

Ruptured Intracranial Mycotic Aneurysm: A Rare Cause of Intracranial Hemorrhage

Keng-He Kong, MD, Kay-Fei Chan, MD

ABSTRACT. Kong K-H, Chan K-F. Ruptured intracranial mycotic aneurysm: a rare cause of intracranial hemorrhage. *Arch Phys Med Rehabil* 1995;76:287-9.

• Intracranial mycotic aneurysm is a rare complication in patients with infective endocarditis occurring in about 2% to 10% of cases. Although the risk of rupture is about 1.7%, it is usually a catastrophic event with a fatality rate of 80%. Neurological deficits secondary to cortical involvement are common, given the frequency of intralobar hemorrhage. We report two cases of intracerebral hemorrhage caused by ruptured intracranial mycotic aneurysms. Both had involvement of the right frontoparietal lobes with resultant left hemiparesis, left homonymous hemianopia, and impairments of cognition and perceptual function. Despite intensive rehabilitation, their functional outcomes were less than satisfactory as they needed assistance in self-care activities and mobility on discharge.

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The incidence of neurological involvement in infective endocarditis ranges from 30%¹ to 40%.² Despite the improvement in prognosis of patients with infective endocarditis with antibiotic treatment, this incidence has remained remarkably the same.³ Clinical manifestations include seizure, delirium, stroke, meningoencephalitis, and even peripheral neuropathy. Intracranial infective aneurysm is an uncommon complication occurring in about 2% to 10% of patients with infective endocarditis.⁴⁻⁶ The true incidence is probably much higher because many infective aneurysms remain asymptomatic and some resolve with antibiotic treatment. Most of them remain asymptomatic with only about 1.7% rupturing with subsequent intracranial hemorrhage.⁷ We report two cases of intracerebral hemorrhage caused by ruptured intracranial mycotic aneurysms as a result of bacterial endocarditis, their neurological deficits and functional outcomes after rehabilitation.

CASE 1

This 25-year-old female clerk with mitral valve prolapse complicated by mitral regurgitation was admitted to a neurosurgical facility for sudden onset of drowsiness and left hemiparesis. Clinical examination showed impaired conscious level, conjugate deviation of both eyes to the left and a left hemiparesis with associated hyperreflexia. A grade 3/6 pansystolic murmur of mitral regurgitation was heard over the cardiac apex and there were splinter hemorrhages over the left index and middle fingers. She was afebrile and no other stigmata of bacterial endocarditis was noted. There was a history of dental extraction 4 weeks before admission. A diagnosis of bacterial endocarditis with intracranial hemorrhage was made.

From the Department of Rehabilitation Medicine, Tan Tock Seng Hospital, Singapore.

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Reprint requests to Kay-Fei Chan, MD, Registrar, Department of Rehabilitation Medicine, Tan Tock Seng Hospital, C/O Ang Mo Kio Community Hospital, 17, Ang Mo Kio Avenue 9, Singapore 2056.

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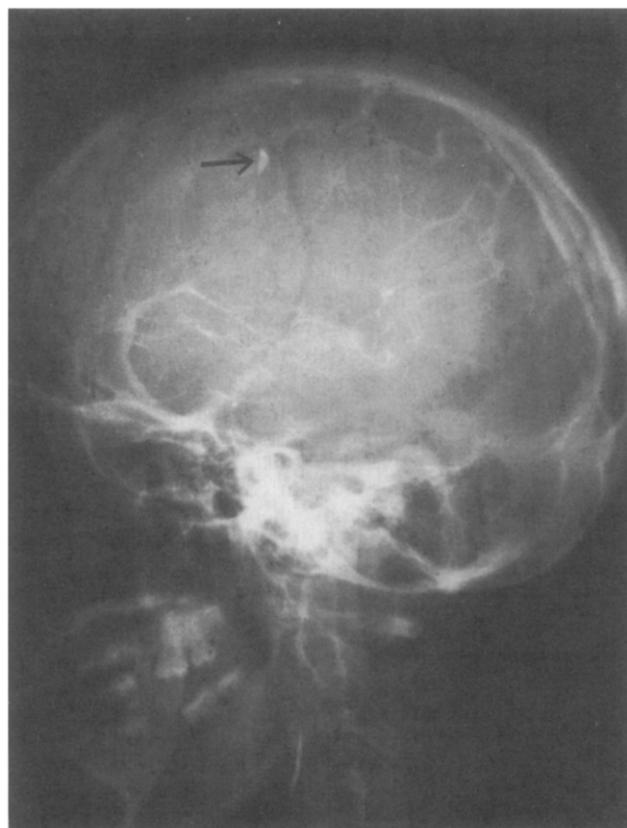


