

# Case Report

## Intracranial hemorrhage in infective endocarditis: A case report

Fahad Aziz<sup>1</sup>, Saira Perwaiz<sup>1</sup>, Sudheer Penupolu<sup>1</sup>, Sujatha Doddi<sup>1</sup>, Srinivas Gongireddy<sup>2</sup>

<sup>1</sup>Resident Internal Medicine, Jersey City Medical Center, NJ, USA; <sup>2</sup>Attending Physician Internal Medicine, Jersey City Medical Center, NJ, USA

**ABSTRACT** Cerebral hemorrhage occurs rarely in infective endocarditis. Here, we present an interesting case of infective endocarditis complicated by severe cerebral hemorrhage. Later, his blood culture grew *S. bovis*. To the best of our knowledge, this is the first ever reported case of *S. bovis* infective endocarditis complicated by extensive intracranial hemorrhage.

**KEY WORDS** Cerebral Hemorrhage; Infective endocarditis; *S. bovis*.

*J Thorac Dis 2011;3:134-137. DOI: 10.3978/j.issn.2072-1439.2010.11.13*

### Introduction

Intracranial hemorrhage (ICH) occurs in about 5% of patients with infective endocarditis (1-4). These hemorrhages are usually attributed to ruptured mycotic aneurysms, even when no aneurysm is demonstrable (1). As mycotic aneurysms are sometimes obliterated by the hemorrhages that they produce, their arteriographic and even pathologic demonstration is not always possible (2,5,6). However, ICH in infective endocarditis can also result from septic erosion of the arterial wall with rupture but without a well-delineated aneurysm (7). Surgical intervention in this situation is usually not feasible, as the necrotic fusiform segment cannot be repaired but requires resection with or without pedicle bypass grafting (8,9). Additionally, hemorrhagic transformation of ischemic brain infarcts can result in ICH, which is often massive in anticoagulated patients (10-12).

### Case reports

37-year old AAM with no significant PMH presented to the ER with the c/o fever since the last 4 days which was intermittent, high grade and associated with chills and rigors. It was periodic occurring usually at night and was relieved with Tylenol. He also had complaint of loss of appetite with weight loss of 5 pounds in the last 1 week. Recent medical history was significant

for cervical pain radiating to the arms, which was worked up recently at another hospital and was found to be secondary to degenerative cervical discs seen on the MRI. Other than the history of fever the review of systems was negative for any recent sore throat or other symptoms of URI. There was no urinary or bowel complaints or symptoms suggestive of CNS involvement. Also there was no history of recent travel, sick contacts or insect bites. There was no history of smoking or iv drug abuse or alcohol abuse. He was not sexually active for last 1-year and previously was in a monogamous relationship.

On Examination the patient was an average built man with fever of 102.6 F and tachycardic at 120. He was hemodynamically stable and did not appear to be in any apparent distress. Physical examination was normal other than hepatosplenomegaly. Liver was smooth, 6 inches below costal margin and nontender. Spleen was 2 inches below left costal margin and non-tender on palpation.

The initial workup revealed WBC of 10.4 with normal differential and Anemia with an Hb of 10 and a platelet count of 90,000. Basic metabolic profile was within normal limits other than hyponatremia with Na of 127 and mildly deranged LFT's with total bilirubin of 2.4 and Direct bilirubin 1 mg/dl. The total protein was also elevated with an elevated globulin component with Albumin being only 2.7 and total protein of 11.4. A urine analysis and CXR done were normal. A septic workup was ordered to rule out the source of fever along with a workup for anemia. The patient was admitted to medical floors and was started on Empiric Antibiotics.

Following day the patient remained febrile and physical examination revealed a diastolic murmur in aortic area which was new from the day of admission. The blood cultures grew Gram-positive cocci in clusters and the patient was started on vancomycin and Gentamycin for presumed Infective Endocarditis. A rapid test for HIV was negative. A 2 D Echo was performed the same day, which demonstrated a large mobile

No potential conflict of interest.

Corresponding to: Fahad Aziz, MD. Internal Medicine, Jersey City Medical Center, NJ 07302, USA. Tel: 347-461-6570; Fax: 347-461-6570. Email: Fahadaziz.md@gmail.com.

Submitted Nov 17, 2010. Accepted for publication Dec 16, 2010.

