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Cervical lymphadenitis due to mycobacterium bohemicum

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detected in CSF. Transudation from blood is unlikely, because the intrathecal SAA of IgG antibody to *C. pneumoniae* was 14 times higher than the blood SAA. A ratio of intrathecal SAA to blood SAA that is >2 supports the hypothesis that the immune reaction is more important within the CNS and strongly suggests the intrathecal presence of the microorganism [3].

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References

1. Grayston JT. *Chlamydia pneumoniae* (TWAR). In: Mandell GL, Bennett JE, Dolin R, eds. *Mandell, Douglas and Bennett's principles and practice of infectious diseases*. 4th ed. New York: Churchill Livingstone, 1995: 1696–701.
2. Schuller E, Sagar HJ. Local synthesis of CSF immunoglobulins. A neuro-immunological classification. *J Neurol Sci* 1981;51:361–70.
3. Schuller E. L'analyse de l'immunité intrathécale. *Presse Med* 1988;17: 155–60.
4. Fryden A, Kihlstrom E, Maller R, Persson K, Romanus V, Ansehn S. A clinical and epidemiological study of "ornithosis" caused by *Chlamydia psittaci* and *Chlamydia pneumoniae* (strain TWAR). *Scand J Infect Dis* 1989;21:681–91.
5. Sundelof B, Gnarpe H, Gnarpe J. An unusual manifestation of *Chlamydia pneumoniae* infection: meningitis, hepatitis, iritis and atypical erythema nodosum. *Scand J Infect Dis* 1993;25:259–61.
6. Socan M, Beovic B, Kese D. Chlamydia pneumonia and meningoencephalitis. *N Engl J Med* 1994;331:406.
7. Korman TM, Turnidge JD, Grayson ML. Neurological complications of chlamydial infections: case report and review. *Clin Infect Dis* 1997;25:847–51.

Cervical Lymphadenitis Due to *Mycobacterium bohemicum*

Nontuberculous mycobacterial lymphadenitis, traditionally imputed to *Mycobacterium scrofulaceum*, has been shown in recent years to be often due to members of the *Mycobacterium avium* complex [1]. Very recently, however, a number of previously unrecognized mycobacteria have been found to be involved in such pediatric disease. We report here a case of laterocervical lymphadenitis due to the most recently recognized mycobacterial species, *Mycobacterium bohemicum* [2].

An 11-year-old boy was hospitalized because of a 3-week history of nonaching swelling in the left submandibular gland, with no associated erythema. Hematologic findings were normal, and serology was negative for cytomegalovirus, toxoplasmosis, and HIV.

An echographic scan showed 2 lymph nodes of ~2 cm in diameter. Histologic examination of a needle aspirate revealed granulomatous inflammation and acid-fast bacilli. A mycobacterium grew in cultures of a subsequent needle aspirate, in both radiometric and solid media, and treatment with amikacin, clarithromycin, and ethambutol was initiated.

Two weeks later, however, the whole lymphonodal pack was removed, and a mycobacterium from the excised material was again isolated in culture. The isolate, which was scotochromogenic, had biochemical and cultural features grossly compat-

ible with *M. scrofulaceum* (semiquantitative catalase findings were the only ones noncompatible).

High-performance liquid chromatography of cell-wall mycolic acids revealed a pattern that, although similar, did not overlap that of the representative profile of this species. Finally, the genetic sequencing of hypervariable regions of 16S rRNA revealed identity with the newly described *M. bohemicum*. This finding was confirmed by the compatibility of almost all standard features (our isolate differed from the type strain only in that it failed to grow at 25°C) and by a mycolic acid profile in high-performance liquid chromatography that overlapped that of the reference strain for this novel species.

Antimicrobial susceptibility testing in liquid radiometric medium showed the following MICs ($\mu\text{g/mL}$): amikacin, 2; ciprofloxacin, 1; clarithromycin, 2; rifabutin, 0.12; rifampin, 2; streptomycin, 2; and ethambutol, 8. These MICs revealed full susceptibility to all antimicrobials tested, with the exception of ethambutol.

A previously reported case of mycobacterial lymphadenitis, attributed at that time to an unknown mycobacterium [3], can now be ascribed to *M. bohemicum* on the basis of agreement of phenotypic characteristics (biochemical and lipidic patterns) and genotypic features. The MICs against that isolate were very close to those presented in this case report.

The pathogenic role of *M. bohemicum*, questionable in the case of pulmonary isolation that gave rise to the new-species description [2], therefore seems plausible in both reported cases of pediatric infection.

This new agent adds to the list of mycobacteria responsible for cervical lymphadenitis in childhood. The similarity of its phenotypic traits to those of *M. scrofulaceum* suggests the possibility that at least some cases traditionally attributed to that species could instead be due to *M. bohemicum* and once more

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emphasizes the role of genetic sequencing in the recognition of microorganisms.