Fig 1—Four-vessel angiogram showing mycotic aneurysm (arrow) at the junction of the right middle cerebral and anterior cerebral arteries.

Computed tomography (CT) scan of the brain showed a large right frontoparietal hematoma with midline shift to the left. Four-vessel angiogram showed a mycotic aneurysm at the junction of the right middle cerebral and anterior cerebral arteries (fig 1). Evacuation of the intracranial clot with excision of the ruptured mycotic aneurysm was performed the following day. Laboratory studies showed mild anemia, leucocytosis, and microscopic hematuria. Histology of the aneurysm showed severe inflammation of the vessel wall with neutrophilic and lymphocytic infiltrates, features

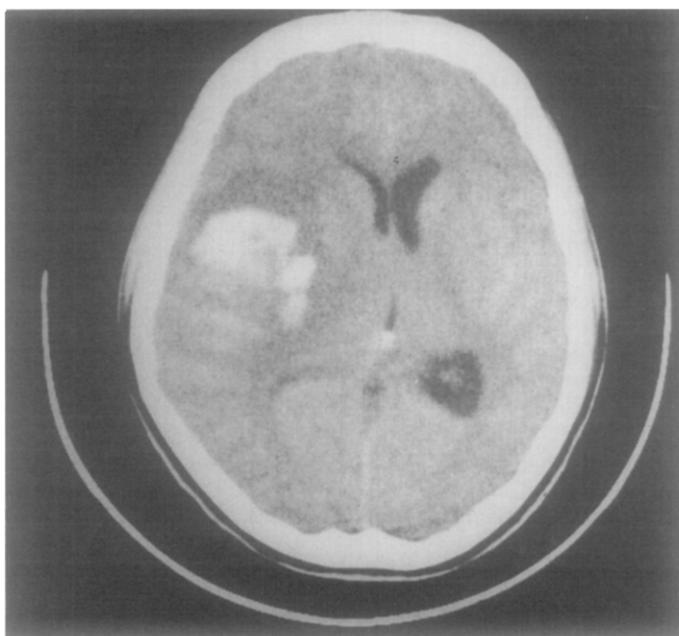


Fig 2—CT scan brain showing large right frontoparietal haematoma with midline shift.

consistent with a mycotic aneurysm. Even though the echocardiographic examination was negative for vegetations and six consecutive blood cultures were negative for bacterial growth, she was treated for bacterial endocarditis with antibiotics for 6 weeks, in view of the other clinical and laboratory findings. Six weeks after admission, she was transferred to rehabilitation for further management. Clinically, there was a left hemiparesis, left homonymous hemianopia, left neglect, and visuospatial deficits. Cognitive testing also showed difficulties in the planning, sequencing, and organization of tasks. Functionally, she needed assistance in all self-care activities and was only able to ambulate with moderate assistance. An intensive rehabilitation program consisting of physiotherapy, occupational therapy and cognitive therapy with emphasis on compensatory measures was started. Functional improvements were made and by the time of discharge 6 weeks after rehabilitation, she was independent in all her self-care activities except for bathing and toileting whereby minimal to moderate assistance was needed. She was able to ambulate under supervision with the assistance of an ankle-foot orthosis and a walking stick. On outpatient review 2 years after the intracranial bleed, she was functionally the same. No further intracranial events were noted.

