Does This Patient Have an Acute Thoracic Aortic Dissection?

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Context The diagnosis of acute thoracic aortic dissection is difficult to make and often missed.

Objective To review the accuracy of clinical history taking, physical examination, and plain chest radiograph in the diagnosis of acute thoracic aortic dissection.

Data Sources A comprehensive review of the English-language literature was conducted using MEDLINE for the years 1966 through 2000. Additional sources were identified from the references of retrieved articles.

Study Selection The search revealed 274 potential sources, which were reviewed for pertinence and quality. Articles included were original investigations describing the clinical findings for 18 or more consecutive patients with confirmed thoracic aortic dissection. Twenty-one studies were identified that met selection criteria.

Data Extraction Critical appraisal and data extraction were performed by the author.

Data Synthesis Most patients with thoracic aortic dissection have severe pain (pooled sensitivity, 90%) of sudden onset (sensitivity, 84%). The absence of sudden pain onset lowers the likelihood of dissection (negative likelihood ratio [LR], 0.3; 95% confidence interval [CI], 0.2-0.5). On examination, 49% of patients have an elevated blood pressure, 28% have a diastolic murmur, 31% have pulse deficits or blood pressure differentials, and 17% have focal neurological deficits. Presence of a diastolic murmur does little to change the pretest probability of dissection (positive LR, 1.4; 95% CI, 1.0-2.0), whereas pulse or blood pressure differentials and neurological deficits increase the likelihood of disease (positive LRs, 5.7 and 6.6-33.0, respectively). The plain chest radiograph results are usually abnormal (sensitivity, 90%); hence, the presence of a normal aorta and mediastinum decreases the probability of dissection (negative LR, 0.3; 95% CI, 0.2-0.4). Combinations of findings increase the likelihood of disease.

Conclusions The presence of pulse deficits or focal neurological deficits increases the likelihood of an acute thoracic aortic dissection in the appropriate clinical setting. Conversely, a completely normal chest radiograph result or the absence of pain of sudden onset lowers the likelihood. Overall, however, the clinical examination is insufficiently sensitive to rule out aortic dissection given the high morbidity of missed diagnosis.

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CLINICAL SCENARIOS

Case 1

A 64-year-old man with a history of hypertension presents to the emergency department after sudden onset of severe, anterior chest pain. On examination, he is alert but uncomfortable. His blood pressure is normal and identical in both arms. His chest is clear and careful cardiac auscultation fails to reveal a diastolic murmur. A chest radiograph reveals a small pleural effusion but is otherwise unremarkable.

Case 2

A 59-year-old woman is brought to the emergency department after the sudden onset of tearing chest pain. On examination, she is alert and oriented. Her blood pressure is identical in both arms. Results of her cardiac and pulmonary examinations are normal but she has a dense left-sided motor deficit. A portable chest radiograph raises the question of a widened mediastinum.

A man . . . was seized with a pain of the right arm and soon after of the left, . . . after these there appeared a tumor on the upper part of the sternum . . . He was ordered to think seriously and piously of his departure from this mortal life, which was very near at hand and inevitable.

J. B. Morgagni, 1761[1]

There is no disease more conducive to clinical humility than aneurysm of the aorta.

Sir William Osler, c 1900[2]

Why Is Clinical Examination Important?

Acute thoracic aortic dissection, one of the most common and serious diseases of the aorta, carries a high morbidity and mortality rate when it is not recognized and treated promptly. Autopsy series conducted before the era of modern treatment estimated that 40% to 50% of patients with dissection of the proximal aorta died within 48 hours.[3] For those fortunate enough to survive the initial 48 hours, the disease was thought to carry a 90% 1-year mortality rate.[3,4] Since the introduction of modern treatment regimens, the fatality rate has declined dramatically. Patients with proximal ascending dissections who rapidly undergo surgery in experienced tertiary centers have a 30-day survival rate of 80% to 85% and a 10-year survival of 55%.[4,5] Likewise, patients with dissection of the descending aorta treated with aggressive anti-

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hypertensive therapy have a 30-day survival rate greater than 90% and a 10-year survival rate of 56%.[4-6] Realization of the dramatic benefits of medical intervention is dependent upon rapid establishment of the diagnosis of dissection.

Approximately 4.6 million patients per year present with chest pain to emergency departments in the United States (8.2% of all emergency department visits).[7] While advanced imaging techniques can reliably establish the diagnosis of thoracic aortic dissection in high-risk populations, it is obviously inefficient, uneconomic, and unrealistic to image every patient complaining of chest pain. Indiscriminate use of diagnostic imaging in poorly chosen patients with very low pretest probability of having dissection has been predicted to yield up to an 85% rate of false-positive results depending on the imaging modality chosen.[8] On the other hand, misdiagnosis of acute thoracic aortic dissection as unstable angina or myocardial infarction can have disastrous iatrogenic consequences should the patient receive anticoagulants or thrombolytic therapy.[9] Physicians are therefore acutely dependent upon the clinical history, examination, and plain chest radiograph to determine which patients require further study.

Traditionally, clinical diagnosis of thoracic aortic dissection has been inaccurate. Physicians correctly suspect the diagnosis in as few as 15% to 43% of presentations when initially evaluating patients with dissection.[3,10,11] Diagnostic delay of more than 24 hours after hospitalization occurs in up to 39% of cases.[12] When the diagnosis is made, not infrequently it is an incidental discovery made during an advanced imaging procedure intended to assess for other diagnoses.[13,14] Autopsies reveal the correct diagnosis is still missed in more than 10% of patients.[13]

The purpose of this review is to offer physicians an evidence-based foundation for using the clinical history, physical examination, and plain chest radiograph to assess the likelihood of thoracic aortic dissection.

