How To Approach Aortic PAU and IMH

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Disclosures

• none
Natural history and outcome of patients with intramural hematomas and penetrating aortic ulcers

Albeir Y Mousa, Joseph Bozzay and Ali F AbuRahma

Abstract
All pathologies of acute aortic syndromes should be precisely diagnosed for prompt therapy. Intramural hematomas, as well as penetrating ulcers can be encountered in these patients. Presentations, clinical scenarios, and proper management are outlined in this review, which sums up available current literature to provide the vascular specialist with an adequate understanding of these unique syndromes.

A PAU is a disruption in the intima with invasion of the media that can occur in arterial territory and appears as an outpouching (crater), but with no obvious dissection. On the other hand, IMH is a localized thickening of the aortic wall secondary to rupture vasa vasorum and localized bleeding within the media, without obvious intimal disruption that can result from a PAU, but not vice versa. Pathology details were out-
Acute Aortic Syndromes

AD: Aortic Dissection
IMH: Intramural Hematoma
PAU: Penetrating Atherosclerotic Ulcer

Aneurysm
The natural history and outcomes for thoracic and abdominal penetrating aortic ulcers

Shaun M. Gifford, MD, Audra A. Duncan, MD, Lawrence E. Greiten, MD, Peter Gloviczki, MD, Gustavo S. Oderich, MD, Manju Kalra, MRBS, Mark D. Fleming, MD, and Thomas C. Bower, MD, Rochester, Minn

Objective: The objective of this report was to define the natural history of penetrating aortic ulcers (PAUs) in the descending thoracic and abdominal aorta.

Methods: Data from consecutive patients with PAU from January 1, 1998 to December 31, 2012 were retrospectively reviewed. Computed tomography (CT) scans were analyzed for anatomic changes. End points analyzed were changes in size, development of symptoms or signs of rupture, morbidity, and mortality.

Results: Ninety-three patients were identified; 57 were followed up with two or more CT studies 3 months apart (group 1), and 20 had immediate repair (group 2). Sixteen had one CT scan and no intervention or follow-up and were excluded from analysis. In group 1, mean age was 78 years (29 men, 28 women), with 28 descending thoracic aorta and 29 abdominal aorta PAUs. Fifty patients were asymptomatic, whereas five had pain and two had emboli. Mean follow-up was 38 months (range, 3-108 months). Ulcer growth rate was as follows: length, 2.0 mm/y; depth, 1.2 mm/y, and aortic diameter, 2.2 mm/y. Thirteen (25%) went on to repair at a mean of 37 months after diagnosis because of size (54%; 7/13), rapid growth (31%; 4/13), and high-risk morphology (15%; 2/13). During surveillance, 11 patients died, 10 of unrelated causes, and 1 of rupture after refusing repair. All repairs in group 1 were endovascular. The 30-day surgical mortality was 0%. One patient had an access site complication requiring bypass after descending thoracic aorta PAU repair. At a mean follow-up of 32 months, all ulcers were excluded on CT; one (8%) had a type II endoleak. Group 2 included 13 men and seven women with a mean age of 70 years, with 12 descending thoracic and eight abdominal aorta PAUs. Repair indications were rupture (n = 3), symptoms (n = 10), or size (n = 7) and included one open and 19 endovascular repairs with 0% 30-day mortality. Major complications (3/20; 15%) included myocardial infarction, site disruption, and hematoma; four of 20 patients had type II endoleaks.

Conclusions: PAU growth rate and risk of rupture are low. Endovascular repair of symptomatic, ruptured, and large PAUs is safe and effective with excellent long-term results. For asymptomatic PAUs, serial CT surveillance is associated with a low rate of rupture or complications. J Vasc Surg 2016;63(1):118-28

Fig 3. Consolidated Standards of Reporting Trials (CONSORT) diagram demonstrating penetrating ulcer population in the study. CT, Computed tomography.
Diagnosis

• Clinical Symptoms
  – pain

• Imaging
  – TEE
  – ?MRA
  – CTA-gold standard
INDICATIONS FOR INTERVENTION
Presentation, complications, and natural history of penetrating atherosclerotic ulcer disease

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Objectives: Increased utilization of computed tomography angiography (CTA) has increased the radiologic diagnosis of penetrating atherosclerotic ulcers (PAUs), which are defined as the ulceration of atherosclerotic plaque through the internal elastic lamina into the aortic media. However, the presentation, treatment indications, and natural history of this disease process remain unclear.

