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Anaesthetic Management of Anti-NMDA Receptor Encephalitis with Super-Refractory Status Epilepticus Undergoing Laparoscopic Ovarian Cystectomy and Tracheostomy: A Case Report

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ABSTRACT

Anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis is an autoimmune encephalitis predominantly affecting young women and frequently associated with ovarian teratoma. Anaesthetic management is challenging due to the interaction of commonly used agents with NMDA receptor pathways, and the risk of seizures, autonomic instability, and super-refractory status epilepticus (SRSE). We report the perioperative management of a 22-year-old woman with anti-NMDAR encephalitis who underwent laparoscopic ovarian cystectomy and subsequent tracheostomy. Despite immunotherapy and multiple antiepileptic agents, she progressed to SRSE requiring ketamine infusion. Both procedures were performed under propofol-based total intravenous anaesthesia (TIVA) with remifentanil, dexmedetomidine, midazolam, and continued ketamine infusion as antiepileptic therapy. Bilateral transversus abdominis plane block provided opioid-sparing analgesia for the laparoscopic procedure. Intraoperative monitoring included invasive arterial pressure, central venous pressure, point-of-care metabolic testing, and processed EEG. No epileptiform activity or haemodynamic instability was observed during either procedure. Subsequently, the patient weaned off adjunctive anit-epileptics and achieved seizure freedom one month postoperatively. This case illustrates the importance of multidisciplinary planning, disease-specific anaesthetic drug selection, invasive monitoring, and cautious EEG interpretation in anti-NMDAR encephalitis complicated by SRSE.

Keywords: Anti-NMDR encephalitis; Super refractory status epilepticus

CASE REPORT

A 22-year-old woman with no prior medical history presented with a subacute onset of unstable emotion and auditory hallucination. She developed recurrent generalised convulsion, was intubated, and was admitted to the intensive care unit (ICU). Cerebrospinal fluid (CSF) analysis confirmed anti-NMDAR IgG antibodies thereby establishing the diagnosis of anti-NMDAR encephalitis. A Computed Tomography (CT) scan of pelvis identified a 2 x 2.3 x 3.2cm right adnexal cystic lesion, consistent with dermoid cyst. First-line immunotherapy including methyprednisolone, Intravenous immunoglobulin (IVIG) and Rituximab was initiated.

Electroencephalography (EEG) showed moderate to severe cerebral dysfunction and extreme delta brush. She was commenced on multiple antiepileptics including Lamotrigine, Lacosamide, Clonazepam, Valporate, Levetiracetam, and ketogenic diet. Despite treatment, seizure activity escalated and the patient progressed to super-refractory stratus epilepticus, fulfilling established SRSE criteria. Ketamine infusion was subsequently commenced at a rate of 1.2-7.5mg/kg/hour for a body weight of 50kg. A multidisciplinary team consisting of neurology, gynecology, ICU and anaesthesiology was convened for further management.

Laparoscopic cystectomy was performed within one week of the radiology findings becoming available. The anaesthetic goals were to minimise NMDA receptor antagonism, prevent seizure, and maintain haemodynamic and autonomic stability. Anaesthesia was conducted under general anaesthesia with bilateral transverse abdominis plane (TAP) block. Standard monitoring, invasive arterial blood pressure, central venous pressure monitoring, point-of-care blood tests (for glucose, blood gases and electrolyte) and EEG monitoring (Sedline) were used. Anaesthesia was maintained by total intravenous anaesthesia with propofol (TCI Marsh effect site 2-2.8ug/ml), remifentanil (0.1 - 0.15 ug/kg/min), dexmedetomidine (0.2-0.3 ug/kg/hr) and midazolam (3mg total bolus dose).

Rocuronium was used as the neuromuscular blocking agent. In addition to the TAP block (levobupivacine 0.25%, 40ml total volume for bilateral block) prior to surgical incision, fentanyl (150mcg total bolus dose) and paracetamol (1gm) were used for analgesia. Ketamine infusion of 1.4mg/kg/hr was continued from ICU as anti-epileptic.

