Our findings should be interpreted with caution because the study was not population-based, and, indeed, most of the patients were highly selected by a self-reporting mechanism. These cases are, however, quite similar to those described by Haywood et al. [11] and to additional cases known to us that are still in litigation and were unavailable for this study. Moreover, despite its limitations, self-reporting on a Web site has proven to be a useful method for gathering cases that come from a wide geographic area and would otherwise not come to the attention of investigators interested in a specific disease entity.

Acknowledgments

We are deeply indebted to Jacqueline Roemmele and Donna Batt-dorff of the National Necrotizing Fasciitis Foundation for their assistance in completing this study.

Alan L. Bisno, 1,2 Franklin R. Cockerill III, 2 and Carmen T. Bermudez 1

1 Department of Medicine, University of Miami School of Medicine, and 2 Veterans Affairs Medical Center, Miami, Florida; and 3 Divisions of Clinical Microbiology and Infectious Diseases, Mayo Clinic, Rochester, Minnesota

References


Table 1. Physical findings for 15 patients with Group A streptococcal necrotizing fasciitis at the first outpatient visit and at admission to the hospital.

<table>
<thead>
<tr>
<th>Finding</th>
<th>1st Visit</th>
<th>Admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate &gt;90</td>
<td>8 (73)</td>
<td>12 (80)</td>
</tr>
<tr>
<td>Systolic BP &lt;100</td>
<td>2 (18)</td>
<td>9 (60)</td>
</tr>
<tr>
<td>Temperature &gt;37.7°C</td>
<td>5 (45)</td>
<td>5 (33)</td>
</tr>
<tr>
<td>Localized edema</td>
<td>4 (36)</td>
<td>13 (87)</td>
</tr>
<tr>
<td>Localized erythema</td>
<td>3 (27)</td>
<td>7 (47)</td>
</tr>
<tr>
<td>Cutaneous lesion</td>
<td>2 (18)</td>
<td>8 (53)</td>
</tr>
</tbody>
</table>

a Includes only the 11 patients for whom primary care records were available for review.

b One patient with varicella and 1 with an abrasion.

was localized pain. When there is an accompanying history of preceding (often nonpenetrating) trauma, the clinical picture of necrotizing fasciitis may closely mimic that of musculoskeletal strain. Other patients may manifest nonspecific symptoms such as feverishness, malaise, chills, and arthralgia that are easily confused with a viral syndrome. An unexpected finding was the frequency of gastrointestinal symptoms, such as nausea, vomiting, and diarrhea, which were present in 7 (47%) of the patients on initial outpatient presentation.

Although the streptococci that cause necrotizing fasciitis are predominantly group A, rarely this entity may be due to strains of other serogroups, including group B [5, 6], as was the case for 1 of our patients.

Inflammation at the fascial level is associated with intense discomfort that is often unrelated by analgesic agents such as NSAIDs or even narcotics. (Indeed, there are theoretical reasons to postulate that NSAIDs may even exacerbate the condition [7].) This finding, particularly if accompanied by tachycardia and even low-grade fever, should prompt additional studies (e.g., WBC count and sedimentation rate or quantitation of C-reactive protein) that might suggest the presence of a bacterial infection, as opposed to a musculoskeletal problem or viral infection. In addition, such patients should be followed up promptly, either by telephone contact or another visit.

Nevertheless, it must be acknowledged that in some cases it may simply be impossible to diagnose early streptococcal necrotizing fasciitis confidently on the basis of clinical examination and the routine blood studies generally performed in the primary care setting. The utility of imaging studies in diagnosing early streptococcal necrotizing fasciitis is uncertain, although some authors have reported promising results with MRI in differentiating cellulitis from necrotizing fasciitis [8, 9]. Stamenkovic and Lew [10] performed frozen-section biopsy within 0–4 days (average, 21 h) of the onset of symptoms of necrotizing fasciitis, which led to earlier diagnosis and a statistically significant improvement in mortality when compared with cases in which the diagnosis was made on clinical grounds alone. A less invasive test that is applicable to problematic cases is clearly required.
Transverse Myelitis Associated with Probable Cat-Scratch Disease in a Previously Healthy Pediatric Patient

We evaluated a pediatric patient for transverse myelitis associated with *Bartonella henselae* infection. There was no adenopathy in our patient, but the diagnosis was made serologically. It is necessary to keep cat-scratch disease in mind even in the absence of typical findings.

