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A Twenty Two Months Toddler of Anti-NMDA Receptor Encephalitis Presenting with Total Insomnia for 4 days: The First Toddler Case in World Literature

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Abstract

Anti-N-methyl-D-aspartate receptor encephalitis (anti-NMDARe) is the most frequent autoimmune encephalitis in children and may cause long-term sequelae. It is caused by antibodies directed against the RN1 or RN2 subunits of NMDA receptors. It is characterized by a prodromal phase of unspecific illness with fever that resembles a viral disease. The prodromal phase is followed by seizures, disturbed consciousness, psychiatric features, prominent abnormal movements, and autonomic imbalance. Here, we report a rare case of anti-NMDARe in a 22 months-old girl visited our multidisciplinary neurosciences clinic due to drug resistant total insomnia and aggression for four days without any other prodromal symptoms. The cerebrospinal fluid tested positive for anti-NMDAR antibodies and no association with teratoma or thymoma was found. The patient was treated with intravenous corticosteroid and immunoglobulin therapy concomitantly, which resulted in a prompt clear clinical improvement as she regained the ability to eat, sleep and social interaction with resumption, without behavioral disturbance and was discharged after 7 days from the hospital.

Introduction

Anti-NMDA receptor encephalitis (NMDARE) causes neuropsychiatric symptoms [1,2] resulting in morbidity in 20% [3] and mortality in 10%. [3,4] It is associated with autoantibodies against the GluN1 subunit of NMDA receptors on the neuronal surface, leading to inflammation and dysfunction. This autoimmune encephalitis develops due to the formation and attachment of IgG, particularly IgG1 and G3, to NMDA receptor (NR1) subunit with subsequent internalization of NMDA (glutamate) receptors, reduction of neuronal Ca influx, and decrease in the receptor-dependent synaptic currents.[5] Florance et al. [6] reported the first pediatric patient (age <18 years) with anti-NMDA receptor encephalitis. Although the clinical manifestation of anti-NMDAR encephalitis in children and adults was similar, most children did not have underlying tumors. [7]

Anti-NMDAR encephalitis in young children presents with a distinct, often rapid progression of neuropsychiatric symptoms, frequently starting with a viral-like prodrome (fever, headache) followed by behavioral changes (agitation, irritability, tantrums), seizures, movement disorders (dyskinesia, dystonia), and speech dysfunction, including mutism. [6,7]

EEG in children with anti-NMDAR encephalitis is abnormal in 80–90% of cases, typically showing non-specific, diffuse, slow-wave encephalopathy, focal, or multi-focal slowing. While "extreme delta brush" (EDB) is a signature finding, it is seen in only 6–53% of pediatric cases. Common findings include background slowing (63–100%) and epileptiform discharges (33–56%), with 16–52% experiencing seizures.[10] Brain MRI findings in children with Anti-NMDAR encephalitis are often normal, reported in 40-60% of pediatric cases at onset, making it a diagnosis primarily based on clinical symptoms and CSF antibody testing. When abnormal, findings are typically non-specific, featuring T2/FLAIR hyperintensities in the cortical/subcortical areas and hippocampus. [11] CSF analysis in children with anti-NMDAR encephalitis typically reveals elevated white blood cell counts (lymphocytic pleocytosis) in >90% of cases, elevated protein levels, and positive oligoclonal bands (OCBs). The most definitive diagnostic marker is the detection of anti-NMDAR antibodies in the CSF, which is more sensitive (100% in some studies) and specific than serum. [12] The first-line treatment for anti-NMDA receptor encephalitis is immunotherapy, including methylprednisolone, intravenous immunoglobulins, and plasma exchange. The second-line treatment is immunosuppression, typically with rituximab, azathioprine or cyclophosphamide.[13] Awareness of the disease and recognition of its symptoms are key to the early diagnosis and prompt initiation of treatment which may improve outcomes. With treatment, 75% of patients are estimated to recover completely while 25% may face residual neurodevelopmental impairment or death [14]. We report a case of a toddler with anti-NMDAR encephalitis in which the symptom prompting medical evaluation was total insomnia which has not been previously emphasized in the literature as a presenting feature of anti-NMDAR encephalitis. On prompt clinical diagnosis and starting empirical immunosuppressant and immunomodulating therapy, we were able to discharge her from hospital on the 7th day of admission.

