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DISSECTING ANEURYSM OF THE AORTA

A Review

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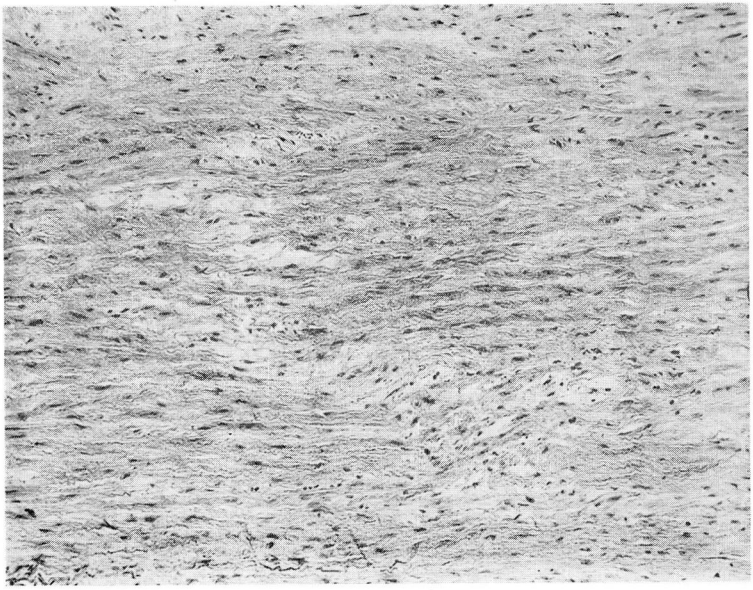
DISSECTING ANEURYSM OF THE AORTA is an intramural separation of the aortic wall by circulating blood that has penetrated and extended into the media. The process may arrest at this stage and heal, progress immediately, or extend at some later date. As the dissecting aneurysm progresses, it may rupture through the adventitia into the pericardial, pleural, or abdominal cavities and may obstruct arteries arising from the aorta. It may rupture back into the lumen and form a double-barreled aorta, which may persist, or the aneurysm may be filled with clot and subsequently heal, obliterating the secondary channel.

Etiology

Although the specific cause of the disease is not known, the lesion that generally is accepted as being precedent to dissecting aneurysm of the aorta is



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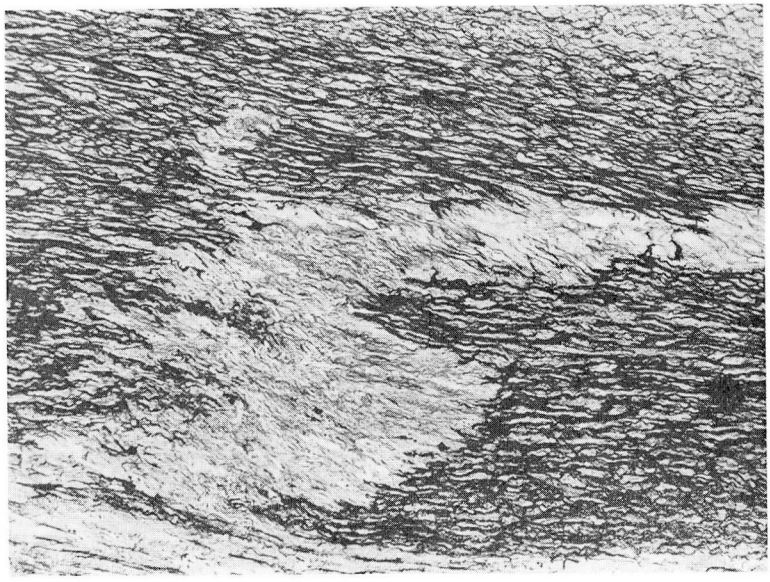

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Fig. 1. Changes in the media. (**A**) Medial degeneration (mucinous); hematoxylin, eosin and methylene blue stain, X80. (**B**) Marked loss of elastic tissue in a corresponding area; Verhoeff's stain, X80.

