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Painless dissecting aneurysm of the thoracic aorta:

Report of eight cases masquerading as gross aortic insufficiency, severe hypertension, myocardial infarction and mediastinal enlargement.

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In a review of 68 patients with dissecting thoracic aorta aneurysm (DTAA), eight patients (11.7%) were found to have painless dissection. Three of the eight patients presented with gross aortic insufficiency (AI). Angiography on a fourth patient, who had severe hypertension, showed Type III DTAA. Three other patients had either dilatation of the ascending aorta or a possible mediastinal mass, according to chest roentgenograms. One patient received treatment for suspected myocardial infarction with cardiogenic shock. A high index of suspicion in patients with unexplained AI, severe hypertension and a mediastinal mass or dilatation of the ascending aorta, coupled with the early use of angiographic studies, will assist in establishing the diagnosis and decide the most appropriate management.

PAIN, often severe, unbearable and unresponsive to average analgesic dosages, is a common symptom affecting 85% to 90% of the patients with thoracic aorta dissection. Without noticeable pain, especially of the typical piercing or ripping nature, the diagnosis of this lethal disorder may be overlooked. Reviewing 68 cases of dissecting aneurysm of the aorta treated at Henry Ford Hospital from 1964 to 1975, we found eight patients (11.7%) free of any significant pain, according to their clinical history. In all of these patients, the absence of pain was a major factor in the delayed establishment of the correct diagnosis.

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Case 1

A 46-year-old black male had been in apparent good health until March 17, 1971, when during a routine check-up his family physician first noted a heart murmur. The man was well until two months prior to his admission to the Henry Ford Hospital in May, 1973, with symptoms of congestive heart failure. At no time did the patient experience any chest pain.

Examination showed a patient in moderate distress with orthopnea. The neck veins were distended at a 30° Fowler’s position. There were rales in both lung bases. The heart revealed a wide thrusting apical impulse at the anterior axillary line, sixth intercostal space. A systolic thrill and palpable lift were present over the second right intercostal space. A Grade IV/VI ejection murmur was best heard over the aortic area radiating to the carotid arteries and to the precordium. The second aortic sound was markedly decreased and was followed by a Grade II/VI diastolic blow which radiated to the left sternal border and to the apex. The blood pressure was 150/70-60 mmHg on both arms. All the peripheral pulses were palpable and equal. The liver was palpable 4 cm below the costal margin. Both legs had + edema.

Figure 1. Case 1 A
Aortic angiogram showing the catheter tip in the true channel which is heavily opacified by contrast material. The arrows outline its separation from the false lumen.
Painless dissecting aneurysm

Laboratory data: Cardiac catheterization revealed moderate pulmonary hypertension of 54/20 mmHg, pulmonary wedge pressure 13 mmHg, cardiac index 2.2 L/min/M^2, central aorta pressure 156/50 mmHg, and left ventricular pressure 158/3-10-20 mmHg. An electrocardiogram showed left ventricular enlargement. A chest roentgenogram revealed cardiomegaly that was predominantly left ventricular and dilatation of the ascending aorta. An aortic root angiogram showed a double aortic channel beginning at the valve and gross aortic insufficiency (Figure 1). The dissection extended to the beginning of the descending aorta, but the distal portion was not definitely seen.

Once the diagnosis was established, the patient was advised to have an operation, in view of the gross aortic insufficiency and congestive failure. The patient refused and has since been on a medical treatment program which includes salt restriction, diuretics, rawolfia preparation, alphamethyldopa, and digoxin. His condition has remained stable, free of congestive failure, but he is unable to work and his activities for over three years have been markedly restricted.
Case 2

A 61-year-old black male with a six-year history of hypertension first developed symptoms of transient cerebral ischemic attacks on October 12, 1970. These were characterized by dizziness and slurred speech. Chest roentgenogram revealed a normal cardiovascular silhouette. All the peripheral pulses were palpable and equal. On November 24, 1972, he developed symptoms of congestive heart failure, but denied having chest pains. On January 25, 1973, he experienced intermittent claudication of the left lower extremity associated with a systolic bruit and decreased pulsations of the left femoral artery. Examination of the heart revealed a harsh Grade III/VI ejection murmur over the aortic area, radiating to the carotid arteries followed by a Grade II/VI diastolic blow best heard along the left sternal border. There was a S3 gallop sound at the apex. A systolic thrill was present at the suprasternal notch. The blood pressure was 140/60 mmHg and the heart rate was 80 per minute with an occasional premature ventricular beat. The lungs were clear to percussion and auscultation.

