



## 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.

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### Thoracic aortic dissection

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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.

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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?

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#### *Usefulness of the history and physical examination*

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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.

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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

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76 were asked, the diagnosis of TAD was made in 91% of the cases [9]. Von  
77 Kodolitsch et al [8] devised a clinical prediction model for the initial pre-  
78 diction of TAD based on history, physical examination, and chest radiogra-  
79 phy. This study of 250 patients with acute chest pain, back pain, or both  
80 and a clinical suspicion for TAD investigated 26 clinical and radiographic  
81 variables. The assessment of these variables permitted the identification of  
82 96% of TAD cases [10]. Box 1 lists independent predictors of TAD [10]. [Q6]  
83 Finally, TAD should be strongly considered in patients with chest pain who  
84 are not responding to the usual therapy for acute coronary syndrome.

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

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

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

- 113 Chest pain of immediate onset
- 114 Tearing or ripping quality of pain
- 115 Pulse differentials
- 116 Mediastinal widening on chest x-ray

117 Kodolitsch et al [8] did find, however, that a blood pressure differential of  
118 greater than 20 mm Hg was an independent predictor of TAD. Two more  
119 recent studies by Lane et al [14] and Pesola et al [15] indicated, however, that  
120 20% of people may have significant interarm blood pressure differentials.  
121 Although a discernable pulse amplitude difference and greater than 20 mm  
122 Hg difference in blood pressure should increase suspicion of TAD, they do  
123 not rule it in. Nevertheless, Sullivan [11] suggested that the performance  
124 of bilateral arm blood pressure measurements proves that the emergency  
125 physician was clearly searching for TAD and that a broad differential  
126 diagnosis was being considered. Traditionally, aortic dissection is seen in  
127 patients with markedly elevated blood pressure; however, 25% of TAD  
128 patients present with an initial systolic blood pressure of less than 100 mm  
129 Hg [16].

### 130 *Age*

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

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

### 150 *Pregnancy*

151 The association of aortic dissection and pregnancy has been well  
152 described in the literature. It is estimated that 50% of all cases of TAD in  
153 women younger than 40 years old are associated with pregnancy [17,18].  
154 Various hemodynamic changes in pregnancy, such as increases in heart rate  
155 and stroke volume, along with elevated progesterone and estrogen levels,  
156 have been shown to induce histologic changes in the arterial wall that may  
157 predispose to dissection [19].

158 Emergency physicians should consider the diagnosis in all pregnant  
159 patients who present with chest or back pain. Because back pain is relatively  
160 common during pregnancy, the diagnosis of TAD can be difficult. If  
161 a pregnant patient presents with a history concerning for TAD, however, the  
162 diagnosis should be pursued. Pain that radiates from the upper to lower  
163 back, especially in association with hypertension, should be considered  
164 suspect and worked up. The key to detecting TAD in this patient population  
165 starts by simply thinking about the diagnosis.

#### 166 *Why is the diagnosis missed?*

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

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

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

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

#### 190 **Box 2. Thoracic aortic dissection risk factors**

191 Hypertension

192 Age

193 Male gender

194 Pregnancy

195 Bicuspid aortic valve

196 Coarctation of the aorta

197 Cocaine

198 to attempt to integrate the two seemingly disparate complaints or falsely  
 199 assigns a preliminary diagnosis that does not explain the patient's symp-  
 200 toms; this has been referred to by some as failure to perform complaint and  
 201 sign integration [11]. Because acute aortic disease may present with a myriad  
 202 of signs and symptoms, emergency physicians should be wary and search for  
 203 chest pain, back pain, or abdominal pain in association with other findings,  
 204 such as stroke symptoms, limb ischemia, or a combination of chest and back  
 205 pain [11]. **Box 3** summarizes risk management recommendations, and **Box 4**  
 206 lists causes of missed or delayed TAD diagnosis [11].

### 207 *Subtle and atypical presentations*

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

### 220 **Box 3. Risk management recommendations for thoracic** 221 **aortic dissection**

- 222 1. Perform a detailed risk factor analysis for every chest pain  
 223 patient.
- 224 2. Perform a thorough physical examination focusing on thoracic  
 225 aortic dissection findings—pulse deficits, blood pressure  
 226 differential, diastolic murmur of aortic regurgitation, and  
 227 neurologic abnormalities.
- 228 3. Understand the limitations of the chest x-ray.
- 229 4. Practice complaint and sign integration (the diagnosis must  
 230 explain the patient's presentation).
- 231 5. Understand that many cases may be subtle and atypical.
- 232 6. Do not exclude lethal aortic diseases such as thoracic aortic  
 233 dissection in young patients.
- 234 7. Documentation should reflect a diligent search for life-  
 235 threatening chest pain etiologies, particularly thoracic aortic  
 236 dissection.