medial degeneration. This most often consists of mucoid changes in the media and focal loss of elastic tissue (Fig. 1); degeneration of muscle also may occur. The intimal rupture, almost universally present, may vary in size from a small defect a few millimeters in diameter to a large and nearly circumferential laceration. Some believe that the initial lesion may be hemorrhage into degenerated media derived from rupture of vasa vasorum. The originating point (Fig. 2) of dissection is predominantly in the ascending aorta, within a few centimeters of the aortic valve. It may occur in the regions of the ligamentum arteriosum and the left subclavian artery. The intramural dissection (Fig. 3) is of variable length and occurs mostly in the outer third of the media (Fig. 4), a circumstance permitting ready access to the adventitia and therefore possible rupture.

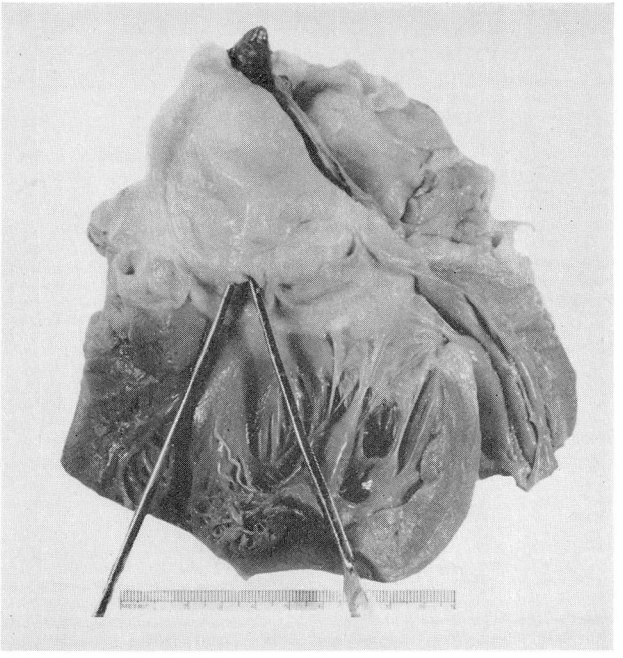


Fig. 2. Area of laceration of intima (marked by probes) at upper margin of aortic sinus.

The precedent disease and weakening of the media is of basic importance in the formation of dissecting aneurysm; however, no single feature may be responsible for all cases. Several factors are worthy of consideration regarding etiology.

Degenerative change. Degeneration of elastic tissue and other medial elements has been a common finding in dissecting aneurysm. Gore[1] has found that the deterioration of the elastic tissue is most common in those patients under 40 years of age. Erdheim[2] described a type of medial degeneration that he designated *medionecrosis aortae idiopathica cystica*, in which accumulations of

mucoid material were considered of primary importance. The significance of this finding is now generally recognized but it has not been observed constantly: Bauersfeld[3] found no cystic medionecrosis in a series of 15 dissecting aneurysms, and Glendy, Castleman, and White[4] found it in 6 of 13. Rottino[5] recognized it as being present but did not accept it as antecedent to the aortic medial degeneration he observed. In three completely studied cases, Moritz[6] found chromo-

