Thoracic Endovascular Aortic Repair for Penetrating Aortic Ulcer: Literature Review

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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 Cochrane databases reporting the results of endovascular repair for penetrating ulcers of the thoracic aorta were systematically searched and reviewed.**

Results. **Thirty-one articles were integrated 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 (65.8%), had a history of smoking (60.4%), and systemic hypertension (90%). Only 9% were asymptomatic at initial presentation. Most cases (76%) occurred among patients with a single ulcer, located in the descending thoracic aorta (81%), 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% (15 of 310). The most frequent complications were endoleaks (8%, 25 of 310) and access problems (16.1%, 26 of 161). After a mean follow-up of 17.7 months (range, 1 to 52), the all-cause mortality was 22.9% (71 of 310), and the aortic-related mortality was 4.1% (13 of 310). During follow-up, new endoleak and ulcer recurrence were observed in 5.4% (n = 15 of 274) and 4.5% (n = 5 of 110), respectively, requiring a new aortic endovascular procedure in 50% (n = 10).**

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

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After a first description by Shennan in 1934, penetrating aortic ulcer (PAU) is integrated into the acute aortic syndrome. Acute aortic syndrome subsumes the entity of PAU, intramural hematoma (IMH), traumatic rupture, and aortic dissection. This heterogeneous group of pathologies have distinct etiologies and pathophysiologic features, but IMH and PAU have similar clinical presentation and treatment. The mechanism of PAU is disruption of the internal elastic lamina that can spread to the media, leading to an IMH. Disruption of the media results in a pseudoaneurysm, and if the adventitia is perforated, transmural aortic rupture occurs as the ultimate stage in the natural history. The natural history of PAU progression remains controversial. There remains debate about how best to identify patients with more malignant vascular pathology requiring surgery and patient with non–life-threatening disease best managed with medical therapy. Despite technical advances, open repair for thoracic PAU is associated with high operative mortality that may reach 16% [1].

Since 1998 [2] and the first description of thoracic endovascular aortic repair (TEVAR) in the management of PAU, a number of successful case reports and series have been published. All the results suggest that TEVAR is now the preferred approach for thoracic PAU, with favorable short-term outcomes. The aim of this article is to define the outcomes for TEVAR of penetrating aortic ulcers with a particular focus on midterm results.

Material and Methods

Search Strategy

A literature search was undertaken to identify all studies published in the past 20 years that reported on thoracic endovascular aortic repair for penetrating ulcer. Candidate studies were sought through a computerized search of the Embase, Medline, and Cochrane databases for the period of January 1994 to March 2014. Key words entered in this search were "thoracic aorta" or "penetrating ulcer" or "penetrating atherosclerotic ulcer" or "endovascular repair." Articles were limited to those published in the English language. Additionally, manual evaluation of the reference lists of the retrieved articles and reviews on this subject area was performed.

Study Selection

Studies were considered for inclusion on the basis of the following criteria: (1) reporting of thoracic endovascular

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aortic repair for management of penetrating ulcers; and (2) reporting clinical outcome with at least 1-month follow up. Studies were excluded on the basis of the following criteria: (1) those containing duplicate data; only the manuscripts with the most recent or the best-documented material from the same patients were used for analysis; and (2) studies including abdominal PAU where it was not possible to separate out the outcomes of the subgroup undergoing TEVAR. Articles were selected for further review and inclusion in the final analysis if they described outcomes for individual patients treated for thoracic PAU.

Data Extraction

Data were extracted regarding age; sex; presence of vascular risk factors; American Society of Anesthesiologists (ASA) score; presence or history of thoracic aortic surgery; associated aortic disease (IMH, aneurysm, dissection, rupture, aortobronchial fistulae); initial clinical presentation; number of ulcers; interval between diagnosis and TEVAR; proximal landing zone; technical success of TEVAR defined by successful exclusion of the ulcer during the initial endovascular procedure; number of stent grafts used with diameter and length; management of the left subclavian artery (coverage with or without rerouting); cerebrospinal fluid drainage; and inhospital and long-term follow-up outcome, including graft-related complications (endoleak, stroke, spinal cord ischemia, and access problems defined as failure to achieve femoral access, dissection, rupture, fistula), non–graft-related complications (sepsis, pneumonia, renal failure, myocardial infarction), early and late open and endovascular reinterventions, ulcer recurrence, length of inhospital stay, and follow-up. When presence or absence of a variable was not explicitly stated in the manuscript, those patients were not included in the analysis. Therefore, the denominator used in the analysis, and reported in the results, indicates the number of patients for whom data were available.