Lung protective ventilation strategy was employed and adjusted to maintain normocapnia. Patient State Index (PSI) was maintained at 35-45, with EEG waveforms predominantly showing slow delta wave and frontal alpha oscillation. No epileptiform discharge was observed throughout the surgery. Surgery lasted 30 minutes with a total blood loss of 20ml. Blood gases near the end of surgery revealed normal parameters. Throughout surgery, the mean arterial pressure was maintained within 20% of the patient's baseline. Normothermia was achieved. She was then kept intubated and transferred back to ICU postoperatively. The ovarian cyst was sent to pathology and the result was consistent with mature ovarian teratoma.

Subsequently, the seizure pattern evolved from generalised tonic-clonic seizures to orofacial dyskinesia and limb twitching, and she acquired a nosocomial infection. In view of prolonged intubation, tracheostomy was arranged. A similar setup was used for the surgery and the anaesthetic goals were achieved. She eventually weaned off most adjunctive anti-epileptic, achieved seizure freedom one month after surgery and began rehabilitation. At a neurology clinic follow-up one year postoperatively, she remained seizure-free and engaged in rehabilitation for motor and cognitive functioning with progress.

DISCUSSION

Incidence and clinical relevance of anti-NMDAR encephalitis

Anti-NMDAR encephalitis is now the most frequently identified form of autoimmune encephalitis. Since it was first described in 2007,[1] meta-analysis reported the prevalence of anti-NMDAR encephalitis 7%.[2,3,4,5,6] It

predominantly affects young females, and the mortality varies from 5–7%.[7] Ovarian teratoma is identified in approximately 58% of adult female patients with anti-NMDAR encephalitis.[8] Seizures affect over half of all patients, with a quarter progressing to refractory or SRSE.[9] Surgical resection of the teratoma, together with immunotherapy, has been identified as the strongest prognostic indicator for favourable longer-term neurological outcome.[8,10.11] Hence, these patients frequently require anaesthetic intervention, such as radiological imaging and lumbar puncture under sedation, teratoma excision, and tracheostomy.[12]

Rationale for TIVA over volatile anaesthesia

With NMDA receptors acting as a pharmacological target of anaesthetic agents, anti-NMDAR encephalitis necessitates caution with various routinely used anaesthetic agents, such as ketamine, nitrous oxide, and at dose-dependent inhibition, volatile agents. Case reports have documented clinical deterioration following volatile anaesthetic exposure in these patients.[13,14] Therefore, the choice of anaesthetic agents should be carefully formulated. Propofol, in contrast, acts predominantly through GABA-A receptor, with negligible and indirect antagonism on NMDA receptor at clinical dose, and shares anticonvulsant properties. This makes TIVA with propofol a rational and commonly preferred technique.[15,16]

Dexmedetomidine also offers pharmacological advantage in anti-NMDAR encephalitis due to its independence from both NMDA and GABA receptor systems. Acting via the central presynaptic alpha-2 agonism, dexmedetomidine reduces noradrenaline release and is effectively providing sympatholysis for paroxysmal sympathetic hyperactivity (PSH).[17] Such haemodynamic stability was demonstrated in the current case report for both teratoma excision and tracheostomy under cautious drug titration with invasive monitoring. Emerging preclinical and clinical evidence suggests a neuroprotective role of dexmedetomidine[18,19] through the attenuation of neuroinflammatory cytokines, reduction of excitotoxic cascade and preservation of cerebral autoregulation. [20] These properties may be beneficial during active autoimmune neuronal injury. Furthermore, dexmedetomidine can be continued as a sedative in ICU postoperatively, which has been reported to be a safe option for prolonged sedation.[21.22]

Specific benefit of opioid-sparing multimodal analgesic

Opioids act indirectly on the NMDA receptor. The NR1 subunit of NMDA receptor is reported to conjugate with the mu-opioid receptor (MOR) as part of the functional complex.[23] Anti-NMDAR encephalitis may disrupt such interaction, potentially reduce opioid analgesic efficacy, and lead to unpredictable dose requirement[15,22]. Apart from analgesia, current evidence found that opioids exhibit immunomodulatory effects via the MOR on natural killer cells, T-cell and lymphocytes.[24] This may theoretically attenuate the intended therapeutic immune response delivered by the immunotherapy received by anti-NMDAR encephalitis patients.