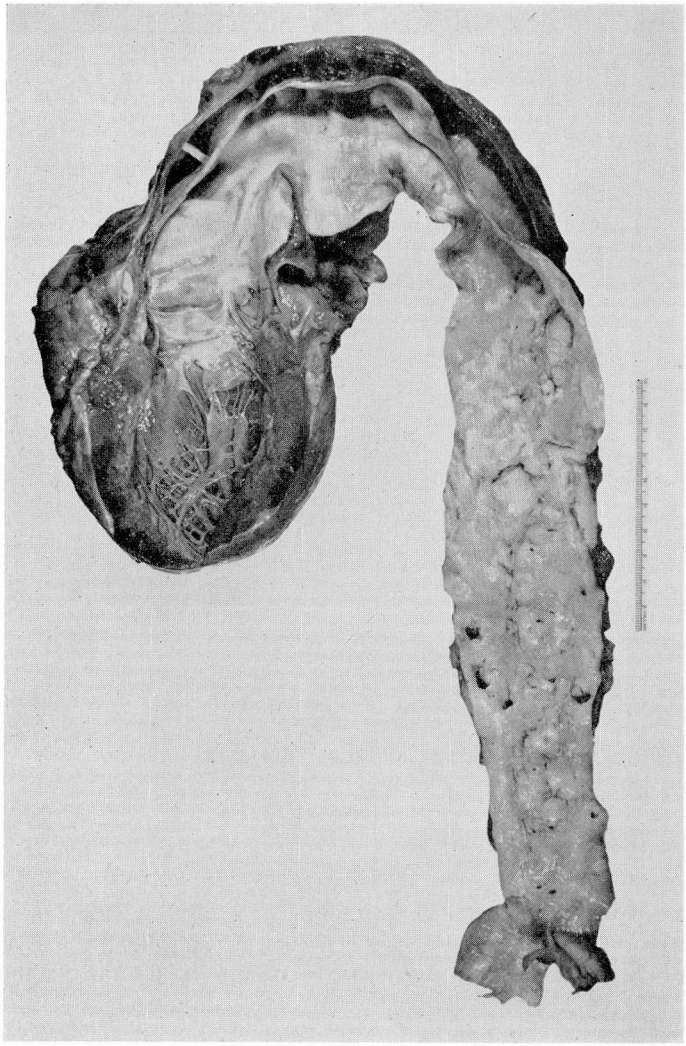
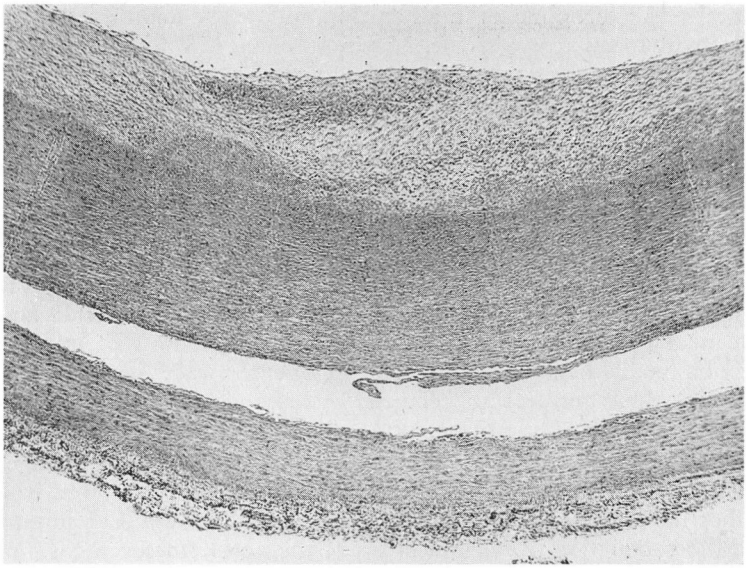


Fig. 3. Intramural dissection in aortic arch showing separation of vascular wall into two leaves. The dissecting aneurysm extended to the level of the diaphragm and had ruptured into the pericardium.



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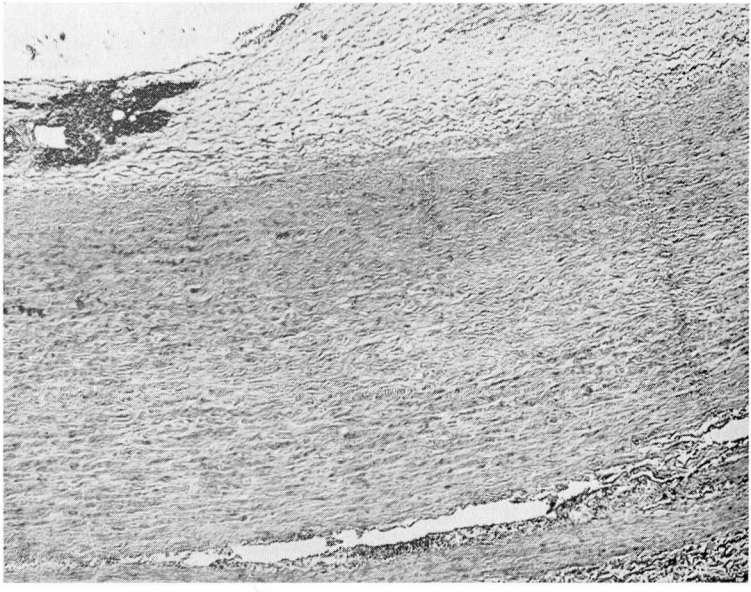

