PEDIATRIC NEUROLOGY BULLETIN
OFFICAL BULLETIN OF AOPN

Email : iapneurologybulletin@gmail.com | www.iapneurologyindia.com

Vol-10 | Dr. Varsha Vaidya, Editor & Neuro Bulletin Incharge | Dr. Amarjeet Wagh, Chief Editor | Dr. Jasodhara Chaudhuri, Editor | December 2025



Dr. Jitendra Sahu
Chairperson AOPN

Greetings from the AOPN Chair desk, the current theme of the neuro bulletin "Auto immune neurological disorders" is exciting and contextual. It is important because all pediatiricians and pedicatric neurologists involved in the care of children with neurological problems should be aware of advances in neuroim-munology . Timely and apt management of these disorders can significantly impact outcomes in these disorders. I appreciate the special efforts of Dr. Varsha Vaidya for the successful release of the Neurobulletin periodically. I must thank editor in chief Dr Amarjeet Wagh & editor Dr. Jashodhara Chaudhuri for this academic activity



Dr. Vinit Wankhede
Secretary, AOPN

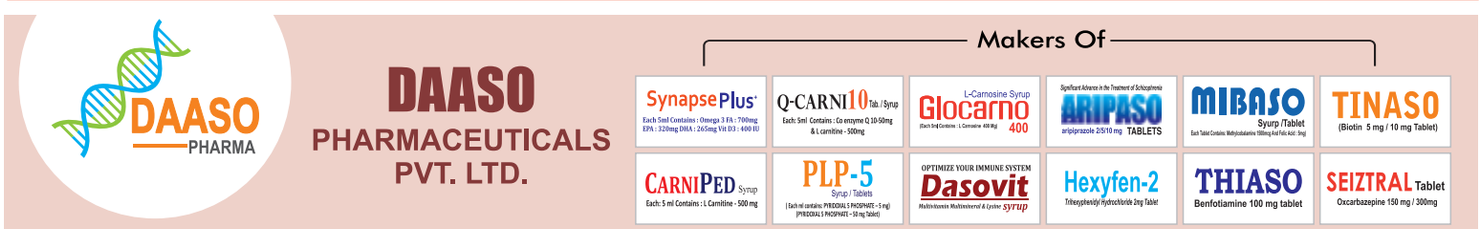
Dear Esteemed Members of AOPN, It is my pleasure to present to you the latest issue of the Neuro Bulletin, a quarterly publication of the Academy of Pediatric Neurology (AOPN). This issue focuses on the important topic of Autoimmune Neurological Disorders, a group of conditions that are increasingly being recognized in children and adolescents. Autoimmune neurological disorders can have a significant impact on the quality of life of affected children and their families. Early recognition and treatment can make a significant difference in outcomes, and it is essential that pediatric neurologists and other healthcare professionals are aware of the latest developments in this field. I hope you find this issue of the Neuro Bulletin informative and useful in your practice. I would like to thank the editorial team especially Dr Amarjeet Wagh, Dr Varsha Vaidya Dr Jasodhara and the contributors for their hard work in bringing out this issue. On behalf of Central AOPN, I am proud to welcome you to 24th Annual National Conference of AOPN at Guwahati Warm regards, Dr. Vinit Wankhede, Secretary General, Central AOPN

AUTO IMMUNE ENCEPHALITIS PATHOPHYSIOLOGY

Dr Jayshree Mahigh, Asst. Professor, KIMS Hospital, Bhubaneswar

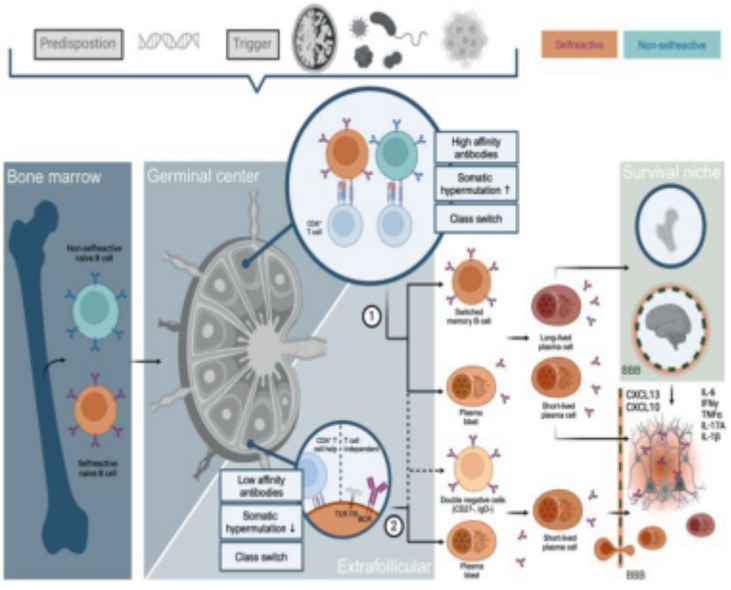
Auto immune Encephalitis is a group of disorders where our immune system target our own neuronal and glial surface antigens in the CNS and rarely the intra cellular antigens.