Figure 2. Case 2
Aortic root angiogram showing the heavily opacified false channel containing the catheter tip (open arrow) and the septum (black arrows) which separates it from the true channel.
Painless dissecting aneurysm

Laboratory data: Electrocardiogram revealed left ventricular and left atrial enlargement. Chest roentgenogram showed generalized cardiomegaly. Cardiac catheterization on February 9, 1973, showed mild elevation of the pulmonary artery pressure to 32/12 mmHg, the central aortic pressure was 145/60 mmHg, and the cardiac index was 2.4 L/min/M². An aortic root injection revealed dissection of the ascending aorta with involvement of the aortic valve (Figure 2). The distal extent could not be determined. There was indirect evidence of aortic insufficiency as shown by the to and fro motion of the contrast material in the descending aorta. The left ventricle could not be entered from the aorta. In view of the findings of a Type I dissection of the ascending thoracic aorta, surgical repair was offered to the patient but he declined. He was treated medically and was re-admitted on May 30, 1974, with increasing symptoms of congestive failure. He again refused surgery. After two years, the patient was lost to follow-up as of January 8, 1975.

Case 3

A 54-year-old Caucasian male carpenter, who was in apparent good health until three weeks prior to admission, complained of exertional dyspnea and orthopnea associated with intermittent fever and hoarseness. He had no history of chest pains. A complete physical examination six months earlier was unremarkable.

During examination, rales were heard in both lung bases. Paralysis of the left vocal cord was initially attributed to aortic aneurysmal dilatation. The apical impulse of the heart was 10 cms to the left of the mid-sternal line. There was a Grade IV/VI ejection murmur over the precordium that was best heard over the aortic area and radiated to the carotid arteries. A Grade III/VI diastolic blow was heard at the aortic area and along the left sternal border. The blood pressure was 112/60-40 mmHg in both arms.

Figure 3. Case 3

Aortic angiogram showing the true lumen more heavily opacified than the false channel. The arrows outline the poorly defined septum between both channels.
Laboratory data: Chest roentgenogram demonstrated an enlarged left ventricle. Cardiac catheterization revealed moderate pulmonary hypertension of 42/16 mmHg and a wedge pressure elevation of 20 mmHg. Cardiac index was 2.6 L/min/m², central aorta pressure 128/48 mmHg and left ventricle 132/0-26-44 mmHg. The aortic root angiogram showed gross aortic regurgitation and the left ventriculogram showed one plus mitral insufficiency. The chest roentgenogram revealed a bilateral nodular infiltrate in the perihilar areas with a “bat-wing” configuration and bilateral pleural effusions compatible with congestive heart failure.

On March 2, 1964, the patient underwent operation for gross aortic insufficiency of unknown etiology, but possibly secondary to valve destruction from endocarditis. At operation, an unexpected dissecting aneurysm of the ascending aorta was found with an intima tear located 4 cms above the aortic valve. The aortic leaflets were normal. He had an uneventful recovery from the surgical repair of the dissection with a Dacron prosthesis.

The patient did well until four months postoperatively when he developed upper thoracic spine and left shoulder pain. Roentgenograms failed to reveal any osseous metastases. A chest roentgenogram showed a lobulated mass in the left lung apex which, in retrospect, was also present in previous films. At thoracotomy an undifferentiated carcinoma of the left lung with involvement of the hilum was found. He was treated with cobalt therapy to no avail and expired on January 29, 1965, as a result of the lung carcinoma. Prior to death, his cardiac condition was good, with an ejection murmur along the left sternal border. A retrospective review of the cineangiogram showed evidence of dissection of the ascending aorta (Figure 3).

