The Importance of Repeated Angiography in the Treatment of Myotic-Embolic 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 "myotic" 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 myotic aneurysms of intravascular origin which he called "myotic-embolic". This is perhaps a more descriptive term for the intravascular entity which we discuss in this paper. Myotic 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 1865 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 myotic 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 50% 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...
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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 13 mm. aneurysm arises from the middle cerebral artery near its bifurcation.
seen. A radiomerecury brain scan done 3 days after the subarachnoid hemorrhage was positive, and showed an irregular area of increased uptake in the left temporal area. An electroencephalogram showed slowing and depression of voltage in the left fronto-temporal region.

One week later, the patient was able to follow simple one step commands, but could utter no understandable words. Additional neurological deficits included a severe right hemiparesis and hemihypaesthesia involving face, arm, and leg. A low grade fever of 100–101° continued during 6 weeks of therapy. On April 17, all antibiotics were stopped and over the next few days the patient became and has remained afebrile.

On May 13, a second left carotid arteriogram was performed (Fig. 2a and b) and an aneurysm was seen at the bifurcation of the left middle cerebral artery. In view of the patient’s poor clinical condition, the site of this aneurysm, and the poor operative experience with mycotic aneurysms, we decided not to attack this lesion surgically.

Course. Since May, 1963, the patient has shown slow steady improvement in his speech, although he remains moderately dysphasic. He has experienced some return of strength in his right side, though he still has a moderately severe right hemiparesis and hemihypaesthesia, greatest in the arm. He is able to feed himself and travels in a wheel chair. His reflexes are increased in the right limbs, a right Hoffman is present, but both plantar reflexes are flexor. He has remained afebrile without antibiotics, continues digoxin, 0.25 mg. daily, and his cutaneous ulcers have healed.

On November 10, 1965, a third left carotid arteriogram was performed (Fig. 3). Four injections were made, one with cross compression of the right carotid. There was no evidence of vascular spasm and no aneurysm was demonstrated.

Discussion

In each of the preceding extensive reviews of mycotic aneurysms of intravascular origin, the intracranial vessels were the fourth most common location, following the aorta, abdominal and peripheral vessels in that order. Although the arteriographic demonstration of a small peripherally located intracranial aneurysm should suggest the possibility of mycotic origin, many mycotic aneurysms arise at the major branching sites of the middle cerebral artery and are radiographically indistinguishable from non-infected aneurysms.

The natural evolution of this entity is not as well known as that of other types of intracranial aneurysms. This is due to its lower incidence, plus the fact that the first sign of trouble is often a catastrophic fatal subarachnoid hemorrhage, as in 4 of the 5 cases seen at the Massachusetts General Hospital between 1953 and 1963 (Table 1). In only one instance (Case #936688) were there earlier neurological signs. The strikingly rapid development of the aneurysm, often less than a month from the time of presumed septic embolization to rupture, the tendency for free bleeding once ruptured, and the coincidence of acute or subacute bacterial endocarditis, usually with heart murmurs and evidence of other peripheral embolic phenomena, have been well documented.7,8,9 In the acute stages of septic necrosis, no actual aneurysm may be demonstrable. This phenomenon was true in our case and also in Case #1045855 in Table 1.

The fact that our case and all cases in Table 1 were receiving intensive antibiotic therapy at the time of rupture suggests that the incidence of this lesion may remain around 2–3% of patients with subacute bacterial endocarditis.
The importance of antibiotic coverage in patients suspected of having acute or subacute bacterial endocarditis cannot be too firmly stressed; yet, since there is rarely warning prior to rupture, and no completely satisfactory prophylaxis, perhaps the most important aspect of this entity for the clinician is the mode of treatment for those individuals who survive their initial rupture.

In a recent report of 5 cases with mycotic aneurysms, Roach and Drake advocated prompt surgical extirpation of the more peripheral aneurysms, but in the absence of a significant intracerebral hematoma, stated that "if the aneurysm arises from a major vessel, a delay of operation must be considered." With the first statement we are in complete agreement. If the artery lies peripherally enough so that its complete occlusion at the point of the aneurysm would not produce an appreciable neurological deficit, then its earliest possible removal seems advisable. However, when the aneurysm arises from a major vessel, and it not uncommonly does arise from the middle cerebral bifurcation of the dominant hemisphere, we advocate conservative initial management with appropriate massive antibiotics. Further therapy, however, should be based on subsequent angiographic findings rather than a predetermined course of later direct surgical attack. The presence of a significant intracerebral hematoma might, of course, necessitate early evacuation.

Early surgical attack when a major vessel is involved is felt to be inadvisable because in the acutely inflamed stage, the aneurysm characteristically has no wall other than cerebral tissue and the parent vessel almost always must be sacrificed. By waiting while massive antibiotic therapy is being carried out, even though the danger of rebleeding is a distinct possibility of really unknown proportion, it can be anticipated that some reparative fibrosis has occurred in both the wall of the aneurysm and feeding vessel, so that the chances of applying a clip or ligature to obliterate the aneurysm without sacrificing the parent vessel are more favorable. If, on repeated arteriograms 1 to 3 months later, the aneurysm is noted to be enlarging, as was the occasion in one case of Roach and Drake, then surgical removal seems to be indicated. However, in 2 situations surgery may not be the most prudent course. Mycotic aneurysms are frequently multiple and if, on repeated angiography, multiple lesions are disclosed, surgery might better be deferred. Also, hopefully, one might encounter the condition noted in our patient in which
the aneurysm had disappeared. Schnider and Cotsonas's comprehensive review of the reported cases also suggests this possibility; 4 of 17 cases with subarachnoid hemorrhage secondary to a ruptured mycotic aneurysm lived without definitive surgical treatment. Du Boulay recently has drawn attention to the dynamic state with reparative fibrosis of an intracranial aneurysm of noninflammatory etiology. Perhaps this process in an inflammatory aneurysm would be more likely to progress to complete obliteration of the lesion than it would in a non-inflammatory aneurysm. Thus, if on repeated angiography the aneurysm appeared to be diminishing in size, it would be wise to defer surgery, or even omit it entirely in those instances where the aneurysm disappeared.

Summary

We have discussed the problem of intracranial mycotic-embolic aneurysms and have summarized 5 fatal cases occurring at the Massachusetts General Hospital between 1953 and 1963. We have also presented an unusual case in which no aneurysm was angiographically demonstrable one day following subarachnoid hemorrhage; a 1.5 cm. aneurysm was seen on carotid arteriography two months later; five months after the hemorrhage there was no sign of the aneurysm. We have emphasized the importance of repeated angiography in determining the wisest course of therapy when the aneurysm is located on major intracranial vessels.

References