Pathophysiology of Thoracic Aortic Dissection

The aortic wall is composed of 3 contiguous tissue layers in sequence from the vessel lumen proceeding outwards: the intima, media, and adventitia. Weakening of these tissue layers can lead to a tear in the intima permitting the entry of blood between the intima and adventitia.[15] Passage of blood into this space can extend the tear and create a so-called false lumen. The majority of these tears take place in the ascending aorta, usually in the right lateral wall where the greatest shear force upon the artery wall is produced by blood expulsed from the heart under high pressure.[3] The tear then extends along the greater curve of the aortic arch and down the descending aorta, though retrograde extension of the tear toward the aortic valve is also possible.[15] Most aortic tears occurring beyond the ascending aorta originate immediately distal to the left subclavian artery.[15] Predisposing factors for the initiation of a thoracic aortic dissection include hypertension,[15] bicuspid aortic valve,[15] coarctation of the aorta,[15] the Marfan syndrome,[16] Ehlers-Danlos syndrome,[17] Turner syndrome,[18] giant cell arteritis,[19] third-trimester pregnancy,[20] cocaine abuse,[21] trauma,[22] intra-aortic catheterization,[23] and history of cardiac surgery, particularly aortic valve replacement.[24]

The clinical features of thoracic aortic dissection are a consequence of the underlying pathophysiologic changes in the aorta. Patients perceive the initial aortic tear as sudden onset of severe ripping or tearing chest pain. The pain is sometimes described as having a migrating quality likely corresponding to extension of the tear along the aorta. Depending on the location of the tear and its direction of extension, patients alternately describe the pain as radiating to the neck, back, or abdomen. Occasional presentations of painless dissection have been reported, though these are usually accompanied by other findings.[25,26]

Retrograde extension of the tear to the aortic valve can result in aortic regurgitation with its characteristic diastolic murmur. Likewise, if the tear communicates with the pericardial space, patients can present with symptoms of acute pericardial tamponade (hypotension, pulsus paradoxus, jugular venous distension, and muffled heart sounds). Syncope or prolonged unconsciousness can be the initial presentation of patients with pericardial tamponade.

The initial aortic tear and subsequent extension of a false lumen along the aorta can occlude blood flow from the true lumen of the aorta into any of the arteries that originate from the aorta. Depending upon which arteries become occluded, patients can present with a variety of corresponding syndromes. These include acute myocardial infarction from occlusion or extension of tear into the coronary arteries (typically the right coronary artery); death, syncope, or hemiplegia after occlusion of one or both carotid arteries; absent peripheral pulses in the major limb vessels secondary to occlusion of the brachiocephalic trunk, left subclavian artery, or distal aorta; anuria from disruption of renal blood flow; and paraplegia or quadriplegia from occlusion of vessels feeding the anterior spinal artery.

Examination for the Signs and Symptoms of Thoracic Aortic Dissection

The classic clinical history for thoracic aortic dissection consists of the sudden onset of severe tearing or ripping chest pain radiating to the interscapular region or low back, occurring in late middle-aged men with a history of hypertension. Physicians therefore need to inquire of patients about the onset, quality, radiation, and intensity of patients' pain. Inquiry should also be made of history or symptoms suggestive of factors that increase the risk of aortic dissection, including hypertension, the Marfan syndrome, bicuspid aortic valve, prior aortic valve replacement, and the other syndromes previously listed.

History taking from patients with thoracic aortic dissection has tended to be

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poor; however, there is evidence that a more thorough history may increase diagnostic yield. A retrospective chart review of 83 patients with subsequently confirmed thoracic aortic dissection revealed that only 42% of conscious patients were asked all of 3 basic questions about their pain (quality, radiation, intensity at onset).[14] One quarter of patients were asked 1 or none of these key questions. If all 3 questions were asked, physicians correctly diagnosed thoracic aortic dissection in 30 of 33 patients (91%); if 1 or more of these questions was omitted, then the correct diagnosis was suspected during the initial evaluation in only 22 of 45 (49%) patients ($P<.001$). In these patients, the diagnosis was made later usually as an incidental finding during imaging procedures intended to diagnose alternative conditions. Unfortunately, the retrospective design of this study cannot preclude the possibility that physicians were simply more likely to ask about additional classic findings when they already had a strong clinical suspicion of thoracic aortic dissection derived from other data including physical examination and plain chest radiograph.

The physical examination should begin with elicitation of vital signs, particularly the blood pressure and pulses on both sides of the body. While checking the blood pressure, the examiner should evaluate for acute pericardial tamponade by assessing for pulsus paradoxus, particularly in a patient with hypotension or jugular venous distension. Frequent allusion is made to the importance of comparing the blood pressure in both arms. While it is essential to seek evidence of vascular occlusion in the arms, the complete examination should include comparison of all major arteries including the carotid and femoral pulses, in addition to the radial pulses.

Most of the published series of patients with thoracic aortic dissection comment only upon the loss or obvious diminishment of pulses rather than particular blood pressure differentials. Older retrospective autopsy series that do refer to blood pressure differentials arbitrarily designate a difference in systolic pressure between arms of 20 mm Hg[3] or 30 mm Hg[27] as significant. However, a convenience sample of 610 patients without thoracic aortic dissection presenting to an emergency department showed that 53% had interarm differences of greater than 10 mm Hg and 19% had differences greater than 20 mm Hg.[28] Nonetheless, a good quality prospective, observational study did find that a blood pressure differential of greater than 20 mm Hg was an independent predictor of dissection.[29] Hence, a blood pressure differential of at least 20 mm Hg ought to be present to be considered significant.

Cardiac auscultation should focus upon detecting the diastolic murmur of aortic regurgitation.[30] A rapid neurological examination directed toward the detection of gross motor and sensory defects such as hemiplegia and paraplegia should ensue.

