Aortic disasters

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“There is no disease more conducive to humility than aneurysm of the aorta.”—Sir William Osler [1]

Thoracic aortic dissection (TAD) and abdominal aortic aneurysm (AAA) are two of the most lethal and high-risk clinical entities in the practice of emergency medicine. Both aortic diseases are associated with high morbidity and mortality rates if the diagnosis is missed or delayed on initial presentation, and both have been shown in multiple studies to manifest in subtle and atypical presentations. Acute disease of the thoracic or abdominal aorta may cause a myriad of symptoms, such as stroke, shock, ischemia of limbs or other organs, chest and back pain, spinal cord ischemia, and flank pain. Emergency physicians should be experts not only in the recognition, treatment, and disposition of patients with acute aortic emergencies, but also in the detection of atypical cases.

This article discusses the subtle nuances of two specific aortic emergencies, TAD and AAA. This article highlights reasons these diagnoses are missed or delayed, reviews some noteworthy subtle and atypical cases, reviews important medicolegal issues, and discusses practical ways to reduce the risk of litigation. Risk management strategies are highlighted to emphasize ways to decrease the risk of missing the diagnosis and the subsequent risk of litigation. Emergency physicians cannot diagnose every case of TAD and AAA, but what they can do is increase their chances of detection and reduce their risk of litigation. This article discusses straightforward tools that can be used to accomplish this. Ultimately, this strategy provides protection for patients and physicians.
Thoracic aortic dissection

Dissection of the thoracic aorta may be one of the most challenging diagnoses to make in clinical medicine today and is responsible for many lawsuits against emergency physicians [2]. More than 2000 cases of TAD occur every year in the United States, and it is the most common acute illness of the aorta [3]. Because hypertension and age are significant risk factors, it would make logical sense that more cases of this deadly entity will be seen as the population ages. Most cases occur in patients 50 to 70 years old with a male predominance. Ascending aortic dissection left untreated carries with it a 75% mortality rate within the first 2 weeks alone [3].

Estimated mortality rates are 1% to 2% per hour for the first 24 to 48 hours [2]. Autopsy series conducted in the 1950s before modern treatment modalities were available revealed a 40% to 50% mortality rate within the first 48 hours [4]. The most common misdiagnoses discussed in the literature include myocardial infarction, atypical chest pain, congestive heart failure, and gastrointestinal bleeding [5]. In data published by Viljanen [6], myocardial ischemia was found to be the number one diagnosis among missed TAD cases.

This discussion aims to answer the following important questions regarding this deadly diagnosis: (1) Are there features of the history and physical examination that are helpful in increasing diagnostic accuracy? (2) Why is the diagnosis of TAD delayed or missed? (3) What are some atypical presentations that should make the emergency physician entertain the diagnosis of TAD? (4) What tools can be used to improve documentation in the chest pain patient, and what should emergency physicians do to reduce risk if the diagnosis is missed?

Usefulness of the history and physical examination

Traditional teaching is that most diagnoses in medicine are made by performing a thorough history and physical examination; in the chest pain patient, this could not be more true. Sixteen studies of more than 1500 patients with TAD looked at the various sensitivities of the history. The pooled sensitivity of pain is approximately 90%. Patients with TAD usually have acute-onset pain that is maximal at the beginning. Sudden-onset chest pain has been shown to have a sensitivity of approximately 84%. Absence of sudden pain has a negative likelihood ratio of 0.3. Pain that is described as “ripping” or “tearing” has been shown to have a likelihood ratio of 1.2 and 10.8 [7,8]. In many cases, there also is characteristic radiation of the pain into the neck, arm, or back and abdomen.

Despite the fact that the history often can mislead the emergency physician, there is some evidence that a better history may increase the diagnostic yield. A retrospective chart review of 83 patients with TAD revealed that only about 40% of alert patients were asked the basic questions about their pain—quality, radiation, and intensity at onset. When all three questions...
were asked, the diagnosis of TAD was made in 91% of the cases [9]. Von Kodolitsch et al [8] devised a clinical prediction model for the initial prediction of TAD based on history, physical examination, and chest radiography. This study of 250 patients with acute chest pain, back pain, or both and a clinical suspicion for TAD investigated 26 clinical and radiographic variables. The assessment of these variables permitted the identification of 96% of TAD cases [10]. Box 1 lists independent predictors of TAD [10]. Finally, TAD should be strongly considered in patients with chest pain who are not responding to the usual therapy for acute coronary syndrome.

