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(CASE REPORT)

Diagnostic and therapeutic difficulties of pediatric encephalitis with a N Methyl D Aspartate in Libreville: about three cases.

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Abstract

Anti-NMDAR antibody encephalitis is an autoimmune neurological disease, which is defined as an inflammation of the brain due to antibodies attached to N-Methyl-D-Aspartate receptors.

This is the most common autoimmune encephalitis and a neurological emergency. It accounts for 4% of all encephalitis cases, making it the fifth most common cause after herpes simplex virus and varicella-zoster virus (VZV) infections. It is rare in pediatrics, with an incidence ranging from 0.07 to 0.085 per 100,000 children in Europe, but in Africa it remains underdiagnosed due to the very high cost of antibody testing in cerebrospinal fluid (CSF) and treatment.

It is characterized by neuropsychiatric symptoms and leads to serious cognitive and psychomotor sequelae if left untreated. Therefore, we share our experience with three pediatric cases to clarify the circumstances of their onset, the various clinical signs, and finally, to highlight the diagnostic and management difficulties we encountered in our setting.

Keywords: Encephalitis; Anti-NMDA antibodies; Children; Sequelae

1. Introduction

Anti-NMDAR antibody encephalitis is an autoimmune encephalitis, defined as inflammation of the brain caused by antibodies bound to N-Methyl-D-Aspartate receptors. It is the most common autoimmune encephalitis and a neurological emergency. It accounts for 4% of all encephalitis cases [1], making it the fifth most common cause of encephalitis after Herpes simplex virus, Varicella-zoster virus (VZV), Mycobacterium tuberculosis, and acute disseminated encephalomyelitis. It is the second most common cause of autoimmune encephalitis in children after acute disseminated encephalitis (ADEM) [1]. It is rare in pediatrics, its incidence varies from 0.07 to 0.085 per 100,000 children in Europe [2], but in Africa it remains difficult to diagnose [3,4].

This encephalitis is characterized by neuropsychiatric symptoms and is most often linked to ovarian teratoma in young women [4,5]. The diagnosis is confirmed by the detection of an antibody directed against a central nervous system (CNS) antigen, either against an intracellular antigen or against a membrane antigen, particularly anti-N-Methyl-D-Aspartate receptor antibodies in the cerebrospinal fluid [6].

Treatment is based on corticosteroid therapy and the administration of intravenous immunoglobulin as first-line treatment, followed by plasmapheresis and, as a third-line treatment, rutiximab [2].

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The outcome is favorable if diagnosis and treatment are early, but the prognosis is sometimes guarded, with the possibility of cognitive sequelae [2,7].

Therefore, we will describe our experience with three pediatric cases hospitalized in our facility, in order to clarify the circumstances of their onset, the various clinical signs, and finally, to highlight the diagnostic and management challenges we encountered in our setting.

2. Clinical Observations

2.1. Case 1

This was a 9-year-old boy, with no significant past medical or surgical history, the third of three children, with normal psychomotor development until then. He was admitted to our department on February 17, 2024, for investigation and management of generalized tonic-clonic seizures associated with psychomotor agitation and abnormal choreic movements with orofacial dyskinesia and lower limb dystonia with a flabbing gait in both hands, all occurring in a febrile context.

The examination upon admission revealed an altered state of consciousness with a pediatric Glasgow Coma Scale score of 13/15, regression of psychomotor skills, motor deficits, and cognitive impairment. The child no longer recognized his mother or those around him and did not respond to his name.

Faced with this symptomatology we initially considered severe malaria, neurological form, which we ruled out by a thick blood smear test which came back negative, then a viral infectious encephalitis, probably herpetic, and finally an autoimmune encephalitis.

We performed a complete blood count (CBC) which revealed a white blood cell count of 120,000/mm^3, hemoglobin level of 10 g/dL, platelet count of 350,000/mm^3, a negative thick blood smear, and a C-reactive protein (CRP) level of 47 mg/dL.

