Endogenous Endophthalmitis Following
Streptococcus pneumoniae Meningitis

Teruhiko Sekiguchi¹² and Akira Inaba²

Abstract

A 67-year-old man was transported to our hospital and diagnosed with pneumococcal meningitis. We immediately administered ceftriaxone and vancomycin according to the guidelines, but did not administer dexamethasone to him because he had been previously administered antibiotics. His left eye became complicated by endogenous endophthalmitis on the next day, which resulted in blindness, although his meningitis rapidly ameliorated. In comparison to other patients who have been reported to recover from complications with endophthalmitis after the combination therapy of antibiotics, corticosteroids and vitreous surgery, we consider that this patient’s poor visual outcome may have been caused by severe inflammation or the breakdown of the blood ocular barrier due to the action of S. pneumoniae. Corticosteroids may be able to successfully treat such inflammation or disruption of the blood ocular barrier.

Key words: bacterial eye infections, bacterial meningitis, pneumococcal meningitis, steroids, corticosteroids, antibiotics

(DOI: 10.2169/internalmedicine.54.4705)

Introduction

Endogenous bacterial endophthalmitis is an infectious disease within the eyeball, caused by bacteremia (1). Streptococcus pneumoniae is one of the most common organisms, and it is associated with a poor visual outcome (2). We herein report a patient presenting with endogenous endophthalmitis caused by pneumococcal meningitis. We also review the pertinent literature and discuss the optimal treatment for pneumococcal endophthalmitis.

Case Report

A 69-year-old man developed a 40°C fever. He had a past history of hypertension and dyslipidemia. He also regularly drank a cup of Japanese sake (equal to 20 g of ethanol) every day, but he had never undergone an operation or had recently experienced any trauma. A few days later, he became drowsy and consulted his home doctor. The doctor brought him to our hospital, after administering 2 g of ceftriaxone. The physical examination indicated marked stiffness in his neck. A blood examination showed an elevation of white blood cells (17,000/μL) and C-reactive protein (22.4 mg/dL). The hepatic enzymes were also high [glutamic oxaloacetic acid transaminase (GOT): 99 U/L, glutamic pyruvate transaminase (GPT): 61 U/L, γ-glutamyl transpeptidase (GTP): 98 U/L], thus suggesting alcoholic hepatitis. HIV-antibody was negative. The cells (polynuclear: 438/μL; mononuclear: 13/μL) and protein (454 mg/dL) were elevated and sugar was not detected in the cerebrospinal fluid (CSF). Streptococcus pneumoniae, sensitive to major beta-lactam antibiotics, was cultured from both his blood and CSF. A diagnosis of bacterial meningitis was thus made. He was immediately treated with 4 g of ceftriaxone and 2 g of vancomycin per day, but according to the guidelines (3, 4), dexamethasone was not administered because he had been treated with antibiotics just prior to this presentation. The brain MRI findings at admission were normal including the orbits.

On the next day, his left eye became rapidly swollen (Fig. 1A). An ophthalmologic examination revealed the an-
duced by corticosteroids. In fact, adjunctive dexamethasone poor prognosis. These inflammatory responses can be responses paradoxically cause severe cell death and result in a A2 independent of TLR2. These host inflammatory responses resulted in no light perception (Fig. 1C, 2). Although his consciousness, temperature and CSF findings almost normalized within a week, his left visual acuity did not show any improvement and finally repeatedly administered. Although his consciousness, temperature and CSF findings almost normalized within a week, his left visual acuity did not show any improvement and finally resulted in no light perception (Fig. 1C, 2).

Discussion

Our patient presented with endogenous ophthalmitis following the onset of pneumococcal meningitis accompanied with bacteremia. The systemic administration of antibiotics was effective against meningitis and he had no risk factors for endogenous endophthalmitis, such as diabetes or cirrhosis (5-7). However, the intensive injection of appropriate antibiotics into eyeball did not improve the endophthalmitis in this case.

Streptococcus pneumoniae have several inducers to activate the host inflammatory response (8, 9). Among them, the cell wall and pneumolysin are regarded as being the main toxic factors. The former induces the production of cytokines, the influx of neutrophils and cell apoptosis through CD14 and Toll-like receptor 2 (TLR2) pathways. The latter activates inflammatory mediators, such as tumor necrosis factor alpha (TNF-α) and enzymes including phospholipase A2 independent of TLR2. These host inflammatory responses paradoxically cause severe cell death and result in a poor prognosis. These inflammatory responses can be reduced by corticosteroids. In fact, adjunctive dexamethasone has been reported to show a significantly better outcome only in pneumococcal meningitis among various types of bacterial meningitis (10).