An analysis of missed TAD litigation by Sullivan [11] noted a common pattern: failure to take and record a risk factor profile for TAD. Sullivan performed a detailed analysis of more than 2500 presentations of diseases that have known, associated risk factors (eg, aortic dissection, myocardial infarction, pulmonary embolism, ectopic pregnancy) and found that risk factors were inquired about in only 432 of the cases. Failure to ask about specific risk factors represents an opportunity to increase the rate of detection of these diseases, particularly TAD [11]. The most important risk factors include hypertension, male gender, advanced age, connective tissue disease, and pregnancy [12]. Other lesser known risk factors include bicuspid aortic valve, Turner syndrome, weight lifting, and methylenedioxymethamphetamine (MDMA or “ecstasy”) use [13]. To convince anyone, especially a jury, that a differential diagnosis was considered in the chest pain patient, a detailed risk factor assessment should be performed and documented. The performance of this detailed analysis gives the impression that the emergency physician was thorough in the approach to the patient. In some cases of missed TAD, a verdict for the defense has been rendered based on the performance of a thorough risk factor assessment.

Physical examination findings that should be looked for and documented include the following: (1) difference in pulse amplitude between arms, (2) presence of aortic diastolic murmur, and (3) blood pressure differentials between arms. For many years, measurement of bilateral blood pressures has been considered an important component of the workup of a patient with suspected TAD. Some authors have recommended that this measurement be performed in triage as a risk management tool. Older studies that designated a difference of more than 20 to 30 mm Hg between arms were based on autopsy series and were arbitrary. A prospective study by Von

**Box 1. Independent predictors of thoracic aortic dissection**

- Chest pain of immediate onset
- Tearing or ripping quality of pain
- Pulse differentials
- Mediastinal widening on chest x-ray
Kodolitsch et al [8] did find, however, that a blood pressure differential of greater than 20 mm Hg was an independent predictor of TAD. Two more recent studies by Lane et al [14] and Pesola et al [15] indicated, however, that 20% of people may have significant interarm blood pressure differentials. Although a discernable pulse amplitude difference and greater than 20 mm Hg difference in blood pressure should increase suspicion of TAD, they do not rule it in. Nevertheless, Sullivan [11] suggested that the performance of bilateral arm blood pressure measurements proves that the emergency physician was clearly searching for TAD and that a broad differential diagnosis was being considered. Traditionally, aortic dissection is seen in patients with markedly elevated blood pressure; however, 25% of TAD patients present with an initial systolic blood pressure of less than 100 mm Hg [16].

Age

A common theme in many cases of missed myocardial infarction and TAD is that the patient is young and may have no obvious, known disease-specific risk factors. One of the most famous cases of a young person who died from an aortic dissection was Jonathan Larson, Tony award–winning writer of the Broadway play Rent. Larson had visited the emergency department twice in 2 days, once with the complaint of chest pain and the second time with abdominal pain. He was found dead in his apartment the night before Rent opened on Broadway. He was 35 years old. Young people can and do have acute aortic disease. Although Larson’s chest x-ray later was interpreted as abnormal, the diagnosis of aortic disease probably was not entertained because he was young and healthy and had no obvious physical examination evidence of a connective tissue disease. It was later determined that Larson had Marfan disease.

Emergency physicians should exercise caution when dealing with young patients with chest pain. Although physical examination may not reveal stigmata of overt connective tissue disease, other risk factors, such as family history, presence of hypertension, and cocaine use, should be considered. All patients, regardless of age, should have the diagnosis of TAD entertained if they present with chest pain.