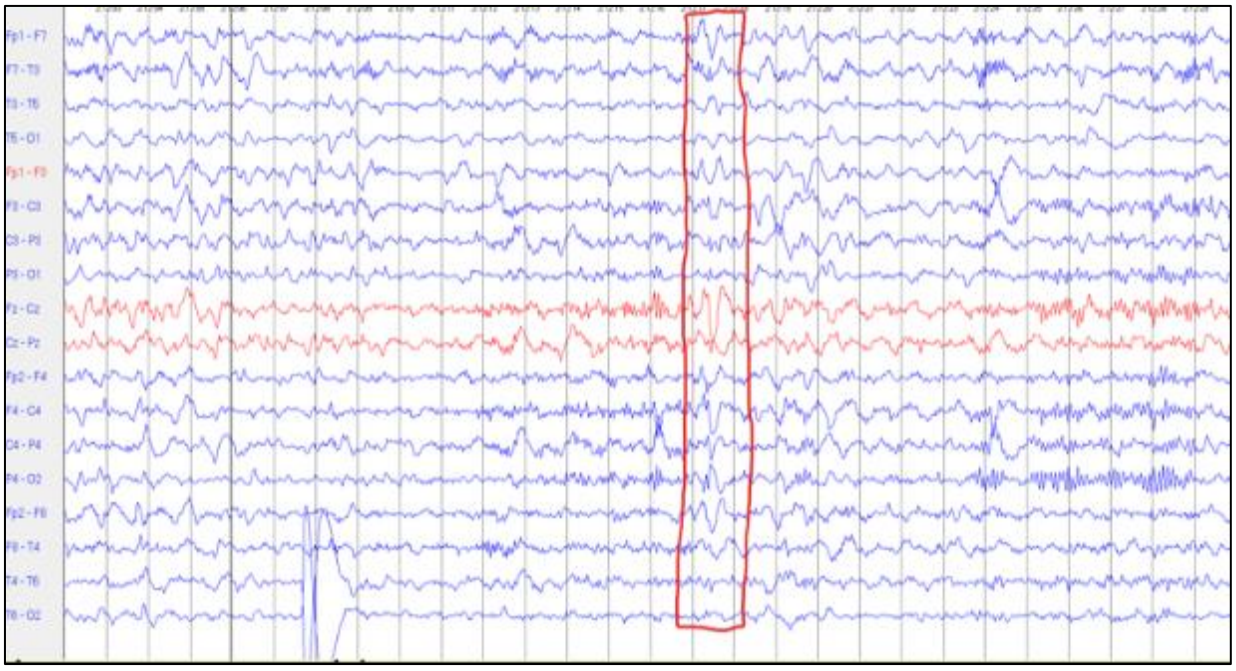

Figure 1 EEG Wakefulness/Sleep: presence of abnormalities in the fronto-central regions

A lumbar puncture was performed and revealed clear, normal-tension cerebrospinal fluid. Cytological and biochemical analysis showed a normal cerebrospinal fluid protein level, cytology with 4 leukocytes/mm^3 and a predominantly lymphocyte differential. Viral and bacteriological studies were negative.

A brain MRI was performed and returned normal results, but the search for anti-NMDA antibodies in the cerebrospinal fluid (CSF) was positive after 3 weeks, thus supporting a diagnosis of NMDAR encephalitis.

An EEG during wakefulness and sleep (Figure 1) showed a fairly well-organized structure, with paroxysmal abnormalities of frontocentral onset consistent with focal-onset epilepsy.

The patient was prescribed sodium valproate at 30 mg/kg/day (400 mg twice daily), acyclovir 500 mg every 8 hours by slow intravenous infusion, haloperidol at 0.1 mg/kg/day, paracetamol 500 mg every 6 hours if fever was present, and corticosteroids at 2 mg/g/day. Given these results of positive NMDA antibodies in the cerebrospinal fluid (CSF) and blood, the patient was initiated with a rutiximab protocol consisting of one course of 375 mg per unit area of body surface area per week for 4 weeks.

Following this treatment and four courses of Rutiximab, the patient's clinical condition improved significantly after one month, with:

The cessation of abnormal movements and seizures; the patient was awake and aware of his surroundings, following his surroundings with his eyes when his name was called. He understood what was asked of him and sometimes attempted to begin to answer with a few words. He was able to move around with assistance.

2.2. Case 2

This was a 5-year-old girl with no significant personal or family medical history.

