Endocarditis Due to Group A β-Hemolytic Streptococcus in Children with Potentially Lethal Sequelae: 2 Cases and Review

Bacterial endocarditis affecting the normal heart is rare in childhood. Here we describe 2 children who developed endocarditis due to group A β-hemolytic Streptococcus (GABHS) that required emergency cardiac surgery. These cases emphasize the importance of considering this diagnosis in children presenting with signs of embolism, for whom urgent intervention may avert catastrophe.

Case 1. A 4-year-old boy presented with a 10-day history of fever and pain in his left foot. On examination he was febrile with purpuric lesions on his left thigh and dusky discoloration of his toes. The cardiovascular system was normal on the basis of clinical examination, but echocardiography revealed a large flail valve attached to a mitral valve leaflet with severe associated regurgitation. He underwent urgent cardiac surgery to excise the vegetation and thrombus and repair the mitral valve. Two cultures of blood and a culture of a scraping from the left foot taken on admission yielded (untyped) GABHS. Culture of a throat swab taken earlier yielded no growth. Ischemia of the foot progressed to necrosis and amputation of 2 toes, leaving a residual deformity that required plastic and orthopedic surgery. He completed a 6-week course of antibiotics and has subsequently remained well.

Case 2. A 33-month-old boy presented with hot, tender, painful swelling in his ankles and right knee 4 weeks after varicella. On examination he was pyrexial, tachycardic, and had splenomegaly. The remainder of the examination, including auscultation of the heart, was normal. He was treated with ibuprofen. Two days later his left leg suddenly became cold with impalpable pulses. A gallop rhythm and an ejection systolic murmur were heard over the precordium. Echocardiography showed perforation of the aortic valve with vegetation on the aortic root and severe aortic regurgitation. Within hours he had 2 short focal seizures involving the left arm and face. CT of the brain revealed a large right-side infarct in the middle cerebral artery territory. Repeated echocardiography showed an abscess at the aortic root. Excision of the aortic valve and homograft replacement of the aortic root was carried out immediately. Blood culture yielded GABHS T12, M-nontypeable. Culture of a throat swab taken earlier yielded no growth. He completed a 6-week course of antibiotics. Cardiac function returned to normal, but he has a residual left hemiplegia.

Our 2 cases emphasize the importance of considering a diagnosis of infective endocarditis in children, even with no prior history of heart disease, who present with signs of embolism, and reemphasize the virulence of GABHS. The organisms most commonly implicated in endocarditis are viridans streptococci and reemphasize the virulence of GABHS. The organisms most commonly implicated in endocarditis are viridans streptococci and Staphylococcus aureus [1]. GABHS endocarditis is unusual in children, with a reported incidence of <5% [2]. The most common sources of GABHS bacteremia are the upper respiratory tract and the skin, especially where damaged (e.g., superinfected varicella lesions [3, 4]).

To our knowledge, the development of fulminant GABHS endocarditis after varicella, as seen in our second patient, has not been described previously. Table 1 summarizes the 6 cases of GABHS endocarditis that we could find in the pediatric literature. Five of the 6 occurred on the left side of previously normal hearts, with predominantly embolic features, as in our cases.

A final point: our second patient was treated with ibuprofen before features of infectious endocarditis appeared. An association has previously been noted between the use of nonsteroidal anti-inflammatory drugs and progression of GABHS infection to an invasive form [10].

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References

Table 1. Clinical features and outcome of 6 cases of group A β-hemolytic Streptococcus endocarditis in children.

<table>
<thead>
<tr>
<th>Patient no. [ref]</th>
<th>Age, sex</th>
<th>Days from onset of diagnosis to diagnosis</th>
<th>Previous heart disease</th>
<th>Heart murmur</th>
<th>Splenomegaly</th>
<th>Roth’s spots or splinters</th>
<th>Emboli</th>
<th>Valve(s) affected</th>
<th>Outcome</th>
</tr>
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<tbody>
<tr>
<td>1 [5] 2 y, M</td>
<td>14</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Peripheral</td>
<td>No</td>
<td>Aortic, mitral</td>
<td>Died</td>
</tr>
<tr>
<td>2 [6] 16 y, M</td>
<td>10</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>NR</td>
<td>Renal/peripheral</td>
<td>No</td>
<td>Aortic</td>
<td>Renal failure; R</td>
</tr>
<tr>
<td>3 [6] 2 y, M</td>
<td>16</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>NR</td>
<td>NR</td>
<td>No</td>
<td>Mitral</td>
<td>Died</td>
</tr>
<tr>
<td>4 [7] 4 moo, F</td>
<td>16</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Aortic</td>
<td>Died</td>
</tr>
<tr>
<td>5 [8] 4 y, F</td>
<td>11</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Cerebral, peripheral</td>
<td>Aortic surgery; R</td>
</tr>
<tr>
<td>6 [9] 3 y, F</td>
<td>5</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Mitral</td>
<td>Left hemiplegia</td>
</tr>
</tbody>
</table>

