Thrombotic Occlusion of Abdominal Aortic Aneurysm
Following Distal Embolization

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SUDDEN THROMBOSIS of an aneurysm of the abdominal aorta is an extremely rare complication. More commonly, abdominal aortic aneurysms rupture and bleed either retroperitoneally, into the free peritoneal cavity, or into adjacent bowel or mesentery. By contrast, the commonest complications of popliteal aneurysms are acute occlusion due to thrombosis of the aneurysm and peripheral embolic occlusion from mural thrombi within the aneurysm.1 Embolic phenomena and thrombosis of an abdominal aortic aneurysm are quite unusual. This report concerns a case demonstrating both of these occurrences.

Case Report

A 64-year-old right-handed insurance broker, was admitted to Montefiore Hospital on August 21, 1960, because of intermittent left hemiplegia and blurred vision. Four hours prior to admission the patient awoke and noticed inability to move the left upper and lower extremities. He had a left facial sag, dysarthria, and impairment of vision: numbers looked hazy on a nearby clock. He had experienced four such episodes each lasting 5 to 10 minutes before admission. In the past the patient enjoyed good health. He saw a physician about 1 year prior to admission because of labile hypertension. No medication was given. There was no history of angina, dyspnea on exertion, or intermittent claudication.

On admission the patient was in no distress. His blood pressure was 160/90 mm. Hg; temperature, pulse, and respiration were normal. There was no impairment of vision and no gross field defect. The carotid arteries were palpable bilaterally. There was no murmur in the carotid region. The femoral pulses and dorsalis pedis pulses were palpable bilaterally. Neurologic examination was within normal limits. Blood cell counts and urinalysis were normal, as were an electrocardiogram, electroencephalogram, and skull and chest roentgenograms.

Toward the end of the examination the patient experienced two transient episodes of left hemiplegia, left supranuclear facial paresis, and dysarthria. There was no sensory deficit on primary or simultaneous stimulation. The patient felt weak and was sweating. The blood pressure at the same time was 110 to 120 systolic and 70 to 80 diastolic. The episodes lasted 3 to 5 minutes. A diagnosis of partial or complete occlusion of the right carotid artery was made. A bilateral carotid angiogram showed partial obstruction of the right internal carotid artery at the bifurcation and patency on the left. When the blood pressure was elevated to 190/100 with Aramine he seemed better.

About 12 hours after admission the patient was taken to the operating room where a right internal carotid endarterectomy was performed under local anesthesia. An arteriosclerotic plaque was found at the bifurcation of the right common carotid artery, partially occluding the origin of the internal carotid artery.

Postoperatively, the blood pressure was at hypotensive levels (90/80) for several days, with transient episodes of right-sided hemiparesis. The blood pressure was controlled with Aramine. Heparin was administered for 9 days postoperatively. The patient was discharged on September 2, 1960, with a stable blood pressure of 160/90. There was a mild central facial paresis, but both carotid arteries were pulsating, and the patient was asymptomatic. Ophthalmodynamometry postoperatively was normal. Histologic report of the operative specimen was “atherosclerotic plaque.”

The patient was readmitted on September 4, 60 hours after discharge, because of severe pain and paresthesias in both lower extremities. Twenty-four hours after discharge he had noticed minor paresthesias in the right foot, and later bluish discoloration of the right toes. Paresthesias in the left foot and leg were followed by excruciating pain in both lower extremities and bluish discoloration in the toes. The pain was barely relieved by 100 mg. of Demerol intramuscularly. For 1 year prior to admission the patient had noted mild gluteal ache bilaterally on walking and relieved by further walking or massage. He had experienced no erection or ejaculation for the past 4 years, but he had libido. There had been no incontinence of urine or feces.

On admission the patient was flat in bed, tossing and turning from side to side. He complained of excruciating pain and paresthesias in both lower extremities. His blood pressure was 130 to 140/90.

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temporal arteries. Vision was normal. The chest was clear to percussion and auscultation. There were no abdominal masses or tenderness. The carotid and temporal arteries were palpable bilaterally. The femoral, popliteal, and dorsalis pedis pulses were not felt. The skin was cold below the umbilicus, the toes were cyanotic, and there was patchy cyanosis in both lower extremities. Motor and sensory modalities were intact. Neurologic examination was within normal limits. The white blood cell count was 15,000 with a shift to the left. Roentgenograms of the abdomen revealed calcification of the abdominal aorta.

A diagnosis of sudden occlusion of the bifurcation of the abdominal aorta was made, probably thrombotic in nature. The patient was taken to the operating room about 4 hours after admission. Under endotracheal general anesthesia, the abdomen was explored and an aneurysm was found in the abdominal aorta. The aneurysm was small, began one inch below the renal arteries, and extended to the aortic bifurcation. It was completely occluded by fresh clot, and no pulsations were palpable in the aneurysm or below. The aneurysm was mobilized and resected, including a 1.7-centimeter portion of both common iliac arteries. Both iliac arteries were occluded by fresh thrombus, and no back flow occurred after the clot was removed from the right side. The right iliac artery was explored more thoroughly and at its bifurcation an embolus was found. When this was extracted, vigorous back flow was present. The thrombus was then removed from the left common iliac artery and good back flow was obtained. A knitted Teflon bifurcation graft was inserted and circulation was re-established to both lower extremities. At the end of the procedure both femoral pulsations were strong, and several hours later both dorsalis pedis pulses were readily palpable.

The patient had an uneventful postoperative course without complications. The cyanosis of the tip of the toes gradually disappeared. No anti-coagulants were given. The pathologic report of the specimen removed was “arteriosclerotic aneurysm of aorta with thrombosis.” The patient was discharged on September 21, 1960, asymptomatic. He was readmitted for the third time on May 5, 1961, for postoperative carotid angiogram, which revealed patency of the right internal and external carotid arteries. The patient was discharged on May 6, 1961, asymptomatic.

Discussion

Jannetta and Roberts recently described the first well-documented instance of sudden, complete thrombosis of an abdominal aortic aneurysm with severe bilateral ischemia of the lower extremities. In their case, the episode occurred 48 hours after a cholecystectomy during which the aneurysm had been palpated by the surgeons, but was not traumatized or mobilized. The etiology of the thrombotic occlusion of the aneurysm could not be accounted for other than by the conjecture that it was probably related to an unexplained sudden shift in position of the mural thrombus, resulting in occlusion of the outlet of the aneurysm.

In the present case, the etiology of this rare complication may be readily explained. A fragment of the mural thrombus that almost always lines a fusiform aneurysm presumably broke off and embolized to the right common iliac artery, resulting in occlusion of the vessel with superimposed thrombosis. There was propagation of the thrombus proximally into the aneurysm, which clotted completely because of poor run-off. This explains the onset of ischemic symptoms in the right foot first, and then the development of the full-blown picture of terminal aortic occlusion the next day. The mural thrombus in fusiform aneurysms is not well organized or firmly adherent to the wall of the aneurysm, because of the dynamic nature of the lesion. Emboli and thrombosis occur commonly in aneurysms of the popliteal and femoral arteries because the loosely adherent mural thrombus is probably disrupted by motion of the overlying joint or muscles.

Summary

The second successfully treated case of sudden complete occlusion of an abdominal aortic aneurysm is presented. The thrombosis was caused by impaired run-off following an embolic occlusion of the right iliac artery. This complication is extremely rare in abdominal aortic aneurysms.

References


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