potential for expansion of the reach and scope of the service.

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DOI: 10.1177/1039856213476123

Acute neuropsychiatric manifestations of anti-N-methyl-D-aspartate receptor encephalitis

Dear Sir,

Autoimmune limbic encephalitis (ALE) is a well-characterized neuropsychiatric syndrome with associated neuroimaging and immunological diagnostic markers.[1] Importantly, because of the predominant psychiatric symptoms in a majority of patients, most patients are seen initially by psychiatrists.[2] Immune-mediated encephalitis can be paraneoplastic and several antibodies to onconeural antigens have been identified.[3] A relatively new clinical subsyndrome of ALE has been identified in young women, often associated with antibodies to the N-methyl-D-aspartate receptor (NMDAR) and usually with a set of striking clinical features, including changes in personality, mood and anxiety symptoms, psychosis, bizarre behavior and catatonia.[2,4] Although more than 50% of cases are associated with a tumor, especially ovarian teratoma, there are cases where no such association could be demonstrated, especially in younger patients.[2,5] The characteristic clinical features of anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis are a prodromal flu-like illness followed by a range of psychiatric, neurological and autonomic symptoms and signs.[4,6]

AS was a 26-year-old Caucasian woman, postpartum four months, who was transferred to our neurology service from a rural hospital for management of suspected complex partial seizures, altered consciousness and aggressive behavior. Two weeks prior to admission to the local hospital, she had three episodes of abnormal limb movements, lip smacking, confusion, and transient difficulty in speech and swallowing. She had normal renal, thyroid and liver function with normal electrolytes, glucose, calcium, magnesium, phosphate and C-reactive protein. A brain CT scan was normal. A provisional diagnosis of complex partial seizure disorder was made and she was given oral sodium valproate and oral clonazepam. In spite of the above treatment, her behavior continued to deteriorate and she was transferred. On examination at arrival to the unit, she was found to have stereotypical oro-lingual movements, mutism and unblinking open eyes. There were several episodes, during which she would walk aimlessly and attack staff and others randomly. Other features observed during the period at various times were dystonic posturing of the left upper limb, stupor and echolalia. The EEG showed non-specific slow wave activity without any lateralizing or epileptiform features. CSF examination showed clear fluid, mononuclear pleocytosis, negative gram staining and culture, negative PCR for HSV, varicella zoster virus, enterovirus, mycobacterium and fungi, and undetectable cryptococci and pneumococcal antigens. CSF protein and glucose levels were normal but revealed oligoclonal bands. NMDAR antibodies were present both in the CSF and serum. A diagnosis of non-paraneoplastic anti-NMDAR encephalitis was considered. She was started on methylprednisolone 1 g per day and also given intravenous immunoglobulin (0.4 mg/kg body weight). Additionally, she was treated with clonazepam, haloperidol and quetiapine from the time of admission. She continued to improve over the next four weeks and was discharged.

This case illustrates the importance of considering a diagnosis of anti-NMDAR encephalitis in young women presenting with acute change in behavior with associated seizures, catatonia and aggressive behavior. There is now an increased recognition of anti-NMDAR encephalitis, and evidence has been accumulating about this condition in various clinical settings such as intensive care,[7–9] and pediatric[5] and neurology[2] units. Pruss et al. reported a prevalence of 1% in adult patients admitted to an intensive care unit.[8] Although structural brain MRI would aid in the diagnosis, the findings may be unremarkable in 50% of patients.[10] An EEG examination is always useful to support the diagnosis, although the findings vary with non-specific, slow, disorganized waves with or without epileptogenic foci.[2] A lumbar puncture must always be carried out and CSF examination performed. In about 80% of cases, CSF abnormalities are evident in the early stage.[2] As illustrated in this woman's case, the findings include lymphocytic pleocytosis, elevated protein and the presence of oligoclonal bands. Anti-NMDAR antibodies are detected in most cases.[2] Anti-NMDAR encephalitis is most commonly seen in women and an association with a tumor: in particular, ovarian teratoma has been reported in women older than 18 years.[2] With this scenario, it is recommended that all cases should be

screened for an underlying tumor by MRI, CT scan, pelvic and transvaginal ultrasound.

It has been suggested that anti-NMDAR antibody-mediated reversible neuronal dysfunction might be the pathogenic mechanism underlying this condition.[1] NMDAR antibodies are usually present in CSF in higher titers than in serum which, together with oligoclonal bands, suggests intrathecal antibody synthesis.[2] The proposed treatments include administration of both IVIg and methylprednisolone. A second line of treatment for those not responding in the first 10 days is the administration of rituximab every week for four weeks, in combination with cyclophosphamide administered with the initial dose of rituximab, followed by four weekly cycles of cyclophosmamide.

To conclude, this case underscores the importance of considering anti-NMDAR encephalitis in the differential diagnosis of a patient presenting with acute neuropsychiatric symptoms.

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DOI: 10.1177/1039856213476122

Mental health legislation and likelihood of harm criteria

Dear Sir,

O'Connor[1] voices three main concerns about the proposal to replace the likelihood of harm criteria in mental health legislation with a capacity and best interests model. In our view, his concerns are ill-founded.

O'Connor worries that incapacity-based detention decisions will be 'more arbitrary'. In fact, though, numerous studies show capacity assessments have very good inter-rater agreement,[2] and as O'Connor notes, currently '[t]here is generally poor reliability … in relation to when schedules … are made and upheld'.

O'Connor also worries that the new model will be 'more restrictive'. Under the common law, a patient can be said to lack decision-making capacity if he or she 'is unable to comprehend and retain the information which is material to the decision, in particular as to the consequences of the decision; or … unable to use and weigh the information as part of the process of making the decision'.[3] Obviously, a simple awareness that one is ill is not, of itself, sufficient to retain legal capacity. Under the new model, a patient who did retain legal capacity (properly understood) around the decision to receive psychiatric treatment would be able to refuse it, and, as O'Connor notes, this is a right that all citizens enjoy regarding all other forms of treatment. Disallowing this right to people with mental illnesses is discriminatory and contravenes our human rights obligations.[4]

Finally, O'Connor worries that law reform might propagate stigma. We strongly disagree. Characterizing people as violent is a major cause of stigma.[5] In contrast, there is no evidence to support the notion that simply acknowledging that people with mental illnesses sometimes lose decision-making capacity is likely to lead to social alienation and discrimination.

Despite the above considerations, O'Connor's final point is sound. We agree that the introduction of a capacity/best interests test into Australian mental health legislation 'requires careful design'. The nature and form of the best interests test warrants particular attention and will need to respect the 'will and preferences' of the person concerned.[6] We also agree that it is vital that there be 'a process of review, calibration, education and training' of clinicians using the modernised Act.

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