Case Report

A Twenty-two months girl youngest third child of non-consanguineous marriage, with no significant past history and

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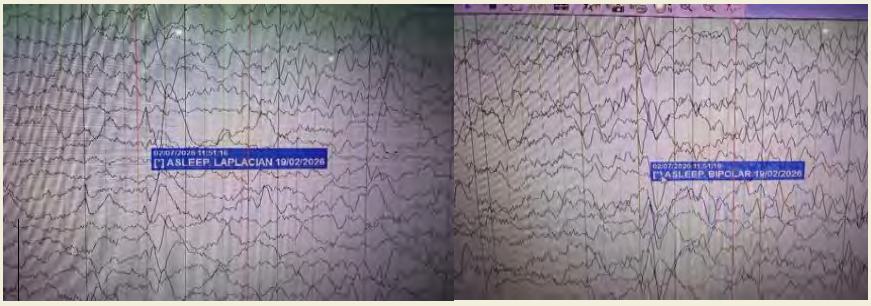

Figure 1A: Evolution of EEG Changes in Anti-NMDAR Encephalitis Over 4 Months.

Background is moderately slow for age, dominated by theta–delta activity. Poorly organized posterior rhythm. Sleep state achieved but architecture is poorly formed. Generalized high-amplitude rhythmic delta activity, intermittent frontal predominance. No definite epileptiform activity.

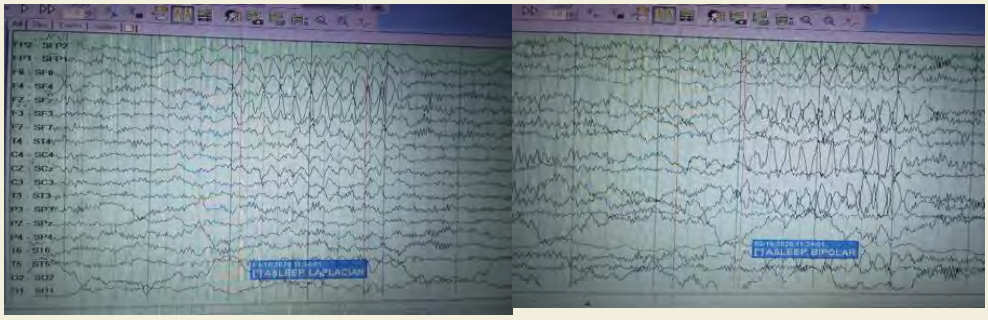

Figure 1B: Background is moderately slow for age, dominated by theta–delta activity. Poorly organized posterior rhythm. Sleep state achieved but architecture is poorly formed. Generalized high-amplitude rhythmic delta activity, intermittent more clear frontal predominance.

Figure 1C: Background is slow for age, dominated by theta–delta activity. Poorly organized posterior rhythm. Sleep state achieved but architecture is poorly formed. Generalized intermittent high-amplitude non-rhythmic delta activity, in frontal predominance. No definite epileptiform activity.

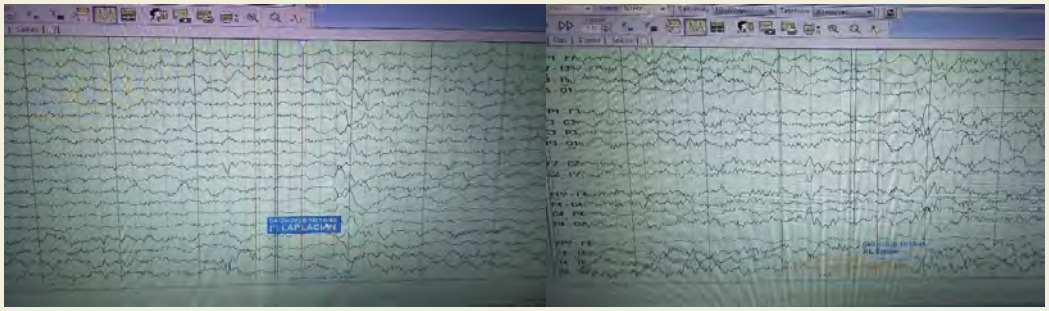

Figure 1D: Background is moderately slow for age, dominated by theta–delta activity. Poorly organized posterior rhythm. Generalized high-amplitude delta activity has decreased as compared with previous EEG record.

