Clinical and pathologic findings of aortic dissection at autopsy: Review of 336 cases over nearly 6 decades

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Abstract Background We aimed to characterize the clinical and pathologic findings of aortic dissection (AD) over a nearly 60-year period.

Methods The Jesse E. Edwards Registry of Cardiovascular Disease database was queried for cardiac specimens from autopsies with AD as a diagnosis and compared 2 cohorts: early (1956-1992) and current (1993-2015).

Results From 1956 to 2015, 338 cases (166 early, 170 current) with AD were included (mean age: 60; 62% male). The AD was 86% type A and 14% type B. Sixty-two percent of cases were under medical care at time of death (61% early, 62% current, *P* = not significant). Of those under medical care, 63% were not diagnosed prior to death (64% early, 62% current, *P* = not significant).

Risks for dissection did not differ between time intervals and include left ventricular hypertrophy, suggestive of hypertension (84%), prior cardiovascular surgery (38%), bicuspid valve (14%), and connective tissue disease (9%). An intimal tear was identified in the ascending aorta in the majority (68%), followed by descending (14%), root (9.5%), and arch (7%). Aortic rupture occurred in 58%, most frequently in the ascending aorta (41%).

Conclusions In a large cardiovascular registry, >60% of cases of AD were not detected clinically and first identified at autopsy. Although diagnostic techniques have significantly improved over the time interval, the percentage of AD discovered at autopsy did not differ from the early to the current era. The most prevalent risk factors for dissection including hypertension and prior cardiovascular surgery remain similar in both time periods. AD death is related to rupture of the aorta in the majority of cases. (Am Heart J 2019;209:108-115.)

Acute aortic dissection (AD) requires emergent diagnosis and treatment; however, frequently, the diagnosis is delayed or missed altogether and not established until autopsy.[1-6] Many of the early seminal reports of acute AD were based in large part on autopsy findings.[1-6] Over time, there have been a significant shift and improvement in testing modality used to diagnose AD, from primarily chest radiography and catheter-based aortography to computed tomography (CT), transesophageal echocardiography, and magnetic resonance imaging, each of which has high sensitivity and specificity for the diagnosis.[7,8] Additionally, since DeBakey described the first successful operation for dissection in 1954, there has also been significant improvement in surgical techniques.[9]

Registries such as the International Registry of Acute Aortic Dissection have greatly informed clinicians regarding the diagnosis and management of patients with dissection.[1] It is known that the diagnosis and treatment of acute AD are frequently delayed and may be affected by variables including atypical symptoms, prior cardiac surgery, and presentation to nontertiary hospitals[10]; however, there is potential in clinical registries to overlook cases that are missed altogether.[1,10] Therefore, although early studies of AD were based in large part on autopsy findings, contemporary autopsy series of AD are relatively infrequent.

Given the significant improvements in imaging and recognition of thoracic aortic aneurysms as well as acute AD, we hypothesized that patients dying from AD and being referred to autopsy may have evolved over time. We therefore sought to examine the

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demographics, clinical risk factors, and diagnosis of AD as well as the pathologic findings in a referral-based cardiovascular (CV) pathology registry. We also explored whether the clinical findings or recognition differed from the early to the later years of the registry.

Methods

The Jesse E. Edwards Registry of Cardiovascular Disease is a national referral center for CV autopsy specimens. Dr Jesse E. Edwards, a prominent cardiac pathologist (1912-2008), initiated the registry at the Miller Hospital in St Paul, MN, in 1960.[11] Currently housed at United Hospital in St Paul, MN, it has been running continuously since its inception with currently >30,000 cases housed at the Registry.

We examined cases from 1956 through November 2015 that were identified by coding for AD on the final report. Cases were excluded if they were surgical specimens or tissue donor referral hearts. Demographic data, clinical data, and pathological features of the dissection including the extent of the dissection and the location of the intimal tear were compared.

One author independently reviewed all of the records available in each case. Data are presented as a percentage (%) of those in which data are available.

