Multiple Cerebral Mycotic Aneurysms Complicating Posttraumatic Pseudomonas Meningitis

Case Report

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While intracranial mycotic aneurysms due to infected emboli are relatively common, we have been able to find only two reported cases1,5 in which this lesion appeared to be due, instead, to meningitis.

Because this complication is so rare we decided to report an additional instance of ruptured mycotic aneurysm secondary to bacterial meningitis.

Case Report

An 18-year-old man was admitted to his local hospital after a motorcycle accident. He was unconscious, bleeding from his right ear and both nostrils. X-rays of the skull showed an extensive fracture spreading horizontally, originating in the midparietal area and extending into the frontal sinus on the left. Ampicillin therapy was started.

The patient regained consciousness within a few hours and his vital signs remained stable with a temperature of less than 100°. During the remainder of this hospitalization he was sporadically drowsy and irritable, but never became disoriented. He complained of severe headaches over the left frontal area and vertex. Because of this and a continuing bloody drainage from his ear and nose, he was transferred to Mary Hitchcock Memorial Hospital 7 days after the accident.

Examination. On admission he was apathetic but irritable. His right ear canal contained dried blood. When he bent forward there was a profuse flow of clear fluid from the left nostril. Neurological examination was normal. In spite of bed rest, penicillin, streptomycin and daily lumbar punctures, the cerebrospinal fluid rhinorrhea continued for a week and it was decided to repair the defect. During the entire preoperative period the patient had been afebrile.

First Operation. A dural laceration was repaired and the fractured anterior frontal fossa was refloored with acrylic resin. The patient remained lethargic during the first postoperative day and by evening had developed ronchi and rales over the bases of both lungs, presumably due to aspiration of vomitus. At this time a tracheostomy was performed, pulmonary toilet was begun and the patient was given dexamethasone. Approximately 12 hours later he was awake and oriented, but during the next 2 hours he progressed into a deep coma with a temperature of 106°. Four blood cultures drawn over this period were sterile.

Second Operation. In the evening of the second postoperative day the bone flap was removed, along with a moderate amount of epidural blood clot. Hypothermia was started. The patient regained consciousness 6 hours later and was noted to have bilateral extensor plantar responses. At this time he was receiving chloramphenicol. Hypothermia was discontinued on the fourth postoperative day.

Before the first two operations the cerebrospinal fluid had shown an increase in protein and red cells, but the sugar concentration was normal and repeated cultures were negative. However, on the third day after the second operation the spinal fluid sugar had dropped to less than 10 mg% and there were 1500 leukocytes per cu mm of spinal fluid. The cells were almost all polymorphonuclear leukocytes. A species of pseudomonas was isolated from the spinal fluid culture on the fifth postoperative day. On the sixth postoperative day, chloram-
phenicol was discontinued and ampicillin was given for an additional 2 weeks. Treatment was stopped after the patient had been afebrile for 10 days. The rhinorrhea did not recur after the operation. When the patient was discharged on the 23rd postoperative day (3 days after therapy was discontinued) he was symptom-free, well oriented, and able to walk. The cerebrospinal fluid sample taken on the last day of therapy was normal except for a sugar concentration of 10 mg%.

Second Admission. The patient was admitted 3 days later with complaints of headache, backache, fever and delirium of several hours’ duration. Physical examination revealed nuchal rigidity with positive Kernig and Brudzinski signs. There was early choking of both optic discs and nystagmus. The cerebrospinal fluid contained 4700 polymorphonuclear leukocytes per cu mm, 275 mg% protein, and sugar of less than 10 mg%; a cerebrospinal fluid culture was sterile. The patient was given ampicillin, but remained febrile for several days with temperatures up to 103° and with variable levels of consciousness.

On the third hospital day a strain of pseudomonas resistant to ampicillin was isolated from the cerebrospinal fluid. Ampicillin was discontinued and penicillin, streptomycin, and sulfoxazole were substituted. The patient became afebrile but developed hallucinations and disorientation. There were bilateral extensor plantar responses and a left abducens paralysis, with continued nuchal rigidity. On the evening of the fourth hospital day, he developed generalized seizures with increased intracranial pressure and he had eye signs suggestive of a lesion in the posterior fossa. He was given urea and anti-convulsants. Later the same day he developed aphasia and a facial paralysis, hemiparesis, and hyper-reflexia, all on the right side.

Third Operation. For relief of increased intracranial pressure, external ventricular drainage was established through a right occipital burr hole. Postoperatively, the drainage fluid was clear at first, and the patient responded to verbal stimuli. Approximately 2 hours later, however, there was a sudden increase in the rate of flow of cerebrospinal fluid which became serosanguinous. At this time he suffered a respiratory arrest. He was resuscitated and was then maintained by a mechanical respirator. Several hours later he had a fatal cardiac arrest.

Autopsy Findings. The pertinent findings were confined to the skull and central nervous system.

A fracture line 2 cm long extended posteriorly from the cribiform plate toward the left clinoid process. This was covered by a patch of synthetic material. There was also a fracture line 4 cm long extending across the anterior pole of the left middle fossa.

There were approximately 100 ml of clotted blood beneath the posterior portion of the temporal lobe on the floor of the middle fossa. There were also approximately 100 ml of partially clotted blood in the subdural and subarachnoid spaces of both posterior fossae enclosing the midbrain and cerebellum.

