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# A Case Report and Literature Review on Suspected Anti-N-Methyl-D-Aspartate Receptor Encephalitis in a Child with Type 1 Diabetes at Al Sabah Children Hospital, South Sudan

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***Abstract*** *Anti-N-Methyl-D-Aspartate (NMDA) receptor encephalitis is a rare form of limbic encephalitis but represents the most common autoimmune encephalitis in children. Its clinical manifestations and disease course differ from adults in terms of tumor association, neurological presentation, and symptom frequency. While associations with autoimmune diseases such as Graves' disease, autoimmune polyglandular syndrome, and type 1 diabetes have been reported in adults, a link with type 1 diabetes has not previously been described in children. We conducted a case review of a 16-year-old adolescent with known type 1 diabetes admitted to Al Sabah Children's Hospital, Juba, South Sudan. Clinical features, treatment, and outcomes were documented. Literature was reviewed to contextualize findings and highlight diagnostic challenges in resource-limited settings. The patient presented with altered consciousness, seizures, psychotic symptoms, and movement disorder. Following initiation of high-dose corticosteroid therapy, the patient achieved complete recovery within four weeks. This case suggests a possible association between type 1 diabetes and anti-NMDA receptor encephalitis in pediatric patients, an observation not previously documented. Anti-NMDA receptor encephalitis should be considered in pediatric patients with type 1 diabetes presenting with neuropsychiatric symptoms. This case emphasizes the need for improved diagnostic capacity, awareness, and access to advanced neuroimaging and serological markers in South Sudan and other resource-limited settings.*

***Keywords*** *Anti-NMDA Receptor Encephalitis, Autoimmune Encephalitis, Pediatrics, South Sudan, Type 1 Diabetes.*

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## Introduction

Anti-N-methyl-D-aspartate receptor autoimmune encephalitis (anti-NMDAR) is the second most common cause of acute demyelinating encephalitis after mixed disturbance encephalitis, surpassing all viral causes [1-5]. Its prevalence in pediatric populations is estimated at 0.6 per 100,000 [6]. Although it can occur across all ages and sexes, it is most frequently observed in children and young adults (15–45 years), with approximately 80% of affected patients being females [2, 7-8]. Anti-NMDAR encephalitis is an antibody-mediated neurological disorder characterized by autoantibodies directed against the GluN1 subunit and the NR2A/NR2B subunits of the NMDA receptor in the hippocampus, leading to the clinical manifestations [9]. The condition may be associated with viral infections, particularly following herpes simplex virus-1 meningoencephalitis, and with certain tumors, most notably ovarian teratomas. However, in pediatric cases, the disease is often idiopathic [10].

Associations with other autoimmune diseases such as Graves' disease, type 1 diabetes, and autoimmune polyglandular syndrome have been reported in adults, reflecting the shared genetic, pathophysiological, and environmental characteristics of autoimmune disorders [11].

Clinically, patients typically present with acute behavioral changes (irritability, tantrums, manic symptoms, behavioral outbursts, sleep dysfunction, hyperactivity, or coma), seizures (focal, motor, complex partial), dystonia, status epilepticus, memory deficits, catatonia, dyskinesia (including orofacial dyskinesia), speech disturbances (mutism or reduced responsiveness), autonomic dysfunction, and hypoventilation [7]. Diagnosis is confirmed by a **10-fold increase in cerebrospinal fluid levels of anti-NMDA receptor antibodies**, which remains the gold standard. Neuroimaging is often normal, with MRI abnormalities present in only 50% of cases, typically showing nonspecific hippocampal hyperintensities on T2/FLAIR. Electroencephalography (EEG) frequently reveals abnormal slow or disorganized activity, and in rare cases, the characteristic "extreme delta brush" pattern [7].

Immunotherapy is the mainstay of treatment, with tumor removal indicated when an underlying neoplasm is identified [12-14]. The disease can be severe in the acute phase, with a mortality rate of about 5%. Relapses occur in approximately 15% of patients. Despite these challenges, functional outcomes are generally favorable, although neuropsychological and psychiatric sequelae remain relatively common [14]. Early administration of immunotherapy improves outcomes and reduces relapse risk [14].

