CASE REPORT

Neuroborreliosis: the Guillain-Barré mimicker

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SUMMARY

A 34-year-old woman presented to the medical admissions unit with progressive ascending weakness of her limbs and areflexia. Diagnosis of Guillain-Barré syndrome was suspected and she was started on intravenous immunoglobulins. Owing to a poor initial response, further exploratory history revealed travel to the New Forest and a possible tick bite; subsequent investigations confirmed positive serology for antibodies against *Borrelia*. The patient's weakness improved with intravenous ceftriaxone for neuroborreliosis, a manifestation of Lyme disease. With inpatient neurorehabilitation, she made good recovery and was able to mobilise with a stick from being completely bed bound 6 weeks after completion of her antibiotics.

BACKGROUND

This case highlights the importance of bearing differential diagnoses in mind when working up patients with neurological conditions. This patient initially presented with suspected Guillain-Barré syndrome (GBS), but further probing and investigation identified a different diagnosis: neuroborreliosis. Neuroborreliosis is a manifestation of Lyme disease, which requires treatment with antibiotics. As the incidence of neuroborreliosis is increasing in the UK and known to mimic many neurological conditions, it is an important differential to bear in mind and should be investigated as part of the initial work up as correct treatment can be started promptly.

CASE PRESENTATION

A 34-year-old woman, otherwise fit and well, presented to the medical admissions unit with a 4-day history of headache, and 'pins and needles' in her hands and legs. There was no evidence of meningism, no rash, no photophobia and no neck stiffness. Initially, on examination, she had a normal gait and a normal cranial nerve examination. Although her upper and lower limb power was 5/5, she was found to be hyporeflexic at her knees and ankles bilaterally. There were downgoing plantars, and there was a slight reduction in light touch and pinprick sensation in her hands, and up to her knees bilaterally. Over the next few days, there was symmetrical ascending progression of weakness, and her lower and upper limb power reduced to 2 of 5 (MRC grade), with bilateral lower limb areflexia. She consequently became bed bound. She also reported of severe sciatica-type pain bilaterally. She had a lumbar puncture and cerebrospinal fluid (CSF) showed a white cell count of 0, a normal protein count of 0.23 (0.10–0.50), normal glucose of 3.7 (2.8–3.9) and normal lactate of 1.8 (1.1–2.4). This was diagnostically unhelpful. Nerve conduction studies conducted 10 days after admission confirmed GBS. As the studies showed active denervation it was thought that recovery could take up to 6 months. Spirometry was advised to monitor respiratory function. The patient was started on intravenous immunoglobulins for 5 days, but there was no improvement noted in her symptoms. She reported further deterioration of her symptoms with development of left-sided lower motor neurone facial weakness and subsequent paralysis. She was reviewed again by the neurology team, who established that a few weeks prior to her symptoms, she had been in the New Forest in the vicinity of Southampton where she had noted a tick bite on her right shin, and described it as a red blister with a central bite and a surrounding red ring. The patient had not previously been questioned about this, and this was new information established after the initial diagnosis of Guillain Barré. Serum *Borrelia* antibody tests were carried out at this point, as Lyme disease could be a contender for her presentation.

INVESTIGATIONS

The patient's initial blood tests including inflammatory markers were normal, along with her initial observations. A CT of the head on admission was normal, and a subsequent MRI of the spine showed a small disc bulge at L5/S1, but no nerve root compression was demonstrated.

Two weeks after initial presentation, we were notified about the presence of IgG oligoclonal bands in the CSF, which is indicative of a systemic inflammatory response such as Guillain-Barré or a systemic infection, however, the initial CSF findings had been unremarkable, which can also be the case in early GBS.

Serial spirometry was conducted during the progressive stage of the patient's symptoms, and this remained stable throughout.

Nerve conduction studies supported GBS. They demonstrated slow nerve conduction velocities (ulnar nerve was 42 m/s with proximal conduction block and common peroneal nerve speed was 32 m/s with proximal conduction block) and delayed F-waves, suggestive of a demyelinating neuropathy. It was also noted that the patient had evidence of active denervation indicating poor prognosis and delay in recovery of up to 6 months.

