

Ocular manifestations as a result of Lyme disease: a case report

KYLIE-ANN MOYNAGH BMedSci (Hons) (Orthoptics) AND ROWENA McNAMARA MSc DBO(T)

Orthoptic Department, Imperial College Hospitals, London

Abstract

Aim: To present the case of a young adult who developed a cranial nerve palsy as a result of Lyme disease.

Methods: The case of a 22-year-old male medical student who contracted Lyme disease after visiting a deer park in Oxford is discussed. A full orthoptic and ophthalmic examination was performed. The case outlines the difficulty the patient experienced in getting a diagnosis and eventually being appropriately treated.

Results: The patient had an isolated sixth nerve palsy which resulted from being bitten by an infected tick at a deer park which caused Lyme disease to develop. The patient developed erythema migrans, a bull's-eye-shaped rash, on his leg where he had been bitten, but believed this to be ringworm. No other obvious symptoms of Lyme disease developed, which resulted in the patient himself investigating further to reach a diagnosis. Once diagnosed the patient was given a course of the antibiotic ceftriaxone intravenously for 4 weeks. Lyme disease is described as a non-contagious uncommon infection that can spread to the skin, heart, joints and nervous system.

Conclusions: Orthoptists and ophthalmic specialists should be aware that if the diagnosis is uncertain Lyme disease could be the cause.

Key words: Borreliosis, Erythema migrans, Lyme disease, Sixth nerve palsy

Introduction

We report the clinical findings of an isolated sixth nerve palsy in a 22-year-old male that was caused by Lyme disease.

Case report

A 22-year-old male medical student attended his general practitioner complaining of intermittent diplopia having had an accidental minor injury (finger poke) to his right eye 4 days previously. The GP commented that there was no bruising, swelling or echymoses in the right eye, but due to the symptom of diplopia referred him to an eye casualty department.

Correspondence and offprint requests to: Kylie Moynagh, Orthoptic Department Western Eye Hospital, 171 Marylebone Road, London NW1 5QH. e-mail: Kylie-Ann.Moynagh@imperial.nhs.uk

Medical and previous ophthalmic history

The patient's general health was excellent. He had had no previous head injuries, was not affected by headaches or dizzy spells, and had no other known medical problems. He also had no family history of ocular or medical problems, but was allergic to penicillin. The patient had been myopic from a young age.

Ophthalmic examination

Full ophthalmic examination in eye casualty revealed no abnormal signs relating to the ocular trauma, and fundus and media were normal. A review appointment was arranged following an orthoptic assessment. However, the patient re-attended casualty 2 days later complaining that the diplopia had worsened and that it was now affecting near and distance fixation. A repeat ophthalmic examination was normal.

Orthoptic examination

The patient attended the Orthoptic Department 8 days after the initial injury, when there was no change in his case history.

His corrected visual acuities were -0.14 logMAR in the right eye and -0.04 logMAR in the left eye. No compensatory head posture was noted on observation.

Cover testing with glasses revealed minimal esophoria with good recovery movement on near fixation with no diplopia and slight left esotropia with constant horizontal diplopia on distance fixation. Normal binocular single vision was confirmed at near fixation with Bagolini glasses and a Frisby stereo-test.

Ocular movements showed restriction on left abduction measuring 18D of esotropia, compared with 2D on right abduction. This was diagrammatically shown on the Hess chart displayed in (Fig. 1). On prism cover test in primary position the patient measured 6D base-out on near fixation and 7D base-out on distance fixation, and was aware of homonymous diplopia using Worth's Lights in the distance fixation.

Diagnosis

As a result of the ocular findings, the patient was diagnosed with a left partial sixth nerve palsy and the symptom was managed with a 7D base-out Fresnel prism on the left lens of his glasses.

The patient was sent back to casualty for further ophthalmic investigation. Full blood count, kidney function test and glucose were all normal. A CT scan showed no evidence of intra- or extra-axial haemor-