## Case Presentation

### History

A 16-year-old male adolescent with a known history of type 1 diabetes mellitus since the age of five, managed with insulin (Mixtard) at 1 IU/kg/day (28 IU in the morning and 14 IU in the evening), presented with acute neurological symptoms. His diabetes was reported to be well controlled.

Two weeks prior, he had been admitted to the Intensive Care Unit at Juba Teaching Hospital with cerebral malaria and diabetic ketoacidosis (DKA), treated successfully, and discharged after seven days. Unfortunately, 5 days after discharged from Juba Teaching Hospital, he was admitted to Mauna Medical Centre for 24 hours treatment for recurrent DKA and cerebral malaria, with no improvement. Ultimately he was referred to Al Sabah Children Hospital.

At presentation, he had a one-day history of high-grade persistent fever unresponsive to paracetamol or tepid sponging. The fever was associated with abnormal body movements characterized by recurrent episodes of flexion

and extension of both upper and lower limbs, each lasting less than one minute. These episodes were accompanied by upward rolling of the eyeballs and drooling of saliva, but without sphincter incontinence. He also reported upper respiratory symptoms (cough and sneezing) five days prior to the onset of neurological manifestations.

### Examination

On arrival, the patient appeared acutely ill and comatose, with a Glasgow Coma Scale score of 6/15. He was febrile (temperature 38.8°C), with mid-sized pupils bilaterally reactive to light. The remainder of the physical examination was unremarkable.

### Investigations

The followings were the investigations requested with their results:

1.Random blood glucose: 433 mg/dl 2. Urinalysis: WBCs 10–15 cells/HPF, numerous pus cells, positive leukocyte esterase, glucose +++, ketones +++ 3. Widal test: reactive for *Salmonella enterica* 4. Complete blood count: WBC 3.5 × 10⁹/L, lymphocytes 40%, granulocytes 78%, hemoglobin 10 g/dl, platelets 420,000/µL 5. Serum electrolytes, urea, and creatinine: within normal limits.

### Initial Impression

The working diagnosis was coma secondary to severe diabetic ketoacidosis, cerebral malaria, enteric fever, and urinary tract infection.

### Differential Diagnosis

Bacterial meningitis and meningoencephalitis.

## Management and Outcome

### Treatment

The patient was started on:

- Artesunate 2.4 mg/kg at 0, 12, and 24 hours, then once daily for 2 days
- Dexamethasone 0.6 mg/kg/day in 4 divided doses
- Cefepime 50 mg/kg every 12 hours
- Vancomycin 20 mg/kg in 30 ml of normal saline every 8 hours
- Maintenance fluids (normal saline) for DKA
- Acyclovir 20 mg/kg every 8 hours via nasogastric tube
- Anticonvulsants:
  - Phenytoin: loading dose 20 mg/kg, reloaded twice with 10 mg/kg each (maximum 40 mg/kg), then 5 mg/kg in 2 divided doses
  - Phenobarbitone: loading dose 20 mg/kg, reloaded twice with 10 mg/kg each (maximum 40 mg/kg), then 5 mg/kg in 2 divided doses
- Regular insulin at 0.1 IU/kg/hour

### Clinical Course

Thirty-six hours after admission, the patient improved from DKA with stabilization of metabolic status. His Glasgow Coma Scale rose to 13/15 (awake but confused). However, he developed left-sided weakness affecting both upper and lower limbs, with complete loss of movement and sensation. Neurological examination revealed loss of the left nasolabial fold, deviation of the mouth to the right, and inability to swallow solids and liquids.

A brain CT scan was requested with an impression of stroke secondary to CNS infection. The scan, performed after 7 days (without contrast), was unremarkable.

On the fifth day of admission, the patient became unresponsive to voice and pain. He developed repeated episodes of movement disorder characterized by lip smacking, limb flailing, eye blinking, and hyperextension of the neck and trunk, each lasting 30–60 minutes.