Pregnancy

The association of aortic dissection and pregnancy has been well described in the literature. It is estimated that 50% of all cases of TAD in women younger than 40 years old are associated with pregnancy [17,18]. Various hemodynamic changes in pregnancy, such as increases in heart rate and stroke volume, along with elevated progesterone and estrogen levels, have been shown to induce histologic changes in the arterial wall that may predispose to dissection [19].
Emergency physicians should consider the diagnosis in all pregnant patients who present with chest or back pain. Because back pain is relatively common during pregnancy, the diagnosis of TAD can be difficult. If a pregnant patient presents with a history concerning for TAD, however, the diagnosis should be pursued. Pain that radiates from the upper to lower back, especially in association with hypertension, should be considered suspect and worked up. The key to detecting TAD in this patient population starts by simply thinking about the diagnosis.

Why is the diagnosis missed?

In many cases, there simply is no way to make the diagnosis of TAD during the initial patient presentation. Manifestations may be subtle, and TAD may masquerade as other diseases. Classic symptoms may be lacking, and patients simply do not report any symptoms that trigger the workup of aortic disease. Case reports of TAD presenting as a stroke, coagulopathy, and gastrointestinal bleeding have been reported. In cases such as this, a definitive diagnosis of TAD simply cannot be made.

Evaluation of risk factors as previously discussed has been shown in several different disease entities to increase the rate of diagnosis. A sound risk management strategy for all chest pain patients is to take and record a detailed risk factor analysis for TAD and for coronary artery disease and pulmonary embolism. Family history of aortic disease and presence of any connective tissue disease should be sought. Hypertension has been shown to be a substantial risk factor and should be assessed carefully. Box 2 lists TAD risk factors [17,18].

Failure to appreciate classic signs and symptoms and poor knowledge base may lead to delays in diagnosis. Certain pain radiation patterns, such as substernal chest pain radiating into the back or abdomen, may not be considered indicative of TAD. Numerous cases have been reported in which classic, “textbook presentations” were not recognized.

Patients with TAD may present with signs and symptoms that seem separate and unrelated. An example is a patient with chest pain and left leg numbness or chest pain and visual problems. The treating physician neglects

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<th>Box 2. Thoracic aortic dissection risk factors</th>
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<td>Hypertension</td>
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<td>Age</td>
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<td>Pregnancy</td>
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<tr>
<td>Bicuspid aortic valve</td>
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<td>Coarctation of the aorta</td>
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to attempt to integrate the two seemingly disparate complaints or falsely assigns a preliminary diagnosis that does not explain the patient’s symptoms; this has been referred to by some as failure to perform complaint and sign integration [11]. Because acute aortic disease may present with a myriad of signs and symptoms, emergency physicians should be wary and search for chest pain, back pain, or abdominal pain in association with other findings, such as stroke symptoms, limb ischemia, or a combination of chest and back pain [11]. Box 3 summarizes risk management recommendations, and Box 4 lists causes of missed or delayed TAD diagnosis [11].

Subtle and atypical presentations

The classic, textbook description of acute, severe, tearing substernal chest pain radiating into the back in association with hypertension seems to be the exception rather than the rule. Classic textbook descriptions are based on population-based studies and frequently seem irrelevant in an individual patient. Box 5 lists atypical presentations of TAD. Symptoms in patients with TAD are more variable than previously thought, and classic findings are often absent [7]. Chest pain that lacks the classic radiation pattern may lead to delays in diagnosis. Further complicating matters is the fact that patients with TAD presenting without pain have been described. In a study of 236 cases of confirmed TAD, 15% (35 of 236) were painless. Fig. 1 is a CT scan of patient with painless TAD (CT scan shows intimal flap only). There was a failure to make the diagnosis before death in 28% (66 of 236) of

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**Box 3. Risk management recommendations for thoracic aortic dissection**

1. Perform a detailed risk factor analysis for every chest pain patient.
2. Perform a thorough physical examination focusing on thoracic aortic dissection findings—pulse deficits, blood pressure differential, diastolic murmur of aortic regurgitation, and neurologic abnormalities.
3. Understand the limitations of the chest x-ray.
4. Practice complaint and sign integration (the diagnosis must explain the patient’s presentation).
5. Understand that many cases may be subtle and atypical.
6. Do not exclude lethal aortic diseases such as thoracic aortic dissection in young patients.
7. Documentation should reflect a diligent search for life-threatening chest pain etiologies, particularly thoracic aortic dissection.
the cases [17]. Delays in diagnosis seem to be common. In a study of 84 TAD patients by Viljanen [6], there was a delay of more than 24 hours in 31% of the proximal TAD cases and in 53% of the distal aortic dissections [6].