There were four aneurysms of the arteries at the base of the brain (Fig. 1). The largest was fusiform, situated on the right antero-inferior cerebellar artery; this had ruptured. With its attached clot, it measured 2.5 by 1.5 cm. There were two intact fusiform aneurysms 1 cm long, one on the left poste-ro-inferior cerebellar artery and the other on a branch of the right antero-inferior cerebellar artery which had its origin proximal to the ruptured aneurysm. There was also an intact saccular aneurysm 0.5 cm in diameter at the origin of the right posterior cerebral artery.

Sections of the ruptured aneurysm showed clotted blood lying adjacent to the necrotic vessel wall, all layers of which were heavily infiltrated with polymorphonuclear leukocytes (Fig. 2). Sections of the other aneu-rysms showed similar changes but without rupture. Stains for elastica showed dissolution and disruption of elastic lamina. Sections stained for bacteria showed focal accumulations of rod shaped organisms in necrotic arterial walls.

Examination of the remainder of the brain showed that the subarachnoid hemorrhage had filled the third and fourth ventricles and had dissected into the midbrain, destroying most of the basis pontis. There was no gross or microscopic evidence of residual purulent meningitis (Fig. 3).

Examination of other organs revealed no evidence of thrombotic or embolic phenomena. There were no endocardial vegeta-
Mycotic Aneurysms with Meningitis

FIG. 1. Diagram of cerebral arteries showing multiple mycotic aneurysms. Arrow indicates ruptured aneurysm. Drawn from a photograph.

FIG. 2. Photomicrograph of a section of ruptured mycotic aneurysm. A thrombus in various stages of organization can be seen in its lumen. H. & E., X 11.

Discussion

We have been able to find reports of only two cases of mycotic aneurysms originating from an intracranial infection extrinsic to an artery. Mitchell and Angrist described a single case of mycotic aneurysm secondary to influenzal meningitis in their series of 36 intracranial aneurysms, 11 of which were mycotic. Their other cases of mycotic aneurysms were associated with bacterial endocarditis or septicemia. Barker reported a case of sphenoid sinusitis with meningitis and cavernous sinus thrombosis associated with bilateral internal carotid artery aneurysms, one of which had ruptured. It should be noted, however, that there are some unreported cases. For example, Dr. George Margolis of this laboratory has shown us sections of two cases of mycotic aneurysms secondary to a purulent meningeal infection.

Intracranial mycotic aneurysms due to a contiguous extrinsic infection are much less common than those due to an intrinsic infection. This is most likely due to differences in pathogenesis. In both cases an infectious angiitis is the initial factor in aneurysm formation. This angiitis probably extends relatively slowly through the thickness of the vessel wall. It is also probable that the integrity of

Cultures of blood and ventricular drainage fluid obtained at autopsy were sterile.
the internal elastic lamina must be compromised before aneurysm formation. In instances of an infectious angiitis originating from within an artery, the infectious process will affect the internal elastic lamina at a relatively early stage. Since the uninvolved peripheral portion of the wall of the artery is not permeable to bacteria, the elastica will be significantly damaged before the infection can spread to the meninges exterior to the vessel. In this way, an aneurysm can form prior to the occurrence of meningitis. Aneurysm formation is not an inevitable consequence of an infectious angiitis; the vessel may undergo fibrosis or thrombosis instead. In cases of aneurysms originating externally, the angiitis must, of necessity, be preceded by a meningitis. Because of the relative slowness of the destruction of the vessel wall, especially the internal elastic lamina, it is quite possible that the meningitis will have killed the patient or will have been cured before an aneurysm can form. Because of this, those instances of aneurysms arising from external infection would be more likely to occur in cases of meningitis caused by organisms of relatively low virulence, which would cause a chronic meningitis. Organisms whose virulence has been attenuated by therapy might also be expected to act in the same way.

It also appears that certain organisms have a predilection for attacking vascular walls, thus leading to aneurysm formation. Manion specifically mentions pseudomonas as such an organism. Although normally considered to be of low invasiveness and virulence, invasion of the walls of small arteries producing a necrotizing arteritis with thrombosis has been reported. The intracranial introduction of this organism has been attributed to contamination of the subarachnoid space by trauma or diagnostic or therapeutic procedures.

In our case, it appears that the antibiotic therapy was successful in the treatment of the meningitis, since no residual disease was found at autopsy and no organisms could be isolated at that time. Unfortunately, the cerebral vessels had already been damaged by the time the bacteria were eradicated from the cerebrospinal fluid and meninges. It seems likely that the signs and symptoms of the terminal episode were due to the rapid expansion and bleeding of an aneurysm rather than to meningitis.

**Summary**

We have reported a case of multiple intracranial mycotic aneurysms which occurred as a complication of post-traumatic meningitis due to a pseudomonas infection. The
trauma consisted of a compound skull fracture involving the frontal sinus. Death was caused by a subarachnoid hemorrhage resulting from the rupture of one aneurysm. This case is unusual because the mycotic aneurysms were caused by an extrinsic infectious process contiguous with the affected arteries rather than by septic emboli.

Acknowledgments

The authors wish to thank Dr. George Margolis for his help in the preparation of this paper, and Mary Layton for preparation of the drawing.

References

4. Margolis, G. Personal communication.