After discussion with the neurologist, serum *Borrelia* antibodies tests were performed, 10 days after initial admission, and results were obtained after a further 2 weeks. Both IgG and IgM antibodies to *Borrelia* were positive, which was consistent with Lyme disease at some time, most likely a recent onset. Treatment was started for

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neuroborreliosis. However, unfortunately, the initial CSF sample was not tested for *Borrelia*-specific antibody production and a repeat lumbar puncture was not carried out as a diagnosis was already made.

DIFFERENTIAL DIAGNOSIS

The initial clinical presentation was typical of GBS. Headache and sciatica-like radicular pain are uncommon in GBS but this does not exclude the diagnosis. Differential diagnosis includes infective or vasculitic polyradiculopathies. However, together with the recent visit to the New Forest, a tick bite, and positive IgM and IgG antibodies for *Borrelia*, Lyme disease mimicking GBS was made.

The important difference in this case would be the use of antibiotics to treat the Lyme disease, which should help in maximising recovery and prevent further complications.

TREATMENT

The patient was initially given 5 days of intravenous immunoglobulin for presumed GBS. This treatment did not make the expected improvement in her condition. On discovering her antibody tests were positive for *Borrelia* she was given 14 days of intravenous ceftriaxone, which is the recommended treatment for Lyme disease with neurological involvement.[1–3]

She also received intensive neurorehabilitation as it was felt that her symptoms could take up to 6 months to recover.

OUTCOME AND FOLLOW-UP

Following completion of 2 weeks of intravenous antibiotics, the patient was discharged to neurorehabilitation. Six weeks following the completion of her antibiotics, from being completely bed bound with quadreparesis, she was mobilising with a stick. Her facial weakness had resolved and her grip had improved. She continued to report 'pins and needles' in her fingers and feet but was slowly improving. She was offered regular physiotherapy to help regain full function.

DISCUSSION

Lyme disease is a leading tick-borne bacterial infection found across the world, with an increasing incidence in the UK, from 0.50/100 000 in 2002 to 1.64/100 000 in 2010.[4] The most common culprits in the UK are *Borrelia burgdorferi, Borrelia afzelli* and *Borrelia garinii*. Recognised locations that constitute a higher risk include the New Forest in Southampton, where our patient had been walking and was exposed to a tick bite. Lyme disease, if untreated, can result in early localised signs such as a target-like erythaema migrans rash, which is reported by almost 90% of patients. Additionally, 50–70% of people report a tick bite leading up to the rash.[4 5] Early disseminated Lyme disease can present weeks or months after the initial tick bite and can cause neurological involvement such as mononeuritis multiplex, radiculopathies and cranical nerve palsies (commonly facial palsy), collectively known as neuroborreliosis. It can also cause cardiac manifestations including myocarditis, cardiac conduction disturbances and cardiomyopathy. Late Lyme disease can present several years after the initial infection and can cause joint, skin and neurological complications.[4]

In the UK, neuroborreliosis is the most common complication of Lyme disease. Most commonly, it can present with acute meningoencephalomyelitis, or acute cranial neuropathy and radiculopathy, and rarely can present with a Guillain-Barré-like picture.[5] Lyme disease should be considered in anyone who presents with symptoms and signs, as above, suggestive of the early disseminated phase of the infection, and these patients should be questioned about possible tick bites in the preceding few months. Blood and CSF samples from these patients should be sent for *Borrelia*-specific antibodies and testing by PCR. Treatment of early localised Lyme disease can be carried out in the community with a course of oral doxycycline. However, severe early disseminated or late Lyme disease warrants intravenous antibiotics, such as ceftriaxone, for 2–3 weeks.[1–4]

GBS is an autoimmune attack on nerve roots and peripheral nerves, often triggered by infections such as *Campylobacter jejuni*. The acute phase can be life-threatening due to respiratory muscle paralysis and cardiac arrhythmias. Beyond this, it can often be associated with a full recovery, though in some patients the recovery process may take up to 2 years, requiring an intense neurorehabilitation programme.[6]