Results

Search Results

Thirty-one articles [2–32] were integrated after a literature search, and identified 310 patients treated by TEVAR for PAU (Fig 1).

Patient Characteristics

Table 1 summarizes the clinical characteristics of the 310 patients included in the study. The mean age was 71.9 ± 6.1 years, 65.8% (204 of 310) were male, 60.4% (110 of 182) had a history of smoking, and 89.1% had systemic hypertension. Thoracic pain was reported in 65.4% (93 of 142) of the cases, collapse in 19.2% (20 of 104), with only 8.9% (11 of 123) completely asymptomatic at initial presentation. Pleural effusion was identified in 35.5% of the cases (37 of 104). The ASA score was 1 or 2 in 14.5% (16 of 110) and 3 or 4 in 85.5% (94 of 110).

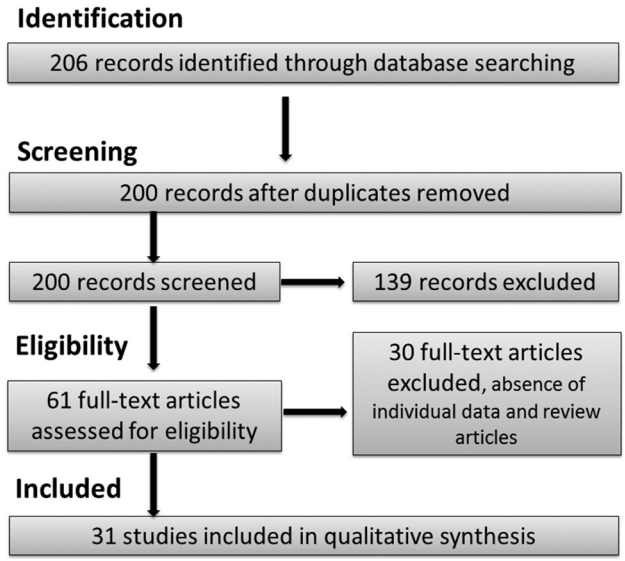

Fig 1. Preferred reporting items for systematic reviews and meta-analyses (PRISMA) flow diagram of the study.

Aorta Characteristics

Most reported cases (76.3%, 55 of 72) occurred in patients with only one ulcer, located in the descending thoracic aorta (81.8%, 207 of 253), and intramural hematoma was identified in 45.3% (64 of 141). The most common associated aortic disease was rupture in 29.7% (88 of 296), aneurysm in 25.3% (32 of 126), and dissection in 8% (8 of 100).

Endovascular Repair

The TEVAR was performed within 48 hours of diagnosis in 55.3% of the patients (93 of 168; Table 1). Technical success was 98.3% (305 of 310). Data on proximal landing zone were available for 253 cases: zone 0 in 6 patients, zone 1 to 2 in 39 patients, and zone 3 to 4 in 207 patients. The left subclavian artery was covered in 21.8% (62 of 284). In the majority of those cases, the left subclavian artery was rerouted (77.4%, 48 of 62). Preventive cerebrospinal fluid drainage was utilized in 24.7% (26 of 105).

Only one device was required in 79.5% (140 of 176), with mean length for that device of 119.1 mm (range, 77 to 200 mm). Overall mean device diameter was 35 ± 2.8 mm. Mean intervention duration was 106 ± 60.2 minutes (n = 108 of 310) and mean inhospital length of stay was 7.6 days (n = 169 of 310).

