NEUROLOGICAL COMPLICATIONS OF LYME DISEASE; A CASE REPORT

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CASE HISTORY

An 81 year old caucasian lady, presented with complaints of severe back pain after returning from her holiday in Yorkshire. The Pain initially developed over her right thigh and then progressed down to her right ankle, buttocks and finally to her back. She was admitted as the pain became excruciating and worsened on lying flat. The patient did not get any relief even after having analgesics. The Pain gradually subsided in 14 days of post admission on treatment with analgesics and antibiotics and was discharged.

The following month she was brought to the accident and emergency department and was diagnosed with urinary tract infection which was treated with ciprofloxacin. Two days later she developed double vision accompanied with headache which progressed to left sided facial weakness.

Her past medical history revealed an attack of Bell’s palsy 5 years prior to this episode for which she was treated with Acyclovir and Steroids.

On Neurological assessment her fundoscopy was normal and visual field intact. On further examination she had bilateral 6th nerve palsy which was more prominent on right side and lower motor neuron lesion of 7th nerve over the left. Both upper and lower limb examination revealed that the tone and power were normal. But when reflexes were tested on reinforcement the triceps showed decreased reflex activity where as biceps exhibited an increased reflex. Similarly reflex activities were tested on the lower limbs which exhibited decreased ankle reflex and flexor plantar response. The sensory perception was tested using light touch, pain and prick which was found to be normal. Vibration sensation was decreased bilaterally over both knees. Gait was normal but mildly ataxic. Romberg’s sign was negative.

On Investigations:- MRI showed periventricular white matter, T2 hyperintensity lesion secondary to small vessel disease, ischemic changes in pons and midbrain.

Cerebrospinal fluid showed lymphocyte: 48, protein .92g/dl, glucose 3.14 mmol/l. no evidence of atypical or malignant cells.

Nerve conduction test was abnormal and both upper and lower limb showed symmetrically decreased amplitude with preserved motor conduction.
parameters. Findings were consistent with Diffuse acquired predominantly symmetrical sensory axonal polyneuropathy.

The blood test showed high lymphocyte count (21.8%), but at the same time urea and electrolyte, liver function test, inflammatory and tumor markers were within normal limits. ANA, ds DNA, ENA and ANCA were negative. C6 ELISA was reactive for lyme’s disease.

Serology done on serum and CSF showed positive IgM immunoblotting that was strongly suggestive of recently acquired CNS lyme disease.

**Treatment**: - was commenced with I.V. Ceftriaxone 2gms OD for 4 weeks and analgesics.

**Progression**: - she had a good recovery from her Lyme meningitis and has no significant residual neurological problems.

**DISCUSSION ON LYME DISEASE**

Lyme disease is caused by a tick borne spirochaete named Borrelia Burgdorferi (fig. 1). In Europe 2 additional species are encountered B.afzelii and B.garinii. . The infectious spirochetes are transmitted to humans through the bite of certain Ixodes species that feed on variety of large mammals particularly deer. The disease is caused by the infection and the body's immune response to infection.

![Fig 1: Borrelia Burgdorferi](image)

**EPIDEMIOLOGY**

In the UK, areas where infection is acquired include:
- The New Forest
- The South Downs
- Parts of Wiltshire and Berkshire

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About 20% of confirmed cases are reported to have been acquired abroad:

- The USA
- France
- Germany
- Austria
- Scandinavia
- Eastern Europe

PRESENTATION:

It should be remembered that some infected people will have no symptoms many patients do not remember being bitten by the often innocuous tick. Progression may be arrested at any stage and presentation depends on the stage of disease at the time of presentation.

1) *Early Lyme Disease*

- The characteristic manifestation is erythema migrans(fig.2):
  - A circular rash at the site of the infectious tick attachment that radiates from the bite, within 2-40 days.
It may be the only manifestation of disease in one third of patients.
- Pyrexia, arthritis, musculoskeletal symptoms and local lymphadenopathy may occur in about two thirds of patients but one third of patients will develop no further symptoms.

You may not even notice you have been bitten. A rash that looks like a “bull’s-eye” target may appear after the tick bite. Late manifestations may even occur months to years later

2) Disseminated Lyme disease occurs in weeks to months later, with:
- Flu-like illness, oligoarthralgia myalgia, multiple erythema migrans and sometimes systemic upset
- Intermittent inflammatory arthritis:
- Central nervous system disorders (15%):
  - These include facial (and other cranial nerve) palsies. These are the most common neurological manifestations in Europe and the USA.
  - Meningism and meningitis may occur alone or with other neurological manifestations. It is usually at the mild end of the spectrum but can be more severe.
  - Mild encephalitis producing malaise and fatigue.
  - Peripheral mononeuritis
  - Lymphocytic meningoradiculitis (or Bannwarth's syndrome
- Cardiovascular problems (10%):
  - This usually presents with syncopal episodes associated with fever.
  - Manifestations include transient atrioventricular block, myocarditis, or chronic dilated cardiomyopathy.
- Occasionally hepatitis, orchitis, uveitis and panophthalmitis.

3) Late manifestations of Lyme disease
- Untreated or inadequately treated Lyme disease can cause late disseminated manifestations weeks to months after infection. These late manifestations typically include prolonged arthritis, polyneuropathy, encephalopathy and symptoms consistent with fibromyalgia.
  - Chronic lyme arthritis - a chronic erosive arthropathy typically involving the knees.
  - Chronic neurological syndromes. Generally these appear to be more common in Europe. These include chronic
neuropathies (usually with paraesthesia and occasionally with pain but not with motor deficit). They may even present as chronic fatigue syndromes, spastic paraparesis or depression.

INVESTIGATIONS

The first step in patients with symptoms consistent with Lyme disease is to obtain an antibody titer. ELISA, PCR, CSF, Blood test, Immunoblot (Western blot) technique are more specific but technically demanding. The antibody titre can either be a total Lyme titer or separate immunoglobulin G (IgG) and immunoglobulin M (IgM) titers.

TREATMENT

Doxycycline 200mg od for 14 days. Or amoxicillin 500mg tds for 14 days. Disseminated disease and arthritis needs minimum of 30 day treatment with doxycycline or amoxiclycine. Neuro Borreliosis is treated with parentral beta lactam antibiotics for 3-4 weeks. Ceftriaxone 2gm iv daily. Cefotaxime 2gm tds or benzyl penicillin 3gm qdid.

PREVENTION

Protective clothing and insect repellents should be used in tick infested areas. Tick collars for pets check skin often when in risky areas. A single dose of doxycycline 200mg po given within 72 hrs of a bite is effective prophylaxis. A recombinant vaccine OspA in adjuvant (3 injections of 30 microgm at 0,1, and 2 or 12 months) offers upto 76 % protection.

REMOVING TICK

Suffocate tick with petroleum jelly then remove by grasping close to mouth parts and twisting off then clean skin.

REFERENCES

[4] Lawrence C, Lipton RB, Lowy FD, Coyle PK, Seronegative chronic