Rarer clinical findings reported in the literature include pulsatile sternoclavicular joint, hoarseness, dysphagia, superior vena cava syndrome, Horner syndrome, bulbar palsies, acute arterial occlusion, deep venous thrombosis, and bilateral testicular tenderness.[31-37]

A plain chest radiograph should be obtained and examined for abnormalities of the aortic silhouette. This is best accomplished with a standing anteroposterior projection. Unfortunately, the majority of chest radiograph findings associated with thoracic aortic dissection are subjective and not defined. Criteria for radiographic features associated with traumatic thoracic aortic dissection have been proposed but have not been adopted or validated in radiological studies of nontraumatic dissections.[38] Radiographic abnormalities may include wide mediastinum, widening of the aortic knob, difference in diameter between the ascending and descending aorta, and blurring of the aortic margin secondary to local extravasation of blood.[39] The chest radiograph might also reveal unilateral or bilateral pleural effusions. The calcium sign, consisting of the separation of intimal calcification from the outer border of the aortic knob by 1 cm or more, is highly suggestive of dissection but present in a minority of cases.[37,40] Comparison with previous chest radiographs of the same patient can help the examiner detect suggestive new changes in the aortic contour.

METHODS

Literature Search and Selection

A structured MEDLINE search including the years 1966 through 2000 was conducted to identify English-language articles examining the accuracy of the clinical history, examination, and chest radiograph in the detection of acute thoracic aortic dissection. Key words used in the search included *physical examination, medical history taking, professional competence, reproducibility of results, observer variation, diagnostic tests, decision support techniques, Bayes theorem, sensitivity, specificity, thoracic aortic dissection, aortic aneurysm,* and *dissecting aneurysm*. Articles focused only on electrocardiograms (ECGs) were not specifically sought because such analyses document a variety of abnormalities seen with thoracic aneurysm but lack the appropriate clinical information for valid sensitivity and specificity estimates. When studies reported the results of ECGs as part of the overall clinical examination, however, these data were collated. Abstracts were reviewed and the full texts of articles that might meet the inclusion criteria were retrieved. The reference lists of reviewed articles were searched to identify additional sources.

All potential articles were reviewed for explicit inclusion and exclusion criteria. Articles were included if they were original studies describing the clinical findings in a series of 18 or more consecutive patients with confirmed dissection of the thoracic aorta (TABLE 1). Acceptable means of confirmation of diagnosis were surgical exploration, autopsy, aortogram, magnetic resonance imaging, computed tomography, or transesophageal echocardiography. The latter 4 imaging studies were included as acceptable gold-standard investigations on the basis of high sensitivity and

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Table 1. Studies Assessing the Accuracy of Clinical Examination for Thoracic Aortic Dissection (TAD)*

Source, y	Clinical Setting, Study Dates	Design	No. of Patient Episodes	Age, Mean (Range), y	Male, %	Type A, %	Level of Quality
Von Kodolitsch et al,[29] 2000	University hospital, 1988-1996	Prospective study of patients presenting to ED with history suggestive of TAD	250 (128 with TAD)	53	78	61	3
Hagan et al,[5] 2000	12 Tertiary centers in 6 countries, 1996-1998	Multinational prospective international registry; cases identified on admission or review of discharge/surgery/ radiology records; 60% of cases referred	464	63	65	62	4
Armstrong et al,[45] 1998	University hospital, 1992-1994	Retrospective review of patients with clinically suspected TAD referred for TEE	75 (34 with TAD)	57 (20-80)	74	91	4
Jagannath et al,[40] 1986	University hospital, 1965-1977	Retrospective review of radiographs†	72 (36 with TAD)	62 (17-85)	Not stated	1/3	4
Slater and DeSanctis,[37] 1976	University hospital, 1963-1973	Retrospective chart review	124	59 (19-81)	73	43	4
Levinson et al,[27] 1950	University hospital, 1935-1947	Retrospective chart review of autopsy cases	58	59 (22-90)	72	. . .	4
Luker et al,[48] 1994	Hospital, 1987-1993	Retrospective review of radiologists' initial chest radiograph readings in cases with subsequently confirmed TAD	75	61 (24-77)	49	47	4
Chan,[43] 1991	University hospital, 1987-1989	Prospective evaluation of utility of transesophageal echocardiography in patients with clinically suspected TAD	40 (18 with TAD)	60	60	. . .	4
Enia et al,[44] 1989	Hospital, 1981-1987	Prospective evaluation of transthoracic echocardiography in patients with clinically suspected TAD	46 (35 with TAD)	58 (34-82)	91	66	4
Itzchak et al,[49] 1975	Hospital, 1960-1973	Retrospective chart review	24	57 (12-86)	75	46	4
Lindsay and Hurst,[50] 1967	University hospital, 1949-1966	Retrospective chart review	62	57 (31-83)	65	65	4
Hume and Porter,[51] 1963	University hospital and medical examiner's office, 1950-1962	Retrospective chart review‡	68	53 (10-79)	79	81	4
Nielsen,[46] 1961	3 Danish hospitals, 1944-1958	Retrospective chart review§	40	66 (36-83)	45	. . .	4
Erb and Tullis,[47] 1960	University hospital, 1950-1960	Retrospective chart review	30	56 (36-85)	67	. . .	4
Pinet et al,[52] 1984	University hospital, 1970-1979	Retrospective chart review	191	58 (19-90)	69	64	4
Pate et al,[53] 1976	Memphis hospitals, dates not given	Retrospective chart review	126	Not reported	79	. . .	4
Miller et al,[54] 1979	University hospital, 1963-1979	Retrospective review of surgically managed cases	73	57 (20-86)	70	73	4
Viljanen,[12] 1986	University hospital, 1964-1985	Retrospective review of surgically managed cases	73	51	66	64	4
Strong et al,[55] 1974	University hospital and VA hospital, 1960-1973	Retrospective chart review	59	60 (26-86)	78	46	4
Mészáros et al,[10] 2000	3 Hungarian towns, 1972-1998	Longitudinal, observational, population-based study‖	86	66 (36-97)	61	86	4
Sullivan et al,[11] 2000	3 University hospital EDs, 1992-1996	Retrospective review of ED patients referred for thoracic imaging	44	65 (36-89)	. . .	61	4

*ED indicates emergency department; TEE, transesophageal echocardiography; and VA, Veterans Affairs. Ellipses indicate information not available. Type A refers to aortic dissections involving the aorta proximal to the subclavian artery.
†Does not include data on the frequency of specific radiographic findings but does report interobserver agreement.
‡Two cases not confirmed by surgery or autopsy.
§Forty cases in which TAD was considered cause of death; also reports additional 18 cases in which TAD was incidental finding on autopsy.
‖Eleven percent of cases were chronic.