Pediatric Auto immune encephalitis(AIE) are commonly caused by the auto antibodies against the neuronal antigens- most common includes- against NMDA, LGI1 and CASPR2, while the auto antibodies against the glial antigens include AQP4, GFAP, MOG, GAD. Some less common neuronal antigens include GABAAR, GABABR, IgLON5.The intra cellular antigens like Hu, Ma2, Ri are targeted primarily by auto reactive T cells, especially in the adult population and are usually associated with solid organ tumors carrying a poorer prognosis. Thus, the knowledge and understanding of the pathophysiology of Auto Immune Encephalitis is of utmost importance for its accurate treatment and prognostication.



B cell autoreactivity is counter selected by central and peripheral tolerance mechanisms to prevent autoimmune responses. In the bone marrow, *central tolerance* eliminates self-reactive B cells through three primary processes: clonal deletion, receptor editing , and induction of anergy. *Clonal deletion* removes highly autoreactive B cells via apoptosis. *Receptor editing* involves secondary recombination to generate non-autoreactive B cell receptors (BCRs). *Anergy induction* renders autoreactive B cells biochemically and genetically less responsive to activation . Despite central tolerance mechanisms, approximately 20 % of autoreactive B cells escape elimination .

Mature naïve B cells migrate to secondary lymphoid organs where several *peripheral tolerance* mechanisms exist to mitigate potential autoimmune reactions. These peripheral checkpoints include the absence of co-stimulatory, pro-inflammatory, or survival signals, as well as active suppression byregulatory T Cells (Tregs). Germinal Centres (GCs)within secondary lymphoid organs are the location where B cells undergo clonal expansion and Somatic Hypermutation (SHM).Ultimately, affinity-matured and class-switched B cells leave the GC either as plasmablasts or memory B cells. To prevent auto reactivity, GCs employ at least two primary *peripheral tolerance* checkpoints. Negative selection eliminates B cells that strongly recognize self-antigens.



Several key observations have suggested that autoantibodies in AIE originate from GC reactions. **First,** histopathological studies of teratomas from patients with Anti NMDA receptor encephalitis (NMDARE) frequently exhibit dense lymphocytic infiltrates, comprising T and B cells surrounding neuronal tissue, resembling GCs. These histologic findings suggest that NMDARE teratomas serve as ectopic sites with GC-like activity. **Second,** class switch recombination (CSR) is a process traditionally associated with GCs . In NMDARE, the predominant IgG subclasses are IgG1, IgG2, and IgG3, whereas IgG4 is absent. In contrast, autoantibodies associated with CASPR2 and LGI1 encephalitis are typically of the IgG4 subclass . This observation is linked to B cells being repeatedly engaged in GC reactions rather than being activated by a single immunization event. **Third,** SHMs are considered a hallmark of GC activity. Evidence shows that autoantibodies derived from the B cells or antibody secreting cells (ASC) in the CSF of NMDAR, LGI1 and CASPR2 encephalitis patients frequently carry SHM. **Lastly,** genetic studies that indicate that some forms of AIE, especially LGI1 encephalitis, are strongly (90%) associated with certain HLA class II genes- HLA-DRB1 * 07:01. . The association of disease with MHC II encoding genes suggests T cell dependent B cell maturation and antibody production, while this association in NMDARE have not been seen.

Extrafollicular pathway provides a rapid, short lasting antibody response. This rapid response is crucial in early defence but is also implicated in the development of autoantibodies in autoimmune conditions. For autoantibodies to exert their pathogenic effects on targets within the CNS, they must cross brain barriers. The CNS, long considered an 'immune-privileged' site, is now understood to actively regulate immune cell passage through barriers (blood-brain barrier (BBB). In many cases of AIE, the BBB remains intact, indicating that B cells and ASC actively infiltrate the CNS and produce autoantibodies locally as well.

Cytokine profiles provide insights into T cell involvement in AIE. Elevated TNF-α and interferon-γ , IL-6 levels in NMDARE indicate Th1 and Th17 involvement, while in LGI1 encephalitis, Th17-associated cytokines are less frequently elevated. Currently, **T cells** are more and more recognized in supporting B cell maturation and promoting inflammation in AIE except in paraneoplastic auto immunity especially in the adult population where T cells produce auto immunity against cytoplasmic/ nuclear antigens like Hu, Ma, Ri.

Microglia, the resident immune cells of the CNS contribute to neuroinflammation, through Chemokine secretion.

Triggers-Three primary hypotheses on how A) Viruses trigger autoimmune response in AIE have been proposed.

Molecular mimicry

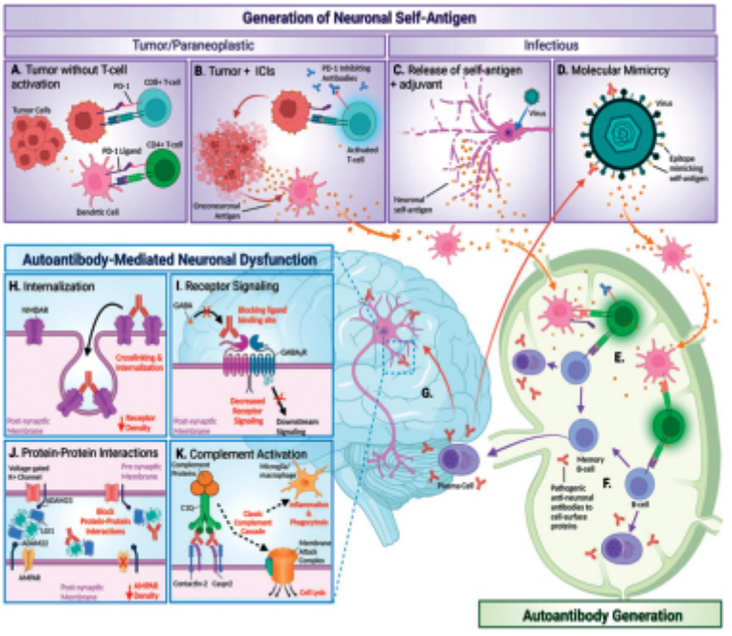
1) This mechanism occurs when viral proteins share structural similarities with self-antigens, potentially inducing cross-reactive autoantibodies.