Available at [www.jthoracdis.com](http://www.jthoracdis.com)

ISSN: 2072-1439 © 2011 Journal of Thoracic Disease. All rights reserved.

vegetation on the non coronary cusp of the aortic valve.

Patient responded to the treatment and was afebrile on day 3 of admission. Blood cultures grew *Strep. Pasteurianus* a sub-species of *strep. Bovis* and the patient's antibiotic were changed to Rocephin and also a workup to rule out GI malignancy was ordered. The following cultures obtained remained negative and patient had no spikes of fever.

The patient was continued on current therapy when on day 6, he started complaining of a severe headache with an episode of vomiting following which he had an episode of seizure. The patient went in deep coma with GCS of 4 and a decerebrate posture.

A CT-Scan Head done was read as diffuse intraventricular hemorrhage, particularly in the right lateral ventricle, with early hydrocephalus of the lateral ventricles.

There was an intraparenchymal hematoma in the right occipital lobe.

There was also minimal scattered subarachnoid hemorrhage. Other findings were minimal right-to-left subfalcine herniation and compression of the brainstem, concerning for early transtentorial herniation.

The patient was intubated and transferred to the ICU for further management. Neurology and Neuro Surgery consults were called and as per recommendations patient was started on mannitol.

CT Angiography was also performed which showed no significant interval change in the large amount of hemorrhage involving the right occipital lobe or the intraventricular extension of the hemorrhage into the lateral ventricles, third ventricle and fourth ventricle. There was hydrocephalus with effacement of the basilar cisterns. After the administration of contrast there were no abnormal vessels in the region of the hemorrhage. The vessels of the circle of Willis, intracranial carotid arteries and basilar artery flow were within normal limits.

Patient's condition deteriorated in the ICU and on examination no brainstem reflexes could be elicited. Patient was declared dead the following day.

## Discussion

ICH complicating infective endocarditis is the result of a spectrum of arterial injury ranging from acute, pyogenic necrosis to large, aseptic aneurysms that may rupture weeks to months after bacteriologic cure. While the term "mycotic aneurysm" has usually been applied to both processes, the difference is not merely semantic. The extremes of this continuum appear to represent different clinical syndromes with distinct therapeutic implications.

Septic emboli appear to be a necessary substrate for ICH, although clinically recognized ipsilateral embolism precedes ICH in only 40% of cases (1,6,8,13-17). Sustained bacteremia in

tricuspid valve endocarditis, even with virulent organisms, does not result in ICH, supporting the necessity of embolic fragments. The offending infected emboli may escape clinical recognition by being small, by incompletely obstructing flow, or by preventing infarction by collateral circulation. Clearly, lack of antecedent clinical brain embolism does not eliminate the risk of ICH, as most hemorrhages occurred without recognized, antecedent embolism.

Symptomatic ICH associated with *S. aureus* occurred within 48 hours of admission. This propensity for early hemorrhages in *S.aureus* endocarditis has been noted by others (1,13). We observed ICH with *S. bovis* infective endocarditis which has never been reported before. The lobar location of ICH on CT is distinctive and should suggest infective endocarditis when occurring in relatively young patients with fever. ICH due to septic arteritis usually occurs during uncontrolled infection. Surgical treatment is difficult, requiring sacrifice of the involved artery, sometimes with microvascular pedicle/bypass surgery, as there is not a well-delineated aneurysmal neck that can be readily clipped (6,8,9).

Dilated mycotic aneurysms usually have no evidence of active infection and may be more amenable to surgical therapy. Many, possibly most, mycotic aneurysms heal spontaneously without rupture during anti- microbial treatment (8,14,18-20). Resolution may take many months (8). However, late rupture of mycotic aneurysm after otherwise successful treatment of infective endocarditis can occur.

While ICH occurs in about 5% of patients during the acute course of infective endocarditis, proven ruptured mycotic aneurysm is reported in only about 1.7% (range 0.8-2.8%) (21-25). Of patients with ICH and ruptured mycotic aneurysms, only about one third undergo surgical repair, due to fatal initial hemorrhage or to multiplicity of aneurysms. The fraction of our patients who could have potentially benefitted from surgical treatment of mycotic aneurysm under ideal circumstances of detection and prediction of rupture is quite small.

