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Tick-borne Encephalitis Identified in Ireland: Diagnosis and Outcome

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Abstract

Presentation

Tick borne encephalitis (TBE) is not endemic in Ireland and diagnostic tests are seldom requested. We describe the first notified case in Ireland. A 50-year-old female returned from Lithuania and presented with fever and new neurologic signs.

Diagnosis

TBE was diagnosed by detection of TBE virus specific antibodies in serum and cerebrospinal fluid (CSF).

Treatment

The patient was managed with observation and supportive care consisting of intravenous fluids and analgesia.

Discussion

The case highlights the importance of awareness of TBE among physicians and travellers to guide appropriate testing and vaccination. TBE is being recognised in non-endemic countries posing an emerging risk to public health.

Introduction

TBE virus (TBEV) is of the *Flaviviridae* family.[1] It is transmitted by the bite of an infected tick, from the *Ixodes* species and is an important pathogen of the central nervous system (CNS) in many parts of Europe and Asia.[2] Infection and subsequent inflammation of the CNS can result in permanent neurologic sequelae and death.[3]

Case Report

A 50-year-old female presented with a seven-day history of generalised headache and poor appetite. On day 12 of her illness she developed fever and rigors with associated dizziness, unsteadiness, and speech difficulty. She had no past medical history and took no regular medications. She had travelled to Lithuania four weeks prior to presentation. While abroad she went mushroom picking in a forest and on the same day noted a large tick fastened to her skin above the xiphisternum. The tick's body was removed, but the head remained in situ, falling off after seven days.

On admission her temperature was 39°C. Relevant neurological findings included a bilateral resting and intention tremor, dysarthria, left lateral nystagmus, reduced power (4/5) in the upper limbs and an ataxic tandem gait. The remainder of the physical examination and vital signs were normal.

Laboratory investigations demonstrated a normal c-reactive protein (CRP) <1mg/l, mild hyponatraemia 133mmol/l, normal renal and liver profiles, and a white cell count (WCC) of 8.4 $x10^9$/L with lymphopenia 0.8 $x10^9$/L. A lumbar puncture revealed a WCC of 17 cells/µL (differential; 64% mononuclear, 36% polymorph cells), with CSF glucose 3.37mmol/l (range 2.22-3.89mmol/l) and protein 73mg/dl (range 15-45 mg/dl). An MRI brain suggested encephalitis (Figure 1).

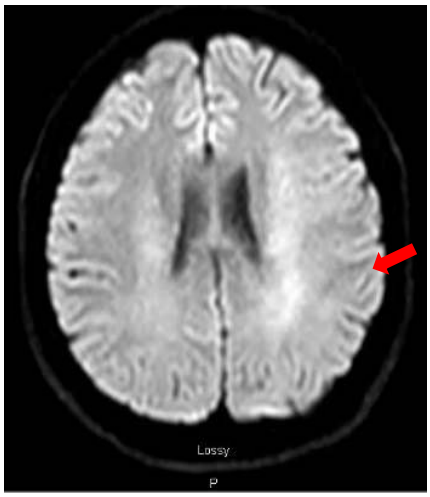

Figure 1: *MRI Brain - Indeterminate restricted diffusion involving the left centrum semiovale extending to the left anterior temporal lobe (indicated by arrow). Appearances would be consistent with encephalitis.*

Based on the patient's travel history and disease course, detection of TBEV antibodies was performed. Samples were sent to the Rare and Imported Pathogens Laboratory, Porton Down, England. Both anti-TBEV IgM and IgG antibodies were strongly positive in serum and CSF. Anti-TBEV IgG titres were quantitated in serum and CSF using an immunofluorescence assay which yielded results of 1:1,000 and 1: 320, respectively. Furthermore, the TBEV viral index in paired CSF: serum samples were markedly raised at 33 (normal < 3), confirming the diagnosis of TBE. The patient had not been vaccinated against TBEV.

Antimicrobials were discontinued and supportive therapy continued consisting of intravenous fluids and analgesia. The patient was discharged after 8 days. Her neurological signs had improved with normalisation of gait and muscle power, some tremor remained. Review at three months revealed some fatigue without any residual neurologic sequalae.

Discussion

TBE is a notifiable disease in European Union (EU) since 2012.[4] The highest notification rates in Lithuania, Slovenia and the Czech Republic, but cases are now being recognised in Switzerland, Scandinavia and England.[5, 6] Information on TBE is lacking for some non-EU countries, including Ukraine, where annual reported cases are far below that of neighbouring countries.[7] TBE exhibits a spring-summer seasonal pattern. The risk of acquisition is highest with outdoor activities, particularly in wooded areas. Transmission occurs within minutes of the bite with an incubation of 7-14 days. The illness has a biphasic pattern, a first phase of non-specific symptoms or influenza-like illness followed by the second phase 1-20 days later involving fever and signs of CNS inflammation such as meningitis, meningo-encephalitis, encephalo-radiculitis, or paralysis.[8]

A confirmed case is defined by symptoms of CNS inflammation with one of the following five laboratory criteria; TBE serum IgM and IgG antibodies, TBE IgM antibodies in CSF, sero-conversion or four-fold increase of TBE antibodies in paired serum samples, detection of TBE viral nucleic acid in a clinical specimen or TBEV from clinical specimen.[9]

This case highlights the importance of awareness of TBE among physicians and to consider it in travellers or refugees. A detailed travel history is needed to direct testing. There is no specific antiviral therapy.[3] If suspected; early discussion with microbiology is necessary for TBEV antibody testing, this allows for overseas analysis and avoid delayed diagnosis. Prevention is key with a need to advise travellers on measures to avoid tick bites as well as consider vaccination.[10] The current increase in incidence of TBE in endemic regions and in countries not previously known to have TBE signals an emerging public health risk.[6]

Declaration of Conflicts of Interest:
None.

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