Perioperative Outcomes

Perioperative outcomes (Table 2) were defined as those occurring within the first 30 postoperative days. The overall 30-day mortality was 4.8% (15 of 310). Of the 15 patients who died within 30 days, 6 had multiorgan failures, 2 acute respiratory distress syndromes, 3 aorta ruptures (2 thoracic, 1 abdominal), 1 retroperitoneal hemorrhage, 1 hemorrhagic cerebrovascular accident, 1 aortoesophageal fistula, and 1 mesenteric ischemia.

The early morbidity rate was 36.4% (113 of 310). Access problems followed by endoleaks were the most common

Table 1. Clinical Characteristics

First Author [Ref]	Year	n	Age (years)	Male % (n)	Smoking History % (n)	DM % (n)	Renal Failure % (n)	CAD % (n)	PAD % (n)	COPD % (n)	AHT % (n)	Previous Aortic Surgery % (n)
Murgo [1]	1998	4	76[a]	75 (3)	…	…	25 (1)	50 (2)	…	25 (1)	100 (4)	…
Brittenden [2]	1999	2	71[a]	100 (2)	…	…	…	50 (1)	50 (1)	50 (1)	…	…
Czermack [3]	2002	2	73[a]	100 (2)	…	…	…	…	…	…	…	…
Faries [4]	2002	1	82	100 (1)	…	…	…	…	…	100 (1)	100 (1)	…
Kos [5]	2002	10	73[a]	70 (7)	60 (6)	20 (2)	0	0	0	0	40 (4)	…
Schoder [6]	2002	8	70[a]	62.5 (5)	…	37.5 (3)	50 (4)	37.5 (3)	…	50 (4)	87.5 (7)	25 (2)
D'Ancona [7]	2003	1	65	100 (1)	…	0	100 (1)	0	0	100 (1)	100 (1)	0
Krohg-Sørensen [8]	2003	2	77[a]	100 (2)	…	…	…	100 (2)	…	…	…	0
Crane [9]	2003	1	82	0	100 (1)	0	0	0	0	0	100 (1)	0
Heidenreich [10]	2003	1	63	0	100 (1)	0	0	0	100 (1)	100 (1)	100 (1)	0
Dake [11]	2004	26	70[a]	69.2 (18)	69.2 (18)	…	11.5 (3)	30.7 (8)	…	50 (13)	76.9 (20)	…
Melissano [12]	2005	2	76[a]	0	50 (1)	0	0	0	0	0	100 (2)	0
Brinster [13]	2006	21	73[a]	33.3 (7)	76.1 (16)	19 (4)	9.5 (2)	19 (4)	…	19 (4)	76.1 (16)	…
Khamaisi [14]	2006	1	75	0	0	0	0	0	0	0	100 (1)	0
Pauls [15]	2007	12	74[a]	75 (9)	33.3 (4)	16.6 (2)	25 (3)	8.3 (1)	8.3 (1)	…	91.6 (11)	…
Attia [16]	2007	3	69[a]	100 (3)	66.6 (2)	0	33.3 (1)	0	…	33.3 (1)	100 (3)	0
Citro [17]	2007	1	78	100 (1)	100 (1)	0	0	0	0	100 (1)	100 (1)	0
Piffaretti [18]	2007	11	73[a]	81.8 (9)	…	…	36.3 (4)	45.4 (5)	…	45.4 (5)	90.8 (10)	…
Botta [19]	2008	19	71[a]	73.6 (14)	36.8 (7)	…	36.8 (7)	…	…	68.4 (13)	84.2 (16)	10.5 (2)
D'Souza [20]	2009	20	67[a]	65 (13)	60 (12)	10 (2)	5 (1)	15 (3)	…	35 (7)	85 (17)	…
Girns [21]	2009	11	71[a]	63.6 (7)	…	…	…	…	…	…	100 (11)	…
Patel [22]	2010	37	72[a]	43.2 (16)	54 (20)	8.1 (3)	…	59.4 (22)	21.7 (8)	43.2 (16)	83.7 (31)	…
Canaud [23]	2010	1	85	100 (1)	0	0	0	0	0	0	100 (1)	0
Komen [24]	2011	1	57	100 (1)	0	0	0	0	0	0	100 (1)	0
Czerny [25]	2011	72	67[a]	70.8 (51)	…	…	13.8 (10)	31.9 (23)	9.7 (7)	27.7 (20)	94.4 (68)	27.7 (20)
Tumelero [26]	2011	2	71[a]	50 (1)	50 (1)	0	0	0	0	0	100 (2)	0
Palombo [27]	2012	13	70[a]	61.5 (8)	…	…	…	…	…	…	100 (13)	…
Chong [28]	2012	1	66	100 (1)	0	0	0	100 (1)	0	0	100 (1)	0
Mestres [29]	2012	22	69[a]	90.9 (20)	86.3 (19)	40.9 (9)	27.2 (6)	50 (11)	4.5 (1)	40.9 (9)	100 (22	4.5 (1)
Siegel [30]	2013	1	82	0	…	100 (1)	100 (1)	0	0	0	100 (1)	0
Esteban [31]	2014	1	63	100 (1)	100 (1)	0	0	0	0	100 (1)	100 (1)	0