Cat-scratch disease is caused by the organism *Bartonella henselae* and is classically described as self-limited regional lymphadenitis. Neurological complications are rare, with estimated reports of 0.17%–3% of patients developing these manifestations [1]. The most common CNS manifestations of cat-scratch disease are coma and the acute onset of seizures [1]. A case of recurrent encephalopathy associated with cat-scratch disease was recently reported, suggesting that the diagnosis be considered for children with unexplained recurrent seizures and altered mental status [1–4].

We recently evaluated a patient who developed transverse myelitis associated with probable *B. henselae* infection. A case of transverse myelitis as a CNS complication of cat-scratch disease was described in an adult patient before the development of specific serology to confirm infection [2]. Our case demonstrates this entity in a pediatric patient in the era of serology-based diagnosis of the disease.

A 7-year-old male was admitted to the Pediatric Intensive Care Unit at West Virginia University Children’s Hospital (Morgantown) for evaluation of lower-extremity weakness. Two days before admission, he complained of gradually worsening pain in his neck, lower back, and below his knees. This pain awakened him from sleep. The evening before admission, he was unable to stand or use his right hand. This weakness progressed to involve his entire right side and eventually his left lower extremity. On the morning of his admission, the patient described decreased sensation in both lower extremities and urinary incontinence. His parents denied periods of altered mental status, and he denied headache, blurred vision, or hearing loss. His parents also denied recent travel, skin rash, tick bite, fever, cough, nausea, vomiting, diarrhea, ill contacts, or recent viral illness.

His medical history revealed no chronic medical conditions and up-to-date immunization status. He lived in Spencer, West Virginia, with his parents and a healthy 6-year-old brother. The family lived in a 2-story house, had gas heat, and city water. The patient’s grandmother was the owner of 2 indoor cats and 1 dog. The ages of the cats were 8 months and 6 years. The patient spent 3 days per week at his grandmother’s home and had sustained multiple cat scratches on his fingers and wrists. Family history was noncontributory.

At the time of physical examination, the patient’s temperature was 38.5°C. The blood pressure was 94/42 mm Hg, pulse rate was 106, and respiration rate was 22. He was in no acute distress, was lying on his back, and was interactive with the examiners. Significant findings included normal reactive pupils and a symmetrical face. He had cervical spine tenderness and right torticollis. There was decreased muscle tone and sensation of the right lower extremity and inability to move his right side. Sensation was also decreased in the left lower extremity. Deep tendon reflexes were 2+ and equal throughout. Babinski’s sign was present bilaterally. Strength was 2/5 in the right upper extremity and 1/5 in the right lower extremity. Strength was 4/5 in the left upper and lower extremities. Cranial nerves were intact, and there were no meningeal signs. There was a papule, which had been present for 2–3 weeks, on the fifth digit of the left hand where the patient had sustained a scratch. There was no lymphadenopathy present.

At admission to the intensive care unit, laboratory studies revealed normal WBC and differential blood cell counts, hematocrit of 33.2%, and normal platelet count. Electrolyte,
blood urea nitrogen, and creatinine concentrations were also normal, as were the lactate dehydrogenase level, prothrombin time, and partial thromboplastin time. The patient’s erythrocyte sedimentation rate was 34 mm/h. CT of the head and spinal cord revealed no evidence of acute hemorrhage. MRI revealed cord edema from the C2 to T10 region of the spinal cord and an abnormal signal extending down to the T10 level (figures 1 and 2). A working diagnosis of transverse myelitis was formulated. Additional diagnostic tests were negative, including serologies for hepatitis, Epstein-Barr virus, Lyme disease, and mycoplasma.