She was hospitalized from June 26, 2025, to July 17, 2025, in the pediatric ward for right-sided focal seizures that subsequently generalized in the context of a post-flu, non-febrile episode. The neurological examination on admission revealed: altered level of consciousness with a pediatric Glasgow Coma Scale score of 12/15 and lethargy; focal seizures that subsequently generalized; dystonia of the upper limbs localized to both hands associated with choreic movements of all four limbs; aphasia; and tetraplegia.

A lumbar puncture was performed, which was clear and normal in blood pressure. Cytobacteriological analysis was unremarkable, with normal protein and glucose levels, and cytology showing an absence of leukocytes. Viral testing was not performed, and bacteriological analysis did not identify any pathogens. Anti-NMDA antibodies were positive. Magnetic resonance imaging was normal. An electroencephalogram (EEG) during wakefulness and sleep (Figure 2) revealed diffuse cerebral dysfunction with high-amplitude spike-wave discharges in the right frontal lobe.

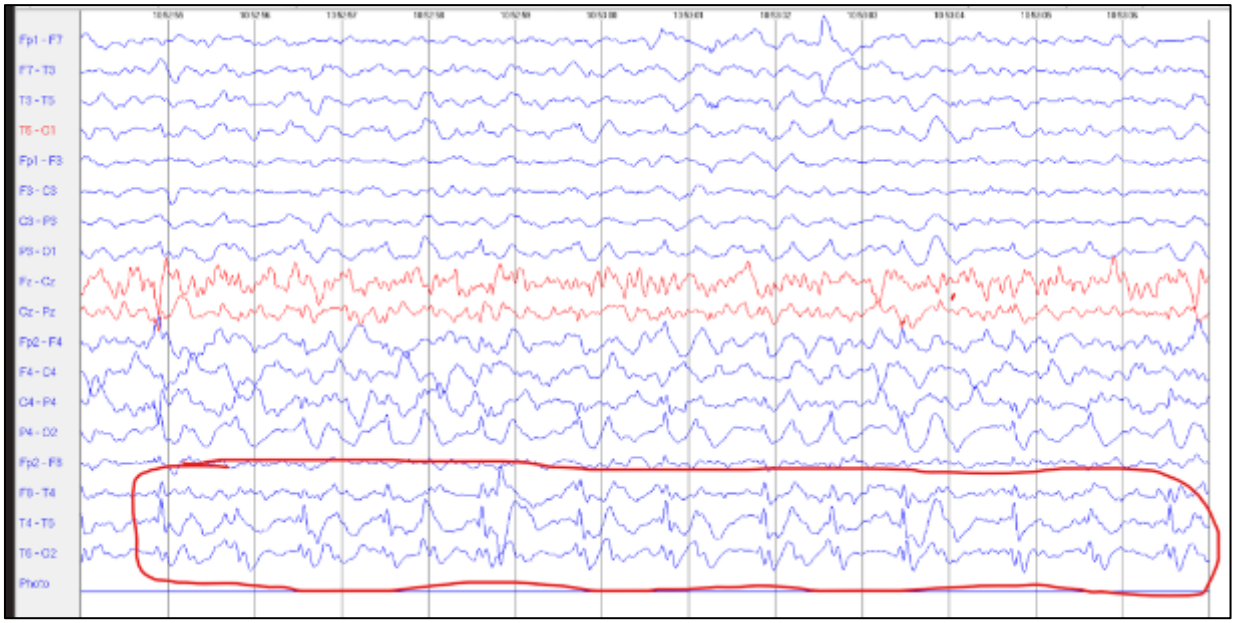

Figure 2 EEG Wakeful/Sleep: diffuse cerebral distress with spike-waves in the right frontal lobe

Based on these results, the diagnosis was NMDAR encephalitis. Treatment consisted of corticosteroid therapy at 2 mg/kg/day as an injectable bolus for 5 days, followed by oral administration. Other treatments included haloperidol drops (6 drops twice daily), oral sodium valproate at 30 mg/kg/day in two divided doses, and an immunosuppressive protocol with rutiximab at a dose of one per week.

The evolution to date is marked by an improvement in motor skills; she walks on her own, orients herself in the house, turns her head when her name is called, executes simple commands, manages to make herself understood by gestures especially for physiological needs, she presents difficulties with fine motor skills, and on the cognitive level persists aphasia, the patient has started psychomotor and speech therapy sessions.

