Glioblastoma: a mimic of NMDA receptor encephalitis

Roshni Mansfield,[1,2] Wisdom Yong,[3] Krishna Shah,[2] Sachid Anand Trip[3,4]

[1]Faculty of Medicine, Imperial College London, London, UK
[2]Ambulatory Emergency Care Unit, Northwick Park Hospital, Harrow, London, UK
[3]Department of Neurology, Northwick Park Hospital, Harrow, UK
[4]Department of Neuroinflammation, Queen Square MS Centre, University College London Hospitals NHS Foundation Trust, National Hospital for Neurology and Neurosurgery, London, UK

Correspondence to
Dr Roshni Mansfield;
roshni.mansfield@nhs.net

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DESCRIPTION

An Indian woman in her 40s without any medical or psychiatric history presented with a seizure. She had become uncharacteristically quiet before suddenly cackling and banging her hands and feet repeatedly. She developed facial and upper limb dystonic posturing including the extension of one arm. The acute onset, short duration, loss of responsiveness and unilateral dystonic posturing were compatible with a frontal lobe seizure. Several episodes occurred during transfer to the hospital, each lasting 3–30 min. CT head imaging was unremarkable (figure 1).

She was discharged and seen in ambulatory care 3 days later. Her lymphocytosis, neutrophilia (described as 'reactive') and elevated creatine kinase (10 006 IU/L) were attributed to recent motor seizure activity, despite the broad differential. An electroencephalogram, brain MRI and neurology appointment were arranged, in accordance with the Trust's 'first fit' pathway, which mandates follow-up within 2 weeks, similar to the established National Institute for Health and Care Excellence guidelines.[1]

Five days later, she was brought back to hospital: she had become increasingly withdrawn, expressing a delusion that 'someone' was controlling her. She had stopped recognising her children.

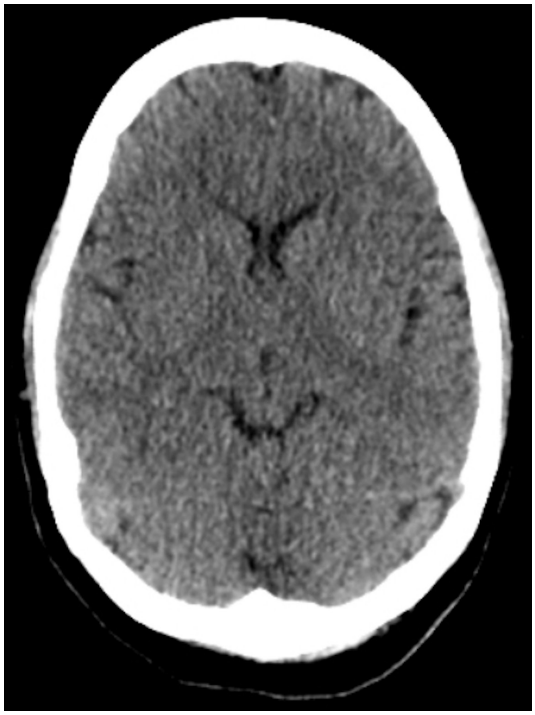

Figure 1 CT head reported as normal.

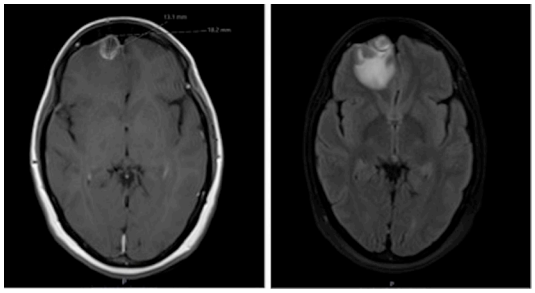

Figure 2 MRI head showing a frontal lobe ring-enhancing lesion (left), with extensive surrounding oedema (right).

On examination, she stared unblinkingly and demonstrated echolalia, echopraxia and stereotyped, repetitive, slow rotation of both wrists. However, she had intervals of apparent lucidity when she was able to answer simple questions. Physical examination was limited by behavioural disturbance, but no other abnormal neurological signs were noted.

This syndrome of frontal lobe seizures, acute psychiatric disturbance, unusual mixed hyperkinetic movement disorder and encephalopathy with normal CT brain imaging was suggestive of N-methyl D-aspartate (NMDA) receptor encephalitis. In particular, her movements did not fit more common disorders such as tremor, chorea, myoclonus, dystonia or tics and had a bizarre appearance that experts have found difficult to classify.[2]

However, an MRI brain revealed a 18×16×17 mm ring-enhancing intra-axial lesion at the frontal pole with extensive surrounding vasogenic oedema and mass effect (figure 2). Given the neutrophilia and lymphocytosis, an infective lesion (particularly a tuberculoma due to her ethnicity) was then considered the foremost differential.

She was transferred to a tertiary neuroscience centre, where investigations interrogating an infectious process were negative, including lumbar puncture, QuantiFERON, cysticercal, HIV and fungal serology. A chest–abdomen–pelvis CT was normal. A brain biopsy revealed a grade IV glioblastoma. She has been treated with levetiracetam, radiotherapy and temozolomide. Her psychiatric and motor symptoms have resolved since treatment and she has been seizure-free for 6 months. We attribute her previous symptoms to ictal activity.

Glioblastoma multiforme accounts for >60% of all adult brain tumours.[3] Although presentation can vary depending on the location of the lesion, this case is unusual in that the initial presentation was a phenocopy of NMDA encephalitis and fits clinical criteria for diagnosis.[4] However, recognised differentials of NMDA receptor encephalitis are

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varied, including infective, psychiatric, metabolic, neoplastic, paraneoplastic, cerebrovascular and other inflammatory disorders.[4] The orbitofrontal location of this lesion explained all the clinical features. The refutation of the top two clinical differentials (NMDA encephalitis and tuberculoma) highlights the importance of having a wide differential diagnosis with unusual neurological presentations.

Learning points

- Simplified pathways for common presentations like 'first fits' can streamline services, but without careful history-taking, complex presentations such as this may be inappropriately funnelled into a standardised pathway.
- A psychiatric presentation with movement disorder and seizure activity may represent a frontal space-occupying lesion, not necessarily NMDA receptor encephalitis.

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