[a] Mean.

Ellipsis (…) represents data unavailable.

AHT = arterial hypertension; COPD = chronic obstructive pulmonary disease; CAD = coronary artery disease; DM = diabetes mellitus; PAD = peripheral artery disease.

Table 2. TEVAR Perioperative Complications

	Perioperative Complications									
First Author [Ref]	Paraplegia % (n)	Stroke % (n)	Sepsis % (n)	Multiorgan Failure % (n)	Pulmonary Complication % (n)	Renal Failure % (n)	Myocardial Infarction % (n)	Endoleak % (n) (types)	Access Problems % (n)	Overall % (n)
Murgo [1]	25 (1)	0	0	0	25 (1)	…	…	1 (4)	0	100 (4)
Brittenden [2]	0	…	…	…	…	…	…	0	50 (1)	50 (1)
Czermack [3]	0	0	…	…	…	…	…	0	0	50 (1)
Faries [4]	0	0	0	0	0	0	0	0	0	0
Kos [5]	10 (1)	0	0	0	0	0	0	4 (1,1,1,2)	0	60 (6)
Schoder [6]	12.5 (1)	…	…	…	…	…	…	1 (2)	37.5 (3)	62.5 (5)
D'Ancona [7]	0	0	0	0	0	0	0	0	0	100 (1)
Krohg-Sørensen [8]	…	…	…	…	…	…	50 (1)	0	50 (1)	0
Crane [9]	0	0	0	0	0	0	0	0	0	0
Heidenreich [10]	0	100 (1)	0	0	0	0	0	0	100 (1)	100 (1)
Dake [11]	0	3.8 (1)	3.8 (1)	3.8 (1)	3.8 (1)	0	0	2 (1,1)	19.2 (5)	42.3 (11)
Melissano [12]	0	0	0	0	0	0	0	0	100 (2)	100 (2)
Brinster [13]	0	0	0	0	0	…	0	0	23.8 (5)	28.5 (6)
Khamaisi [14]	0	0	0	0	0	0	0	0	0	0
Pauls [15]	0	0	…	…	…	…	…	2 (2,3)	16.6 (2)	58.3 (7)
Attia [16]	0	0	…	…	…	…	…	1 (4)	0	33.3 (1)
Citro [17]	0	0	0	0	0	0	0	0	…	0
Piffaretti [18]	0	9 (1)	0	0	0	36.3 (4)	0	0	…	45.4 (5)
Botta [19]	0	0	…	10.5 (2)	10.5 (2)	10.5 (2)	0	1 (2)	5.2 (1)	47.3 (9)
D'Souza [20]	0	0	…	…	…	…	…	3 (1,2,2)	20 (4)	40 (8)
Patel [21]	5.4 (2)	5.4 (2)	0	0	0	2.7 (1)	0	3 (1,2,2)	…	43.2 (16)
Girns [22]	9 (1)	…	9 (1)	9 (1)	18 (2)	…	…	1 (1)	…	54.5 (6)
Canaud [23]	0	0	0	0	100 (1)	0	0	0	0	100 (1)
Komen [24]	0	0	0	0	0	0	0	0	…	0
Czerny [25]	1.3 (1)	2.7 (2)	…	1.3 (1)	…	1.3 (1)	0	5 (1,2,2,3,4)	…	16.6 (12)
Tumelero [26]	0	0	0	0	0	0	0	0	…	0
Palombo [27]	7.6 (1)	0	…	7.6 (1)	7.6 (1)	…	…	0	…	30.7 (4)
Chong [28]	0	0	0	0	0	0	0	0	…	0
Mestres [29]	4.5 (1)	0	0	0	4.5 (1)	4.5 (1)	0	1 (2)	4.5 (1)	40.9 (9)
Soegel [30]	0	0	0	0	0	0	0	0	0	0
Esteban [31]	0	0	0	0	0	0	0	0	0	0