CASE 2

This previously well 22-year-old female clerk with known mitral valve prolapse and mitral regurgitation was admitted for fever and lethargy. Echocardiographic examination of the heart showed vegetations and three consecutive blood cultures grew alpha-streptococci. The diagnosis of bacterial endocarditis was made and she was started on antibiotics. Twelve days after antibiotic therapy, she developed headache, vomiting, progressive drowsiness, and a left hemiparesis. CT brain scan showed a large right frontoparietal hemorrhage with midline shift (fig 2). As the patient's condition deteriorated, an emergency craniotomy was performed. Intraoperatively, two mycotic aneurysms were observed along the branches of the middle cerebral artery and one had ruptured giving rise to the frontoparietal hemorrhage. Evacuation of the clot and excision of both aneurysms were performed.

The patient was transferred to rehabilitation 6 weeks after admission. Clinically, she had signs of a major right hemispheric syndrome. There was a left hemiplegia, left homonymous hemianopia, and neglect. Perseveration and impairment of praxis and memory were also noted. Functionally, she was dependent in most self-care activities and mobility and was also incontinent of bowel and bladder. A graded rehabilitation program with emphasis on cueing and compensatory measures was started. Despite this, only modest functional gains were achieved. On discharge 8 weeks after rehabilitation, she needed supervision to minimal assistance in all self-care activities and was able to ambulate slowly with a quad-stick under close supervision. No significant improvement of her neurological status was observed. On review 1 year after the intracranial bleed, there were no further neurological events and her functional status was unchanged.

DISCUSSION

Many intracranial mycotic aneurysms are asymptomatic till rupture occurs. Rupture is usually a catastrophic event with a fatality rate of 80%.⁸ It can produce either subarachnoid hemorrhage, intraventricular hemorrhage, or more commonly, direct intracerebral destruction of the brain itself. Aneurysmal rupture may be the first presentation of infective endocarditis and this is more common in acute endocarditis where aneurysm formation occurs earlier, compared with subacute infective endocarditis where aneurysm formation occurs more frequently but at a later stage of the disease.^{9,10} Occasionally, there may be cranial nerve involvement secondary to compressive effects from an enlarging aneurysm or signs of meningeal irritation may develop from a slowly leaking aneurysm. Intracranial hemorrhage in infective endocarditis can also result from septic erosion of the arterial wall with rupture but without a well-delineated aneurysm¹⁰ or from hemorrhagic transformation of ischemic brain infarcts especially in those who are anticoagulated.^{11,12}

Embolisation of septic embolic material to cerebral vessels is necessary for the development of infective aneurysm. Molinari and coworkers, in animal studies of septic cerebral embolization noted the formation of infective aneurysms as early as 3 days when antibiotics were not given.¹³ With antibiotic treatment, aneurysm formation occurred at 7 to 10 days. It was also shown that the inflammatory response proceeded from the adventitial surface toward the media, with the internal elastic membrane and intima being affected last. The middle cerebral artery is involved most often, about 4 times more often than either the anterior or posterior cerebral artery. The aneurysms are typically sited at the bifurcation of small secondary branch peripheral arteries and they can be single or multiple.⁸

CT scan of the brain apart from showing intracerebral or subarachnoid hemorrhage does not usually show the diagnosis of infective aneurysm. Angiography is still the ultimate means for establishing diagnosis. Given the high mortality rate of aneurysmal rupture, it has been recommended that routine CT scan and 4-vessel angiogram be performed for any patient with known infective endocarditis who develops a neurological deficit suggestive of embolization.¹⁴

Management of the intracranial infective aneurysm is dependent on factors like the presence or absence of hemorrhage, anatomic location, and clinical course. Healing of

infective aneurysms with antibiotic treatment is well documented and the indications for surgical intervention must be evaluated on an individual basis.^{15,16} Serial follow-up of the aneurysm by angiography and more recently, magnetic resonance imaging (MRI), and sequential CT scan has been recommended while patients are still on antibiotic treatment.¹⁷