By the tenth day, he regained responsiveness with partial improvement in limb movement, swallowing, and restoration of the nasolabial fold. However, he developed psychotic symptoms, including hallucinations and delusions. He repeatedly reported hearing the voice of his elder sister (who had died eight

months earlier) calling him to follow her, expressed fear of imminent death, and exhibited excessive talking for several hours without sleep for more than 16 hours.

**Impression**

Based on the clinical course, the impression was autoimmune encephalitis, most likely anti-NMDA receptor encephalitis. However, due to lack of access to cerebrospinal fluid antibody testing (gold standard), MRI, and electroencephalography in our setting, the diagnosis could not be confirmed.

**Definitive Management**

The patient was initiated on prednisolone 2 mg/kg/day and haloperidol 5 mg orally once daily.

Within 5 days of treatment, he showed a remarkable improvement with reduction in movement disorders, seizures, and behavioral symptoms. After 25 days of admission, his symptoms had completely resolved.

**Outcome**

The patient was discharged home with significant improvement and linked to the outpatient follow-up clinic for continued care.

## Discussion

Anti-N-methyl-D-aspartate receptor autoimmune encephalitis (anti-NMDAR) is the second most common cause of acute demyelinating encephalitis after mixed disturbance encephalitis, surpassing all viral causes. It is characterized by the subacute onset of short-term memory loss, behavioral changes, and seizures involving the medial temporal lobe and amygdalae [1-5, 15, 16].

The condition was first described by Dalmau et al. in 2007 [7]. It occurs across all ages and sexes but is most frequently seen in children and young adults (15–45 years), with approximately 80% of cases reported in females [7, 8]. Our patient, a 16-year-old male adolescent, falls within the commonly affected age group described in the literature.

Anti-NMDAR encephalitis may be associated with viral infections, particularly following herpes simplex virus-1 meningoencephalitis, and with certain tumors, most notably ovarian teratomas. In pediatric cases, however, the disease is often idiopathic [9]. Our patient had preceding upper respiratory symptoms (cough, rhinorrhea, and sneezing), which may suggest a viral trigger. Given that tumor-associated cases are more common in females and older patients, malignancy was considered less likely in this case.

Associations with other autoimmune diseases such as Graves' disease, type 1 diabetes, and autoimmune polyglandular syndrome have been reported in adults, reflecting shared genetic, pathophysiological, and environmental factors [10]. However, no study has documented anti-NMDAR encephalitis in pediatric patients with type 1 diabetes. This case therefore represents the first reported suspected association between type 1 diabetes and anti-NMDAR encephalitis in a child.

The clinical presentation of anti-NMDAR encephalitis differs between adults and children. In adults, psychiatric symptoms (67%), cognitive impairment (19%), and seizures (14%) are the most common initial manifestations, particularly in females. In contrast, children typically present with non-psychiatric symptoms such as seizures (72%), with focal seizures (42%) being the most frequent, followed by status epilepticus and later behavioral disturbances [17]. Our patient initially presented with seizures that progressed to status epilepticus, followed by behavioral changes, consistent with pediatric patterns described in the literature.

The hallmark of anti-NMDAR encephalitis is a 10-fold increase in cerebrospinal fluid antibody titers, which remains the gold standard for diagnosis. Unfortunately, this test was unavailable in our setting. Brain CT findings are typically normal, as was the case for our patient. MRI abnormalities are seen in 40–50%

of patients, usually nonspecific hippocampal hyperintensities on T2/FLAIR sequences, but MRI could not be performed due to financial constraints. Electroencephalography often shows generalized slow or disorganized activity, and in rare cases, the characteristic "extreme delta brush" pattern. However, EEG was also unavailable in our pediatric facility.

Immunotherapy remains the cornerstone of treatment, with corticosteroids, intravenous immunoglobulin, and plasma exchange as first-line options. Rituximab and cyclophosphamide are reserved for refractory cases [12-14]. Our patient was treated with high-dose corticosteroids (prednisolone), to which he responded favorably. Therapy was tapered over four weeks following recovery and subsequently discontinued.