2) Antigen exposure through tissue damage: HSV-1 infection may cause direct neuronal damage and disrupt the BBB, exposing previously hidden NMDAR antigens

3) Polyclonal B Cell Activation: During certain viral infections, B cells can become non-specifically activated.

B) Tumors- Ovarian teratomas (OTs) are found in 30–40 % of NMDARE cases. Autoantibody development might involve tumor-intrinsic factors (e.g., abnormal neuroglial features or NMDA receptor composition). Some AIE, including $GABA_AR$, AMPAR, LGI1 and CASPR2 encephalitis can have underlying thymomas, though no such link has been described for NMDARE. The thymus is central to the negative selection of autoreactive T cells and this mechanism can be disturbed by thymomas, with altered presentation of self-antigens, allowing autoreactive $CD4^+$ T cells to evade tolerance checkpoints.

C) The genetic risk factors of AIE extend beyond HLA associations. In NMDARE and LGI1 risk loci were found in B cell development , pro-inflammatory Cytokine production pathways of microglia and astrocytes associated with BBB integrity.



CLINICAL FEATURES OF AUTOIMMUNE ENCEPHALITIS

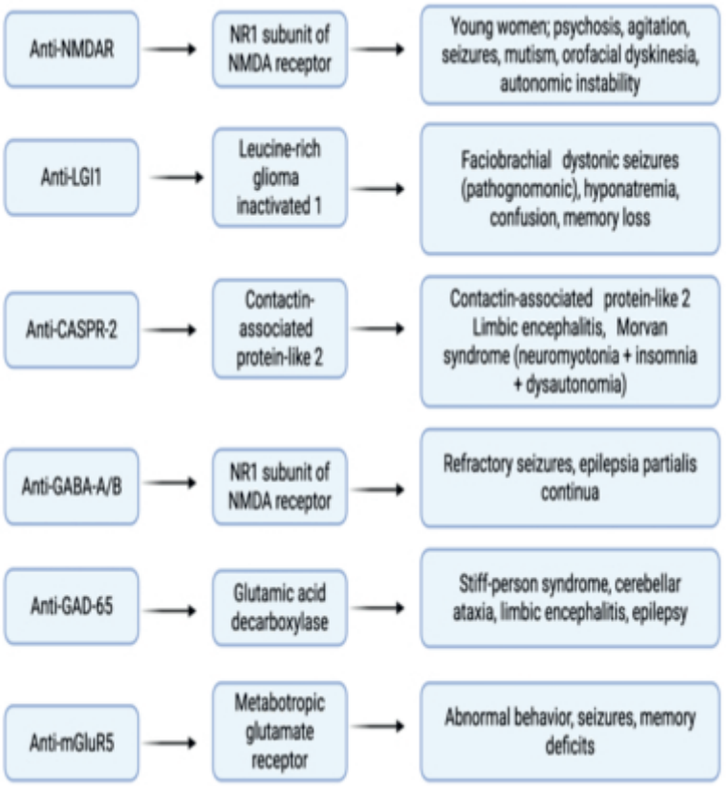
Dr. Rallapalli Anvitha DM Pediatric Neurology fellow, Department of Pediatrics, PGIMER, Chandigarh.
, Dr. Arushi G Saini Additional Professor, Pediatric Neurology, Department of Pediatrics, PGIMER, Chandigarh.