The functional outcome of patients surviving rupture of intracranial infective aneurysms is not well documented. Bohmfalk⁴ in his review of 19 survivors of ruptured infective aneurysms defined outcome as either partially recovered or completely recovered, whereas Frazee¹⁸ in his series of 7 survivors defined outcome as normal or disabled. The functional outcome of the 2 reported cases here was deemed to be less than satisfactory as on discharge, both still needed supervision and assistance in some basic self-care activities and mobility. Much of this is because of the presence of cognitive impairment and perceptual problems secondary to right hemispheric involvement. It is very likely that the functional outcome of patients with ruptured intracranial infective aneurysms is closely dependent on the degree of cerebral damage secondary to the hemorrhage. As most aneurysms are sited in the middle cerebral circulation, rupture inevitably results in intralobar hemorrhage and destruction. As a result, cortical neurological deficits like dysphasia, dyspraxia, hemianopia, perceptual, and cognitive deficits are not uncommon depending on the side and site of hemorrhage. The rehabilitation team should be cognizant of the frequency of these deficits because of their adverse effects on functional outcome.¹⁹

References

1. Jones HRJ, Siekert RG, Geraci JE. Neurologic manifestations of bacterial endocarditis. *Ann Intern Med* 1969;71:21-8.
2. Ziment I. Nervous system complications in bacterial endocarditis. *Am J Med* 1969;47:593-60.
3. Lerner PI. Neurologic complications of infective endocarditis. *Med Clin North Am* 1985;69:385-98.
4. Bohmfalk GL, Story JL, Wissinger JP, Brown WE Jr. Bacterial intracranial aneurysm. *J Neurosurg* 1978;48:369-82.
5. Cantu RC, LeMay M, Wilkinson HA. The importance of repeated angiography in the treatment of mycotic-embolic intracranial aneurysms. *J Neurosurg* 1961;25:189-93.
6. Moskowitz MA, Rosenbaum AE, Tyler HY. Angiographically monitored resolution of cerebral mycotic aneurysms. *Neurology* 1974;24:1103-8.
7. Hart RG, Kagen-Hallet K, Joerns SE. Mechanism of intracranial hemorrhage in infective endocarditis. *Stroke* 1987;18:1048-56.
8. Pruitt AA, Rubin RH, Karchmer AW, Duncan GW. Neurologic complications of bacterial endocarditis. *Medicine* 1978;57:329-43.
9. Weinstein L. Life-threatening complications of infective endocarditis and their management. *Arch Intern Med* 1986;146:953-7.
10. Siekert RG. Neurologic manifestations of infective endocarditis. In: Vinken PJ, Bruyn GW, editors. *Handbook of clinical neurology*. Amsterdam: Elsevier, 1980:469-77.
11. Cerebral Embolism Study Group: Immediate anticoagulation of embolic stroke. *Brain haemorrhage and management options*. *Stroke* 1984;15:779-89.
12. Lierberman A, Hans WK, Pinto R, Isom WO, Kupersmith M, Bear G, et al. Intracranial haemorrhage and infarction in anticoagulated patients with prosthetic heart valves. *Stroke* 1978;9:18-24.
13. Molinari GF, Smith L, Goldstein MN. Pathogenesis of cerebral mycotic aneurysms. *Neurology* 1973;23:325-32.
14. Barrow DL, Prats AR. Infectious intracranial aneurysms: comparison of groups with and without endocarditis. *Neurosurgery* 1990;27:562-73.
15. Morawetz RB, Karp RB. Evolution and resolution of intracranial bacterial (mycotic) aneurysms. *Neurosurgery* 1984;15:43-9.
16. Bingham WF. Treatment of mycotic intracranial aneurysms. *J Neurosurg* 1977;46:428-32.
17. Ahmadi J, Tung H, Gianotta SL, Destian S. Monitoring of infectious aneurysms by sequential computed tomographic/magnetic resonance imaging studies. *Neurosurgery* 1993;32:45-9.
18. Frazee JG, Cahan LD, Winter J. Bacterial intracranial aneurysms. *J Neurosurg* 1980;53:633-41.
19. Wade DT, Hewer RL, Skilbeck CE, David RM. Recovery and rehabilitation of physical loss. In: Wade DT, Hewer RL, Skilbeck CE, David RM, editors. *Stroke: a critical approach to diagnosis, treatment and management*. London: Chapman and Hall Medical, 1985;235-60.