

Always consider the travel history: West Nile virus infection presenting in rural Ireland

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Introduction

Confusion is a very common hospital presentation, as are headaches. When combined, and accompanied by pyrexia, clinicians often consider CNS infections as the potential aetiology. This case discusses such a scenario which took time and deliberation to reach the final diagnosis of West Nile virus encephalitis.

Case report

A 54-year-old man presented to hospital in Ireland with a 48-hour history of headaches, confusion, vomiting, general malaise and fever of 38.5°C. His medical history included gastro-oesophageal reflux disease for which he was taking famotidine, and he had recently completed isoniazid therapy for latent tuberculosis. None of his contacts were unwell. He lived in California and had arrived in Ireland 1 week earlier. Full clinical examination on presentation was normal apart from disorientation to time, place and person, with a Glasgow Coma Scale score of 14/15.

Routine blood tests were all normal, as well as thyroid function tests, haematinics, blood gas, human immunodeficiency virus and syphilis serology, blood cultures, urine culture and SARS COV-2 polymerase chain reaction. Computed tomography scan of the brain was normal, and magnetic resonance imaging of the brain with contrast showed a small number of punctate T2/FLAIR hyperintensities in the deep white matter of the frontal lobes bilaterally that were within normal limits for his age. Lumbar puncture on admission revealed a CSF protein level of 625 mg/litre, glucose level of 3.5 mmol/litre (paired serum glucose level of 5 mmol/litre), white cell count of 147/cm² (99% lymphocytes), and no organisms were seen on microscopy and culture. Polymerase chain reaction of CSF for genetic material of herpes simplex virus 1 and 2, varicella zoster virus, *Escherichia coli*, *Haemophilus influenzae*, *Listeria monocytogenes*, *Neisseria meningitidis*, *Streptococcus pneumoniae*, *Strep. agalactiae*, enterovirus, human parechovirus and *Cryptococcus neoformans* were all negative. Empiric cover for bacterial meningitis and viral encephalitis was started on the day of admission. Lumbar puncture was repeated on day 7 of treatment with largely unchanged CSF results and another negative polymerase chain reaction. Given the patient's history of latent tuberculosis, tests for acid-fast bacilli and tuberculosis culture were performed on the day 7 CSF specimen, which were negative. An electroencephalogram revealed left-sided low amplitude slowing throughout with additional brief bursts of generalised delta, but no periodic discharges or epileptiform abnormalities. The findings were felt to be in keeping with an encephalopathy.

The patient made a complete recovery of symptoms within 24 hours of commencing the empiric treatment, but completed 7 days of ceftriaxone and 14 days of aciclovir. However, the clinicians had yet to elucidate the cause of this illness that was behaving like a viral encephalitis. At this point, the case was re-examined in its entirety and colleagues in microbiology, neurology and infectious diseases were consulted. This led to a serum sample being sent for West Nile virus serology, given the patient's usual residence in California. Serum samples were also sent for dengue fever IgM, tick-borne encephalitis IgM, yellow fever IgM and Japanese encephalitis virus IgM, all of which were negative.

The patient had returned home to California, completely asymptomatic, by the time his West Nile virus serology results were received from the National Virus Reference Laboratory. These found a positive serum IgM and negative IgG, indicating acute West Nile virus infection causing encephalitis as the probable cause as per the Official Journal of the European Union diagnostic criteria (2018). Unfortunately, it was not possible to test for West Nile virus-specific antibodies or DNA in the CSF (usual confirmatory investigation) as the patient had flown home.

How to cite this article:

Maher L, Aziz R. Always consider the travel history: West Nile virus infection presenting in rural Ireland. Br J Hosp Med. 2023. <https://doi.org/10.12968/hmed.2022.0314>

Learning points

- Take a careful and comprehensive travel history in all patients.
- Consider diseases endemic to a patient's country of origin.
- Do not be afraid to expand the differential diagnosis in a challenging case by consulting other subspecialties.
- Neuroinvasive disease caused by West Nile virus infection carries considerable mortality risk and a high risk of long-term troubling neurological symptoms.

Discussion

This case highlights the importance of carefully considering the patient's travel history and liaising with different specialties when faced with a challenging case. Most cases of West Nile virus infection in humans are subclinical, with a minority of patients developing symptoms ranging from fever and myalgia to meningoencephalitis and death. Increased morbidity and mortality are associated with extremes of age and an immunocompromised state (Colpitts et al, 2012). West Nile virus is transmitted to humans via a mosquito bite; mosquitoes acquire the virus via eating an infected bird (Hayes et al, 2005). This is not the first case of West Nile virus infection detected in Ireland, but it is very rare, with the last case reported 10 years ago (Health Protection Surveillance Centre, 2013). However, West Nile virus is endemic throughout much of the USA, with hundreds to thousands of cases of neuroinvasive disease recorded each year (Petersen, 2019). West Nile virus, a subtype of arbovirus, is widely distributed throughout much of the world (Chancey et al, 2015), with 3829 cases reported in Europe between 2010 and 2018 (Young et al, 2021). From a retrospective review of the literature, it seems that antiviral agents are unnecessary in treatment of this infection, with the mainstay being conservative management (Popescu et al, 2020). Neuroinvasive disease from West Nile virus infection can have long-term sequelae; Klee et al (2004) found only 37% of patients made a full recovery within 1 year of infection. This patient was very fortunate to make a full recovery so quickly.

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