Cat Scratch Disease Presenting With Peripheral Facial Nerve Paralysis

ABSTRACT. Acquired peripheral facial nerve paralysis is a relatively common disorder that affects both children and adults. The most frequent nontrauma-related etiologies in otherwise neurologically intact patients are idiopathic (Bell's palsy) and infectious, which includes otitis media, herpes zoster, Lyme disease, herpes simplex virus, Epstein–Barr virus, and *Mycoplasma pneumoniae*.[1–5]

Cat scratch disease (CSD) is typically a subacute, regional lymphadenitis caused by *Bartonella henselae* that is seen in children and young adults. CSD most often has a benign, self-limited course. However, 11% of CSD patients may present atypically, most commonly with Perinaud's oculoglandular syndrome or acute encephalopathy.[6–11]

We present a child with the first reported case of acute facial nerve paralysis in serologically proven CSD with typical lymphadenitis.

ABBREVIATION. CSD, cat scratch disease.

CASE REPORT

A previously healthy 3-year-old boy was admitted to our hospital in October 1996 with a 2-day history of progressive left facial weakness. The child had been intermittently febrile at home (maximum 101.7°F rectal) for 4 days and a left axillary lymphadenitis was diagnosed by his physician 3 days before admission and treated with amoxicillin. There was a history of scratches to the left arm by a 4-month-old kitten 3 weeks before the child's admission and to the right arm during the previous week. There had been no tick or other animal exposures. Interestingly, the patient's cousin, who had been exposed to the same family of cats, was diagnosed with CSD (including osteolytic lesions) 1 year earlier. On examination, the patient was alert and verbal, although cranky. He was afebrile, with a respiratory rate of 20 breaths per minute and a pulse of 88 beats per minute. He weighed 15 kg (50% percentile) and exhibited normal developmental milestones. He had a dense peripheral left facial nerve paralysis (Fig 1). The remainder of the neurologic examination (cranial nerves, strength, deep tendon reflexes, and sensation) was normal. There was mild tympanosclerosis on the right side. The left tympanic membrane was normal. He had a subconjunctival hemorrhage of recent onset on the left side. Otherwise, the conjunctivae were normal and had no injection or granuloma. No preauricular lymph nodes were present. The fundi were normal, and there were no meningeal signs. There was a 2 × 3-cm tender erythematous firm area in the left axilla that was consistent with lymphadenitis. No other adenopathy was noted. There were several erythematous nonblanching papules at the posterior hairline and over both shoulders, and there was a well-healed scratch on the right forearm.

Initial laboratory studies included a white blood cell count of 13 700/mm^3 (43% segmented neutrophils, 2% band forms, 37% lymphocytes, 13% monocytes, 4% eosinophils, and 1% atypical lymphocytes), hemoglobin of 11.3 g/dL, and a platelet count of 391 000/mm^3. Serum alanine aminotransferase level was 30 U/L. Lumbar puncture revealed white blood cell count of 1/mm^3, red blood cells 56/mm^3, glucose 57 mg/dL, and protein 13 mg/dL.

The patient was treated initially with intravenous ceftriaxone (100 mg/kg per day) and oral trimethoprim–sulfamethoxazole (10 mg/kg per day). The next day, prednisone (2 mg/kg per day) was added.

Forty-eight hours after admission, the patient remained afebrile and was playful, with some improvement in the lymphadenitis and no change in the facial paralysis. Lyme disease and Epstein–Barr virus studies were negative, as were blood and cerebrospinal bacterial cultures. The patient was discharged home on trimethoprim–sulfamethoxazole and prednisone with a clinical diagnosis of CSD. Cat scratch (*B henselae*) serum serology later returned elevated at 1:256 (1:64 is considered positive). Cerebrospinal fluid polymerase chain reaction did not detect *B henselae* or *B quintana*. Lymph node biopsy or excision was not felt to be clinically indicated, thus, polymerase chain reaction studies and pathology on the lymph node were not performed.

Ten days after discharge, the skin lesions had resolved, and the lymphadenitis was decreased to 1 cm with no tenderness or erythema. The facial nerve palsy was significantly improved. Soon thereafter, it completely resolved.

COMMENT

The history of previous kitten scratches to the arm and the ipsilateral axillary lymphadenitis was quite consistent with typical CSD for this patient. However, the onset of facial nerve paralysis 1 day after the lymphadenitis presented was interesting and not expected as a typical manifestation of CSD. In the absence of trauma and with an otherwise normal neurologic and tympanic membrane examination, we considered other causes of acquired facial nerve paralysis in children. These included Lyme disease (in an endemic Lyme disease area), Epstein–Barr virus, herpes simplex virus, *M pneumoniae*, and herpes zoster.[1–5] We deemed it likely that the facial nerve palsy was related to the total clinical picture that included the fever and lymphadenitis. Ceftriaxone was begun pending the Lyme disease test results.