Definitions

Cardiac pathology examinations were performed by cardiac pathologists at the Registry over the study interval. Subjects were defined as *dying under medical care* if they were in the hospital or had visited a clinic or hospital with symptoms and documentation of being evaluated by a medical provider prior to their demise (including those sent home from medical facility). Those not under medical care died prior to hospital arrival and had no evidence of medical evaluation by a health care provider for symptoms related to the AD. Cardiomegaly was determined by comparison to published nomograms of normal heart weights for height and weight.[12] The diagnosis of left ventricular hypertrophy (LVH) was made when the thickness of the compact myocardium was greater than 1.5 cm in a normally sized heart or when the heart was determined to be enlarged by comparison to the reference tables.[12] *Significant coronary artery disease* was defined as greater than 75% cross-sectional narrowing. Aortic dissection was identified as the cause of death if there was evidence of rupture or if it was acute and led to secondary complications.

Two autopsy cohorts of similar size were formed for comparison of 2 eras: early (1956-1992) and current (1993-2015). This cut point of 1992-1993 dividing the data set coincided with a period in which there was increased utilization of noninvasive imaging techniques to diagnose AD.[7,13]

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Descriptive statistics are displayed as mean and SD for normally distributed continuous variables. Where continuous variables have skewed distributions, data are reported as median (25th, 75th percentile). Categorical variables are reported as number and percentage with characteristics. Continuous variables were analyzed using Student *t* test or Kruskal-Wallis rank sum tests. Categorical variables were analyzed using Pearson χ^2 or Fisher exact tests. A value of $P < .05$ was considered significant; *P* values are 2-sided where appropriate. Statistical calculations and plots were performed with Stata 14.1 (College Station, TX).

Results

A total of 336 cases (166 early, 170 current) with AS were identified, and the majority (86%) were type A (Table I). The mean age of the total cohort was 60.0 years, and the majority (62%) of the patients were male. Most of the patients were local referrals from the greater Minneapolis/St Paul metro area. Patients in the 2 eras were of similar age and gender but more frequently were referred from outside the local area in the current era (Table I). AD was the cause of death in 86% (84% early, 88% current, *P* = not significant [NS]) of the entire cohort, and 62% of cases were under medical care at time of death (61% early, 62% current, *P* = NS). Of those under medical care, 63% were not diagnosed prior to death and did not differ between the 2 time intervals (64% early, 62% current, *P* = NS).

Clinical risk factors and coexisting conditions

Cardiomegaly with LVH (mean 606 ± 156 g), a marker of preexisting hypertension, was found in 85% and was the most common preexisting clinical risk factor identified, followed by prior CV surgery (38%). Other clinical risk included bicuspid valve (14%), connective tissue disease (9%) (most commonly Marfan syndrome [6%]), aortitis (8%) (syphilitic in 2, giant cell in 1, unknown in remainder), and congenital heart disease (6%). The clinical risk factors did not differ between time periods (Table I). Medial degeneration was identified in 47% at autopsy. Coexistent coronary artery disease was identified in the majority of cases. There was no difference in these pathologic findings between the 2 time intervals.

Type A versus type B

Cases with type A and B dissection did not differ with respect to age or gender. Cases with type B AD were more likely to have died under medical care (Table II). Medial degeneration and bicuspid aortic valve were more

Table I. Overview of 336 autopsy specimens with AD

	# With data	All patients (N = 336)	1956-1992 (n = 166)	1993-present (n = 170)	*P* value
Age (y), mean ± SD	336	60.0 ± 16.6	61.7 ± 16.7	58.3 ± 16.4	.058
Male (%)	*337*	*209 (62.0)*	*105 (63.3)*	104 (60.8)	*.65*
Referral location	313				
Local (%)		280 (87.0)	145 (92.4)	135 (81.8)	.004
Greater MN (%)		17 (5.3)	2 (1.3)	15 (9.1)	
Out of state (%)		25 (7.8)	10 (6.4)	15 (9.1)	
LVH (%)	295	248 (84.1)	114 (84.0)	134 (78.3)	.90
Prior CV surgery (%)	279	105 (37.5)	45 (37.2)	60 (37.7)	.93
Bicuspid aortic valve (%)	303	44 (14.3)	24 (16.6)	20 (12.3)	.28
Connective tissue disorder (%)	252	22 (8.7)	10 (10.1)	12 (7.7)	.51
Marfan syndrome	252	13 (5.1)	6 (6.1)	7 (4.5)	.59
Aortitis (%)	229	18 (7.8)	7 (8.0)	11 (7.7)	.94
Congenital heart disease (%)	220	13 (5.9)	4 (4.8)	9 (6.5)	.59
Medial degeneration (%)	294	136 (46.0)	64 (44.8)	72 (47.1)	.69
Coronary artery disease (%)	307	245 (79.3)	112 (77.8)	133 (80.6)	.54
Died under medical care (%)	263	163 (62.0)	72 (61.0)	91 (62.8)	.77
Premortem diagnosis* (%)	159	59 (37.1)	26 (36.1)	33 (37.9)	.81
Cause of death					
Dissection (%)	288	291 (86.5)	158 (94.6)	133 (77.8)	.35