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Table 2. Sensitivity of the Clinical History in the Diagnosis of Acute Thoracic Aortic Dissection*

Source, y	No. of Patients	Sensitivity, %						
		History of Hypertension	Marfan Syndrome	Any Pain	Chest Pain	Anterior Chest Pain	Posterior Chest Pain	Back Pain
Von Kodolitsch et al,[29] 2000	128	77	7	100†	. . .	76	. . .	50‡
Hagan et al,[5] 2000	464	72	5	96	73	61	36	53
Armstrong et al,[45] 1998	34	. . .	. . .	94	74	. . .	. . .	56
Slater and DeSanctis,[37] 1976	124	65	5	94	91	43	38	76
Levinson et al,[27] 1950	58	59	. . .	78	47	. . .	9	36
Lindsay and Hurst,[50] 1967	62	. . .	. . .	90	. . .	61	14	13
Hume and Porter,[51] 1963	68	89	4	97	59	59	33	43
Nielsen,[46] 1961	40	18	3	65	. . .	54	. . .	8
Erb and Tullis,[47] 1960	30	53	7	70	40	. . .	. . .	. . .
Pinet et al,[52] 1984	191	53	7	96	63	. . .	30	. . .
Pate et al,[53] 1976	126	. . .	. . .	88	63	. . .	38	22
Chan,[43] 1991	18	56	. . .	78	. . .	. . .	. . .	. . .
Strong et al,[55] 1974	59	75	3	. . .	. . .	32	. . .	25
Sullivan et al,[11] 2000	44	70	0	98	66	. . .	. . .	. . .
Enia et al,[44] 1989	35	80	. . .	. . .	. . .	. . .	. . .	. . .
Mészáros et al,[10] 2000	72	67	. . .	92	. . .	64	. . .	10
Pooled sensitivity (95% CI)	NA	64 (54-72)	5 (4-7)	90 (85-94)	67 (56-77)	57 (48-66)	32 (24-40)	32 (19-47)

*CI indicates confidence interval; NA, not applicable. Ellipses indicate data not available.
†Presence of pain inclusion criterion for study.
‡Posterior chest or lower back pain.

specificity.[41,42] Articles were excluded if more than 15% of their cohorts included trauma patients, patients with chronic thoracic aortic dissection (defined as a dissection presumed to have occurred more than 14 days prior to presentation), patients with abdominal aortic aneurysms, or if the study selectively included patients with only proximal or distal dissections.

Retrieved studies were graded for quality using criteria similar to that used in previous articles in this series but modified to include only consecutive series. Level 1 studies were defined as prospective, blinded examinations of a large number (>100) of independently selected consecutive patients. Level 2 studies were of identical criteria but included fewer than 100 patients. Level 3 studies were large, prospective investigations but included nonindependently selected patients. Level 4 studies were retrospective reviews of nonindependently selected patients.

Study Characteristics

A total of 274 studies were identified by the search strategy of which 21 studies met inclusion criteria (Table 1). No level 1 or level 2 studies were located. One study met level 3 criteria; the remaining 20 were level 4. One large series was self-described as prospective in conception and definition of clinical parameters.[5] An unknown percentage of its patients, however, were identified by physician review of discharge records, echocardiography, and surgical databases. This study was consequently classified conservatively as level 4.[5] Approximately half the investigations, including the 1 level 3 study, were specifically designed to elucidate the clinical presentation of acute aortic dissection. The remaining reports were either designed to test new imaging modalities or to study the outcomes of medical or surgical management of patients with thoracic aortic dissection. In each case, however, these studies included data on patients' clinical findings at the time of diagnosis. The studies varied considerably in the number and detail of components of the clinical history or examination that were reported. Only the prospective level 3 study explicitly defined the criteria used to establish whether a given clinical finding was present or absent.[29]

These studies assessed a total of 1848 patients, aged 10 to 97 years. The major limitation of all the studies is that patients were selected for inclusion either retrospectively after confirmation of diagnosis by a reference standard study or prospectively on the basis of the presenting clinical picture. Therefore, in all these studies the reference standard and clinical examination were not applied independently of one another. This biases the results of the studies to overestimate the sensitivity of clinical findings because more obvious cases are preferentially included in such series. In addition, physicians performing the reference standard procedure were not blinded to the results of the clinical examination and vice versa. This too could lead to overestimation of sensitivity.

Only 4 studies included control groups.[29,43-45] While these investigations can be used to generate data for specificity in addition to sensitivity, their estimations of specificity are heavily influenced by their inclusion biases. The specificities derived from these studies should be interpreted with caution as they only reflect the specificity

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Abdominal Pain	Sudden-Onset Pain	Severe Pain	Ripping or Tearing Pain	Migrating Pain	Syncope
22	79	86	62	44	10
30	85	91	51	17	9
27	88	93	7	. . .	6
4	93	94	. . .	71	5
40	. . .	. . .	. . .	. . .	14
11	. . .	. . .	. . .	. . .	. . .
49	. . .	. . .	. . .	. . .	. . .
33	76	. . .	. . .	. . .	16
17	. . .	. . .	. . .	. . .	. . .
. . .	. . .	89	. . .	6	. . .
. . .	88	. . .	. . .	. . .	10
. . .	78	. . .	. . .	39	. . .
27	. . .	. . .	. . .	. . .	. . .
34	. . .	. . .	. . .	. . .	2
. . .	. . .	. . .	. . .	. . .	. . .
10	. . .	. . .	. . .	. . .	14
23 (16-31)	84 (80-89)	90 (88-92)	39 (14-69)	31 (12-55)	9 (8-12)

for a given sign or symptom among patients similar to those included in the studies (ie, those with a full clinical syndrome suggestive of thoracic aortic dissection). These studies likely overestimate sensitivity and underestimate specificity by selecting patients for inclusion due to the presence of the particular sign being considered, thereby creating cohorts with artificially high prevalence of the finding.