237  
238**Box 4. Causes of missed or delayed thoracic aortic dissection diagnosis**

- 239 Inadequate knowledge base
- 240 Failure to detect a classic presentation
- 241 Failure to perform a detailed risk factor profile of thoracic aortic
- 242 dissection
- 243 Failure to integrate the patient's signs and symptoms

244 the cases [17]. Delays in diagnosis seem to be common. In a study of 84  
 245 TAD patients by Viljanen [6], there was a delay of more than 24 hours in  
 246 31% of the proximal TAD cases and in 53% of the distal aortic dissections  
 247 [6].

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

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

269

**Box 5. Atypical presentations of thoracic aortic dissection**

- 270 Painless
- 271 Isolated abdominal pain
- 272 Stroke
- 273 Paralysis
- 274 Syncope



275 Fig. 1. Proximal aortic dissection. Chest CT scan of a 34-year-old man who presented with  
 276 nausea and vomiting, but no chest pain.

[Q14]

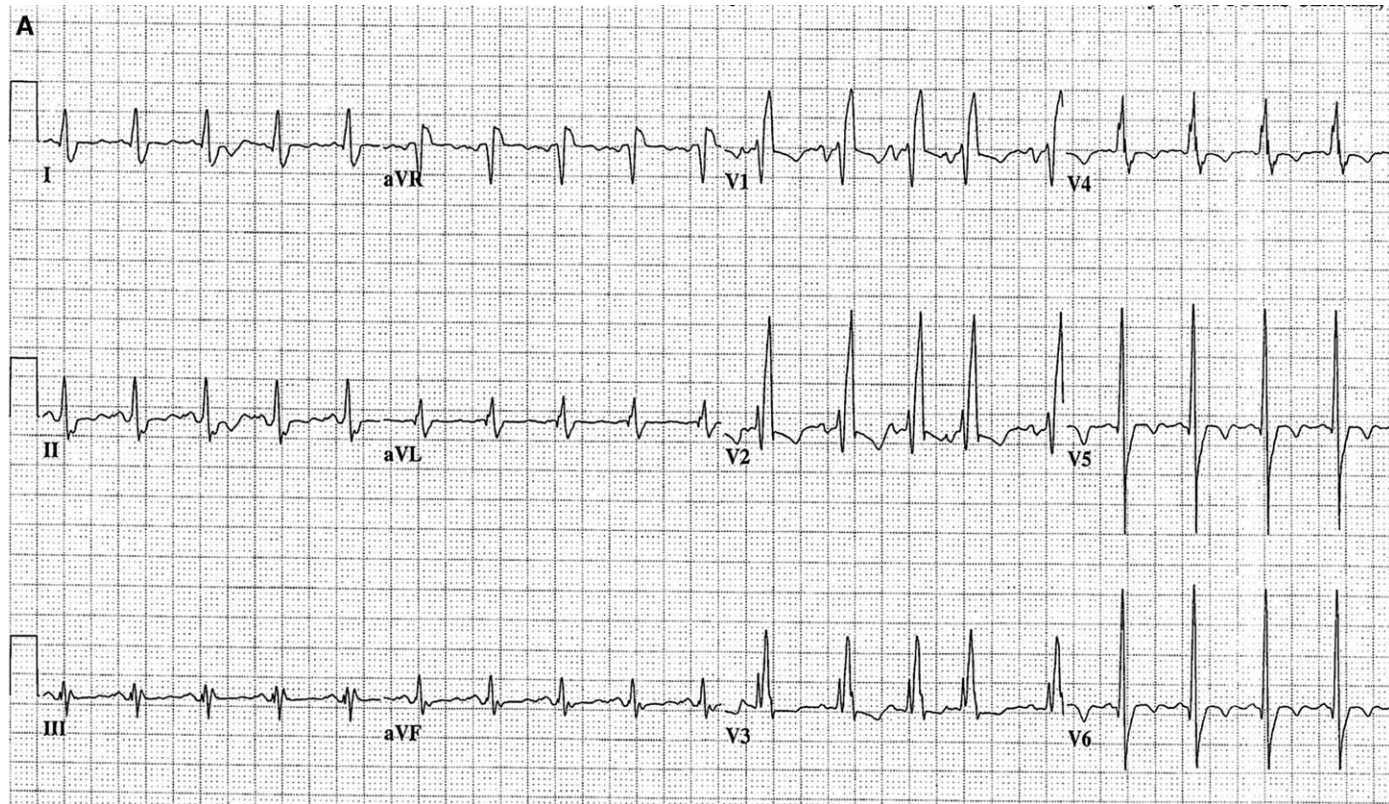
277 such as this may be detected by transesophageal echocardiography [22]. In  
 278 particular, cases of cocaine-induced TAD may lack many of the typical pain  
 279 referral patterns and resemble an acute coronary syndrome or some other  
 280 chest pain entity.

