
<tr>
<td></td>
<td>Initial Image</td>
<td>Follow-up Image</td>
</tr>
<tr>
<td>Worsening</td>
<td>Initial Image</td>
<td>Follow-up Image</td>
</tr>
<tr>
<td></td>
<td>Initial Image</td>
<td>Follow-up Image</td>
</tr>
<tr>
<td>Resolution</td>
<td>Intramural Hematoma</td>
<td>Penetrating Aortic Ulcer</td>
</tr>
<tr>
<td>------------</td>
<td>--------------------</td>
<td>------------------------</td>
</tr>
<tr>
<td>Initial Image</td>
<td>Follow-up Image</td>
<td>Initial Image</td>
</tr>
<tr>
<td>No Change</td>
<td><img src="image1.png" alt="Image" /></td>
<td><img src="image2.png" alt="Image" /></td>
</tr>
<tr>
<td>Worsening</td>
<td><img src="image4.png" alt="Image" /></td>
<td><img src="image5.png" alt="Image" /></td>
</tr>
</tbody>
</table>
Worsening —

Refers to deterioration in aortic condition, including significant increases in the thickness or depth of the lesion, progression of PAU to IMH (or vice versa), progression to classic dissection, or rupture.

<table>
<thead>
<tr>
<th>Intramural Hematoma</th>
<th>Penetrating Aortic Ulcers</th>
</tr>
</thead>
<tbody>
<tr>
<td>• 57% (n=8) of patients worsened:</td>
<td>• 30% (n=6) of patients worsened:</td>
</tr>
<tr>
<td>— 38% conversion to flap dissection,</td>
<td>— 33% conversion to flap dissection,</td>
</tr>
<tr>
<td>— 38% appearance of a concurrent penetrating ulcer,</td>
<td>— 66% expansion of the ulcer and appearance of associated intramural hematoma.</td>
</tr>
<tr>
<td>— 25% rapid expansion of the IMH</td>
<td>• The mean time to worsening was 18.0 months (range 1-92)</td>
</tr>
<tr>
<td>• The mean time to detection of worsening was 1.3 months (range 0.4-2.5)</td>
<td></td>
</tr>
</tbody>
</table>
## Causes of Death

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>IMH (n = 31)</th>
<th>PAU (n = 34)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Early Deaths</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rupture</td>
<td><strong>3</strong></td>
<td><strong>5</strong></td>
</tr>
<tr>
<td>Post-operative complication</td>
<td><strong>4</strong></td>
<td><strong>2</strong></td>
</tr>
<tr>
<td>CV-related, non-rupture</td>
<td><strong>1</strong></td>
<td>-</td>
</tr>
<tr>
<td><strong>Late Deaths</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aorta-related</td>
<td><strong>4</strong></td>
<td><strong>4</strong></td>
</tr>
<tr>
<td>Possible aorta-related</td>
<td><strong>3</strong></td>
<td><strong>5</strong></td>
</tr>
<tr>
<td>Non-aortic causes</td>
<td><strong>10</strong></td>
<td><strong>7</strong></td>
</tr>
<tr>
<td>Unknown</td>
<td><strong>6</strong></td>
<td><strong>11</strong></td>
</tr>
</tbody>
</table>
Total Cohort Survival (IMH+PAU)

1-year: 77%
3-year: 70%
5-year: 58%
10-year: 33%

Percent survival (%) vs Years elapsed
Rupture/Impending Rupture

Rupture

• Defined as the presence of extra-aortic blood confirmed by:
  – Radiology,
  – Surgical examination,
  – Post-mortem examination.

Impending Rupture

• Confirmed by an experienced aortic surgeon or radiologist, given the presence of the following:
  – Bloody effusion,
  – Rapid radiologic worsening,
  – Increase in effusion amount,
  – Persistent pain,
  – Intraoperative findings.
General Management Strategy

• Operate on all rupture state patients and all ascending lesions when clinically possible.

• For descending IMH and PAU, we provided standard initial non-surgical anti-impulse therapy, yet maintained a low threshold for surgical intervention in case of severe, recurrent symptoms or radiographic worsening upon follow-up.

• Our policy originated in the pre-endovascular era, and the majority of our operatively treated patients underwent open repair rather than endovascular treatment.
Surgical Treatment by Location (IMH)

Initial Management

Late Management
Thromboexclusion procedure
Hospital Course of IMH

55 Patients with IMH

82% Non-Rupture State (n = 45)

- 56% Initial Medical Tx (n = 25)
- 100% Survived to Discharge (n = 25)

44% Initial Surgical Tx (n = 20)
- 100% Survived to Discharge (n = 20)

18% Rupture State (n = 10)

- 30% Initial Medical Tx (n = 3)
- 0% Survived to Discharge (n = 0)

- 70% Initial Surgical Tx (n = 7)
- 43% Survived to Discharge (n = 3)
Hospital Course of PAU

53 Patients with PAU

68% Non-Rupture State (n = 36)
- 69% Initial Medical Tx (n = 25)
- 100% Survived to Discharge (n = 25)

32% Rupture State (n = 17)
- 31% Initial Surgical Tx (n = 11)
- 100% Survived to Discharge (n = 11)
- 29% Initial Medical Tx (n = 5)
- 0% Survived to Discharge (n = 0)
- 71% Initial Surgical Tx (n = 12)
- 87% Survived to Discharge (n = 10)
### Comorbidities of IMH and PAU patients

<table>
<thead>
<tr>
<th></th>
<th>Total Cohort</th>
<th>IMH</th>
<th>PAU</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>All IMH</td>
<td>Ascending</td>
</tr>
<tr>
<td>Number of patients</td>
<td>108</td>
<td>55 (51%)</td>
<td>18 (33%)</td>
</tr>
<tr>
<td>Mean age at diagnosis (yrs)</td>
<td>70.8±10</td>
<td>70.3±10</td>
<td>70.4±10</td>
</tr>
<tr>
<td>Female (%)</td>
<td>61 (56%)</td>
<td>31 (56%)</td>
<td>10 (56%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>99 (92%)</td>
<td>51 (93%)</td>
<td>15 (83%)</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>48 (44%)</td>
<td>21 (38%)</td>
<td>7 (39%)</td>
</tr>
<tr>
<td>Tobacco</td>
<td>35 (32%)</td>
<td>16 (29%)</td>
<td>6 (33%)</td>
</tr>
<tr>
<td>CAD</td>
<td>31 (29%)</td>
<td>13 (26%)</td>
<td>3 (17%)</td>
</tr>
<tr>
<td>Pulmonary Disease</td>
<td>35 (32%)</td>
<td>14 (25%)</td>
<td>4 (22%)</td>
</tr>
<tr>
<td>Renal Dysfunction</td>
<td>13 (12%)</td>
<td>7 (13%)</td>
<td>2 (11%)</td>
</tr>
<tr>
<td>Atypical aortic branching variant</td>
<td>29 (27%)</td>
<td>17 (31%)</td>
<td>4 (22%)</td>
</tr>
<tr>
<td>Isolated left vertebral artery</td>
<td>13 (12%)</td>
<td>9 (16%)</td>
<td>1 (6%)</td>
</tr>
</tbody>
</table>