Elipsis (…) represents data unavailable.

Table 3. Midterm and Long-Term Outcomes

First Author [Ref]	Mean Follow-Up (months)	Mortality			Survival %			Late Outcome >30 Day		
		Overall % (n)	<30 Day % (n)	Aortic Related % (n)	1 Year	3 Years	5 Years	New Endoleak % (n)	Disease Evolution % (n)	New Aortic Procedure % (n)
Murgo [1]	6	50 (2)	0	0	…	…	-	25 (1)	50 (2)	0
Brittenden [2]	12	0	0	0	100	…	…	0	0	0
Czermack [3]	15	5 (1)	0	0	50	…	…	0	100 (2)	1
Faries [4]	18	0	0	0	100	…	…	0	0	0
Kos [5]	9	10 (1)	0	10 (1)	…	…	…	10 (1)	…	0
Schoder [6]	14	37.5 (3)	0	12.5 (1)	100	…	…	0	12.5 (1)	0
D'Ancona [7]	1	0	0	0	…	…	…	…	…	…
Krohg-Sørensen [8]	12	0	0	0	100	…	…	…	…	…
Crane [9]	12	0	0	0	100	…	…	0	0	0
Heidenreich [10]	1	0	0	0	…	…	…	…	…	…
Dake [11]	51	30.7 (8)	11.5 (3)	3.8 (1)	85	76	70	3.8 (1)	…	3.8 (1)
Melissano [12]	27	0	0	0	100	…	…	0	0	0
Brinster [13]	14	4.7 (1)	0	0	…	…	…	0	…	0
Khamaisi [14]	1	0	0	0	…	…	…	…	…	…
Pauls [15]	28	0	0	0	…	…	…	1	…	0
Attia [16]	14	0	0	0	100	…	…	…	…	…
Citro [17]	6	0	0	0	…	…	…	…	…	…
Piffaretti [18]	15	9 (1)	0	0	90	90	…	…	…	…
Botta [19]	22	31.5 (6)	10.5 (2)	0	83	72	66	10.5 (2)	…	5.2 (1)
D'Souza [20]	24	10 (2)	0	5 (1)	…	…	…	0	0	0
Patel [21]	32	37.8 (14)	5.4 (2)	10.8 (4)	…	…	…	10.8 (4)	…	10.8 (4)
Girns [22]	32	27.2 (3)	27.2 (3)	9 (1)	…	…	…	…	…	0
Canaud [23]	5	0	0	0	…	…	…	0	0	0
Komen [24]	12	0	0	0	100	…	…	…	…	…
Czerny [25]	42	23.6 (17)	4.1 (3)	1.3 (1)	93	…	72	4.1 (3)	…	1.3 (1)
Tumelero [26]	25	0	0	0	…	…	…	…	…	…
Palombo [27]	16	38.4 (5)	7.6 (1)	15.3 (2)	74.5	…	…	0	0	0
Chong [28]	1	0	0	0	…	…	…	…	…	…
Mestres [29]	52	31.8 (7)	4.5 (1)	4.5 (1)	…	…	61.2	9 (2)	…	9 (2)
Siegel [30]	1	0	0	0	…	…	…	…	…	…
Esteban [31]	12	0	0	0	100	…	…	0	0	0

Elipsis (…) represents data unavailable.

early complications (16.1% [26 of 161] and 8% [25 of 310], respectively). Endoleaks were described as type 1 in 9 cases, type 2 in 10, type 3 or 4 in 6 cases.

