IDCases 12 (2018) 117–118

Contents lists available at ScienceDirect

IDCases

journal homepage: www.elsevier.com/locate/idcases

Case illustrated.

# Lyme disease presenting with multiple cranial neuropathies on MRI☆

Pierre-Philippe Piché-Renaud[a], Helen Branson[b], E. Ann Yeh[c], Shaun K. Morris[d,*]

[a] Pediatrics Resident, Sainte-Justine Hospital, University of Montreal, Canada
[b] Division of Radiology, Hospital for Sick Children, University of Toronto, Canada
[c] Division of Neurology, Hospital for Sick Children, University of Toronto, Canada
[d] Division of Infectious Disease, Hospital for Sick Children, University of Toronto, Canada

ARTICLE INFO

*Keywords:*
Lyme disease
Neuroborreliosis
Facial nerve palsy
Cranial neuropathies

ABSTRACT

We present the case of a 10-year old patient from southeastern Ontario with severe bilateral facial palsy. MRI was performed that showed extensive symmetric enhancement of cervical cranial nerve roots and multiple cranial nerves (III, V, VI, VII, VIII, X and XII). Lumbar puncture was performed that revealed pleocytosis and elevated proteins in the cerebrospinal fluid. Serology confirmed the diagnosis of neuroborreliosis. The patient was treated with a 4-week course of IV ceftriaxone, following which he returned to baseline.

A previously healthy 10-year old boy presented in late August with a one-day history of bilateral facial palsy and an otherwise normal neurological examination. Three weeks prior to presentation, he experienced an evanescent rash on his chest with a spike of fever. While the family was aware of the presence black-legged ticks in their local area located in southeastern Ontario, the family and child denied any recent history of tick bite.

MRI of the brain was performed and revealed bilateral symmetric enhancement of cervical cranial nerve roots and multiple cranial nerves including the oculomotor (III), trigeminal (V), abducens (VI), facial (VII), vestibulo-cochlear (VIII), vagus (X) and hypoglossal (XII) (Fig. 1). Cerebrospinal fluid testing revealed a white blood cell count of 133 cells/μL (81% lymphocytes, 13% neutrophils). CSF protein was elevated at 177 mg/dL (normal < 60 mg/dL) and glucose was 43.2 mg/dL (normal 45–79.3 mg/dL) with a serum glucose of 91.9 mg/dL. Bacterial culture plus molecular testing for herpes simplex virus and enterovirus were negative. Blood was also sent for serologic testing for various viral and bacterial infections.

Methylprednisolone (30 mg/kg/day for 5 days) was administered for a presumed diagnosis of a neuroinflammatory disorder and, after no improvement in the facial palsy was seen, was followed by IVIG (2 g/kg). On day 6, initial serology results came back positive for Lyme IgG and IgM by Western blot and a diagnosis of neuroborreliosis or early-disseminated

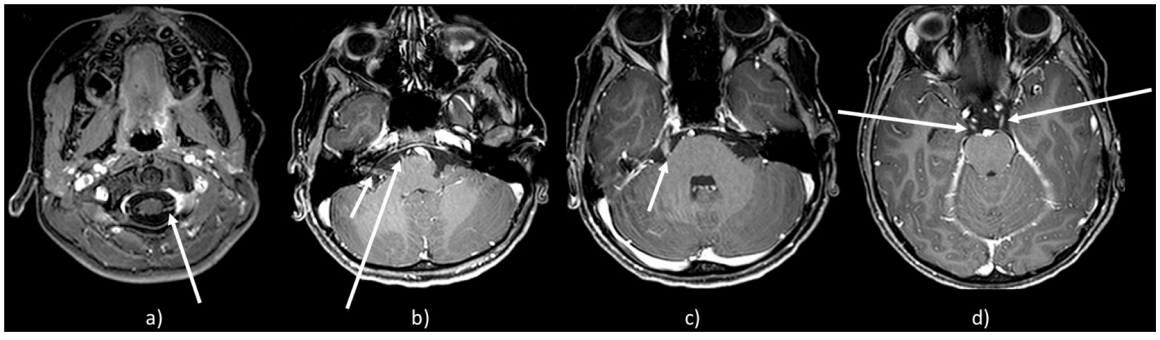


**Fig. 1.** Post-gadolinium axial SPGR images demonstrate multiple enhancing nerves including a) ventral and dorsal cervical nerve roots b) CN VII/VIII in acoustic canal and CN VI (cistern) c) CN V (cistern) d) bilateral CN VI.

☆ Consent for this publication was obtained from the patient and his parents.

* Corresponding author at: Hospital for Sick Children, University of Toronto, 555 University Ave, Toronto, Ontario, M5G1X8, Canada.
*E-mail address:* shaun.morris@sickkids.ca (S.K. Morris).

https://doi.org/10.1016/j.idcr.2018.04.004
Received 30 March 2018; Received in revised form 4 April 2018; Accepted 4 April 2018

Lyme disease with neurological involvement was made. The patient received a 4-week course of IV ceftriaxone (75 mg/kg/day) following which he returned to his baseline condition with no ongoing palsy.

Lyme disease is the most common tick-borne infection in North America and is caused by the spirochete *Borrelia burgdorferi* (1,2). Frequently associated neurological complications of its early disseminated stage include unilateral facial palsy and aseptic meningitis (3,4). Rarely, multiple cranial nerve involvement is seen (5). In this case, multiple cranial nerve involvement on MRI was unexpected, and suggests the need for careful radiological evaluation of the whole neuraxis, including cranial nerves, in cases of CNS lyme disease.

## Declaration of interests

None.

## References

[1] Wormser GP, Dattwyler RJ, Shapiro ED, Halperin JJ, Steere AC, Klempner MS, et al. The clinical assessment, treatment, and prevention of lyme disease, human granulocytic anaplasmosis, and babesiosis: clinical practice guidelines by the Infectious Diseases Society of America. Clin Infect Dis 2006;43(9):1089–134.
[2] Onyett H. Canadian Paediatric Society ID, immunization C. Lyme disease in Canada: focus on children. Paediatr Child Health 2014;19(7):379–88.
[3] Shapiro ED. Lyme disease. N Engl J Med 2014;371(7):684.
[4] Hildenbrand P, Craven DE, Jones R, Nemeskal P. Lyme neuroborreliosis: manifestations of a rapidly emerging zoonosis. AJNR Am J Neuroradiol 2009;30(6):1079–87.
[5] Chaturvedi A, Baker K, Jeanmonod D, Jeanmonod R. Lyme disease presenting with multiple cranial nerve deficits: report of a case. Case Rep Emerg Med 2016;2016. 7218906.