Data Analysis

Summary measures for the sensitivity for components of the clinical examination for acute thoracic aortic dissection used published raw data from the reported trials that met criteria. Only 4 studies included specificity data that allowed construction of likelihood ratios (LRs). A random effects model was used to generate conservative summary measures and confidence intervals (CIs) for the sensitivity and LRs.[56] For LRs, a summary measure is reported only when there are more than 2 studies. The uncertainty in these measures is reflected in the broad CIs around the estimates. Interobserver agreement was calculated and interpreted using the κ statistic of Landis and Koch.[57] Fast Pro version 1.8 software was used for the meta-analysis (Academic Press, San Diego, Calif).

RESULTS

Accuracy of the Clinical History

Risk Factors. Sixteen studies examining 1553 patients report sensitivities for various components of the clinical history in TABLE 2. Most patients with dissection have a previously documented history of hypertension (sensitivity, 64%); however, the positive LR of this history is 1.6 (95% CI, 1.2-2.0). The pooled prevalence of the Marfan syndrome in this group of studies was 5% (95% CI, 4%-7%). Given that the Marfan syndrome only afflicts 0.02% to 0.03% of the general population,[58] the high prevalence of the Marfan syndrome in these series is suggestive of a markedly increased risk associated with this disorder, though the frequency of the Marfan syndrome detected in these series likely reflects the inclusion biases of these studies. The one controlled study that assessed for the Marfan syndrome generated a positive LR of 4.1.[29]

Symptoms. The majority of patients presented with pain (pooled sensitivity, 90%) of severe intensity (sensitivity, 90%) that occurred suddenly (sensitivity, 84%). All other recorded clinical symptoms were present in a low to moderate proportion of patients (Table 2). Patients were most likely to have anterior chest pain (sensitivity, 57%); however, pain was frequently experienced elsewhere including the posterior chest (32%), back (32%), and abdomen (23%). Likewise, migrating and ripping or tearing pain were only present in 31% and 39% of patients, respectively.

The presence of pain of sudden onset is not diagnostic (positive LR, 1.6; 95% CI, 1.0-2.4). The absence of this history, however, substantively decreases the probability of an acute thoracic aortic dissection (negative LR, 0.3; 95% CI, 0.2-0.5). Physicians should be cautious about relying too heavily on the absence of sudden pain to exclude aortic dissection because the inclusion biases of these studies likely overestimate the sensitivity.

Pain of a tearing or ripping sensation may also be diagnostically useful. Two studies found almost identical specificities of 94% and 95% for this historical feature.[29,45] While the reported specificities were almost identical, the positive LRs generated by these 2 studies differed considerably (1.2 vs 10.8, TABLE 3) reflecting significant heterogeneity in the sensitivity for this history reported by the 2 investigations. The retrospective study found that only 7% of patients had noted tearing or ripping pain.[45] By contrast, the better-quality larger, prospective study, in which physicians were asked to query predefined clinical symptoms of each patient, reported a sensitivity of 62%.[29] This figure is more consistent with the other large study with prospectively defined clinical symptoms in this series[5] and with the pooled sensitivity for this symptom (Table 2). Therefore, it seems reasonable to suspect that the higher reported sensitivity and LR are the more accurate data. Migratory pain has performance characteristics that are simi-

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lar to tearing or ripping pain. The positive LR for the presence of this quality was 7.6 (95% CI, 3.6-16.0) in one study[29] but only 1.1 (95% CI, 0.5-2.4) in the other.[43] Additional studies of independently selected patients that prospectively ask about the sensation of tearing or ripping, and migration of pain are needed to confirm the high LR for these findings. Description of pain as sharp was slightly more prevalent than tearing or ripping; however, this descriptor was only elicited in 2 studies and had a positive LR near unity.[5,45]

Accuracy of the Physical Examination

Physical examination findings classically associated with thoracic aortic dissection are typically present in less than half of all cases (TABLE 4). However, when present, signs of thoracic aortic dissection can be helpful. Among the most useful is a pulse differential between carotid, radial, or femoral arteries. While the pooled sensitivity for this sign is only 31%, a deficit in 1 of these pulses compared with the contralateral side is strongly suggestive of dissection (positive LR, 5.7; 95% CI, 1.4-23.0).[29,44,45] Focal neurological deficits, though present in only 17% of cases, may also be helpful. Specificity for this sign is high in the 2 studies in which it has been measured (positive LR, 6.6-33.0; Table 3).[29,45] The absence of a pulse deficit or focal neurological deficit does not appreciably alter the likelihood of thoracic aortic dissection.

The presence or absence of a diastolic murmur is not helpful. Only one third of patients with thoracic aortic dissection have a diastolic murmur (sensitivity, 28%). The positive and negative LRs (positive LR, 1.4; 95% CI, 1.0-2.0; negative LR, 0.9; 95% CI, 0.8-1.0) are close to 1, suggesting that the presence or absence of a diastolic murmur should not be considered helpful.[29,43-45] Unfortunately, these studies do not comment upon whether the diastolic murmurs identified were known to be new or old. It is possible that if a diastolic murmur was known to be new that it might have greater diagnostic utility.