Case 4

A 58-year-old Caucasian male, who was admitted to the hospital with a four-day history of decreased urinary output and malignant hypertension, claimed no history of chest or abdominal pain. At examination, his blood pressure was 300/175 mmHg and a Grade III/VI ejection murmur was heard over the precordium.

Laboratory data: A renal arteriogram, performed on August 16, 1968, showed patent renal arteries and raised the question of dissecting aneurysm. The arch aortogram showed the presence of a large dissecting thoracic aorta aneurysm with the point of entrance in the mid-thoracic area extending distal to the diaphragm. The patient was treated medically in an attempt to control his hypertension, but he developed progressive renal failure and expired from uremia three years and six months after the diagnosis was established.

Figure 4. Case 6

Noncontinuous monitor electrocardiographic lead showing marked slowing of the rate with a shift to a lower pacemaker on strip 2 (probable A-V junctional rhythm). On strip 5 standstill follows the isolated aberrant beat.
Painless dissecting aneurysm

Case 5
This 72-year-old Caucasian male was known to be diabetic and hypertensive for 19 years. A thoracic aorta aneurysm was suspected during a routine chest roentgenogram. He had no history of chest pain. The angiogram confirmed the presence of dissection of the ascending aorta including the arch. Because of his age and lack of aortic valve involvement, he was treated medically and did well for four years, but then was lost to follow-up.

Case 6
A 62-year-old Caucasian male was admitted to the coronary care unit because of sudden onset of weakness, hypotension, diaphoresis, and mild epigastric discomfort with emesis. Symptoms occurred shortly after lunch. He was a known hypertensive for one year. On admission, the patient was diaphoretic and cyanotic. His blood pressure was 94/70 mmHg with pulse rate of 110/minute. The heart sounds were distant. No murmurs or gallops were heard. Serial electrocardiograms revealed ST-T changes in leads I, AVL and V4, V5 compatible with subendocardial or non-transmural ischemia or infarction. The serial creatine-phosphokinase measurements were 50, 150 and 85 units; the serum glutamic-oxalacetic transaminase levels were 2,250, 8,400 and 6,200 units; the serum lactic dehydrogenase levels were 1,360, 1,760 and 1,360 units. The hemoglobin was 16.0 gms%, the leukocyte count was 26,000 per cubic mm with 86% neutrophiles. Treatment was given for probable myocardial infarction with

Figure 5. Case 7 A
Postero-anterior chest film showing cardiomegaly and mediastinal widening.
Figure 5. Case 7 B
Lateral view showing the anterior prominence of the mediastinal widening.
Painless dissecting aneurysm

cardiogenic shock and azotemia. Death three days later followed asystole preceded by marked sinus bradycardia unresponsive to isoproterenol and atropine (Figure 4). At autopsy a dissecting aneurysm of the ascending aorta was found extending to its root with cardiac tamponade with a hematoma of the lower portion of the interatrial septum. The coronary arteries showed severe atherosclerosis but no obstruction was present. No definite evidence of acute myocardial infarction was found.

Case 7

A 52-year-old black woman with a three-year history of labile hypertension was admitted to the hospital on February 24, 1969, because of a one-year history of persistent nonproductive cough associated with intermittent loss of voice and shortness of breath. She gave no history of chest pain. The examination of this obese woman in mild respiratory distress showed bilateral rhonchi, wheezing and basilar rales in the lungs. There was

Figure 6. Case 7
Aortic angiogram showing a large ascending aortic aneurysm.
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no vocal cord paralysis. The heart had a Grade II/VI ejection murmur best heard at the left sternal border. Blood pressure was 230/140 mmHg in the right arm and 200/130 mmHg in the left arm, and the pulse rate 96/minute and regular. Peripheral pulses were palpable and equal bilaterally.

The chest roentgenogram revealed a large anterior mediastinal mass (Figure 5). The radiologist thought this was not related to the aorta but suggested additional studies. A thoracic aortogram showed a large ascending aortic aneurysm (Figure 6) which at operation was confirmed to be dissecting in nature. Following surgery, the patient developed cardiogenic shock and expired several hours later with intractable pump failure.