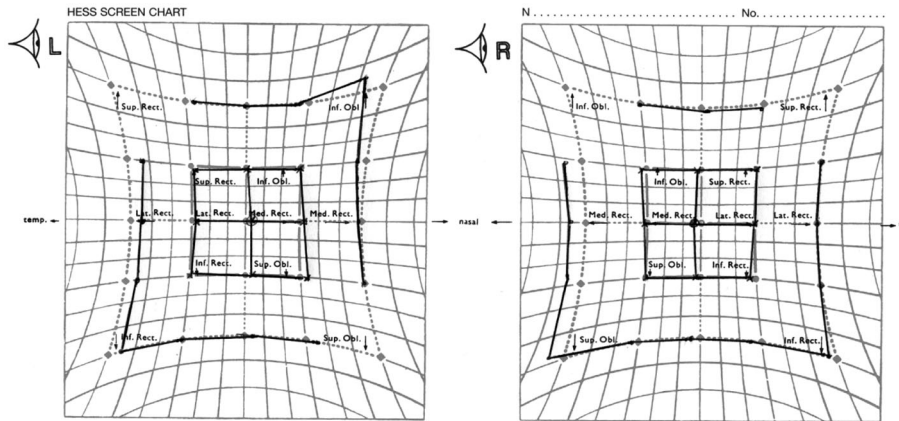


Fig. 1. Hess chart at the first orthoptic examination.

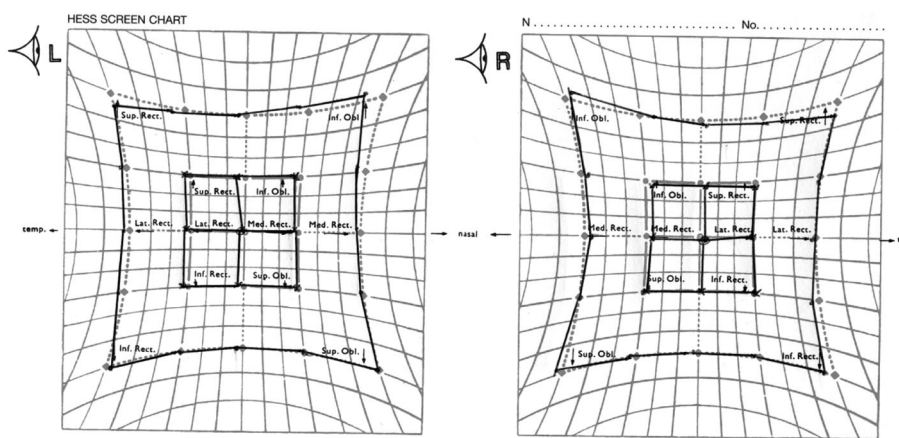


Fig. 2. Hess chart at the second orthoptic examination 6 weeks later.

rhage, normal intracranial appearances and normal appearance of the bony orbits and orbital contents.

The doctor in casualty did ask the patient at this stage if he had been bitten by a tick, but the patient replied that he had not; however, he did mention that he had a rash on his thigh which he thought at the time was ringworm. With the diagnosis of the sixth nerve palsy the patient was advised to attend a general casualty department and seek the opinion of a neurologist. No neurological signs were detected and the patient was reassured by the neurologist that the cranial nerve palsy would resolve spontaneously.

The patient returned to the Orthoptic Department 6 weeks later, with no diplopia and full left abduction. The Hess chart also showed normal eye movements (Fig. 2).

On reflection the patient realised that the rash on his leg had started coincidentally at the time of the eye injury. After his own investigation he discovered that this rash was possibly an erythema migrans, a bull's-eye-shaped rash associated with the early symptoms of Lyme disease (Fig. 3). He looked further into Lyme disease and remembered that he had visited a deer park in Oxford 3 weeks previous to the onset of the diplopia and that the bull's-eye rash had appeared 2 days after the visit.

The patient was now certain of his diagnosis and wanted the relevant investigations to prove it.

He attended his GP again with his suggested diagnosis

but was told he was being paranoid and to stop worrying. This led the patient to discuss his concerns with a rheumatologist, who finally believed his case and got him admitted immediately. Appropriate culturing blood tests and clinical manifestations confirmed it was Lyme disease and he was prescribed intravenous ceftriaxone for 4 weeks.

Discussion

What could have caused this isolated left sixth nerve palsy in this young patient? The differential diagnosis in this case includes the minor blunt injury the patient received to his right eye; however, neither the GP nor the casualty doctor found any visible signs of trauma 4 days after the injury. There was no ocular or medical history and the patient had no history of head trauma.

Other possible causes of sixth nerve palsies in this age group are space-occupying lesions, multiple sclerosis, hypertension and diabetes.¹ These were ruled out by medical and ophthalmic investigations.

None of these diagnoses was consistent with the symptoms the patient was displaying and doctors were unsure about his condition, but advised him that the diplopia would resolve spontaneously. The patient being a medical student with access to medical books and the internet sought his own explanation.