Anti-NMDAR encephalitis can be severe in the acute phase, with a mortality rate of approximately 5%. Relapses occur in about 15% of patients. Despite these risks, long-term outcomes are generally favorable, although neuropsychological and psychiatric sequelae are relatively common [12-14]. Early initiation of immunotherapy improves prognosis and reduces relapse risk. Our patient responded well to corticosteroid therapy within a short duration, further supporting the clinical impression of autoimmune encephalitis.

## Conclusion

This case demonstrates the importance of considering anti-NMDA receptor encephalitis in pediatric patients with type 1 diabetes or other autoimmune diseases who present with refractory status epilepticus, psychiatric symptoms, and movement disorders. Despite the absence of confirmatory tests, advanced imaging, and EEG, the patient responded well to corticosteroid therapy, highlighting the importance of clinical vigilance. The case underscores the urgent need to strengthen intensive care services, diagnostic infrastructure, human resource capacity, and health financing mechanisms to improve outcomes for complex pediatric neurological conditions in South Sudan.

At the same time, the case highlights the systemic challenges faced in South Sudan: limited intensive care capacity, lack of access to cerebrospinal fluid antibody testing, MRI, and EEG, shortages of trained pediatric neurologists, and absence of health insurance mechanisms to cover advanced diagnostics and prolonged ICU care. These gaps underscore the urgent need for policy and infrastructure development to improve outcomes for children with complex neurological conditions.

## Recommendations

The followings are the agreed recommendations by the authors:

1. **Intensive Care Unit strengthening:** Establish an ICU unit and equip with necessary equipment like ventilatory support, monitoring equipment, and trained pediatric critical care staff.
2. **Diagnostic facilities:** Establish access to CSF antibody testing, MRI, and EEG to enable timely and accurate diagnosis of autoimmune encephalitis.
3. **Human resources:** Invest in training pediatric neurologists, and ICU nurses in recognition and management of autoimmune neurological disorders.
4. **Health insurance:** Introduce affordable insurance schemes to reduce out-of-pocket costs for families requiring advanced diagnostics and prolonged ICU care.
5. **Policy development:** Formulate national policies to integrate rare autoimmune diseases into child health programs, ensuring funding for diagnostics, treatment, and follow-up.

### Limitations

1. **ICU constraints:** Limited critical care beds and equipment restricted monitoring and supportive management.

2. **Diagnostic gaps:** Absence of CSF antibody testing, MRI, and EEG prevented definitive diagnosis.
3. **Human resource shortages:** Few trained pediatric neurologists and ICU specialists hindered comprehensive care.
4. **Financial barriers:** Lack of health insurance forced reliance on out-of-pocket payments, limiting access to advanced investigations.
5. **Policy gaps:** No national framework exists for rare pediatric autoimmune diseases, leaving hospitals without structured support.

### Lessons Learned

1. **Clinical suspicion is vital:** In the absence of confirmatory tests, careful clinical observation and response to immunotherapy can guide diagnosis.
2. **ICU readiness matters:** Strengthening pediatric ICU services is essential for managing prolonged seizures, movement disorders, and altered consciousness.
3. **Diagnostics drive outcomes:** Access to antibody testing, MRI, and EEG would have allowed earlier confirmation and tailored management.
4. **Human resources are critical:** Training and retaining pediatric specialists is key to improving recognition and treatment of rare neurological conditions.
5. **Insurance and policy support:** Affordable health insurance and national policies are needed to reduce financial barriers and ensure equitable access to care.

## Declaration

Written informed consent for publication was obtained from the patient's parents.

## Competing interests

None.

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## Ethical Approval

Not applicable. This is a case report.

## Author's contributions

JP drafted the case report, literature review, and conclusion with recommendations, while JL and KS reviewed, expanded it, and edited. All reviewed the final manuscript and agreed on the final version.

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None.

## Data Availability

The data generated and analyzed from this case report is available on reasonable request from the authors of this case report.

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