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Case Report

A pediatric case with peripheral facial nerve palsy caused by a granulomatous lesion associated with cat scratch disease

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Abstract

Background: Cat scratch disease is a common infectious disorder caused by *Bartonella henselae* that is transmitted primarily by kittens. It typically exhibits a benign and self-limiting course of subacute regional lymphadenopathy and fever lasting two to eight weeks. The most severe complication of cat scratch disease is involvement of the nervous system, such as encephalitis, meningitis, and polyneuritis. Peripheral facial nerve palsy associated with *Bartonella* infection is rare; few reported pediatric and adult cases exist and the precise pathogenesis is unknown.

Case report: A previously healthy 7-year-old boy presented with fever, cervical lymphadenopathy, and peripheral facial nerve palsy associated with serologically confirmed cat scratch disease. The stapedius muscle reflex was absent on the left side and brain magnetic resonance imaging revealed a mass lesion at the left internal auditory meatus. The patient's symptoms and imaging findings were gradually resolved after the antibiotics and corticosteroids treatment.

Conclusions: The suspected granulomatous lesion was considered to have resulted from the host's immune reaction to *Bartonella* infection and impaired the facial nerve. This is the first case report providing direct evidence of peripheral facial nerve palsy caused by a suspected granulomatous lesion associated with cat scratch disease and its treatment course.

Keywords: *Bartonella henselae*; Cat scratch disease; Facial nerve palsy; Magnetic resonance imaging; Minocycline

1. Introduction

Cat scratch disease (CSD) is a human infection caused by *Bartonella henselae* (*B. henselae*), a gram-negative bacillus commonly found in feline erythrocytes and fleas. *B. henselae* can contaminate feline saliva and infect humans through biting or scratching [1]. As *Bartonella* species are difficult to culture, the serological study of specific immunoglobulin (Ig)G is generally recommended for CSD diagnosis. A positive IgM test suggests acute infection, although the production of IgM is brief in CSD [2].

CSD generally afflicts children and young adults as a benign and self-limiting course lasting two to eight weeks [1,3,4]. Regional lymphadenopathy manifests one to two weeks after infection mainly in ipsilateral

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upper extremities, the neck, and the jaw [1,5]. While the prognosis of CSD is generally good, it may become complicated with such neurological conditions as encephalitis, meningitis, and polyneuritis [5,6]. Peripheral facial nerve palsy associated with *Bartonella* infection is very rare; only a few published pediatric and adult cases of CSD exist, and its precise pathogenesis is unknown [3,4,7,8]. We herein describe the clinical course of a 7-year-old boy who had fever, cervical lymphadenopathy, and peripheral facial nerve palsy associated with serologically confirmed CSD.

2. Case report

An otherwise healthy 7-year-old boy presented with a remittent fever reaching 40 degrees Celsius, left cervical lymphadenopathy of 1–2 cm in size, and left facial nerve palsy. He had experienced a transient fever with subsequent erosive lesions on the left cheek one month prior that recurred with left facial nerve palsy at crying a day before admission (Fig. 1). Initial laboratory findings included white blood cell (WBC) count 8200/μL and C-reactive protein (CRP) 0.2 mg/dl. Ultrasonography (US) revealed left cervical and parotid gland lymph node swelling. He was diagnosed as having peripheral facial nerve palsy and began treatment with intravenous acyclovir and prednisolone for 5 days followed by oral prednisolone for 11 days. His fever improved transiently but soon recurred with deteriorated laboratory findings of WBC count 24,200/μL and CRP 3.8 mg/dl. Despite switching to from acyclovir to flomoxef and azithromycin, the patient's high fever, cervical lymphadenitis, and facial palsy persisted. He was transferred to our hospital at 23 days after the onset of facial nerve palsy.

On admission, history taking on the erosive abrasion on his left cheek revealed that his family had started keeping a kitten three months prior to symptom onset. Left-side palsy prevented the boy from closing his eye and lifting the corner of the mouth and eyebrow. According to the Yanagihara grading system [9] for facial palsy, his palsy was scored 8 (ranging from the score 0, complete palsy, to 40, no palsy) Other cranial nerve functions and neurological signs were normal, and no meningeal signs were observed. Laboratory findings disclosed WBC count 9400/μL, CRP 1.8 mg/dl, and no pleocytosis in the cerebrospinal fluid. US revealed multiple swollen lymph nodes at the left neck and parotid gland. Magnetic resonance imaging (MRI) identified a mass lesion at the left internal auditory meatus (Fig. 2a; arrow). The stapedius muscle reflex as tested by an otolaryngologist was absent on the left side, which was consistent with mass lesion causing facial nerve compression at the level of the facial nerve canal. Based on his history and findings, the boy was prescribed minocycline after obtaining informed consent from his parents about a possible teething issue. He was also administered ceftriaxone since Lyme disease could not be completely ruled out. His fever, lymphadenopathy, and laboratory findings all improved within a week. Oral prednisolone and amoxicillin were continued for two weeks since his facial nerve palsy persisted and the mass lesion remained enhanced in serial MRI on day 33 (Fig. 2b–d; arrows). Afterwards, the symptoms of facial nerve palsy slowly but gradually improved and became resolved at six months after onset. Follow-up MRI on day 70 showed the disappearance of the mass lesion at the left internal auditory meatus (Fig. 2e). Immunofluorescence assays revealed that *B. henselae*-specific IgM had been negative but IgG was greater than 1:1024 in previously obtained serum.

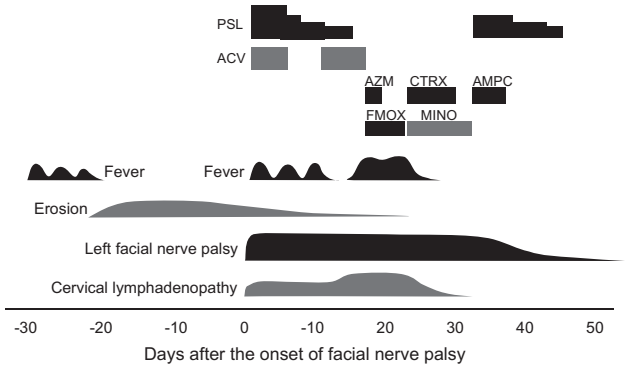

Fig. 1. Clinical course. Fever and cervical lymphadenopathy improved after the administration of CTRX and MINO, followed later by facial nerve palsy resolution by PSL. ACV, acyclovir; AMPC, amoxicillin; AZM, azithromycin; CTRX, ceftriaxone sodium; FMOX, flomoxef sodium; MINO, minocycline hydrochloride; PSL, prednisolone.

3. Discussion

The most common neurological manifestation of CSD is acute encephalopathy, which occurs in 2–3% of patients and is more prevalent in adults. Seizures, cerebellar ataxia, hemiparesis, myelitis, hearing loss, abductor nerve palsy, and aphasia have all been associated with encephalopathy as well [10]. Other neurological manifestations in the absence of encephalopathy are uncommon but may include neuroretinitis and peripheral neuritis [10]. Peripheral facial nerve palsy associated with *Bartonella* infection is very rare, with only a few reported pediatric [3,7] and adult cases [4,8]. One report described a 9-year-old boy with parotitis complicated with partial facial nerve palsy of the mandibular marginal branch coursing through the parotid gland. The cause of the palsy was suspected to be direct compression by the parotid gland [7]. Another case exhibited peripheral facial nerve palsy associated with a granulomatous lesion in the parotid gland that was detected by MRI [4]. The remaining reports suggested a pathophysiology of disseminated *B. henselae* or bacterial spreading [3], but the above considerations on patho-

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