BJPsych | The British Journal of Psychiatry (2012) 200, 428–430

# Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

## Contents



## Encephalitis and psychosis

Barry *et al* reported four cases of anti-*N*-methyl-D-aspartate (NMDA) receptor antibody encephalitis that presented psychiatrically.[1] This report was welcome in highlighting the importance of immunologically mediated encephalitides (or synaptopthies), both primary autoimmune and paraneoplastic, that has emerged over recent years. However, two points are worthy of emphasis.

First, the aetiological association of anti-NMDA receptor antibody encephalitis with ovarian neoplasms was perhaps understated in the paper. In a large study by Dalmau *et al*, around 50% of cases were associated with ovarian neoplasms and 80% of such patients improved following tumour removal and first-line immunotherapy, whereas only 48% of patients without an identified tumour responded as well to first-line immunotherapy.[2] Therefore, the identification and resection of ovarian tumours in patients with this syndrome is a primary concern.

Second, Barry *et al* conclude that it is important to consider anti-NMDA receptor antibody encephalitis in new-onset psychosis associated with catatonia, seizures and dyskinesia, and that it is unclear whether there is a pure psychiatric presentation. Zandi *et al* explored this question prospectively in 46 unselected patients with new-onset psychosis, finding anti-NMDA receptor antibodies in 2 patients.[3] It was also found that there were no clinical features that differentiated between antibody positive and negative patients. Also of note, this study identified one patient positive for anti-voltage-gated potassium channel antibodies (probably, in fact, anti-leucine-rich, glioma inactivated 1 (LGI1)). It is recognised that psychosis may be a feature of autoimmune encephalitides associated with serum antibodies against a number of proteins, including LGI1 and glutamic acid decarboxylase. Further psychiatric studies are required to determine whether a screen for antibodies associated with encephalitis should be routine in new-onset psychosis.


1 Barry H, Hardiman O, Healy DG, Keogan M, Moroney J, Molnar PP, et al. Anti-NMDA receptor encephalitis: an important differential diagnosis in psychosis. *Br J Psychiatry* 2011; **199**: 508–9.

2 Dalmau J, Lancaster E, Martinez-Hernandez E, Rosenfeld MR, Balice-Gordon R. Clinical experience and laboratory investigations in patients with anti-NMDAR encephalitis. *Lancet Neurol* 2011; **10**: 63–74.

3 Zandi MS, Irani SR, Lang B, Waters P, Jones PB, McKenna P, et al. Disease-relevant autoantibodies in first episode schizophrenia. *J Neurol* 2011; **258**: 686–8.


**Nicholas Moran**, Consultant Neurologist, Kent & Canterbury Hospital, Canterbury/King's College Hospital, London, UK. Email: nfm10@aol.com

**doi: 10.1192/bjp.200.5.428**

**Authors' reply:** We thank Dr Moran for highlighting the importance of immunologically mediated encephalitis when considering differential diagnoses for atypical psychosis. Dr Moran suggests that the aetiological association of anti-NMDA receptor encephalitis with ovarian neoplasms, in particular teratomas, was perhaps understated in our case series of four patients, where we reported ovarian pathology (a dermoid cyst) in one patient.[1] By contrast, Dalmau *et al*'s original series of 100 cases identified ovarian teratomas in 54 of the 58 cases with ovarian pathology and early removal of such tumours was associated with better outcomes.[2] However, more recent studies have not observed such high rates of ovarian pathology.[3,4] In a series of 44 patients with anti-NMDA receptor encephalitis, Irani and colleagues found tumours in 9 patients of which 8 cases were ovarian teratomas. Furthermore, 25% of cases overall were male. In keeping with Dalmau and colleagues, the identification and removal of an ovarian tumour was associated with a better outcome,[2] although the best outcome was predicted by adequate immunotherapy during initial illness.[4]

As noted previously, it is still unclear whether there is a purely psychiatric presentation to this disorder. However, the constellation of symptoms including some or all of catatonia, dyskinesias and seizures with psychosis certainly warrants anti-NMDA receptor antibody testing. We agree with Dr Moran that future studies are required to determine whether routine screening for NMDA receptor antibodies is indicated for atypical presentations, treatment-resistant cases and first-onset psychosis.


1 Barry H, Hardiman O, Healy DG, Keogan M, Moroney J, Molnar PP, et al. Anti-NMDA receptor encephalitis: an important differential diagnosis in psychosis. *Br J Psychiatry* 2011; **199**: 508–9.

2 Dalmau J, Gleichman AJ, Hughes EG, Rossi JE, Peng X, Lai M, et al. Anti-NMDA-receptor encephalitis: case series and analysis of the effects of antibodies. *Lancet Neurol* 2008; **7**: 1091–8.

3 Florance NR, Davis RL, Lam C, Szperka C, Zhou L, Ahmad S, et al. Anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis in children and adolescents. *Ann Neurol* 2009; **66**: 11–8.

4 Irani SR, Bera K, Waters P, Zuliani L, Maxwell S, Zandi MS, et al. N-methyl-D-aspartate antibody encephalitis: temporal progression of clinical and paraclinical observations in a predominantly non-paraneoplastic disorder of both sexes. *Brain* 2010; **133**(Pt 6): 1655–67.


**Helen Barry**, Department of Psychiatry, Royal College of Surgeons in Ireland, RCSI Education and Research Centre, Beaumont Hospital, Dublin 9, Ireland. Email: helenbarry@rcsi.ie; **Kieran Murphy**, **David Cotter**, Department of Psychiatry, Royal College of Surgeons in Ireland, RCSI Education and Research Centre, Beaumont Hospital, Dublin, Ireland

**doi: 10.1192/bjp.200.5.428a**

## 2012 and still no Holy Grail

Allan and colleagues provided a helpful reappraisal on the use of neurostimulatory treatments for depressive illness.[1] In their words, the Holy Grail of treatment would be one as effective as electroconvulsive therapy (ECT), but better tolerated and ideally without the need for general anaesthesia. They concluded that ECT has not yet been supplanted, but we wonder whether the authors were aware of how pertinent this observation is for the year 2012.

Electroconvulsive therapy may not be in use in England by the fiscal year 2011–12. Practitioners in ECT will have seen the graph to support this suggestion at various educational events in recent years. It is based on an extrapolation of data that used to be collected by the Department of Health in quarterly surveys of the number of ECT treatments administered in England. The last two surveys were in the fourth quarter of 1998–99, and the fourth