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

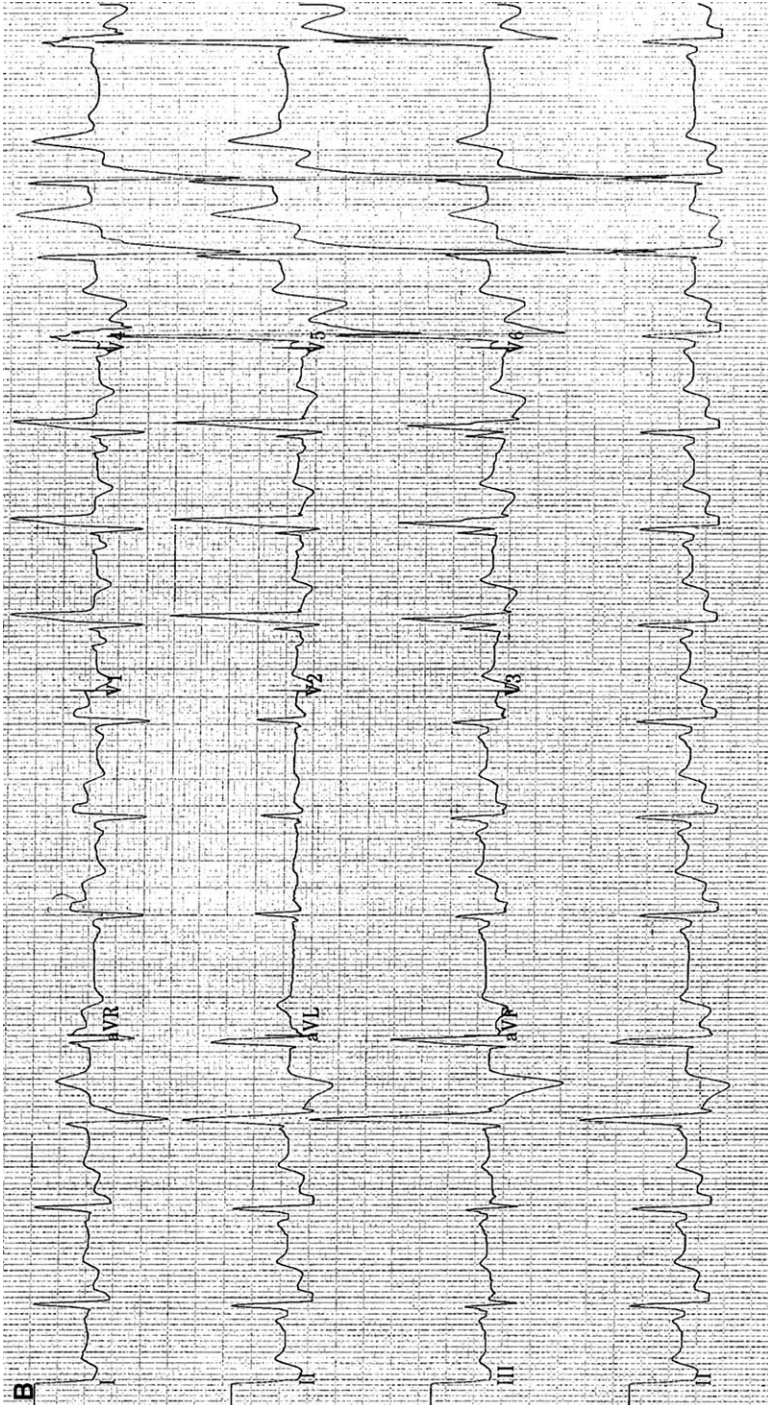
291 TAD in some cases may cause extremity ischemia. A useful acronym  
 292 proposed by Pacifico and Spodick [23] is *ILEAD*—ischemia of the lower  
 293 extremity in aortic dissection [24]. Patients who present with evidence of  
 294 extremity ischemia in association with chest pain should have the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