Patients' blood pressure on presentation is not helpful. While approximately half of patients present with elevated blood pressure (pooled sensitivity, 49%; 95% CI, 41%-57%), an equal proportion are either hypotensive or normotensive. Only 1 study permitted calculation of a LR for hypertension; however, this study confirmed its low diagnostic yield (positive LR, 1.3 for systolic blood pressure >150 mm Hg).[29] Pericardial rub is rarely present (pooled sensitiv-

Table 3. Accuracy of Clinical Findings for Thoracic Aortic Dissection in Consecutive Patients Preselected for High Clinical Suspicion of Dissection Referred for Advanced Imaging*

Symptom or Sign	Source, y	Positive Likelihood Ratio (95% CI)	Negative Likelihood Ratio (95% CI)
History of hypertension	Chan,[43] 1991†	1.5 (0.8-3.0)	0.7 (0.4-1.3)
	Enia et al,[44] 1989‡	1.1 (0.7-1.6)	0.7 (0.4-2.4)
	Von Kodolitsch et al,[29] 2000§	1.8 (1.4-2.3)	0.4 (0.3-0.6)
	Summary	1.6 (1.2-2.0)	0.5 (0.3-0.7)
Sudden chest pain	Chan,[43] 1991†	1.0 (0.7-1.4)	0.98 (0.3-3.1)
	Armstrong et al,[45] 1998‖	1.5 (1.1-1.9)	0.3 (0.1-0.8)
	Von Kodolitsch et al,[29] 2000§	2.6 (2.0-3.5)	0.3 (0.2-0.4)
	Summary	1.6 (1.0-2.4)	0.3 (0.2-0.5)
"Tearing" or "ripping" pain	Armstrong et al,[45] 1998‖	1.2 (0.2-8.1)	0.99 (0.9-1.1)
	Von Kodolitsch et al,[29] 2000§	10.8 (5.2-22.0)	0.4 (0.3-0.5)
Migrating pain	Chan,[43] 1991†	1.1 (0.5-2.4)	0.97 (0.6-1.6)
	Von Kodolitsch et al,[29] 2000§	7.6 (3.6-16.0)	0.6 (0.5-0.7)
Pulse deficit	Armstrong et al,[45] 1998‖	2.4 (0.5-12.0)	0.93 (0.8-1.1)
	Enia et al,[44] 1989‡	2.7 (0.7-9.8)	0.63 (0.4-1.0)
	Von Kodolitsch et al,[29] 2000§	47.0 (6.6-333.0)	0.62 (0.5-0.7)
	Summary	5.7 (1.4-23.0)	0.7 (0.6-0.9)
Focal neurological deficit	Armstrong et al,[45] 1998‖	6.6 (1.6-28.0)	0.71 (0.6-0.9)
	Von Kodolitsch et al,[29] 2000§	33.0 (2.0-549.0)	0.87 (0.8-0.9)
Diastolic murmur	Chan,[43] 1991†	4.9 (0.6-40.0)	0.8 (0.6-1.1)
	Armstrong et al,[45] 1998‖	1.2 (0.4-3.8)	0.97 (0.8-1.2)
	Enia et al,[44] 1989‡	0.9 (0.5-1.7)	1.1 (0.6-1.7)
	Von Kodolitsch et al,[29] 2000§	1.7 (1.1-2.5)	0.79 (0.6-0.9)
	Summary	1.4 (1.0-2.0)	0.9 (0.8-1.0)
Enlarged aorta or wide mediastinum	Chan,[43] 1991†	1.6 (1.1-2.3)	0.13 (0.02-1.00)
	Armstrong et al,[45] 1998‖	1.6 (1.1-2.2)	0.42 (0.2-0.9)
	Von Kodolitsch et al,[29] 2000§	3.4 (2.4-4.8)	0.31 (0.2-0.4)
	Summary	2.0 (1.4-3.1)	0.3 (0.2-0.4)
Left ventricular hypertrophy on admission electrocardiogram	Chan,[43] 1991†	0.2 (0.03-1.9)	1.2 (0.9-1.6)
	Von Kodolitsch et al,[29] 2000§	3.2 (1.5-6.8)	0.84 (0.7-0.9)

*CI indicates confidence interval.
†A total of 18 (n = 40) patients with thoracic aortic dissection.
‡A total of 35 (n = 46) patients with thoracic aortic dissection.
§A total of 128 (n = 250) patients with thoracic aortic dissection.
‖A total of 34 (n = 75) patients with thoracic aortic dissection.

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ity, 6%; 95% CI, 3%-13%). Assessment for pulsus paradoxus and jugular venous distension is not enumerated in any of the studies.

Electrocardiographic findings consistent with acute myocardial infarction do not rule out aortic dissection. New Q waves or ST-segment elevation were noted in 7% of admission ECGs (Table 4). Similarly, normal ECGs were documented in 8% to 31% (mean, 22%) of patients.[5,10,11,37,46,47] The remaining ECGs had a variety of other abnormalities including left ventricular hypertrophy, atrial fibrillation, and nonspecific ST-segment changes. As part of the clinical evaluation, ECGs have not been studied well but seem to have little utility for detecting or ruling out thoracic aortic dissection.

Table 4. Sensitivity of the Physical Examination in the Diagnosis of Acute Thoracic Aortic Dissection*

		Sensitivity, %							
Source, y	No. of Patients	Elevated BP	Diastolic Murmur	Pulse Deficit	Pericardial Rub	Congestive Heart Failure	Focal Neurological Deficit	Shock	New MI on ECG
Von Kodolitsch et al,[29] 2000	128	41	40	38	. . .	. . .	13	12	2
Hagan et al,[5] 2000	464	49	32	15	. . .	7	5	16	3
Armstrong et al,[45] 1998	34	. . .	15	12	. . .	. . .	32	26	11
Slater and DeSanctis,[37] 1976	124	36	32	31	. . .	. . .	19	10	3
Levinson et al,[27] 1950	58	66	28	19	5	. . .	16	22	32
Enia et al,[44] 1989	35	. . .	49	49	. . .	. . .	. . .	. . .	. . .
Lindsay and Hurst,[50] 1967	62	29	35	45	. . .	. . .	23	13	. . .
Hume and Porter,[51] 1963	68	68	4	34	. . .	. . .	. . .	10	. . .
Nielsen,[46] 1961	40	. . .	. . .	. . .	. . .	. . .	. . .	30	10
Erb and Tullis,[47] 1960	30	. . .	27	72	0	. . .	13	. . .	25
Pinet et al,[52] 1984	191	. . .	35	55	12	. . .	. . .	38	. . .
Pate et al,[53] 1976	126	37	21	33	. . .	. . .	13	21	. . .
Miller et al,[54] 1979	73	58	64	. . .	. . .	29	12	. . .	. . .
Viljanen,[12] 1986	73	. . .	29	37	. . .	. . .	22	30	. . .
Chan,[43] 1991	18	. . .	22	. . .	. . .	. . .	. . .	. . .	. . .
Strong et al,[55] 1974	59	66	20	34	. . .	. . .	. . .	5	. . .
Sullivan et al,[11] 2000	44	. . .	. . .	12	. . .	. . .	14	. . .	2
Itzchak et al,[49] 1975	24	. . .	. . .	21	. . .	. . .	21	. . .	. . .
Mészáros et al,[10] 2000	66	44	11	20	2	. . .	41	36	9
Pooled sensitivity (95% CI)	NA	49 (41-57)	28 (21-36)	31 (24-39)	6 (3-13)	15 (4-33)	17 (12-23)	19 (15-26)	7 (4-14)