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Tularemia Presenting with Ataxia

Infectious, parainfectious, and postinfectious processes are important considerations in the differential diagnosis of cerebellar ataxia. Cerebellar dysfunction can be a prominent or early feature of infection by both viruses and bacteria. Varicella is the classic cause of cerebellar ataxia in childhood [1]. Other organisms or infections that have been associated with cerebellar ataxia include influenza virus [2], leptospirosis, *Mycoplasma pneumoniae* [3], *Coxiella burnetii* (Q fever) [4], Coxsackie virus [5], ECHO virus [6], enterovirus, *Legionella pneumophila* [7], Lyme disease [8], typhus, Rocky Mountain spotted fever, Epstein-Barr virus (infectious mononucleosis) [9], and typhoid fever [10]. In this report, we describe a patient with tularemia, whose clinical presentation was predominantly one of gait ataxia. Cerebellar dysfunction that mainly involved the vermis was present during neurological examination.

A 61-year-old man presented to a hospital in Memphis with a 2-day history of difficulty walking. He also described a 1-week history of progressively more severe fevers and chills and the presence of decreased appetite, myalgias, generalized headache, and photophobia. The patient had not had nausea, vomiting, diarrhea, rash, angina, chest pain, cough, or sore throat in the days before presentation.

The patient's medical problems included coronary artery disease and hypercholesterolemia. His medications were lovastatin, metoprolol, and enteric-coated aspirin. He was retired and lived in the city of Memphis. He did not hunt or have other hobbies or obligations that required him to traverse any of the wooded areas in the tristate area around the city of Memphis. Two weeks before presentation, a friend had given him a piece of deer meat that he had refrigerated before cooking and eating the following day.

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References

1. Wolinsky E. Mycobacterial lymphadenitis in children: a prospective study of 105 nontuberculous cases with long-term follow-up. *Clin Infect Dis* 1995;20: 954–63.
2. Reischl U, Emler S, Horak Z, et al. *Mycobacterium bohemicum* sp. nov., a new slow-growing scotochromogenic mycobacterium. *Int J Syst Bacteriol* 1998;48:1349–55.
3. Tortoli E, Kirschner P, Springer B, et al. Cervical lymphadenitis due to an unusual mycobacterium. *Eur J Clin Microbiol Infect Dis* 1997;16: 308–11.

Vital signs at presentation included blood pressure of 128/61 mm Hg, pulse rate of 72, respiratory rate of 20, and temperature of 39.5°C. During general physical examination, the patient was found to have decreased breath sounds and crackles at the lung bases. The abdomen was soft but mildly distended with mild right upper quadrant and midabdomen tenderness to deep palpation. There was no rebound tenderness or hepatosplenomegaly. There was no pharyngitis, nuchal rigidity, lymphadenopathy, rash, or joint effusions.

Neurological examination revealed that the patient was slightly delirious with decreased eye contact and attention span, incomplete orientation to time, and mildly impaired short- and long-term memory. He exhibited moderately severe staccato dysarthria. There was no nystagmus. Hip and shoulder girdle muscles were weak (4+/5) and mildly tender to palpation. Muscle stretch reflexes were symmetrical, and there were no upper motor neuron signs. Mild ataxia was noted on finger-to-nose, heel-to-shin, and rapid-alternating-movement testing. There was marked truncal ataxia with titubation present in both the seated and standing positions. Gait ataxia was moderately severe and clearly out of proportion to the degree of hip girdle weakness.

Pertinent findings of laboratory studies at admission included the following: aspartate aminotransferase, 210 U/L (normal range, 11–47 U/L); alanine aminotransferase, 60 U/L (7–53 U/L); γ -glutamyl transpeptidase, 51 U/L (11–50 U/L); lipase, 7694 U/L (2.3–50 U/L); amylase, 1474 U/L (35–118 U/L); total leukocytes, $5.8 \times 10^3/\mu\text{L}$ ($3.8 \times 10^3/\mu\text{L}$ to $9.8 \times 10^3/\mu\text{L}$); hematocrit, 38.1% (40.7%–50.3%); platelets, $85 \times 10^3/\mu\text{L}$ ($140 \times 10^3/\mu\text{L}$ to $440 \times 10^3/\mu\text{L}$); and creatine kinase, 1273 U/L (30–220 U/L). The leukocyte profile included 8% lymphocytes, 4% monocytes, no eosinophils, 25% band forms, and 63% neutrophils. A chest radiograph showed an infrahilar density. Analysis of CSF obtained by lumbar puncture revealed a protein concentration of 76 mg/dL (normal range, 15–45 mg/dL), rare lymphocytes, no RBCs, a glucose concentration of 46 mg/dL, and normal results of stains and cultures. CT of the abdomen and MRI of the brain (without contrast medium) were unremarkable. CT of the chest showed small bilateral pleural effusions, consolidation within the left lower lobe of the lung,