[Q7]

296 Cases of aortic dissection have been noted to have a frequent association  
 297 with mild neurologic symptoms. Neurologic deficits are found in approxi-  
 298 mately 18% to 30% of cases [25]. In some cases, neurologic deficits may  
 299 occur without any chest pain. Neurologic presentations include acute stroke,  
 300 bilateral lower extremity paralysis secondary to dissection involving the  
 301 greater radicular artery, unilateral lower extremity numbness, and hoarse-  
 302 ness [26–30]. Cerebral ischemia and stroke is the most common neurologic  
 303 manifestation associated with TAD and has been reported in 5% to 10% of  
 304 patients. Spinal cord involvement in TAD may result in various spinal cord

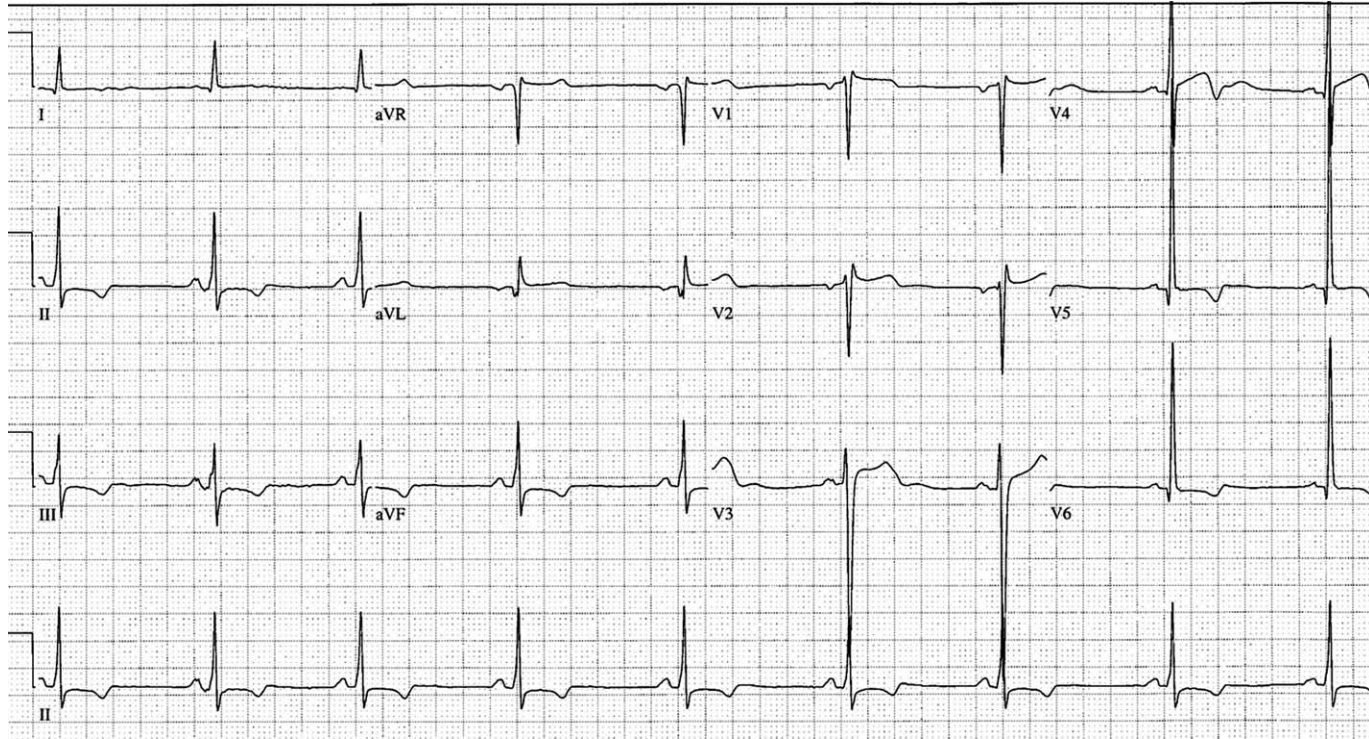


305 Fig. 2. Case 1—a 51-year-old man with left-sided chest pain and diaphoresis Baseline electrocardiogram (ECG) (A) and ECG on presentation (B). The patient  
306 was treated with aspirin,  $\beta$ -blockers, and heparin. The patient decompensated and was found to have a proximal thoracic aortic dissection and right coronary  
307 artery occlusion by cardiac catheterization.



DTD 5.0

Fig. 2 (continued)



309 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  
310 the inferior leads. Right coronary artery occlusion was found in the operating room during repair.



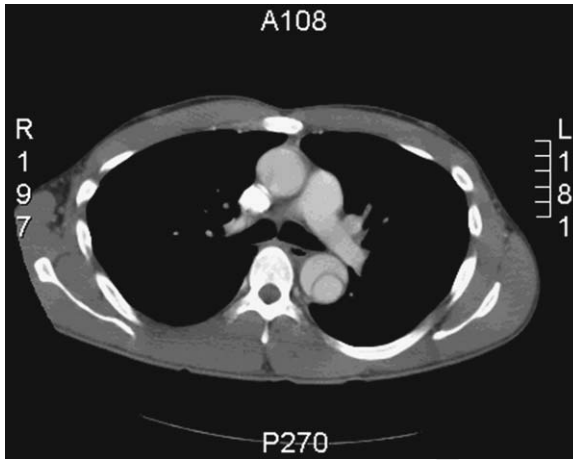
311 Fig. 4. Echocardiogram shows large pericardial effusion secondary to proximal thoracic aortic  
 312 dissection and hemopericardium. This 44-year-old woman presented with chest pain and  
 313 syncope.

314 syndromes, including transverse myelitis and spinal cord infarction, and  
 315 may present with paraplegia or quadriplegia [31]. These spinal cord  
 3 syndromes tend to occur more often in cases of distal TAD. The diagnosis  
 3 of TAD should be entertained in any patient with neurologic signs and  