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Fig. 4. Channels of dissection. (**A**) Wide split near outer third of media; hematoxylin, eosin and methylene blue stain, X25. (**B**) Margin of channel in outermost media near adventitia. A focus of hemorrhage is present in the thickened intima; hematoxylin, eosin and methylene blue stain, X50.

tropic substance in the aortic media in all and, in addition, demonstrated it in the wall of the pulmonary artery in one. Mucinous change of a milder degree than observed in the dissecting aneurysms was found in 10 per cent of adult aortas and appeared to increase in intensity with advancing age. The underlying cause of medial degeneration is not known. Gore[1] has suggested that it may be a metabolic defect involving elastic tissue. Experimental evidence indicates that impairment of the blood supply to the aortic wall can lead to cystic degeneration as shown by Schlichter[7] who found definite cystic degenerative changes in the media of the aorta in dogs, one to six weeks old, after searing the adventitia.

Rottino[5] studied 12 cases of aortic dissecting aneurysm and found the medial degeneration to be characterized by loss of muscle, elastic tissue, and collagen. The medial changes predisposed to rupture of the vasa vasorum and the formation of a medial hematoma that split the diseased wall. An inflammatory reaction was lacking and healing occurred by loose scar formation and by regeneration of muscle and elastic tissue.

Amromin, Schlichter, and Solway[8] emphasized the importance of changes in the vasa vasorum, finding them sclerotic, hypertrophic, and narrowed in 7 of 12 cases. They believed that the resultant ischemia of the media is an important factor in the production of medionecrosis of the aorta. In seven cases of dissecting aneurysm of the aorta McCloskey and Chu[9] found alterations in the vasa vasorum consisting of medial hypertrophy and splitting of the internal elastic membrane of small arteries, medial hypertrophy with intimal proliferation and endothelial swelling of the arterioles, and endothelial swelling and proliferation of the capillaries.

Congenital anomalies. Coarctation of the aorta commonly is associated with medial degeneration of the aortic wall. Hamilton and Abbott[10] in a review of autopsy findings in 200 cases of coarctation of the aorta found that 16.5 per cent of the patients had died from aortic dissecting aneurysm. Schnitker and Bayer[11] reported that 31.9 per cent of 141 patients with dissecting aneurysm of the aorta, less than 40 years of age, had either true coarctation or definite narrowing of the aorta. A congenital bicuspid aortic valve may be found with dissecting aortic aneurysm. Gore[1] found this association in 9 of 32 patients less than 40 years of age.

Arachnodactyly also has been reported to predispose to dissecting aneurysm of the aorta.[12]

Hypertension. Hypertension usually is present in patients who develop dissecting aneurysm of the aorta, but many investigators regard it as of secondary importance. David, McPeak, Vivas-Salas, and White[13] found dissecting aneurysm of the aorta in 17 patients, all of whom had hypertension (blood pressure above 140/90 mm. Hg). Schnitker and Bayer[11] reviewed 580 case reports of patients having dissecting aneurysm and found the blood pressures often were not recorded, but of the 44 patients less than 40 years of age, on whom such determinations were made, only 50 per cent were hypertensive. It is doubtful that even an extremely high blood pressure could cause dissection of the aorta, inasmuch as Klotz and Simpson[14] found at autopsy that a pressure of more than 1000 mm. Hg was necessary to rupture an aorta.