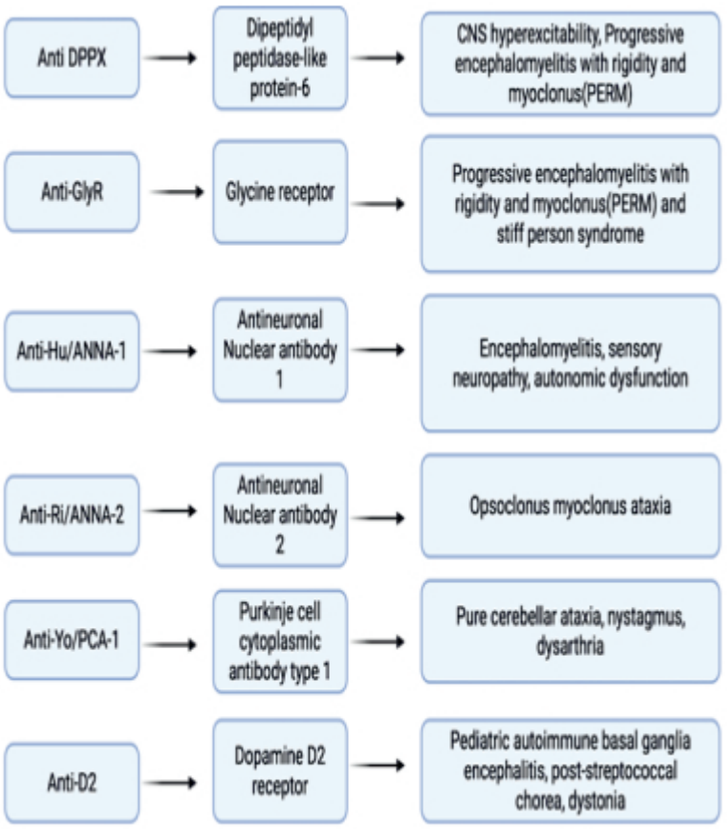
- Autoimmune encephalitis in children presents as a subacute, polysymptomatic encephalopathy dominated by behavioral change, seizures and movement disorders.
- Autoimmune encephalitis in children typically evolves over days to weeks and can mimic infectious or primary psychiatric disease.
- Presentation and severity vary by age and antibody subtype, with many forms responding to immunotherapy.
- Seizures are a hallmark and often the initial and the most common manifestation occurring in 80% of children. Seizures can vary from focal and focal-to-bilateral seizures to generalized seizures, seizure clusters, status epilepticus, and epilepsiapartialis continua. Psychiatric and behavioral disturbances are frequent nearly 50% of patients. It includes agitation, psychosis, mood instability, aggression, poor academic performance, and catatonia, often evolving rapidly following a brief prodromal phase.
- Some degree of cognitive impairment is seen in majority of patients and is considered a cardinal symptom.
- Movement abnormalities are heterogenous occur in one third patients and may present as dyskinesias, chorea, dystonia, ataxia, tremor, or repetitive orofacial stereotypies.
- The course of the disease is predictable and evolves in stages.
- Older children and adolescents tend to manifest more pronounced psychiatric symptoms, depression, and memory impairment, which may mimic a primary psychiatric illness along with autonomic symptoms. Autonomic features include hypoventilation, hypotension or bradycardia. After

few weeks, altered sensorium, seizures and movements disorder appear. In adolescent girls, evaluation for an underlying tumor, particularly an ovarian teratoma, is essential.

In contrast, in younger children, the presentation is usually dominated by flurry of seizures and movement disorder along with behavioral changes such as irritability and autistic like regression.

Different antibody subtypes can have characteristic presentations.(1)(2)





Autoimmune encephalitis should be suspected in a child with acute to subacute presentation of varied symptoms such as seizures, encephalopathy, behavioral abnormalities and movement disorders. Different clinical phenomenology can predict the antibody type even though NMDAR encephalitis is the most common in children.

Investigations in Autoimmune Encephalitis in Children: A Review

Dr. Alpana Kondekar, Dr. Snehal Shinde Affiliation: Professor(Additional), Department of Pediatrics, T.N. Medical College & B.Y.L. Nair Hospital, Mumbai

Autoimmune encephalitis (AE) is an important and treatable cause of acute encephalitis. Diagnosis of AE in a developing child is challenging because of overlap in clinical presentations with other diseases and complexity of normal behavior changes. Early identification is crucial as AE is potentially reversible with prompt immunotherapy. The investigation of AE relies on a combination of clinical suspicion and multi-modal testing, including cerebrospinal fluid (CSF) analysis, neuroimaging, electroencephalography (EEG), and antibody profiling.

Approach to Investigations in Pediatric Autoim-mune Encephalitis (AE)

1. Clinical Suspicion and Initial Assessment

Autoimmune encephalitis should be suspected in a child presenting with:

- Subacute onset (<3 months) of encephalopathy, seizures, movement disorders, behavioral or psychiatric changes, or developmental regression.
- Associated with neurologic dysfunction (altered mental status, cognitive decline, abnormal movements, speech disturbance, sleep problems, autonomic instability).

Table 1. Proposed classification criteria for possible, definite antibody-positive and probable antibody-negative pediatric AE

Categorical features of AE	Specific diagnostic features	Possible AE	Probable antibody-negative AE	Definite antibody-positive AE
1. Evidence of acute or subacute symptom onset	Onset of neurological and/or psychiatric symptoms over ≤3 months in a previously healthy child	Yes	Yes	Yes
2. Clinical evidence of neurologic dysfunction	Features include: • Altered mental status/level of consciousness or EEG with slowing or epileptiform activity (focal or generalized) • Focal neurologic deficits • Cognitive difficulties† • Acute developmental regression • Movement disorder (except tics) • Psychiatric symptoms • Seizures not explained by a previously known seizure disorder or other condition	≥2 features present	≥2 features present	≥2 features present
3. Paraclinical evidence of neuroinflammation	Features include: • CSF inflammatory changes (leukocytosis>5 cells/mm^3 and/or oligoclonal bands) • MRI features of encephalitis • Brain biopsy showing inflammatory infiltrates and excluding other disorders	Not available	=1 features present	=1 features present
4. AE serology	Presence in serum and/or CSF of well-characterized autoantibodies associated with AE	No	No	Yes
5. Exclusion of other etiologies	Reasonable exclusion of alternative causes, including other causes of CNS inflammation	Yes	Yes	Yes