2.3. Case 3

This was a 16-month-old boy who was hospitalized in August 2025 for agitation and generalized tonic seizures with upward gaze following an episode of febrile gastroenteritis. On examination, he presented with irritability, motor restlessness, hemibalism, and a general regression of developmental milestones, including loss of head control, walking, and speech; he was no longer able to say "papa" or "mama." A lumbar puncture was unremarkable, but the serum NMDA antibody test was positive.

A wakeful and sleep EEG (Figure 3) showed a fairly well-organized tracing with a left temporoparietal irritative focus with secondary generalization, associated with bursts that sometimes gave the tracing a fragmented appearance. This appearance is consistent with epileptic encephalopathy.

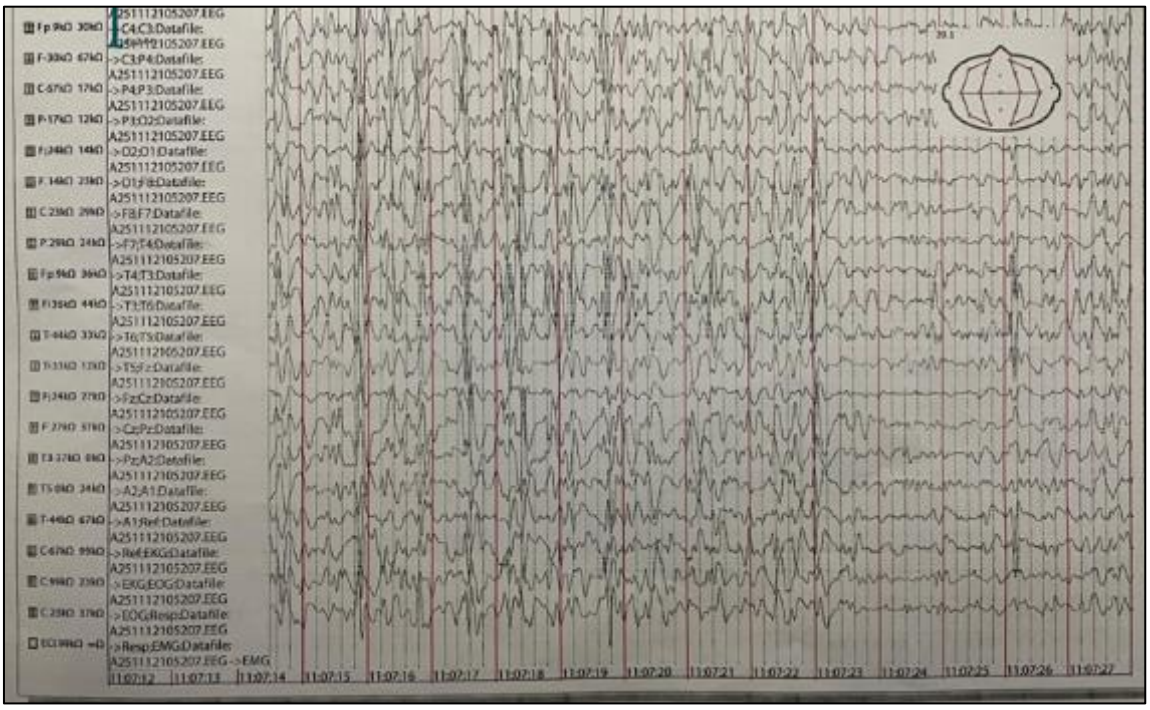

Figure 3 Wakeful/Sleep EEG: presence of pontine-waves in the left temporoparietal with generalization associated with bursts sometimes giving a fragmented appearance to the burst tracing

A brain MRI was normal.

Treatment consisted of corticosteroid therapy at 2 mg/g/day, initially administered as an intravenous bolus for 5 days, followed by oral administration. Other treatments included haloperidol drops: 4 drops twice daily, oral sodium valproate at 30 mg/kg/day in two divided doses, and an immunosuppressant protocol with rutiximab, administered once weekly for 4 weeks.

