Cerebral Bacterial Aneurysm and Indications for Cerebral Angiography in Infective Endocarditis

Masaru YAMADA, Yoshio MIYASAKA, Hiroshi TAKAGI*, and Kenzoh YADA

Department of Neurosurgery, Kitasato University School of Medicine, Sagamihara, Kanagawa; *Department of Neurosurgery, Yamato City Hospital, Yamato, Kanagawa

Abstract

Six infective endocarditis patients who developed cerebral bacterial aneurysm were reviewed to clarify the indications and timing for cerebral angiography to achieve early detection of unruptured aneurysms. All cerebral bacterial aneurysms were confirmed either angiographically or at autopsy. All patients were treated conservatively. Four patients died due to ruptured aneurysm. Four of the six patients showed the signs and symptoms of cerebral and/or systemic embolism, followed by rupture or detection of cerebral bacterial aneurysm. Prodromal signs and symptoms of embolism in patients with infective endocarditis should be considered as indicators for cerebral angiography to detect cerebral bacterial aneurysms before rupture.

Key words: cerebral bacterial aneurysm, infective endocarditis, diagnosis, cerebral angiography, embolic symptom

Introduction

Intracranial hemorrhage caused by ruptured cerebral bacterial aneurysms in patients with infective endocarditis is frequently fatal, resulting in as much as 80% mortality. The early diagnosis of cerebral bacterial aneurysms and surgical treatment before rupture are extremely important. This retrospective study reviewed six conservatively treated patients with infective endocarditis who developed cerebral bacterial aneurysms to determine the indications and the best timing for angiographic procedures intended to detect the aneurysm before rupture.

Clinical Materials and Methods

Eighty-four patients with diagnosis of infective endocarditis based on fever, positive blood culture, a relevant cardiac murmur, echocardiographic findings, and predisposing heart disease between 1979 and 1990 were reviewed. Four patients with ruptured and two with unruptured cerebral bacterial aneurysm were included in this study. Table 1 summarizes the clinical details of the patients, three males and three females, aged 16 to 51 years (mean 32 yrs). The initial examination found intracranial episodes of subarachnoid hemorrhage, subcortical hematoma, and cerebral embolism. The diagnosis of cerebral bacterial aneurysm was confirmed either by angiography (Cases 1, 2, 5, and 6) or by autopsy (Cases 3 and 4).

Results

All patients were treated conservatively with appropriate antibiotics selected according to the results of blood cultures. The organisms responsible for the infective endocarditis were Klebsiella pneumoniae, Staphylococcus aureus, α-streptococci, and Streptococcus viridans in Cases 1, 3, 4, and 5, respectively, but were undetermined in Cases 2 and 6. In Case 2, the antibiotics administered were ineffective against the systemic infection, and the aneurysm ruptured (Table 1). In Cases 1, 3, and 4, the antibiotics administered were effective against the systemic infection, but the aneurysms ruptured. In Cases 5 and 6, the antibiotics inhibited the systemic infection, and the aneurysms remained unruptured. Only in these two patients did the aneurysms become smaller or disappear, and both survived. All four patients with ruptured aneurysms died, two of the initial episode of hemorrhage, and two of the second.
Table 1 shows the signs and symptoms of cerebral or systemic embolism before aneurysm rupture or before detection of unruptured cerebral bacterial aneurysms (Cases 1-4) or before detection of unruptured cerebral bacterial aneurysms (Cases 5 and 6). **Neurological symptoms after cerebral embolism episodes. ACA: anterior cerebral artery, IVH: intraventricular hematoma, MCA: middle cerebral artery, PCA: posterior cerebral artery, SAH: subarachnoid hemorrhage, SCH: subcortical hematoma, SDH: subdural hematoma, TIA: transient ischemic attack.

**Table 1 Clinical summary of six patients with cerebral bacterial aneurysms**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age/Sex</th>
<th>Clinical presentation</th>
<th>Site of aneurysm</th>
<th>Symptoms of embolism*</th>
<th>Consciousness level after initial hemorrhage</th>
<th>Survival time</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>29/M</td>
<td>SAH</td>
<td>ACA</td>
<td>TIA, cerebral infarct, petechiae</td>
<td>alert</td>
<td>7 wks</td>
<td>death 1 day after rebleeding</td>
</tr>
<tr>
<td>2</td>
<td>24/M</td>
<td>SAH</td>
<td>MCA</td>
<td>—</td>
<td>somnolent</td>
<td>4 wks</td>
<td>death 12 days after rebleeding</td>
</tr>
<tr>
<td>3</td>
<td>35/F</td>
<td>SCH (temporal)</td>
<td>MCA</td>
<td>Osler's node, petechiae</td>
<td>coma</td>
<td>3 days</td>
<td>death</td>
</tr>
<tr>
<td>4</td>
<td>37/M</td>
<td>SCH (frontal), SDH, IVH</td>
<td>ACA</td>
<td>—</td>
<td>coma</td>
<td>1 day</td>
<td>death</td>
</tr>
<tr>
<td>5</td>
<td>16/F</td>
<td>cerebral embolism</td>
<td>MCA, PCA</td>
<td>cerebral infarct, splinter hemorrhage</td>
<td>hemiparesis**</td>
<td></td>
<td>good recovery</td>
</tr>
<tr>
<td>6</td>
<td>51/F</td>
<td>cerebral embolism</td>
<td>MCA</td>
<td>cerebral infarct</td>
<td>hemiparesis**</td>
<td></td>
<td>moderate disability</td>
</tr>
</tbody>
</table>