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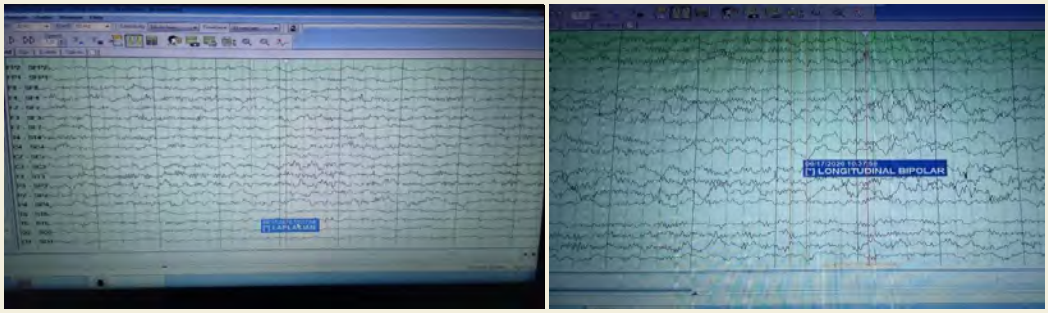

Figure 1E: Background is moderately slow for age, dominated by theta–delta activity. Poorly organized posterior rhythm. Generalized high-amplitude delta activity has almost disappeared.

Figure 2A: At admission. Bilateral symmetrical areas of T2/FLAIR hyperintensities in bilateral basal ganglia metabolic/ toxic encephalopathy.

Figure 2B: MRI. After 03 months: Complete radiological resolution of previously noted bilateral basal ganglia signal abnormalities.

developmentally normal child presented to our multidisciplinary OPD with total drug-resistant insomnia for last four days. The patient was a typically developing child born after a full-term uncomplicated gestation and delivery. She had no prior history of injury, ingestion, psychosocial stressors, change in appetite, involuntary movements, or seizures. Three months prior to this presentation, she had been evaluated by his primary care physician after a three-day febrile illness and was diagnosed with a viral infection. Since that time, she had been absolutely well and did not visit any health facility. For the current illness, she was initially evaluated and treated for insomnia by various pediatric specialists with risperidone and melatonin, but her symptoms were worsening over four days and not had slept for a single hour over last four nights. However, over these four days she became extremely agitated and started biting herself and her carers. The family history was negative for neurologic or psychiatric illness.

In the clinic she had episodic outbursts of excessive, inconsolable crying and distress that were described "as she was having nightmares while awake". During these episodes of extreme distress, she was bursting into tears and requiring physical restraint by family members against combativeness. The patients' initial physical examination revealed normal anthropometry, vital signs, cardiovascular, pulmonary, and abdominal findings. She did not have dysmorphic features or a rash. Examination of the cranial nerves, muscle bulk, tone, reflexes and strength were normal, but she was not straightening her legs. There was no sign of meningitis on physical examination. Initial differential for this clinical presentation was broad including insomnia secondary to pain from corneal abrasion or unidentified traumatic or infectious source, traumatic brain injury, developmental disorder with regression, encephalopathy due to acute intoxication, acute psychosis, postinfectious or parainfectious intravenous and autoimmune encephalopathy. Spot EEG was done in the clinic and urgent cerebral MRI was requested. Electroencephalogram (EEG) was done under sedation with oral ChlorHydrate in the dose of 70mg per kg, as she was very dysphoric and agitated. Her EEG was non-specific, revealing diffuse delta slowing with occasional paroxysmal high amplitude delta bursts of 1-4 seconds with superadded fast beta activity but no definite extreme-delta-brush discharges were documented [Figure 1A]. She was admitted to the general ward under pediatric neurology services with support from consulting services for further evaluation and management given his altered mental status