The recognized clinical manifestations of CSD continue to increase and include lymphadenitis, the oculoglandular syndrome, encephalopathy, endocarditis, bacillary angiomatosis and peliosis, skeletal lesions, transient maculopapular rash, erythema nodosum, hemolytic anemia, thrombocytopenia, atypical pneumonia, glomerulonephritis, and disseminated hepatic and splenic lesions.[6–9] The most common neurologic mani-festation of CSD is acute encephalopathy, which occurs in 2% to 3% of patients and is more common in adults than in children. Seizures, cerebellar ataxia, hemiparesis,

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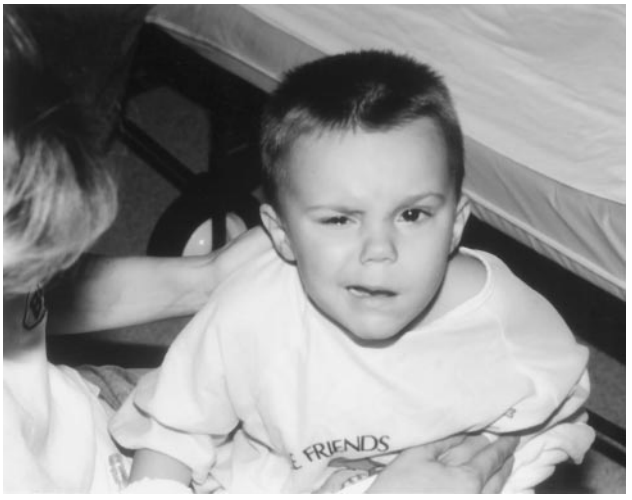

Fig 1. Left facial nerve paralysis noted at admission to hospital.

myelitis, hearing loss, sixth nerve palsy, and aphasia all have been associated with CSD encephalopathy. Up to 20% to 30% of patients with CSD encephalopathy may have cerebrospinal fluid pleocytosis.[12,13] Recently, *B quintana* has been associated with central nervous system pathology.[14]

Abnormal neurologic findings in the absence of encephalopathy in CSD are uncommon but may include neuroretinitis and peripheral neuritis of the extremities (pain, edema, and paresthesias).[13] Facial nerve paralysis in proven CSD has not been clearly established in the literature. There is one case report of a 9-year-old boy with parotitis associated with a partial facial nerve paralysis of the marginal mandibular branch (off the cervicofacial branch) of the facial nerve that courses through the parotid gland. The diagnosis was based on a lymph node biopsy that was "consistent with a condition such as CSD," coupled with the retrospective recall of cat contact without known scratches.[15] A CSD case series focusing on CSD encephalopathy mentions facial nerve paralysis in two children, 18 months and 8 years of age, in association with CSD oculoglandular syndrome, which includes conjunctivitis, conjunctival granuloma, and preauricular lymphadenitis. It is specifically mentioned that these cases were not studied as exhaustively as those with CSD encephalopathy and that the diagnosis was made clinically with no specific details given.[13] The physical findings of the oculoglandular syndrome or parotitis were not seen in our patient. We did not perform additional studies to eliminate neuroretinitis, but decreased visual acuity was not suspected by history or physical examination.

Given the history and clinical course of illness in this child, coupled with the strongly positive serology to *B henselae*, we are confident that all findings, including the facial nerve palsy, were attributable to CSD. The clinical criteria for diagnosing CSD may vary, and when atypical features are present that have broad differential diagnoses, it is important to have serologic confirmation when making this diagnosis. Although evidence for antibiotic efficacy in CSD is largely anecdotal, our decision to treat with trimethoprim–sulfamethoxazole was based on published reports and on our own experience with this antimicrobial agent.[11,16] We also prescribed a short course of oral steroids to possibly decrease the inflammation of the seventh cranial nerve.

Transient facial nerve paralysis is certainly not a common finding in CSD. With the concomitant skin findings, this case could be considered evidence of disseminated CSD. An alternative explanation for the facial nerve involvement is that in addition to kitten scratches to the arm resulting in the lymphadenitis, this child may have been scratched about the face and neck (thus the skin lesions on initial examination) or had had direct inoculation into his oral/nasal mucosa or conjunctiva with contiguous spread resulting in the facial nerve palsy.

Facial nerve paralysis has not been reported previously in typical CSD. Based on this case, we believe that CSD at least should be considered in a child with significant kitten exposure who presents with acquired facial nerve palsy even without lymphadenitis, parotitis, or oculoglandular syndrome. The list of differential diagnoses of acquired facial nerve paralysis is quite lengthy, but it does not currently include CSD. We present this case to alert clinicians to this neurologic sequela for CSD. The wide spectrum of presentations in CSD should continue to keep pediatric caregivers ever-vigilant in considering the diagnosis of CSD and inquiring about exposure to cats.

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