Cardiac complications of TAD are particularly common and include aortic regurgitation (found in 18–50% of cases). Acute aortic regurgitation is the second most common cause of death in patients with TAD after rupture into the pericardium with subsequent tamponade. Myocardial infarction is a relatively rare complication of proximal aortic dissection and is estimated to occur in approximately 1% to 7% of all TAD cases. If the coronary ostia do become involved, lesions of the right coronary artery leading to inferior ST-segment elevation myocardial infarction are most common [20,21]. An illustrative case is that of a 51-year-old man who presented with left-sided chest pain and diaphoresis. His electrocardiogram (ECG) on presentation showed ST-segment depression in the inferior leads, suggesting myocardial ischemia. He was treated with heparin, aspirin, and a glycoprotein IIb/IIIa inhibitor and had the diagnosis of proximal TAD made at the time of cardiac catheterization (Fig. 2). Fig. 3 is an ECG showing T-wave inversions in the inferior leads in a young man with cocaine-associated TAD.

One presentation that warrants mentioning is isolated substernal chest pain that occurs without the characteristic radiation pattern. This type of presentation most likely occurs secondary to the formation of an intimal tear in the proximal aorta without dissection. Without distal dissection, pain may be confined to the anterior chest and lead to a delay in diagnosis. Cases

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<th>Box 4. Causes of missed or delayed thoracic aortic dissection</th>
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<td>Inadequate knowledge base</td>
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<td>Failure to detect a classic presentation</td>
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<td>Failure to perform a detailed risk factor profile of thoracic aortic dissection</td>
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<td>Failure to integrate the patient’s signs and symptoms</td>
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<th>Box 5. Atypical presentations of thoracic aortic dissection</th>
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<tbody>
<tr>
<td>Painless</td>
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<tr>
<td>Isolated abdominal pain</td>
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<td>Stroke</td>
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<td>Paralysis</td>
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<td>Syncope</td>
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such as this may be detected by transesophageal echocardiography [22]. In particular, cases of cocaine-induced TAD may lack many of the typical pain referral patterns and resemble an acute coronary syndrome or some other chest pain entity.

Painless TAD may present as syncope and is thought to occur secondary to acute cardiac tamponade. The emergency physician must generate a differential diagnosis rapidly in patients presenting with syncope. Entities that should be considered in the differential diagnosis include myocardial infarction, pulmonary embolism, gastrointestinal bleeding, and central nervous system lesions or hemorrhage. The diagnosis of TAD should be entertained early because rapid bedside ultrasound may detect a hemorrhagic pericardial effusion and cardiac tamponade [23]. Fig. 4 shows an echocardiogram of a patient with syncope secondary to proximal TAD and cardiac tamponade.

TAD in some cases may cause extremity ischemia. A useful acronym proposed by Pacifico and Spodick [23] is ILEAD—ischemia of the lower extremity in aortic dissection [24]. Patients who present with evidence of extremity ischemia in association with chest pain should have the diagnosis of TAD entertained.