Neurologic events such as stroke and paraplegia occurred in 2.4% (7 of 287) and 2.9% (9 of 308). Paraplegia remained permanent in only 1 case; in other cases, complete resolution of neurology occurred subsequent to cerebrospinal fluid drainage.

The incidence of postoperative sepsis, multiorgan failure, renal failure, and pulmonary complication were, respectively, 1.2% (2 of 157), 2.3% (6 of 261), 4% (9 of 221), 4.7% (9 of 189); and 1 patient had myocardial ischemia. Three cases of postoperative aortic rupture and 2 cases of fistula were described. Aortic dissection was not observed. One patient required surgical conversion with thoracic stent-graft explantation during their postoperative course.

Midterm Outcomes

Midterm outcomes (Table 3) were defined as those occurring after 30 days. After a mean follow-up of 17.8 months (range, 1 to 52), the overall mortality rate was 22.9% (71 of 310) and the aortic-related mortality was 4.1% (13 of 310). Survival rates at 1, 3, and 5 years were, respectively, 91.1% (164 of 180), 79.3% (56 of 70), and 67.3% (139 of 206). During the follow-up, new endoleak was observed in 5.4% (15 of 274) and ulcer recurrence in 4.5% (5 of 110) of cases, necessitating a new aortic endovascular procedure in 50% (10 cases).

Comment

Penetrating ulcer of the thoracic aorta represents about 7.6% of cases in acute aortic syndrome, but the exact incidence remains unknown [33]. Most patients included in the study were in their seventies and the majority were men. This cohort tends to have numerous comorbidities such as previous history of smoking (60%), systemic hypertension (90%), and a high ASA score (ASA = 3 or 4 in 85%). Thoracic pain is the most frequent symptom (65%), and pleural effusion was observed in 35% of the cases. The clinical presentation may imitate aortic dissection but since the advent of computed tomography scanning and magnetic resonance imaging, it is possible to distinguish between the pathologies. The typical radiodiagnostic features of PAU are an atherosclerotic aorta with erosion of the intima and associated central ulceration. The media, exposed to pulsatile pressure can locally dissect, creating an IMH. In this study, IMH were coexistent with PAU in 45% of cases. The Stanford study [12] demonstrated that PAU with IMH is associated with more progressive disease and greater complications.

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 aortic complications, we only identified 3 cases of postoperative aortic rupture, 2 cases of fistula, and no aortic dissection. Surgical conversion during the postoperative course with thoracic stent-graft explantation was required in 1 patient because of a persistent type 1 endoleak [25]. Endoleaks are the second most frequent complication reported (8%), including type 1 in 9 cases, type 2 in 10, and type 3 or 4 in 6 cases. Most reported cases (76%) occurred in patients with only one ulcer, located in the descending thoracic aorta (81%), and only one device was required in 79.5% of patients. That may explain a short operative time of 106 minutes, and a mean hospital stay of 7 days.

Severe atherosclerosis was present in most of the cases [34], and cardiovascular risk factors were frequent. That could explain the 16.1% rate of access problems, results congruent with a recent American national registry review of 1,154 patients undergoing elective TEVAR, in which 10% of the patients required iliac access with retroperitoneal exposure [34].

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.

This study has certain limitations. Most of the reported articles are cases reports and short series, including retrospective studies with missing data. But the suspected very low incidence of PAU explains the small numbers.

Although indications for intervention in the management of PAU are still debated, in agreement with recent guidelines [35, 36], we think that asymptomatic patients with an incidental finding of PAU on imaging should simply be observed. For patients with persistent or recurrent symptoms, aortic diameter greater than 55 mm or an increase of more than 10 mm per year at the level of the ulcer, and associated aorta diseases (dissection, aneurysm, IMH, rupture, fistula), TEVAR is indicated.