Case 8

A 45-year-old black male, with known hypertension for two years, was found to have dilatation of the ascending aorta by chest roentgenograms. He had sustained a stab wound of the left anterolateral chest when hospitalized on August 10, 1975; he reported no past history of chest pain nor angina.

At examination, his blood pressure was 140/90 mmHg in both arms. Auscultation of the heart revealed a Grade II/VI ejection murmur over the left sternal border followed by a Grade II/VI descrescendo diastolic murmur. The peripheral pulses were palpable and equal bilaterally. The

Figure 7. Case 8
Aortic angiogram showing the heavily opacified true channel (arrows) compressed anteriorly and medially by the large false channel.
Painless dissecting aneurysm

Patient was treated uneventfully with a thoracotomy for a left hemothorax. Following his recovery he underwent a left cardiac catheterization which revealed a central aorta pressure of 150/80 mmHg and a left ventricular pressure of 150/4-8-14 mmHg. The thoracic aortogram showed a dissection of the aorta (Figure 7) starting just above the ring of the aortic valve and extending distally to the left common femoral artery. A 1 to 2+ aortic valve regurgitation was present. The left ventricular contractions were poor to fair. The coronary arteries were normal. The electrocardiogram revealed left ventricular hypertrophy by voltage criteria. Following thoracic surgery consultation, it was decided to treat the patient medically with careful control of hypertension and close follow-up for possible early operation if symptoms or clinical findings should deteriorate.

Discussion

Painless dissecting aneurysm of the aorta represents a diagnostic challenge because it masquerades as numerous clinical syndromes. These result from the great variety of branch vessels that may be involved as well as by the degree of compression of the organs in the vicinity of the expanding hematoma and their proximal extension with aortic valve deformity. The most extensive review of the literature by Hirst et al., 1 349 of 409 cases (85%) of dissecting aneurysms of the aorta were recorded to have pain at the onset of symptoms. In two more recent series 56 of 62 cases (90%) published by Lindsay and Hurst,1 and 41 of 45 patients (89%) of the group studied by Dalen et al.3 suffered from significant pain. Needless to say, a detailed history is mandatory in order to rule out a possible antecedent bout of significant pain easily forgotten by patients with chronic dissection.

In our present report of painless thoracic aorta dissections, three of the eight patients presented with the clinical manifestations of gross aortic insufficiency and varying degrees of congestive heart failure. The diagnosis was established by angiography in the first two cases and unexpectedly discovered at surgery in the third case. The finding of a murmur of aortic regurgitation not present in previous examinations, particularly when accompanied by prominent systolic pulsations and thrill at the aortic area and a history of hypertension, is highly diagnostic of dissection. In cases of unexplained aortic insufficiency, a careful study of the angiograms, looking specifically for the intimal tear with the dividing septum and double lumen, could prevent an erroneous diagnosis as it occurred in our third patient. The absence of a definite tear or double lumen would favor the diagnosis of aneurysm due to cystic medial necrosis without dissection in some instances.

Autopsy revealed Type I dissection of the ascending aorta and cardiac tamponade in still another patient admitted to the coronary care unit in cardiogenic shock with abnormal electrocardiogram and serum enzymes, but without significant pain. It is not uncommon that patients who have acute dissection of the ascending aorta are frequently thought to have an acute myocardial infarction on the initial examination and this possibility should always be kept in mind in the differential diagnosis. Another patient who was admitted with severe hypertension was discovered to have dissection of the descending aorta during angiographic studies performed as part of the hypertensive work-up. In three additional cases, a mediastinal or ascending aorta dilatation associated with long-standing hypertension was the diagnostic clue that prompted angiographic confirmatory studies of aortic dissection. A review of serial chest films to detect the increasing aortic size is very helpful. As suggested by Cohen and Littman,4 dissection of the aorta can occur with few clinical clues and those that are present can be misleading. A high index of suspicion in patients with unexplained aortic insufficiency, severe hypertension, dilatation of the ascending aorta and/or anterior mediastinal mass coupled with the early use of angiographic studies will assist in establishing an early diagnosis and in the selection of the most appropriate management.
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References