## 10-MINUTE CONSULTATION

# Tick bite and early Lyme borreliosis

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This is part of a series of occasional articles on common problems in primary care. The *BMJ* welcomes contributions from GPs

A 48 year old man removed two ticks attached to his leg while walking in the Scottish Highlands. A week later he develops a rash and, worried about Lyme disease, consults you

Fig 1 | Fully engorged female *Ixodes ricinus* (courtesy of Dr Alan S Bowman, University of Aberdeen)

### What you should cover

*Epidemiology*—Lyme borreliosis is the commonest tick-borne infection in the northern hemisphere. It is relatively uncommon in the UK overall (about 1200 cases in 2009), but marked geographical variation is observed. Risk is highest in rural forested areas and heathland such as the Highlands (incidence 56.35/100 000 in 2009-10[1]), Lake District, and New Forest. Some 15–20% of infections are acquired in Europe or the US. There has been a steady rise in cases diagnosed in the UK over the past decade.[2 3]

*Risk assessment*—Ask about duration of hard bodied (*Ixodes*) tick attachment, with or without engorgement (fig 1). Transmission of pathogenic *Borrelia* species is unlikely if ticks are attached for <24 hours and unengorged.[2 4]

*Clinical features*—Erythema migrans (fig 2) occurs in 90% of symptomatic Lyme borreliosis 2–40 days after exposure.[2-4] Classic erythema migrans is annular with central clearing (differential diagnosis includes ringworm and erythema multiforme), but in the early stages it can be homogeneous and easily confused with cellulitis or insect bite hypersensitivity, and multiple lesions can follow haematogenous spread.[4] Non-specific febrile illness without rash occurs in 7% of early Lyme borreliosis in the US.[3] Neuroborreliosis can occur in early infection and usually presents with meningitis or cranial nerve palsies (such as facial nerve).[2-4] Cardiac involvement (heart block) is extremely rare.[2-4]

### What you should do

- Examine the rash.
- Discuss testing. Erythema migrans is a clinical diagnosis and does not require serological confirmation. Serology is indicated only for diagnostic uncertainty or neurological involvement. Such patients (and any with immunocompromise) should be discussed with an infection specialist. Paired blood samples taken at a four week interval may be required since seroconversion can take several weeks.[2]

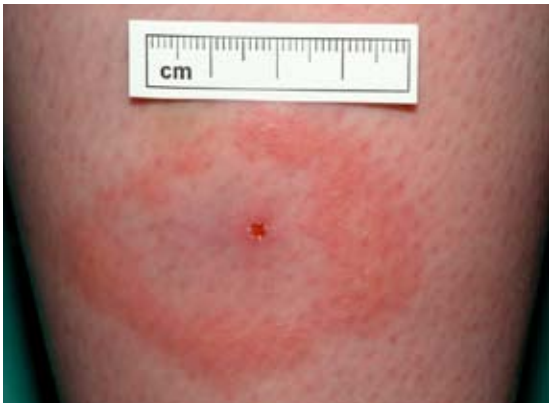


Fig 2 | Classic erythema migrans. Note the central clearing

- Asymptomatic individuals with tick bite should not be tested for Lyme borreliosis (false positives occur because of past resolved infection and cross reactive antibodies), nor should they receive prophylactic treatment (see below).[2] Clinical features should be explained with advice to return if symptoms develop.[2]
- Treat erythema migrans for 14 days (range 14–21 days)[4] with oral doxycycline (100 mg twice daily) or amoxicillin (500 mg three times daily). For pregnant or breastfeeding women or children aged <12 years, the British Infection Association lists alternative treatments.[2] (See the *British National Formulary* for paediatric dosing.[2])
- Explore the patient's concerns. Early Lyme borreliosis has a good clinical outcome.[2-4] Cure was observed in 95% of those treated for erythema migrans in a prospective study.[5] Non-specific symptoms such as fatigue or headache are common in the general population[6] and are no more likely in people treated for Lyme borreliosis at 6-12 months.[5 7]
- Give advice on prevention:
  - Cover skin with long sleeved clothing in forested areas. Insect repellents such as DEET applied to skin and permethrin treatment of clothing are also effective.
  - Check carefully at least daily for ticks, and remove them gently (without twisting) by grasping as close to the skin as possible with tweezers or a commercial tick removal device. Using nail polish, match ends, etc, to remove ticks can increase the risk of transmission by irritating or rupturing the tick, causing injection or release of infected material.[2]
  - Prophylactic treatment of tick bites is rarely indicated in the UK. It may be indicated in special circumstances such as immunocompromise[2] or after exposure to tick bites in specific regions such as parts of New England, USA, where tick infection prevalence is >20% and exposure occurred <72 hours previously.[4] Discuss chemoprophylaxis with an infection specialist.