In conclusion, TEVAR of penetrating ulcer has excellent short-term and midterm results. The endovascular approach should be used as first line treatment of thoracic aortic ulcers when surgery is indicated. It is clear, however, that the middle and long-terms results are significantly affected by patient comorbidity and severe atherosclerosis, as the aortic mortality is very low and the rate of access problems high.

References

1. Ganaha F, Miller DC, Sugimoto K, et al. Prognosis of aortic intramural hematoma with and without penetrating atherosclerotic ulcer: a clinical and radiological analysis. Circulation 2002;106:342–8.

2. Murgo S, Dussaussois L, Golzarian J, et al. Penetrating atherosclerotic ulcer of the descending thoracic aorta: treatment by endovascular stent-graft. Cardiovasc Intervent Radiol 1998;21:454–8.
3. Brittenden J, McBride K, McInnes G, Gillespie IN, Bradbury AW. The use of endovascular stents in the treatment of penetrating ulcers of the thoracic aorta. J Vasc Surg 1999;30:946–9.
4. Czermak BV, Waldenberger P, Perkmann R, et al. Placement of endovascular stent-grafts for emergency treatment of acute disease of the descending thoracic aorta. AJR Am J Roentgenol 2002;179:337–45.
5. Faries PL, Lang E, Ramdev P, Hollier LH, Marin ML, Pomposelli FB. Endovascular stent-graft treatment of a ruptured thoracic aortic ulcer. J Endovasc Ther 2002;9(Suppl 2):II20–4.
6. Kos X, Bouchard L, Otal P, et al. Stent-graft treatment of penetrating thoracic aortic ulcers. J Endovasc Ther 2002;9(Suppl 2):II25–31.
7. Schoder M, Grabenwöger M, Hölzenbein T, et al. Endovascular stent-graft repair of complicated penetrating atherosclerotic ulcers of the descending thoracic aorta. J Vasc Surg 2002;36:720–6.
8. D'Ancona G, Bauset R, Normand JP, Turcotte R, Dagenais F. Endovascular stent-graft repair of a complicated penetrating ulcer of the descending thoracic aorta: a word of caution. J Endovasc Ther 2003;10:928–31.
9. Krohg-Sørensen K, Hafsahl G, Fosse E, Geiran OR. Acceptable short-term results after endovascular repair of diseases of the thoracic aorta in high risk patients. Eur J Cardiothorac Surg 2003;24:379–87.
10. Crane JS, Cowling M, Cheshire NJ. Endovascular stent grafting of a penetrating ulcer in the descending thoracic aorta. Eur J Vasc Endovasc Surg 2003;25:178–9.
11. Heidenreich MJ, Neschis DG, Costanza MJ, Flinn WR. Endovascular repair of a penetrating thoracic aortic ulcer by way of the carotid artery. J Vasc Surg 2003;38:1407–10.
12. Demers P, Miller DC, Mitchell RS, Kee ST, Chagonjian L, Dake MD. Stent-graft repair of penetrating atherosclerotic ulcers in the descending thoracic aorta: midterm results. Ann Thorac Surg 2004;77:81–6.
13. Melissano G, Astore D, Civilini E, Chiesa R. Endovascular treatment of ruptured penetrating aortic ulcers. Ann Vasc Surg 2005;19:270–5.
14. Brinster DR, Wheatley GH, Williams J, Ramaiah VG, Diethrich EB, Rodriguez-Lopez JA. Are penetrating aortic ulcers best treated using an endovascular approach? Ann Thorac Surg 2006;82:1688–91.
15. Khamaisi M, Anner H, Marincheva G, Leitersdorf E, Bogot N. Stent graft in treatment of penetrating thoracic aortic ulcer. J Am Soc Echocardiogr 2006;19:835.e4–e6.
16. Pauls S, Orend KH, Sunder-Plassmann L, Kick J, Schelzig H. Endovascular repair of symptomatic penetrating atherosclerotic ulcer of the thoracic aorta. Eur J Vasc Endovasc Surg 2007;34:66–73.