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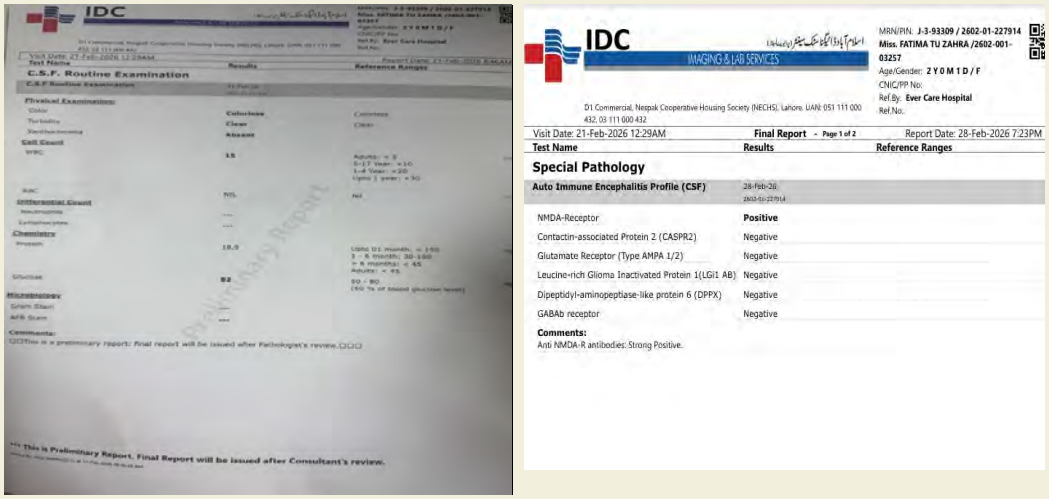

IDC

C.S.F. Routine Examination

Test Name | Results | Reference Ranges

Physical Examination:

Color — Colorless

Clarity — Clear

Xanthochromia — Absent

Cell Count

WBC — 15 — Adults: < 5; 5-17 Years: < 10; 1-4 Year: < 20; Upto 1 year: < 30

RBC — Nil — Nil

Differential Count

Neutrophils — ...

Lymphocytes — ...

Chemistry

Protein — 18.0 — Upto 01 month: < 150; 1 - 6 month: 30-150; > 6 months: < 45; Adults: < 45

Glucose — 52 — 50 - 80 (60 % of blood glucose level)

Microbiology

Gram Stain — ...

AFB Stain — ...

Comments:

This is a preliminary report. Final report will be issued after Pathologist's review.

*** This is Preliminary Report. Final Report will be issued after Consultant's review.

IDC

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MRN/PIN: J-3-93309 / 2602-01-227914

Miss. FATIMA TU ZAHRA /2602-001-03257

Age/Gender: 2 Y 0 M 1 D / F

CNIC/PP No:

Ref.By: Ever Care Hospital

Ref.No.

Visit Date: 21-Feb-2026 12:29AM | Final Report - Page 1 of 2 | Report Date: 28-Feb-2026 7:23PM

Test Name	Results	Reference Ranges
Special Pathology		
Auto Immune Encephalitis Profile (CSF)	28-Feb-26 2602-01-227914	
NMDA-Receptor	**Positive**	
Contactin-associated Protein 2 (CASPR2)	Negative	
Glutamate Receptor (Type AMPA 1/2)	Negative	
Leucine-rich Glioma Inactivated Protein 1(LGI1 AB)	Negative	
Dipeptidyl-aminopeptiase-like protein 6 (DPPX)	Negative	
GABAb receptor	Negative	

Comments:
Anti NMDA-R antibodies: Strong Positive.

Figure 3: CSF Report on admission.

and concern for acute encephalopathy. CSF autoimmune panel was sent and urgent non-contrast cerebral MRI was requested. Urgent routine CSF analysis and was normal and after 18 hours of admission contrast cerebral MRI report was: bilateral symmetrical areas of T2/FLAIR hyperintensities in bilateral ganglia [Figure 2A]. Possibility of metabolic/toxic encephalopathy was considered in differentials.