Fig. 3. Erythema migrans.

Lyme disease, also known as Lyme borreliosis, is named after the village of Old Lyme in Connecticut. It is an infection caused by a number of species of the spirochete bacterium *Borrelia*. In the United States the main disease-causing species is *Borrelia burgdorferi*, named after Willy Burgdorfer who identified the bacterium in 1982. In Europe *Borrelia afzeli* and *Borrelia gerinii* also cause the disease. Lyme disease is classed as a zoonosis as it is transmitted to humans from a natural reservoir among rodents and deer by a bite from the infected wood ticks that feed on both hosts. Allen Steere realised in 1978 that the disease is tick-borne.

The NHS² describes Lyme disease as a non-contagious uncommon infection; the Health Agency notes 1000 to 2000 cases in the United Kingdom each year. The NHS warns that the tick population is at its highest, and symptoms can most often occur, between May and September.

The early signs and symptoms of Lyme disease include fever, headache, fatigue, depression, facial weakness and a characteristic bull's-eye-shaped skin rash known as erythema migrans (Fig. 3). Winterkorn³ describes Lyme disease as having three stages: stage 1 features the local erythema migrans that may be accompanied by 'flu-like symptoms; stage 2 is the dissemination of the spirochete to many organs of the body, especially the skin, heart, joints and nervous system; and finally stage 3 can follow a disease-free period and may continue to produce clinical manifestations, for example Lyme arthritis, for years.

Conjunctivitis and photophobia have been described as being first ocular manifestations of Lyme disease, but cranial nerve involvement can occur outlining disturbance to the central nervous system.⁵ Other ocular signs include episcleritis and optic neuritis, and nervous system involvement can affect various cranial nerves unilaterally or bilaterally.⁵ There is no consistent opinion on when these signs occur.

There is difficulty culturing *Borrelia* bacteria in the laboratory, and therefore diagnosing Lyme disease is typically based on the clinical findings and history of exposure to tick-populated areas. However, Winterkorn³ describes *Borrelia burgdorferi* being successfully isolated from blood, synovial and spinal fluid, and also the retina and the vitreous of the eye.

Our patient had no 'flu-like symptoms or facial weakness and discovered through his own investigation

that only 50% of people infected with Lyme borreliosis develop such symptoms. However, he did have the characteristic bull's-eye-shaped rash. This shows that Lyme disease is not straightforward and other manifestations can occur, such as this isolated sixth nerve palsy in our patient. A case was published of a 40-year-old man who developed an isolated sixth nerve palsy at the end of treatment for Lyme disease, but there was some suggestion that it was due to a delayed response to antibiotic treatment or that the patient coincidentally developed the nerve palsy due to other causes.⁴

It was fortunate that our patient was a medical student who persevered with seeking a diagnosis and was eventually treated. If Lyme disease is not diagnosed and therefore left untreated, later misdiagnosed symptoms may involve the joints (arthritis), heart and central nervous system. Fatigue, encephalitis, meningitis, muscle pain and loss of memory can also arise.

The treatment for Lyme disease depends on the stage of diagnosis. Patients who are diagnosed early can be treated with antibiotics including doxycycline. Those diagnosed late are treated with the antibiotic ceftriaxone either orally or intravenously (as given to our patient) for a minimum of 4 weeks. Kalish *et al.*⁶ state that all patients with cranial nerve palsy as a result of Lyme disease should receive antibiotic treatment to prevent later complications, regardless of whether antimicrobial treatment was given.

Mastrianni *et al.*⁴ and MacDonald⁵ highlight the spectrum of ophthalmologic disease in Lyme disease that is still not defined and usually left to be explored only by non-ophthalmologic specialties. Both these articles provide evidence that Lyme disease presents in unusual ways, and Mastrianni *et al.*⁴ describe it as the next greatest imitator after syphilis.

Conclusion

This case report illustrates an unusual presentation of an isolated sixth nerve palsy in a young male as a result of Lyme disease. It was due to the persistence of the patient that treatment was given. A heightened awareness of this condition would have enabled the patient to receive treatment earlier.

Orthoptists and ophthalmic specialists should always consider Lyme disease when the diagnosis is uncertain and should ask the patient relevant questions in their case history. They should also be aware that even if the patient's idiopathic cranial nerve palsy resolves spontaneously, a systemic condition could still be active. Finally, hospitals near woodland areas could provide patient information on Lyme disease.

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