 3 symptoms, especially if they occur in conjunction with chest pain. Many  
 3 cases of missed TAD could have been diagnosed if the treating physician  
 320 would have attempted to integrate the patient's signs and symptoms.

321 Isolated abdominal aortic dissection has been described previously in  
 322 case reports. Farber et al [32] evaluated 10 cases of aortic dissection confined  
 323 to the abdominal aorta. Only five patients were hypertensive on presenta-  
 324 tion. In a study by Hsue et al [33], patients with cocaine-associated TAD  
 325 were more likely to have distal aortic dissection and to present with  
 326 abdominal pain. In a study of 398 cases of TAD, Hirst et al [34] showed that  
 327 2.5% were confined to the distal thoracic and abdominal aorta. Fig. 5 shows  
 328 a CT scan of a patient with isolated abdominal pain after cocaine use. The  
 329 CT scan shows a Stanford B TAD. TAD and abdominal aortic dissection  
 330 may present with isolated abdominal pain. Considering the diagnosis,  
 331 particularly in younger, hypertensive patients with risk factors, such as  
 332 cocaine, may make the difference between diagnosis and a poor outcome.

### 333 *International Registry of Aortic Dissection study*

334 One of the largest studies to date to examine the clinical characteristics of  
 335 TAD cases was the International Registry of Aortic Dissection (IRAD)  
 336 performed by Hagan et al [4]. In this study, 464 cases of TAD were  
 337 examined for the history, physical examination, and management. Of these  
 338 cases, 62.3% were proximal dissections. Classic physical examination find-  
 339 ings, such as the murmur of aortic regurgitation and a pulse deficit, were



340 Fig. 5. Abdominal CT scan shows dissection of the abdominal aorta in a 34-year-old  
341 hypertensive man with abdominal pain after cocaine use.