### PATIENT INFORMATION

- Health Protection Scotland. What do I need to know about ticks and tick borne diseases? www.documents.hps.scot.nhs.uk/giz/general/tick-factsheet-2009-04.pdf
- The Royal Parks, Health Protection Agency. Tick bites and Lyme disease. www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1271256716650
- Wolters Kluwer Health. Patient information: what to do after a tick bite to prevent Lyme disease (beyond the basics). www.uptodate.com/contents/patient-information-what-to-do-after-a-tick-bite-to-prevent-lyme-disease

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Previous articles in this series
- A child with neck swelling (*BMJ* 2012;344:e3171)
- Otitis externa (*BMJ* 2012;344:e3623)
- Blood stained nappy (*BMJ* 2012;344:e3496)
- Blepharitis (*BMJ* 2012;344:e3328)

**USEFUL READING**

- British Infection Association position statement[2]
- Health Protection Agency. Lyme borreliosis/Lyme disease. www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/LymeDisease/
- Health Protection Scotland. Lyme disease. www.hps.scot.nhs.uk/giz/lymedisease.aspx

We thank Dr Alan S Bowman, University of Aberdeen, for providing the image in fig 1.

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Patient consent obtained.

1 Slack GS, Mavin S, Yirrell D, Ho-Yen DO. Is Tayside becoming a Scottish hotspot for Lyme borreliosis? *J R Coll Physicians Edinb* 2011;41:5-8.
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5 Cerar D, Cerar T, Ruzic-Sabljic E, Wormser GP, Strle F. Subjective symptoms after treatment of early Lyme disease. *Am J Med* 2010;123:79-86.
6 McAteer A, Elliott AM, Hannaford PC. Ascertaining the size of the symptom iceberg in a UK-wide community-based survey. *Br J Gen Pract* 2011;61:e1-11.
7 Skogman BH, Croner S, Nordwall M, Eknefelt M, Ernerudh J, Forsberg P. Lyme neuroborreliosis in children: a prospective study of clinical features, prognosis, and outcome. *Pediatr Infect Dis J* 2008;27:1089-94.

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## A PATIENT'S JOURNEY

# Lyme neuroborreliosis

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This is one of a series of occasional articles by patients about their experiences that offer lessons to doctors. The *BMJ* welcomes contributions to the series. Please contact Peter Lapsley (plapsley@bmj.com) for guidance

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- At either end of the tube (*BMJ* 2012;344:e2971)

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- Management of tick bites in primary care http://bit.ly/MAEMBX
- Secret diary of a Lyme worrier 6—pre-Lyme/post-Lyme http://bit.ly/NJIStS

This patient describes her experience of Lyme neuroborreliosis, its eventual diagnosis, and its profound effect on her life

It was during a 280 mile drive to a remote village in the Yorkshire Dales that I was first aware of an ache in my right thigh. Four days later, it had spread to my ankle and buttocks. Next day I was in agony with horrendous back pain. For the next two weeks I barely slept. Lying down was impossible. I got no relief from painkillers. A 24 hour bout of foul diarrhoea was distressing, and I felt ill. I visited a chiropractor, used cold packs, returned home, saw my general practitioner, and had a blood test.