Cases of aortic dissection have been noted to have a frequent association with mild neurologic symptoms. Neurologic deficits are found in approximately 18% to 30% of cases [25]. In some cases, neurologic deficits may occur without any chest pain. Neurologic presentations include acute stroke, bilateral lower extremity paralysis secondary to dissection involving the greater radicular artery, unilateral lower extremity numbness, and hoarseness [26–30]. Cerebral ischemia and stroke is the most common neurologic manifestation associated with TAD and has been reported in 5% to 10% of patients. Spinal cord involvement in TAD has not been described. However, the incidence of spinal cord injury in patients with TAD is unknown.
Fig. 2. Case 1—a 51-year-old man with left-sided chest pain and diaphoresis. Baseline electrocardiogram (ECG) (A) and ECG on presentation (B). The patient was treated with aspirin, β-blockers, and heparin. The patient decompensated and was found to have a proximal thoracic aortic dissection and right coronary artery occlusion by cardiac catheterization.
Fig. 3. Case 2—a 34-year-old man with abdominal pain. The patient developed chest pain 1 hour after arrival. Electrocardiogram shows T-wave inversions in the inferior leads. Right coronary artery occlusion was found in the operating room during repair.
syndromes, including transverse myelitis and spinal cord infarction, and may present with paraplegia or quadriplegia [31]. These spinal cord syndromes tend to occur more often in cases of distal TAD. The diagnosis of TAD should be entertained in any patient with neurologic signs and symptoms, especially if they occur in conjunction with chest pain. Many cases of missed TAD could have been diagnosed if the treating physician would have attempted to integrate the patient’s signs and symptoms.

Isolated abdominal aortic dissection has been described previously in case reports. Farber et al [32] evaluated 10 cases of aortic dissection confined to the abdominal aorta. Only five patients were hypertensive on presentation. In a study by Hsue et al [33], patients with cocaine-associated TAD were more likely to have distal aortic dissection and to present with abdominal pain. In a study of 398 cases of TAD, Hirst et al [34] showed that 2.5% were confined to the distal thoracic and abdominal aorta. Fig. 5 shows a CT scan of a patient with isolated abdominal pain after cocaine use. The CT scan shows a Sanford B TAD. TAD and abdominal aortic dissection may present with isolated abdominal pain. Considering the diagnosis, particularly in younger, hypertensive patients with risk factors, such as cocaine, may make the difference between diagnosis and a poor outcome.

**International Registry of Aortic Dissection study**

One of the largest studies to date to examine the clinical characteristics of TAD cases was the International Registry of Aortic Dissection (IRAD) performed by Hagan et al [4]. In this study, 464 cases of TAD were examined for the history, physical examination, and management. Of these cases, 62.3% were proximal dissections. Classic physical examination findings, such as the murmur of aortic regurgitation and a pulse deficit, were
detected in only 31.6% and 15.1% of cases. Traditionally, it is thought that the chest x-ray would be abnormal in cases of TAD. In this study, 12.4% of chest x-rays were interpreted as completely normal. Patients with TAD frequently present without a classic history and physical examination. Although from a risk-management standpoint it makes good sense to examine and document the presence or absence of an aortic murmur and a pulse deficit, they are unreliable findings [4,6]. Box 6 lists suggestive findings of TAD; see also Box 1 earlier for independent predictors of TAD.

**Abdominal aortic aneurysm**

AAA is a high-risk clinical entity that may present in many different ways. Presentations ranging from asymptomatic, to flank pain simulating renal colic, to peritoneal rupture and shock all have been described. Some patients initially may be hemodynamically stable, allowing the emergency physician to make a definitive diagnosis before aneurysmal rupture occurs.

The elderly population is especially at risk for AAA. Some authors estimate that 4% to 8% of all patients older than age 65 have an AAA [35]. Ruptured AAA is the 13th most common cause of death, leading to an estimated 10,000 to 15,000 deaths/year in the United States. Of patients with

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<th>Box 6. Suggestive findings of thoracic aortic dissection</th>
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<tr>
<td>Chest pain and neurologic abnormalities</td>
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<tr>
<td>Chest pain in association with back or abdominal pain</td>
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<td>Chest pain that radiates into the back</td>
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Fig. 5. Abdominal CT scan shows dissection of the abdominal aorta in a 34-year-old hypertensive man with abdominal pain after cocaine use.
AAA, 40% to 50% die before they reach the hospital, and the overall mortality rate of a ruptured AAA is greater than 90% [35]. There is a marked increase in mortality when the diagnosis is delayed or missed [36–38]. In one study, the mortality rate was 35% when the diagnosis was at least initially suspected, whereas the mortality rate was 75% when there was no suspicion before the diagnosis was made [38].

