Case Reports and Technical Note

Coarctation of the Aorta and Cerebral Aneurysm
Report of Two Cases

RICHARD G. ROBINSON, Ch.M., F.R.C.S.
Neurosurgical Unit, Dunedin Hospital and Department of Surgery, University of Otago
Medical School, Dunedin, New Zealand

Eppinger in 1871 is credited with first describing the association of coarctation of the aorta and cerebral aneurysm. Later Hamby in 1952 said that the combination was common. Pool and Potts do not mention the matter in their book on cerebral aneurysms. We are reporting two cases.

Case Reports

Case 1. This 35-year-old man was transferred to the Neurosurgical Unit on February 25, 1966, from another hospital to which he had been admitted on February 22. While on the lavatory he had developed severe headache and collapsed to the floor without losing consciousness. A lumbar puncture had shown a blood-stained cerebrospinal fluid. There had been no previous episodes, but in the last 2 months he had had frequent headaches and had felt tired and generally irritable.

Originally he denied any previous illness, but his wife remembered that at the ages of 20 and 21 years he had been examined for military service and had been declined on account of high blood pressure. Two years later he had been examined for a life insurance policy and had been passed as fully fit.

Examination. He was a small wiry man who did not have any abnormal neurological signs. There was mild arteriovenous nipping of the retinal vessels. The blood pressure in the arms was 200/100 mm Hg, in the thigh, 150/100 mm Hg. The femoral pulses were feeble and delayed. Bounding pulsations could be felt from the periscapular arteries. A systolic bruit could be heard all over the precordium.

Radiographs of the chest showed rib notching (Fig. 1). The heart shadow was not enlarged. An aortogram done through the right femoral artery demonstrated a coarctation of the aorta 3 cm distal to the left subclavian artery. The catheter passed through the coarctation but it was impossible to get enough contrast medium into the cerebral arteries for diagnostic purposes. Tracings taken from the aortic catheter gave a pressure of 180/70 mm Hg above the lesion and 100/75 mm Hg below it.

Three days later bilateral carotid arteriograms were done. The right external carotid artery was absent, and its branches arose from the internal carotid artery. The right cerebral artery did not fill from the right internal carotid artery. The left carotid arteriogram demonstrated a lobulated aneurysm 1 cm in diameter arising from the

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posterior aspect of the anterior communicating artery (Fig. 2). Both anterior cerebral arteries filled from the left side.

The aneurysm was considered to be an unsatisfactory lesion to treat. It was uncertain whether the defect in the circle of Willis was due to spasm or to arterial anomaly. On April 4, the coarctation was resected. The immediate postoperative course was normal and he was discharged home on April 14.

Course. He was readmitted to Dunedin on May 17 for reassessment. Observations of the blood pressure in the arms gave readings between 170 to 140 mm Hg systolic and 100 to 90 mm Hg diastolic. The carotid arteriograms were repeated and showed no change from those done in March. It was concluded that the deficiency of the anterior cerebral circulation was developmental and not due to spasm. It was decided to continue the conservative management of the cerebral aneurysm.

He was seen on August 2, 1966. He had returned to work and was doing a full day's work without complaint. He was taking serpentine 5 mg twice a day because the blood pressure was still elevated. A casual blood pressure taken from the right arm was 170/90 mm Hg.

Comment. This patient had a common developmental anomaly of the circle of Willis and another known anomaly of the internal carotid artery in the neck. As might be expected in a patient his age, resection of the coarctation of the aorta did not completely control the hypertension.

Case 2. This 19-year-old bricklayer was admitted to the Neurosurgical Unit on June 15, 1966, from Grey Hospital, Greymouth, by air ambulance. He had been admitted to that hospital on June 10 after having developed a severe headache while sitting in a motor car. Until this illness he had done hard physical labor and had indulged in strenuous sports with ease. He was found to have an elevated blood pressure and signs of coarctation of the aorta. A lumbar puncture produced heavily blood-stained cerebrospinal fluid.

Examination. There were no abnormal neurological signs, and the retinal vessels were normal. The blood pressure in the right arm was 150/100 mm Hg and in the left thigh was 130/100 mm Hg. The right femoral pulse was absent, and the left was feeble and delayed. There was marked pulsation from the arteries around the scapula.

Radiographs of the chest showed notching of the ribs. The heart size was normal. Bilateral carotid arteriograms were normal on the right side, but on the left showed a large aneurysm at the bifurcation of the internal carotid artery. There was a second aneurysm of medium size arising from the junction of the left anterior cerebral artery and the anterior communicating artery (Fig. 3).

Course. It was considered that the cerebral aneurysms were not suitable for surgical treatment. Suddenly on the evening of June 22 he had another subarachnoid hemorrhage and died. Autopsy permission was refused.

Although coarctation of the aorta was not proved by surgery or autopsy, the physical signs were so characteristic as to make any other diagnosis unlikely.

Discussion

Coarctation of the aorta of the adult type is one of the less common congenital abnormalities of the heart and great vessels. The incidence in general autopsy studies has varied from 1 in 2500 to 1 in 1176. Maude Abbott in 1928 delineated the syndrome of coarctation of the aorta. There was a 4:1 ratio of male to female, 74% died before the