*Of those who died under medical care.

Table II. Comparison of autopsy cases with type A versus type B AD

	# With data	Type A (n = 289)	Type B (n = 47)	*P* value
Age (y), mean(SD)	336	59.5 ± 16.4	62.7 ± 18.2	.24
Age range		18-92	13-88	N/A
Male (%)	*336*	177 (61.5)	30 (63.8)	.76
Female (%)		111 (38.5)	18 (36.2)	
Referral location	313			
Local (%)		239 (82.7)	29 (83.0)	N/A
Greater MN (%)		16 (5.5)	1 (2.1)	
Out of state (%)		19 (6.6)	6 (12.8)	
No data (%)		15 (5.2)	1 (2.1)	
Died under medical care (%)	261	137 (60.1)	26 (78.8)	.038

commonly associated with type A dissection than with type B (Supplemental Table I).

Age

The mean age of AD cases in patients with congenital disease was younger than those without congenital disease (43.2 ± 22.7 years vs 60.0 ± 16.7 years; $P < .001$). Similarly, the mean ages of AD in cases with connective tissue disorder and bicuspid valve disease were younger than those without (40.7 ± 12.7 years vs 61.0 ± 16.5 years, $P < .001$ and 48.7 ± 16.2 years vs 61.4 ± 16.2 years, $P < .001$, respectively). Conversely, the mean age of AD cases in patients with prior CV surgery was older than those without surgery (63.3 ± 15.6 vs 57.8 ± 17.8, $P = .009$).

Activity at time of death

Sixty cases had relevant data regarding activity at time of onset of AD: 10 patients (17%) were sleeping, 25 patients (46%) were engaging in minimal activity (driving, watching TV, eating dinner, etc), 20 patients (33%) were engaging in strenuous activity (shoveling snow, lifting heavy objects, sexual intercourse), and 5 patients were demonstrating emotional/mental strain as a possible precipitating factor.

Premortem evaluation

Of the 162 cases known to be under medical care at time of death, 114 had data available regarding diagnostic testing performed prior to death (Supplementary Table II). The primary modalities used in the early cohort were electrocardiogram, chest radiography, and invasive aortography (n = 13, 22%), whereas CT (n = 28, 50%) and echocardiography (n = 22, 39%) were more commonly used in the current cohorts.

Aortic dissection location and complications

Intimal tear was present in every case and was identified in the ascending aorta in the majority (227,

Table III. Rupture location

	Number of cases (N = 236)
Hemopericardium	175
Pleural space	48
Mediastinum	38
Retroperitoneal	8
Abdominal cavity	3

Table IV. Complications of AD (cause of death)

	All patients (N = 336)
Aortic rupture	196 (58%)
Ascending aorta	139 (41)
Descending aorta	20 (6)
Aortic root	20 (6)
Aortic arch	13 (4)
Abdominal aorta	4 (1)
AD specific complications	24 (7%)
Acute myocardial infarct	14 (4)
Gastrointestinal bleed/fistula	10 (3)
AD without rupture	71 (21%)
Sepsis/shock	8 (2)
Coagulopathy	8 (2)
Surgical complications	14 (4)
CVA	4 (1)
Cardiopulmonary arrest	32 (10)
Comfort care	5 (2)
Undetermined	45 (14%)

CVA - cerebrovascular accident

68.0%), followed by the descending (47, 14%), aortic root (32, 10%), aortic arch (24, 7%), and abdominal aorta (3, 1%). Twenty-six percent of patients (86 of 336) had their dissections confined to the ascending aorta (DeBakey type II dissection). Aortic rupture was frequently observed as shown by anatomic level of rupture in Table III. The pericardium (hemopericardium) was the most common location as seen in 174 patients (52%) overall. Of the cases with hemopericardium from which an identified rupture site was located, 89% was from the ascending aorta, 8% from aortic root, and 3% from the aortic arch.