Methods: The radiology database at a single university hospital was searched prospectively for the CTA diagnosis of PAU from January 2008 to June 2009. All scans were interpreted by a cardiovascular radiologist. Information on PAU characteristics and need for surgical repair due to PAU disease was collected. PAU stability or progression was assessed by follow-up CTA, if available. Only PAUs in the aortic arch, descending thoracic aorta, and abdominal aorta were included.

Results: Three hundred eighty-eight PAUs were diagnosed by CTA interpretation. PAU location was in the aortic arch in 27 (6.8%) cases, the descending thoracic aorta in 243 (61.3%) cases, and the abdominal aorta in 138 (29.7%) cases. Two hundred twenty-four (57.7%) PAUs were isolated (without saccular aneurysm or intramural hematoma); 102 (27.8%) PAUs had associated saccular aneurysms and 56 (14.4%) PAUs had associated intramural hematoma. Rupture was present in 16 (4.1%) cases. Fifty (12.9%) PAUs underwent repair with thoracic endovascular aortic repair (TEVAR) (n = 30), endovascular aneurysm repair (EVAR) (n = 10), or open surgery (n = 10); primary indications for repair were saccular aneurysm (n = 26), rupture (n = 16), and persistent or recurrent symptoms (n = 8). Even if initially treated conservatively with resolution of pain, symptomatic PAU disease was more likely to progress than asymptomatic PAU disease (36.2% vs 7.8%, P < .001). Follow-up CTA was available for 87 PAUs, 20 (23.0%) of which demonstrated radiographic disease progression as a mean follow-up of 8.4 ± 10.3 months. Symptomatic PAU disease was more likely to progress than asymptomatic disease (42.9% vs 16.7%, P = .029).

Conclusions: For PAUs diagnosed on CTA at a single institution, 4.1% were ruptured and 12.9% underwent repair. Close follow-up imaging appears to be indicated for PAUs, particularly in the case of symptomatic disease, which is more likely to require repair and to undergo radiographic progression. (J Vasc Surg 2012;66:108-119.)

Table I. Characteristics of patients with penetrating atherosclerotic ulcer disease

<table>
<thead>
<tr>
<th></th>
<th>All PAU disease (n = 388)</th>
<th>Isolated PAU disease (n = 224)</th>
<th>PAUs with intramural hematoma (n = 56)</th>
<th>PAUs with saccular aneurysm (n = 198)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>73.2 ± 8.9</td>
<td>73.5 ± 8.4</td>
<td>70.5 ± 10.6</td>
<td>73.8 ± 8.7</td>
<td>.047</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>234 (60.2%)</td>
<td>148 (65.8%)</td>
<td>29 (51.8%)</td>
<td>71 (65.7%)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>283 (72.8%)</td>
<td>150 (66.9%)</td>
<td>51 (90.9%)</td>
<td>82 (80.4%)</td>
<td>.034</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>161 (44.5%)</td>
<td>86 (42.4%)</td>
<td>25 (43.0%)</td>
<td>50 (49.0%)</td>
<td>.541</td>
</tr>
<tr>
<td>CAD</td>
<td>153 (42.3%)</td>
<td>87 (42.9%)</td>
<td>18 (31.6%)</td>
<td>48 (47.1%)</td>
<td>.161</td>
</tr>
<tr>
<td>Symptomatic</td>
<td>69 (17.9%)</td>
<td>10 (4.5%)</td>
<td>45 (80.4%)</td>
<td>14 (13.1%)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

CAD, Coronary artery disease; PAU, penetrating atherosclerotic ulcer.