Table 2. Tiered Approach to Testing

Tier	Timing / Context	Tests Included
Tier 1: Initial workup (urgent)	At presentation of acute/subacute encephalopathy	MRI brain ± spine, EEG, CSF (cells, protein, OCB), basic labs, infectious workup
Tier 2: Autoimmune workup	Once infection excluded	Serum and CSF neuronal antibody panels, thyroid antibodies, ANA
Tier 3: Extended evaluation	If atypical, refractory, or relapsing	PET/SPECT, tumor screening, repeat antibody testing, neuropsychological evaluation

Table 3. **Stepwise Diagnostic Approach for suspected AE**

Step	Investigation Type	Purpose / Key Findings	Typical Findings in AE
A. Neuroimaging	**MRI brain (± spine) with contrast**	To identify inflammatory or demyelinating lesions; rule out structural mimics	T2/FLAIR hyperintensities in limbic system, basal ganglia, or cortex; may be normal early.Normal MRI is seen in up to 50% of anti-NMDA receptor AE. It may show changes after autoimmune encephalitis a few days later.
	Functional imaging (PET/SPECT)	Assess brain metabolism and inflammation when MRI is normal FDG-PET can detect focal or multifocal brain abnormality.	It can detect brain abnormalities in earlier stages of disease; it was found to be more sensitive than MRI brain. Bilateral temporal lobe hypermetabolism and bilateral occipito-parietal hypometabolism are the most common patterns seen.
B. CSF analysis (lumbar puncture)	**Cell count, protein, glucose, oligoclonal bands**	CSF analysis should include- total cell count with differential, proteins, glucose level, CSF/serum glucose ratio, IgG index, oligoclonal bands, viral panel studies including HSV ½ PCR, varicella zoster PCR, bacterial/fungal cultures, cytology, flow cytometry, neuronal autoantibody panel.	CSF findings in autoimmune encephalitis include lymphocytic pleocytosis(20-200cells but can be as high as 900 cells), elevated CSF Proteins, elevated IgG index and positive intrathecal oligoclonal bands.
	CSF antibody testing	Detection of neuronal surface or intracellular antibodies	Often positive for NMDAR, GABA_A/B, MOG, etc. ******Refer to table no 3,4for the details**
C. Blood investigations	**Basic labs**	Rule out metabolic or systemic causes	CBC, ESR/CRP, electrolytes, liver/renal function
	Autoimmune panel	Assess for systemic autoimmunity	ANA, anti-thyroid, anti-dsDNA, complement levels
	Serum neuronal antibody panel	Detect AE-related antibodies	Anti-NMDAR, MOG, LGI1, CASPR2, D2R, etc. (interpret with caution; serum-only positivity may be nonspecific)

Step	Investigation Type	Purpose / Key Findings	Typical Findings in AE
D. EEG	**Standard or prolonged video EEG**	To assess encephalopathy and seizure activity	Generalized or focal slowing, epileptiform discharges, "delta-brush" pattern in NMDAR-AE
E. Infection screening	**PCR and serology**	Exclude infectious encephalitis (e.g., HSV, EBV, VZV, Mycoplasma)	Negative results help strengthen AE diagnosis
F. Tumor screening (if indicated)	**Ultrasound, MRI, CT, or PET**	Identify paraneoplastic source (rare in children but possible in adolescents)	Ovarian teratoma (NMDAR), thymoma, germ cell tumor
G. Neurocognitive and psychiatric assessment	**Formal testing**	Evaluate cognitive, memory, and behavioural impact	Variable deficits depending on antibody and brain regions involved
H. Brain Biopsy	**Invasive**	biopsy may be needed for mass lesion to rule out neoplastic etiology	Pathological findings in autoimmune encephalitis is non-specific, includes T-cell and or B-cell perivascular and parenchymal infiltrates with secondary gliosis

Supportive and Follow-Up Investigations

- Serial MRI and EEG to monitor response to immunotherapy.
- Neurocognitive and psychiatric follow-up for rehabilitation and academic planning.
- Consider immune status and vaccine review before long-term immunosuppression.

TABLE 4. Common Antibodies Tested in Autoimmune Encephalitis

Antibody	Target Antigen / Receptor	Preferred Sample	Key Features / Associations in Children
Anti-NMDA receptor (NMDAR)	NR1 subunit	**CSF > Serum**	Most common in children; psychiatric symptoms, seizures, dyskinesias; ovarian teratoma rare in children.
Anti-GABA_A receptor	GABA_A receptor	**CSF**	Multifocal seizures, MRI with cortical/subcortical lesions.
Anti-GABA_B receptor	GABA_B receptor	**Serum + CSF**	Limbic encephalitis, often paraneoplastic in adults (rare in children).
Anti-AMPA receptor	AMPA receptor (GluR1/2)	**Serum ± CSF**	Rare in children; memory loss, seizures.
Anti-mGluR5	Metabotropic glutamate receptor 5	**Serum ± CSF**	Rare; "Ophelia syndrome," Hodgkin lymphoma (adults).

