The Importance of Repeated Angiography in the Treatment of Mycotic-Emolic Intracranial Aneurysms

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An aneurysm caused by an invading organism, either from the adjacent tissues outside the vessel wall or (as far more commonly occurs) from within, has been known as "mycotic" since Osler first applied the term to a case in 1885 to focus attention on its inflammatory nature. Eppinger, in his classic monograph on this entity, discussed the pathogenesis of mycotic aneurysms of intravascular origin which he called "mycotic-embolic". This is perhaps a more descriptive term for the intravascular variety which we discuss in this paper. Mycotic aneurysms occur most commonly with vegetative bacterial endocarditis, either acute or subacute, and rarely with septicemias of other origins. Early accounts of their occurrences include the report of a case of "Rheumatism" in 1851, and in 1869 a case of subarachnoid hemorrhage secondary to a ruptured aneurysm in a patient with heart valve vegetations. Although the incidence of this lesion has been reduced since the introduction of antibiotics, it still accounts for 2.5-4.5% of all intracranial aneurysms.

The purpose of this paper is to discuss the significance of repeated angiography in the treatment of mycotic intracranial aneurysms as illustrated by an unusual case in which no aneurysm was demonstrable 1 day after a subarachnoid hemorrhage. An aneurysm, 1.5 cm. in diameter, was seen 2 months after the hemorrhage, but was not seen in an angiogram done 3 months later.

Case Report

A 41-year-old man was admitted to the Boston Veterans Administration Hospital for the 6th time on March 3, 1965, with complaints of daily fevers of 100-102°, night sweats, anorexia, weight loss of 20 pounds, and progressively increasing shortness of breath for the preceding 2 months. History. In 1955, the patient had been in an automobile accident and sustained severe burns involving both arms and legs and the body up to the waist. Since then, he had been hospitalized 3 times, in 1962 and 1963, and most recently in January, 1964, for skin ulcers which had involved both legs and were, on several occasions, septic, requiring I.V. and I.M. penicillin therapy for healing. There was no documented history of rheumatic fever or other cardiac abnormalities in his past.

Examination. Physical examination revealed an alert, cooperative, cachectic 41-year-old man with blood pressure of 100/40, pulse, 96, and temperature, 100.6° rectally. Extensive scar tissue and multiple skin graft sites were evident on the arms and legs. There were mild flexion contractures. A loud systolic murmur was heard over the aortic area and was transmitted into the neck. There was also a loud diastolic murmur heard best over the aortic area and left sternal border. The spleen was palpated 2 finger breadths below the left costal margin, but no hepatomegaly or evidence of peripheral embolic phenomena was noted.

Laboratory data included a white blood cell count of 7,600 with 59% polys and 41% lymphs; the urinalysis, blood urea nitrogen, and electrolytes were normal. The EKG showed first degree A-V block with pulmonale P waves. Seven blood cultures were taken before starting antibiotics and grew out gamma Streptococcus group D. The diagnosis of subacute bacterial endocarditis with secondary aortic insufficiency was made, and digitalization plus I.V. penicillin, 24 million units and I.M. streptomycin, 2 gm. per day, were started. Two days after admission, multiple cutaneous petechiae were noted but cleared in the next week.

Daily fever spikes of 101-103° persisted. On the morning of March 16, the patient suddenly complained of a severe headache and became unresponsive. On regaining consciousness a few moments later, he was aphasic; his right side including the face was flaccid. A lumbar puncture revealed grossly bloody cerebrospinal fluid with an opening pressure of 210 mm. of CSF; cultures were negative. A left carotid arteriogram (Fig. 1a and b) was done on March 17. Although the left Sylvian vessels were displaced slightly medially, there was no shift of the anterior cerebral vessels across the midline. There was an increased vascularity in the parietal area but no aneurysm was
Fig. 1. March 17, 1965. Left carotid arteriograms showing slight medial displacement of the insular vessels and increased vascularity in the posterior parietal area.

Fig. 2. May 13, 1965. Repeat left carotid arteriograms. A 15 mm. aneurysm arises from the middle cerebral artery near its bifurcation.