Dissecting aneurysm of the aorta occurring in hypertensive patients during methonium therapy recently has been reported by Beaven and Murphy.[15] Necropsy findings in 44 hypertensive patients who died after methonium or pentolinium therapy showed that nine had dissecting aneurysm of the aorta, an incidence that the authors believed was beyond chance expectation. Of the 44 patients, 34 had malignant hypertension and 10 benign hypertension. Of the nine patients with dissecting aneurysm, six had had malignant hypertension and three benign hypertension. The authors suggested three possible explanations of this association: (1) Treatment prolonged life and allowed this complication to develop; (2) the fluctuations in the blood pressure encouraged the development of dissecting aneurysm; and (3) the hypotensive agents possibly had a specific biochemical effect on the aorta. Present experience suggests that the first of these explanations is probably the best.

Atherosclerosis. Atherosclerosis is present in the majority of cases of dissecting aneurysm of the aorta, but probably is of only minor etiologic importance inasmuch as it is such a common pathologic change in the aorta. Atherosclerosis is a disease primarily of the subintimal connective tissue and extends to involve the media adjacent to the intima, while a dissecting aneurysm involves primarily the outer portion of the medial layer. Bauersfeld[3] has observed that when intimal tears from a dissecting aneurysm are present in association with atherosclerosis they usually are found between two atherosclerotic plaques rather than in them. An intimal tear usually is located within the first few centimeters of the ascending aorta, an area less commonly involved with atherosclerotic plaques than is the rest of the aorta.

Pregnancy. In women less than 40 years of age there appears to be a distinct relationship between pregnancy and dissecting aneurysm of the aorta. Schnitker and Bayer[11] found that 35 per cent of the reported patients less than 40 years of age who had dissecting aneurysm of the aorta were women, and 49 per cent of those women were pregnant. In 20 women the dissection occurred antepartum, in 2 postpartum, and in only 2 at the time of labor.

Granulomatous giant-cell aortitis. Two cases of dissection with rupture of the aorta have occurred apparently as a direct complication of granulomatous giant-cell aortitis. Magarey[16] described a 75-year-old woman who allegedly was in good health prior to an unexpected and sudden death on a commode, and McMillan[17] described a 58-year-old man who died shortly after developing a left hemiparesis. Histologically in both instances medial changes were predominant and consisted of granulomatous reaction with giant-cell formation in the areas of destruction and an infiltration of lymphocytes and plasma cells. Giant cells formed a striking feature and were observed to be in close relation to fragments of elastic tissue.

Syphilis. Syphilis at one time had been suggested as a possible predisposing factor but is no longer so regarded. In their review of cases of dissecting aneurysm, Schnitker and Bayer[11] found only five (3.5 per cent) with a history of syphilis. Shennan[18] reported an incidence of 10 per cent in a series of 218 cases. Weiss[19] has pointed out that syphilitic aortitis tends to fuse the layers of the aorta, thereby actually hindering dissection. In Bauersfeld's[3] series of dissecting

aneurysms there were two cases with syphilitic involvement of the aorta, in each of which dissection, involving the ascending aorta, stopped at the beginning of the syphilitic aneurysm.

Incidence

The true incidence of dissecting aneurysm of the aorta is unknown. Only 30 cases were seen at the Massachusetts General Hospital in 49 years (1897 to 1946).[13] The incidence at autopsy is reported as about 1 in 500 cases. Regarding the age of the patients, the greatest frequency of dissecting aneurysm appears to be between the fifth and seventh decades of life; however, cases have been reported in which the age incidence ranged from 14 months to 100 years. Schnitker and Bayer[11] found that 24 per cent of their patients were less than 40 years of age. The sex ratio for occurrence of dissecting aneurysm is about 1 to 3 in favor of males.

Clinical Features

Symptoms. Pain is by far the most frequent symptom. It occurs suddenly and usually is severe from the onset. Patients describe it as sharp, burning, searing, or tearing. Usually it is not related to physical effort or activity and it is located in the anterior part of the chest, in the epigastrium, or frequently in both regions. Pain also may begin in or spread to the intercapsular region, neck, midback, flank, or sacrum, and occasionally it may extend into the upper or lower extremities.