Anti-Glycine receptor (GlyR)	Glycine receptor α1	**Serum ± CSF**	Progressive encephalomyelitis with rigidity/myoclonus (PERM).
Anti-DPPX	Dipeptidyl-peptidase–like protein 6	**Serum**	CNS hyperexcitability, diarrhea, weight loss; rare in children.
Anti-LGI1	Leucine-rich glioma-inactivated 1	**Serum > CSF**	Rare in children; faciobrachial dystonic seizures.
Anti-CASPR2	Contactin-associated protein 2	**Serum > CSF**	Peripheral nerve hyperexcitability, rarely pediatric.
Anti-MOG	Myelin oligodendrocyte glycoprotein	**Serum**	Demyelinating phenotypes (ADEM, seizures, cortical encephalitis).
Anti-GFAP	Glial fibrillary acidic protein	**CSF ± Serum**	Meningoencephalomyelitis, optic disc swelling, ataxia.
Anti-AQP4	Aquaporin-4	**Serum**	NMOSD; sometimes overlaps with AE-like presentations.
Anti-AMPA/NMDA dual positivity	—	**CSF + Serum**	May indicate broad autoimmune response or overlapping syndromes.

Interpretation Principles

- **CSF testing is more sensitive** for many antibodies (e.g., NMDAR, GABA_A); always perform both serum and CSF testing.
- **Negative antibody testing does not exclude AE —** diagnosis is primarily clinical supported by paraclinical evidence.
- **Correlation is key:** Always interpret antibody results in the context of clinical, MRI, EEG, and CSF findings.
- **Repeat testing** may be necessary if suspicion remains high with initial negative results.

TABLE 5. Pitfalls and interpretation tips of serum and CSF antibody titres in AE

Issue	Explanation / Example
Serum-only low-titer antibodies	May represent non-specific binding; interpret cautiously without supportive clinical findings.
CSF-negative but serum-positive LGI1/CASPR2	Still valid; these are typically peripheral in origin.
CSF-only NMDAR antibody	Common — do not exclude AE if serum negative.
Coexisting MOG or AQP4 antibodies	May suggest mixed demyelinating-autoimmune encephalitic spectrum.
Post-infectious false positives	Occasionally seen after viral encephalitis (e.g., HSV).

MANAGEMENT STRATEGIES IN PEDIATRIC AUTOIMMUNE NEUROLOGICAL CONDITIONS

Dr Juhi Gupta, Assistant Professor (Pediatric Neurology), SMS Medical College, Jaipur, India
Dr Tanya Sood, Senior Resident, Pediatrics, JLN Medical College, Ajmer, India

Autoimmune neurological diseases involve immune-mediated injury to the central or peripheral nervous system, driven by autoreactive antibodies or cytokines targeting neuronal or glial antigens.Autoimmune neurological disorders affecting the central nervous system (CNS) includes infection-triggered encephalopathy syndromes (ITES), autoimmune encephalitis, demyelination syndromes including acute disseminated encephalomyelitis (ADEM), neuromyelitis optica spectrum disorder (NMO-SD), MOG antibody-associated disease (MOGAD), and multiple sclerosis (MS). Autoimmune peripheral nervous system (PNS) disorders include Guillain Barre Syndrome (GBS), chronic inflammatory demyelinating polyneuropathy (CIDP), myasthenia gravis, etc. Immunomodulation also has a role in certain other neurological disorders such as paediatric acute neuropsychiatric syndrome (PANS), opsoclonus myoclonus ataxia (OMA), etc. The current article focuses on management of autoimmune encephalitis (figure 1) and demyelinating disorders.

1. Acute Phase: First-line Immunotherapy

Prompt initiation of therapy is crucial to limit irreversible neuronal damage. First-line immunotherapy should be offered to all children with autoimmune neurological disorders, unless they are back to baseline at the time of diagnosis.

- **High-dose corticosteroids –** suppress transcription of proinflammatory cytokines and restore blood–brain barrier integrity. Usually given as intravenous methyl-prednisolone pulse (IVMP) therapy at 30mg/kg/day (up to 1gram/day) for 3-5 days followed by oral steroid taper. Oral steroid taper duration depends on the severity, response and adverse reactions.
- **Intravenous immunoglobulin (IVIg) –** modulates complement activation, neutralizes autoantibodies, and interferes with Fc receptor-mediated injury. IVIg is given at 2 gram/kg over 2-5 days. For autoimmune encephalitis, IVIg should be the part of first-line immunotherapy, especially in severe cases. In certain conditions like MOGAD, IVIG is used monthly to prevent recurrence.
- **Therapeutic plasma exchange (TPE) –** removes circulating autoantibodies and immune complexes while resetting immune homeostasis. TPE should be strongly considered in severe cases of autoimmune encephalitis and demyelinating disorders especially NMO-SD if no improvement is seen on IV steroids. TPE should precede IVIg if both are being used.

2. **Second-line Immunotherapy**

For children who fail to improve approximately two weeks after initiation of two or more first-line immunotherapy, should be offered second-line immunosuppression.

- **Rituximab-** It is the second-line immunotherapy of choice for autoimmune encephalitis.
- **Cyclophosphamide-** It is another effective second-line therapy, which is considered if rituximab is not available or is contra-indicated.
- **Tocilizumab –** This IL-6 inhibitor should be used only in most refractory cases of autoimmune encephalitis who have failed to respond to rituximab and/or cyclophosphamide after 1-3 months.
- In multiple sclerosis, various disease modifying therapies are used depending upon availability, affordability and adverse effect profile (Table 1).

