SHORT REPORT

Facial nerve palsy associated with *Rickettsia conorii* infection

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Abstract

Facial nerve palsy has been occasionally attributed to infectious agents, but *Rickettsiae* species have not been documented as causative agents. We report two adolescent girls with facial nerve palsy and serological evidence of *R conorii* infection. These cases indicate that rickettsioses should be included among the causes of facial nerve palsy, particularly in endemic areas.

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Keywords: Bell's palsy; facial nerve palsy; Rickettsia conorii; rickettsial infections

Acute unilateral facial nerve palsy has occasionally been attributed to infectious agents, including the herpes, mumps, rubella, influenza, respiratory syncytial, human immunodeficiency, and coxsackie viruses, and *Borrelia burgdorferi*, *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and group B streptococci.[1] A nonspecific immunological response to infection, leading to facial nerve compression and degeneration has been suggested as the pathogenic mechanism. *Rickettsiae* species have not been implicated in the aetiology of facial nerve palsy. We report two cases of facial nerve palsy in adolescents with serological evidence of *Rickettsia conorii* infection.

Case reports

CASE 1

A 14 year old girl was admitted with a six day history of fever, cough, and conjunctivitis. Physical examination revealed bilateral nonpurulent conjunctivitis, palpable liver and spleen 3 cm subcostally, cervical lymphadenopathy, and right facial weakness. Blood pressure was 110/60 mm Hg. Chest auscultation, examination of the remaining cranial nerves, and tympanoscopy were normal. Tympanometry revealed a normal tympanogram of type A in both ears. The acoustic reflexes, measured by ipsilateral and contralateral stimulation, were absent in the right and normal in the left ear. White blood cell count was 13.7×10^9/l; neutrophil count, 6.98×10^9/l; haemoglobin, 1.72 mmol/l; and platelet count, 292×10^9/l. Erythrocyte sedimentation rate was 11 mm/h; alanine aminotransferase, 45 U/ml; and aspartate aminotransferase, 68 U/ml. Chest *x* ray was normal. Serology for Epstein–Barr virus, cytomegalovirus, herpesviruses 1 and 2, adenovirus, coxsackie viruses, *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and *Borrelia burgdorferi* was negative for acute infection. IgM and IgG antibodies against *R conorii* at titres of 1/960 and 1/3840 respectively were detected by indirect immunofluorescence test. IgM antibodies against *R typhi* and *Coxiella burnetii* at a titre of 1/100 and IgG antibodies at titres of 1/960 and 1/400 respectively were also detected.

Oral clarithromycin and prednisolone 2 mg/kg were administered. The fever subsided, but the facial weakness progressed to paralysis over the next three days. Prednisolone was continued at the same dose for two more days and subsequently tapered within five days. Two weeks later IgM and IgG antibodies against *R conorii* were 1/3840 and 1/15 360 respectively, thus confirming acute *R conorii* infection. Titres for *R typhi* and *Coxiella burnetii* remained low. Facial palsy subsided three months later.

CASE 2

A 12 year old girl was admitted with a five day history of progressive right facial side weakness, following a flu like febrile illness two weeks previously. Physical examination revealed a drooped mouth corner, impaired facial muscle movement, and eye closure on the right side. Liver and spleen were palpable at 4 cm and 3 cm respectively. Blood pressure was 125/70 mm Hg. Examination of the other cranial nerves and tympanoscopy was normal. Tympanometry revealed a normal tympanogram of type A in both ears. The acoustic reflexes, measured by ipsilateral and contralateral stimulation, were absent in the right and normal in the left ear. White blood cell count was 5×10^9/l; neutrophil count, 1.85×10^9/l; haemoglobin, 1.89 mmol/l; and platelet count, 390×10^9/l. Erythrocyte sedimentation rate was 31 mm/h; alanine aminotransferase, 48 U/ml; and aspartate aminotransferase, 60 U/ml. Serology for Epstein–Barr virus, cytomegalovirus, herpesviruses 1 and 2, adenovirus, coxsackie viruses, *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and *Borrelia burgdoferi* was negative for acute infection. Indirect immunofluorescence test was positive for *R conorii*, with an IgM titre of 1/400 and IgG titre of 1/960. IgG antibodies against *R typhi* and

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Coxiella burnetii at titres of 1/400 and 1/100 respectively were also detected.

Oral prednisolone 2 mg/kg was administered for five days and gradually tapered within five days. A repeat serology for *R conorii* two weeks later revealed IgM antibody titre of 1/1600 and IgG antibody titre of 1/3840, thus confirming a recent *R conorii* infection. Four months later, on a follow up evaluation, the girl had normal right facial nerve function; IgM and IgG titres against *R conorii* were 1/100 and 1/1600 respectively.

Discussion

Mediterranean spotted fever, the typical systemic *R conorii* infection, usually presents with spiking fever, myalgia, headache, hepatosplenomegaly, and a maculopapular or purpuric rash. However, atypical cases have been reported.[2 3] The disease is considered endemic in southern Europe, and epidemiological studies in certain parts of Greece have shown considerable seroprevalence.[4]

Our first patient was admitted with a diagnosis of lower respiratory tract infection. Facial palsy was noted later in the course of her illness. She was investigated for rickettsial infection because of history of animal contact with the household dogs of the family. The second patient presented with typical Bell's palsy and a preceding upper respiratory tract infection. She was investigated for rickettsial infection because of the presence of mice on the family's farm. Tick bite signs were not present in either patient.

Neurological involvement in rickettsial infections is encountered in a minority of cases with severe systemic manifestations. Disturbances of consciousness, meningoencephalitis, and a case of Guillain–Barré syndrome in an adult patient with *R conorii* infection have been reported.[5 6] *Rickettsiae* are not currently included among the aetiological agents of facial nerve palsy. In a series of *R conorii* infection cases in children from Sicily, two cases of facial nerve palsy were noted; however, clinical details of these two patients were not reported.[3] Six patients with facial nerve palsy were hospitalised in our department during 1999. Two of these had a clinical presentation and serological confirmation of acute Epstein–Barr virus infection; serological evidence of *R conorii* infection was present in two others. In the remaining two cases no aetiological agent was found.

The indirect immunofluorescence test is generally considered to be a sensitive and specific test for confirming the diagnosis of *R conorii* infection. Titres above 1/150 for IgM and above 1/400 for IgG specific antibodies are suggestive of acute infection. As low titres of specific IgM antibodies may be present in many systemic diseases,[3 4] confirmation of the diagnosis requires a fourfold increase of the specific antibody titre in a second sample, three to four weeks after the onset of symptoms.

Tetracyclines and chloramphenicol, given early in the course of the disease, are considered to be the treatment of choice for all rickettsial infections. Our patients did not receive any specific treatment, as confirmation of the diagnosis was rather delayed. A short course of corticosteroids was administered, although their role in the treatment of facial nerve palsy remains controversial.[7]

In conclusion, rickettsial infection should be included among the causes of facial nerve palsy in endemic areas, particularly in patients with a history of animal contact. The outcome of such cases associated with rickettsioses seems excellent.

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