Alternatively, corticosteroids may repair any rupture in the blood ocular barrier. Endogenous bacterial endophthalmitis occurs when bacteremia exists and the blood ocular barrier is crossed by bacteria from the bloodstream (5). As mentioned above, Streptococcus pneumoniae meningitis causes the release of proinflammatory mediators, such as TNF-α, interleukin (IL)-1β, IL-6 (11). TNF-α disrupts the blood ocular barrier at the level of the pigment epithelium (12). Corticosteroids are reported to successfully repair the breakdown of the blood ocular barrier in an animal model (13). The discrepancy observed between the outcome of meningitis and that of endophthalmitis in our patient may therefore have been due to a disruption of the blood ocular barrier.

Even after antibiotic clear therapeutic guidelines for bacterial meningitis with blood-brain-barrier penetrating third-generation cephems were established, some patients still suffered from complicating endogenous endophthalmitis. Since the Endophthalmitis Vitrectomy Study suggested that the management of postoperative bacterial endophthalmitis after cataract surgery should be the combined therapy of immediate vitrectomy, intravitreal antibiotics and (systemic and focal) corticosteroids in 1995 (14), the outcome of endophthalmitis has improved (15). However, no clear treatment protocol for endogenous endophthalmitis has yet been established. For example, the efficacy of both intravitreal antibiotics and corticosteroids still remains controversial (5). We could find three case reports about adult endogenous pneumococcal endophthalmitis complicated by meningitis since 1999 (16-18) (Table). Combined therapy of intravenous and intravitreal injection of antibiotics, intravenous or focal injection of corticosteroids and vitreous surgery was able to slightly improve the visual acuities of the patients.

Figure 1. (A) The left eye became opaque and swollen on day 4. (B) The anterior part of left vitreous body is markedly clouded and high-echoic. Retinal detachment (black arrow) is also suggested by ultrasonography (B-mode). (C) The inflammation finally spread to all of the intraorbital tissues.
Figure 2. The clinical courses of both meningitis and endophthalmitis. Note that the endophthalmitis progressed rapidly even though the abnormalities of the cerebrospinal fluid ameliorated within a week.

Table. Clinical Features of Previously Reported Endogenous Endophthalmitis Cases Following Pneumococcal Meningitis since 1999.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Case</th>
<th>Background</th>
<th>Side</th>
<th>Systemic ABX</th>
<th>Focal ABX</th>
<th>Corticosteroids</th>
<th>Outcome of visual acuity</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>56/M</td>
<td>Asplenic</td>
<td>Bil</td>
<td>CTRX VCM</td>
<td>None</td>
<td>topical injection</td>
<td>R: LP, L: 20/200</td>
</tr>
<tr>
<td>17</td>
<td>40/F</td>
<td>Travel</td>
<td>Bil</td>
<td>CTRX VCM</td>
<td>AMK VCM</td>
<td>Beta-parabulbar injection</td>
<td>R: &lt;1/10, L: &lt;1/10</td>
</tr>
<tr>
<td>18</td>
<td>44/M</td>
<td>None</td>
<td>Bil</td>
<td>ABPC CTX VCM</td>
<td>CAZ VCM</td>
<td>DEXA-IV</td>
<td>R: NLP, L: 20/80</td>
</tr>
<tr>
<td>Our case</td>
<td>69/M</td>
<td>Alcoholic</td>
<td>L</td>
<td>CTRX VCM</td>
<td>CAZ VCM</td>
<td>None</td>
<td>R: Normal, L: NLP</td>
</tr>
</tbody>
</table>


described in these reports. Although the patient reported by Torii et al. received dexamethasone in order to avoid brain edema (18), this treatment may unexpectedly reduce the inflammatory responses, simultaneously repairing the breakdown of the blood ocular barrier, or both.

We herein presented a patient presenting with endogenous endophthalmitis following the onset of pneumococcal meningitis who unfortunately resulted in no light perception in the affected eye. We did not add the corticosteroids to our patient even after the onset of endophthalmitis, because the retrospective case series of pneumococcal endophthalmitis by Miller et al. did not find any significantly better visual
outcome in the eyes injected with dexamethasone (2). However, most of their cases had an exogenous origin and the number of patients was also relatively small. *Streptococcus pneumoniae* are known to cause especially severe inflammations. We suggest that corticosteroids should be used to effectively treat any complicating intraocular inflammation or to repair a breakdown of the blood ocular barrier caused by pneumococcal meningitis.

The authors state that they have no Conflict of Interest (COI).

References