Syncope may be the initial symptom but when the patient regains consciousness there usually is severe pain. A few cases have been reported in which syncope and pain were absent. The mechanism of syncope has been attributed to involvement of the depressor nerve endings in the aortic arch, and to cerebral ischemia secondary to pooling of large quantities of blood in the false passage of the aorta or, more commonly, to blockage or dissection of the innominate and common carotid arteries. Neurologic symptoms occur in about one half of the cases and symptoms of acute arterial insufficiency are frequent. Other symptoms may occur and include dyspnea, nausea, vomiting, orthopnea, oliguria, hemoptysis, ankle edema, melena, hematemesis, and hematuria.

Physical findings. There are few pathognomonic signs. Nissim[20] considers a reduplicated pulsation in an artery to be diagnostic. It is believed to be due to a difference in the rate of propagation of the pulse wave through the lumen of the involved artery because of its dissected coats. Shennan[18] has described a delay in conduction of the pulse beat, which he considers significant. In dissecting aneurysm of the aorta, the delay in the pulse beat when compared with the apical beat can occur in all arteries because of a weakening of the pressure of the blood stream passing through the involved aorta.

From the nature of dissecting aneurysm of the aorta it is easy to understand the lesser or greater involvement of various parts of the body. A patient having this condition usually is in acute distress and frequently appears to be in shock,

but the blood pressure is within normal limits or may be elevated. The pulse rate not infrequently is normal or slow and the temperature ranges between 99 and 103 degrees F. if the patient survives more than 48 hours.

Since most dissecting aneurysms begin in the ascending portion of the aorta, the heart may be involved in a variety of pathologic processes. Myocardial infarction may occur when there is obliteration of the coronary arteries by proximal extension of the dissection. Pericarditis occurs in approximately one third of the cases: it may result from an adventitial tear in the intrapericardial portion of the aorta, allowing a seepage of blood into the pericardial sac which may cause cardiac tamponade and death, or it may result from uremia if the dissecting aneurysm has occluded both renal arteries. Aortic insufficiency occurs in approximately 25 per cent of these patients. Cardiac enlargement from pre-existing hypertension usually is present.

Various pulmonary changes may occur when the dissection involves the thoracic aorta distal to the pericardial reflection. Pleural effusion is a relatively common finding and usually occurs on the left side. There are two explanations for its frequent occurrence on the left side: the aorta is in close proximity to the left pleural cavity and when there is rupture through the adventitia in this region it usually results in hemothorax; and dissection along the pulmonary artery may occur following the line of least resistance, which is along the left main pulmonary artery. Pulmonary congestion and atelectasis are frequent findings.

When neurologic manifestations are present they often facilitate the diagnosis. Involvement of the convex surface of the arch of the aorta may occlude the carotid artery and produce clinical symptoms resembling those of cerebrovascular accident—homolateral blindness with contralateral flaccid paralysis, hemianesthesia, and aphasia, called *carotid hemiplegia* by Weisman and Adams.[21]

Obstruction of the subclavian artery or arteries may result in ischemic necrosis of peripheral nerves, manifested by a pulseless, cold, weak extremity with anesthesia and loss of tendon reflexes. The changes often are temporary, lasting minutes to hours. Occlusion of an iliac artery may produce similar temporary symptoms in the lower extremity. Green and Saphir[22] reported a case in which the findings indicated an acute iliac occlusion, except for ecchymosis of the lower abdominal wall resulting from extension of the dissection into the deep inferior epigastric artery.

The intercostal, lumbar, and sacral arteries frequently are involved when the dissection spreads down the descending aorta. Occlusion of the ostia of the intercostal arteries in the lower thoracic and abdominal aorta may produce ischemic spinal necrosis or hemorrhagic infarction, resulting in flaccid paralysis and loss of sensation below the spinal level involved. Several factors determine whether the blood supply to the anterior spinal artery will be impaired. Anatomically the segmental arteries vary as to their site of origin from the posterior wall of the aorta. The dissection usually occurs on the anterior wall and extends in a spiral fashion. Weisman and Adams[21] noted that most of their cases with neurologic complications were in elderly patients in whom the circulation already had been impaired by atherosclerosis.