AD death was most commonly due to rupture (58%) as shown in Table IV. The most common rupture site was the ascending aorta, but it also occurred in the root, descending thoracic aorta, and aortic arch. Rupture of ascending aorta was cause of death in 7 of the 9 patients with bicuspid valve. Of 187 cases that died of AD without clinical knowledge of AD until autopsy, 155 (83%) died of rupture. Deaths related to AD but not due to rupture included coronary ischemia (4%) and gastrointestinal ischemia or bleeding (3%). The most common cause of death in patients without aortic rupture was sudden cardiopulmonary arrest/arrhythmia, which accounted for 10% of causes of death, although several other etiologies

Table V. Branch Vessel Dissection

Aortic Dissection, extra aortic vessel involvement	Number of Cases
Total cases with extra aortic vessel involvement	142
Brachiocephalic	50
Subclavian Artery	46
Left Only	39
Right Only	2
Both	1
Not Recorded	4
Common Carotid	29
Left only	19
Right only	7
Both	2
Not Reported	1
Renal Arteries	26
Left only	12
Right only	4
Both	11
Unspecified Renal Artery	10
Celiac artery	13
SMA/IMA artery	18
Iliac Artery*	30

*Data regarding specific iliac involvement is incomplete.

also contributed (surgical complications, sepsis, coagulopathy, stroke). Healed dissections were found in 7% of cases, (n = 25, 20 type A, 5 type B), each of whom was not recognized clinically and died from non–AD-related causes including intracerebral hemorrhage, subdural hematoma, non–AD-related myocardial infarction, rejection of orthotropic heart transplant, and septic shock.

Branch vessel involvement data were available for a subset of the overall group (Table V). In this group, the frequency of aortic dissection extending to involve extra-aortic vascular branches occurred in roughly 45% of cases. The most common sites of involvement were the arch vessels (brachiocephalic, subclavian, carotids) followed by the iliac, renal, and mesenteric vessels. Data regarding specific iliac artery involvement were not consistently available.

A significant subgroup (n = 101) of the population had undergone prior cardiac surgery as demonstrated in Table VI. The surgical procedure involved the ascending aorta in approximately half the case including some patients who died from acute complications during the index AD. AD complications also occurred days to years after surgery. Rupture was the cause of death in 50% of the postsurgical group and occurred distal to the repaired ascending aorta after both type A AD as well as elective ascending aorta repairs. Eleven cases that had undergone coarctation repair developed AD, and 4 of these cases ruptured in ascending aorta. AD occurred just superior to the aortic valve after aortic valve replacement (AVR) and at saphenous vein graft ostium or aortic cannulation sites for coronary artery bypass graft (CABG) cases. In 3 cases,

Table VI. Aortic Dissection cases occurring after Cardiac surgery

	Number of cases	Ruptures	Median time until death (months)*	Number of healed (incidental) dissections
Type A Dissection Surgery	20	10		3
Acute Type A	13	9	0.13 (0.03, 0.92)	0
Prior Type A	7	1	180 (40, 240)	3
Elective Ascending Aorta Surgery	29	11		7
Ascending Aorta Alone	4	1	0.46 (0.23, 11.99)	3
Prior Aortic Surgery + CABG	6	3	0.46 (0.03, 144)	2
Prior Aortic Surgery + CABG + AVR	6	1	72 (0.03, 203.99)	0
Aortic Surgery + AVR	13	6	0.46 (0.03, 96)	2
Non Aortic Cardiac Surgery	34	21		4
AVR alone	10	4	66 (0.16, 120)	1
AVR + CABG	8	4	0.15 (0.03, 0.59)	1
CABG alone	14	11	0.57 (0.07, 192)	2
MVR	2	2	56 (5, 108)	0
Descending Aorta Surgery	18	7		2
Acute Type B	3	1	0.07 (0.03, 0.33)	1
Prior Type B	4	1	15.5 (1.97, 96)	1
Coarctation Repair	11	5	10.5 (0.03, 420)	0

• All Variables reported as median (minimum, maximum).

a fistula formed between the descending aortic graft and the gastrointestinal tract, leading to bleeding. Cardiac tamponade occurred after cardiac surgery in 16 of the 70 cases (23%) (excluding cases of acute AD and descending aorta surgery).