*BP indicates blood pressure; MI, myocardial infarction; ECG, electrocardiogram; NA, not applicable; and CI, confidence interval. Ellipses indicate data not available.

Table 5. Sensitivity of the Plain Chest Radiograph in the Diagnosis of Acute Thoracic Aortic Dissection*

		Sensitivity, %				
Source, y	No. of Patients	Abnormal Aortic Contour	Pleural Effusion	Displaced Intimal Calcification	Wide Mediastinum	Abnormal Chest Radiograph Findings
Von Kodolitsch et al,[29] 2000	128	76†	13	. . .	. . .	. . .
Hagan et al,[5] 2000	427	50	19	14	62	88
Armstrong et al,[45] 1998	34	. . .	. . .	. . .	86	100
Slater and DeSanctis,[37] 1976	116	96	9	9	. . .	96
Luker et al,[48] 1994	75	76	. . .	8	. . .	85
Chan,[43] 1991	18	. . .	. . .	. . .	94	. . .
Pinet et al,[52] 1984	191	. . .	. . .	. . .	56	. . .
Pate et al,[53] 1976	87	. . .	10	. . .	70	90
Earnest et al,[39] 1979	74	66	27	7	11	93
Viljanen,[12] 1986	73	. . .	. . .	. . .	75	. . .
Strong et al,[55] 1974	59	54	. . .	2	34	95
Sullivan et al,[11] 2000	31	42	. . .	. . .	. . .	84
Itzchak et al,[49] 1975	24	88	17	4	83	. . .
Pooled sensitivity (95% CI)	NA	71 (56-84)	16 (12-21)	9 (6-13)	64 (44-80)	90 (87-92)

*CI indicates confidence interval; NA, not applicable. Ellipses indicate data not available.
†Mediastinal and/or aortic widening.

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Accuracy of the Plain Chest Radiograph

Pooling of 13 studies permitted analysis of 1337 radiographs. Only 3 studies commented on the proportion of portable vs conventional radiographs. The proportions of portable radiographs reported in these investigations were 24%, 61%, and 80%.[29,45,48] Radiographic findings classically associated with thoracic aortic dissection are not reliably present (TABLE 5). However, most patients with thoracic aortic dissection do tend to have abnormal findings on chest radiographs (sensitivity, 90%) so that a completely normal radiograph helps to lower the likelihood of the diagnosis. In particular, absence of wide mediastinum and abnormal aortic contour decreases the probability of disease (negative LR, 0.3; 95% CI, 0.2-0.4, Table 5).

Interobserver and intraobserver agreement for physician assessment of radiographs has been reported in 2 studies, both using radiologists as participants. Agreement was generally found to be fair ($\kappa = 0.25$ for intraobserver agreement on suspicion for aortic dissection[48]; $\kappa = 0.23$-0.33 for interobserver agreement on presence of wide mediastinum, irregularities of the aortic contour, and pleural effusion[40]). These low rates of interobserver agreement underscore the lack of validated standards for defining the radiographic features of aortic dissection.

Table 6. Positive Likelihood Ratio of Aortic Dissection in Patients With Combinations of Findings*

No. of Findings	Positive Likelihood Ratio (95% Confidence Interval)
0	0.1 (0.0-0.2)
1	0.5 (0.3-0.8)
2	5.3 (3.0-9.4)
3	66.0 (4.1-1062.0)

*Data from Von Kodolitsch et al.[29] Findings include aortic pain (severe, sudden-onset tearing pain), blood pressure or pulse differential between arms, and/or wide mediastinum on chest radiograph.

Accuracy of Combinations of Findings

Most clinical findings associated with thoracic aortic dissection are insensitive when considered in isolation. Combinations of findings, though not often found, markedly increase the accuracy of clinical assessment for thoracic aortic dissection. The single level 3 study described increasing accuracy of progressive combinations of findings (TABLE 6).[29] For example, aortic pain alone (pain of sudden onset, tearing, or ripping in character or both) has a positive LR of 2.6; the presence of both aortic pain and pulse or blood pressure differentials increases the positive LR to 10.5 (95% CI, 1.4-80.1). Further addition of mediastinal or aortic widening on chest radiograph clinches the diagnosis with a positive LR of 66.0 (95% CI, 4.1-1062.0). Unfortunately, this diagnostically valuable triad was present in only 27% of patients. Conversely, patients without any findings from the triad (aortic pain, pulse of blood pressure differential, and mediastinal widening) are unlikely to have a thoracic aortic dissection given a negative LR of 0.07 (95% CI, 0.03-0.17). However, 4% of patients in this category, without any of the above signs, were nonetheless ultimately diagnosed with aortic dissection. Given the high morbidity of a missed diagnosis, even such a pronounced negative LR is insufficient to defer diagnostic imaging if thoracic aortic dissection is still clinically suspected.