342 detected in only 31.6% and 15.1% of cases. Traditionally, it is thought that  
343 the chest x-ray would be abnormal in cases of TAD. In this study, 12.4% of  
344 chest x-rays were interpreted as completely normal. Patients with TAD  
345 frequently present without a classic history and physical examination. Al-  
346 though from a risk-management standpoint it makes good sense to examine  
347 and document the presence or absence of an aortic murmur and a pulse  
348 deficit, they are unreliable findings [4,6]. Box 6 lists suggestive findings of  
349 TAD; see also Box 1 earlier for independent predictors of TAD.

### 350 Abdominal aortic aneurysm

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

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

#### 360 **Box 6. Suggestive findings of thoracic aortic dissection**

- 361 Chest pain and neurologic abnormalities
- 362 Chest pain in association with back or abdominal pain
- 363 Chest pain that radiates into the back

364 AAA, 40% to 50% die before they reach the hospital, and the overall  
 365 mortality rate of a ruptured AAA is greater than 90% [35]. There is a  
 366 marked increase in mortality when the diagnosis is delayed or missed [36–38].  
 367 In one study, the mortality rate was 35% when the diagnosis was at  
 368 least initially suspected, whereas the mortality rate was 75% when there was  
 369 no suspicion before the diagnosis was made [38].

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

### 377 *Usefulness of the physical examination*

378 The physical examination of patients with suspected AAA may be helpful  
 379 but is fraught with problems. If rupture has occurred in the retroperitoneum  
 380 or if the patient is obese, the examination may appear normal. One of the  
 381 most helpful findings is detection of an abnormally widened aortic pulse  
 382 [40]. Detection of the aortic pulse to the right of the midline also has been  
 383 shown to be useful. Caution should be exercised because only 76% of  
 384 aneurysms greater than 5 cm are palpable [40–42]. Box 7 lists physical  
 385 examination findings of AAA [11].

[Q8]

### 386 *Why is the diagnosis missed?*

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

#### 392 **Box 7. Physical examination findings of abdominal aortic** 393 **aneurysm**

394 Unexplained hypotension or altered level of consciousness  
 395 Presence of pulsatile abdominal mass  
 396 Aortic pulsation to the right of the midline  
 397 Left lower quadrant abdominal mass with tenderness and  
 398 distention  
 399 Abdominal bruit  
 400 Absent or diminished lower extremity peripheral pulses  
 401 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 Risk factor assessment plays an important role not only in evaluating the  
408 patient, but also in convincing potential juries that the emergency physician  
409 was thorough in the approach to patient care. Cases have been identified  
410 that lacked a thorough risk factor analysis, which subsequently led to  
411 plaintiff verdicts. What can the emergency physician do to decrease poten-  
412 tial risk? AAA rupture is a diagnosis that must be approached with a high  
413 index of suspicion because history has shown that a significant proportion  
414 of diagnoses are delayed or missed. Eliciting a thorough history should  
415 enable emergency physicians to risk stratify patients and know when even  
416 low-risk patients need a prompt evaluation. Patients with atherosclerotic  
417 disease also are at risk for aneurysm formation. Advanced age is a well-  
418 established risk factor; AAA rarely occurs in patients younger than age 50.  
419 The disease also is more prevalent in men, smokers, and hypertensive  
420 individuals. The strong familial association may be less well known. Webster  
421 et al [44] found that that 39% of patients with AAA have first-degree  
422 relatives with this condition. The failure to elicit a personal history of  
423 aneurysm in a patient who presents with a history consistent with rupture  
424 could be detrimental. Finally, patients with connective tissue disorders, such  
425 as Ehlers-Danlos syndrome and Marfan syndrome, must be identified and  
426 recognized for their increased risk because of vascular structural defects,  
427 particularly of the aorta. **Box 8** lists AAA risk factors [54]. [Q9]

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

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

435 Hypertension

436 Atherosclerotic vascular disease

437 Connective tissue disease

438 Family history in first-degree relative

439 Advanced age (mean age of diagnosis 67)

440 Smoking

441 Diabetes

442 White race

443 hospital with a diagnosis of abdominal pain and possible gastrointestinal  
 444 bleeding. In the hospital, the patient had a ruptured AAA and died. A  
 445 couple of things can be learned from this case. First, there was no attempt to  
 446 integrate the abdominal pain, syncope, and bleeding. What did the treating  
 447 physician think was the cause of the severe abdominal pain? Second, any  
 448 elderly patient with abdominal pain, particularly if associated with syncope,  
 449 should have a rapid evaluation to rule out a ruptured AAA. The diagnosis  
 450 of AAA should be entertained in all older patients with syncope, even if an  
 451 alternative diagnosis is more likely. Box 9 is an overview of AAA litigation  
 452 [11].