Discussion

In a referral-based CV registry spanning approximately 60 years, a number of notable findings are identified. Firstly, in cases under medical care at time of death, the majority of cases are not diagnosed premortem, and this did not differ between early and more recent time periods. Secondly, the most common risk factors for AD include hypertension, prior CV surgery, and bicuspid aortic valve, which have not changed significantly over the study time interval. Thirdly, an intimal tear was identified in the ascending aorta in the vast majority (68%) of patients. Finally, the cause of death in dissection was most frequently due to aortic rupture, which most frequently occurs in the ascending aorta.

Historically, aortic dissection was often a diagnosis first recognized at autopsy or surgery for a separate condition.[3] In autopsy series, the recognition of AD premortem is widely variable and depends on the population studied and the definitions used for diagnosis. Of the patients that were under medical care at the time of their demise in this series, 62% were not recognized prior to autopsy, and this did not differ in the current era. In a recent single-institution series, Roberts et al report that, in 23%, the diagnosis of AD was made at autopsy or at surgery for another condition and suggest that this is likely an underestimate given the relatively low number of autopsies currently performed.[14] Spitell et al, in a case series from the Mayo clinic over a 10-year period, revealed that AD was diagnosed at autopsy in 17 cases (28% of the series).[15] In the current era when autopsies are less common as autopsy rates have declined by 50% over the past 40 years,[14,16] it is unknown what variables influence physicians to order autopsies beyond an unexpected death. Many of the cases in this registry are generated by medical examiner's cases, and thus, strict comparison of percentages to tertiary hospital autopsy cohorts is not possible, but despite this bias, it is notable that AD is first recognized at autopsy in the majority of cases.

When compared to contemporary clinical series, the characteristics of affected cases in this autopsy series are similar including Stanford classification (86% type A), age, and gender.[1] Considering LVH as a hypertension equivalent, undertreated hypertension remains an important risk factor for aortic dissection as noted in prior clinical and autopsy series.[1-4,17,18] Over one-third of this series had a history of prior CV surgery, which is much higher than the 18% in the International Registry of Acute Aortic Dissection database (1996-1998) and may reflect a referral basis of this autopsy registry.[1] The higher prevalence of prior surgery may also relate to less typical presentations and delays (or lack thereof) in recognition and treatment.[10,18] A heightened index of suspicion for AD in these patients is necessary given less typical presentations.[18] This cohort with prior CV surgery demonstrates that AD may occur at highly varied intervals post aortic and other cardiac surgeries in some cases related to preexisting aortopathies in cases of repaired coarctation and AVR.[18] In other cases, the instrumentation from cannulation or bypass grafts was the nidus for subsequent AD.[18] Although some have suggested that AD

patients with prior CV surgery are less prone to rupture, we have confirmed in this series that rupture is indeed the cause of death in a significant proportion of these cases.[19]

A bicuspid aortic valve was found in a significant subset (14%; 4 of whom had undergone cardiac surgery) and is similar to that found in an autopsy series of type A AD[2] though more than reported in most clinical series[16,20] and more likely to be associated with type A AD.[6] Marfan syndrome was noted in about 4% of the group, similar to earlier reports.[1-3] Patients with connective tissue disease, congenital disease, and bicuspid valve were younger, as reported previously.[2,20] Less common conditions associated with dissection including aortic coarctation and aortitis were noted rarely.[2-4] Medial degeneration, a marker of inherent weakness of the aortic wall related to many of the aortopathies,[3] was described in approximately half of the cases and more prevalent in type A than type B.

