JISC update Aug - Sept 2018

Case discussion: of possible malignant Catatonia

A young patient with no psychiatric history (or family history) presented with fluctuating psychiatric symptoms on their way home from a Mediterranean holiday. They initially presented as distant and paranoid with incongruent affect (in response to good exam grades and acceptance into the University of choice). Their mental state deteriorated quickly with significant paranoia, agitation, delusions of witch craft, posturing, elation and visual hallucinations. They were admitted to the acute Trust where they became so disturbed as to require RT and sedation on ICU in order to conduct an MRI head, LP and EEG.

Their mental state was still extremely poor once extubated with extreme agitation and lots of IM benzos/promethazine and 5mg IM haloperidol twice. They were started on a quetiapine with little effect; and then olanzapine; and responded well and tolerated 15mg + 1mg clonazepam BD.

Other findings include:

Fluctuating temperatures (continually between 38-40)
Normal CK
Slight leucocytosis
Raised platelets
No rigidity (at any point)
A posterior (cerebellar) stroke on MRI.
Slightly elevated C3 and C4
Normal EEG x 2
Normal LP x 2

We have been extensive in our investigations for autoimmune, neurological, rheumatological, haematological and infectious causes. Nothing else has returned as positive but we are still a/w NMDA, K+ and Ca2+ gated channels.

Our Neurologists think this is a primary psychiatric illness; and do not think steroids are indicated. The question of stopping olanzapine and ECT for malignant catatonia or NMS has been raised.

Response 1: That this is similar to several anti NMDA receptor antibody cases: The severity of behaviour disturbance, delusions, visual hallucinations, automatic instability with temperature fluctuations and catatonic posturing. This clinician would suggest a review of the EEG looking for nonspecific slowing, any other soft abnormalities. Many of the EEGs in their NMDA cases reported in first instance as normal but the abnormal on second look. She says it would be interesting to see if there are any orofacial or other dyskinesia type movements, reversed sleep cycle etc. The presence of confusion and fluctuating mental state (if present) should help evidence the probable organic aetiology to neurology; as well as the clear similarity to many autoimmune encephalitis cases in literature.

Response 2: reminds us about ovarian (sometimes microscopic) tumours associated with the AI limbic encephalitis picture and NMDA Abs.

Response 3: a similar case; clarified when the antibodies finally came back. EEG/LP and MRI were poor diagnostic tools. She also recommends scanning the ovaries.

Response 4: Also thinks it sounds like autoimmune encephalitis unless there is another obvious precipitant. He asks is there any campylobacter or teratoma? He adds that the differential might be substance related?
The cerebellar stroke and pyrexia sound organic rather than functional.

Response 5: Says given age and symptoms, empirical steroids treatment is indicated until NMDA and caspr bloods back; and recommends a pelvic USS to look for an ovarian teratoma. Recent US guidelines talked about prophylactic steroid treatment until otherwise in this group.

Response 6: Thinks this is organic. Would PET be useful to look for primary? Functional psychosis certainly wouldn't explain the fevers although there may be two concurrent illnesses. Is it worth trial of another antipsychotic to cover for first episode psychosis?

Response 7: Notes the consensus around this being autoimmune encephalitis; and adds that the unanswered question is does ECT has a role in treatment? His personal view is yes; with limbic/autoimmune encephalitis, lethal/malignant catatonia and NMS being overlapping syndromes. As ECT has a good track record in catatonia and NMS, 'and it is therefore a reasonable treatment option in suspected "encephalitis"', and can be given with steroid treatment.

Response 8: If this is NMDA-RE, the evidence base suggests outcome is directly related to time to Plasma exchange, or IVIG (hence the need to expedite the tests). Approximately 40-50% patients require ITU. The older literature suggested a 40-50% mortality prior to the discovery of the antibodies (we presume a proportion were labelled encephalitis lethargica patients then) - I wonder if ITU is now saving these patients?

Response 9: Asks if there any recent history of emotional problems? Have there been recreational drugs? Could a sympathomimetic type like cocaine have caused the posterior cerebellar stroke?

Response 10: I recall a case a long time ago of a young person who took cocaine in Africa and became psychotic. A PET scan showed an ischaemic temporal lobe (from the vasoconstrictive effects of cocaine) and over time as this improved his psychotic symptoms resolved.

Emotional lability post-stroke

A stroke physician colleague has just asked a liaison psychiatrist for some advice on the treatment of emotional lability in a woman in her 70s who had large right-sided parietal CVA 6 months ago. She has left hemiparesis affecting face, arm and leg, some associated dysarthria; but appears cognitively intact and is not dysphasic. She was commenced on mirtazapine whilst an inpatient on a stroke unit because of outbursts of crying. She continues to have episodes of uncontrollable crying which is both distressing/embarrassing to her and to her family, although she denies low mood or emotional triggers to the outbursts.

I wondered whether any of you can offer medication advice?

Response 1: Has had experience with similar patients with dementia, emotional lability and bladder irritability at a hospice. Amitriptyline worked well.

Response 2: SSRIs have some (limited) evidence in pathological laughter and crying post CVA. It might be worth switching to citalopram or sertraline.

Response 3: has found valproate to be helpful, anecdotally

Response 4: again anecdotal experience - 'I find Sertraline has been helpful'

Response 5: 'It does appear that she has pseudobulbar affect. There are reports of response on dextrometharphan, an antitussive with some antidepressant effect'.

Is ECT safe to give alongside delirium?

This is a patient with a 3-month history in hospital with failure to thrive secondary to reduced oral intake.

The patient has psychotic depression with pseudo dementia on review. He and his family agreed to emergency ECT; but then he developed aspiration pneumonia with delirium which is now improving on antibiotics. He however still has some delirium, inflammatory markers normalising. Medical colleagues and his family are keen to proceed as they worry about deterioration; is there any reason not to proceed?

Response 1: Has learnt not to delay ECT; especially if the pneumonia and delirium are further signs of bodily deterioration secondary to a malnourished state. It's worth being very open with all involved, especially family, about the risks of death and I rest quite a lot on the anaesthetic opinion on fitness to proceed.

Response 2: Says that this seems a relatively common problem of missed depression leading to life threatening deterioration. She suggests weighing up possible causes of aspiration and the impact of ECT. If his mood lifts and he doesn't get affected by sustained cognitive change then it should help his quality of life. If his brain is more vulnerable to the effect of ECT cognitively then there is another risk-benefit decision. You can certainly see the impact of nutritional decline in older adults and how that can lead to frailty that is hard to recover. But she has also seen several cases where the decline is caught earlier and ECT returns people to good functioning.

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