As this toddler was previously absolutely normal, she was empirically initiated intravenous methylprednisolone pulses (30 mg/kg/day) and immunoglobulin (400 mg/kg/day) for five days, considering a provisional diagnosis of autoimmune encephalitis. Eventually, CSF NMDA receptor antibody by fixed cell-based (semiquantitative) assay came back strongly positive [Figure 3]. With prompt immunotherapy with methylprednisolone concomitantly, the child showed marked improvement and after 5 days of pulse therapy she was discharged on oral steroids (hydrocortisone 1mg/kg 12 hourly) with some intermittent agitation. She was mobile and recognizing all her family members. She is being monitored regularly and after one month of her discharge, her detailed evaluation revealed her normal basal developmental (normal for her chronological age), but her EEG (Figure 1B) was still abnormal. Though she had no apparent convulsions, considering the possibility of subclinical seizures her oral Levetiracetam was continued and being followed monthly.

On her subsequent monthly visits, we haven't documented any residual neurological, behavioral, or sleep-related symptoms after one month of her discharge from the hospital. However, her asleep EEGs were abnormal, more or less of the same pattern till 17th JUNE,2026 (5th EEG- (Figure 1E). As, at 4 months of follow-up her EEG was absolutely normal and clinically and neurologically she was normal for her age. After this her hydrocortisone is decreased to 0.5 mg/kg/12 hourly, mycophenolate mofetil (MMF) in the dose of 10 mg /kg/12 hourly is commenced and levetiracetam is continued along with other supportive therapies.

Her long-term management plan is further tapering oral steroids over next 6 months and increasing mycophenolate mofetil to 20 mg/kg/12hourly along with other supportive therapies and close clinical monitoring. Our plan is to continue immunosuppressant/immunomodulating therapies for two years, if she remains asymptomatic. Due to financial constraints, we will repeat her MRI. Brain and CSF immune panel at one year after the hospital discharges and earlier if she becomes symptomatic

Discussion

The first pediatric case was reported by Höftberger et al. [15] in 2015, highlighting the clinical features and antibody profiles in children. The initiating triggers are varied and include viral infections and tumors [16], which cause the formation of autoantibodies to glutamate NMDARs, leading to an autoimmune response with both neurological and neuropsychiatric sequelae. This autoimmune encephalitis is a disorder with characteristic clinical features that are predominantly seen in young adults and children with or without teratomas [6, 17]. Most patients have five stages of clinical presentation: a prodromal phase, psychotic and/or seizure phase, unresponsive and/or catatonic phase, hyperkinetic phase, and gradual recovery phase. [18] In contrast our patient had no other symptom except total insomnia and agitation. As children with anti-NMDAR encephalitis have multiple symptoms, and monosymptomatic cases are present in only 1% of patients that is why anti-NMDAR encephalitis is unlikely to be a cause of isolated psychosis and is usually accompanied by seizures [7]. Seizures are a common presentation of anti-NMDA receptor encephalitis in children and young men [7,19]. Approximately 70% of patients develop seizures, with reported frequencies of 57% to 82% [20]. Seizures as the sole manifestation occurs in only 23% of children. Most likely, as in our toddler was predominantly monosymptomatic of total insomnia without any complaint of seizures and her diagnosis was being missed.

The diagnosis of anti- NMDAR encephalitis (ANMDARE) is based on clinical suspicion, supported with EEG findings, MRI Brain findings and confirmed by the demonstration of antibodies in the serum or, more sensitively, the cerebrospinal fluid. [7] The

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electroencephalogram (EEG) is almost always (90–100%) abnormal in cases of ANMDARE and typically shows generalised or pre dominantly frontotemporal slowing [6, 7]. In agreement similar findings of EEG were documented in our toddler.