The main question at this point is whether we are looking at a patient who developed GBS following an immune response triggered by Lyme disease, or if this is a case of Lyme disease mimicking GBS, or, indeed, if the two coexisted. There are very few cases in the literature about neuroborreliosis and Lyme disease. There was a case report from 1982 about a 28-year-old man who presented with progressive peripheral neuropathy in the context of Lyme disease and the same question arose with regard to the aetiology.[7] Interestingly, there is a case of a 4-year-old boy who suffered from suspected GBS with serum anti-*Borrelia* antibody titres. He was started on intravenous ceftriaxone and also given intravenous immunoglobulin, and complete recovery was noted after treatment.[8] In this case, it is uncertain, however, as to which treatment helped full recovery, the immunoglobulin or the antibiotics. In addition, there was a 19-year-old girl who presented with suspected GBS with positive serum titres for anti-*Borrelia* antibodies, where they observed a clinical stabilisation of her weakness with intravenous ceftriaxone allowing neurorehabilitation to take place.[9] In the latter cases, it would appear that neuroborreliosis was mimicking Guillain-Barré. However, another case has been reported whereby a patient was treated for neuroborreliosis but then went on to develop Guillain-Barré despite antibiotic therapy, as confirmed by clinical findings and nerve conduction studies.[10] This is consistent with Guillain-Barré being triggered by Lyme disease.

Initially, our patient presented with signs and symptoms that progressed in the manner of Guillain-Barré; the ascending paralysis, areflexia and facial palsy. An initial lumbar puncture showed normal CSF findings apart from the oligoclonal bands. We would have expected an elevated protein count in Guillain-Barré, but in early illness normal CSF protein can be found. Our working diagnosis was supported by the nerve conduction studies showing demyelinating neuropathy but we also saw early denervation, which was unusual for Guillain-Barré. With the accompanying headache, radicular pain (both of which had initially not been considered as being important, but which later became more relevant) and a poor response to the intravenous immunoglobulin, we questioned the validity of Guillain-Barré and explored alternative diagnoses. Lyme disease became a more likely differential diagnosis, and subsequent questioning of the patient divulged a likely exposure.

The IgM and IgG antibodies against *Borrelia* were positive, suggesting early disseminated Lyme disease, and we gave the patient a 2-week course of intravenous ceftriaxone, in addition to the previous intravenous immunoglobulin she had received for suspected GBS. In our patient, we also noted good recovery 6 weeks after having taken intravenous antibiotics, when previously, with suspected Guillain-Barré alone, it was thought it could take up to 6 months. Important to note is that some

Guillain Barré variants, which have denervation, may have poorer prognosis.

On balance, we feel that this is a case of Lyme disease mimicking Guillain-Barré rather than Guillain-Barré caused by Lyme disease. As there was no response to the intravenous immunoglobulin, we do not believe there was co-existing Guillain-Barré and Lyme disease.

Lyme disease is known as a great mimicker and it is therefore a vital diagnosis to bear in mind, especially as it can manifest with such disabling complications. The incidence of neuroborreliosis is increasing in the UK, but there is often a delay in requesting serological tests, and therefore treatment, for this condition.[11] Increased education and awareness can help with prevention of hospitalisation and complications associated with disseminated and late Lyme disease, and, ultimately, reduce patient distress. In patients with suspected demyelination and Guillain-Barré-like symptoms, consideration of Lyme disease can ensure quicker treatment, stabilise weakness and help maximise recovery.

Learning points

- Neuroborreliosis can mimic Guillain-Barré syndrome and also trigger Guillain-Barré, and therefore must be considered a key differential in any suspected case.
- Lyme disease is increasing in the UK, therefore it is essential to bear its wide manifestations in mind.
- Neuroborreliosis is the commonest complication of Lyme disease in the UK and requires treatment with intravenous antibiotics.

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