Table III. Predictors of disease progression of penetrating atherosclerotic ulcers by serial computed tomography angiography

<table>
<thead>
<tr>
<th></th>
<th>PAUs that progressed (n = 20)</th>
<th>PAUs that did not progress (n = 67)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>71.7 ± 6.7</td>
<td>71.2 ± 8.8</td>
<td>.828</td>
</tr>
<tr>
<td>Thoracic aortic location</td>
<td>12 (60.0%)</td>
<td>42 (62.7%)</td>
<td>.510</td>
</tr>
<tr>
<td>Total aortic diameter (mm)</td>
<td>32.8 ± 12.0</td>
<td>31.3 ± 7.1</td>
<td>.599</td>
</tr>
<tr>
<td>PAU neck (mm) a</td>
<td>12.3 ± 7.0</td>
<td>12.9 ± 6.5</td>
<td>.775</td>
</tr>
<tr>
<td>PAU depth (mm) a</td>
<td>5.9 ± 3.3</td>
<td>7.3 ± 4.3</td>
<td>.230</td>
</tr>
<tr>
<td>Through intimal calcification</td>
<td>12 (60.0%)</td>
<td>28 (41.8%)</td>
<td>.239</td>
</tr>
<tr>
<td>Male gender</td>
<td>11 (55.0%)</td>
<td>37 (55.2%)</td>
<td>.999</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>7 (35.0%)</td>
<td>19 (30.6%)</td>
<td>.930</td>
</tr>
<tr>
<td>Hypertension</td>
<td>17 (85.0%)</td>
<td>45 (72.6%)</td>
<td>.409</td>
</tr>
</tbody>
</table>

Symptomatic                    | 9 (45.0%)                     | 12 (17.9%)                          | .029    |

*Only assessed for isolated penetrating atherosclerotic ulcers (PAUs) and PAUs with intramural hematoma.
Thoracic Endovascular Repair of Complicated Penetrating Aortic Ulcer: An 11-Year Single-Center Experience

Rolf Alexander Järosi, MD, Riccardo Gorla, MD, Konstantinos Tsagakis, MD, Philipp Kahler, MD, Michael Horacek, MD, Florian Bruckschen, MD, Daniel-Sebastian Dohle, MD, Heinz Jakob, MD, Thomas Schlosser, MD, Holger Eggebrecht, MD, Eduardo Bossone, MD, and Raimund Erbel, MD

Abstract
Purpose: To analyze an 11-year single-center experience of treating complicated penetrating aortic ulcer (PAU) using thoracic endovascular aortic repair (TEVAR). Methods: This study included 63 consecutive patients (mean age 69 ± 11.5 years; 40 men) with complicated PAU (42 symptomatic, 22 with rupture) who underwent TEVAR between 2002 and 2013. The PAUs were located in the aortic arch (n=11), the descending thoracic aorta (n=45), and the thoracoabdominal aorta (n=9). Results: TEVAR was performed within 14 days of diagnosis in 33 (52.3%) cases (9 ruptures treated immediately); the other 30 (47.7%) patients had an average interval between diagnosis and intervention of 40.2±9 days. Technical success was 98.4% (62/63). One patient had a type I endoleak after stent-graft repair of a PAU in the aortic arch without great vessel transposition; another procedure was required for carotid-subclavian bypass and proximal stent-graft extension. No patient experienced spinal cord ischemia after TEVAR. Five (7.9%) patients died in-hospital; 3 had severe cardiac complications, 1 died from complications of aortic rupture, and the other succumbed to septic shock. Mean follow-up was 45.6±47.2 months, during which 12 (19.0%) patients needed a secondary intervention because of late endoleaks (n=4, 6.3%) or new complications due to disease progression. Multivariate analysis indicated that a PAU depth >15 mm was an independent predictor of mortality (hazard ratio 6.92, p=0.03). In the biomarker analysis, symptomatic patients had significantly higher D-dimer and troponin levels compared to asymptomatic patients ([59.5±460.7 vs 283.2±85.2 pg/L (p=0.016) and 0.22±0.61 vs 0.022±0.03 ng/mL (p=0.04), respectively]. Conclusion: Patients with PAU suffer from underlying severe atherosclerotic disease and have a significant number of cardiovascular comorbidities that lead to relevant mortality and morbidity after TEVAR. As a PAU diameter >15 mm represented high risk for disease progression, these patients may be candidates for early intervention. D-dimer levels may help identify patients at risk and with progression of PAU.