The patient was empirically treated with intravenous steroids (doses of which were tapered over 3 weeks) and a 7-day course of acyclovir. Repeated MRI of the brain and spinal cord was performed on day 2 of admission; this study revealed decreased edema. CSF was obtained from the cisterna magna; analysis of the fluid revealed 30 WBCs (88% lymphocytes), 6 RBCs, protein level of 40 mg/dL, and glucose level of 72 mg/dL. The myelin basic protein level was elevated at 13.6 ng/mL (normal level, <5 ng/mL). CSF was also sent for determination of enterovirus titers and PCR analysis for herpesvirus; results of these tests were normal. Routine, aerobic, anaerobic, fungal, and acid-fast cultures of CSF did not reveal organisms. Serum titers of indirect fluorescent antibody to *B. henselae* and *Bartonella quintana* were evaluated; titers of IgG and IgM antibodies to both organisms were 1:2048 and <1:20, respectively. The specimen was tested at the Microbiology Division of American Medical Laboratories (Cypress, CA). Titers in convalescent-phase sera were not determined.

The patient showed slow neurological recovery and after 9 hospital days was discharged to a rehabilitation center; he spent 3 weeks at the center, where he received aggressive muscle strength training and occupational therapy directed at fine motor skills in writing and utensil use. The patient reportedly spent 3 additional months in an outpatient physical and occupational therapy program under the guidance of a pediatric neurologist. He is currently attending public school and has functionally made a full recovery, maintaining an active lifestyle and participating in organized sports at age 10 years.

Cat-scratch disease usually presents as subacute regional lymphadenopathy, and causation is determined with serological testing for *B. henselae*. Rarely is the diagnosis made with culture isolation of the organism. Most of the large series of manifestations of cat-scratch disease were based on diagnosis by skin testing, but now more cases can be confirmed with serology. Close contact with or a scratch from a kitten is the most common predisposing factor in the development of infection. A number of reports describe the natural history of atypical disease [3, 4]. Hepatic and splenic involvement, fever of unknown origin, and CNS involvement are often implicated in atypical disease [3, 4]. Regional lymphadenopathy from the primary inoculation site is the rule even in atypical disease, but it is not

Figure 2. Sequential sagittal T2-weighted fast-spin echo images of the cervical and thoracic spine of a 7-year-old boy with probable cat-scratch disease. Images reveal a diffuse process involving the intramedullary portion of the spinal cord from the level of C2 through T10 with diffuse increased signal intensity; this appearance is most consistent with a diffuse inflammatory process such as myelitis. Arrows indicate the peak area of cord edema.
Influenza A–Associated Encephalopathy with Bilateral Thalamic Necrosis in Japan

Two cases of acute encephalopathy in young children clearly showed evidence of influenza A virus infection and bilateral thalamic lesions. Influenza-associated encephalopathy with bilateral thalamic lesions has mostly been reported in Japan; it differs from Reye’s syndrome in several respects. Other factors in addition to influenza virus infection may have contributed to the etiology of encephalopathy in our case patients.

It is estimated that >100 children die of influenza-associated encephalopathy (influenza encephalopathy) in Japan every year; these cases are typically associated with sudden onset of high-grade fever, severe convulsions, rapidly progressive coma, and death within 2 or 3 days [1–5]. Cases of influenza encephalopathy in Japan resemble acute cases of influenza A virus–associated encephalopathy reported by Delorme et al. [6], rather than cases of Reye’s syndrome or postinfluenza encephalopathy seen during an influenza epidemic [7]. There are clinical distinctions between influenza encephalopathy and Reye’s syndrome. In influenza encephalopathy, there is no history of taking aspirin; there is rapid loss of consciousness, with coma ensuing within 24 h; convulsions occur in almost all patients in the early stage of onset; and hyperammonemia is rarely seen. Moreover, neuroimaging of patients with influenza encephalopathy often reveals bilateral thalamic lesions, which have not been demonstrated in cases of Reye’s syndrome. In the winter season of 1998–1999, we encountered 2 fatal cases of typical influenza A encephalopathy with characteristic brain lesions.

A 3-year-old girl (case patient 1) became ill with a cough on 12 January 1999; she had a high-grade fever develop on 13 January. On 14 January, she was treated with cefdinir (antimicrobial),