To date, the child's progress has been marked by improvement in motor and cognitive development. They are learning to sit and stand, have resumed playing, are aware of their surroundings, have good social interaction, recognize their name by eye contact, try to grasp objects, recognize their parents, and say words, although they do not yet say "papa" or "mama."

3. Discussion

NMDAR antibody encephalitis remains relatively uncommon in sub-Saharan Africa, particularly in Gabon. It is most often associated with ovarian teratoma in young women or is responsible for a psychiatric syndrome [4, 5].

The diagnostic criteria for NMDAR antibody encephalitis are based primarily on:

The main symptoms, which are: behavioral disturbances, cognitive dysfunction, seizures, and abnormal movements, most often orofacial with dyskinesias, present in 80% of cases [1, 2]. EEG findings and abnormalities in CSF analysis are also considered [5]. These criteria are found in the literature [3, 6, 7, 8] as in our clinical case.

Paraclinical examinations are of limited value. Brain MRI is useful and can reveal abnormalities in patients with anti-NMDAR antibody encephalitis, but in our study, the brain MRI was normal, a finding confirmed by some authors who have reported abnormal MRI findings in 31% of cases [1,2]. EEG abnormalities are frequent, as in all cases in our bibliography and in the literature [3,6,7,8]. Diagnostic delay in our setting was a major problem because, in our cases, the cost of brain MRI and anti-NMDA receptor antibodies, along with the average three-week wait for confirmation of antibody presence in the CSF, is responsible for the diagnostic delay in our setting and explains, on the one hand, why this pathology is underdiagnosed in Africa, as in our case and in the literature [3,4]. On the other hand, the very high cost of treatment further reinforces and justifies the hypothesis put forward earlier.

The occurrence of complications such as cognitive and motor sequelae, as observed in the case of observation 2 who is recovering with difficulty, makes this entity a diagnostic and therapeutic emergency.

4. Conclusion

Anti-NMDAR antibody encephalitis in pediatric patients is a rare but serious disease and constitutes an emergency.

Early diagnosis is crucial and is based on symptoms, cerebrospinal fluid (CSF) analysis, and the detection of antibodies in the CSF. Treatment often involves immunomodulators and specialized care.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflicts of interest.

Statement of ethical approval

This work was conducted in accordance with the recommendations of the Declaration of Helsinki regarding medical research involving human subjects, in order to protect the privacy and confidentiality of personal information concerning the patients involved in our research. Ethical approval was obtained

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

Author contributions

All authors contributed to the writing of this manuscript and read and approved the final version.

References

[1] Graus F, Titulaer MJ, Balu R, Benseler S, Bien CG, Cellucci T et al. A clinical approach to diagnosis of auto immune encephalitis. Lancet Neurol 2016; 15: 391-404.

[2] Lamblin F, Joubert B, Honnorat. Current developments in the diagnosis and treatment of autoimmune encephalitis. Elsevier 2023: 1-21.

[3] Mansou A, Djoman A, Gro Bi A, Djiv Ohessoun A, N'gatta P, Kamara I et al. Encephalitis A Anti-Nmdar Antibodies: A pediatric case report in a resource-limited country. Dakar Med 2024; 68(3):275-277.

[4] Sd Tchameni C, Samr H, nna A, Amadou, Fgum B, Ngo Pambo C J R, Bâ H. Autoimmune encephalitis with anti-NMDA receptor antibodies associated with an ovarian teratoma: a case report. Health Research Afr 2025 3(8):91-94

[5] Marsters C, Iskhakova S, Powe L, Budhram A. First psychotic episode caused by anti-N-methyl-D-aspartate receptor antibody encephalitis. JAMC 2023; 195 (23): 823-826.

[6] Moualek D, Mouhouche L, Hechan N, Bouakaz I. Autoimmune encephalitis with anti-NMDAR and anti-VGKC antibodies: Study of 2 cases. Sante-dzcom 2020;11-27.

[7] Beniacine Z, Bicha Z, Bouderda Z. NMDA Antibody Encephalitis in an Infant: A Case Report. Rev Med Alg 2019; 4 (17): 54-57.

[8] Azoum M Ben, Tatencloux S, Deva K, Blanc P. Two pediatric cases of autoimmune encephalitis with anti-NMDA receptor antigen. Arch Ped. 2014: 1-4.