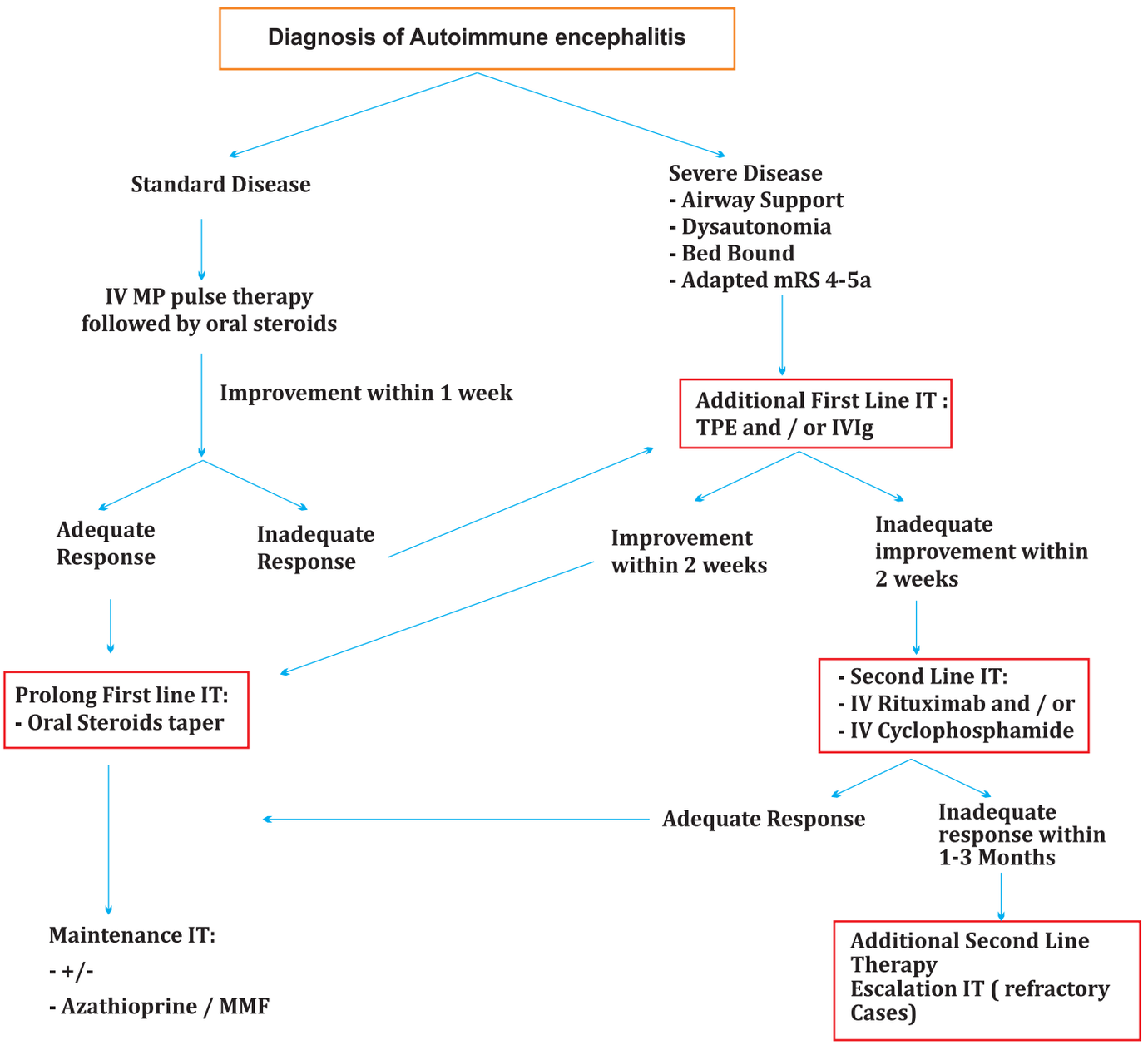
3. **Maintenance and Long-Term Management**

Once disease activity is controlled, long-term immunomodulation with oral drugs like azathioprine or mycophenolate mofetil (MMF) aims to prevent relapse and promote neural recovery. Clinical and radiological monitoring helps balance disease control with minimizing drug-related toxicity.

Supportive therapy—including physiotherapy, seizure control, and cognitive rehabilitation remains essential to improve quality of life. Multidisciplinary co-ordination among neurologists, immunologists and rehabilitation teams enhances outcomes.

In conclusion, management of autoimmune neurological disorders revolves around early recognition, rapid immunotherapy and structured long-term care.

Management algorithm for autoimmune encephalitis



DAASO PHARMA

DAASO PHARMACEUTICALS PVT. LTD.