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Knowledge and Compassion Focused on You
PAU and Effusion
Intramural Hematoma and Penetrating Ulcers: Indications to Endovascular Treatment

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KEYWORDS
Penetrating aortic ulcer; Intramural hematoma; TEVAR; Stent-graft; Acute aortic syndrome

Abstract Intramural hematoma (IMH) of the aorta and penetrating aortic ulcer (PAU) are important variants forms of classic double-barrel aortic dissection in patients presenting with acute aortic syndrome. Recent insights provided by modern high-resolution imaging are currently challenging previous pathophysiologic concepts underlying IMH and PAU, suggesting a close relationship of both entities. Thoracic endovascular aortic repair (TEVAR) offers a less invasive approach to the treatment of affected patients with very encouraging early to midterm results. This review discusses current indication for TEVAR in IMH and PAU patients in the view of an improved understanding of these diseases.

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So far, the natural history of IMH is not fully understood. Published data are somewhat conflicting with some reporting a rather favorable outcome of IMH as compared to classic dissection, whereas others have reported similar mortality and complication rates to double-barrel dissection. Complications of IMH are quite common and include progression towards overt false-lumen dissection in 28–47% of cases, early aneurysm formation or (contained) rupture in 20–45% of patients. Patients may, however, show spontaneous reabsorption of IMH under medical treatment, although regression is less common. Predictors of progression include recurrent or persisting pain, and presence of penetrating aortic ulcer, while younger age, aortic diameter <4.0–4.5 cm, and hematoma thickness <1.0 cm portend a better prognosis. Dissection, involvement of the ascending aorta (type A-IMH) demands urgent surgical repair in most of the patients due to the risk of rupture or progression to frank dissection. It
Clinical importance of minimal enhancement of type B intramural hematoma of the aorta on computed tomography imaging

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ABSTRACT

Objective: To investigate the instability, morphology, natural course, and prognostic value of enhancement of the thrombosed false lumen on contrast-enhanced computed tomography (CT) scans in patients with type B intramural hematoma of the aorta (IMH).

Methods: A total of 65 patients (42 men, mean age, 75 years) with type B IMH were evaluated retrospectively. On initial CT scans, attenuation of the false lumen (AFL) was determined before enhancement, and in the early and delayed phases of contrast enhancement. Then enhancement of the false lumen (EFL) was calculated (AFL in the delayed image - AFL in the precontrast image). The Cox proportional hazards model was employed to estimate the including death or surgical repair.

Results: The mean AFL for precontrast CT, arterial phase enhanced CT, and delayed phase enhanced CT was 59.8 ± 108, and 63.7 ± 111 Hounsfield units, respectively, whereas the mean EFL was 7.4 was the only independent predictor of IMH related events (n = 23, hazard ratio, 1.008; 95% confidence interval 1.004 to 1.012). IMH-related death/surgical repair (n = 10, hazard ratio, 1.11; 95% confidence interval 1.00 to 1.24).

Conclusions: In patients with IMH, EFL is the most powerful predictor of IMH-related events or surgical repair.

Fig 5. Multidetector computed tomography (MDCT) images of a 74-year-old man with type B intramural hematoma of the aorta (IMH). The attenuation of the false lumen (AFL) for plain computed tomography (CT) scan, arterial phase contrast-enhanced CT, and delayed phase contrast-enhanced CT was 55, 59, and 65 HU, respectively. The enhancement of the false lumen (EFL) is 13 HU. A, At the onset, arterial phase contrast-enhanced coronal CT scan shows an intimal defect (arrow) with a depth of 1 mm. B, One month later, contrast-enhanced coronal CT scan reveals that the intimal defect has progressed to an ulcer-like projection (ULP, arrow) with a depth of 7 mm. C, Three months later, contrast-enhanced coronal CT scan demonstrates progressive enlargement of the ULP (arrow). After that, stent-graft placement was performed. D, After stent-graft placement, contrast-enhanced coronal CT scan shows disappearance of the ULP.
Rupture upon Presentation
Delayed Ruture-3days
Management