Table 1 shows the signs and symptoms of cerebral or systemic embolism before aneurysm rupture or before detection of an unruptured aneurysm. Cerebral embolism manifested as cerebral infarct (hemiparesis) in three cases, including one transient ischemic attack. Systemic embolism manifested as petechiae (2 cases), Osler’s node (1), and splinter hemorrhage (1). Four (67%) of the six patients showed prodromal signs of cerebral or systemic embolism that are considered important indications for cerebral angiography.

**Illustrative Case**

**Case 1:** Eight days before admission, a 29-year-old right-handed male noticed a transient episode of motor weakness in the right upper extremity and dysarthria followed by high fever and transient hematuria. He developed severe headache and vomiting 3 days later. On the day before admission, he had noticed motor weakness of the left upper extremity.

Physical examination on admission revealed petechial hemorrhages in the nail beds of the fingers and a diastolic cardiac murmur suggesting mitral stenosis. Neurological examination revealed monoparesis of the left upper extremity. Echocardiography showed an abnormality in the left atrium, but cardiology with catheterization disclosed no abnormal mass. The next day, nuchal rigidity developed, and lumbar puncture revealed bloody cerebrospinal fluid. Cerebral angiography demonstrated a small aneurysm in the distal portion of the left anterior cerebral artery (Fig. 1). A blood culture was negative.

He was treated conservatively by administration of several antibiotics (penicillin G, cephalosporins, and aminoglycosides), which reduced the fever. Seven weeks after admission, he suddenly became comatose with pupils dilated, and died on the following day. Autopsy revealed infective endocarditis of the mitral valve, multiple infarction of the spleen, and a large subcortical hematoma in the left parieto-occipital area caused by rupture of the cerebral bacterial aneurysm in the left anterior cerebral artery.

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Discussion

Whether conservative treatment of cerebral bacterial aneurysms is preferable to a more aggressive surgical approach is still controversial. The conservative alternative has been used in our institute, since cerebral bacterial aneurysm was regarded as part of the systemic infection, so treatment of the infection was treatment of the aneurysm. However, retrospective studies of conservatively treated cases have shown that, once the aneurysm had ruptured, the outcome was highly unfavorable. Frazee et al.\(^1\) reported that none of five surgically treated patients died, but six of eight treated nonsurgically did die. Brust et al.\(^3\) reported good outcomes after surgery in 17 patients with 28 cerebral bacterial aneurysms. Bohmfalk et al.\(^2\) and Ojemann et al.\(^9\) reported lower mortality after elective surgery for cerebral bacterial aneurysms than after conservative treatment. These reports suggest that the outcome after surgical treatment is considerably better. However, selection of the appropriate surgical treatment is another outstanding issue, and the resolution of cerebral bacterial aneurysms by medical methods also demands consideration.\(^8,9\) Cerebral angiographic studies to clarify the natural course of cerebral bacterial aneurysms are necessary.

The indications and timing for cerebral angiography to identify a cerebral bacterial aneurysm before rupture during the treatment of infective endocarditis is a fundamental problem.\(^1,3-5,8,10,12,13\) Some authors suggest any patient with symptoms of cerebral embolism should undergo angiography,\(^2,7\) while others consider that only patients with significant signs and symptoms not responding to antibiotics,\(^4\) abnormal findings on neurological examination, computed tomography, and cerebrospinal fluid examination,\(^3,10\) or all asymptomatic infective endocarditis patients when circumstances permit\(^2\) should be examined. In our series, there were four patients (67%) with symptoms of systemic embolism (Osler's node, petechiae, and splinter hemorrhage) or cerebral embolism (transient ischemic attacks or cerebral infarct) before the cerebral bacterial aneurysm was detected. Two of the four patients with aneurysm that later ruptured showed these signs and symptoms. Therefore, symptoms of systemic embolism as well as abnormal neurological events should be considered as important early diagnostic factors for cerebral bacterial aneurysm.

Improvement of the mortality and morbidity in infective endocarditis patients requires early reaction to prodromal signs and symptoms, including cerebral angiography to detect aneurysms before rupture, and a more aggressive treatment approach incorporating aneurysm surgery. However, cerebral bacterial aneurysms which grow rapidly after negative results from angiography or nonaneurysmal vasculitis may still cause problems.\(^3\) The outcome following surgery of unruptured cerebral bacterial aneurysms should also be examined.

References


Address reprint requests to: M. Yamada, M.D., Department of Neurosurgery, Kitasato University School of Medicine, 1-15-1 Kitasato, Sagamihara, Kanagawa 228, Japan.