The improved accuracy of combinations of clinical findings may further be inferred from a holistic view of the 4 studies that selected patients for inclusion on the basis of an overall clinical picture suggestive of thoracic aortic dissection. Despite the relative rarity of thoracic aortic dissection compared with other acute causes of pain, approximately half the patients selected for these studies turned out to have thoracic aortic dissection (pooled sensitivity, 52%). By comparison, only 0.003% of patients presenting to an emergency department with acute back, chest, or abdominal pain are eventually diagnosed with dissection.[29] This implies that a full clinical history, examination, and radiograph substantially selects for patients with acute dissection. Furthermore, among patients

Table 7. Final Diagnoses in Patients With Clinical Syndromes Suggestive of Thoracic Aortic Dissection But Without Thoracic Aortic Dissection on Further Study*

Diagnosis	No. (%) of Patients			
	Von Kodolitsch et al,[29] 2000 (N = 122)	Enia et al,[44] 1989 (N = 11)	Armstrong et al,[45] 1998 (N = 41)†	Eagle et al,[33] 1986 (N = 51)‡
Acute coronary syndrome	18 (15)	2 (18)	8 (20)	12 (24)
Chest wall syndrome	18 (15)	. . .	. . .	. . .
Mediastinal cyst or tumor	. . .	. . .	. . .	4 (8)
Neuroradicular syndrome	1 (0.8)	. . .	. . .	. . .
Pulmonary disease	1 (0.8)	. . .	. . .	. . .
Hypertensive crisis	11 (9)	. . .	. . .	. . .
Gastrointestinal disease (esophagitis, PUD, gastritis, pancreatitis)	12 (9.8)	. . .	. . .	2 (4)
Pneumothorax	2 (1.6)	. . .	. . .	. . .
Pulmonary embolism	6 (4.9)	1 (9)	. . .	1 (2)
Pleuritis	5 (4.0)	. . .	. . .	1 (2)
Pericarditis	7 (5.7)	4 (36)	3 (7)	3 (6)
Nondissecting aneurysm	. . .	1 (9)	13 (32)	4 (8)
Aortic plaque rupture and/or intramural hemorrhage	. . .	. . .	9 (22)	. . .
Valvular pathology	. . .	. . .	4 (10)	5 (10)
Arteriosclerotic emboli	. . .	. . .	. . .	1 (2)
No definitive diagnosis	4 (3.3)	3 (27)	14 (34)	14 (28)

*PUD indicates peptic ulcer disease. Ellipses indicate data not available.
†Some patients without thoracic aortic dissection were given multiple diagnoses.
‡Included 55 patients with suspected thoracic aortic dissections but negative aortograms; 4 patients were false negative cases and later demonstrated to have thoracic aortic dissection.

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referred for aortic imaging who turn out not to have an acute dissection, approximately half to three quarters are diagnosed with alternative serious diseases that can potentially be identified by imaging intended to confirm the diagnosis of thoracic aortic dissection (TABLE 7).[29,33,43-45,59] The clinical syndrome suspicious for thoracic aortic dissection, while far from pathognomonic for acute dissection, does detect patients with serious disease that merit advanced diagnostic imaging.

THE BOTTOM LINE

Despite the large number of case series describing patients with thoracic aortic dissection, the clinical examination for thoracic aortic dissection has yet to be prospectively scrutinized in an independent, blinded study. The extant data permit estimation of the sensitivity of clinical history, physical examination, and plain chest radiography but likely overestimates the accuracy of the clinical examination by selectively including more obvious cases. A small number of studies have included control populations and may therefore estimate the specificity of components of the clinical examination; however, the accuracy of these data is again limited by the lack of independence between the selection of patients for study and clinical findings.

Given the high, rapid mortality associated with undiagnosed thoracic aortic dissection, prospective, independent studies of the clinical examination are needed to aid physicians in determining which aspects of the clinical examination ought to be relied upon to refer patients rationally for further diagnostic studies. Until then, the current literature permits the following limited conclusions about the clinical examination:

- Most patients with thoracic aortic dissection have severe pain of abrupt onset. The absence of pain of sudden onset substantively decreases the probability of dissection (negative LR, 0.3; 95% CI, 0.2-0.5); however, the study design of the reports included in this article precludes accurate assessment of the sensitivity and specificity of these features. The presence of tearing or ripping pain (positive LR, 1.2-10.8) or pain that migrates (positive LR, 1.1-7.6) may prove useful, but additional data are required to know whether they are reliable features of the clinical history.
- Physical findings associated with thoracic aortic dissection tend to be present in a third or fewer cases; however, pulse deficits (positive LR, 5.7; 95% CI, 1.4-23.0) or focal neurological deficits (positive LR, 6.6-33.0) greatly increase the likelihood of thoracic aortic dissection in the appropriate clinical setting. The presence or absence of a diastolic murmur is not useful (positive LR, 1.4; negative LR, 0.9).
- A normal aorta and mediastinum on chest radiograph helps exclude the diagnosis (negative LR, 0.3; 95% CI, 0.2-0.4) but no particular radiographic abnormality is dependably present.
- The presence of the above findings in combination increases the positive LR for dissection but the absence of even multiple findings does not definitively exclude the diagnosis. Clinical history, examination, and radiography can help rule in aortic dissection but are not sufficiently accurate to rule out the disease.

SCENARIO RESOLUTION

Case 1

The patient's clinical history of sudden onset of severe chest pain is worrisome. His history of hypertension slightly increases his risk of a thoracic aortic dissection. The absence of a diastolic murmur, blood pressure differential, neurological deficit, and widened mediastinum does not reliably exclude the diagnosis of thoracic aortic dissection. Given the high mortality of untreated or mistreated thoracic aortic dissection, this patient merits further advanced imaging.

Case 2

The presence of a neurological deficit in a patient with a clinical history consistent with thoracic aortic dissection is a specific finding. This patient has a very high likelihood of having an acute thoracic aortic dissection and ought to undergo urgent diagnostic imaging to locate and delineate the suspected lesion.

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