### 453 *Must-know presentations*

454 It has been known for many years that AAA may masquerade as renal  
 455 colic. Urologic symptoms are estimated to be present in 10% of patients  
 456 with AAA and are estimated to be the most common misdiagnosis for this  
 457 group of patients [36]. The mechanism for the development of urologic  
 458 symptoms is thought to be due to irritation of the retroperitoneal ureter or  
 459 direct compression of the ureter by the expanding or rupturing aneurysm  
 460 [46]. Among patients older than age 65 who were referred to urologists for  
 461 workup of renal colic, 10% were found to have an AAA [47]. All older  
 462 patients and patients with connective tissue disease who present with flank/  
 463 back pain or signs and symptoms of nephrolithiasis should have AAA ruled  
 464 out. Chart reviews and retrospective analyses have shown repeatedly symp-  
 465 tomatic or ruptured AAAs to be misdiagnosed as genitourinary pathologies.  
 466 One review found that 7 of 23 patients with AAA were diagnosed er-  
 467 roneously with urinary obstruction, infection, or stone [41,48]. Another  
 468 study discovered 10 of 44 patients with AAA who had received the same  
 469 inappropriate diagnosis [49]. Although the pain associated with AAA is  
 470 typically constant and localized to the abdomen, numerous reviews detail  
 471 histories of radiation to one or both flanks and colicky-type pain. Also,  
 472 emergency physicians should not be misled to a diagnosis of ureteral colic by  
 473 the presence of microscopic hematuria because it can be seen in ruptured  
 474 AAA. To avoid potential catastrophic misdiagnoses and litigation, any

### 475 **Box 9. Overview of abdominal aortic aneurysm litigation**

476 Poor risk factor analysis  
 477 Poor recognition of flank pain (“kidney stone”) presentations  
 478 Failure to obtain imaging studies to evaluate the aorta  
 479 Delay in proper management  
 480 Failure to recognize presentations such as syncope  
 481 Failure to consult a vascular surgeon or arrange timely outpatient  
 482 follow-up

483 elderly patient with flank pain and suspected genitourinary disease should  
484 be evaluated for aortic pathology. In most cases, this evaluation involves  
485 performing an abdominal CT scan or ultrasound. Fig. 6 shows a CT scan of  
486 a patient who presented with flank pain and hematuria. Box 10 lists unusual  
487 presentations of AAA.

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

502 AAA frequently is misdiagnosed as diverticulitis, and it is estimated that  
503 12% of AAAs are diagnosed initially as diverticulitis [36,41,48,49]. Most  
504 AAAs rupture into the left retroperitoneum or peritoneum and may lead to  
505 tenderness or a left lower quadrant mass. In cases in which diverticulitis is in  
506 the differential diagnosis, emergency physicians should consider the diag-  
507 nosis of AAA. With more widespread availability and liberal use of CT,  
508 it would seem logical that the rate of this misdiagnosis would decrease.  
509 Patients with suspected diverticulitis, particularly patients at high risk for



510 Fig. 6. Abdominal CT scan shows large abdominal aortic aneurysm in a 70-year-old man who  
511 presented with left flank pain and hematuria.

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513**Box 10. Unusual presentations of abdominal aortic aneurysm**514  
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Musculoskeletal complaints (thigh or groin pain)  
 Bilateral testicular pain  
 Unexplained inguinal pain  
 Femoral neuropathy  
 Abdominal pain and urge to defecate

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AAA, should have appropriate imaging studies performed in the emergency department.

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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].

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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.

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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].

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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

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**Box 11. Risk management recommendations for abdominal aortic aneurysm**

1. Consider the diagnosis in every older patient with abdominal, back, or flank pain.
2. Perform a risk factor analysis for abdominal aortic aneurysm on every patient with abdominal or back pain.
3. Consider the diagnosis in every older patient with “renal colic” presentations and in cases of unexplained hypotension or syncope.
4. Use caution in diagnosing kidney stones in patients older than age 65.
5. Elderly patients with abdominal pain are high-risk patients.
6. Documentation should reflect a diligent search for abdominal aortic aneurysm.

characteristic for other disease entities. 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].

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**Summary**

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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].

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**References**

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