Then on 13 September 2009 I developed double vision. My doctor took one look and asked that I be admitted to Ealing Hospital right away. The back pain had subsided by the time I was assessed by the neurologist, Dr Mark Weatherall. My double vision persisted, however, the left side of my face had become paralysed, and I had become unsteady on my feet. I had numerous further tests and scans. The good news was that there was no sign of a brain tumour, and I was transferred to Charing Cross Hospital for further investigation.

So how did I feel? I had no pain, and the bed rest and care extended by the hospital staff worked wonders. However, the double vision and facial palsy made life rather difficult. I found that I could function if I wore spectacles with the right lens covered over. Eating and drinking improved as the left side of my face began to recover. I was mobile, though unsteady on my feet, and judging the distances between objects was unreliable. The hospital staff were wonderful and the daily routines reassuring. All in all I felt that I was in good hands, and convinced that I would recover.

Dr Weatherall and his team worked through all manner of possibilities. Even I, whose medical knowledge is, at best, scanty, was aware of the unusual nature of my case. I had more scans and a second lumbar puncture and came to realise that, though each member of the team might spend only a few minutes with me, behind the scenes serious professional discussions were going on. Then one day one of the team appeared at my bedside and asked me where I had spent my holidays, and seemed delighted when I told her. And—hey presto—a solution was in sight. Where had I been in the past year? New England, USA, for one; the Dordogne, for another; and the North Yorkshire moors. Take your pick. Could I remember being bitten? Yes—I'd assumed they were mosquito bites. I'd never heard of Lyme disease and had no idea that the gentle deer I'd admired so much on my holidays carried ticks that could do me harm.

I was treated with daily intravenous ceftriaxone for four weeks. Back at Ealing, Professor Bill Lynn, the infectious diseases consultant, monitored the effect of this treatment and adopted me as an outpatient. I wanted to know more about Lyme disease, and he patiently explained. He had spent three years in a hospital in Boston, USA, where the incidence of Lyme disease was higher than in the UK, where only about 40 cases a year were reported. I discovered that the disease had first been identified in 1974 in the town of Lyme, Connecticut; hence its name.

Over the following year things steadily improved. The improvised eye patch was long gone. My brain seemed

### A DOCTOR'S PERSPECTIVE

All professors have an aphorism. When I was a medical student at Cambridge, our professor of medicine, Tim Cox, would tell us, "If you don't think it, you won't diagnose it." In this case, we did not diagnose Lyme disease until we had thought of it, and we thought of it only because some results did not quite fit our working diagnosis of Miller Fisher syndrome.

When I first met Joyce, I already knew that her brain scan was normal; she was areflexic with bilateral abducens nerve palsies and a left lower motor neurone facial palsy. Miller Fisher syndrome seemed plausible, but when the results of cerebrospinal fluid tests came back, the patient had significant lymphocytosis. I reread the literature on Miller Fisher syndrome and found that this was described; but it didn't feel right. Joyce mentions her feeling that serious professional discussions were going on, and she was absolutely right. I was constantly canvassing other people's opinions on the case. It was my colleague Jane Pritchard whose unequivocal rejection of my working diagnosis led us to request further tests, and it was my registrar Una Sherrin who first considered Lyme disease, elicited the relevant history, and sent off samples to the reference laboratory in Southampton. Enzyme linked immunosorbent assay (ELISA) was positive for *Borrelia burgdoferi*, and IgM immunoblotting was positive in both cerebrospinal fluid and serum, confirming the diagnosis. Joyce had been to several places where Lyme disease could be contracted.However, the organism strain and the timing of her symptoms indicated that she had been infected on her holiday in the Dordogne.