AAA is more common in people in their 60s and 70s, but younger patients, especially ones with underlying connective disease, are at increased risk as well. Because rupture, either into the retroperitoneal space or into the peritoneum, may cause rapid patient decompensation and death, emergency physicians should consider this diagnosis early in all patients who are at risk who present with compatible signs and symptoms, such as unexplained abdominal, back, or flank pain [39].

**Usefulness of the physical examination**

The physical examination of patients with suspected AAA may be helpful but is fraught with problems. If rupture has occurred in the retroperitoneum or if the patient is obese, the examination may appear normal. One of the most helpful findings is detection of an abnormally widened aortic pulse [40]. Detection of the aortic pulse to the right of the midline also has been shown to be useful. Caution should be exercised because only 76% of aneurysms greater than 5 cm are palpable [40–42]. Box 7 lists physical examination findings of AAA [11].

**Why is the diagnosis missed?**

Sullivan [11] reviewed in detail the reasons physicians miss the diagnosis of AAA. Sullivan previously reviewed litigation against emergency physicians and highlighted several reasons there is either a delay or a missed diagnosis. One of the principal reasons for a delay in diagnosis is that the classic triad of abdominal or back/flank pain, hypotension, and a pulsatile

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**Box 7. Physical examination findings of abdominal aortic aneurysm**

- Unexplained hypotension or altered level of consciousness
- Presence of pulsatile abdominal mass
- Aortic pulsation to the right of the midline
- Left lower quadrant abdominal mass with tenderness and distention
- Abdominal bruit
- Absent or diminished lower extremity peripheral pulses
- Cyanotic toes
402 abdominal mass is relatively rare and has been estimated to occur in only
403 30% to 50% of patients [44]. In addition, AAA commonly masquerades as
404 other common problems, such as renal colic or diverticulitis. Lastly, AAA
405 can cause many signs and symptoms that suggest a disorder in another
406 system. AAA may cause urologic, gastrointestinal, or vascular symptoms.
407
408 Risk factor assessment plays an important role not only in evaluating the
409 patient, but also in convincing potential juries that the emergency physician
410 was thorough in the approach to patient care. Cases have been identified
411 that lacked a thorough risk factor analysis, which subsequently led to
412 plaintiff verdicts. What can the emergency physician do to decrease poten-
413 tial risk? AAA rupture is a diagnosis that must be approached with a high
414 index of suspicion because history has shown that a significant proportion
415 of diagnoses are delayed or missed. Eliciting a thorough history should
416 enable emergency physicians to risk stratify patients and know when even
417 low-risk patients need a prompt evaluation. Patients with atherosclerotic
418 disease also are at risk for aneurysm formation. Advanced age is a well-
419 established risk factor; AAA rarely occurs in patients younger than age 50.
420 The disease also is more prevalent in men, smokers, and hypertensive
421 individuals. The strong familial association may be less well known. Webster
422 et al [44] found that that 39% of patients with AAA have first-degree
423 relatives with this condition. The failure to elicit a personal history of
424 aneurysm in a patient who presents with a history consistent with rupture
425 could be detrimental. Finally, patients with connective tissue disorders, such
426 as Ehlers-Danlos syndrome and Marfan syndrome, must be identified and
427 recognized for their increased risk because of vascular structural defects,
428 particularly of the aorta. Box 8 lists AAA risk factors [54].

429 A common theme in litigation against emergency physicians relates to the
430 failure to integrate the patient’s signs and symptoms. Consider the following
431 case: A 64-year-old man presented with a chief complaint of abdominal pain
432 and rectal bleeding. Before he presented to the hospital, he had a syncopal
433 episode at home in the bathroom. Physical examination revealed periumbil-
434 ical and left lower quadrant abdominal pain. He was admitted to the

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**Box 8. Risk factors for abdominal aortic aneurysm**

- Hypertension
- Atherosclerotic vascular disease
- Connective tissue disease
- Family history in first-degree relative
- Advanced age (mean age of diagnosis 67)
- Smoking
- Diabetes
- White race

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hospital with a diagnosis of abdominal pain and possible gastrointestinal bleeding. In the hospital, the patient had a ruptured AAA and died. A couple of things can be learned from this case. First, there was no attempt to integrate the abdominal pain, syncope, and bleeding. What did the treating physician think was the cause of the severe abdominal pain? Second, any elderly patient with abdominal pain, particularly if associated with syncope, should have a rapid evaluation to rule out a ruptured AAA. The diagnosis of AAA should be entertained in all older patients with syncope, even if an alternative diagnosis is more likely. Box 9 is an overview of AAA litigation [11].