17. Attia C, Villard J, Boussel L, et al. Endovascular repair of localized pathological lesions of the descending thoracic aorta: midterm results. Cardiovasc Intervent Radiol 2007;30:628–37.
18. Citro R, Bossone E, Patella MM, et al. Endovascular treatment of painless aortic ulcer detected by transesophageal echocardiography as an incidental finding. J Cardiovasc Med 2007;8:1049–51.
19. Piffaretti G, Tozzi M, Lomazzi C, et al. Penetrating ulcers of the thoracic aorta: results from a single-centre experience. Am J Surg 2007;193:443–7.
20. Botta L, Buttazzi K, Russo V, et al. Endovascular repair for penetrating atherosclerotic ulcers of the descending thoracic aorta: early and midterm results. Ann Thorac Surg 2008;85:987–92.
21. D'Souza S, Duncan A, Aguila F, et al. TEVAR for non-aneurysmal thoracic aortic pathology. Catheter Cardiovasc Interv 2009;74:783–6.
22. Patel HJ, Williams DM, Upchurch GR, Dasika NL, Deeb GM. The challenge of associated intramural hematoma with endovascular repair for penetrating ulcers of the descending thoracic aorta. J Vasc Surg 2010;51:829–35.
23. Girn HR, McPherson S, Nicholson T, Mavor AI, Homer-Vanniasinkam S, Gough MJ. Short series of emergency stent-graft repair of symptomatic penetrating thoracic aortic ulcers (PTAU). Vasc Med 2009;14:123–8.
24. Canaud L, Hireche K, Berthet JP, Branchereau P, Marty-Ané C, Alric P. Endovascular repair of aortic arch lesions in high-risk patients or after previous aortic surgery: midterm results. J Thorac Cardiovasc Surg 2010;140:52–8.
25. Komen N, Vercauteren S, de Roover D. Aortic hammer syndrome. J Endovasc Ther 2011;18:497–500.
26. Czerny M, Funovics M, Sodeck G, et al. Results after thoracic endovascular aortic repair in penetrating atherosclerotic ulcers. Ann Thorac Surg 2011;92:562–6.
27. Tumelero RT, Duda NT, Tognon AP, Panata L, Teixeira JC, Giongo JB. Endovascular stent-graft treatment of penetrating aortic ulcer. Arq Bras Cardiol 2011;96:e20–3.
28. Palombo D, Lucertini G, Robaldo A, Pane B, Spinella G. Treatment of penetrating aortic ulcer by endoprosthesis: a single center experience. Int Angiol 2012;31:54–61.
29. Chong F, Winter M, Puckridge P, Penhall A, Joseph M. A rare case of penetrating atherosclerotic ulcer of the aorta. Heart Lung Circ 2012;21:105–7.
30. Mestres G, Rodríguez R, García-Madrid C, et al. Endovascular treatment of penetrating aortic ulcers: midterm follow-up. Rev Esp Cardiol (Engl Ed) 2012;65:54–9.
31. Siegel Y. Penetrating atherosclerotic aortic ulcer rupture causing a right hemothorax: a rare presentation of acute aortic syndrome. Am J Emerg Med 2013;31:755–7.
32. Esteban C, Pérez P, Muchart J, Sampere J, Martorell A, Llagostera S. Treatment of a complicated penetrating ulcer of the descending thoracic aorta using a scalloped stent graft. Ann Vasc Surg 2014;28:264.e9–e11.
33. Eggebrecht H, Plicht B, Kahlert P, Erbel R. Intramural hematoma and penetrating ulcers: indications to endovascular treatment. Eur J Vasc Endovasc Surg 2009;38:659–65.
34. Tsilimparis N, Dayama A, Perez S, Ricotta JJ. Iliac conduits for endovascular repair of aortic pathologies. Eur J Vasc Endovasc Surg 2013;45:443–8.
35. Erbel R, Aboyans V, Boileau C, et al. 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). Eur Heart J 2014;35:2873–926.
36. Evangelista A, Czerny M, Nienaber C, et al. Interdisciplinary expert consensus on management of type B intramural haematoma and penetrating aortic ulcer. Eur J Cardiothorac Surg 2015;47:209–17.