AD site and complications

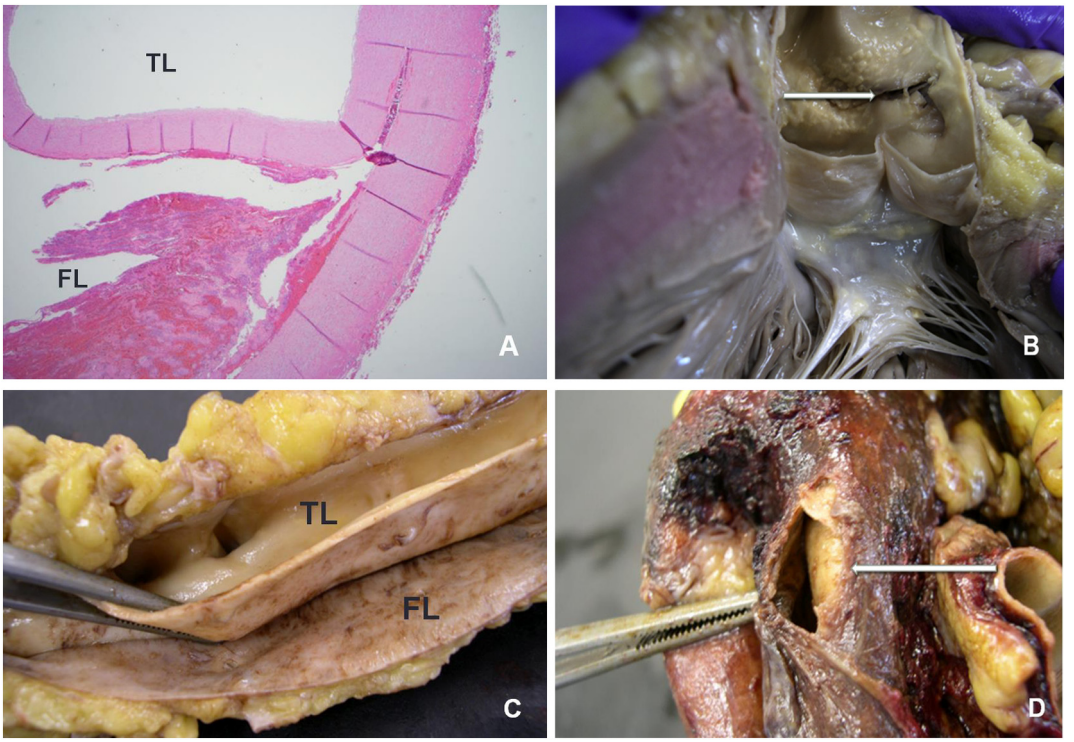
These pathologic findings provide understanding of the clinical presentation and the cause of rapid demise in many patients with AD. The intimal tear, the inciting event in the progression of AD, is located most frequently in the ascending aorta and was identified in each case[2-5,21] (Figure 1, *A-C*). Death in type A AD has been estimated to occur at rate of 1% hourly and as shown here is most frequently related to fatal rupture of the proximal aorta[3,4,17] (Figure 1, *D*). Only timely recognition and treatment can prevent this catastrophic event. Of the cases with documented aortic rupture, rupture into the pericardium was most common as seen in nearly two-thirds of the cases, followed by rupture into pleural space. This pattern of rupture is similar when compared to data previously seen in prior autopsy series.[3,5,22] The dissection flap may involve and lead to ischemia of the coronary, cerebral, mesenteric, or peripheral extremities as described in earlier series.[3] Clinicians caring for AD patients recognize that these patients frequently suffer from multiorgan complications as shown in this series including acute coronary syndromes, cardiac arrest, gastrointestinal bleeding, and sepsis.

