Progression of catatonia in anti-NMDA receptor encephalitis: lessons from five cases

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Background

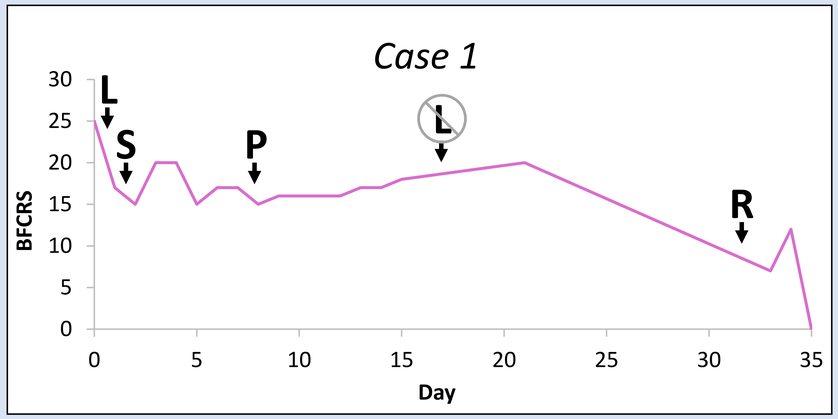
Catatonia can occur in nearly one-third of patients with anti-NMDA receptor encephalitis (anti-NMDARE), but little consensus exists about its treatment approach. Five cases of catatonia secondary to anti-NMDARE are presented and the treatment outcomes are compared.

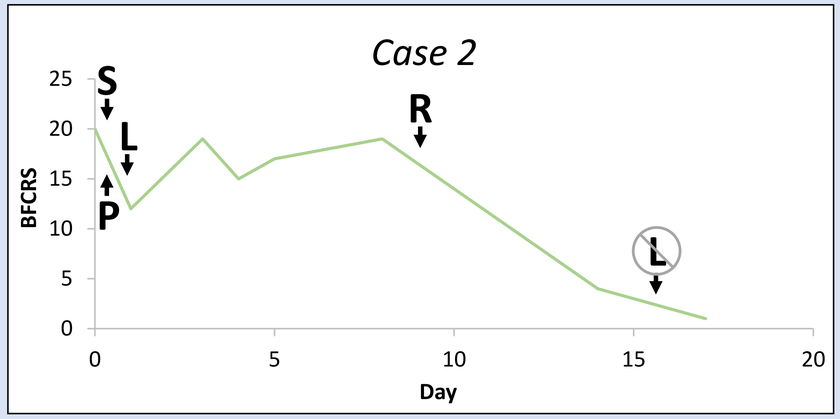
Results

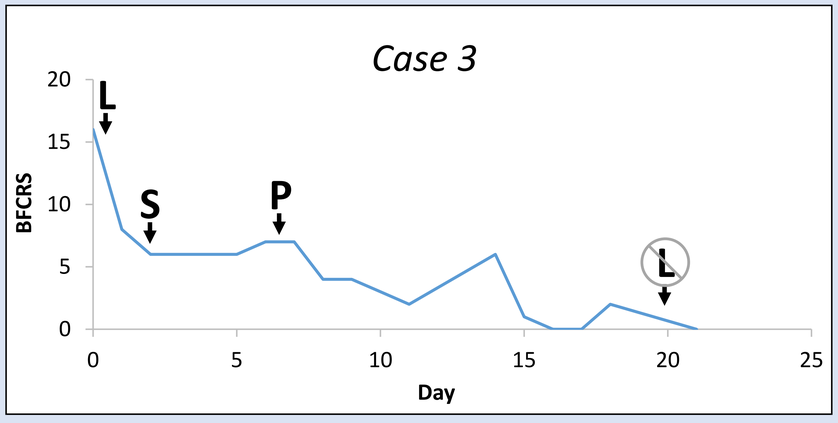
Variable	N	%
Female	5	100%
Confirmed CSF IgG	5	100%
Ovarian teratoma	2	40%
Concurrent delirium	5	100%
Concurrent autonomic instability	5	100%
Antipsychotic-emergent catatonia	3	60%
Catatonia resolution due to:		
Lorazepam	1	20%
Treatment of ANMDARE	3	60%
Treatment of ANMDARE and ECT	1	20%

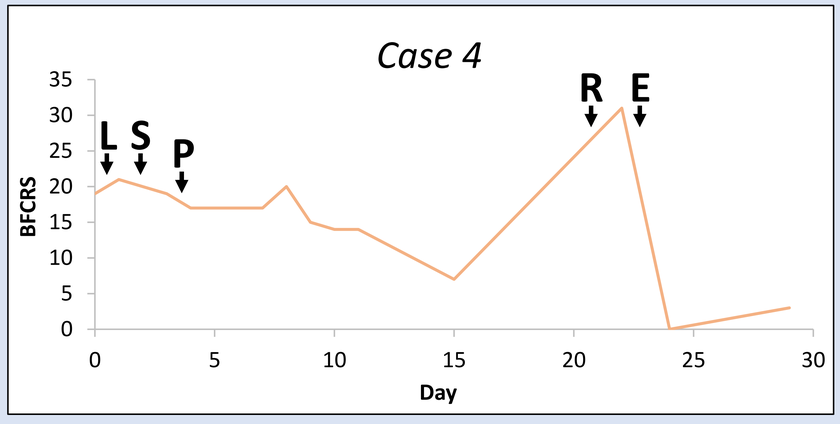
Variable (unit)	Mean	Range
Age (y)	27.4	21 to 38
Peak lorazepam dose/24h	7.5 mg	4.5 to 12

Catatonia Progression









Treatment: **L** Lorazepam **S** Steroid **P** Plasmapheresis **R** Rituximab **E** ECT

- Bush-Francis Catatonia Rating Scale (BFCRS) scores were not available for 1 case.
- *Case 5* initially had improvement of catatonia due to lorazepam, but needed escalation of care due to status epilepticus; lorazepam was not continued. Later use was ineffective. Catatonia ultimately resolved after rituximab initiation.

Discussion & Implications

- Lorazepam seemed to be less effective as ANMDARE progressed.
- We speculate that there may be a “point of futility” in administering lorazepam; this idea should be explored using more rigorous methodology.
- Catatonia treatment should be balanced with delirium risk from lorazepam, particularly if the lorazepam is no longer helpful.
- As in other medical etiologies of catatonia, identifying and treating the underlying cause is paramount.

References

References available via QR code:

The authors have no disclosures.