Medical
- Pain control
- Anti-Hypertensives
- Surveillance Imaging

Intervention
- TEVAR
  - Except ascending/arch involvement
- EVAR
**Intramural Hematoma and Penetrating Ulcers: Indications to Endovascular Treatment**

H. Eggebrecht*, B. Plicht, P. Kahlert, R. Erbel

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Submitted 4 September 2009; accepted 4 September 2009

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**Keywords**
Intramural hematoma; Intermural hematoma; TEVAR; Stent-graft; Acute aortic syndrome

**Abstract**
Intramural hematoma (IMH) of the aorta and penetrating aortic ulcer (PAU) are important variant forms of classic double-barrel aortic dissection in patients presenting with acute aortic syndrome. No clear imaging criteria exist for aortic dissection; therefore, clinical correlation is mandatory.

**Table 1. Overview of other published studies on TEVAR in PAU patients.**

<table>
<thead>
<tr>
<th>Authors</th>
<th>n</th>
<th>Technical success</th>
<th>Complete sealing of PAU</th>
<th>Neurologic complications</th>
<th>In-hospital mortality</th>
<th>Additional endovascular procedures required</th>
<th>Aorta-related mortality during follow-up</th>
<th>Mean duration of follow-up (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dake et al. 1994</td>
<td>5</td>
<td>5/5 (100%)</td>
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<td>0</td>
<td>0</td>
<td>0</td>
<td>11.6</td>
</tr>
<tr>
<td>Murgu et al. 1998</td>
<td>4</td>
<td>4/4 (100%)</td>
<td>3/4 (75%)</td>
<td>1 (25%)</td>
<td>1 (25%)</td>
<td>0</td>
<td>0</td>
<td>7.7</td>
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<tr>
<td>Britenden et al. 1999</td>
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<td>2/2 (100%)</td>
<td>2/2 (100%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>Manley et al. 2000</td>
<td>1</td>
<td>1/1 (100%)</td>
<td>1/1 (100%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Not specified</td>
</tr>
<tr>
<td>Salter et al. 2001</td>
<td>4</td>
<td>4/4 (100%)</td>
<td>4/4 (100%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>8.5</td>
</tr>
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<td>Haulon et al. 2002</td>
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<td>2/2 (100%)</td>
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<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>Pittet et al. 2002</td>
<td>1</td>
<td>1/1 (100%)</td>
<td>1/1 (100%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>Schöder et al. 2002</td>
<td>8</td>
<td>8/8 (100%)</td>
<td>8/8 (100%)</td>
<td>1 (13%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>14.1</td>
</tr>
<tr>
<td>Kos et al. 2002</td>
<td>10</td>
<td>10/10 (100%)</td>
<td>9/10 (90%)</td>
<td>1 (10%)</td>
<td>0</td>
<td>0</td>
<td>1 (12.5%)</td>
<td>9</td>
</tr>
<tr>
<td>Faries et al. 2002</td>
<td>1</td>
<td>1/1 (100%)</td>
<td>1/1 (100%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>18</td>
</tr>
<tr>
<td>Ganaha et al. 2002</td>
<td>6</td>
<td>6/6 (100%)</td>
<td>6/6 (100%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
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</tr>
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<td>Eggebrecht et al. 2003</td>
<td>10</td>
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<td>9/10 (90%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>24.4</td>
</tr>
<tr>
<td>Crane et al. 2003</td>
<td>1</td>
<td>1/1 (100%)</td>
<td>1/1 (100%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>12</td>
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<tr>
<td>Demers et al. 2004</td>
<td>26</td>
<td>26/27 (92%)</td>
<td>24/26 (92%)</td>
<td>2 (8%)</td>
<td>3 (12%)</td>
<td>1 (4%)</td>
<td>1 (4%)</td>
<td>Not specified</td>
</tr>
<tr>
<td>Eggebrecht et al. 2005</td>
<td>22</td>
<td>22/22 (96%)</td>
<td>21/21 (96%)</td>
<td>1 (5%)</td>
<td>1 (5%)</td>
<td>0</td>
<td>0</td>
<td>Not specified</td>
</tr>
<tr>
<td>Brinster et al. 2006</td>
<td>21</td>
<td>21/21 (100%)</td>
<td>21/21 (100%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>27</td>
</tr>
<tr>
<td>Dalalinas et al. 2007</td>
<td>18</td>
<td>18/18 (100%)</td>
<td>18/18 (100%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>14.4</td>
</tr>
<tr>
<td>Botta et al. 2008</td>
<td>19</td>
<td>19/19 (100%)</td>
<td>19/19 (100%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Not specified</td>
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<tr>
<td>Geißbüs et al. 2008</td>
<td>48</td>
<td>45/48 (94%)</td>
<td>Not specified</td>
<td>2 (4%)</td>
<td>7 (15%)</td>
<td>4 (9%)</td>
<td>3 (2%)</td>
<td>14.3</td>
</tr>
</tbody>
</table>