Tuohy, Boman, and Berdez[23] have stressed the diagnostic importance of wandering paralysis and vacillating sensory disturbances occurring below the level of the umbilicus.

Involvement of the mesenteric arteries may cause symptoms and signs that simulate an acute abdominal surgical emergency. There may be severe abdominal pain, hematemesis, melena, or signs of intestinal obstruction. Occlusion of the renal arteries usually results in infarction of one or of both kidneys. Symptoms of acute renal colic may be simulated and there may be albuminuria, hematuria, oliguria, or anuria, which result in a uremic death.

Laboratory findings. David and associates[13] stated that none of the electrocardiograms in their 17 cases of dissecting aneurysm of the aorta could be considered normal. Patterns of left ventricular strain and changes suggesting coronary disease predominated; the pattern of acute myocardial infarction was not found. The most common electrocardiographic pattern according to Levinson, Edmeades, and Griffith[24] is that of left ventricular hypertrophy. The pattern of myocardial infarction may occur when there is involvement of the dissecting process into the media of the coronary arteries, resulting in thrombosis and occlusion with myocardial infarction. The pattern of pericarditis may occur as the result of conditions previously described. Prolongation of Q-T intervals, sagging of RST segments, and T-wave flattening or inversion may occur as a result of electrolyte disturbances that are associated with uremia.

Leukocytosis usually is present. The white blood cell counts range from normal to 30,000 per cu. mm., with an increase in neutrophils. Anemia may result from bleeding into the aortic wall. Spinal fluid examination usually reveals clear fluid, but rarely it may be bloody from an associated spinal subarachnoid hemorrhage.

The most common finding on a thoracic roentgenogram is widening of the supracardiac shadow. This usually is progressive and involves the ascending or descending aorta. Occasionally a double-barreled shadow (especially on laminagrams) and areas indicating pleural effusion are seen. The angiocardiogram may be diagnostic, showing a dense concentration of the contrast medium in the main channel and a lateral or medial less dense concentration in the false channel.

Differential Diagnosis

Glendy, Castleman, and White[4] reported that myocardial infarction, embolism to some part of the body, and an acute abdominal surgical emergency are the conditions most likely to be confused with dissecting aneurysm of the aorta. Myocardial infarction is the most difficult to differentiate, but awareness of certain points may help to clarify the problem. In many cases of myocardial infarction, but rarely in cases of dissecting aneurysm, a history of angina is present. In myocardial infarction pain tends to develop gradually; in dissecting aneurysm pain is immediate and overwhelming. Radiation of pain down one or both arms occurs with myocardial infarction; whereas, with dissecting aneurysm radiation of pain is widespread to head, back, abdomen, and legs, but rarely to

arms. In addition, the persistence of hypertension, and obstruction of some part of the peripheral arterial circulation favors a diagnosis of dissecting aneurysm. Perhaps most important are the daily electrocardiograms that usually will confirm the diagnosis of myocardial infarction. Serum transaminase concentrations may provide further help in the differentiation.

Embolism to the aortic bifurcation, iliac, cerebral, or pulmonary arteries may be confused with dissecting aneurysm. The severe pain either in the chest or in the abdomen, with little if any dyspnea, the sudden onset of symptoms without evidence of previous heart disease or thrombophlebitis which might provide a source for embolism, and the usually uncontrollable course of the illness help to distinguish dissecting aneurysm in most instances.

An abdominal surgical emergency may be simulated by dissecting aneurysm when it involves the abdominal aorta. However, when dissecting aneurysm is present, the pain is likely to extend downward into both flanks. The presence of hypertension and the absence of previous symptoms related to the abdominal viscera or specifically to the kidneys indicate the possible presence of a dissecting aneurysm.