Enthusiasm for detection of unruptured mycotic aneurysms is tempered by the uncertain risk of rupture. Many case reports confirm that many unruptured mycotic aneurysms, including large and enlarging mycotic aneurysms, fully heal following antimicrobial therapy. The prevalence of unruptured intracranial mycotic aneurysms in patients with endocarditis is unknown.

Current recommendations concerning the indications for surgery for unruptured mycotic aneurysms are entirely arbitrary (26).

We believe that brain emboli complicating infective endocarditis result in ICH by at least 3 different mechanisms: 1) sterile emboli can cause infarcts that undergo secondary hemorrhagic transformation that is usually mild and asymptomatic in the absence of anti- coagulation therapy; 2) septic emboli during uncontrolled infection, particularly

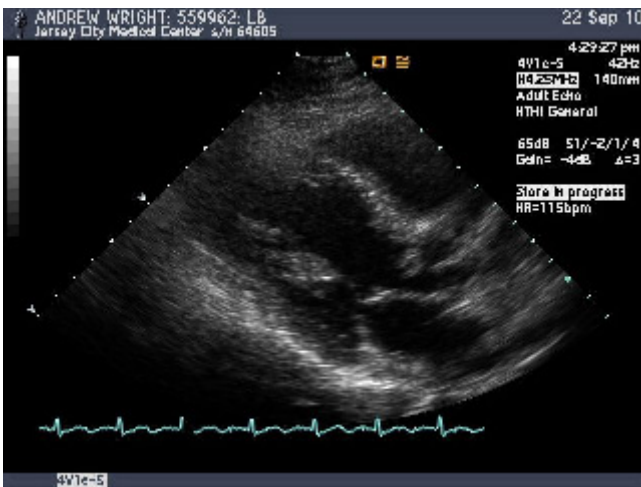


Fig 1. A large vegetation on the aortic valve

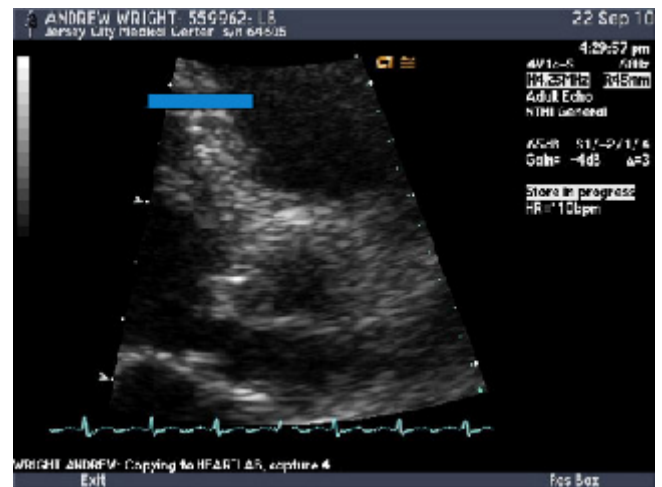


Fig 2. A large vegetation on the aortic valve



Fig 3. Intra-ventricular hemorrhage, particularly in the right Lateral ventricle, with early hydrocephalus of the lateral ventricles. There is minimal scattered subarachnoid hemorrhage.



Fig 4. Large right occipital hemorrhage with intraventricular extension. There is hydrocephalus and effacement of the basilar cisterns, which is unchanged. There is no visualized abnormal vasculature in= the right occipital region or elsewhere within the brain to suggest arterial venous malformation.

with virulent organisms, can cause acute, erosive arteritis with rupture; and 3) septic emboli during effective antimicrobial therapy and/or associated with nonvirulent organisms can injure the arterial wall, leading to subacute development of aneurysms that are often aseptic at the time of rupture. *S. aureus* is the most common organism underlying symptomatic ICH;

these hemorrhages usually occur early, during uncontrolled infection (1,2,13). To the best of our knowledge this is the first ever reported case of ICM secondary to *S. bovis* infective endocarditis. Mycotic aneurysms amenable to surgery appear to underlie only a fraction of brain hemorrhages in patients with infective endocarditis.