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**Total** 209 204/209 (98%) 129/135 (96%) 8 (4%) 15 (7%) 9 (5%) 3 (2%)
Thoracic Endovascular Aortic Repair for Penetrating Aortic Ulcer: Literature Review

Background. The aim of the study was to provide a literature review of thoracic endovascular aortic repair (TEVAR) outcomes for penetrating ulcer of the aorta.

Methods. Relevant articles in the Embase, Medline, and Cachete databases reporting the results of endovascular repair for penetrating ulcers of the thoracic aorta were systematically searched and reviewed.

Results. Thirty-one articles were identified after a literature review, and 310 patients treated by TEVAR for penetrating ulcers of the aorta were identified. In this cohort, most patients were male (68.4%), had a history of smoking (60.4%), and systemic hypertension (60%). Only 9% were asymptomatic at initial presentation. Most cases (95%) occurred among patients with a single ulcer, located in the descending thoracic aorta (53%), with associated intramural hematoma in 45%. The technical success of TEVAR was 98.3%. Surgical conversion during the postoperative period with stent-graft explantation was required in 1 patient. The overall 30-day mortality was 4.8% (13 of 310). The most frequent complications were endoleaks 0%, 25 of 310 and access problems (16.1%, 26 of 161). After a mean follow-up of 17.7 months (range, 1 to 32), the all-cause mortality was 23.9% (71 of 301), and the aortic-related mortality was 4.3% (13 of 310). During follow-up, new endoleak and ulcer recurrence were observed in 4.4% (n = 13 of 273) and 4.5% (n = 5 of 110), respectively, requiring a new aortic endovascular procedure in 50% (n = 159).

Conclusions. Thoracic endovascular aortic repair of penetrating ulcer has excellent short-term and midterm results. The endovascular approach should be the first-line management for aortic ulcer when intervention is indicated.

Since the first use of TEVAR for PAU in 1998 [9], our literature review identified 310 patients with PAU treated by TEVAR, when patient-level data were available. The endovascular approach had favorable outcomes: 30-day mortality of 4.8%, and 1-year survival rate of 91.1%. This minimally invasive technique is associated with a high rate of technical success (98.3%). Focusing on serious

Midterm outcomes (mean 17.8 months) with an overall mortality of 22.9% and an aortic-related mortality of 4.1% are a reflection of both the effectiveness of the technique and also the high comorbidity status of this cohort of patients. That is also clear from the 67.3% 5-year survival. Czerny and associates [26] confirmed that most of the deaths in their study were cardiovascular and cancer related. The midterm efficacy of TEVAR in the context of PAU is also evident from the low rate of endoleak (5.4%) and ulcer recurrences (4.5%). Only half of these patients actually required a new aortic endovascular procedure. Hence, TEVAR can be considered as an effective midterm treatment for 95.4% of patients.
Aortic remodeling after thoracic endovascular aortic repair for intramural hematoma

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Background: Intramural hematoma (IMH), penetrating atherosclerotic ulcer (PAU), and aortic dissection comprise a spectrum of acute aortic pathologies. Although thoracic endovascular aortic repair (TEVAR) has increasingly been applied to aortic dissection, there is a paucity of data on the anatomic effect of TEVAR for IMH. Our goal was to investigate the extent of aortic remodeling after TEVAR.