Epileptogenic abnormalities are less common, seen in 24–50% of cases, and are possibly more common during the early stage of the illness [21]. In concordance no definite epileptogenic abnormalities were present in our patient. The EEG finding of extreme delta brush (EDB) [17], extreme beta brush [6], and brief rhythmic discharge [7] have been identified as relatively novel and potential biomarkers for ANMDARE. However, none of these findings were present in EEG of our patient. MRI- Brain, an essential tool in diagnosing and monitoring ANMDARE, can be normal in up to 60% of patients. [22, 23] Common MRI manifestations include cortical T2-hyperintensities and cortical atrophy, notably in limbic regions. [7,21,24] When MRI abnormalities are present, they are most found in the temporal, frontal, and parietal lobes. [25] Though our patient was evaluated on sixth day of her illness, we documented bilateral basal ganglion encephalitis. MRI-Brain abnormalities, usually T2-hyperintense lesions, occur in one-third of children and adults with NMDARE [1,2]: T2-hippocampal lesions associated with worse outcomes in adults, but not in children.[26] In agreement, though basal T2-FLAIR images showed very prominent basal ganglion encephalitis, our patient was discharged on 7th day of hospitalization and MRI-Brain after three months from discharge showed complete resolution of basal ganglion hyperintensities. Severe or total insomnia can be the initial or primary presenting feature of autoimmune encephalitis (AE) in pediatric patients, even if the classic diagnostic signs are missing early on, as profound sleep disruption is a known, critical marker of neuroinflammation in children [6]. This clinical presentation, afebrile toddler with aggression and EEG findings supported with specific MRI. Brain findings (basal ganglion encephalitis) in a normally developing toddler prompted us for the diagnosis of AE and starting empirical treatment.

Cerebrospinal fluid (CSF) analysis is the cornerstone for diagnosing pediatric NMDAR encephalitis, with detection of IgG antibodies against the GluN1 subunit of the NMDAR in the CSF being the definitive confirmatory test. It is highly sensitive, often revealing CSF-specific oligoclonal bands (60% of cases), lymphocytic pleocytosis, and elevated protein, making it more reliable than serum alone for diagnosis. [13, 17, 27] Cell-based assays (CBA) are the gold standard for detecting anti-NMDAR antibodies in children with autoimmune encephalitis, typically testing for IgG against the GluN1 subunit in CSF (highly sensitive), which was positive in our patient but no other abnormality of CSF was documented. Conversely to intracellular antigens, cell surface or synaptic proteins antibodies cannot be detected by standard immunoblot or ELISA and require the use of cell-based assays. The inclusion of antibodies in the CSF is vital, since serum testing is less reliable in the diagnosis of anti-NMDAR AE (14% false negative results) [13,21]

As specific antibody test results can be delayed, treatment for suspected AE is often given empirically. Early initiation of immunotherapies has been shown to improve outcomes and reduce relapses. [1,28] Once the diagnosis is confirmed, the treatment approach to anti-NMDAR encephalitis generally involves escalation of immunotherapy alongside teratoma removal where applicable. [1, 24,29] First line immunotherapies include high-dose steroids, intravenous immunoglobulins (IVIG) and plasma exchange (PLEX), sequentially or concomitantly. Second-line immunotherapies, such as rituximab, azathioprine or cyclophosphamide are used for refractory cases. Empirically, we used high-dose steroids (Methylprednisolone 30 mg /kg/day single dose and IVIG (400 mg per kg /day for 5 days) Concomitantly. Our patient responded very well, confirming that early start and concomitant use of steroids and immunoglobulins are more effective. This is consistent with a previous report by Titulaer et al. [4], confirming that these first-line therapies are broad in their mechanisms of action and can therefore be utilized empirically. However, to the best our knowledge no toddler in the world literature with such severe total insomnia has been managed in general ward and discharged in almost normal health state after seventh day of admission (11th day after symptoms onset). Therefore, regardless of antibody status or duration of symptoms, once a diagnosis of possible or definite AE is made, initiating therapy as soon as possible is indicated. Children have been reported to recover faster than adults, usually within six months. [30] Toddlers have a good prognosis, with full recovery in 67% of patients and no reports of mortality. [31]

In conclusion, because infants and toddlers cannot verbally report hallucinations or delusions, their symptoms are frequently misattributed to viral prodromes, severe temper tantrums, or primary psychiatric issues. Accurate Anti-NMDAR Encephalitis diagnosis in toddlers relies heavily on identifying these non-classical features, as young children rarely present with the textbook adult psychosis. Instead, the presentation is dominated by neurological and developmental red flags. [6] We believe that ANMDARE is probably under reported in infants and toddlers. Early recognition and empirical aggressive immunosuppressive therapies are essential in order to improve outcomes.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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