Must-know presentations

It has been known for many years that AAA may masquerade as renal colic. Urologic symptoms are estimated to be present in 10% of patients with AAA and are estimated to be the most common misdiagnosis for this group of patients [36]. The mechanism for the development of urologic symptoms is thought to be due to irritation of the retroperitoneal ureter or direct compression of the ureter by the expanding or rupturing aneurysm [46]. Among patients older than age 65 who were referred to urologists for workup of renal colic, 10% were found to have an AAA [47]. All older patients and patients with connective tissue disease who present with flank/back pain or signs and symptoms of nephrolithiasis should have AAA ruled out. Chart reviews and retrospective analyses have shown repeatedly symptomatic or ruptured AAAs to be misdiagnosed as genitourinary pathologies. One review found that 7 of 23 patients with AAA were diagnosed erroneously with urinary obstruction, infection, or stone [41,48]. Another study discovered 10 of 44 patients with AAA who had received the same inappropriate diagnosis [49]. Although the pain associated with AAA is typically constant and localized to the abdomen, numerous reviews detail histories of radiation to one or both flanks and colicky-type pain. Also, emergency physicians should not be misled to a diagnosis of ureteral colic by the presence of microscopic hematuria because it can be seen in ruptured AAA. To avoid potential catastrophic misdiagnoses and litigation, any

Box 9. Overview of abdominal aortic aneurysm litigation

- Poor risk factor analysis
- Poor recognition of flank pain ("kidney stone") presentations
- Failure to obtain imaging studies to evaluate the aorta
- Delay in proper management
- Failure to recognize presentations such as syncope
- Failure to consult a vascular surgeon or arrange timely outpatient follow-up
elderly patient with flank pain and suspected genitourinary disease should be evaluated for aortic pathology. In most cases, this evaluation involves performing an abdominal CT scan or ultrasound. Fig. 6 shows a CT scan of a patient who presented with flank pain and hematuria. Box 10 lists unusual presentations of AAA.

Five percent of symptomatic AAA patients present with neurologic symptoms [50]. A history of unexplained syncope in an older patient should make one think of the diagnosis of AAA [50]. Often, syncope is associated with some other finding, such as heme-positive stools, causing the physician to conclude that a gastrointestinal bleed is the most probable diagnosis. Expansion of the retroperitoneal hematoma also can cause nerve root compression with resultant neuropathy. Anatomically the nerves most susceptible to this compression and ischemia are the femoral and obturator nerves. Although it is a rare occurrence, a ruptured AAA should be considered in the differential diagnosis of patients with peripheral neuropathy in the lower extremity and especially anterior thigh pain and numbness associated with weakness of hip and knee flexion [51,52]. An appreciation of this relatively rare, but well-described presentation may make the difference between a diagnosis and a delay in diagnosis and subsequent poor outcome.

AAA frequently is misdiagnosed as diverticulitis, and it is estimated that 12% of AAAs are diagnosed initially as diverticulitis [36,41,48,49]. Most AAAs rupture into the left retroperitoneum or peritoneum and may lead to tenderness or a left lower quadrant mass. In cases in which diverticulitis is in the differential diagnosis, emergency physicians should consider the diagnosis of AAA. With more widespread availability and liberal use of CT, it would seem logical that the rate of this misdiagnosis would decrease. Patients with suspected diverticulitis, particularly patients at high risk for

Fig. 6. Abdominal CT scan shows large abdominal aortic aneurysm in a 70-year-old man who presented with left flank pain and hematuria.
AAA, should have appropriate imaging studies performed in the emergency department.