Makers Of

Synapse Plus
Each 5ml Contains : Omega 3 FA : 700mg
EPA : 320mg DHA : 265mg Vit D3 : 400 IU

Hexyfen-2
Trihexyphenidyl Hydrochloride 2mg Tablet

L-Carnosine Syrup
Glocarno 400
(Each 5ml Contains : L Carnosine 400 Mg)

TINASO
(Biotin 5 mg / 10 mg Tablet)

PLP-5
Syrup / Tablets
(Each ml contains: PYRIDOXAL 5 PHOSPHATE – 5 mg)
(PYRIDOXAL 5 PHOSPHATE – 50 mg Tablet)

Q-CARNI10 Tab. / Syrup
Each: 5ml Contains : Co enzyme Q 10-50mg
& L carnitine - 500mg

OPTIMIZE YOUR IMMUNE SYSTEM
Dasovit
Multivitamin Multimineral & Lysine syrup

MIBASO
Syurp /Tablet
Each Tablet Contains: Methylcobalamine 1500mcg And Folic Acid : 5mg

CARNIPED Syrup
Each: 5 ml Contains : L Carnitine - 500 mg

Significant Advance in the Treatment of Schizophrenia
ARIPASO
aripiprazole 2/5/10 mg TABLETS

THIASO
Benfotiamine 100 mg tablet

SEIZTRAL Tablet
Oxcarbazepine 150 mg / 300mg

Table 1: Disease Modifying Drugs for Multiple Sclerosis in Children

Drug	Level of evidence	Mechanism of Action	Administration	Typical Dose / Schedule	Monitoring	Key Side Effects
Natalizumab	Prospective observational study	Anti–α4β1 integrin monoclonal antibody; inhibits T/B-cell migration into CNS	IV	6mg/kg IV monthly; 300 mg per dose max	JC virus antibodies every 6 months	Infections, hypersensitivity, PML
Fingolimod	PARADIGMS clinical trial; FDA/EMA approved (0–17 yrs)	Sphingosine-1-phosphate receptor modulator; prevents lymphocyte migration from lymph nodes	Oral	≤40 kg: 0.25 mg/day; >40 kg: 0.5 mg/day	ECG, eye exam, CBC, LFT	Bradycardia, macular edema,varicella infection, hepatotoxicity, leukopenia
Rituximab	Retrospective observational studies	Anti-CD20 monoclonal antibody; B-cell depletion	IV	375 mg/m² or 500 mg/m² (Max 1000mg/dose) every 6 months (after 2 induction doses; 2 weeks apart)	CBC, LFT, immunoglobulin levels, CD19 levels every 6 months	Infusion reactions, hypogammaglobulinemia, infections
Teriflunomide	TERIKIDS trial; double-blind RCT	Inhibits DHODH → reduces T/B-cell penetration in CNS	Oral	<40 kg: 7 mg/day; ≥40 kg: 14 mg/day	Screen for latent TB, LFT, BP	Alopecia, paresthesia, GI upset, liver toxicity
Interferon β	REPLAY phase 4 study (ongoing); EMA approved >12 yrs	Reduces proinflammatory cytokines; limits immune cell trafficking into CNS	IM / SC	Interferon β1a: 30 µg IM weekly or 22-44 µg SC 3× weekly Interferon β1b: 250 µg SC EOD	CBC, LFT	Flu-like symptoms, injection site reaction, depression, thyroid dysfunction
Glatiramer acetate	ITEMS Study,Retrospective observational study; EMA approved > 12 yrs	Shifts Th1 → Th2 immune profile (anti-inflammatory)	SC	20 mg daily or 40 mg 3× weekly	ECG, LFT	Flushing, tachycardia, hepatotoxicity

Abbreviations: PML: Progressive Multifocal Leucoencephalopathy; FDA: Food& Drug Administration; EMA: European Medical Agency; DHODH: dihydro-orotate dehydrogenase; CNS: Central Nervous System; SC; Subcutaneous; IM: Intramuscular; GI: Gastro-intestinal; LFT: Liver Function Test; CBC: Complete Blood Count; EOD: Every Other Day

IMAGE GALLERY



National neurology cme Shillong



Maha AOPN NPEC Nagpur



EAST module Sambhaji Nagar



Odisha State neuro Pedicon



West Bengal State Neuro Pedicon



Installation ceremony, central AOPN, Trivandrum



Karnataka neuro Pedicon



Tamil Nadu AOP N installation



Hyderabad aopn office, Practice module launch

DAASO PHARMA

Thanking you

Your Academic Partner for Neurobulletin

DAASO PHARMA

Makers Of

Synapse Plus+
Each 5ml Contains : Omega 3 FA : 700mg
EPA : 320mg DHA : 265mg Vit D3 : 400 IU

Hexyfen-2
Trihexyphenidyl Hydrochloride 2mg Tablet

L-Carnosine Syrup
Glocarno 400
(Each 5ml Contains : L Carnosine 400 Mg)

TINASO
(Biotin 5 mg / 10 mg Tablet)

PLP-5
Syrup / Tablets
(Each ml contains: PYRIDOXAL 5 PHOSPHATE – 5 mg)
(PYRIDOXAL 5 PHOSPHATE – 50 mg Tablet)

Q-CARNI10 Tab. / Syrup
Each: 5ml Contains : Co enzyme Q 10-50mg
& L carnitine - 500mg

OPTIMIZE YOUR IMMUNE SYSTEM
Dasovit
Multivitamin Multimineral & Lysine syrup

MIBASO
Syurp /Tablet
Each Tablet Contains: Methylcobalamine 1500mcg And Folic Acid : 5mg)

CARNIPED Syrup
Each: 5 ml Contains : L Carnitine - 500 mg

Significant Advance in the Treatment of Schizophrenia
ARIPASO
aripiprazole 2/5/10 mg TABLETS

THIASO
Benfotiamine 100 mg tablet

SEIZTRAL Tablet
Oxcarbazepine 150 mg / 300mg