## References

1. Pniitt AA, Rubin RH, Karchmer AW, Duncan GW. Neurologic complications of bacterial endocarditis. *Medicine* 1978;57:329-43.
2. Royden-Jones H, Siekert RG, Geraci JE. Neurologic manifestations of bacterial endocarditis. *Ann Intern Med* 1969;71:21-8.
3. LeCam B, Guivarch G, Boles JM, Garre M, Cartier F. Neurologic complications in a group of 86 bacterial endocarditis. *Eur Heart J* 1984;5:s97-100.
4. Dreyer NP, Fields BN. Heroin-associated infective endocarditis. *Ann Intern Med* 1973;78:699-702.
5. Lerner PI. Neurologic complications of infective endocarditis. *Med Clin N Am* 1985;69:385-98.
6. Roach MR, Drake CG. Ruptured cerebral aneurysms caused by microorganisms. *N Engl J Med* 1965;273:240-4.
7. Siekert RG. Neurologic manifestation of infective endocarditis. In: Vinken PJ, Bruyn GW, editors. *Handbook of Clinical Neurology*. Amsterdam: Elsevier; 1980. p. 469-77.
8. Morawetz RB, Karp RB. Evolution and resolution of intracranial bacterial (mycotic) aneurysms. *Neurosurgery* 1984;15:43-9.
9. Day AL. Extracranial-intracranial bypass grafting in the surgical treatment of bacterial aneurysms: Report of two cases. *Neurosurgery* 1981;9:583-8.
10. Cerebral Embolism Study Group. Cardioembolic stroke, early anticoagulation, and brain hemorrhage. *Arch Intern Med* 1987;147:636-40.
11. Lieberman A, Hass WK, Pinto R, Isom WO, Kupersmith M, Bear G, et al. Intracranial hemorrhage and infarction in anticoagulated patients with prosthetic heart valves. *Stroke* 1978;9:18-24.
12. Cerebral Embolism Study Group. Immediate anticoagulation of embolic stroke. Brain hemorrhage and management options. *Stroke* 1984;15:779-89.
13. Weinstein L. Life-threatening complications of infective endocarditis and their management. *Arch Intern Med* 1986;146:953-7.
14. Moskowitz MA, Rosenbaum AE, Tyler HR. Angiographically monitored resolution of cerebral mycotic aneurysms. *Neurology* 1974;24:1103-8.
15. Katz RI, Goldberg HI, Selzer ME. Mycotic aneurysm. *Arch Intern Med* 1974;134:939-42.
16. Laguna J, Derby BM, Chase R. *Cardiobacterium hominis* endocarditis with cerebral mycotic aneurysm. *Arch Neurol* 1975;32:438-9.
17. Wilson WR, Geraci JE, Danielson GK, Thompson RL, Spittell JA Jr, Washington JA II, et al. Anticoagulant therapy and central nervous system complications in patients with prosthetic valve endocarditis. *Circulation* 1978;57:1004-7.
18. Leipzig TJ, Brown FD. Treatment of mycotic aneurysms. *Surg Neurol* 1985;23:403-7. Bingham WF. Treatment of mycotic intracranial aneurysms. *J Neurosurg* 1977;46:428-37.
19. Cantu RC, LeMay M, Wilkinson HA. The importance of repeated angiography in the treatment of mycotic-embolic intracranial aneurysms. *J Neurosurg* 1966;25:189-93.
20. Bamford J, Hodges J, Warlow C. Late rupture of a mycotic aneurysm after cure of bacterial endocarditis. *J Neurol* 1986;233:51-3.
21. Dean RH, Meacham PW, Weaver FA, Waterhouse G, O'Neil JA. Mycotic embolism and embolomycotic aneurysms. *Ann Surg* 1986;204:300-7.
22. Wilson LR, Lie JT, Houser OW, Piepgras DG, Geraci JE. The management of patients with mycotic aneurysms. *Curr Clin Top Infectious Dis* 1981;2:151-83.
23. Frazee JG, Cahan LD, Winter J. Bacterial intracranial aneurysms. *J Neurosurg* 1980;53:633-41.
24. Garvey GJ, Neu HC. Infective endocarditis - an evolving disease. *Medicine* 1978;57:105-27.
25. Bohmfalk GL, Story JL, Wissinger JP, Brown WE. Bacterial intracranial aneurysm. *J Neurosurg* 1978;48:369-82.
26. Hourihane JB. Ruptured mycotic intracranial aneurysm. *Vase Surg* 1970;4:21-9.