AAA may cause a myriad of vascular presentations. Peripheral emboli to the lower extremities, leading to the “blue-toe syndrome,” are the initial manifestation of AAA in 5% of patients. This presentation may cause an ischemic, painful extremity or cyanotic toes due to atheroembolism. Emboli also may involve the mesenteric and renal arteries, leading to intestinal ischemia and hematuria and renal failure [53].

A multitude of risk management recommendations have been described by various authors. The first is to consider the diagnosis of AAA in all patients at risk for the disease, especially if older than age 65 years and if connective tissue disease is present. High-risk presentations of this disease include unexplained syncope, abdominal pain, diaphoresis, flank/back pain, and unexplained hypotension and shock. The most common misdiagnoses that have been reported in the literature include renal colic and diverticulitis. The emergency physician should have a low threshold to perform an ultrasound or CT scan on all older patients who present with abdominal pain to exclude the diagnosis.

Having an awareness of and respect for the difficulty of diagnosing symptomatic AAA is key to understanding the steps that must be taken urgently to avoid missed and delayed diagnoses. Patients in whom AAA is considered should be given high priority for a thorough history and rapid physical examination and further diagnostic evaluation. Because these patients have a great potential for a poor outcome, the case should be documented carefully. Specific aspects of the search, such as the evaluation for a pulsatile mass on abdominal examination, must be included so that the thought process of the physician can be realized retrospectively. It should be clear from the medical record that the emergency physician entertained the diagnosis of AAA and took appropriate steps to rule it out. Anyone who reviews the chart should be able to see that a diligent search for an AAA was made. Box 11 summarizes risk management recommendations [54].

As highlighted earlier, numerous retrospective studies have shown AAA to be misdiagnosed as other disease processes, whether urologic, gastrointestinal, or musculoskeletal in origin. Physicians repeatedly have been misled by patient presentations or laboratory findings that were more
Hematuria may lead to the premature diagnosis of renal colic. Emergency physicians must remain constantly vigilant in searching for the correct diagnosis and avoiding the pitfalls that lead to detrimental events and litigation. Patients with recognized risk factors or whose presentation suggests AAA must be evaluated by one of the accepted methods—CT, ultrasound, or MRI. One cannot accept an alternative diagnosis without first confirming a normal aorta. Emergency medicine is a practice of ruling out and appropriately treating the processes that pose an imminent threat; determining the most likely diagnosis is a secondary concern.

**Issues with follow-up**

A review of case law by Sullivan [11] identified problems with referral and follow-up as a potential source of litigation against emergency physicians. Some cases have been filed secondary to the treating physician not recognizing the importance of timely follow-up. In most cases, this follow-up should be in the form of direct discussion with a vascular surgeon who is told of the diagnosis and the size of the aneurysm. Because risk of rupture is directly related to aneurysm size, emergency physicians should be aware of the potential risk of sending a patient home with instructions to “see your doctor.” If an AAA is discovered incidentally and is not thought to be symptomatic, instructions for follow-up are mandatory and time specific. Documentation of these expectations must be clear on the medical record to transfer some responsibility to the patient should he or she not comply and have a poor outcome. Timely surgery is crucial; operative mortality for unruptured aneurysms carries a mortality rate of less than 5%, whereas emergency surgery after rupture is greater than 50% [53].
Summary

TAD and AAA are two of the highest risk disease entities in emergency medicine. Emergency physicians should be vigilant in their approach to patients who have symptoms compatible with acute aortic disease. In chest and abdominal pain presentations, the chart must look like there was a search for the TAD and AAA. By having a sound knowledge of atypical cases; having an appreciation for how subtle TAD and AAA can be; and recording and documenting a thorough history, physical examination, and risk factor profile, the emergency physician may reduce substantially the risk of missing a diagnosis and subsequently being sued. Emergency physicians cannot diagnose every case of acute aortic disease; what they can do is practice with a sound understanding of risk management principles and consider these diagnoses in all patients with chest, back, or abdominal pain. Ultimately, this strategy would provide protection for the patient and the physician [54].

References