NOTE. NR, not reported; R, recovered; ref, reference.
Paradoxical Reaction Syndrome Complicating Aural Infection Due to Mycobacterium tuberculosis during Therapy

A patient with an aural mass and progressive unilateral deafness caused by a locally extensive process due to *Mycobacterium tuberculosis* was evaluated. A discussion of this uncommon infection [1] and an early complication during antituberculosis therapy are presented.

A 21-year-old Pakistani man presented with an aural mass and hearing loss in the right ear. The patient complained of right ear pain, headache, tinnitus, and recurring mucopurulent discharge from the external ear for 3 months. His medical history was unremarkable, except for bilateral retinal detachment caused by a locally extensive process due to *M. tuberculosis* during early childhood. He denied cough, weight loss, or night sweats. He may have received bacille Calmette-Guérin vaccine as a child.

The patient was well nourished and afebrile. Physical examination revealed a pale fungating mass that bled easily and totally obliterated the right external auditory canal. Cervical and periauricular lymph nodes were nonpalpable. The mastoid process was normal. Neurological examination was unremarkable, except for complete conductive deafness. Laboratory studies disclosed the following: leukocyte count, 4400/μL; hemoglobin level, 13.8 g/dL; platelet count, 201,000/μL; and erythrocyte sedimentation rate, 13 mm H2O/h. Serological testing for HIV type 1 was negative. A chest radiogram was normal, and an MRI of the head with iv gadolinium contrast revealed thrombosis of the left parietal superficial cortical veins. A persistently high opening pressure of 27 cm H2O during lumbar puncture, examination of CSF was negative, and culture of CSF for mycobacteria was negative. Cultures of multiple induced sputum specimens were negative for mycobacteria. The patient was treated for 24 months with daily oral rifampin (600 mg), ethambutol (1200 mg), and ofloxacin (400 mg twice a day).

Within 10 days after initiation of treatment with antituberculous medications, the patient’s course was complicated by the new onset of complex partial seizures and worsening headache. Repeat MRI of the head showed a new ischemic infarction in the temporoparietal area, and magnetic resonance venography revealed thrombosis of the left parietal superficial cortical veins. A persistently high opening pressure of 27 cm H2O was noted during repeated lumbar puncture, although CSF examination and CSF cultures remained negative. No change was noted during the neurological examination. Treatment with high doses of dexamethasone was instituted, and the headaches resolved. The corticosteroid dose was tapered over 5 months and then was discontinued.

He did not have a symptomatic recurrence. The mass in the auditory canal resolved completely, although scarring of the tympanic membrane and discontinuity of the middle ear ossicles were permanent. At the end of therapy, complete resolution of the intracranial process was noted during repeated MRI (figure 1B). However, the unilateral conductive hearing loss failed to improve.

By the turn of the 20th century, the natural progression of this locally destructive infection due to *M. tuberculosis* was well established [2]. After the introduction of antimycobacterial agents, the rate of *M. tuberculosis* infection among pediatric patients with otitis media had significantly declined from 3%-4% to 0.05%-0.9% [3-5].

Expansion of a cerebral mass (tuberculoma) after therapy for *M. tuberculosis* infection or development of multiple new brain lesions during treatment of tuberculous meningitis has acid-fast bacilli and necrotizing granulomas. Analysis of smears of the right ear discharge was positive for acid-fast bacilli, and, 3 weeks later, *M. tuberculosis* was isolated and identified. This strain of *M. tuberculosis* was resistant to isoniazid, streptomycin, and ethionamide. Aside from an opening pressure of 29 cm H2O during lumbar puncture, examination of CSF was negative, and culture of CSF for mycobacteria was negative. Cultures of multiple induced sputum specimens were negative for mycobacteria. The patient was treated for 24 months with daily oral rifampin (600 mg), ethambutol (1200 mg), and ofloxacin (400 mg twice a day).

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Clinical Infectious Diseases 2000;30:625-7
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