Consistent with other evidence, the incorporation of bilateral TAP block in the current case report, opioid-sparing strategy could mitigate the risk of central hypoventilation, which is one of the potentially fatal complications of anti-NMDAR encephalitis.[22] Another benefit of such a strategy is facilitating early neurological assessment by preventing delayed emergence and controlling confounding factors for neurological examination, such as EEG studies.[25,26]

Level of monitoring

Despite growing incidence, there is still a lack of current guidelines on providing optimal anaesthetic care for anti-NMDAR encephalitis. Paroxysmal sympathetic hyperactivity was found intraoperatively with statistically significant tachy-brady arrhythmia and blood pressure fluctuation 30 minutes into anaesthesia.[27] A case report showed even temporary pacemaker implantation was required in a patient with severe autonomic instability.[28] Therefore, arterial line monitoring with central venous line access can be beneficial especially in cases that anticipate hemodynamically instability due to PSH, major fluid shift surgery, and the need for vasoactive drugs administration.

EEG monitoring is recommended in multiple case reports. The use of bilateral 4-channel acquisition with a density spectral array may be advantageous as it facilitates the detection of pathological EEG patterns of the disease.[29] However, the interpretation of EEG waveform for monitoring the depth of anaesthesia should be undertaken with caution. The high specific EEG pattern of extreme delta brush may confound conventional EEG monitoring.

Ketamine and Magnesium sulphate (MgSO4) in SRSE: a reframed indication

In stable disease, Ketamine is generally avoided as an anaesthetic agent due to its direct receptor antagonism. The use of Ketamine in anti-NMDAR encephalitis should be carefully justified. In vitro research has shown that prolonged status epilepticus invokes receptor trafficking responses and paradoxically upregulates synaptic NMDA receptors, while simultaneously internalising GABA-A receptor.[30] Such a phenomenon contributes to progressively treatment- resistant status epilepticus and diminished benzodiazepine anti-epileptic efficacy.[31,32] By targeting the upregulated NMDA receptors, case series reported that Ketamine infusion was effective in controlling SRSE after failure of multiple anti-epiletics and immunotherapy.[33] This is consistent with the current case, which demonstrated no seizure activity intraoperatively with ketamine infusion. The use of ketamine with EEG monitoring warrants caution in the interpretation of disease progression. Ketamine independently generates an EEG oscillatory pattern of the delta-alpha complex, which can resemble extreme delta brush.[34] DSA was found to be valuable for distinguishing disease-related EEG change from seizure activity and from spectral activity of anaesthetic agents and antiepileptics.[35]

MgSO4, another commonly used drug in daily anaesthetic practice,also acts on NMDA receptors and inhibits glutaminergic pathway to exhibit anti-epileptic effect. Limited evidence supports its safety profile in anti-NMDAR encephalitis with SRSE. MgSO4 has a particular role in obstetric anaesthesia; with young females predominantly affected by anti-NMDAR encephalitis, its use should be further explored. A case report documented no adverse maternal and fetal outcome with the use of MgSO4 for seizure control and fetal neuroprotection in a primigravid patient with anti-NMDAR encephalitis induced SRSE undergoing combined caesarean delivery and teratoma excision.[36,37]

CONCLUSION

Anti-NMDAR encephalitis complicated by SRSE presents a unique anaesthetic challenge because the disease process and several routinely used anaesthetic agents converge on NMDA receptor pathways. This case

demonstrates that a carefully selected TIVA-based technique, combined with opioid-sparing multimodal analgesia, invasive haemodynamic monitoring, and EEG surveillance, can facilitate safe perioperative management for both laparoscopic ovarian cystectomy and subsequent tracheostomy.

Continued ketamine infusion, although theoretically concerning in stable anti-NMDAR encephalitis, may be justified in the specific setting of established SRSE when used as an antiepileptic strategy.
Individualised drug selection, close multidisciplinary coordination, and cautious interpretation of processed EEG indices against the background of abnormal disease-related EEG patterns are essential to optimise outcomes in this increasingly recognised condition.

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