Although AD is usually fatal if untreated, some patients survive as demonstrated by healed dissections which did not play a role in the patient's death occurring in 7% of the total cohort (including 20 with type A), which is also consistent with earlier series.[3,4] Aortic dissection localized to the ascending aorta (DeBakey type II dissection) was more frequently observed (26%) than has been reported in clinical series but similar to prior autopsy series and potentially relates to a referral basis toward autopsy with this subgroup less likely to have branch vessel involvement and therefore typical manifestations of AD.[2,9,23]

Several factors which influence the diagnosis and treatment of aortic dissection have significantly evolved over the past 60 years. Hypertension likely plays a causal role in the initiation of the intimal tear and intramural hematoma formation and remains the most important risk for AD, despite an abundance of evidence and society-based recommendations for treatment.[24] Imaging techniques which establish the diagnosis have been developed and evolved over the study period. Until the advent of aortography in 1939, aortic dissection was almost exclusively a postmortem diagnosis. Aortography was initially the primary diagnostic test for AD; however, in the early 1990s, there was a fundamental change in the imaging tools used to diagnose dissection with rapid expansion of the noninvasive techniques of echocardiography, CT, and magnetic resonance imaging.[7,13] Our series was consistent with this paradigm shift, as imaging modalities shifted from chest radiograph/electrocardiogram/aortography being the primary diagnostic modalities used in the early cohort compared to CT and echocardiography being more commonly applied in the current cohort.[25] Aside from diagnostics, the study period saw the advent of CV surgery which was revolutionized in the 1950s and 1960s with the invention and refinement of the cardiopulmonary bypass circuit, and the first AD surgery by DeBakey occurred in the 1950s.[9,26] Surgical strategies for type A AD have continued to evolve, and surgical risk has decreased with improvements in surgical techniques, cardiac anesthesia, cerebral protection, and perioperative care.[8,25] Endovascular stents treatment for type B is also being increasing used but were not seen in the current series.[25]

In 1956, Hirst discussed the improved recognition of dissection in that era compared to previous historical data and the importance of considering the diagnosis with a variety of clinical scenarios.[3] The clues to the diagnosis, including symptoms of sudden severe pain, in conjunction with physical examination findings of diastolic murmur or neurologic findings, especially in patients with conditions known to increase risk, have been recognized since the 1950s.[3] Current guidelines have emphasized that these elements should be combined by clinicians with the aim of improving recognition.[8,27-29] However, despite greatly enhanced access to highly sensitive noninvasive imaging tools in the current era leading likely to increased recognition of predisposing conditions, these data suggest that imaging is still underused at the time of medical presentation in a notable number of cases. From a clinical standpoint, the fact that many aortic dissections remain unrecognized at time of death speaks to the need for heightened suspicion of AD in patients presenting with symptoms, risk factors or physical findings.[8,10] Educational efforts focused on recognition of aortic dissection have improved times to recognition and treatment of AD, especially at referral hospitals where clinicians may less commonly encounter the diagnosis.[30] Patient advocacy groups such as

Figure 1



A, Microscopic H&E stain of aortic dissection showing true (TL) and false lumen (FL) containing thrombus.**B**, An intimal tear is visualized in the proximal ascending aorta immediately above the left and noncoronary sinotubular ridge.**C**, An aortic dissection is seen with the true (TL) (top) and false lumen (FL) visualized.**D**, Adventitial tear with aortic rupture (of ascending aorta).

Thoracic Aortic Disease Coalition, John Ritter Foundation, and others also focus efforts on recognition of AD by both medical professionals but also for patients in higher-risk groups to recognize symptoms at onset and advocate for their own testing when necessary.

Aortic intramural hematoma (IMH) is a variant of AD in which a hematoma in the aortic media is visualized by imaging in the absence of an intimal tear or false lumen.[31] Within this series, cases of IMH likely either progressed to a typical AD or ruptured as a significant percentage of IMH cases are prone to do. In the early years of this series, one case of ascending AD and rupture had presented the day prior with chest pain and was noted to have "aortic wall thickening" and pericardial effusion only to become hypotensive and arrest the following morning. Although clinical information was often not complete, it is possible that other cases had IMH as well. Some of the healed dissections, particularly those in the descending aorta, may have been IMH.

Limitations of the study include the fact that as this is an autopsy study and it is retrospective in nature with variable amount of clinical data provided by the primary pathologist. Furthermore, the cases examined at the Jesse E. Edwards Registry are referred based on individual provider discretion and reflect a referral bias. It is unknown which factors lead a clinician to recommend that the family consider an autopsy for a hospitalized patient and whether there is inherent bias in the differences over time. Despite these limitations, this study provides valuable information regarding aortic dissection over the last 60 years.

Appendix. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ahj.2018.11.006.

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