Levinson, Edmeades, and Griffith[24] state that the sudden appearance of an aortic diastolic murmur in a patient with hypertension who has severe chest pain is almost always pathognomonic of a dissecting aneurysm of the aorta. Various causes of the murmur have been cited. In Resnick and Keefer's[25] case, it appeared that the functional similarity of the false sac to an arteriovenous shunt and the regurgitation through the intimal tear above the aortic valve produced the murmur. At autopsy, an intimal opening was found just above the aortic valve. Hamman and Apperly[26] attribute the murmur to stretching or dilatation of the aortic ring. Levinson, Edmeades, and Griffith[24] believe that an intimal tear just at or above the aortic ring loosens the commissure and the corresponding cusp drops to a lower level within the ring. A hematoma at or above the aortic ring will distort it sufficiently to cause unequal closure of the valve leaflets. This murmur must be differentiated from that caused by a ruptured aortic valve cusp that is associated with tachycardia and previous cardiac damage from syphilis, rheumatic fever, or bacterial endocarditis. Thoracic pain and shock usually are absent in endocarditis.

Prognosis

Dissecting aneurysm of the aorta is associated with a high mortality. Approximately 25 per cent of the patients die within the first 48 hours after onset of symptoms. Half of the patients survive for 3 to 60 days; and the remaining 25 per cent of the patients may live, but usually sustain a second dissecting aneurysm that results in death. About 10 per cent of the latter group survive because the aneurysm ruptures farther down the aorta back into the lumen and forms a double-barreled aorta.

Treatment

Heretofore there has been no specific treatment for the disease. Treatment consisted mainly of supportive measures including bed rest, liberal use of morphine and oxygen, and blood transfusions in case of profound shock. Anticoagulant therapy is contraindicated.

Gurin, Bulmer, and Derby,[27] in 1935, surgically treated a dissecting aneurysm located in an iliac artery by excising the internal wall of the aneurysm at the distal point of advance. This procedure allowed the blood in the aneurysm to flow back into the true lumen, but the patient died from uremia. Other surgical attempts to repair dissections [28-31] have consisted of closure by suturing a ruptured dissecting abdominal aneurysm, and by fenestration of the internal wall of the aneurysm. Heretofore, all of the patients who were reported as surgically treated died postoperatively, either from uremia or from rupture of the aorta,[27,28,30] except those reported recently by DeBakey, Cooley, and Creech,[29] and Swann and Bradsher.[31] Four of DeBakey, Cooley, and Creech's six patients with dissecting aneurysm recovered completely. More recently they have reported[32] that 13 patients with dissecting aneurysm of the aorta have been treated surgically. Of the 13, 10 have survived operation and apparently have remained well up to one and one-half years postoperatively. Their technic included transection of the aorta as close to the origin of the dissection as possible, creating a re-entry of the false lumen into the true lumen proximally, and obliteration of the false lumen distally by suturing the inner and outer layers of the aorta around its full circumference. Occasionally in cases in which the dissection begins near the left subclavian artery, the proximal portion of the aneurysm, including the origin, is resected and replaced with a homograft. The distal portion is obliterated as described above. Swann and Bradsher[31] recently reported successful treatment of a patient with acute dissecting aneurysm of the thoracic aorta by fenestration of the internal wall of the aneurysm and external wrapping of the aorta with nylon cloth. The patient apparently was well six months after surgery.

Shaw[30] believes that the ideal mechanical solution of the problem of dissecting aneurysm involves repair of the primary tear in the proximal aorta as well as evacuation of blood and thrombus from the lumen of the aneurysm. To locate the tear and control the bleeding during repair are formidable problems, since the dissection usually starts in the region of or proximal to the great vessels of the aorta. Solution of the problems will be accomplished along with more accurate and early diagnosis and improved methods of controlling hypothermia and circulatory arrest.

Certainly, any patient who develops dissecting aneurysm of the aorta should be considered a vascular surgical emergency because of the serious mechanical problem it presents and the ultimately grave prognosis on conservative management.

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