Methods: A retrospective chart review from 2006 to 2012 was conducted on patients who underwent TEVAR for IMH. Data were collected from the electronic medical record. Radiology images were reviewed and primary data points included diameter (TLD) and volume measurements for aortic true lumen and total aortic diameter (TAD) and volume at the site of maximal pathology. Aortic remodeling was evidenced by a TAD/TLD ratio closest to 1.0. Patients with no imaging beyond 30 days postoperatively were excluded.

Results: During the 6-year period, 44 patients underwent TEVAR for IMH. Twenty-five patients had an IMH with concomitant PAU. There were 25 (58%) female patients. Mean age was 71 ± 11 years, and 40 (91%) patients had hypertension. Operative indications included intratable pain in 31 (70%), rapidly progressing IMH or conversion to dissection in 13 (30%), rupture in 10 (23%), and uncontrolled hypertension in 6 (14%). Technically successful TEVAR was performed in all patients with 42 (95%) reporting complete relief of symptoms. The 30-day mortality rate was 9% with a 5% rate of permanent paraplegia or paraparesis. At a mean follow-up of 26 months, there were no additional aortic-related deaths and overall survival was 80% with a reintervention rate of 11%. For our imaging analysis, 10 patients were excluded because of lack of follow-up imaging beyond 30 days. At a mean follow-up of 13 months, all measured data points were statistically improved from before to after TEVAR: thickness of IMH (12 mm vs 4 mm; P = .01), mean TLD (35 mm vs 37 mm; P = .04), mean TAD (47 mm vs 42 mm; P = .02), TAD/TLD ratio (1.35 vs 1.14; P < .01), and IMH volume (103 cm³ vs 14 cm³; P < .01). The mean Δ in TAD/TLD ratio from before to after TEVAR for the reintervention group was 0(29) (P = .65). Analysis of patients with isolated IMH and those with concomitant PAU revealed no statistical differences.

Conclusions: TEVAR is safe and effective in treating IMH and based on longitudinal computed tomography scan analysis, aortic remodeling is evidenced by normalization of all measured indices. (J Vasc Surg 2014;60:929–36.)

Fig. 1. a, Close-up imaging demonstrating an aortic intramural hematoma (IMH). b, Postoperative findings 11 months after thoracic endovascular aortic repair (TEVAR) with near resolution of IMH.

Table III. Pre- and postoperative aortic diameters and volume measurements.

<table>
<thead>
<tr>
<th></th>
<th>Preoperative</th>
<th>Postoperative</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>TAD, mm</td>
<td>47 ± 12</td>
<td>42 ± 10</td>
<td>.03</td>
</tr>
<tr>
<td>TLD, mm</td>
<td>36 ± 9</td>
<td>37 ± 9</td>
<td>.04</td>
</tr>
<tr>
<td>IMH thickness, mm</td>
<td>12 ± 3</td>
<td>4 ± 2</td>
<td>.01</td>
</tr>
<tr>
<td>TAD/TLD ratio</td>
<td>1.35 ± 0.14</td>
<td>1.13 ± 0.14</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Volume of IMH, cm³</td>
<td>103 ± 62</td>
<td>18 ± 17</td>
<td>&lt;.01</td>
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</table>

IMH, Intramural hematoma; TAD, total aortic diameter; TLD, true lumen diameter.

Data are presented as mean ± standard deviation.
Conclusions

• **PAU/IMH-Spectrum of AAS**
  – Not always benign
  – Predictors of worse outcome

• Intervention warranted