Lyme disease is caused by spirochaetes of the *Borrelia* species, transmitted to humans by the bites of *Ixodes* ticks. Different strains cause different patterns of disease, and the European *B burgdorferi garinii* is particularly associated with neurological manifestations, including cranial nerve palsies and meningoradiculitis (Bannworth's syndrome, first described in France in 1922). A total of 953 laboratory confirmed cases were reported to the Health Protection Agency in 2010, with the total numbers of those infected in the UK being thought to lie between 2000 and 3000 cases annually. Cases of Lyme disease have been rising steadily over recent years, probably because of better surveillance, increased awareness of the disease, and increasing infection rates caused by travel to endemic areas such as the Dordogne and eastern Europe (although most cases are contracted in the UK). Whether Lyme disease causes chronic neurological problems is controversial, but it seems likely that it is responsible for some cases of chronic fatigue and encephalopathy.

The lessons of this case are a bit old fashioned: cast your differential diagnosis wide; don't dismiss results that don't fit your diagnosis; many brains are better than one (even a neurologist); and the answer, more often than not, lies in the history. In addition, despite what we are conditioned to think at medical school, Lyme disease in the UK is not simply confined to the New Forest. It extends to other forested areas in the south of the country—such as Exmoor, the South Downs, and Thetford Forest in East Anglia—and further north in the Lake District, Yorkshire moors, and Scottish Highlands. Cases have even been reported in the Home Counties. The importance of taking a comprehensive travel history in complex cases cannot be overstated.

**Mark W Weatherall**

to be trying to get my eyes to focus, and most of the time I tended to forget about it. Bright lights and rapid movements still brought on a feeling of vertigo. I got painful cramps in my legs at night, and an occasional odd sharp sting somewhere in my body, rather like a ping made by letting go of a rubber band. I fancifully thought that it was another nerve reconnecting. Both consultants continued to monitor my progress at intervals. The antibiotics sparked off serious diarrhoea, which lasted about six months. I began to feel my age. Balance was uncertain. Walking became slower, and I tired quickly. I couldn't raise myself from a sitting position in the bath, and stairs presented a challenge.

We moved house. An excellent retirement village gave me a fresh start. I continue to get a lot of painful cramp, especially at night, and my leg muscles look different. I still need to wear sunglasses in bright light. I was interested to discover that I can no longer swim: I can perform leg and arm actions separately, but can't coordinate them. Lack of exercise has led to weight increase. Otherwise I feel fine and enjoy a lively social life. We take holidays here and abroad as before. I am not quite as I was before Lyme disease, but then I am two years older. When my friends commiserate and say what a terrible time I've had, I tell them that it was quite an adventure which, in a strange way, I enjoyed, and that I'd met lots of dedicated people who restored my faith, if that were needed, in our NHS. I am a fortunate 80 year old woman.

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## ANSWERS TO ENDGAMES, p 48 For long answers go to the Education channel on bmj.com

### PICTURE QUIZ
### An acquired source of seizures

1 A solitary cystic lesion is seen at the junction of the right frontal and parietal lobes, with an eccentric nodule within.
2 Adult onset seizures in a high risk patient with an intracerebral cystic lesion are suggestive of neurocysticercosis (parasite cysts in the brain).
3 A diagnosis of neurocysticercosis relies on establishing the risk of exposure and is supported by imaging techniques, immunological tests, and histological examination, although this investigation is rarely needed.
4 This patient needs antiepileptic drugs and steroids to control symptoms and complications. Antiparasitic agents are not usually indicated in patients with a solitary lesion.

### CASE REPORT
### A painful swollen finger

1 Flexor sheath infection, also known as infectious flexor tenosynovitis or pyogenic tenosynovitis
2 Puncture wound and spread of infection proximally through the flexor sheath.
3 Skin commensals *Staphylococcus aureus* and group A *Streptococcus*.
4 Wound swab, removal of rings from the fingers, intravenous antibiotics, tetanus booster, radiographs, high hand elevation, and urgent referral to hand surgery unit for debridement.
5 Stiffness, contracture, and necrosis necessitating amputation are the main complications in flexor sheath infection.

### STATISTICAL QUESTION
### Observational study designs

Nested case-control study (answer *d*) best describes the study design used.